IV. ARGUMENTS PRESENTED BY THIRD PARTIES

A. BRAZIL

1. Introduction

4.1 Brazil explains that the proceeding challenges the WTO-consistency of France's 1 January 1997 Decree, which bans the manufacture, processing, sale and possession for sale, importation, exportation, domestic marketing, offer and transfer of all varieties of asbestos fibres and products containing them (the Decree or the ban).[1] The ban has four narrow exceptions that apply where no substitutes exist for chrysotile products. The substitute products that do exist generally are more expensive than chrysotile products. Thus, the ban clearly operates to create a commercial advantage for substitute products. According to Brazil, a ban is the most trade restrictive of measures. Therefore, the justification for any ban must be subject to the strictest scrutiny, especially as applied to a developing country such as Brazil. The ban has ended Brazilian exports of uncontaminated chrysotile to France. In 1994 and 1995, France imported from Brazil 1,100 and 1,500 metric tonnes of uncontaminated chrysotile, respectively. Since the ban took effect in 1997, France has not imported any chrysotile from Brazil.

4.2 According to Brazil, the importance of this proceeding extends far beyond the French ban – the proceeding is a test case. Will other WTO Members be allowed to ban products of developing countries that can be safely used with appropriate, tested precautions simply to appease the public? Modern economies use hundreds of products that present health risks if they are misused, but that present no risks if they are used properly. Uncontaminated chrysotile is one of them; if properly used, uncontaminated chrysotile presents no health risk. Similar products include organic fibres, man-made fibres, benzene, mercury, ammonia, nearly all forms of pesticide, etc. Societies regulate these products to ensure they are used safely so as to protect the health of workers handling them directly and of the general population which is exposed to them indirectly. The same treatment is appropriate for uncontaminated chrysotile. Uncontaminated chrysotile—the only asbestos fibre Brazil mines and exports – is the safest by far of all asbestos fibres. In particular, it is much safer than amphibole, the asbestos responsible for current health problems from past exposure. All of the asbestos that Brazil mines, produces and exports is uncontaminated chrysotile. For this reason, Brazil's chrysotile products are among the safest in the world. The medical explanation for these facts is set forth in detail in a recent bio-persistence study by Dr. David S. Bernstein, an expert in fibre toxicology (indeed, the EC often seeks his expertise on this topic).[2]

4.3 Brazil asserts that the primary issue in this proceeding is not - as the EC would suggest - whether asbestos can be hazardous to human health. It can. Years of misuse and unsafe utilization of the most hazardous form of asbestos – amphibole - have caused significant damage to health. All countries, including Brazil, regret the harm to human health caused by decades of exposure earlier this century to amphibole produced and used worldwide. Brazil understands well the basis of the public outcry, experienced in many countries (including Brazil), that led the French Government to commission the INSERM Report[3] (a study focusing on the health effects of earlier, unsafe uses of amphibole asbestos) and then to ban asbestos. France imposed the ban only one day after INSERM released its Report. The Report was commissioned and released to provide a scientific "cover" for a political decision that had already been taken. However, as a review of the INSERM Report demonstrates, the causes of asbestos-related health problems in France are past uses, especially in the

[1]Decree No. 96-1133, dated 24 December 1996, (J.O. dated 26 December 1996).

[2]David S. Bernstein, *Summary of the Final Reports on the Chrysotile Bio-Persistence Study* (Geneva Switzerland; 2 October 1998).

[3]INSERM, *Effets sur la santé des principaux types d'exposition à l'amiante*, Les Éditions INSERM, Paris, 1997 (INSERM Report).

spraying of brittle amphibole on to fireproof buildings and, until quite recently, warships (flocking). Given the long latency period between exposure to amphibole and the onset of any related diseases, workers who were victims of heavy exposure with virtually no protection 30 years ago are experiencing serious health problems today. The INSERM Report is based on analyses of these workers' health. The INSERM Report does not focus on data from studies of modern uses of chrysotile. Moreover, in the Report, INSERM concedes that it was unable to produce "scientifically certain" conclusions, but could present only an "aid to understanding" based on "plausible, though uncertain, estimates.[4]" Quite simply, the INSERM Report is an inadequate basis for the ban.

4.4 Brazil argues that it has a deep appreciation of the desire - indeed, the need - for the French Government to address public concern and protect public health. Brazil also understands the frustration of being unable to remedy or even mitigate the health consequences of past exposure from unsafe use of amphibole, and the frustration of being unable to take measures to remedy or decrease exposure from flocked amphibole asbestos that is already in French buildings (because disturbing flocking increases exposure). However, when France approved the WTO Agreement, it agreed not to restrict trade merely to appease domestic sentiment, no matter how strong. Brazil cannot accept France's adoption of a politically motivated measure that will neither (i) make those already sick from asbestos exposure healthy; nor (ii) reduce risk to the healthy beyond existing levels of protection guaranteed by modern, controlled uses of chrysotile. As the European Commission recently stated:

> [V]arious national organisations, including the Health and Safety Executive in the United Kingdom, have made very disturbing projections about the numbers of deaths which are likely to be attributable to asbestos over the next few decades. However, it is important to note that these figures relate to past exposures to mixed asbestos types, including the fibres which have already been banned. It would be wrong to use these statistics alone to justify a ban on the marketing and use of chrysotile because such a ban would not lead to a lower risk of exposure for workers to asbestos which is already in place, nor would it reduce the number of deaths which are occurring today as a result of past exposure to asbestos.[5]

4.5 Modern uses of asbestos are or should be limited to chrysotile, which most parties, including INSERM, agree is safer than other forms of asbestos. Moreover, modern uses are or should be confined to products in which the fibres are bonded in a finished product and, thus, cannot escape, e.g., asbestos-cement products.[6] For these and other reasons, modern uses are quite safe; they involve

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[4]See para. 4.30 below.

[5]Official Journal of the European Communities, C 135/108 (14 May 1999) (30 September 1998 answer of Mr. Bangemann to Written Question E-2736/98 of Christine Oddy (PSE)). See also Official Journal of the European Communities, C 13/123 (18 January 1999) (24 July 1998 answer of Mr. Bangemann to Written Question E-1950/98 of Anita Pollack (PSE)) ("[I]t is important to mention that a new ban would not lead to a lower risk of exposure to existing asbestos for workers, nor would it reduce the number of deaths from past exposure to asbestos. Possible contamination from asbestos in existing buildings (e.g. in relation to maintenance activities and asbestos removal operations) will remain an important cause of exposure to workers for many years.").

[6]Brazil notes that in the chrysotile-cement industry, the largest present-day use of chrysotile, the manufacturing process uses a water slurry mixture of chrysotile and cement. No dust or pollution is created during this process. See also American Lung Association, *Asbestos*, pp. 2 and 3 (http://www.lungusa.org/air/envasbestos.html) ("Asbestos is rarely used alone, and it is generally safe when combined with other materials with strong bonding agents. As long as the material remains bonded so that fibers are not released, it poses no health risk."); National Cancer Institute, (1996), p. 3 (http://www.ncih.nih.gov./clinpdq/risk/Questions_and_Answers_About_Asbestos_Exposure.html) ("Asbestos that is bonded into finished products such as walls, tiles, and pipes poses no risk to health as long as it is not damaged or disturbed (for example, by sawing or drilling) in such a way as to release fibers into the air . . .. [N]o fiber type can be considered harmless, and proper safety precautions should always be taken by people working with asbestos.").

exceedingly low levels of exposure (that often do not exceed even the "natural" levels in ambient air). Chrysotile is used in a very wide variety of products. It is used as a flame retardant, to strengthen friction materials (e.g., truck brakes) and to create cement pipes for carrying water that are far less subject to corrosion, cracking and breaking than traditional cement pipes. In most applications, chrysotile is used because it *increases* public safety; thus, using other, less-efficient products in its place often decreases public safety. The use of chrysotile as a fire retardant needs no explanation. However, a discussion of its use in friction materials may be illuminating. Chrysotile is used primarily in truck brake pads, drum brakes and brake blocks to control heat build-up, thus maximizing friction and stopping power. It is the preferred product for this application. As one of the authors of an American Society of Mechanical Engineers (ASME) study commissioned by the EPA concluded:

> (a) The "replacement/substitution of asbestos-based with non-asbestos brake linings will produce grave risks"; and
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> (b) "the expected increase of skid-related highway accidents and resultant traffic deaths would certainly be expected to overshadow any potential health-related benefits of fiber substitution."[7]

4.6 Brazil pleads that chrysotile's numerous public safety benefits - the many contributions it makes to societies around the world - not be ignored in this proceeding, as they were when France passed its ban. In Brazil's view, the primary question in this proceeding is quite narrow - is a complete ban necessary to protect public health or can public health be ensured by regulating modern, controlled uses of chrysotile and chrysotile products? The answer arrived at by those countries in the Americas that have examined the issue closely, ranging south from Canada, to the United States, to Brazil, is that public health can be ensured by regulating modern controlled uses. France may, of course, take measures that are designed to, and actually do, protect its citizens. However, the ban does not meet even this very generous characterization of the general rule set forth in the WTO Agreement on Technical Barriers to Trade (the TBT Agreement). France must not be allowed to impose a ban on imports and safe, modern uses of chrysotile as a response to public pressure. That the ban does not apply to man-made fibres produced in France, which the available scientific data show present greater risks when their use is not controlled and which have not been proven safer, confirms that the basis for the ban may be political and economic, but is *not* scientific or medical.

4.7 Brazil argues that in many respects, the French reaction is identical to that of the United States Environmental Protection Agency (the EPA) promulgated in 1989, when it banned asbestos under pressure from panicked U.S. public opinion. The EPA was unable to justify its ban scientifically to the United States Court of Appeals for the Fifth Circuit. After lengthy legal proceedings, the Fifth Circuit ordered the EPA to reverse its decision and to acknowledge publicly that modern products containing chrysotile enclosed in a matrix of cement or resin do not pose any detectable risk to public health.[8] (Today, although amphiboles are prohibited in the United States, a number of products containing non-brittle chrysotile are permitted, including the products manufactured by Brazil and previously manufactured in France from Brazilian chrysotile.) Unfortunately, France has adopted a measure that unnecessarily and to no good effect impedes international trade.

4.8 Brazil makes the following claims regarding the ban: (1) the ban is inconsistent with Article 2.2 of the TBT Agreement because it creates unnecessary obstacles to trade and is more trade restrictive than necessary; (2) the ban is inconsistent with Article XI of the General Agreement on

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In developing countries such as Brazil, the availability of low-cost, high-quality building and piping materials, such as chrysotile-cement products, is crucial. Substitute products are more expensive and thus less available to those who need them most.

[7] See *Corrosion Proof Fittings v. EPA*, 947 F.2d 1201, 1224, n.25 (5th Cir. 1991) (*Corrosion Proof*) (written testimony of Mr. Arnold Anderson, ASME).

[8] *Id.*

Tariffs and Trade 1994 (GATT 1994) because it is a quantitative restriction that is not excused by the exceptions in Article XI:2 or Article XX; (3) the ban is inconsistent with Article 2.8 of the TBT Agreement because it applies to asbestos but not to man-made fibres or other substitute products and thus operates as a technical regulation setting forth an unnecessary design or descriptive characteristic; (4) the ban is inconsistent with Article 2.4 of the TBT Agreement because international standards for producing and using chrysotile and chrysotile products exist and France should have used them; (5) the ban is inconsistent with Article III:4 of the GATT 1994 and Article 2.1 of the TBT Agreement (national treatment) because it does not apply to domestic man-made fibres and other substitute products, which are like products to chrysotile; and (6) the ban is inconsistent with Article I:1 of the GATT 1994 and Article 2.1 of the TBT Agreement (MFN) because, insofar as it bans imports of chrysotile and chrysotile products, but not imported like product substitutes, it improperly discriminates among imports.

2. Factual Aspects

4.9 **Brazil** concurs with practically all aspects of Canada's presentation, agreeing (i) that the French ban was passed in response to public outcry in France over the deaths associated with the intensive exposure to amphibole that had taken place early on in the century; (ii) with the circumstances and risks of exposure presented by Canada. In particular, it agrees with the statement that exposure, even in asbestos product plants, has decreased significantly and that, apart from existing flocked amphibole, current exposure is limited, or could be limited, entirely to chrysotile; in contrast, past exposure and current exposure from past uses (e.g. flocking) included exposure to amphibole; (iii) that current levels of exposure to modern uses of chrysotile are not significant and are not associated with substantial health risks; (iv) with the fact that current controlled-use policies and standards which are accepted internationally are sufficient to ensure the health of chrysotile workers and others exposed to chrysotile and to guarantee their safety; and, (v) with Canada's argument that the INSERM Report has many defects and that it was not the reason for France's ban on modern, controlled uses of chrysotile and chrysotile products.

4.10 Brazil considers that a "battle of experts", with one side presenting experts who support banning chrysotile and the other presenting experts who oppose banning chrysotile would be, in this case, both uninformative and unnecessary because the INSERM Report and the Synthesis[9], as a matter of law, not fact, cannot support the ban.[10] This Report and the Synthesis have several defects that render them utterly incapable of supporting the ban.[11] INSERM has not conducted original research, but merely based itself on existing studies and, furthermore, it has not examined all existing studies as it has deliberately excluded those that have established a distinction between chrysotile and amphiboles. More specifically, the shortcomings of the INSERM Report include the following. First, the Report completely fails to examine the modern uses of chrysotile and chrysotile products and, thus, ignores the current state of the industry. Instead, it focuses on the health effects of exposure to amphibole that took place in previous decades. INSERM concedes that it does not have "direct, certain scientific" data on the health risks associated with current levels of exposure to the modern uses of any form of asbestos, much less chrysotile.[12] In short, INSERM does not examine current uses and exposure levels and does not distinguish among the different levels of risk associated with the

[9]INSERM, *Rapport sur les effets sur la santé des principaux types d'exposition à l'amiante, Expertise collective INSERM*, Paris, 1997, (hereinafter "INSERM Report"); INSERM, *Effets Sur la Sante des Fibres de Substitution à l'Amiante-Synthèse, Expetise collective INSERM*, Paris, 1998, (hereinafter "Synthesis").

[10]Brazil concurs with Canada that the weight of all available scientific evidence, including the INSERM Report, leads to the conclusion that the ban serves no purpose other than restricting trade.

[11]Brazil notes that, because it is only the INSERM Report which preceded the ban, the ban must be supported by the Report alone. Brazil has discussed both the Synthesis and the Report because the former underscores some of the defects of the latter.

[12]INSERM, (1998), *Effets sur la sante des fibres de substitution à l'amiante-synthèse*, Paris, p. 226.

different types of asbestos fibres (chrysotile, the only type produced and exported by Brazil, as well as used in it, is accepted as being the safest of asbestos fibres, even by INSERM itself).[13]

4.11 Second, the INSERM Report fails to examine the efficiency of the ways in which worker exposure has been reduced through the use of air filters in mines and plants[14], and employing masks, laundry services, etc. Third, it does not even compare the risks of the past to the risks associated with man-made fibres[15] and substitute products (such as ductile iron or polyvinyl chloride (PVC) pipes).[16] By the time INSERM began to examine substitutes, the ban had already been in effect for 1.5 years and, in any case, INSERM issued only a synthesis and not a complete report on these substitutes. INSERM concedes in its Report that it lacked the data required to recommend the banning of chrysotile and only to allow its substitutes.[17] INSERM emphasizes that because it is the structure (size and shape) of fibres that makes them toxic when inhaled, any substitute fibre must be viewed as dangerous to human health.[18] Finally, INSERM concedes that, although the health data it applied to chrysotile are from past, massive and prolonged exposure to amphibole, the data being collected for substitutes is based on much lower levels of exposure, replicating modern conditions. Most telling is that INSERM states that toxicity levels for "asbestos" as a whole (and not merely for chrysotile) would yield similar results to those obtained for substitutes if similar testing conditions had been used.[19]

4.12 Brazil further argues that INSERM uses a linear risk model to assume illogically and without any evidence that a threshold does not exist for safe exposure.[20] France and INSERM are forced to commit this methodological error (the assumption) due to the fact that they had data from past prolonged exposure to amphibole but not to current, much lower exposure to chrysotile.[21] To justify

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[13]Ibid., p. 409 ("France used asbestos much later and to a much lesser degree than other countries, and doubtlessly the asbestos used contained a lower proportion of amphibole-type fibres. Because of these differences, it is not possible to simply transpose to France the results of projections concerning mesothelioma [and cancer] cases prepared recently for Great Britain.").

[14]Brazil notes that the Cana Brava Mine, in Brazil, for example, has an exceedingly complex and effective air filtration system. The mine is the first and only asbestos mine in the world to have been certified as complying with ISO 14001. It was certified by Det Norske Veritas of Rotterdam, the Netherlands.

[15]See, e.g., Cossette, M., *Substitutes for Asbestos*, 4 December 1998; Anderson, A., *Fibres in Friction Materials*, December 1998; Davis, J.M.G., *The Biological Effects of Fibres Proposed as Substitutes for Chrysotile Asbestos: Current State of Knowledge in 1998*, 1998; *INSERM Synthesis*. Brazil notes that these studies demonstrate that substitute fibres, both when manufactured and used, are likely to present health risks similar to those from chrysotile.

[16]See *Corrosion Proof*, 947 F.2d pp. 1226-27 (even while banning asbestos, the EPA conceded that ductile iron pipes and PVC pipes present health (cancer) risks "similar" to those presented by asbestos-cement pipes).

[17]INSERM, *Effets sur la sante des fibres de substitution à l'amiante-synthèse*, Paris, 1998, pp. 376 and 428. Brazil notes that the European Commission has also recognized this as an important issue: "There is a key scientific issue which Member States and the Commission agree still needs to be clarified. This is an assessment of the relative risk posed by the substitutes in comparison to the risk posed by chrysotile." Official Journal of the European Communities, C 13/35 (18 January 1999) (11 June 1998, Answer of Mr. Bangemann to Written Question P-1451/98 of Peter Skinner (PSE)).

[18]INSERM Synthesis, p. 2.

[19]Ibid., p. 33.

[20]According to Brazil, the assumption is contrary to logic because a threshold must exist given that asbestos is ubiquitous in water and air. Only those who have suffered intensive, prolonged, exposure have contracted asbestos-related diseases.

[21]See also *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Health Effects Institute - Asbestos Research (1991) at pp. 6-9, para. 6.2.2 (Health hazards caused by asbestos at levels encountered in buildings today are "on the order of 50,000 times lower than industrial exposure levels of the past."); *Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario* (1984), Background Briefing Notes No.1 - "Health Effects of Asbestos" (Current exposure of the general public is "thousands of times less" than occupational levels in the

the ban on the modern uses of chrysotile, France/INSERM had to assume that significant risk is present at all levels of exposure, even at those that are insignificant, out of political self-interest. INSERM adopted the linear risk model despite the fact that studies cited by the European Communities (hereinafter "EC") themselves indicate that "*bricoleurs*" are not at risk. The study conducted by Iwatsubo *et al.*[22] indicates that low, sporadic, intermittent and cumulative exposure of up to 0.5 fibres/ml-years does not present increased risk of mesothelioma. In commenting upon the results of an earlier study, the authors note that "no significant risk was observed for those whose exposure was intermittent".

4.13 Brazil argues that a close examination of the INSERM Report reveals that: (i) prolonged exposure to amphibole (its past uses) is associated with severe health problems (a proposition with which everyone agrees); (ii) substitute fibres have similar structures and, thus, when subject to scientific scrutiny, are expected to have similar health effects at similar levels of exposure; (iii) insufficient data exists on the health effects of current levels of exposure to chrysotile and substitute fibres, but the available data suggests that their health effects would be the same; and (iv) the Report does not purport to be as conclusive as France would have all believe; rather, to overcome (iii) above, INSERM extrapolated from the data used in (i), which as it itself concedes "does not produce scientifically certain knowledge, but only an aid to understanding the implications for risk management.[23]" Brazil contends that the ban has been based on the irrelevant data described above. France employs the linear risk model as a tool to make data on past uses relevant to the imposition of the ban. However, INSERM researchers themselves recognize the limitations of this model and clearly state that it cannot produce "scientifically certain knowledge," but can only serve as an "aid to understanding," based on "plausible, though uncertain, estimates.[24]" These "conclusions" do not support significant trade restrictions, much less the ban. Rather, they are merely a call for further research.

4.14 Brazil argues that recent research focusing on uncontaminated chrysotile demonstrates why it presents no health risks whatsoever. According to Dr. David Bernstein's medical explanation[25], the serpentine (braided) structure of chrysotile leads it to unravel in the lungs (whereas the tubular structure of amphibole and substitute fibres does not allow them to unravel and is unchanging); and once unravelled, the smaller and thinner particles are more easily and rapidly enveloped by macrophages and/or expelled from the lungs. Dr. Bernstein's research demonstrates that uncontaminated Brazilian chrysotile of less than 20 microns (the length that has been associated with pathogenicity for all fibres) is very quickly cleared. The clearance half-time is 1.3 days (and is 2.4 days for fibres of a length of 5-20 microns). He concludes that, once in the lung, chrysotile fibres defibrillate (or unravel), breaking down into shorter fibres. According to Dr. Bernstein, this result "is in stark contrast to amphibole asbestos where a portion of the fibres longer than 20 [microns] remains indefinitely or with synthetic mineral fibres where even very soluble fibres are removed by dissolution in the lung with half-times greater than this."[26] He concludes that uncontaminated chrysotile's lack of bio-persistence suggests that it has "little if any toxicological effect."[27] However, it is of course a fact

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three decades during and after World War II (p. 3); the best estimates are that exposure of current occupants of asbestos-containing buildings "is 1,000 to 10,000 times lower than the average exposure of insulation workers in the past" (Volume 2 p. 585).

[22]Iwatsubo Y. et al., *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study*, American Journal of Epidemiology, 1998, Vol. 148, N° 2.

[23]INSERM Report, pp. 239 and 414..

[24] *Ibid.*, pp. 239 and 232.

[25]Bernstein D., *Summary of the Final Report on the Chrysotile Bio-Persistence Study*, Geneva, 2 October 1998 (document presented by Brazil to the Panel).

[26]Ibid., p. 4.

[27]Ibid., p. 10.

that if used improperly, uncontaminated chrysotile could be dangerous, but that would be the case for virtually all products in existence and not just chrysotile.

4.15 Brazil indicates that it mines, produces and exports only uncontaminated chrysotile and chrysotile products, and subjects mining, production and use to strict regulations. In 1990, it signed the ILO Convention and Recommendation Concerning Safety in the Use of Asbestos (Convention 162 and Recommendation 172). To ensure safety in the mining, manufacture and use of chrysotile and chrysotile products and to meet its ILO obligations, Brazil passed a primary law[28] and decree[29] on asbestos. In addition, the production and use of chrysotile and chrysotile products is governed by "national tripartite (government-industry-workers) agreements". These set exposure limits, and processes of production and safety procedures to be used to guarantee worker safety. Finally, the Brazilian Asbestos Association (ABRA), a watchdog organization comprised of asbestos producers and sellers, further regulates the safety of, and trade in, chrysotile and chrysotile products.

4.16 Brazil explains that the ILO Convention and Recommendation are international standards that establish safety procedures for the handling of chrysotile and chrysotile products. They follow the ILO Code of Practice on Safety in the Use of Asbestos.[30] The goal of the Code is to prevent the risks of exposure to asbestos and its harmful effects and to provide practical control procedures for its use. Convention 162 and Recommendation 172 recommend the controlled and safe use of asbestos. Their wording clearly indicates that the replacement of asbestos fibres should only take place when it is established that this is necessary to protect worker health and when replacement is technically feasible. The replacement of chrysotile asbestos fibres contained in modern materials or products (i.e. where it is sealed in a matrix and cannot be released into the environment) is not necessary since these products do not pose any detectable health risks. International standards, such as Convention 162 and Recommendation 172, recommend the regulation of asbestos on the basis of the type of asbestos fibre employed, the products in which certain fibres are included, and their planned use. Thus, Convention 162 and Recommendation 172 stipulate the prohibition of crocidolite and materials containing friable asbestos for flocking[31], but permit many uses of chrysotile, including those central to this dispute (asbestos-cement and friction products). They allow countries to prohibit other specific uses if national authorities deem this necessary for worker protection, but only on condition that substitute products be subjected to a thorough scientific examination of their health effects.[32]

4.17 In 1995, Brazil passed Law No. 9055 to discipline the extraction, industrialization, use, commercialization and transportation of asbestos and of asbestos-containing products, as well as of natural and synthetic fibres of any source used for the same purpose. The Law (i) bans the processing and use of all types of asbestos, except chrysotile and chrysotile-containing products; (ii) bans the crushing and spraying (flocking) of all types of asbestos, including chrysotile, and of all substitute fibres; (iii) provides the framework for the tripartite agreements in that it sets deadlines for the government's confiscation of the operating licences of companies that do not execute the tripartite agreements, establishes medical inspection requirements for workers, and sets exposure limits for those who work with chrysotile and substitute fibres subject to annual reduction. (In compliance with Article 2.4 of the TBT Agreement, the exposure limits are determined based, in part, on the recommendations of "international entities which are scientifically accredited"); (iv) prohibits miners

[28]Brazilian Law No. 9055 of 1 July 1995.
[29]Brazilian Decree No. 2350 of 15 October 1997.
[30]*Safety in the Use of Asbestos*, Code of Practice, International Labour Organization, Geneva, 1990.
[31]Convention 162, Article 12.
[32]Article 12 of Recommendation 172 states:

(1) The competent authority, wherever necessary for the protection of the workers, should require the replacement of asbestos by substitute materials, wherever possible.

(2) *Before being* accepted for use in any process, all potential substitute materials should be *thoroughly evaluated* for their *possible harmful effects* on health. The health of workers exposed to such materials should be continuously supervised, if judged necessary. (Emphasis added.)

and wholesalers from supplying chrysotile or substitute fibres to companies that do not comply with all provisions of the Law; (v) applies special restrictions to the use of chrysotile and substitutes in products currently considered to be the riskiest, such as textiles; (vi) calls for research into the health effects of chrysotile and substitute fibres and provides financing for the effort; and (vii) provides for prompt Department of Justice action against infractions.

4.18 Brazilian Decree No. 2350 implements the Law, and (i) requires that prior to marketing, all products containing chrysotile of imported or national origin bear a "seal of compliance to the Brazilian System of Certification", and provides for the development of the certification system; (ii) requires research into and confirmation of the health effects of chrysotile and its substitutes; (iii) establishes additional requirements for the tripartite agreements which apply to all mines and companies producing chrysotile and chrysotile products; (iv) establishes requirements for monitoring and controlling the use of chrysotile and its substitutes, and ensures that a record is kept of the exposure measurements made by companies while guaranteeing access to them; and (v) establishes a permanent National Commission on Amianthus (NCA) to ensure the safety of workers involved in the chrysotile or substitute fibre industry. The Decree also establishes certain bodies, such as the NCA, composed of government and industry officials as well as workers, to ensure worker safety.

4.19 The tripartite agreements (otherwise known as *The National Agreements for the Furtherance of the Safe Use of Asbestos*) are required by both the Law and Decree. They are executed by the Federal Government of Brazil, the industries involved (e.g. the mining or asbestos-cement industry) and the workers in the industry (through their unions). They establish mandatory medical procedures inspection and safety measures, as well as exposure limits. They also give workers certain rights, both individual and collective, within their industries. Their objective is to continuously work towards improved worker safety and to decrease exposure limits as well as actual exposure. First, tripartite agreements set the maximum permissible exposure limits to 0.30f/cm3, with 50 per cent of all measurements being below 0.10 f/cm3 (and without there being any constant exposure above 0.3 f/cm3, even when the workers exposed have special breathing equipment). Second, they require the use of specific "collective protection" procedures to protect workers. The procedures are to include the installation of air filter and exhaust systems, the use of wet processes in the handling of chrysotile (which reduces dust release and, thus, exposure), the sealing of workspaces and processes to limit exposure, the demarcation of areas of exposure for warning, the prohibition of dry sandpapering processes, the implementation of a daily programme for the washing, wetting or vacuum cleaning of production sites, and provisions for a change of work clothes (which may not be taken off site), of laundry services and showers for employees. Third, the agreements require employers to provide workers with individual protection equipment that complies with relevant standards. Fourth, they also require them to conduct regular and detailed environmental evaluations of working conditions as well as to medically inspect their employees. All results are to be filed with the Control Commission on the Safe Use of Asbestos and with the Brazilian Asbestos Association, known as ABRA. The Control Commission is comprised of plant workers elected by their peers. Fifth, they require the provision of worker education programmes to communicate the health risks of exposure to chrysotile, the measures which can be taken to reduce exposure, and the "multiplier effect" which tobacco smoking has on exposure. Sixth, they make ABRA responsible for providing the companies with technical assistance regarding controls and preventive measures.

4.20 Founded in 1984, ABRA is an industry watchdog group composed of companies from Brazil's asbestos industry. Its main goal is to oversee industrial activity in order to ensure that ABRA members comply with the Law, the Decree and the tripartite agreements, as well as to educate workers, wholesalers and end-users of chrysotile asbestos and asbestos products on safe use. To accomplish this goal, ABRA has an extensive, independent, monitoring programme. Biannually, it conducts spot measurements at the facilities of its members. It maintains an ISO 9000-certified laboratory and sends control samples once a year to independent laboratories in Edinburgh (AFRICA) and Paris (LHCF) to ensure the accuracy of its measurements. If the company that has been tested

fails to meet the applicable exposure limits, ABRA sends it a letter and informs its suppliers. It then provides the company with a maximum number of days in which to comply, and instructs its suppliers to withhold chrysotile and/or chrysotile products from the company until it is able to notify its compliance. The agreement restates the requirements of both the Law and Decree, and develops certain safety procedures. In exchange for compliance (and dues), ABRA serves as a low-cost repository for state-of-the-art safe-use technologies, covering areas such as plant, air filter and process design. It attempts to encourage as well as facilitate safe use, with its overarching objective being to regulate the industry in such a manner as to render additional government regulation unnecessary. The regulatory regime (consisting of the Law, the tripartite agreements and ABRA itself), aligns the self-interests of the industry with those of its workers. The industry and the workers individually, as well as through Safety Commissions and Unions, cooperate to reduce health risks. The result of this cooperation has been the creation of an extremely safe workplace with very low exposure levels. In general, this system encourages individual plants to exceed applicable requirements in order to guarantee worker and user safety. At the Capivari Asbestos Cement Plant, which is the largest chrysotile-cement plant in South America, the on-site doctor did not report a single case of asbestos-related disease among the employees whose contact with asbestos had been limited to the plant.

4.21 With respect to regulation of asbestos in the United States, Brazil asserts that, in response to public outcry based on sensationalist media reports on the dangers of asbestos, the Environmental Protection Agency (EPA) banned asbestos in 1989.[33] It prohibited "at staged intervals, the future manufacture, importation, processing, and distribution in commerce of asbestos in almost all products […]". In reaction, a United States company that manufactured asbestos pipes, Corrosion Proof Fittings, filed suit against EPA arguing that the ban was not based on scientific and medical information. In a 1991 decision, the United States Court of Appeals for the Fifth Circuit called for the lifting of the ban and ordered EPA to issue new rules grounded in science.[34] The Fifth Circuit concluded that EPA had presented "insufficient evidence to justify its asbestos ban."[35] Specifically, it found that the EPA had failed to (i) consider all of the necessary and relevant evidence, and (ii) "give adequate weight to statutory language requiring it to promulgate the least burdensome, reasonable regulation" that would protect human health.[36] Similarly, France has failed to (i) examine existing evidence on the modern, controlled uses of chrysotile, (ii) assess the danger associated with substitute products, and (iii) impose a regulation that is not more restrictive than necessary. In 1993, EPA lifted the ban and issued new provisions regulating the production and use of asbestos and asbestos products.[37] Based on a thorough scientific and medical review, EPA then authorized more asbestos products (18) than it banned (6). None of the uses that are banned are at issue in this proceeding. Of the authorized uses, two are central to Brazil's exports to France and had previously been allowed (they include chrysotile-cement products and chrysotile friction materials).[38] Under existing regulations, the United States produced 6,890 metric tonnes of chrysotile and imported 20,900 metric tonnes in 1997.[39] In the same year, it consumed nearly 21,000 metric tonnes of chrysotile, exported unmanufactured fibre for a total value of US$5,690,000 and manufactured products for a total of US$197,000,000.[40] Public health has not suffered in the United States and public outcry did not resume. United States regulations ban the dangerous uses of asbestos, and regulate those that are safe.

[33]EPA Final Rule, 54 Fed. Reg. 29460 (1989).
[34]*Corrosion Proof v. EPA*, 947 F.2d 1201 (5th Circuit 1991).
[35]Ibid., p. 1215.
[36]Ibid.
[37]EPA Final Rule, 58 Fed. Reg. 58964 (1993).
[38]The current US regulations on this topic are set forth at 40 C.F.R. part 763, Sub-Part I (1998).
[39]United States Government Geological Survey, Minerals Yearbook 1997, Volume I at 4-5.
[40]Ibid.

3. Legal Aspects

4.22 Brazil argued that, as it turns to Brazil's legal arguments, the Panel should recall Brazil's complex system of regulation that ensures public safety. The Panel should serve the same role regarding France's political decision which the Fifth Circuit served regarding EPA's political decision - that of a neutral arbitrator. Brazil understands that the *Corrosion Proof* decision does not in the least bind the Panel - the procedures, legal standards and status of the parties are quite distinct. However, the court there faced similar circumstances and issues, and in the face of contrary public sentiment, issued a very focused, well-reasoned opinion, which is precisely what Brazil seeks here.

(a) The Agreement on Technical Barriers to Trade

(i) Article 12 of the TBT Agreement

4.23 Brazil argues that a prohibition of the trade and use of a product, such as France's ban, is the most restrictive of all possible trade measures and must be closely scrutinized by the Panel. It requests the Panel to devote particular attention to the ban on imports from Brazil, which is a developing country (and from Zimbabwe, a least-developed country). In general, WTO agreements provide for the special and differential treatment of developing and least-developed country exports. In the context of the TBT Agreement, special provisions are set forth in Article 12, which obliges Members that are developing technical regulations and standards to consider the special needs of developing and least-developed countries and to provide them with differential treatment. Article 12.2 obliges France to "take into account the special development, financial and trade needs" of developing countries and of least-developed countries, when developing its technical regulations. France did not meet this obligation. Rather, it adopted an outright ban that advantages French producers of substitute fibres and products to the detriment of Brazil's chrysotile and chrysotile product producers (and to Zimbabwe's detriment as well). Moreover, the ban has not contributed to improving public health in France.

4.24 France violated Article 12.3 which covers the "preparation and application" of technical regulations and standards. Article 12.3 requires France to ensure that its technical regulations "do not create unnecessary obstacles to exports" from developing countries such as Brazil (and from least-developed countries such as Zimbabwe). However, France's ban applies to Brazilian (and Zimbabwean) exports and creates, to say the least, an "obstacle" to their trade. The obstacle is "unnecessary" because it does not contribute to the supposed objective of increasing safety. The only trade permissible under the ban is that of chrysotile and chrysotile product substitutes. The risks associated with substitute fibres are unknown, but they are suspect. Meanwhile the risks associated with the modern, controlled uses of chrysotile, are zero.

(ii) Article 2.2 of the TBT Agreement

4.25 Brazil argues that the ban is inconsistent with Article 2.2 of the TBT Agreement because it is more trade restrictive than necessary to fulfil a legitimate objective. Once it is established that the Decree is a "technical regulation", the EC must demonstrate (and have the burden of proving) that four different conditions have been met if they are to argue that the ban is in fact consistent with Article 2.2.[41] To defend the ban, the EC must demonstrate to the satisfaction of the Panel that (i) the objective of the ban is "legitimate", (ii) that it "fulfils" this legitimate objective, (iii) that it is not

[41]According to Brazil, general rules of pleading, but also Article 2.5 of the TBT Agreement confirm that France has the burden of justifying its trade restrictive measure. According to Article 2.5, a standard shall be "rebuttably presumed not to create an unnecessary obstacle to trade" when it pursues a legitimate objective and is "in accordance with relevant international standards." France cannot take advantage of this exception to normal rules of pleading because, as demonstrated below, the ban is contrary to relevant international standards.

"more trade restrictive than necessary" to fulfil the legitimate objective, and (iv) that France evaluated the health effects (i.e. "the risks non-fulfilment would create") on the basis of "available scientific and technical information". According to Brazil, the ban meets only the first of these four conditions.

4.26 Brazil argues that the Decree is a "technical regulation" within the meaning of the TBT Agreement. The ban sets out certain (i) product characteristics, (ii) process and production methods, (iii) administrative provisions, as well as (iv) packaging, marking and labelling requirements with which compliance is mandatory. Article 1 of the Decree prohibits the production, importation, exportation, manufacture, transformation, sale and offer for sale of all types of asbestos fibres and asbestos-containing products (except those temporarily excepted from the ban by virtue of Article 2.I). Thus, the ban is explicitly directed at product characteristics (asbestos and asbestos-containing products) and at process and production methods (all forms of production, manufacture and transformation of asbestos and asbestos-containing products). Both the prohibition imposed by Article 1 and the procedures for implementing and reviewing the entitlement to the exceptions set out in Articles 2.II and 3 of the Decree are "applicable administrative provisions" relating to product characteristics and process and production methods. Article 4 of the Decree prescribes certain marking and labelling requirements for those few asbestos-containing products excepted under Article 2. Compliance with the ban is mandatory and violations are penalized under Article 5. Brazil argues that both France and the EC have conceded that the Decree is a technical regulation. In WTO document G/TBT/Notif.97.55, dated 21 February 1997, the French Government notified the ban to the TBT Committee as a technical regulation. Paragraph 3 of the Notification indicates that the ban was being notified under Articles 2.9.2 and 2.10.1 of the TBT Agreement, both of which establish notification obligations for technical regulations. The European Commission has also recognized that the ban is a technical regulation both in a 15 April 1997 document justifying the French ban and during the 8 July 1998 consultations on this dispute. Therefore, both France and the EC concede that the ban falls within the scope of paragraph 1 of Annex 1 of the TBT Agreement and is a technical regulation.

4.27 Brazil does not contest that the objective of protecting the health of French workers and consumers is a "legitimate objective" within the meaning of Article 2.2 of the TBT Agreement. However, it argues that the ban imposed by the Decree creates an unnecessary obstacle to trade. It does not in reality fulfil its stated objective, and is more trade-restrictive than necessary to protect the health of French workers and consumers. In using the word "fulfil" (as in the requirement that "technical regulations shall not be more trade-restrictive than necessary to fulfil a legitimate objective"), the text of Article 2.2 requires the existence of a rational link between the regulation and its stated objective.[42] However, this rational link is absent as the ban does nothing to accomplish its objective. It does not make those who are now sick healthy and removing it would not make any of those now healthy, sick. The lack of a rational link between the ban and its purported objective is demonstrated by the following: (i) that asbestos-related health risks are due to old and already prohibited uses of asbestos; (ii) that there are no detectable health risks associated with modern uses

[42]Brazil notes that, while no WTO panel or Appellate Body reports have addressed this issue under the TBT Agreement, relevant precedents under the SPS Agreement exist: In *Japan - Apples*, the Appellate Body found that an SPS measure was justified only if the Member imposing the measure demonstrated a "rational relationship" between the SPS measure and available scientific information. *Japan - Measures Affecting Agricultural Products* (22 February 1999), WT/DS76/AB/R, para. 84; similarly, in *EC - Hormones*, the Appellate Body required the EC to establish "an objective relationship between two elements, that is to say, an objective situation that persists and is observable between an SPS measure and a risk assessment." *EC - Measures Concerning Meat and Meat Products (Hormones)* (16 January 1998), WT/DS26/AB/R, para. 189; the Appellate Body has also held that a finding that an SPS measure is not based on an actual assessment of health risks is "a strong indication" that the measure does not really protect health but is instead "a trade-restrictive measure in the guise of an SPS measure." *Australia - Measures Affecting Importation of Salmon* (20 October 1998), WT/DS18/AB/R, para. 166. This is precisely the case with the ban.

of chrysotile; and (iii) that health risks associated with substitute fibres remain unknown and are suspect.

4.28 Brazil asserts that the health risks addressed in the INSERM Report are based on past exposure to high levels of asbestos fibres (largely amphiboles) and to exposure to old uses of asbestos, such as flocking. In prohibiting future importation and sale of chrysotile and modern chrysotile-containing products, the ban does nothing to address the effects (today) of exposure between 1940 and the early 1960s to extremely high levels of asbestos, mainly amphibole fibres. It does not cure workers who now suffer because of long-term exposure in the past to amphibole, the use of which was banned in France in 1994, or to unregulated concentrations of fibres that are "50,000 times" higher than the modern-day internationally recognized controlled-use level of 1 f/ml.[43] Likewise, prohibiting the future importation and sale of chrysotile and modern chrysotile-containing products does nothing to address the effects of exposure to (or the disturbance of) friable asbestos, mainly amphibole, in French buildings prior to the 1978 French ban on flocking. This was recognized by European Commissioner, Mr. Bangemann, who, in response to a question posed by the European Parliament, responded that "[I]t is important to mention that a new ban would not lead to a lower risk of exposure to existing asbestos for workers, nor would it reduce the number of deaths from past exposure to asbestos."[44]

4.29 Brazil maintains that there are no detectable health risks associated with modern uses of chrysotile. There is no rational link between the ban and its purported objective because modern uses of uncontaminated chrysotile are safe. Prior to the ban, more than 90 per cent of the chrysotile imported into France was used in the manufacture of chrysotile-cement products.[45] Currently, the chrysotile is bound to the cement and encapsulated in it, without there being any loose or friable fibres. Furthermore, most chrysotile-cement products are produced in such a way that sawing or drilling are unnecessary, and in the few instances when either or both are required, widely recognized and well-established procedures have been developed for these tasks which prevent fibre release.[46] Similarly, in all other modern uses of chrysotile, the fibres are sealed, bonded or encapsulated in the product. In no instance are loose, friable fibres allowed to be. Brazil contends that France does not have credible evidence to suggest that (i) sealed, bonded or encapsulated chrysotile poses a health risk, (ii) concentrations of chrysotile fibres at or below the internationally-recognized controlled use level of 1 f/ml present a health risk, and (iii) controls do not eradicate all risk throughout a product's life-cycle (from mining to manufacture, distribution, sale and use, and eventual disposal). On the other hand, much science-based research concludes that the level of chrysotile encountered in the workplace today, or in buildings, presents no detectable health risk. After an exhaustive study of the existing scientific literature, the Health Effects Institute concluded in 1991 that the health hazards created by asbestos at the levels commonly encountered today are "unlikely to be large enough to be actually

[43]Health Effects Institute – Asbestos Research, *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Cambridge, 1991, pp. 6-9.

[44]Official Journal of the European Communities, C 13/123 (18 January 1999) (24 July 1998 answer of Mr. Bangemann to Written Question E-1950/98 of Anita Pollack (PSE)).

[45]Le Déaut J.-Y. and Revol H., *L'amiante dans l'environnement de l'homme: ses conséquences et son avenir*, Office parlementaire d'évaluation des choix scientifiques et technologiques, Assemblée nationale no. 329 / Sénat no. 41, 16 October 1997.

[46]ISO 7337, §§ 4 and 5 (pp. 2-9): Brazil notes that the cutting of plates or tiles for roofing is not a source of emission if ISO-7337 is followed. ISO-7337 addresses the use of chains to break pipes through pressure, low-speed saws, saws equipped with a vacuum dust extractor, and, also, proper wetting of the materials prior to any action. The cutting or grinding of all cement pipe (even that which does not contain chrysotile) emits silica in the air, in the absence of proper controls. The International Association for Research on Cancer (IARC) rates silica as a Type 1 carcinogen (for man), like asbestos. The worker who cuts any cement pipe therefore has an interest in following ISO-7337.

observed and measured.[47]" This conclusion (which was reached by an independent United States health watchdog), confirmed the 1984 findings of the Ontario Royal Commission.[48] Similarly, in the case brought by Corrosion Proof Fittings against the EPA, the Fifth Circuit made the following comment on the risk of asbestos products relative to toothpicks:

> "As the petitioners point out, the EPA regularly rejects, as unjustified, regulations that would save more lives at less cost. For example, over the next 13 years, we can expect more than a dozen deaths from ingested *toothpicks* - a death toll more than twice what the EPA predicts will flow from the quarter-billion-dollar bans of asbestos pipe, shingles, and roof coatings."[49]

Brazil concludes that, because there are no detectable risks attributable to modern uses of chrysotile, there is no rational link between the French ban and its purported objective.

4.30 Brazil asserts that the French ban induces consumers to use chrysotile substitutes, whose health risks are unknown, in place of chrysotile, whose risks are known. In his 1998 paper on the biological effects of substitute fibres, Dr. J. M. G. Davis concluded that "replacement [of chrysotile by substitute fibres] is premature in the present state of our knowledge .... The need for full toxicology testing of new fibre products is recommended before these products are marketed."[50] This conclusion was shared by the European Communities Directorate General for Consumer Protection Policies, which stated that "there is no significant epidemiology base to judge the human health risks [of substitute fibres] … hence the conclusion that [the uses of] specific substitute materials pose a substantially lower risk to human health, particularly public health, than the current use of chrysotile, is not well founded … ".[51] The INSERM Report itself acknowledges that the risks associated with substitute fibres are unknown. INSERM "urgently" cautions against their use until further scientific tests are conducted. It states that "[T]he absence of epidemiological data concerning the long-term safety of these substitute products should not obscure the results of experimental systems indicating the possibility that pathological modifications could result. It is urgently important that suitable research into this area be conducted prior to the widespread use of substitute fibres."[52] Despite this urgent warning from its own experts, the French Government banned chrysotile and not its substitutes the day after it received the INSERM Report. Thus, the French Government knowingly shifted consumption from chrysotile used in modern ways, and for which there is no detectable health risk, to substitute fibres for which "experimental systems indicate the possibility that pathological modifications could result". Brazil concludes, therefore, that the ban does not "fulfil" a legitimate objective as required by Article 2.2 of the TBT Agreement. The rational link between the ban and its stated health objective does not exist because, as demonstrated above, (i) asbestos-related health risks are due to old, already prohibited, uses of asbestos, and not to the modern uses of chrysotile, (ii) no detectable health risks are associated with the modern uses of chrysotile, and (iii) substitute fibres, whose health risks are unknown, will replace chrysotile.

4.31 Brazil further argues that even if there were a rational link between the ban and the purported objective, the French ban would nonetheless be inconsistent with Article 2.2 of the TBT Agreement

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[47]Health Effects Institute – Asbestos Research, *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Cambridge, 1991, pp. 6-9.

[48]Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario (1984), vol. 2, p. 585.

[49]*Corrosion Proof v. EPA*, 947 F.2d 1201 (5th Circuit 1991. See also L. Budnick, *Toothpick-Related Injuries in the United States, 1979 Through 1982*, 252 J. Am. Med. Ass'n., 10 Aug. 1984, p. 796 (which shows that toothpick-related deaths average approximately one per year).

[50]Davis J.M.G., *The Biological Effects of Fibres Proposed as Substitutes for Chrysotile Asbestos: Current State of Knowledge in 1998*, p. 1 and 5.

[51]European Commission, DG XXIV, Opinion on a Study Commissioned by Directorate General III on Recent Assessments of Hazards and Risks Posed by Asbestos and Substitute Fibres (9 February 1998), p. 1.

[52]INSERM Report, p. 434.

because it is "more trade-restrictive than necessary to fulfil a legitimate objective, taking account of the risks non-fulfilment would create.[53]" A ban is the most trade-restrictive measure possible. It could be justified only if France were able to prove that there was no reasonably available, less trade-restrictive, alternative. France cannot do so. Controlled use policies demonstrably fulfil the objective of protecting the health and safety of French workers and consumers. In assessing whether the ban is more trade-restrictive than necessary, within the meaning of Article 2.2, the Panel should examine both the risks of non-fulfilment and whether a less trade-restrictive measure is available to fulfil the objective.

4.32 Brazil argues that available scientific and technical information does not support the imposition of the ban. Article 2.2 provides that, in assessing the risk that a technical regulation is meant to address, Panels should consider, *inter alia*, relevant scientific and technical information, related processing technology and intended end uses of products. The risk to be avoided in the dispute at hand is the risk of illness resulting from exposure to (a) modern uses of chrysotile and chrysotile-containing products and (b) the disturbance of previously installed friable asbestos (largely amphibole) in buildings. Illnesses associated with old, previously banned, uses of asbestos are not relevant to this analysis. Moreover, they cannot be addressed through the present ban on trade, domestic sale and use. The INSERM Report, which provides the supposed scientific justification for the ban, does not assess the health effects of current levels of exposure to modern uses of chrysotile. To determine the health risk associated with exposure to low levels of bonded, sealed and encapsulated chrysotile in chrysotile-cement and other modern applications, it applies the same risk of exposure associated in previous decades with higher levels of exposure to friable asbestos (largely amphibole). There is no scientific logic for such an extrapolation. The INSERM Report itself concedes that its conclusions are not "scientifically certain" but are merely "plausible, though uncertain, estimates". Several other scientific reports concur that there is no detectable health risk from modern uses of chrysotile.[54]

4.33 Brazil explains that all modern-day uses of chrysotile involve bonding, sealing or encapsulation. Such uses, or modern products, do not contain loose, friable chrysotile fibres – which were the cause of past asbestos-related illnesses. The risk associated with modern use is undetectable. Most modern products are manufactured to specifications well known in the building and public works trades, so that sawing or drilling operations are seldom necessary. When sawing or drilling are necessary, there are well-established procedures to ensure that workers are not exposed to fibre release. Thus, Brazil argues that neither available scientific information, intended end-uses nor processing technology, necessitate a ban on chrysotile. Brazil argues that while the term "necessary" has not yet been interpreted in the context of Article 2.2 of the TBT Agreement, the interpretation provided by the Panel in the case on *Section 337 of the Tariff Act of 1930* is instructive:

> "[I]t was clear to the Panel that a contracting party cannot justify a measure inconsistent with another GATT provision as 'necessary' in terms of Article XX:(d) if an alternative measure which it could reasonably be expected to employ and which is not inconsistent with other GATT provisions is available to it. By the same token, in cases where a measure consistent with other GATT provisions is not reasonably available, a contracting party is bound to use, among the measures reasonably available to it, that which entails the least degree of inconsistency with other GATT provisions."[55]

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[53]Brazil notes that, similarly, under the SPS Agreement, in determining whether an SPS measure is more trade-restrictive than required, the authorities must evaluate whether an alternative, less trade-restrictive, SPS measure would achieve the importing country's appropriate level of protection. *See Australia - Measures Affecting Importation of Salmon* (20 October 1998), WT/DS18/AB/R, paras. 208-210.

[54]See paragraph 4.29 above, regarding the conclusions of the American Health Effects Institute, the Royal Commission and the U. S. Court of Appeals for the Fifth Circuit.

[55]*United States – Section 337 of the Tariff Act of 1930*, adopted on 7 November 1989, BISD 36S/345, pp. 392-93, para. 5.26.

4.34 Brazil argues that the focus is on the range of measures "reasonably available" to France. Just as under Article XX(d), under Article 2.2 of the TBT Agreement, the French ban cannot be justified as "necessary" since a less trade-restrictive measure that fulfils the legitimate objective is available. There are numerous examples of controlled use that are both readily available and effective in addressing the health risks associated with the modern uses of chrysotile. First, the ILO Convention and Recommendation Concerning Safety in the Use of Asbestos (Convention 162 and Recommendation 172) establish procedures to ensure safety in the handling of chrysotile and chrysotile products. Second, the ILO's 1990 Code of Practice on Safety in the Use of Asbestos details appropriate controlled use procedures to ensure worker safety with respect to all chrysotile-containing products currently in use. Third, Brazil, the United States and Canada have demonstrated that controlled-use policy is effective in eliminating the health risks attributable to the modern uses of chrysotile. Controlled use policy is less restrictive than a ban. Trade and sales are permitted as long as appropriate safety measures are employed in the manufacture, installation and use of chrysotile-containing products. While complying with safety regulations could be expensive for firms, the decision of whether or not to use chrysotile or substitutes under the safety regulations should be determined by the marketplace and not by government. Given the availability of controlled use policy and its effectiveness in addressing the legitimate public health objective which France wishes to achieve, the ban is inconsistent with Article 2.2 in that it is more trade-restrictive than necessary to fulfil its objective.

(iii) Article 2.4 of the TBT Agreement

4.35 Brazil contends that the French ban is inconsistent with Article 2.4 of the TBT Agreement because it ignores appropriate and effective international standards. Article 2.4 obliges France to base its technical regulations on existing international standards, or on any "parts of them", that would be effective and appropriate in any given circumstance. France violated this obligation when it banned chrysotile and chrysotile products, ignoring existing international standards that would have been both appropriate and effective. To establish that France has not violated Article 2.4, the EC must show that: (i) there are no international standards that apply to asbestos; (ii) if international standards exist, that the ban is consistent with them; or (iii) if international standards exist and the ban is inconsistent with them, that the international standards would not have been an effective or appropriate means of accomplishing France's stated objective. The EC cannot make such arguments.

4.36 Brazil argues that a number of international standards apply to chrysotile and chrysotile products, including ILO Convention 162 and Recommendation 172, on the types of asbestos that can be used (only chrysotile) and how, and the International Organization for Standardization's (ISO) 7337 standard, entitled Chrysotile Cement Products Guidelines for On-Site Work, regarding the proper installation and use of chrysotile-cement products. The fact that the ISO 7337 standard is an applicable international standard is beyond doubt. Annexes 1 and 3 to the TBT Agreement expressly recognize the authority and status of ISO as an international standard-setting body, and the ISO 7337 standard directly governs the primary chrysotile product group. Each of these documents state that chrysotile products may be manufactured and used, but only under controlled conditions and in modern applications. Each of the standards sets out specific controls to guarantee the safety of workers and end-users. They have been incorporated into Brazilian legislation as well as that of many other countries, including the United States and Canada. The ban is inconsistent with these international standards because it bans all imports, manufacture, use, etc., of chrysotile and chrysotile products, whereas these permit their use in modern applications. They only subject them to safety controls. Current international standards provide an effective and appropriate means of fulfilling France's stated objective and the EC cannot argue otherwise. ILO and ISO standards are "appropriate" for France's stated objective since they were specifically drafted to protect the health of industrial workers, the general public and others who may come into contact with asbestos. ILO and ISO standards would also be "effective" in achieving France's stated objective since they have successfully

protected human health in economies as diverse as those of Brazil, the United States and Canada. The EC would be hard pressed to provide evidence of a deterioration in the health of citizens from Brazil, the United States or Canada due to adherence to ILO or ISO standards.

4.37 Brazil states that a closer examination of the term "ineffective and inappropriate means" is justified. The text of Article 2.4 clarifies that this exception is to be quite narrowly construed and applied. Were it not to be so, Article 2.4 would be rendered useless.[56] Members would all too easily claim that the applicable international standard was "inappropriate". Second, the Article provides examples of the situations in which exceptions to the use of international standards are allowed. These include when an international standard would be ineffective or inappropriate due to fundamental climatic or geographical factors or fundamental technological problems. Thus, a Member may ignore an international standard only if the standard will not achieve the results it seeks because of its unique conditions in terms of climate, geography, or its economy (i.e. level of technological development). No such conditions exist in France. The EC would be unable to present any evidence to suggest that different conditions apply to France so as to make the standards followed by Brazil, the United States and Canada inappropriate or ineffective for it. France ignored ILO and ISO standards because it wished to ban chrysotile to appease public opinion and advantage domestically-produced and substitute products.

(iv) Article 2.8 of the TBT Agreement

4.38 Brazil argues that the French ban is inconsistent with Article 2.8 of the TBT Agreement because it establishes design requirements for products. France's ban is inconsistent with this obligation because, by prohibiting chrysotile and its use in any product, the ban sets out an impermissible "design or descriptive characteristic". To establish that France has not violated Article 2.8, the EC must demonstrate that (i) the ban is a performance requirement; or, in this case, (ii) that adopting a performance requirement would not have been "appropriate". The Communities can demonstrate neither of these points. The ban sets out an impermissible "design or descriptive characteristic" because it regulates on the basis of the content and description of a product. France has banned chrysotile and products containing it but has not banned competing fibres and products that contain them. Therefore, the ban advantages French-produced substitute fibres, products which are "like"[57] chrysotile, and chrysotile containing products. The ban does not contain regulations based on the performance of a product. Rather, it states that certain products may be imported and sold only if they do not contain a certain input, namely chrysotile. Article 2.8 obliges France to adopt a performance requirement "whenever possible". In the case of chrysotile, France could have adopted any of a number of performance requirements that would have enabled it to achieve its stated objective.

4.39 According to Brazil, France could have adopted, for example, detailed regulations regarding the importation, production, modern use and disposal of chrysotile and substitute fibres and their products (as France had previously done and as do Brazil, the United States, Canada and many other countries). Alternatively, France could have established a single, never-to-be-exceeded exposure level to apply to the manufacture, use and disposal of chrysotile and substitute fibres, and to their products. France could, and should, have adopted a performance requirement for chrysotile and the products which contain it. Instead, it adopted a design or descriptive requirement and violated Article 2.8 of the TBT Agreement. Were any other findings to be reached by the Panel, it would allow Member countries to take the much easier route of banning, rather than regulating, products which they claim

[56]Brazil notes that interpretations that render a treaty provision null or void, or consign it to "inutility" are to be avoided whenever possible. See *United States – Standards for Reformulated and Conventional Gasoline* (20 May 1996), WT/DS2/AB/R, p. 23.

[57]Brazil notes that paragraphs 4.42-4.43 below demonstrate that the man-made substitute fibres and products are like products to chrysotile and chrysotile products.

create health risk. The TBT Agreement is based on the assumption that certain products present risks and that those risks are to be managed through standards. To allow a Member to ban, instead of to regulate, products due to perceived risks would render the Agreement meaningless.

(b) The General Agreement on Tariffs and Trade

(i) Article XI of the GATT

4.40 Brazil submits that the ban is also inconsistent with GATT Article XI because it is a quantitative restriction that is not permitted by the WTO. The ban includes (i) a prohibition of the sale in France of chrysotile and chrysotile products, which is a violation of GATT Article III:4, and (ii) a prohibition of the importation of chrysotile and chrysotile products. In fact, paragraphs I and II of Article 1 of the ban prohibit "the import […] of all kinds of asbestos fibres […] whether or not these substances are incorporated into materials, products or devices". The latter aspect violates Article XI. In reference to paragraph 1 of Article XI, Brazil argues that the ban on importation is not a "duty, tax or other charge," but is a "prohibition or restriction" that France has instituted and maintained on the importation of chrysotile from Brazil. Indeed, the ban is the most restrictive of all quantitative restrictions in that it sets a quota at the level of zero imports.[58] Therefore, the portion of the Decree that bans imports is inconsistent with Article XI:1.[59] Brazil further argues that none of the three exceptions contained in paragraph 2 of Article XI apply to the ban. Simply put, the ban is an outright prohibition of all imports, supposedly imposed to protect public health. It is not a "standard or regulation for the classification, grading or marketing" of chrysotile or chrysotile products.[60] Moreover, whether chrysotile is a "commodity" within the meaning of this exception is questionable. All three exceptions only relate to agricultural products.

(ii) Article III of the GATT and Article 2.1 of the TBT Agreement

4.41 Brazil argues that the ban is inconsistent with France's national treatment obligations under GATT Article III:4 and Article 2.1 of the TBT Agreement. The national treatment obligations of Article III:4 and TBT Article 2.1 are violated when a law, regulation or requirement (or a technical regulation) that affects the internal sale, offering for sale, purchase, transportation, distribution or use of any imported product, accords less favourable treatment to the imported product than that accorded to "like" domestic products. Each of these criteria are satisfied with respect to the ban. The ban is indisputably a law and its three implementing "Arrêtés" are regulations. For the purpose of Article 2.1 of the TBT Agreement, the ban is a technical regulation. Article 1 of the Decree bans, among other things, the manufacture, processing, sale, offer for sale, distribution and use of all varieties of asbestos fibres and all asbestos-containing products (except for the few temporary exceptions permitted by its Article 2). Thus, it indisputably meets the second criterion for the application of GATT Article III:4

[58]The fact that Article XI applies to the ban is further confirmed by Article XI:2(b) which applies to "import […] prohibitions," among other restrictions. *See also Japan – Trade in Semi-Conductors*, L/6309, adopted 4 May 1988, BISD 35S/126, para. 104 (finding Article XI:1 "comprehensive" and applicable to all types of non-tariff prohibitions).

[59]The applicability of Article XI:1 to such circumstances has been confirmed by various panels under the GATT 1947 and GATT 1994. *See, e.g., United States Manufacturing Clause*, L/5609, adopted 15/16 May 1984, BISD 31S/74, 88, para. 34; *Japan – Trade in Semi-Conductors*, L/6309, adopted 4 May 1988, BISD 35S/116, 152-53, para. 102; *United States – Import Prohibition of Certain Shrimp and Shrimp Products*, WT/DS58/R (15 May , 1998), paras. 7.11 to 7.17.

[60]Brazil notes that a GATT Panel has held that a ban (which, of course, precludes marketing) is not "related" to marketing under Article XI:2(b). *See Canada - Measures Affecting Exports of Unprocessed Herring and Salmon*, L/6268, adopted 22 March 1988, BISD 35S/98, 112, paras. 4.2-4.3 (rejecting Canadian argument that a ban on exports of certain unprocessed fish was related to marketing, and finding that, to fall under Exception Two, the regulation in question must apply to "marketing as such," and that Exception Two does not apply to just any regulation facilitating foreign sales).

and TBT Article 2.1. It provides less-favourable treatment to chrysotile and chrysotile-containing products (which, prior to the ban, were imported from Brazil), than that which it does to French products used as asbestos substitutes (and which are not banned).[61]

4.42 Finally, the ban itself recognizes that the so-called "substitute fibres", and products incorporating them, are "like" chrysotile and chrysotile-containing products. The few exemptions permitted by Article 2 of the ban apply when no substitute fibre is equivalent in terms of its end-use to chrysotile.[62] In other words, whenever a French substitute fibre can replace chrysotile, chrysotile is banned. There can be no more convincing proof that chrysotile and substitute fibres are "like" products. Even if the ban did not by its own terms prove the "likeness" of French substitute fibres to imported chrysotile, analysis of the precedents set under GATT demonstrate that chrysotile and substitute fibres are indeed alike, as are chrysotile and substitute fibre-containing products. Using the criteria identified by the Appellate Body in the case on *Taxes on Alcoholic Beverages* for establishing "likeness"[63], it is self-evident that the end uses of chrysotile and substitute fibres are the same. The fibres are used solely because they emulate the desired characteristics of chrysotile in particular products. With regard to "consumers' tastes and habits", chrysotile and substitute fibres are not consumer goods. They are used solely as inputs in certain products (primarily in various cement products today). Industrial consumers purchase substitute fibres rather than chrysotile based on considerations of cost and availability. They can do so because substitute fibres are intended to emulate chrysotile's characteristics.

4.43 Brazil asserts that the same reasoning applies to the assessment of the products' properties, nature and quality. Substitute fibres are "like" chrysotile precisely because they emulate chrysotile's characteristics. An additional criterion to determine likeness was added after *Border Tax decision – tariff classification.*[64] As previously noted, almost all chrysotile is used as an input into various cement products. Chrysotile and other fibre-cement products are classified under the same Harmonized Tariff System heading (which is number 68.11). In all instances, the six and eight digit classification of chrysotile and other fibre-containing cement products are the same. Therefore, France's conduct violates GATT Article III:4 and the TBT Agreement's Article 2.1, and is inconsistent with France's national treatment obligation.

(iii) Article I of the GATT and Article 2.1 of the TBT Agreement

4.44 The most-favoured-nation obligations of Articles I:1[65] and 2.1 are violated "with respect to all matters referred to in paragraphs 2 and 4 of Article III" (or, for purposes of the TBT Agreement, Article 2.1), whenever any "advantage, favour, privilege or immunity" is granted to a product from one country and is not "accorded immediately and unconditionally" to a "like product" from other WTO Members. This happens to be the case with the French ban. As previously demonstrated, Brazil argues that the ban violates GATT Article III:4 and, for the purposes of Article 2.1 of the TBT Agreement, is a technical regulation. The fact that substitute fibres may be imported into France

[61]According to Brazil, the absence of imports because of the imposition of a ban does not provide a valid basis for asserting that GATT Article III:4 (and TBT Article 2.1) cannot be applied. Interpretations that render a treaty provision null or void, or consign it to "inutility" are to be avoided whenever possible. See *United States – Standards for Reformulated and Conventional Gasoline* (20 May 1996), WT/DS2/AB/R, p. 23.

[62]Brazil notes that the EC acknowledges this when explaining that the French ban does not include chrysotile diaphragms for use in chlorine environments because substitutes cannot safely be used.

[63]*Japan - Taxes on Alcoholic Beverages* (4 October 1996), WT/DS8/AB/R, p. 20, quoting *Report of the Working Party on Border Tax Adjustments* (2 December 1970) BISD 18S/87, 102, para. 18.

[64]Brazil notes that this criterion was first cited in *EEC – Measures on Animal Feed Proteins*, L/4599, adopted 14 March 1978, BISD 25S/49, 63, para. 4.2.

[65]Brazil recognizes that Canada has not alleged a violation of GATT Article I:1. However, as demonstrated, the French ban violates the most-favoured-nation obligations of both that Article and of Article 2.1 of the TBT Agreement.

while chrysotile imports are banned constitutes an "advantage, favour, privilege or immunity." This advantage is accorded to imported substitute fibres but is denied to imported chrysotile, which is banned.

(iv) Article XX of the GATT

4.45 Brazil argues that the General Exceptions of Article XX do not excuse the Decree. To obtain an exception under Article XX, the EC must establish that (i) the ban does not "constitute a means of arbitrary or unjustifiable discrimination between countries where the same conditions prevail", (ii) that it is not a "disguised restriction on international trade", and (iii) that it is "necessary to protect human life or health." The EC cannot argue that the ban meets these conditions. As demonstrated above, the ban discriminates between like products, without advancing its stated objective. Therefore, it is a "means of arbitrary or unjustifiable discrimination". Also, it disadvantages imports of chrysotile, but not imports of man-made fibres. Countries like Brazil (and Canada) produce both chrysotile and substitute fibres. Therefore, the criterion of "discrimination between countries where the same conditions prevail" is obviously satisfied. Similarly, as previously demonstrated, the ban is a "disguised restriction on international trade". Although it masquerades as a measure designed to protect public health, it is an outright product ban that is designed to quell public outrage and advantage domestic and European manufacturers of substitute fibres and products. Moreover, it cannot be argued that the ban is "necessary" to protect human life or health. For these reasons, the EC should not be granted recourse to Article XX.

B. UNITED STATES

1. Introduction

4.46 The United States' submission first discusses the facts concerning the health risks of exposure to chrysotile asbestos, and reduction of these risks through regulation. In this connection, the United States supplies information correcting certain errors and mischaracterizations in Canada's description of the United States regulation of asbestos and the former United States ban and phaseout on asbestos-containing products. United States regulations are not at issue in this proceeding. The United States' submission nevertheless seeks to set the record straight because of Canada's assertions concerning United States policy. Following a factual discussion, it addresses the legal provisions that the Panel has been asked to interpret.

4.47 In the view of the United States, chrysotile asbestos is a toxic material that presents a serious risk to human health. Chrysotile asbestos is no less toxic than other forms of asbestos. A regulatory approach that treats all forms of asbestos on a par with each other is scientifically justified. France, like all other Members of the WTO, has the right to set its own desired level of protection against risks arising from exposure to asbestos, and its regulation on asbestos appears neither discriminatory nor unnecessarily trade restrictive in ensuring that level of protection. The United States currently relies on specified work practices and other controls (including a limited ban) to reduce the risk to human health from asbestos exposure. However, the United States does not consider its approach to be the only appropriate one for regulation of asbestos. Specification of work practices and other controls does not avoid all the risks associated with a hazardous material such as chrysotile asbestos. First, "controlled use" does not eliminate all the risks associated with asbestos. Although it is generally true that asbestos contained in a cement matrix does not present substantial risks while that product is intact, the same is not true during the production, installation, maintenance, removal, or disposal of that product. Second, in many cases a matrix containing asbestos does not remain intact during its useful life. Moreover, while the bulk of Canada's submission focuses on cement-matrix applications, it also acknowledges that chrysotile asbestos is currently used in brake linings and spun fibres for the production of insulating tissues or cords. Significant health risks attend the manufacture and repair of such substances. Finally, even the best work practice is effective only to the extent that it is followed;

accidents, use of improper techniques, and intentional non-compliance are virtually inevitable in the use of these products. For these reasons, France's ban on the manufacture, processing, distribution in commerce, export, import and sale of asbestos and its products appears to be a WTO-consistent response to the risks posed by the use of asbestos.

4.48 As for the legal issues: In the view of the United States, Canada has not met its burden of proof with respect to *any* violation by the French Decree of provisions of the GATT or the Agreement on Technical Barriers to Trade ("TBT Agreement"). In particular, Canada has not shown that imported asbestos and asbestos-containing products are "like products" with respect to substitute fibres and products containing them which are of French origin. As a finding that these products are not "like products" eliminates any violation of Article III:4, and Article XI:1 is simply irrelevant to the analysis of this measure, there does not appear to be any violation of the GATT 1994. With respect to the TBT Agreement, the United States disagrees with the EC position that the TBT Agreement is inapplicable to the French Decree. The Panel should reject the EC's position and find that the Decree is a "technical regulation" within the meaning of Annex 1 of the Agreement; any other result will open up a loophole which could entirely nullify the TBT Agreement. Nevertheless, in the United States view Canada has not proven any violation of Articles 2.2, 2.4, 2.8 or 2.1 of the Agreement. Finally, Canada has not met the particularly high burden of proof for cases of non-violation nullification and impairment.

2. Factual Aspects

4.49 The **United States** argues that asbestos - whether chrysotile or in other forms[66] - is a toxic substance. In United States lexicography, it is a "Class A carcinogen", meaning a substance whose carcinogenic properties have been proven conclusively.[67] The IPCS has reached the same conclusion: "[E]xposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner."[68] The IPCS Report also concludes: "[C]ommercial grades of chrysotile have been associated with an increased risk of pneumoconiosis, lung cancer and mesothelioma in numerous epidemiological studies of exposed workers."[69] In regulating asbestos, the United States treats chrysotile asbestos the same as any other recognized form of the substance.[70] The findings presented by Stayner *et al*.[71] support the decision not to distinguish between chrysotile and other forms of asbestos. This study concluded that it is prudent to treat chrysotile with virtually the same level of concern as the amphibole forms of asbestos, based on the evidence of a significant lung cancer risk, the fact that workers are generally exposed to a mixture of fibres, and the lack of conclusive evidence for the "amphibole hypothesis".[72] More recent confirmation of the hazardous nature of chrysotile was

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[66]The United States notes that its arguments focus on chrysotile asbestos, as that is the subject of the Canadian challenge.

[67]United States Environmental Protection Agency ("EPA"), Integrated Risk Information System (IRIS), Asbestos Substance File (1993) (www.epa.gov/ngispgm3/iris/subst/0371.htm#II) (includes summary of weight-of evidence classification and human carcinogenicity data, including data showing the carcinogenicity of chrysotile asbestos).

[68]*IPCS Environmental Health Criteria 203 – Chrysotile Asbestos*, WHO, 1998, p. 144. (The IPCS document cites numerous studies supporting this conclusion).

[69]*IPCS Environmental Health Criteria 203 – Chrysotile Asbestos*, WHO, 1998, p. 7.

[70]*Airborne Asbestos Health Assessment Update*, p. 118 (EPA, June 1986) (concluding that "while differences in pleural mesothelioma risk attributable to fibre type may exist, they are much less than differences attributable to other factors").

[71]Stayner, L. T., Dankovic, D. A., and Lemen, R. A., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis*, 86 American Journal of Public Health, 179-186, 1996.

[72]The United States notes that the "amphibole hypothesis" postulates that the mesotheliomas among the workers exposed to chrysotile may be explained by confounding exposures to amphiboles, and that chrysotile may have a lower carcinogenic potency than amphiboles.

provided by Landrigan, concluding on the basis of an epidemiological study undertaken in Quebec that "chrysotile asbestos is still indisputably a human carcinogen.[73]" Concerning exposure to asbestos, IARC stated in 1976 that "at present it is not possible to assess whether there is a level of exposure in humans [to asbestos] below which an increased risk of cancer would not occur."[74] The IPCS reaffirmed this conclusion specifically with regard to chrysotile asbestos in 1998, stating: "[N]o threshold has been identified for carcinogenic risks" with regard to chrysotile asbestos.[75] That means that it cannot be assumed that any exposure, no matter how small, to asbestos is safe. Canada questions France's scientific approach, attacking the use of a "linear risk model". The United States takes issue with Canada's criticism of the INSERM Report's use of a linear dose-response model to estimate cancer risk. The use of such a model is entirely appropriate when it comes to estimating the risk of cancer from exposure to asbestos.

4.50 The United States notes that it is not in a position to draw definitive conclusions concerning the regulatory process in France and the actual factual basis for the French Decree. However, generally speaking, regulatory decision-making relating to carcinogens involves two components: risk assessment and risk management. Risk assessment defines the adverse health consequences of exposure to toxic agents. Risk management combines the risk assessment with the directives of regulatory legislation, together with socioeconomic, technical, political, and other considerations, to reach a decision as to whether or how much to control future exposure to the suspected toxic agents.[76] Risk assessments are carried out independently from considerations of the consequences of regulatory action.[77] A risk assessment involves, among other things, quantitative and/or qualitative estimation of risks associated with low levels of exposure to carcinogens. While it is always preferable to rely on human data, epidemiological studies are often either not available or sufficiently definitive, particularly regarding the specific exposure levels involved, and thus often cannot be relied upon as the sole basis for a risk assessment.[78] In addition, because testing of thousands of animals would be necessary in order to have the sensitivity to detect any but large effects, it generally is not practical to measure risks at low exposure levels directly in animal experiments.[79] Accordingly, a number of mathematical models have been developed to extrapolate from high dose animal studies to low human doses.[80]

4.51 In the United States, models or procedures that incorporate low-dose linearity have been adopted when data and information are limited and when there is uncertainty regarding the mechanism of carcinogenic action.[81] While low-dose linearity may not be appropriate for all carcinogenic risk

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[73]Landrigan, P. L., *Asbestos - Still a Carcinogen,* 338 New England Journal of Medicine 1619 (28 May 1998).

[74]*Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, Vol. 14, IARC, 1976, p. 81.

[75]*IPCS Environmental Health Criteria 203 – Chrysotile Asbestos*, WHO, 1998, p. 144.

[76]*Final Guidelines for Carcinogen Risk Assessment*, 51 Federal Register 33992, 33993, col. 3 (EPA, 24 Sept. 1986).

[77]51 Federal Register 33992, 33993, col.3 (24 September 1986).

[78]The United States notes that, as the 1986 EPA carcinogen risk assessment guidelines point out: "It should be recognized that epidemiological studies are inherently capable of detecting only comparatively large increases in the relative risk of cancer. Negative results from such studies cannot prove the absence of carcinogenic action … ". (51 Federal Register 33992 (24 September 1986), pp. 33995-96). Canada's statement that "no epidemiological study to date has detected a higher health risk [than the linear risk model] resulting from low-level exposures" must be viewed in this light.

[79]51 Federal Register 33992 (24 September 1986), p. 33993, col. 3.

[80]51 Federal Register 33992 (24 September 1986), p. 33997. See also EPA Proposed guidelines for carcinogen risk assessment, 61 Federal Register 17960, 17962 (23 April 1996). Although these most recent guidelines are not yet final, they demonstrate that EPA's reassessment of the issues is similar to the approach taken previously.

[81]51 Federal Register 33992 (24 September 1986), p. 33997, col.3.

assessment, it is commonly used in the United States as a default methodology. This methodology is supported by scientific studies and is a reasonably protective approach in the face of uncertainty.[82] The use of a linear model is appropriate for a quantitative estimation of the risks associated with low levels of exposure to asbestos because of the observed linearity of the response in occupational studies. The United States has adopted this approach, in addition, because of the incomplete understanding of how asbestos causes diseases in humans.[83] In assessing the risk from asbestos, EPA notes that "[d]irect evidence for linearity of response with asbestos exposure is available from seven studies (two of the same plant) that compared lung cancer mortality to the cumulative total dust exposure in asbestos workplaces"[citations omitted].[84] Similarly, the limited data that exist for mesothelioma also indicate a linear relationship.[85] The IPCS states: "there was a clear dose-response relationship, with crude rates of mesotheliomas (cases/1000 person-years) ranging from 0.15 for those with cumulative exposures less than 3530 million particles per cubic meter years … to 0.97 for those with exposures of more than 10,590 mpcm-years […].[86]" After identifying and defining the adverse effects of asbestos through the risk assessment process, the next step is to make risk management decisions. A risk management decision, while taking into account the scientific findings of the risk assessment process, also includes a country's choice on whether and how much to regulate a toxic agent. It is at this stage that a country selects measures and regulations that will achieve its chosen level of protection relating to the health of its people.

4.52 In its arguments, Canada has referred to United States regulations concerning asbestos. Because its description of the U.S. regulatory approach is substantially inaccurate, the United States proceeds to set the record straight. The U.S. regulatory approach at present includes a mix of control measures, including bans and required work practices. This approach involves a number of complex statutes, some of which require the consideration of cost, feasibility and other factors besides human health. Almost all control measures are designed to protect workers and building occupants from exposures resulting from contact with asbestos in installed products. Although France's approach to the same problem is different, this different approach is also reasonable under the circumstances.

4.53 Canada references the 1989 rule promulgated by the EPA prohibiting the future manufacture, importation, processing and distribution in commerce of asbestos in almost all products ("the Asbestos Ban and Phase-Out Rule").[87] Several of Canada's statements on this point are factually inaccurate. According to the United States, the Asbestos Ban and Phase-Out Rule was in large part vacated and remanded to EPA by the United States Court of Appeals for the Fifth Circuit in a case entitled *Corrosion Proof Fittings vs. Environmental Protection Agency*[88], based on the court's view that EPA had not appropriately addressed cost-benefit issues. Contrary to the claims made by Canada that EPA was incapable of scientifically justifying its ban, and that the risks posed by asbestos were not supported by scientific facts, the court specifically agreed with EPA's scientific judgment by recognizing that "[a]sbestos is a toxic material, and occupational exposure to asbestos dust can result in mesothelioma, asbestosis, and lung cancer."[89] Indeed, in the record of rulemaking, EPA provided, among other things, a number of scientific studies and reports on the health risks of asbestos, including: Airborne Asbestos Health Assessment Update[90], Report to the United States Consumer Product Safety Commission by the Chronic Hazard Advisory Panel on Asbestos[91], Asbestiform

[82] 61 Federal Register 17960 (23 April 1996), p. 17965.
[83] *IPCS Environmental Health Criteria 203 – Chrysotile Asbestos*, WHO, 1998, p. 7.
[84] *Airborne Asbestos Health Assessment Update*, EPA, June 1986, p. 23.
[85] *Airborne Asbestos Health Assessment Update*, EPA, June 1986, pp. 23-30.
[86] *IPCS Environmental Health Criteria 203 – Chrysotile Asbestos*, WHO, 1998, p. 8.
[87] 54 Federal Register 29460-29513, 12 July 1989.
[88] *Corrosion Proof Fittings v. Environmental Protection Agency*, 947 F.2d 1201 (5th Cir. 1991).
[89] Ibid., p. 1207.
[90] *Airborne Asbestos Health Assessment Update* (EPA, June 1986).
[91] *Chronic Hazard Advisory Panel on Asbestos*, U.S. Consumer Product Safety Commission, July 1983.

Fibres: Non-occupational Health Risks[92] and "Short-term asbestos work exposure and long-term observation.[93]" These studies and reports were discussed in the preamble to the Rule.[94] The court based its decision on procedural flaws in the EPA rulemaking process and on the court's own interpretation of the applicable United States statutory risk/benefit balancing standard for promulgating such rules, not on any disagreement with EPA's findings concerning the health hazards posed by asbestos. After the judicial remand, EPA imposed a more limited ban on asbestos-containing products, including a ban on any new uses of asbestos.[95] That ban remains in place.

4.54 The United States notes that Canada makes much of the argument that asbestos entrained in a cement matrix does not present "detectable risk.[96]" However, Canada's focus ignores the risks presented by asbestos products throughout their life-cycle. The most significant sources of exposure to asbestos, and therefore risk from asbestos-containing products derive from their manufacture, installation, repair, removal, and disposal, including disposal of products containing asbestos in a cement or resin matrix. Moreover, while Canada appears to acknowledge France's concern about protecting health in general terms, it appears to disagree about how protective France should be. By arguing that certain small risks are equivalent to zero risks, the Canadian submission implicitly questions the sovereign authority of a WTO Member to determine the appropriate level of protection for its citizens. What Canada or the United States might consider to be adequate protection in a particular context is not necessarily what other countries must choose. To put it another way, Canada concedes that a ban is acceptable for "certain uses where exposure cannot be controlled to an acceptable degree". The United States agrees, but submits that it is up to each Member to determine what that "acceptable degree" is. Canada indicates that among the most important commercial applications of asbestos are as part of brake linings or clutches and in the form of spun fibres for the production of insulating tissues or cords. In connection with its analysis of "friction products", which include brake linings and clutches, the United States court in the Corrosion Proof Fittings case recognized that "[w]orkers are exposed to asbestos during the manufacture, use, repair and disposal of these products" and that, in the asbestos ban and phase-out rule, "EPA demonstrates that the population exposure to asbestos in this area is great."[97] The court agreed with EPA's determination that friction products containing asbestos pose a risk to human health.[98]

4.55 With respect to Canada's argument that a major commercial application for asbestos is as a reinforcement material for cement, plastic, or rubber, the United States asserts that, in the Asbestos Ban and Phase-Out Rule, EPA made certain determinations respecting worker exposure to these products that were not questioned by the Corrosion Proof Fittings court. EPA determined that the manufacture, installation, repair, and disposal of flat and corrugated asbestos-cement sheet expose

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[92] *Asbestiform Fibres: Non-Occupational Health Risks*, NAS, NRC, 1984.

[93] Seidman, H., Selikoff, I .J., Hammond E. C., *Short-Term Asbestos Work Exposure and Long-Term Observation*, 330 Annals of the New York Academy of Sciences 61-89, 1979.

[94] 54 Federal Register 29460 (12 July 1989), p. 29468-70.

[95] 40 Code of Federal Regulations (CFR) 763.165-763.169 (59 FR 33208, 28 June 1994).

[96] The United States notes that it is not entirely clear what Canada means by the term "undetectable risk". The presence of asbestos fibres in the air or other media can be detectable or undetectable. A risk can be significant, insignificant, or non-existent. It appears that Canada uses the term "undetectable risk" to refer to a risk that Canada deems insignificant. Significance, however, is a judgment call that can only be made by the regulatory authority responsible for public health and safety. It is up to France to determine what level of risk to the French people from asbestos (or any other hazard) is significant.

[97] *Corrosion Proof Fittings v. Environmental Protection Agency*, 947 F.2d 1201 (5th Cir. 1991), p. 1224.

[98] Significant damages have been awarded in U.S. courts with respect to brake applications of asbestos. In 1985, a retired brake mechanic who was dying of mesothelioma won a verdict of $2 million in a court action against Raybestos Manhattan. See McDonald AD, et al., *Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant*, 41 Br J Ind Med 151-157, 1984; Newhouse M. L. and Sullivan K. R., *A Mortality Study of Workers Manufacturing Friction Materials: 1941-86*, 46 Br J Ind. Med, 176-179, 1989.

workers to asbestos.[99] Similarly, EPA determined that the manufacture and installation of asbestos-cement pipe provide "primary routes of exposure" of workers to asbestos from these products, and workers may also be exposed during the removal of asbestos-cement pipe.[100] The United States generally agrees with Canada's assertion that as long as asbestos is held within a cement or resin matrix in an undisturbed state, there is minimal exposure to the fibres - but only while the matrix retains its integrity. Much asbestos has been installed in United States buildings. Because of the high health risk from disturbed building materials and the reduced risk from intact asbestos-containing materials, EPA has issued guidance recommending management of asbestos-containing materials in place.[101] Unfortunately, cement and resin matrices do not remain undisturbed. Putting aside significant releases that occur during the manufacturing process[102], releases of asbestos can occur when, for example, asbestos-cement pipes are installed (which requires cutting the material), and when asbestos-containing material (such as cement) deteriorates, as through peeling, cracking, or crumbling. Fibre release could also result when the material is dry, has the capacity to be crumbled, pulverized or reduced to powder by hand pressure, or is subject to sanding, grinding, cutting or abrading.[103]

4.56 The United States asserts that a cement matrix in which asbestos is bound can undergo a natural process of erosion or degradation resulting in asbestos fibre release: "[T]he release of fibres from external asbestos-cement products [such as siding] due to weathering can be an important external source of asbestos contamination that can be carried into or can infiltrate into the building environment". This has been acknowledged by the 1991 Health Effects Institute-Asbestos Research (HEI-AR) report, *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*.[104] As reported in the HEI-AR document, researchers found that weathered asbestos-cement sheet products washed from gutters and onto walkways were an important source of chrysotile carried by foot or wind into a classroom.[105] The report also cited a research finding of increased ambient air concentrations in the vicinity of buildings with asbestos-cement products on their exterior.[106] Mere maintenance of some asbestos bound in a matrix may disturb the matrix and thereby create additional exposures to the asbestos fibres. For example, in an EPA study[107] conducted at 17 schools in New Jersey involving spray buffing of resilient floor tile containing asbestos, airborne asbestos concentrations were approximately five times higher during than before spray-buffing with high-speed machines, whereas spray-buffing with low speed machines showed a two-fold increase. For school maintenance workers, the maximum estimated eight-hour time-weighted average exposure concentration was 0.093 f/cc. Similarly, routine spray-buffing and wet-stripping as well as ultra-high speed burnishing and wet-stripping of asbestos-containing resilient floor tile can result in elevated

[99]54 Federal Register 29460-29513 (12 July 1989), p. 29491.

[100]54 Federal Register 29460-29513 (12 July 1989), pp. 29496-97.

[101]*Managing Asbestos in Place: A Building Owner's Guide to Operations and Maintenance Programs for Asbestos-Containing Materials*, EPA, July 1990.

[102]The United States notes that, for example, a study of mortality among long-term employees of an Ontario asbestos-cement factory found a substantially increased risk of death from lung cancer and mesothelioma. Finkelstein, M. M., *Mortality Among Long-Term Employees of an Ontario [Canada] Asbestos-Cement Factory*, 40 Br. J. Ind. Med. 138-44, 1983.

[103]The United States notes that this has been recognized by EPA's asbestos National Emission Standard for Hazardous Air Pollutants (NESHAP), promulgated under §112 of the Clean Air Act, 42 U.S.C. 7412. 55 Federal Register 48406, 48408-09 (Nov. 20, 1990), codified at 40 CFR part 61, subpart M.

[104]*Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Health Effects Institute-Asbestos Research Report, 1991, p. 4-32.

[105]Ibid., pp. 4-32 and 4-33.

[106]Ibid., pp. 4-33.

[107]*Project Summary: Airborne Asbestos Concentrations During Buffing of Resilient Floor Tile*, EPA, October 1993, p. 4.

levels of airborne asbestos.[108] Part 2 of the Kominsky study demonstrates that the ultra high-speed burnishing and wet-stripping procedures were associated with a maximum estimated eight-hour time-weighted average exposure concentration of 0.275 f/cc to operations and maintenance staff.[109] Likewise, the HEI-AR Report states that: "buffing, wax stripping, and other abrasive treatments may cause the release of particulate material from the surface of the floor tile".[110]

4.57 The United States' regulatory programme on asbestos is largely aimed at controlling exposure to asbestos when it is no longer bound in a matrix. United States regulations address renovation and demolition of buildings[111] and identification and management of asbestos-containing material in schools.[112] Regulations of the Occupational Safety and Health Administration (OSHA) of the Department of Labor address worker exposures to asbestos including manufacture, installation, renovation, removal and custodial work where workers come into contact with asbestos-containing material. OSHA has established a permissible exposure limit and mandated extensive work practice controls, enclosures, hazard communication, training, medical and other industrial hygiene practices to protect both workers contacting asbestos and other workers who are nearby. Enforcing and complying with these regulations entail a significant commitment of resources by both the public and private sectors. Even within a cement or resin matrix, the risk from asbestos is not negligible. According to an analysis conducted in 1991 by the Health Effects Institute-Asbestos Research[113], janitors, custodians and maintenance workers exposed to ambient asbestos fibre levels of 0.1 f/ml (the permissible exposure limit currently allowed by OSHA regulations) were subject to an estimated increased risk of death from cancer of 2 in 1,000. The same analysis estimated that building occupants (school children and office workers) exposed to airborne asbestos fibres from asbestos-containing materials (presumably many of which, as building materials, would have been encased in cement or resin) have a lifetime cancer risk from such asbestos exposure of 4 to 60 per 1,000,000. Contrary to Canada's suggestion, such risks are not equivalent to zero. Each country must determine for itself what level of protection from risks of exposure to asbestos it wishes to achieve, i.e. what risks to its population it is willing to accept. It is not for another country to tell France that certain risks to its population are not significant. By way of illustration, the United States regulates risks on the order of 1 in 100,000 (1×10^{-5}) or 1 in a million (1×10^{-6}) in a number of instances. Canada concedes that "[t]he principle of controlled use also means that certain uses for which exposure cannot be controlled to an acceptable degree would be banned". The discussion above demonstrates that exposure to asbestos even in a cement or resin matrix cannot be controlled sufficiently to eliminate all risk.

4.58 The United States argues that both of the parties have mischaracterized the substitutes for asbestos and asbestos-containing products. Some products that currently contain asbestos may be manufactured simply by removing the asbestos, thus eliminating any substitution of risk. Alternatively, a wide variety of fibrous substances are being used commercially as replacement materials for asbestos-containing products. These include the man-made mineral fibres (consisting of glass fibres, rock wool, slag wool, refractory ceramic fibres), selected organic fibres (e.g., aramid, carbon/graphite, polyolefin), and several naturally occurring mineral fibres other than asbestos (e.g., wollastonite, sepiolite, palygorskite). The potential health effects for these non-asbestos fibres

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[108]Kominsky J. R., Freyberg R. W., Clark P. J., Edwards A; Wilmoth, R. C., Brackett, K. A., *Asbestos Exposures During Routine Floor Tile Maintenance. Part 1: Spray-Buffing and Wet-Stripping; Part 2: Ultra High Speed Burnishing and Wet-Stripping*, 13 Appl. Occup. Environ Hyg. 101-112 (February 1998).

[109]Ibid., pp. 107-112

[110]*Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Health Effects Institute-Asbestos Research Report, 1991, pp. 4-70.

[111]Asbestos NESHAP, 40 CFR 61.145.

[112]Regulations issued under the Asbestos Hazard Emergency Response Act (AHERA), 15 USC 2641 et seq.: 40 CFR part 763, subpart E.

[113]*Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Health Effects Institute-Asbestos Research Report, 1991, pp. 8-9 - 8-10.

have been evaluated by EPA,[114] the IARC[115] and the IPCS.[116] Although limited health effects information exists for many of these fibres, available data do not indicate that these fibres are as toxic as chrysotile asbestos. For example, none of these fibres have been found to cause either malignant or non-malignant respiratory diseases similar to those associated with asbestos exposure in humans. Unlike asbestos fibres, these substitute fibres have not been classified as carcinogenic to humans or known human carcinogens. The only fibre that has been shown to be more hazardous than asbestos fibres is erionite. Erionite, however, is not known to be available in commerce at this time.[117]

4.59 The United States notes that Canada continually urges that "controlled use" will bring the risk associated with chrysotile asbestos to "undetectable" levels. It also compares the risk from asbestos to that of other products and activities, concluding that many are more risky than asbestos. Canada overstates the efficacy of "controlled use". For example, Canada states that where it is necessary to cut chrysotile-cement materials on site, "the use of tools that almost entirely eliminate emissions (low-speed saws, with water injection or equipped with suction units), and the wearing of a mask by the operator guarantee their safety". United States regulations recognize, however, that masks may not be sufficient in some situations and require the use of a supplied-air respirator which is obviously more cumbersome and costly.[118] When a government makes its choice whether to ban a product or to opt for controlled use, it must necessarily take into account the anticipated effect of the regulations on its population. Beyond the obvious point that "almost" eliminating emissions of asbestos fibres is not the same as eliminating them, it must be acknowledged that 100 per cent compliance with "controlled use" of asbestos is not a realistic expectation due to the burdensome nature of certain work practices concerned. Even the best work practice is effective only to the extent that it is followed; accidents, use of improper techniques, and international non-compliance are virtually inevitable in the use of these products.[119]

4.60 The United States argues that Brazil has made a number of unjustified and inaccurate assertions concerning the U.S. Environmental Protection Agency's former ban on all forms of asbestos and asbestos-containing products in the United States, and a domestic court decision concerning the EPA ban. Of course, this U.S. domestic court did not rule on the consistency of the EPA ban with the TBT Agreement or the GATT. It only dealt with whether EPA had complied with the risk/benefit balancing requirements of the U.S. Toxic Substances Control Act (TSCA). The risk/benefit standard in this U.S. statute is irrelevant to the Panel's present task of determining whether France's ban on asbestos is consistent with the WTO Agreement. France has not adopted such a risk/benefit balancing standard as the basis for a ban. First, Brazil misrepresents the domestic court decision and the situation following, repeating errors made by Canada. The court in 1991 upheld EPA's determination that "[a]sbestos is a toxic material, and occupational exposure to asbestos dust can result in mesothelioma, asbestosis, and lung cancer". The court based its decision not on any disagreement with EPA's findings concerning the health hazards posed by asbestos, but instead on procedural flaws in the EPA rulemaking process and on the Court's own interpretation of the statutory risk/benefit

[114] *Health Hazard Assessment of Non-Asbestos Fibres*, EPA, 1988.

[115] *Man-Made Mineral Fibres and Radon: Monographs on the Evaluation of Carcinogenic Risks to Humans*, Volume 43, pp. 39, 148-52, IARC 1988.

[116] *Asbestos and other Natural Mineral Fibres*, IPCS, 1986, Environmental Health Criteria 53, International Programme on Chemical Safety, World Health Organization, Geneva; *Man-Made Mineral Fibres* (IPCS 1988), Environmental Health Criteria 77, International Programme on Chemical Safety, World Health Organization, Geneva; *Selected Synthetic Organic Fibres*, (IPCS 1993), Environmental Health Criteria 151, International Programme on Chemical Safety, World Health Organization, Geneva.

[117] In addition, see the US answer to question 4 of the EC (contained in Annex II, Section II.A.4).

[118] 40 CFR 763.121(h) (EPA regulations covering employees of certain state and local governments conducting asbestos abatement projects); 29 CFR 1926.1101(g)(2)(v) (OSHA asbestos regulations for construction).

[119] In addition, see the US answer to question 2 of the EC (contained in Annex II, Section II.A.4).

balancing required by TSCA. Because the court agreed with EPA on the health effects of asbestos, EPA did not, after the court decision, need to carry out a "thorough review of scientific and medical data" as a basis to authorize or ban the products Brazil has listed. All EPA did, pursuant to the court's instructions, was to determine which product categories were no longer manufactured, imported, or processed when the rule was issued. For all such products the ban was maintained. EPA also banned new uses of asbestos.[120]

4.61 Second, contrary to what is alleged by Brazil, the United States has not determined that controlled-use policy effectively eliminates the health risk attributable to modern-day uses of chrysotile. Third, Brazil has downplayed the health risk from asbestos by lifting a quotation out of context from the Health Effects Institute report, in a manner that does not accurately reflect the discussion of mathematical models found in this section of the HEI report.[121] The United States addressed these issues as described above at paragraph 4.40. Finally, concerning Brazil's discussion of the French Decree, the United States notes that Brazil concedes that the objective of the Decree - protection of the health of workers and the public - is a legitimate objective within the meaning of Article 2.2 of the TBT Agreement. The United States agrees with Brazil on this point but would also note that France has a right to set its "legitimate objective" to establish the protection of the health of French workers and consumers at the level it deems appropriate. However, when alleging that there is no "rational link" between the French Decree and health protection, stating that the Decree will not make those now sick healthy, and removing it would not make any of those now healthy sick, Brazil conveniently omits the function of the Decree in preventing future exposure and future disease that would result from that exposure.

3. Legal Aspects

4.62 For the reasons below, the United States suggests that the Panel should find that Canada has failed to meet its burden of proof that the French Decree violates any provision of the WTO Agreement. The Panel should also find that Canada has failed to make a showing that the French Decree gives rise to non-violation nullification or impairment of benefits accruing to Canada.

(a) The General Agreement on Tariffs and Trade

(i) Article XI of the GATT

4.63 With respect to Canada's argument that the Decree violates Art. XI:1 because it imposes an absolute prohibition or restriction on imports, the United States agrees with the EC that Article XI is simply not relevant to these proceedings, and the Decree should be analyzed under Article III instead. The Decree regulates characteristics of asbestos and asbestos-containing products. It applies to all asbestos, and is applied to imported products "at the time or point of importation," in the words of the Note *Ad* Article III.

(ii) Article III of the GATT

4.64 With respect to Canada's allegation that the Decree violates the national treatment obligations embodied in GATT Article III:4, the United States argues that, to show a violation of Article III, there must be discrimination - that is, unlike treatment of like products. Yet the relevant domestic and imported products here are not "like products" for the purposes of Article III:4. As the EC has noted, the classic statement of the factors relevant in determining what constitutes a "like product" is found

[120]For the details, see 40 Code of Federal Regulations 763.160, 763.165-763.169 (59 Federal Register 33208 (28 June 1994).

[121]Health Effects Institute – Asbestos Research, *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge*, Cambridge, 1991, pp. 6-9.

in the Working Party report on Border Tax Adjustments of 1968, which defined like products in terms of "the product's end-uses in a given market; consumers' tastes and habits, which change from country to country; the product's properties, nature and quality".[122] The United States generally agrees with the EC's analysis that the properties, nature and quality of asbestos and asbestos-containing products on the one hand, and substitutes on the other, are not "like". The substitutes are by definition substitutable for asbestos and asbestos-containing products for certain uses, but that does not mean that they are "like products".

4.65 According to the United States, Canada has not made the correct product comparison for the purpose of determining whether the relevant products are "like products" under Article III:4. In considering a regulation that bans asbestos and requires the use of substitutes, the relevant products to compare are the following: (i) asbestos must be compared to substitute fibres; and (ii) products containing asbestos must be compared to products that do not contain asbestos but which perform the same function. Where the asbestos elements of a product were inessential, the substitute product may consist of the same product minus the asbestos element (e.g., a kitchen hot pad with thick cotton padding but no asbestos); or the same product redesigned to eliminate the need for asbestos; or a similar product which uses different fibres (e.g., a hot pad made of glass fibre); or a similar product made of what the EC describes as "classic materials" (e.g., a trivet made of cast iron, ceramic or plastic). The physical correspondence between the two classes of products is therefore considerably weaker than Canada has assumed. Canada has failed to show that asbestos and asbestos-containing products and the substitutes have the same "properties, nature and quality". The known severe adverse human health effects of asbestos are another reason why asbestos-containing products are not "like" the substitutes for which adverse health effects have not been demonstrated. The substitute fibres differ considerably in physical structure and properties from chrysotile asbestos and thus cannot be considered "like products." For example, while chrysotile is a naturally occurring mineral which is crystalline in nature, the man-made mineral fibres (MMMF) are amorphous (non-crystalline) silicates which are produced from a liquid melt of different starting materials (e.g., slag, natural rock, glass, clays). Furthermore, unlike chrysotile asbestos, MMMF do not split longitudinally into smaller fibrils of smaller diameter, but may break transversely into shorter segments.[123]

(iii) Article XXIII:1(b) of the GATT

4.66 The United States argues that Canada has failed to meet the special burden imposed by Article 26.1 of the Dispute Settlement Understanding on parties making claims of non-violation nullification or impairment. The United States has been one of the strongest proponents of the non-violation remedy, as an essential safeguard for bargained-for market access rights against frustration by government actions. But the requirements of the non-violation remedy are not satisfied here. As the EC have noted, the text of Article XXIII:1(b) establishes three elements that a complaining party must demonstrate in order to make a cognisable claim under Article XXIII:1(b): (i) application of a measure by a WTO Member; (ii) a benefit accruing under the relevant agreement; and (iii) nullification or impairment of the benefit as a result of the application of the measure. Canada, as the complaining party, has the burden of presenting detailed evidence in support of all three elements. In the present case, there is no dispute that the French Decree is a measure of a Member. The question is simply whether Canada has a legitimate expectation of benefits accruing to it. The precedents are clear that for expectations to be legitimate, they must take into account all measures of the party making the concession that could reasonably have been anticipated at the time of the concession.

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[122]BISD 18S/102, para. 18.

[123]*Man-Made Mineral Fibres*, IPCS Environmental Health Criteria 77, 1988, pp. 11-12; *Man-Made Mineral Fibres and Radon: Monographs on the Evaluation of Carcinogenic Risks to Humans*, Vol. 43, IARC, 1988, pp. 39-53; *Asbestos and Other Natural Mineral Fibres*, Environmental Health Criteria 53, IPCS, 1986, pp. 22-24.

4.67 The United States considers that, as a matter of principle, the Panel should reject the possibility of a finding of non-violation nullification or impairment with respect to health and safety regulations that respond to the development of scientific knowledge concerning health risks. Members do not have a legitimate expectation that regulatory measures will stay static in the face of expanding scientific knowledge concerning health risks, and changing societal decisions concerning the level of acceptable risk. Canada is also in a poor position to argue that an asbestos ban was unforeseeable at the time it negotiated the tariff concessions on asbestos. The dangers posed to human health by asbestos are notorious and have been so for many years. Pliny, the ancient Roman author, described the "diseases of slaves" as including exposure to the textile processes of preparing and weaving asbestos, and even referred to the use of a transparent bladder skin as a respirator to avoid inhalation of dusts by slaves.[124] As of the time of the first GATT negotiating round in 1947, asbestosis had already (in the 1920s) been identified as a distinct condition caused by asbestos.[125] By 1935 asbestosis was widely recognized as a mortal threat affecting a large fraction of those who regularly worked with the material.[126] In addition, by the mid-1940s there were indications that exposure to asbestos in animals and humans was associated with lung tumours.[127] Thus Canada should have reasonably expected subsequent regulatory action (such as a ban) by a GATT contracting party as a result. As of the Dillon Round of 1960-61, Canada had even more reason to foresee the possibility of restrictive regulations of asbestos. An international symposium of experts on the pathogenesis of lung cancer in 1953 published its conclusions and recommendations in a journal which editorialised: "[I]t seems to be beyond discussion that cancer of the lung is sometimes caused by occupational exposure to asbestos."[128] In addition, two major studies had been published in 1955 on cancer in the textile industry demonstrating the relationship between asbestosis and lung cancer.[129] Since the early 1960s, the hazards posed by asbestos - and particularly chrysotile asbestos - have become even more widely known and documented.[130]

(b) The Agreement on Technical Barriers to Trade

4.68 With respect to Canada's arguments that the French ban on asbestos is inconsistent with a number of provisions in the Agreement on Technical Barriers to Trade (TBT), the United States argues that Canada has misread the relevant TBT articles. The interpretation of the TBT Agreement on which Canada's arguments are based attempts to read into the Agreement obligations that do not exist. The United States urge the Panel to reject that interpretation. The EC, on the other hand, have argued that the French Decree is not a "technical regulation" because it is a categorical ban on asbestos and products containing asbestos. They have argued that general bans, and specifically this product ban, are not "technical regulations" because they allegedly do not "lay down product characteristics or their related processes and production methods" within the meaning of paragraph 1 of Annex 1 of the TBT Agreement. The United States disagree with the EC's view on this point. In this instance, the Decree lays down "product characteristics […] with which compliance is mandatory". The characteristics in question are that the product may not contain any asbestos if it is to be marketed, offered for sale, imported, exported, etc. in France. Compliance with the exclusion of asbestos is mandatory except if the French Government has accorded a derogation, in which case adherence to the terms of the derogation is mandatory. In any event, the French Decree is a technical

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[124]Castleman B. I., *Asbestos: Medical and Legal Aspects*, 4th ed., 1996, p. 1.

[125]Lilienfeld, D. E., *The Silence: The Asbestos Industry and Early Occupational Cancer Research - A Case Study*, 81 American Journal of Public Health 791, 792 (1991).

[126]Castleman B. I., *Asbestos: Medical and Legal Aspects*, 4th ed., 1996, p. 39.

[127]Lilienfield, D. E., *The Silence: The Asbestos Industry and Early Occupational Cancer Research – A Case-Study*, 81 Am. J. Pub. Health 791, 794 (1991); Castleman B. I., *Asbestos: Medical and Legal Aspects*, 4th ed., 1996 pp. 53-65, 135.

[128]Castleman B. I., *Asbestos: Medical and Legal Aspects*, 4th ed., 1996 pp. 94, 95.

[129]Ibid. pp. 97, 98.

[130]See *IPCS Environmental Health Criteria 203 – Chrysotile Asbestos*, WHO, 1998, and sources cited therein.

regulation within the meaning of the TBT Agreement and is subject to the substantive rules of the TBT Agreement. The EC's interpretation of Annex 1 would open up a loophole of potentially huge dimensions in the TBT Agreement. Measures having a very significant impact on trade - for instance, regulations limiting the characteristics of spreadable butter or wool - could simply be redefined as product bans. Similarly, the EC argument would mean that a regulation on the safety of infant toys that excluded any parts below a certain size (to prevent choking) would not be a "technical regulation" nor would regulations excluding water from being added to ham. The provisions of the TBT Agreement would then be rendered a nullity. Such a reading of the TBT Agreement is impermissible as a matter of treaty interpretation, and undesirable as a matter of trade policy. This does not mean that the French Decree does not satisfy the requirements of the TBT Agreement, however. As discussed below, although the TBT Agreement applies to the French Decree, Canada has failed to make a case that the French Decree violates any of the provisions it has cited.

(i) Article 2.1 of the TBT Agreement

4.69 The United States argues that, for the reasons discussed in relation to Article III of the GATT, these products are not "like products." Furthermore, since the ban is applied without discrimination as to the source of the product, discrimination between foreign sources is not an issue.

(ii) Article 2.2 of the TBT Agreement

4.70 The United States notes that Article 2.2 provides a key element of the disciplines in the TBT Agreement. From the standpoint of the United States, certain aspects of Article 2.2 are particularly important with respect to health and safety regulation. The first sentence of Article 2.2 is important because it recognizes that in certain circumstances, technical regulations may create necessary obstacles to trade, and the creation of such necessary obstacles is consistent with the TBT Agreement. We note that the "legitimate objectives" enumerated (non-exhaustively) in Article 2.2 specifically include protection of human health or safety. Article 2.2 also recognizes that in assessing the risks that may arise from non-fulfilment of a legitimate objective, a government may consider a number of elements, including available scientific and technical information, related processing technology or the intended end-uses of a product.

4.71 The obligation in Article 2.2 that technical regulations are not to be more trade restrictive than necessary to fulfill a legitimate objective should be interpreted in a manner similar to Article 5.6 of the Agreement on Sanitary and Phytosanitary Measures (SPS).[131] Such a reading is supported by the sixth clause of the Preamble to the TBT Agreement, which provides that: "[R]ecognizing that no country should be prevented from taking measures necessary … for the protection of human, animal or plant life or health, [or] of the environment […] at the levels it considers appropriate […]." The United States argues that the preamble is part of the context of Article 2.2 in the sense of Article 31 of the Vienna Convention on the Law of Treaties, and provides an authoritative indication of the TBT Agreement's object and purpose for the purposes of treaty interpretation. Thus, in order for a Member to show that a government's technical regulation is more trade-restrictive than required, it would need to show that there is another measure that is reasonably available, fulfils the regulating Member's legitimate objectives, and is significantly less restrictive to trade. Accordingly, the complaining party should be required to identify a specific alternative measure that is reasonably available - as a Member is not required to do what is unreasonable. Furthermore, the alternative measure must make a significant difference from a trade perspective. There should be no need to adopt an alternative measure if it makes only an insignificant difference in terms of trade. Most

[131]Article 5.6 provides in relevant part "when establishing or maintaining sanitary or phytosanitary measures to achieve the appropriate level of sanitary or phytosanitary protection, Members shall ensure that such measures are not more trade-restrictive than required to achieve their appropriate level of sanitary or phytosanitary protection, taking into account technical and economic feasibility".

importantly, the complaining party must demonstrate that the alternative measure fulfils the government's objectives. Canada has failed to demonstrate that its preferred alternative to the French ban – i.e. "controlled use" of asbestos and asbestos products – fulfils the French Government's stated "legitimate objective" of protection of human health.

4.72 Canada alleged that the Decree does not address the "true problem" of asbestos in France which Canada identifies as the flocking of asbestos. Yet it is not for Canada to determine what France's "true problem" is. It is up to France to determine what level of protection to afford its citizens. Second, Canada alleges that the French Decree violates Article 2.2 because it fails to acknowledge the "scientific reality" that chrysotile encapsulated in a matrix is harmless. Yet as discussed above and as demonstrated by the EC, encapsulated asbestos is not harmless at all, as the encapsulation can easily be breached, and is likely to be breached during the product's life cycle, resulting in release of fibres and elevated risk to human health. Canada alleges that the French Decree violates Article 2.2 because it replaces use of chrysotile – an allegedly harmless product – with substitutes whose health risks are unknown. The United States fundamentally objects to this reading of the TBT Agreement. Canada is implicitly arguing that any regulatory action negatively affecting trade in a product has to be tested against the hypothetical risks engendered by use of likely alternative products. This test has no basis whatsoever in the TBT Agreement.

(iii) Article 2.4 of the TBT Agreement

4.73 Canada has asserted that Article 2.4 requires a panel to determine: (i) whether a technical regulation on chrysotile is required; (ii) whether there are international standards concerning chrysotile, (iii) whether the international standards are effective and appropriate to achieve the objective; and (iv) whether the Decree is based on international standards. Under this analysis, Canada concludes that France adopted the most restrictive measure possible despite the fact that the international community has developed standards representing a less restrictive approach (i.e., controlled use). This analysis misreads Article 2.4. First and fundamentally, Article 2.4 does not contemplate that a panel determine whether a technical regulation is or is not required. The burden of proof is on Canada to demonstrate that international standards exist and are relevant. In regard to the ILO standard, both the ILO Convention 162 and Recommendation 172 allow participating countries to choose the approach they find to be appropriate to protect workers from asbestos hazards. Indeed, the Provisional Record to the 72nd Session of the International Labour Conference, which adopted Convention 162, states concerning Article 10 of Convention 162 (which Canada now claims to condition a ban on a finding concerning the risk posed by product substitutes): "The Government member of Canada saw nothing in Article 10 that would prevent any country from doing whatever it wanted with respect to asbestos." [132]

4.74 The United States argues that Brazil's interpretation of Article 2.4 of the TBT Agreement ignores the fact that climate, geography and fundamental technological problems are listed as examples, not an exhaustive list, of reasons that an international standard may be an "ineffective and inappropriate means" of achieving a Member's legitimate objective.

C. ZIMBABWE

1. Introduction

4.75 As an important producer and exporter of chrysotile (white) asbestos fibre and products containing chrysotile asbestos and also as a developing country in need of foreign exchange, Zimbabwe argues that it has a substantial interest in the outcome of this proceeding. In fact, the

[132]International Labour Conference Provisional Record, 72nd Session, Geneva, 1986, 29/1: Fourth Item on the Agenda: Safety in the Use of Asbestos, pp. 29/8.

present dispute is of such importance to Zimbabwe's asbestos industry and indeed its whole economy that the Government of Zimbabwe has decided, for the first time ever, to have recourse to the dispute settlement mechanism of the WTO. Zimbabwe is of the view that the ban of chrysotile asbestos and products containing chrysotile asbestos by France is unjustified and contrary to relevant rules of the World Trade Organization (WTO). The ban should therefore be lifted without delay. Zimbabwe believes that it is not incumbent upon it as a third party to this dispute to set out in full the case against the responding party, i.e. the EC. Accordingly, Zimbabwe will limit itself in this submission to the Panel to addressing a number of factual and legal aspects of this dispute that it feels are of particular importance to the outcome of this proceeding. Zimbabwe argues that the complaining party in this case, i.e. Canada, has made a compelling case with respect to both the factual and legal issues in dispute as to why the ban on chrysotile asbestos and products containing chrysotile asbestos is inconsistent with relevant WTO rules and must be withdrawn immediately.

2. Factual Aspects

4.76 **Zimbabwe** asserts that the chrysotile asbestos industry is of great economic importance to its economy. Zimbabwe ranks among the world's largest producers of chrysotile asbestos. In Africa, Zimbabwe is the number one producer of chrysotile asbestos. It produces a high-quality chrysotile asbestos fibre and has sufficient underground reserves for at least another 25 years and infrastructure to continue operations for many more years to come. Chrysotile asbestos currently accounts for about 18 per cent of Zimbabwe's mineral production index of volume and value. Crocidolite (blue) and amosite (brown) asbestos are not mined in Zimbabwe. As a developing African country, Zimbabwe relies primarily on natural resource products and other primary products for much of its export revenue. In terms of the revenue it generates, chrysotile asbestos is second only to gold as far as the mining sector is concerned. As much as 95 per cent of the country's total asbestos fibre production is exported. In 1998, for example, 150,000 tonnes of chrysotile asbestos were exported out of a total production of some 175,000 tonnes, generating foreign exchange in excess of ZW$1.5 billion. In addition to the export of chrysotile asbestos fibres, more than 7,500 tonnes of asbestos-cement products, valued at over ZW$30 million, were exported. Zimbabwe's sole producer of chrysotile asbestos fibre is African Associated Mines. The European Union in general, and Spain and France in particular, have traditionally been important export markets for African Associated Mines.

4.77 Zimbabwe argues that African Associated Mines suffered a dramatic (more than 50 per cent) drop in its sales to France in 1996. The setback suffered by African Associated Mines in the French market is directly attributable to the French Government's actions. It should be pointed out in this connection that in mid-1996 the French Government announced its intention to ban asbestos. Before that, i.e. towards the end of 1995, the French Government had already announced a programme to reduce the risks associated with exposure to asbestos. There is therefore clear evidence that the French ban on asbestos and products containing asbestos has had a direct and damaging impact on Zimbabwe's asbestos industry. The significance of the asbestos industry to Zimbabwe cannot be overstated. The country has immensely benefited from its existence. African Associated Mines directly employs about 6,000 people in Zimbabwe, which amounts to about 20 per cent of total employment in the mining industry. The industry indirectly sustains more than 70,000 people in and around the mining towns of Zvishavane and Mashava. There are no other industries in these towns, meaning that a decline of the asbestos industry would cause dislocation with all its attendant social consequences. It should be borne in mind in this context that the Zimbabwean economy has faced considerable difficulties in the past decade and has not been able to create a sufficient number of new jobs. Out of a labour force of 5 million people, only 1.4 million people are gainfully employed. Apart from generating revenue for the Government of Zimbabwe, the asbestos industry has injected dynamism into the country's economy. In addition to the salaries and wages paid by the companies engaged in the mining and marketing of asbestos and asbestos products, the asbestos industry's more than 300 suppliers of goods and services receive payments of around ZW$600 million each year,

including over ZW$150 million for the state-owned Zimbabwe Electricity Supply Authority (ZESA) and the National Railways of Zimbabwe.

4.78 It is apparent from the foregoing that a ban on asbestos would have severe repercussions for the Zimbabwean economy. In fact, as has been demonstrated, the ban on asbestos by France has already impacted negatively on the Zimbabwean economy. It must be mentioned in this connection that Zimbabwe views with great concern the potentially wider implications of the French ban on the use of chrysotile asbestos. While it is true that most countries, including the United States, still do not generally prohibit the use of chrysotile asbestos or products containing chrysotile asbestos, there is the probability that other governments may be tempted to follow the French example if the French measure were upheld by the WTO. Indeed, the European Union has just announced - without awaiting the outcome of a WTO ruling - that it will move to ban the use of chrysotile asbestos in all its member States.[133] Zimbabwe wished the WTO to be aware of the wider implications of the decision it would render in this dispute.

4.79 Zimbabwe argues that the risks involved in the use of chrysotile asbestos can be adequately controlled. It appears that the concerns that governments have with respect to the use of chrysotile asbestos relate to airborne asbestos dust or respirable asbestos fibres, as they may have an effect on human health. For this reason, the United Nations Environment Programme (UNEP), the International Labour Organization (ILO), and the World Health Organization (WHO), within the Framework of an Inter-Organization Programme for the Sound Management of Chemicals, commissioned a Task Group of international experts to make an evaluation of the risks for human health from exposure to chrysotile asbestos and to make recommendations for health protection and further research. The report of the Task Group was published in 1998.[134] One of the Task Group's main conclusions was that "[e]xposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner".[135] The Group acknowledged, however, that it was not possible to provide quantitative estimates of risks to humans given the dearth of information and data.[136] Furthermore, the Group cautioned that there was a need for further epidemiological studies of populations exposed to pure chrysotile so as to clearly and reliably be able to distinguish between chrysotile and amphibole exposure.[137] In other words, there is a possibility that the available data may actually overestimate the risks to humans from exposure to chrysotile asbestos.[138] What is quite clear from the Task Group's conclusion - and this is crucial - is that the risks to humans are conditional on exposure as well as on doses or concentrations.[139] The key objective for any responsible government must therefore be to reduce exposure. This said, it should be borne in mind that chrysotile asbestos is a natural product. It is present in the air we breathe and in the water we

[133]This information is based on a report by Reuters News Agency, dated 6 May 1999.

[134]WHO, *IPCS Environmental Health Criteria 203 - Chrysotile Asbestos*, Geneva, 1998.

[135]Ibid., p. 144.

[136]Ibid., pp. 7 and 144. Zimbabwe notes that, in this regard, it is misleading for the EC to cite the 1998 Task Group report for the proposition that there is an international consensus that no threshold of exposure can be identified below which no risk to humans exists. The Task Group in fact merely stated that it could not identify any such threshold on the basis of the available data. See WHO, *IPCS Environmental Health Criteria 203 - Chrysotile Asbestos*, Geneva, 1998, pp. 7 and 144.

[137]Ibid., p. 145.

[138]Zimbabwe notes that this is especially true of applications of chrysotile-containing products in industries such as construction, on which the EC has placed great emphasis in its Submission, because "studies have not been in general able to distinguish between chrysotile and amphibole exposure". See WHO, *IPCS Environmental Health Criteria 203 - Chrysotile Asbestos*, Geneva, 1998, pp. 122 and 112.

[139]According to Zimbabwe, the EC confirms this when stating: "[L]es principales données qui ont été présentées illustrent le caractère ubiquitaire de l'amiante en milieu de travail qui peut, à des niveaux d'exposition suffisamment élévés, entraîner de nombreux cas de maladies mortelles".

drink. Exposure is therefore inevitable, and no ban can change that.[140] With these facts at hand, the question arises as to whether the French ban of asbestos is justifiable given the information within the public domain. Zimbabwe believes that what is at the heart of this dispute is the risk of occupational exposure to cement containing chrysotile asbestos. This is because prior to 1997, i.e. before the French ban was implemented, around 90 per cent of French imports of chrysotile asbestos fibres were used for the production of asbestos-cement.

4.80 It is the submission of Zimbabwe that transportation and storage of imported chrysotile asbestos fibre do not entail a risk of exposure provided that there is proper packaging. Another possible activity involving a risk of exposure is the production of chrysotile asbestos-cement itself. In Zimbabwe, this risk has been contained, as demonstrated by the monitoring done by a group of independent experts for Turnall Fibre Cement Company Limited, which is a Zimbabwean company engaged in the manufacture of chrysotile asbestos-cement. The focus of the research has been on the health hazards related to asbestos during the process of manufacture. This research has been going on for more than ten years and so far there have been no reported cases of risks to human life. It should be mentioned here that the EC has adduced as relevant evidence a very recent study by the U.K. Health and Safety Commission which is alleged to demonstrate that notwithstanding the application of control measures, "primary users" of chrysotile asbestos fibres, i.e. workers in asbestos-cement factories, showed a higher mortality rate in relation to asbestos-related lung cancer and mesothelioma. Zimbabwe views this study with considerable scepticism in view of the fact that there are long latency periods involved in the above-named diseases and that the current "cases" go a long way back to a time when the control measures implemented were far less sophisticated than they are now.

4.81 Zimbabwe asserts that a risk of exposure may also be incurred by workers or any person, for that matter, during installation, maintenance and repair of asbestos-containing products. The risks involved in the use of asbestos-containing products can be adequately controlled, even taking into account France's high level of protection against health risks, thus making a ban unnecessary.[141] In fact, the 1998 Task Group report supports this conclusion when stating that "[n]on-friable products and appropriate technological controls greatly reduce fibre release.[142]" It can thus be said that the risk of occupational exposure (i) is a function of the nature of the product and (ii) the risk inherent in that product can in any event be further reduced through appropriate control measures. Regarding the products at issue, i.e. products made from asbestos-cement, the first thing that should be noted is that asbestos-cement does not contain friable asbestos. Moreover, and equally importantly, products made from asbestos-cement are products of high density and thus chrysotile asbestos fibres are firmly blended into the final product. This reduces to a minimum the likelihood of fibres being released into the air and thereby posing a health hazard to human beings. The ILO came to the same conclusion in a report released in 1985: "[l]a manipulation de produits contentant de l'amiante dans lesquels les fibres d'amiante sont solidement fixées dans un liant de telle sorte qu'il ne puisse pas se former de poussières ne présente pas de danger pour la santé.[143]"

4.82 It emerges therefore that when products made from asbestos-cement are used and handled properly, the risks associated with their use are minimal. The recommendation of the 1998 Task Force was to the same effect. It recommended that appropriate control measures be

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[140]WHO, *IPCS Environmental Health Criteria 203 - Chrysotile Asbestos*, Geneva, 1998, pp 2 and 129 et seq.

[141]For a more detailed discussion of this point, see Zimbabwe's arguments with respect to GATT Article XX .

[142]WHO, *IPCS Environmental Health Criteria 203 - Chrysotile Asbestos*, Geneva, 1998, p. 28.

[143]Bureau International du Travail, *La sécurité dans l'utilisation de l'amiante*, Conférence internationale du Travail, Rapport VI (1), 71ème session, 1985, Genève, p. 29.

implemented wherever occupational exposure might occur.[144] Among the control measures which might be used to minimize exposure to chrysotile asbestos are engineering controls, special work practices (including workplace hygiene), and protective equipment, such as technical appliances which eliminate or minimize the formation of asbestos dust, as well as protective respiratory equipment or special protective clothing. That risk control is in fact an effective means of dealing with chrysotile asbestos-related health concerns is borne out by the following passage taken from the report of the 1998 Task Group: "[d]ata from industries where control technologies have been applied have demonstrated the feasibility of controlling exposure to levels generally below 0.5 fibres/ml. Personal protective equipment can further reduce individual exposure where engineering controls and work practices prove insufficient."[145] In light of the foregoing, it is the contention of Zimbabwe that the combined use of high-density products made from asbestos-cement, which inherently are low-risk products, coupled with adequate risk control measures minimize the risk of exposure to asbestos dust. Whatever residual risk may remain does not, in Zimbabwe's view, justify an outright ban on chrysotile asbestos.

3. Legal Aspects

4.83 It is the submission of Zimbabwe that the French ban of chrysotile asbestos is contrary to WTO rules and should be lifted without any delay. It is the view of Zimbabwe that the French Decree constitutes a technical regulation within the meaning of the Agreement on Technical Barriers to Trade. As such, it must be in accordance with Article 2.2 of the TBT Agreement and hence must not be "more trade-restrictive than necessary to fulfil a legitimate objective". By totally banning the import of chrysotile asbestos, the French legislation contravenes the express language of this Article. Furthermore, in the event of the French Decree being found to fall outside the ambit of the TBT Agreement, the Decree contravenes the provisions of GATT Article III:4, as it discriminates against imported asbestos in favour of other like products which are used in France for the same purpose. In the same vein, the French Decree cannot be justified under the terms of GATT Article XX(b), as claimed by the EC.

(a) The Agreement on Technical Barriers to Trade

4.84 Zimbabwe disagrees with the view of the EC that the Decree does not fall within the scope of the TBT Agreement. For a mandatory measure to come within the scope of the TBT Agreement, it must be a "technical regulation". The Decree clearly is a mandatory measure. Notwithstanding the EC's claim to the contrary, it is the submission of Zimbabwe that the Decree, to the extent that it applies to products containing chrysotile asbestos, qualifies as a technical regulation within the meaning of Annex 1 of the TBT Agreement. The argument of the EC that for the TBT Agreement to be applicable, the Decree should have specified which particular products were covered by the ban is without any merit. It is the view of Zimbabwe that such an interpretation is overly restrictive. Annex 1 of the TBT Agreement talks about "product characteristics" in general. Nowhere does it state that the national legislator should adopt only product-specific regulations. Even ignoring this point, Zimbabwe fails to understand why a Member should be precluded from laying down horizontal rules applicable to a group or groups of products which call for the same regulatory approach. In fact, it appears that there would be little merit in forcing Members to specifically enumerate all products covered by a particular regulation when it is in the nature of things that new products would regularly have to be added to the list due to, for example, technological developments. From a public policy perspective, this would seem to be a rather inefficient and costly approach to adopt.

4.85 Zimbabwe argues that the second reason advanced by the EC in support of its argument that the TBT Agreement is not applicable in this case is also without any merit. According to the EC, the

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[144]WHO, *IPCS Environmental Health Criteria 203 - Chrysotile Asbestos*, Geneva, 1998, p. 144.
[145]Ibid., p. 144.

ordinary meaning of the noun "characteristic" supports the view that, for the TBT Agreement to be applicable, product characteristics must be positively defined. Applying this reading of the TBT Agreement to the present case, the EC argues that "not containing chrysotile asbestos" should not be seen as the equivalent of a product characteristic. Zimbabwe finds this reasoning of the EC very tenuous. According to the Shorter Oxford English Dictionary, the noun "characteristic" designates a "distinguishing quality or peculiarity".[146] Zimbabwe believes that, without doing injustice to these terms, a product's "distinguishing quality or peculiarity" can lie in the fact that it does not contain asbestos. The absence of any trace of asbestos clearly sets apart a product in terms of its qualities from another product which contains asbestos.[147] In any event, Annex 1 does not actually require positive product characteristics. Zimbabwe submits that its interpretation of Annex 1 is also in conformity with the relevant context of Annex 1 of the TBT Agreement. All the Agreements annexed to the WTO Agreement are part of the relevant context.[148] Thus, Article 2(f) of the Agreement on Rules of Origin obliges Members to ensure that "their rules of origin are based on a positive standard". From this it follows that where Members wanted to give a special meaning to a term - in this case, to the term "standard" - they used appropriate language to reflect their intention. Members did not adopt that approach as far as Annex 1 of the TBT Agreement is concerned.[149]

4.86 Given the object and purpose of Annex 1 of the TBT Agreement, Zimbabwe wonders what would be the rationale of a rule which compels Members to define product characteristics positively when all they care about is a negative characteristic. Why, for example, should France have to positively define the characteristics of a host of products when its only regulatory concern is with the asbestos contained in those products? It is the submission of Zimbabwe that its interpretation is also in conformity with the jurisprudence of the WTO Appellate Body. Thus, according to the Appellate Body, the term "measure" as it appears in various WTO agreements is to be understood to include a government's failure to act.[150] In other words, a "negative" measure, i.e. a failure to act, counts as a measure no less than a "positive" measure. By token of the same reasoning, the term "characteristics" should encompass negative characteristics. In view of the above reasons, Zimbabwe joins Canada in believing that the general term "product characteristics" lends itself to an interpretation which includes negative characteristics.

4.87 Having demonstrated that the Decree qualifies as a technical regulation under the TBT Agreement to the extent that it bans products containing chrysotile asbestos, Zimbabwe now turns to show that the same is true also with respect to the Decree's ban on the use of chrysotile asbestos fibres as such. The EC has expressed the view that the French ban on the production and importation of chrysotile asbestos fibre is not a technical regulation within the meaning of Annex 1 of the TBT Agreement because, just like the ban on asbestos-containing products, the ban on asbestos fibres is general (rather than specific) and lays down negative characteristics (rather than positive ones) or, for that matter, does not lay down any characteristics. As the issues of specificity and of "positive vs. negative standards" have already been discussed, the following submissions will focus on whether or not the French Decree lays down product characteristics with regard to the ban on asbestos fibres. Zimbabwe contends that the matter is more complex than the EC makes it out to be. To be sure, an independent and isolated ban on sales, say, of all cigarettes would not normally be considered

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[146]The Shorter Oxford English Dictionary on Historical Principles, Oxford, 1993.

[147]In this connection, Zimbabwe notes that it is a truism that it is often easier to define objects negatively than to come up with an exact and exhaustive positive definition.

[148]Panel Report, *Japan – Measures Affecting Agricultural Products*, adopted on 19 March 1999, WT/DS76/R, para. 8.111.

[149]"A treaty interpreter is not entitled to assume that [the use of different words in different places] was merely inadvertent on the part of the Members who negotiated and wrote that Agreement". See *EEC - Measures Concerning Meat and Meat Products (Hormones)*, Appellate Body Report, adopted on 13 February 1998, WT/DS26/AB/R, WT/DS48/AB/R, para. 164.

[150]See *Guatemala – Anti-Dumping Investigation Regarding Portland Cement From Mexico*, Appellate Body Report, adopted on 25 November 1998, WT/DS60/AB/R, footnote 47.

a technical regulation. Yet the situation as it presents itself in this dispute is quite unlike that. As Canada rightly pointed out, unlike cigarettes, asbestos fibres *per se*, i.e. as products in their own right, serve no useful purpose. It is the products containing asbestos fibres which have commercial use and value. By necessary implication, when it comes to dealing with the health hazards of asbestos, the concern of policymakers and the law should be with products containing asbestos fibres, not with asbestos fibres, *per se.* If the products containing asbestos disappear, so will asbestos fibres.

4.88 Zimbabwe considers that the Decree is fully consistent with this straightforward principle. The EC does not contest this. On the contrary, the EC sets out the objective of the Decree as follows: "[l]'interdiction de l'amiante, en France et dans d'autres pays, n'a pas pour objectif de supprimer les quelques 0,0002 fibres/ml qui existent 'naturellement' dans l'air. L'interdiction vise simplement à protéger l'ensemble des travailleurs et des utilisateurs de l'amiante qui sont souvent exposés à des valeurs très supérieures [...] pour des opérations courantes d'intervention sur des matériaux contenant de l'amiante-ciment."[151] The EC explains the rationale of its asbestos-control policy in the following terms: "[l]a politique adoptée en France en 1996 vise en tout premier lieu au remplacement des matériaux contenant de l'amiante par d'autres matériaux sans danger [...]".[152] It clearly emerges from these two quotes that the Decree aims at asbestos-containing products, not at asbestos fibres *per se.* The inference that can be drawn from this is that the import ban - just like the corresponding ban on domestic production - does not perform an independent function, but a subsidiary one. Indeed, the EC expressly states that nothing would change if the import ban - and, by implication, the ban on domestic production - were lifted. Imported and domestically produced asbestos fibres could still not be sold on French territory - because no products containing them could be sold. The following sentence pinpoints this underlying logic of the French ban: "[l]e but est donc bien d'arrêter la diffusion d'amiante le plus en amont possible".[153] The ban on asbestos fibres is thus based on considerations of administrative efficiency, which is arguably only a secondary objective pursued by France. Again, this is confirmed by the EC: "[l]'interdiction d'importation a simplement pour but de rendre plus efficace, en termes de contrôle, l'interdiction d'utilisation [which is France's primary goal]".[154]

4.89 Zimbabwe asserts that, for the foregoing reasons, it should be readily apparent that the ban on chrysotile asbestos fibre is very closely related to the ban on asbestos-containing products. Assuming the ban on chrysotile asbestos fibres could be viewed in isolation, it could possibly be argued that it does not, *stricto sensu*, lay down product characteristics. As Zimbabwe has demonstrated, however, such a line of reasoning is unwarranted and misses the point. The ban on asbestos fibres is an integral part of the Decree. In fact, it is part and parcel of the same Article of the same Decree. Zimbabwe therefore submits that for purposes of this proceeding there is one single, indivisible regulatory package - the Decree - whose consistency the Panel needs to examine with the TBT Agreement. Zimbabwe is of the view that the Decree falls within the ambit of the TBT Agreement. This view is buttressed by the reasoning of another Panel which faced a comparable situation. In the *Kodak/Fuji* film case, the Panel had to decide whether a measure that had not been directly brought up under Article 4 of the DSU could nevertheless be within the Panel's terms of reference. The Panel found that such a measure was not within the Panel's terms of reference, unless it was "subsidiary" or "closely related" to the measure that was properly before the Panel.[155] By way of analogous reasoning, Zimbabwe argues that the French ban on asbestos fibres is "subsidiary" and "so closely related" to the ban on asbestos-containing products - which, as shown, qualifies as a technical

[151]See Section III.B of this Report.
[152]Ibid.
[153]See Section III.C of this Report.
[154]Ibid.
[155]*Japan - Measures Affecting Consumer Photographic Film and Paper*, Panel Report, adopted on 22 April 1998, WT/DS44/R, para. 10.8.

regulation within the meaning of the TBT Agreement - that it can reasonably be found to form an integral part of the latter, and thus constitute a technical regulation in, and of, itself.[156]

4.90 Zimbabwe further submits that treating the ban on asbestos fibres and the ban on asbestos-containing products as separate and "unrelated" could give rise to unreasonable results. Such a situation could in fact arise in the present case. It could be envisaged, for instance, that the ban on asbestos fibres might be found to be consistent with the provisions of the GATT, while the ban on asbestos-containing products might be found to violate the provisions of the TBT Agreement because - to use but one example - it is more trade-restrictive than necessary to fulfil a legitimate governmental objective. Zimbabwe submits that such an outcome would be unreasonable and could undermine the practical effectiveness of the TBT Agreement. Taken to its logical conclusion, such a situation would imply, on the one hand, that France could not produce asbestos-containing products domestically as a result of the ban on imported or domestically-produced asbestos fibres. On the other hand, France would be required to lift its ban on imports of asbestos-containing products and adopt instead a less trade-restrictive measure which, in practice, would mean that a certain quantity of asbestos-containing products would cross the border into French territory. France would thus have no choice but to idly sit and watch as other countries take advantage of the business opportunities offered by the French domestic market. Zimbabwe is of the view that the drafters of the TBT Agreement did not and could not have intended such a result.

4.91 It is therefore the submission of Zimbabwe that the TBT Agreement applies to the French Decree in its entirety, i.e. with regard to the ban on asbestos-containing products as well as the ban on asbestos fibres. The French legislation does not meet the requirements of Article 2.2 of the TBT Agreement, as amply demonstrated by Canada. Zimbabwe adopts the arguments presented by Canada in this connection and would like to support the views expressed therein by also relying on the arguments presented below on whether or not the French measure is necessary within the meaning of GATT Article XX(b).

(b) The General Agreement on Tariffs and Trade

(i) Article III of the GATT

4.92 Zimbabwe argues that, in the alternative, and in addition to the claimed violations of the TBT Agreement, the Decree violates GATT Article III:4. Zimbabwe submits that chrysotile asbestos fibres and, at a minimum, cellulose fibres, aramid fibres and glass fibres are "like products" within the meaning of Article III:4. The EC confirms that cellulose and aramid fibres count among those fibres which are most frequently used to substitute asbestos fibres in the manufacture of cement.[157] Cellulose, aramid and glass fibres are all produced in France.[158] Whereas they may lawfully be sold in that country, the importation and sale of asbestos fibres is prohibited. There is thus no doubt that

[156]Zimbabwe believes that its interpretation of the TBT Agreement also finds support in Article 1.6 of the TBT Agreement. This Article states that "[a]ll references in this Agreement to technical regulations […] shall be construed to include any amendments thereto and any additions to the rules or the product coverage thereof, except amendments and additions thereof, except amendments and additions of an insignificant nature". This provision clearly establishes that the term "technical regulation" as used in the TBT Agreement is not to be given a narrow reading, but one which promotes the Agreement's effectiveness.

[157]Zimbabwe notes that, with regard to glass fibres, this follows from the definition of HS tariff position 68.11.

[158]In Zimbabwe's view it does not matter for the purposes of an inquiry under Article III:4 whether domestic production of the "like product" is substantial or small. Nowhere does Article III:4 lay down a requirement that domestic production needs to be substantial.

asbestos fibres are accorded "less favourable treatment" than cellulose, aramid and glass fibres, despite the fact that they are "like products".[159]

4.93 Zimbabwe notes that the EC contests that asbestos fibres, cellulose, aramid and glass fibres are "like products" within the meaning of Article III:4. It is well established in WTO jurisprudence that the determination of whether or not products are "like products" must be made in accordance with such criteria as the products' physical characteristics and the products' end-use.[160] It is equally clear from WTO jurisprudence that any such determination can only be made on a case-by-case basis, i.e. taking into account the specific and unique circumstances of each case.[161] Regarding the first criterion, i.e. physical characteristics and properties, the EC claims that cellulose, aramid and glass fibres are not sufficiently similar to asbestos fibres in that their chemical composition is different. In this connection, Zimbabwe wishes to recall that the EC has acknowledged that the chemical composition of all varieties of asbestos fibres is different as well. This did not preclude the EC, however, from concluding that chrysotile asbestos fibres and amphibole asbestos fibres were "like products". Zimbabwe submits that the same logic applies and extends to cellulose, aramid and glass fibres.

4.94 Even ignoring the inconsistency in the reasoning of the EC, Zimbabwe does not believe that the differences pointed out by the EC are significant enough to make the relevant products "unlike" within the meaning of Article III:4. Zimbabwe wishes to recall, first of all, that "likeness" does not require that products be "identical in all respects".[162] The second thing that should be noted is that the significance that is attached to differences in physical characteristics depends on the particular circumstances of each case. In this case, as has previously been stated, the starting-point of any analysis must be the fact that chrysotile asbestos fibres, as products in their own right, serve no useful purpose.[163] Chrysotile asbestos fibres are predominantly used as "inputs" in the manufacture of fibre-cement products. It follows that substitute fibres like cellulose, aramid or glass fibres, on the one hand, and asbestos fibres, on the other hand, should not be compared to each other as products in their own right. Instead, asbestos fibres and the relevant substitute fibres should be compared to each other as products incorporated into cement. It is obvious that if this approach is adopted, as it should be, the differences identified by the EC become minor ones and irrelevant. The EC essentially makes the point that cellulose and aramid fibres are, on average, less fibrillose and larger in diameter than asbestos fibres and that only asbestos fibres are internationally recognized as "category I" products, i.e. as products that have been shown to cause cancer. With regard to these alleged varying degrees of health risk associated with the fibres at issue, it should be noted that whatever differences exist between the relevant products become far less relevant when the fibres are blended with other materials to produce cement and other related products.[164] As explained by Zimbabwe, any remaining risks arise from improper handling and manipulation of cement-products and not from the cement-products themselves. Beyond that, Zimbabwe is not convinced that much significance should be

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[159]Zimbabwe considers that the Decree falls obviously within the scope of Article III:4 inasmuch as it is a regulation which affects the internal sale of asbestos fibres.

[160]*Japan – Taxes on Alcoholic Beverages*, Appellate Body Report, adopted on 1 November 1996, WT/DS/8/AB/R, WT/DS10/AB/R, WT/DS11/AB/R, p. 20.

[161]Ibid., p. 20.

[162]*Japan – Taxes on Alcoholic Beverages*, Report of the Panel, adopted on 1 November 1996, WT/DS/8/R, WT/DS10/R, WT/DS11/R, para. 6.21

[163]This is precisely why, in the view of Zimbabwe, the different tariff classification of chrysotile asbestos fibres, on the one hand, and of cellulose and aramid fibres, on the other hand, cannot provide any useful guidance for purposes of determining "likeness" in this case. The Appellate Body has in fact confirmed in its report on *Japan – Taxes on Alcoholic Beverages*, adopted on 1 November 1996, WT/DS/8/AB/R, WT/DS10/AB/R, WT/DS11/AB/R, p. 22, that the value of tariff classification as a criterion for establishing "likeness" must be assessed on a case-by-case basis.

[164]According to Zimbabwe, it should be borne in mind in this context that diameter and fibrillosity are in any event relevant only to the extent that these characteristics correlate with health risks to humans.

attached to the fact that only asbestos fibres are listed by the WHO as a "category I" product. In fact, even the EC concedes that there is a lingering uncertainty about the risks involved in the use of alternative fibres. Zimbabwe submits that the fact that there are to date no known negative effects on human health from the use of alternative fibres does not necessarily mean that they are risk-free.[165] Zimbabwe notes that the EC shares that view, for it expressly acknowledges that "… un risque indétectable n'est pas égal à une absence de risque".[166]

4.95 With regard to the second criterion, i.e. commonality of end-uses, Zimbabwe submits that asbestos, cellulose, aramid and glass fibres serve "substantially identical end-uses".[167] Their chemical resistance and reinforcing capabilities make them almost perfect substitutes for asbestos fibres. It is therefore not the case that chrysotile asbestos fibres are unique products, as the EC would have the Panel believe. As previously noted, the EC, in fact, acknowledges that cellulose and aramid fibres are commonly used substitutes for asbestos fibres.[168] Moreover, like Canada, Zimbabwe believes that the structure of the Decree is at least suggestive of the substitutability of asbestos fibres with other fibres. This becomes clear if the French Decree is seen in terms of the functioning of the political process. If very close substitutes had not been available to the principal users of asbestos fibres at the time the Decree was signed into law, it is reasonable to assume that they would have lobbied the French Government and in all likelihood would have secured a broader exception (allowing the continued use of asbestos fibres) than the one that is now in the Decree.[169] In light of the foregoing considerations Zimbabwe believes that asbestos fibres and cellulose, aramid and glass fibres should be regarded as "like products" within the meaning of GATT Article III:4.

(ii) Article XX of the GATT

4.96 Zimbabwe argues that the Decree is not justified under paragraph (b) of Article XX because it is not "necessary to protect human […] health".[170] More particularly, the Decree does not satisfy the necessity requirement. GATT 1947 case law has established that a measure qualifies as "necessary" within the meaning of Article XX if there is "no alternative measure consistent with the General Agreement, or less inconsistent with it, which [a Member] could reasonably be expected to employ to achieve its […] policy objectives".[171] Zimbabwe believes that it is sufficient for it to establish that - even assuming that asbestos fibres posed more of a health risk to humans - there are less trade-restrictive measures available to France to achieve its health objective. The EC claims that in order for France to achieve its health policy objective there was no measure reasonably available to it other than an outright ban on chrysotile asbestos fibres. In particular, the EC submits that control measures used to minimize exposure to chrysotile asbestos fibres are not sufficient to ensure that France reaches its high level of protection. It also argues that control measures are impracticable in the case of the large group of "secondary users" of asbestos fibres, i.e. those workers and do-it-yourself people who, in the absence of control measures, may be exposed to chrysotile asbestos dust during installation, maintenance and repair of products containing chrysotile asbestos. The

[165]Zimbabwe notes that this is all the more true in view of the fact that the kind of diseases at issue here involve long latency periods.

[166]See Section III.B of this Report.

[167]*United States – Taxes on Petroleum and Certain Imported Substances*, adopted on 17 June 1987, BISD 34S/136, para. 5.1.1.

[168]According to Zimbabwe, the same is true for glass fibres.

[169]Zimbabwe notes that the fact that there is an additional and special temporary exemption in Article 7 of the Decree for certain used and agricultural vehicles precisely suggests that no equivalent and affordable substitutes existed at the time the Decree was signed into law and that the sectors concerned successfully lobbied the Government to provide for a temporary exemption.

[170]Zimbabwe notes that the question to be answered by the Panel here is whether it was necessary for France to discriminate between asbestos fibres and "like" domestic fibres in order to protect human health.

[171]*Thailand - Restrictions on Importation of and Internal Taxes on Cigarettes*, Panel Report adopted on 7 November 1990, BISD 37S/200, para. 75.

problem is compounded, according to the EC, by the fact that in many instances "secondary users" do not have any information as to whether they are dealing with products that contain asbestos. The EC submits that even if they were given that information, control measures are costly and turn what would otherwise be a simple operation into a costly, complicated and awkward one. Furthermore, the EC believes that "une fois mis sur le marché, il n'existe plus aucun moyen raisonnable de contrôler l'usage de l'amiante et, en particulier, de contrôler des opérations banales (découpage, sciage …) que de nombreuses personnes peuvent être amenées à réaliser".[172]

4.97 Zimbabwe is not convinced by the arguments of the EC. First of all, regarding the effectiveness of control measures, Zimbabwe believes that the observance of certain work practices and the use of technical appliances in accordance with the ISO standard 7337, for example, would be sufficient to meet the maximum exposure level acceptable to France. The EC argues that, even where special technical equipment is used when high-risk activities are undertaken, peak exposure levels to asbestos would still exceed the French maximum level. What the EC fails to mention, however, is that, as argued by Canada, the wearing of protective respiratory equipment and humidification of the materials during those activities could significantly reduce the exposure - so much so, in fact, that the respect of the French maximum level of exposure would be ensured. Regarding the argument of the EC that mandatory control measures are impracticable because they are too costly, Zimbabwe contests the relevance of such considerations. After all, whether or not these costs are too high, is a matter to be left to the dictates of the market. If the producers of asbestos-cement face insufficient demand for their products because of expensive control measures imposed on their customers, they will go out of business or diversify into the production of cement using alternative fibres. Likewise, Zimbabwe does not see any merit in the argument that control measures make certain work procedures complicated and awkward. Where certain practices are imposed by law, the question of whether they are appreciated by those who must follow them becomes meaningless.[173] It certainly does not in itself provide a rationale for trade-restrictive measures.

4.98 While Zimbabwe recognizes that it may not be readily apparent to an inexperienced person whether or not he/she is handling a product containing asbestos fibres, it is by no means justification for instituting a far-reaching ban on products which might contain asbestos fibres. It is the contention of Zimbabwe that it would be possible under the WTO legal framework for Members to impose a disclosure requirement, which would enable purchasers to make informed decisions as to whether or not they purchase products containing asbestos fibres. Where the materials have already been installed or incorporated, say, in a building, Zimbabwe does not see why there could not be, for instance, an asbestos warning message next to the evacuation instructions on a notice board of that building. Moreover and specifically with respect to the work of plumbers, electricians and the like, Zimbabwe does not see why the owner of an installation or building could not be required to make available some sort of map which would document in which parts of the installation asbestos is present.[174] With reference to the concern of the EC that the use of asbestos-containing products cannot sufficiently be controlled, especially when it comes to "secondary users" of such products, Zimbabwe again does not think that banning all imports of such products would solve the problem. In fact, it would raise more problems than it would solve. To begin with, if indeed the French Government is so concerned about do-it-yourself users of asbestos-containing products, it could have

[172]See Section III.C of this Report.

[173]Zimbabwe notes that, by way of analogy, it might be added here that many people think that the wearing of seatbelts in cars makes driving more "complicated" and awkward. Yet many countries made the wearing of seatbelt a legal requirement.

[174]In this connection, Zimbabwe recalls that a tenant or house owner who wants to drill a hole in a wall to hang up a painting, for instance, also needs to know exactly where the electrical wiring and installations are, lest he/she wants to put his/her life at risk.

easily banned the sale of such products in all do-it-yourself outlets.[175] Furthermore, as a supporting measure, it could have also restricted the handling of asbestos-made products to certified experts, thus eliminating contact with asbestos by inexperienced people.[176] The protection of workers, such as electricians and plumbers, could also have been ensured relatively easily. The French Government could have, for example, required certification, which would only be bestowed upon an individual once he/she had successfully followed information and training courses on the use and handling of asbestos-containing products. The French Government could also have laid out the precise work practices and technical appliances that must be used in all contacts with asbestos-containing products. To ensure compliance, the regulations could authorize the imposition of heavy fines or a custodial sentence in the event of a wilful disregard of the government's regulations. Needless to say, it is also open to a Member to run information campaigns, so as to raise awareness among workers of the risks of asbestos fibres and the procedures to be observed in all contacts with such fibres. It is clear from the foregoing that the French Government had a number of alternative measures at its disposal which would have interfered less with trade and at the same time would have assisted in realizing its overriding objective of protecting the health and safety of its citizens.

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[175]Zimbabwe notes that, after all, in most countries, drugs cannot be bought in supermarkets, but only in pharmacies upon production of a doctor's prescription.

[176]To give another analogous example, Zimbabwe notes that in many countries the installation of ceiling lamps and other electrical appliances may only be carried out by certified electricians.

V. PANEL'S CONSULTATION WITH SCIENTIFIC EXPERTS

A. DETERMINATION OF THE PROCEDURE

5.1 The **Panel** noted that the dispute before it raised scientific and technical issues. At the first substantive meeting, the Panel informed the parties of its intention to seek the opinion of individual scientific experts except where, in the light of the parties' written rebuttals, it concluded that such a procedure was not necessary. The areas in which the Panel wished to obtain information included the circumstances of exposure to chrysotile asbestos and the associated risks, as well as the effectiveness of the controlled use of chrysotile. The Panel invited the parties to submit their comments to it in writing, particularly regarding the areas on which the experts were to be consulted, the possible approaches to such a procedure and the international or other bodies that could usefully be consulted in order to identify suitable experts.

5.2 In a letter to the Panel dated 14 June 1999, **Canada** proposed, in regard to the possible approaches to a procedure for consultation with individual experts, that five requirements should be met, each one intended to ensure observance of the right of the parties to be heard at all stages of the procedure: (i) the Panel should consult the parties on the choice of scientific experts; (ii) the Panel should seek the opinion of the parties concerning the formulation of the questions to be put to the experts; (iii) the Panel should provide the parties with an opportunity to make written comments on a draft report by each of the experts; (iv) the parties should be able to question each of the experts on the content of his final report at a meeting with the Panel; (v) the parties should be given the possibility to make written comments on the conclusions set out in the final report of each expert and their legal implications. Like the Panel, Canada also believes that the areas on which the scientific experts should be consulted ought to include the circumstances of exposure to chrysotile asbestos and the risks associated with present applications as well as risk management by the controlled use of chrysotile asbestos. The experts should also be consulted in two other areas, namely the comparative toxicity of the different types of asbestos fibres and substitute fibres, and risk assessment methods, including the question of whether there are exposure thresholds below which the risk is undetectable in practice. In Canada's opinion, there are four specializations that in one way or another cover the above-mentioned areas and from which the experts should be drawn. These are toxicology, epidemiology, risk analysis and occupational health. Given the scientific characteristics of the dispute, Canada would wish that each question be submitted to more than one expert, and that each expert submit an individual report. As regards the international institutions that could usefully be approached in order to identify suitable experts, Canada believes that they should be consulted in order to come up with a sampling of experts in the above-mentioned domains. The main selection criteria and hence the best guarantee of impartiality should be that experts must have conducted recognized and independent research into chrysotile asbestos. The international organizations that could be approached included the World Health Organization, the International Labour Office and the International Organization for Standardization. Once a list of prospective candidates has been drawn up with the help of the international organizations, the parties should then be able to submit their own list of names of specialists who could act as scientific experts in the areas mentioned above.

5.3 In a letter dated 14 June 1999, the **European Communities** were of the opinion that the scientific issues raised in this dispute were simple and clear. The DSU rules on the burden of proof also provided the Panel with sufficient guidance in dealing with the factual and scientific issues raised by the parties to the dispute. With respect to the general selection procedures and criteria, the European Communities believed that the Panel's use of experts for obtaining scientific and technical advice should respect the general principles of law. In particular, it should be transparent, avoid conflicts of interest, reinforce the integrity of the dispute settlement mechanism and foster public confidence in the outcome of the dispute. In the view of the European Communities, the Panel can in this case establish only an expert review group under the terms of Appendix 4 to the Dispute

Settlement Understanding. Indeed, the measure at issue in the present dispute is one that must be examined strictly in terms of the GATT 1994, to the exclusion of the Agreement on Technical Barriers to Trade. Article 13:2 of the DSU provides as follows: "… With respect to a factual issue concerning a scientific or other technical matter raised by a party to a dispute, a Panel may request an advisory report in writing from an expert review group. Rules for the establishment of such a group and its procedures are set forth in Appendix 4". The establishment of an expert review group is the only option provided under the DSU for panels wishing to obtain information on scientific matters. The first sentence of Article 13:2 applies only to situations in which a Panel wishes to obtain factual or technical but not scientific information. In their context, the ordinary meaning of the terms, as well as the object and purpose of Article 13:2, first and second sentences, clearly lead to the conclusion that panels are not authorized to deviate from the procedure laid down by Appendix 4 to the Dispute Settlement Understanding. Whether the request comes from a party or arises at the initiative of the Panel itself makes no difference. Strictly scientific matters cannot be resolved by means and/or procedures other than those envisaged in Appendix 4 to the Dispute Settlement Understanding. The *chapeau* to Appendix 4 to the Dispute Settlement Understanding also confirms this interpretation by providing that the rules and procedures set forth in the Appendix "… shall apply to expert review groups established in accordance with the provisions of paragraph 2 of Article 13", that is, regardless of whether the Panel bases itself on the first or second sentence of that Article. This interpretation is supported by the fact that, if the Agreement on Technical Barriers to Trade (TBT) should be applicable (which is not the case), Article 14:2 of that Agreement explicitly prescribes that panels establish only a technical expert group (which is equivalent to an expert review group). In such a case, the procedural rules set forth in Annex 2 to the TBT Agreement must apply. Annex 2 to this latter agreement and Appendix 4 to the Dispute Settlement Understanding are almost identical. Moreover, by virtue of Article 1:2 and Appendix 2 to the DSU, only Article 14:2 of the TBT Agreement is applicable.

5.4 The European Communities also point out that the previous cases in which panels requested the opinion of scientific experts all came under the Agreement on the application of Sanitary and Phytosanitary Measures, which is not applicable in this case. Those previous cases are therefore irrelevant to the present dispute. The dispute concerning *Shrimp* is the only other case for which the opinion of scientific experts was requested under the GATT 1994. But this example *per se* is not enough to set a valid precedent applicable to all cases, especially because the parties to the *Shrimp* dispute apparently did not request the exclusive application of Appendix 4 to the Dispute Settlement Understanding. The result is that, in the present case, should the Panel decide to seek the scientific opinion of external experts, it can do so only under Article 13:2, second sentence of the Dispute Settlement Understanding or under Article 14:2 of the TBT Agreement.

5.5 According to the European Communities, Appendix 4 to the DSU and/or Annex 2 to the TBT Agreement lay down almost identical rules on the establishment of an expert review group. These rules must all be observed in this dispute. Moreover, to ensure that the aforementioned principles are respected, the European Communities believe that the Panel should observe the following specific criteria when choosing scientific experts: (i) the experts should not be citizens of the parties to the Dispute; (ii) the Panel should select scientific experts in different areas of specialization in order to ensure coverage of all the areas identified by it. These areas are: the human health hazards posed by asbestos, especially chrysotile asbestos; the inapplicability of a threshold; the circumstances of exposure and the question as to whether what is known as "controlled use" can eliminate the potential hazards to human health; (iii) the European Communities believe that if the Panel decides to request information, it should consult at least five experts so that more than one expert will have the requisite expertise and provide answers to the questions in the various areas identified by the Panel. In the light of the number of experts that the Panel should consult, only scientists with proven expertise in the realm of asbestos should be selected; (iv) the experts should be drawn mainly if not exclusively from the International Agency for Research on Cancer (IARC), a specialized agency of the WHO. The IARC has studied asbestos from all possible angles and should

therefore be well placed to propose experts covering all the areas in which questions could be posed. The Panel should also explore the possibility of consulting the International Labour Office (ILO) in the event that the IARC is unable to cover all the areas in question; (v) the experts chosen must have no link whatsoever, present or past, with the industry producing asbestos or substitute products. They must furthermore clearly demonstrate the lack of any conflict of interest. The parties should receive at the outset the *Curricula Vitae* of all the candidates proposed and should have at least ten working days in which to verify the skills, expertise and possible conflicts of interest of the candidates; (vi) the Panel should also request the opinion of the parties as to the aim of the consultation with experts, the type and nature of the questions to be put to them; (vii) the aim of the consultation should be to further the knowledge of the scientific considerations germane to this dispute. Therefore, and in accordance with the provisions of the Dispute Settlement Understanding, the questions to be put by the Panel must have a direct and strict bearing only on the scientific aspects of the case. The questions may not relate to legal problems nor to any problem of interpretation of any WTO Agreement under examination.

5.6 Having taken cognizance of the comments from the parties, the **Panel** decided to consult the experts on an individual basis, pursuant to paragraphs 1 and 2, first sentence, of Article 13 of the Understanding on Rules and Procedures Governing the Settlement of Disputes. The Panel convened the parties to a meeting on 10 July 1999 to acquaint them with the procedure it intended to follow and to give them the opportunity to state their opinions on the matter. The Panel recalled Article 13 of the Dispute Settlement Understanding which, among other things, provides that:

> "Each panel shall have the right to seek information and technical advice from any individual or technical body which it deems appropriate." […]
>
> "Panels may seek information from any relevant source and may consult experts to obtain their opinion on certain aspects of the matter."

5.7 At that meeting, the Panel told the parties that, in its opinion, Article 13 of the Dispute Settlement Understanding empowered it to seek such information and technical advice as it deemed fit in a given matter; in particular, a panel was free to determine whether it was necessary or appropriate to establish an expert review group. In the case at hand, the consultation of experts acting in their own right seemed to it to be the most appropriate form of consultation. The Panel intended to seek information concerning the circumstances of chrysotile exposure and the attendant hazards. In the circumstances, the Panel indicated that it would structure its questions around the following main topics: the pathogenicity of chrysotile, the relative pathogenicity of amphiboles, chrysotile and substitute products; the assessment and management of risks associated with the use of chrysotile; the effectiveness of controlled use of chrysotile.

5.8 The Panel then presented to the parties the procedure that it intended to follow, which is the same used by previous panels that had consulted experts selected on an individual basis:

- The experts will be placed under the authority of the Panel. They will be consulted on a personal basis and not as representatives of a government or organization. Their opinion will be strictly in the nature of advice; it will not be binding on the Panel;

- the number of experts to be chosen by the Panel will be decided depending on the number of matters on which an opinion will be sought, as well as the number of matters on which each expert can give an opinion;

- the Panel intends to request names from the World Health Organization (WHO), the International Labour Organization (ILO), the International Programme on Chemical

Safety (IPCS), the International Agency for Research on Cancer (IARC), the International Organization for Standardization (ISO), and from the parties;

- the Panel does not intend to appoint experts who are citizens of one or other of the parties to the dispute, unless the parties consent to their appointment or the Panel believes that it would otherwise be impossible for it to secure the specialized scientific advice needed;

- the Secretariat will request the persons suggested to submit a *curriculum vitae*. The *curricula vitae* will be transmitted to the parties. The parties may not establish contact with the experts suggested;

- the parties will have an opportunity to make comments and to state any major objections they may have to any expert under consideration. The Panel will inform the parties of the experts it chooses;

- the experts will receive all the relevant elements of the communications on a confidential basis;

- the Panel will prepare draft questions for the experts. They will be communicated to the parties. The parties will have the opportunity to comment on the questions proposed or to suggest additional questions before they are sent to the experts. The Panel will then draw up a definitive list of questions which will be sent to the experts and simultaneously to the parties;

- each expert will receive all the questions. He will be requested to reply to the questions falling within his sphere of competence and, if necessary, to indicate the areas on which he does not feel competent to reply. The experts will be invited to provide written answers; copies of those answers will be transmitted to the parties. The parties will have an opportunity to make written comments on the replies from the experts and the replies will be included in the Panel's final report;

- should the Panel deem it fitting, either on its own initiative or at the request of a party, a meeting may be held with the experts immediately before the second substantive meeting. Before the meeting, the Panel will ensure that: (i) experts are made privy to the parties' comments on their replies; (ii) the experts each receive the replies of the other experts to the Panel's questions;

- the minutes of the meeting with the experts will be submitted to the parties and to the experts so that they may make corrections. The corrected version will be attached to the Panel's final report.

5.9 The Panel gave the parties the opportunity to transmit their written comments to it.

5.10 In a letter dated 19 July 1999, **Canada** recalled all the points that it had notified to the Panel in its letter of 14 June 1999. Canada agrees with the Panel as to the nature of the information and advice that it intends to seek from the scientific experts. It nevertheless believes that the experts best qualified to reply to the Panel's questions concerning the circumstances of exposure to chrysotile and the associated hazards are to be found in the areas of toxicology, epidemiology, risk assessment and occupational safety. In addition to the opportunity given to the parties to make written comments on the experts' replies, the Panel should also provide for the possibility of a final written submission by the parties following the second substantive meeting. As regards the stipulation that the scientific experts may not be citizens of any of the parties to the dispute, Canada believes that this procedural

rule, established in Appendix 4 to the Dispute Settlement Understanding, normally applies only to the establishment of an expert review group. In the *Hormones* case, the Appellate Body stated in that connection: " … once the Panel has decided to request the opinion of individual scientific experts, there [was] no legal obstacle to the Panel drawing up, in consultation with the parties to the dispute, ad hoc rules for those particular proceedings".[1] As the agreement of the two parties to the dispute is required if the selection of citizens of one of the parties is to be allowed, Canada is surprised at the refusal of the European Communities to allow the selection of their citizens. Canada is prepared to consider the selection of experts who are citizens of the European Communities despite the refusal of the European Communities to consider experts from Canada. In this dispute, if the citizens of the parties are automatically excluded, the Panel risks facing a situation in which it will be unable to select the experts with the best scientific knowledge considering the nature of the advice being sought. Canada therefore requests the European Communities and the Panel to reconsider their decision with regard to the non-participation of citizens of the parties.

5.11 Moreover, Canada cannot accept that, as demanded by the European Communities, the experts must clearly demonstrate the absence of any conflict of interest. It is not incumbent upon a prospective expert to prove his impartiality, instead he is merely required to fill out a disclosure form concerning his interests, relationships and any matters that may affect his independence. This form is provided for in the document entitled *Rules of Conduct for the Understanding on Rules and Procedures Governing the Settlement of Disputes*.[2] Once the persons approached as potential experts have filled out their disclosure forms, the parties to the dispute may oppose any candidate who has disclosed an interest, relationship or matter that may place him in a situation of conflict of interest. The Panel is empowered to decide whether the information disclosed in the form really places a candidate expert in a situation of conflict of interest and to uphold a party's objection to an expert's candidature. The approach taken by the Panel in the *Shrimp* case should be followed in this instance. Having noted that in their disclosure forms three of the experts approached had disclosed what might be considered as potential conflicts of interest, the Panel nevertheless decided to confirm their appointments "being of the view that the disclosed information was not of such a nature as to prevent the individuals concerned from being impartial in providing the scientific information expected of them. The Panel has also taken into account the disclosed information when evaluating the answers provided. The Panel underlined that, in making its choice, it had been guided primarily by the need to gather expertise of the best quality and covering as wide a field as possible. In [the circumstances specific to this case], it was difficult – if not impossible – to reconcile this need with an agreement by all the parties to the dispute on each and every individual concerned".[3] Canada is surprised at the European Communities' insistence on the absence of any link between the experts and producers of chrysotile asbestos but not between the experts and anti-asbestos pressure groups. No one opposes the principles of independence and impartiality of experts or the observance of the rules on conflicts of interest. The single pertinent consideration remains the way in which these principles should be applied in this particular instance.

5.12 In a letter dated 19 July 1999, the **European Communities** took note of the Panel's decision to consult individual scientific experts pursuant to Article 13:1 of the Dispute Settlement Understanding. The European Communities contest the legal basis of the Panel's decision. Under the international customary principles of treaty interpretation, a systematic interpretation of Articles 13:1 and 13:2 of the Dispute Settlement Understanding suggests that as far as scientific matters are concerned, the preferred option in the Dispute Settlement Understanding is the establishment of an expert review group. The term "scientific matter" appears only in the second sentence of Article 13:2

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[1] *EC Measures Concerning Meat and Meat Products (Hormones)* Report of the Appellate Body, WT/DS26/DS48/AB/R, adopted on 13 February 1998, para. 148.

[2] WT/DSB/RC/1, of 11 December 1996.

[3] *United States – Import Prohibition of Certain Shrimp and Shrimp Products,* Report of the Panel, WT/DS58/R, adopted on 6 November 1998, para. 5.7.

of the Dispute Settlement Understanding, which envisages only the constitution of an expert review group. The drafting history of the WTO Agreements also confirms this interpretation.[4] The three previous cases in which panels sought the opinion of scientists in their own right all had to do with matters arising under the SPS Agreement, Article 11:2 of which expressly mentions "scientific" matters and envisages the possibility of consulting experts individually.[5] Canada furthermore requests that the TBT Agreement be applied to the measure at issue here. It is worth noting that Article 14.2 of the TBT Agreement provides only for the possibility of consulting a technical expert group. This Agreement contains no provision equivalent to Article 13:1 of the Dispute Settlement Understanding or to Article 11:2 (first sentence) of the SPS Agreement. The very terms of Article 14.2 of the TBT Agreement are therefore different from Articles 13:1 and 13:2 (first sentence) of the Dispute Settlement Understanding and from Article 11:2 of the SPS Agreement. This difference is not accidental.[6] It denotes the clear intention of the WTO Members to settle scientific or technical matters in the framework of the TBT Agreement only by establishing an expert review group. The decision of the Panel to consult experts on a personal basis is also contrary to Article 1:2 of the Dispute Settlement Understanding, which provides as follows:

> "To the extent that there is a difference between the rules and procedures of this Understanding and the special or additional rules and procedures set forth in Appendix 2, the special and additional rules and procedures in Appendix 2 shall prevail."

5.13 As explained above, there is a clear difference between Article 13:1 and 13:2 (first sentence) of the DSU, invoked in this case by the Panel, and Article 14:2 of the TBT Agreement. The special rules and procedures mentioned in Appendix 2 to the DSU, namely Article 14:2 of the TBT Agreement, which provides for the establishment of a technical expert group, should thus be applied in the present case, should the Panel judge the TBT Agreement to be applicable.[7] Therefore, the European Communities consider the Panel's decision contrary to the letter, object and purpose of Article 14:2 of the TBT Agreement (if the latter is applicable), in conjunction with Article 1:2 of the DSU, and to Article 13:2 (second sentence) of the DSU. Besides, from a systematic point of view, the Panel's decision renders useless and obsolete the provisions of the Dispute Settlement Understanding and of the TBT Agreement regarding expert review groups, which are clearly the option preferred by WTO Members and the only one for which rules of procedure have been drawn up in the WTO for the settlement of "scientific" questions.[8] At this stage, the European Communities are therefore obliged to reserve all their rights on this issue. They would also request the Panel, in keeping with current WTO practice and for the sake of transparency and due process, to state in writing the criteria and the reasons for its decision to call on individual scientific experts and the reasons for which is has not entertained the arguments put forward by the European Communities, and to communicate this information to the parties to the dispute.

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[4] According to the European Communities, confirmation can also be found in the *chapeau* to Appendix 4 to the Dispute Settlement Understanding which provides that "[the following rules and procedures] shall apply to expert review groups established in accordance with the provisions of paragraph 13", that is, regardless of whether it is the first or the second sentence of this Article that is being used by the Panel.

[5] That also explains the Appellate Body's reasons for its finding on that matter in the *Hormones* case. See the report AB/1997-4, para. 147.

[6] As the Appellate Body found in *Hormones* (para. 164), "a treaty interpreter is not entitled to assume that such usage was merely inadvertent on the part of the Members who negotiated and wrote that Agreement".

[7] The third sentence of Article 1:2 of the Dispute Settlement Understanding is not applicable in this case, as the GATT 1994 does not contain contradictory rules and procedures on this matter.

[8] The Panel's interpretation is also contrary to one of the corollaries of the general rule of interpretation set out in the 1969 Vienna Convention, which is that the interpretation must give meaning and effect to all the terms of a treaty. As held by the Appellate Body in the *Gasoline* case "an interpreter is not free to adopt a reading that would result in reducing whole clauses or paragraphs of a treaty to redundancy or inutility" (AB-1996-1, page 22). Specifically, the Panel has so far refrained from providing explicit substantive reasons for its choice of consultation with individual experts over the establishment of an expert review group.

5.14 As regards the type of scientific background and specializations, the European Communities take the view that the experts should be cancer specialists, in particular in lung cancer and mesothelioma. They should also be epidemiologists experienced in the area of asbestos and cancer. The European Communities are not clear as to what type of scientific discipline would encompass those persons who would be required to provide advice regarding "risk evaluation and management in the use of chrysotile" and "the effectiveness of the controlled use of chrysotile", nor what type of technical expertise they should have. If such experts exist, they should be able to provide information about all the categories of persons who could come into contact with asbestos and asbestos-containing products, such as those working in maintenance, repair and construction (for example, carpenters, plumbers, heat repairers, workers in insulating materials, do-it-yourself enthusiasts, etc.). The European Communities believe that the scientists chosen should also have expertise in the inspection of houses, buildings and factories for the presence and possible removal of asbestos. Obviously, such experts cannot be allowed to have any link, whether direct or indirect, with the industries producing asbestos or those producing the equipment for reducing the risk of asbestos fibre inhalation. Such a link would seem particularly possible if the experts were to be designated by the ISO. The European Communities consider that at least two experts should be designated for each scientific domain and each area of questions. That is a minimum prerequisite for a balanced view and for not being entirely dependent on the views of just one person. At all events, the overall number of experts should not be less than six.

5.15 The European Communities have expressed their wish to receive copies of the letters to be sent by the Panel to the aforementioned institutions under this point and of their replies. The experts appointed should not be nationals or residents of the parties to the dispute. The European Communities consider that all the candidates must submit a detailed *curriculum vitae* in time so as to enable the parties to verify their scientific credentials, experience and independence. The candidates must therefore clearly indicate in their *curriculum vitae* whether in the course of their professional life they have worked for or provided advice, in whatever form, to the industries producing asbestos, asbestos-containing products and substitute products or to the industry producing "controlled use" equipment. In addition, the selected experts must complete a disclosure form concerning potential conflict of interest, pursuant to the *Rules of Conduct for the Understanding on Rules and Procedures Governing the Settlement of Disputes* adopted (WT/AB/WP/3, Annex II, page 16, 28 February 1997). The disclosure form must contain all the information indicated in the illustrative list appearing in Annex II to the *Rules of Conduct* mentioned above. It should also explicitly contain information as to whether the expert has done any type of paid or unpaid work (scientific research, consulting, expert advice, participation in the board of directors or board of management, etc.) for the enterprises engaged in the extraction, production, processing of or trade in asbestos, asbestos-containing products or substitute products, or for enterprises producing the equipment intended for "controlled use".

5.16 It is the opinion of the European Communities, that the Panel should, for example, request that the disclosure form further indicate: (i) the expert's professional situation (job in an enterprise or institute connected to the asbestos, substitute products or "controlled use" equipment industries); (ii) whether the expert is a member of the board of directors, board of management or any other supervisory body within an enterprise, association, institution or interest group linked with the industries producing asbestos, substitute products or equipment for "controlled use"; (iii) whether he has conducted scientific research or provided expert advice at the request of or under contract to an enterprise, association, institution or interest group connected with the industries producing asbestos, substitute products or a "controlled use" equipment.[9] If the aforementioned clarifications and

[9] As far as the European Communities are concerned, additional support for the proposition that the term conflict of "interest" should be interpreted as broadly as possible may be drawn from Article III.1 of the *Rules of Conduct* mentioned above, and in a systematic interpretation (by analogy) of the following provisions:

information are not given in the *curriculum vitae* and in the disclosure form, the parties will not be in a position to exercise their rights and make the type of comments being requested of them by the Panel. Therefore, the European Communities consider that the issue of the scientific credentials, experience and, in particular, that of the independence and impartiality of the experts, are of paramount importance and that they need to be reflected in the Panel's decision on the selection and consultation of scientific experts. They therefore wish to reserve their rights until completion of the selection procedures. The parties should be allowed sufficient time to enable them to make effectively known to the Panel their views on the above issues. Specifically, they should be given sufficient time to make known their views on the list of potential experts to be chosen by the Panel and to submit their comments on the written replies from the experts to the questions put to them by the Panel.

5.17 In a letter to the parties dated 2 August 1999, the **Panel** confirmed its intention to consult experts individually, in application of Article 13 of the DSU. The Panel carefully examined the arguments advanced by the parties concerning the expert consultation procedures, in particular, the European Community argument that Article 13.2 of the DSU Agreement requires the constitution of a technical expert group as envisaged in Appendix 4 to the DSU for the purposes of consultation with experts on scientific matters. Article 13 of the DSU provides, among other things, that "each Panel shall have the right to seek information and take advice from any individual or body which it deems appropriate" and that "Panels may seek information from any relevant source and may consult experts to obtain their opinion on certain aspects of the matter". In addition, Article 13.2 prescribes that panels "may" request an advisory report in writing from an expert review group specifically though not exclusively to examine a factual issue concerning a scientific matter. The Panel deems this text to allow for the establishment of such an expert group, while not ruling out consultation of experts on an individual basis, both with regard to a scientific matter "or other technical matter". This interpretation of Article 13:2 of the DSU seems to the Panel to be perfectly in line with the text of this provision, interpreted in accordance with Article 31 of the Vienna Convention on the Law of Treaties, and with the interpretation given by the Appellate Body that Article 13 of the DSU does not prevent panels from consulting with individual experts and leaves to the sound discretion of a panel the determination of whether the establishment of an expert review group is necessary or appropriate.[10]

5.18 The Panel also considered the European Community's argument that, if the measure at issue should be deemed to fall under the TBT Agreement, which the Communities contest, Article 14.2 of that Agreement would require the establishment of an expert review group for any scientific or technical matter, and the EC position that pursuant to Article 1:2 of the DSU, that provision would prevail over those of Article 13 to the DSU. Article 14:2 of the TBT Agreement is among the provisions mentioned in Appendix 2 to the DSU and which, under Article 1:2 of that Understanding, will prevail over the provisions of the Understanding to the extent that there is a difference between the two. The Panel notes, however, that it is only "to the extent that there is a difference" between the rules and procedures of the Understanding and a special or additional rule or procedure in Appendix 2 to the DSU that the latter will prevail. Yet, as stated by the Appellate Body, it is only where the provisions of the DSU and the special or additional rules of Appendix 2 cannot be read as complementing each other that the special or additional provisions will prevail over those of the DSU,

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Articles 8:2, 8:3 and 17:3 of the DSU, paras. 2 and 3 of Appendix 4 to the DSU, as well as paras. 2 and 3 of Annex 2 to the TBT Agreement.

[10] See the Reports of the Appellate Body in *European Communities – Measures Concerning Meat Products (Hormones)* (WT/DS26/26-DS48/AB/R), para. 147 (" … in disputes involving scientific or technical issues, neither Article 11.2 of the SPS Agreement, nor Article 13 of the DSU prevents panels from consulting with individual experts. Rather, both the SPS Agreement and the DSU leave to the sound discretion of a panel the determination of whether the establishment of an expert review group is necessary or appropriate") and *Argentina – Measures Affecting Imports of Footwear, Textiles, Apparel and Other Items* (WT/DS56/AB/R) para. 84 ("Article 13 of the DSU enables a panel to seek information and technical advice as it deems appropriate in a particular case, and (…) the DSU leaves 'to the sound discretion of a panel the determination of whether the establishment of an expert review group is necessary or appropriate'.").

that is, in a situation where the two provisions would be mutually incompatible.[11] In the present case, Article 14:2 of the TBT Agreement provides that a panel "may" establish a technical expert group. Like Article 13:2 of the DSU, this text envisages the possibility of establishing a technical expert group and lays down the procedures that would be applicable in the event. Nevertheless, it does not exclusively prescribe the establishment of a technical expert group, and this possibility, in our opinion, is not incompatible with the general authorization given under Article 13 of the DSU to consult with individual experts. The two provisions can be read as complementing each other.

5.19 The Panel believes that in this case the consultation of experts on an individual basis is the more appropriate form of consultation, inasmuch as it is the one that will better enable the panel usefully to gather opinions and information on the scientific or technical issues raised by this dispute. Considering in particular the range of areas of competence that might be required, it is appropriate in this case to gather information and different individual opinions rather than asking for a collective report on the various scientific or technical matters in question. In the light of the foregoing, the Panel wishes to underline that its decision to consult experts on an individual basis is without prejudice to the applicability of the TBT Agreement to the measure in question, on which the parties disagree.

B. SELECTION OF EXPERTS

5.20 The Panel has requested the assistance of five institutions in identifying experts. The institutions concerned are the World Health Organization (WHO), the International Labour Organization (ILO), the International Programme on Chemical Safety (IPCS), the International Agency for Research on Cancer (IARC) and the International Organization for Standardization (ISO). The parties have also submitted names to the Panel. The Secretariat then requested those of the proposed experts who were prepared to participate to submit to it a detailed *curriculum vitae.* Those *curricula vitae* were forwarded to the parties, who were able to convey to the panel their comments concerning the potential experts and to indicate, where appropriate, whether they had any major objections to any of them. Upon careful examination of the *curricula vitae* and the comments of the parties, the Panel accepted the following four experts, whose nominations were not opposed by the parties:

- Dr. Nicholas H. de Klerk, Senior Research Fellow, Department of Public Health, University of Western Australia, Australia;

- Dr. Douglas W. Henderson, Professor of Pathology, Head of the Department of Anatomical Pathology, Flinders Medical Center and The Flinders University of South Australia, Australia;

- Dr. Peter F. Infante, Director, Office of Standards Review, Health Standards Programme, Occupational Safety and Health Administration, Washington D.C., United States;

- Dr. Arthur W. Musk, Clinical Professor of Medicine and Public Health, University of Western Australia, and Physician, Department of Respiratory Medicine, Sir Charles Gairdner Hospital, Nedlands, Australia.

5.21 The experts were asked to acquaint themselves with the *Rules of Conduct for the Understanding on Rules and Procedures Governing the Settlement of Disputes*[12], paying special

[11] See the Report of the Appellate Body in *Guatemala – Anti-Dumping Investigation Regarding Portland Cement from Mexico* (WT/DS60/AB/R), paras. 65 and 66.

[12] WT/AB/WP/3, of 28 February 1997.

attention to Annex 2 (Illustrative list of information to be disclosed). No expert has disclosed any circumstance that could be considered as the potential source of a conflict of interest.

5.22 In consultation with the parties, the Panel prepared precise questions which it submitted to each expert individually. The experts were requested to answer only those questions that they considered to be within their domain(s) of competence. Written communications by the parties, transcriptions of their oral statements, as well as the references they submitted to the Panel were transmitted to the experts for their information. The written answers from the experts have been forwarded to the parties, who have had a chance to comment on them. The questions posed by the Panel and the answers given by the experts are contained in section V.C. The observations of the parties are reproduced in section V.D.

5.23 On 17 January 2000, the experts were invited to discuss with the Panel and the parties their written answers to the questions and to provide additional information. Annex VI to this report contains the minutes of the meeting.

C. QUESTIONS BY THE PANEL AND COMMENTS BY THE SCIENTIFIC EXPERTS

5.24 The Panel requested the experts to comment on the areas of difference between the parties highlighted in the first paragraph of each question, as well as to address the specific points listed. The Panel encouraged the experts to indicate, to the extent possible, key points on which they considered that (i) there is scientific proof, (ii) there is broad agreement among experts, (iii) there is uncertainty and/or a range of divergent opinions among experts.

1. Introductory Comments by Dr. Henderson

(a) Introduction

5.25 This introduction sets out a general summary of prevailing knowledge and uncertainties on asbestos-related disorders, with emphasis on mesothelioma and lung cancer, together with discussion of both the amphiboles and commercial chrysotile, patterns of exposure, and some brief details of *in vivo* and *in vitro* experimental studies.

5.26 This introduction has two purposes: (i) to provide a general background and broad perspective to the questions and answers that follow; and (ii) to correct some inaccuracies and errors in the documentation supplied already to the WTO. In so doing, I have tried to broaden the perspective beyond the classical Canadian studies on the Quebec chrysotile miners and millers, and beyond the INSERM Report. A number of the general discussions in this introduction have been truncated after the issue has been put into context, and some of these discussions are then continued and amplified in my specific responses to the questions. This has produced some iteration of some points, but I believe that the advantages - avoidance of the potential for distortion created by answers without adequate background information - outweigh any disadvantages. The division of my report into these sections also provides an opportunity to indicate the relative importance of epidemiological studies versus *in vivo* or *in vitro* experimental models in the formulation of my opinions and answers.

5.27 At the outset, I emphasize that Australia (including Western Australia) is no longer an asbestos producer. Production of crocidolite at the Wittenoom blue asbestos industry stopped in 1966. None has been produced or exported since. Crocidolite was used in asbestos-cement products in Australia until 1966 when its use was discontinued, but imported amosite was used in these products until 1984 [NICNAS 99][13]. The use of chrysotile in fibro-cement products was discontinued in 1987.

[13]For complete references, see Annex III to the Panel Report.

5.28 As stated repeatedly in the documentation provided to the WTO, asbestos has the capacity to induce at least five benign pleuropulmonary disorders, and two cancers: parietal pleural fibrous plaques; benign asbestos pleuritis with effusion; diffuse pleural fibrosis; rounded atelectasis; asbestosis; primary lung cancer; malignant mesothelioma. The essential characteristics of these disorders are discussed in the documentation submitted to the WTO and lie beyond the scope of this report; if further details are required, standard texts should be consulted [26-30]. There is no persuasive or compelling evidence that asbestos of any type causes cancers other than lung cancer and mesothelioma, with the arguable exception of cancer of the larynx. At this stage, it is sufficient to point out that: " ... there is an exposure-response relationship for all chrysotile-related diseases. Reduction of exposure through introduction of control measures should significantly reduce risks. Construction and demolition operations may present special control problems". [EHC 203, p 141].

(b) Malignant mesothelioma – Introduction and general observations on asbestos and mesothelioma

5.29 Malignant mesothelioma is a cancer of the mesothelial cells that line the serosal membranes of the major body cavities, namely the pleura, the peritoneum, the pericardium and the tunica vaginalis testis; the constituent neoplastic cells characteristically express the phenotype of a recognized pattern of differentiation of the mesothelium, whether epithelioid, sarcomatoid or both (biphasic), as revealed by conventional light microscopy, mucin immunohistochemistry, immunohistochemistry or electron microscopy, or a combination of these techniques [31-33]. Like other forms of cancer, mesothelioma has the capacity for local invasion of tissues such as the chest wall or lung, with confluent serosal spread in most cases but not all, and in some instances distant metastasis [31], with an almost invariably fatal outcome. Mesothelioma is resistant to conventional cancer therapies (e.g. radiotherapy or chemotherapy), but some long-term survivals have been recorded following radical surgery (pleuropneumonectomy) in patients in good physical condition and with early-stage disease [34-43]; radical surgery of this type is not a treatment option for the majority of mesothelioma patients.

5.30 Most mesotheliomas encountered in the 1990s are a consequence of prior occupational exposure to asbestos [24], including bystander exposure. The relationship between asbestos - especially one or more of the amphibole varieties - and mesothelioma is accepted by virtually all authorities as causal. In this respect, asbestos fulfils all *The Bradford Hill Criteria* for the establishment of causality (e.g. please see Stolley and Lasky [44]).

5.31 The following points about asbestos and mesothelioma are worth emphasis:

(i) Inhalation of asbestos fibres represents the overwhelming cause of mesothelioma in industrialized societies, so much so that the incidence of mesothelioma is usually considered to be an index of those societies' past usage of asbestos.

According to Peto *et al*. [24]:

> "The great majority of mesotheliomas are caused by asbestos, and the much higher incidence in men indicates that most are due to occupational rather than environmental exposure. The incidence continues to rise approximately as the third power of time since first exposure to asbestos for many decades after exposure has ceased (Peto *et al*., 1982), and most patients are men first exposed 30 or more years ago. A country's mesothelioma rate is therefore a quantitative indicator of its population's past exposure — mainly occupational — to asbestos." [p 666].

5.32 Boffetta [15] claims that:

> "Asbestos is the only established risk factor of mesothelioma. Because of the rarity of the disease and the specificity of the causal association, all cases occurring among asbestos-exposed workers are attributed to this exposure." [p 476; please see following discussion].

4.33 The asbestos exposure may take the following forms: (i) direct or indirect occupational exposure (including bystander exposure); (ii) domestic exposure: e.g. household contacts of asbestos workers, such as wives who washed the asbestos-contaminated work clothes of their husbands [45-47]; (iii) environmental exposure: this category includes those who lived downwind of asbestos industries or in townships contaminated by asbestos [45-47]. For example, = 27 mesotheliomas have been recorded among those who lived at Wittenoom as children (the roads, airstrip and school yards were surfaced with crocidolite tailings from the mine, and children often played in the mine tailings).

> "The tumor [mesothelioma] is more often seen in workers who have only moderate or small amount of asbestos in their lungs, and who show little, if any, clinical or radiologic evidence of pulmonary fibrosis. This amount of asbestos may be inhaled not only by professional asbestos workers, but also by those who handle products containing only a small proportion of asbestos, those who do not handle asbestos at all but merely work alongside asbestos workers such as craftsmen employed in the building industry — carpenters, electricians, etc. — those who have relatives who carry asbestos home in their workclothes and those who live close to asbestos plants." [47] [p 295].

5.34 No history of asbestos exposure is obtainable in about 15-25 per cent of mesothelioma cases [31, 48]. Nonetheless, absence of a history of exposure does not equate to absence of exposure, and evidence indicates that many of these mesotheliomas are in reality attributable to asbestos inhalation — e.g. remote, brief or forgotten exposure, or alternatively, the individual may be unaware that he (the male:female ratio is about 8:1) was in fact exposed to asbestos: (i) from my own series of mesotheliomas, 79 per cent of the request forms that accompanied the biopsies on which the diagnosis was made gave a positive history of past asbestos exposure; clinical review of the remaining 21 per cent yielded a history of asbestos exposure in a substantial proportion, including some for whom the original history stated that there was no exposure, so that my estimate of the proportion for whom a positive history of exposure was eventually obtained is = 85-90 per cent. This estimate is in reasonable agreement with figures in the 1999 Report for the Australian Mesothelioma Register [AMR 99], where 85 per cent of mesotheliomas had a history of asbestos exposure; (ii) Leigh *et al.* [49] found measurable asbestos fibre levels (> 200,000 fibres per gram dry lung tissue) in 81 per cent of the 28 per cent of Australian mesothelioma cases that had no history of occupational or environmental exposure to asbestos.

(ii) Putative or possible factors other than asbestos implicated in the induction of mesothelioma

5.35 In the reply to Question 3 from the European Communities, Canada makes the following statements:

> "... Canada wishes to inform the European Communities of the considerable body of evidence contradicting their statement that asbestos in all forms (amphiboles and chrysotile) is the only known factor that can cause mesothelioma or pleural cancer. ... A number of studies suggest other potential risk factors that may have been under-estimated in epidemiological studies in industrialized countries. ... A number of artificial fibres cause mesothelioma when they are injected into the pleura and peritoneum of laboratory animals. Note also that the International Agency for Research on Cancer (IARC) has classified refractory ceramic fibres as probable carcinogens, partly because of instances of mesothelioma induced by inhalation and injection in animal experiments. The SV40 virus readily induces mesothelioma when injected into animals; studies suggest that the virus contaminated anti-polio (poliomyelitis) vaccines from 1955 to about 1963 and may induce mesothelioma with or without the help of asbestos fibres. Some studies of humans report the presence of the simian SV40 virus in the biological tissue of mesothelioma victims. Ionizing radiation used in cancer therapy and perhaps occupational exposure to radiation have induced mesothelioma. ... [E]rionite has been shown to be even more toxic than crocidolite in causing mesothelioma; it has killed large numbers of villagers in

Turkey. Erionite is a mineral fibre but does not belong to the asbestos family."

5.36 Possible factors other than asbestos implicated as contributory or causative for rare mesotheliomas are tabulated below:

TABLE 1: PUTATIVE OR POSSIBLE RISK FACTORS AND MEDIATORS OF RISK OF MESOTHELIOMA OTHER THAN ASBESTOS

Factor	Comments
Erionite	Very high incidence of mesothelioma due to environmental exposure in Turkey (restricted geographic localization only).
Chronic inflammation	Pleural scars (tuberculosis, pleurisy, therapeutic pneumothorax, familial Mediterranean fever); see following discussion.
Radiation	Single cases after Thorotrast injection or radiotherapy; causality unproven. One case in atomic bomb survivor.
Beryllium	Two doubtful cases described.
Vegetable fibres	No proof in humans.
Hereditary factors	Familial cases (explicable by common asbestos exposure ± unidentified genetic susceptibility factors, including association with other cancers in first-degree relatives).
Immunological factors	Rapidly progressive cases in patients with HIV infection; very rare — single case(s) only.
Dietary factors	Provitamin A, ß-carotene may decrease the risk (unproven).
Viruses	Mesotheliomas in animals. Simian virus 40 (SV40) DNA sequences reported in mesotheliomas; see following discussion

Modified from Hillerdal [20].

5.37 There are anecdotal reports of mesothelioma following radiation, including radiotherapy for childhood cancer such as Wilms' tumour [50-56]. In addition, excess rates of mesothelioma have been reported among both Danish and German patients exposed to radioactive thorium dioxide (Thorotrast) for radiological procedures [57, 58], although a similar but smaller Japanese study found no such excess [59]. Neugut *et al*. [60] investigated women with breast cancer and patients with Hodgkin's disease, many of whom had been treated by radiotherapy (RT):

> "The authors performed a retrospective cohort study utilizing 251,750 women registered with breast carcinoma in the Surveillance, Epidemiology, and End Results Program of the U.S. National Cancer Institute from 1973-1993, 24.8% of whom received RT as part of their initial management, and 13,743 people with Hodgkin's disease, 50.6% of whom received RT as part of their initial management. RESULTS: Six cases of malignant pleural mesothelioma were found: two in breast carcinoma patients treated with RT and four found in women not treated with RT. No cases occurred in the patients with Hodgkin's disease. The overall estimated relative risk for malignant pleural mesothelioma after RT was 1.56 (95% confidence interval, 0.18-5.63). CONCLUSIONS: To the authors' knowledge, this is the first controlled study to investigate thoracic radiation exposure and malignant pleural mesothelioma, and no association was found." [abstract].

5.38 I am also aware of at least one mesothelioma in a patient with HIV infection (AIDS) [61]. Other mesotheliomas have occurred many years after chronic inflammatory lesions of the pleura — e.g. chronic empyema or packing of the pleural cavity with leucite spheres as treatment for tuberculosis [62, 63], and there are a few reports of an association between familial Mediterranean fever (FMF) and mesothelioma (about eight cases only; possibly related to recurrent FMF serositis [64-67]). However, cases of this type are exceptional and most cases of "post-inflammatory" mesothelioma with a short interval between inflammation and tumour (e.g. = 2-3 years by analogy with the criteria for the diagnosis of benign asbestos pleuritis [33, 68, 69]), are probably

mesotheliomas that presented with a burst of inflammatory activity, followed by a period of quiescence [70].

5.39 In addition, background asbestos exposure represents a confounding factor for some cases associated with radiation and immunodeficiency: (i) in one report on mortality among 260 plutonium workers, all six mesotheliomas occurred in individuals who had also sustained asbestos exposure [71]: "... no apparently elevated causes of death except for six cases of mesothelioma and six cases of astrocytoma glioblastoma multiforme. The mesothelioma cases had a documented occupational exposure to asbestos ..." [extract from abstract]; (ii) in one of my own cases, the patient had been treated for Hodgkin's disease by mantle radiotherapy 10 years before the diagnosis of his primary pericardial mesothelioma, but he also had a background of occupational exposure to asbestos; (iii) in another case — a pleural mesothelioma in a renal transplant recipient — the patient had also sustained earlier occupational exposure to asbestos.

(iii) Erionite and mesothelioma in Turkey

5.40 Erionite (a fibrous zeolite) represents a naturally occurring fibrous mineral implicated in the induction of mesothelioma in certain villages (notably Karain and Tuskoy) in the Cappadocian region of Turkey [72, 7 3], and in Turkish emigrants [74]. So far as I am aware this represents a restricted geographic pocket of mesothelioma cases induced by erionite used as stucco or whitewash in buildings, so that the inhabitants were exposed to high concentrations of erionite fibres from birth. Erionite has no relevance to the broader mesothelioma problem in Western Europe, North America, and Australia. Nonetheless, in its physical properties erionite has similarities to the amphibole varieties of asbestos and it has been suggested that its greater mesotheliomagenicity is related to a greater surface area (200 m^2 per gram) than crocidolite (8-10 m^2 per gram), due to the presence of pores in the crystal lattice (see Roggli and Brody [75]); such differences in surface topography might correlate with differences in free radical generation at the surface of fibres.

(iv) Simian virus 40 (SV40) and mesothelioma

5.41 Recently, a voluminous literature has grown rapidly on the detection of SV40 DNA in up to 60 per cent of human mesotheliomas [76-87] and some other tumours, such as papillary carcinoma of the thyroid [88], osteosarcomas and brain tumours [83, 89-91]. These observations followed an initial finding that SV40 could induce mesothelioma in hamsters when injected into the pleural cavity [92], and the later demonstration that SV40 could inactivate the tumour suppressor genes p53 and the retinoblastoma gene (Rb) via the large T antigen (TAG) [80, 82, 93, 94]. For humans, early poliomyelitis vaccines contaminated with SV40 were a potential source for the SV40 DNA [82-84]. The following points on this interesting association are also worth emphasis:

- It has been suggested that the presence of SV40 might explain: (i) why mesothelioma only develops in a relatively small proportion of asbestos-exposed individuals (usually < 10 per cent); and (ii) why no history of asbestos exposure is obtainable on a sizeable minority of mesotheliomas [95]. However, almost all the mesotheliomas in which SV40 DNA has been found were asbestos-associated; to the best of my knowledge, there is no reported case-control analysis of SV40-associated mesotheliomas where asbestos fibre counts were not elevated above reference values, with the exception of a recent study by Mayall *et al.* [96] (please see following discussion). Therefore, the existing data do not adequately address either (i) or (ii): there are many other possible explanations for these observations.

- In other studies, SV40 or TAG could not be detected within mesotheliomatous tissue [97-99]. Galateau-Sallé *et al.* [100] found that SV40 was present not only in mesotheliomas but also in benign inflammatory disorders of the pleura and non-

neoplastic lung tissue. In an as yet unpublished investigation carried out in collaboration with Prof. Alec Morley in the Department of Haematology-Oncology at the Flinders University, we have also identified SV40 in mesotheliomas, and in non-neoplastic pleural lesions, normal tissues and colon cancers, casting doubt on the specificity of the association.

- Two epidemiological studies have shown no increase in the incidence of bone or brain tumours — or mesotheliomas — 30 years after the use of polio vaccines contaminated with SV40 [101, 102], although in a later study using SEER data[14], Fisher *et al.* [103] reported an increased frequency of these tumours in subjects who had received SV40-contaminated poliomyelitis vaccines.

- The evidence so far only points to SV40 as a possible co-factor for asbestos in the genesis of mesothelioma [96]. For example, Mayall *et al*. [96] detected SV40 sequences in five of seven asbestos-associated mesotheliomas, but none of four mesotheliomas that were not asbestos-related (investigated by fibre burden analysis of lung tissue, using electron microscopy). However, the evidence in favour of SV40 as a co-factor for mesothelioma induction is still inconclusive and non-persuasive, and in humans the SV40 may represent an innocent bystander or passenger: the criteria for causality [44] have not been fulfilled.

  "It remains to be shown whether the presence of SV40 contributes significantly to malignant transformation or whether certain human neoplasms provide a microenvironment that favors viral replication in humans with latent SV40 infection." [91] [last sentence of abstract].

5.42 The point of these comments is that the evidence for a role of SV40 in the development of mesothelioma is inconclusive, and most of SV40-associated cases still represent asbestos-associated mesotheliomas. Although the literature contains anecdotal reports of mesothelioma following radiation, some of these cases (e.g. among plutonium workers) are complicated by coexistent asbestos exposure and it is worth emphasizing that these cases are rare: together they add up to only a small fraction of 1 per cent of the total burden of mesotheliomas in industrialized societies, for which asbestos remains the overwhelming cause. As emphasized already, there is general agreement that the incidence of mesothelioma in various nations is a reflection of the past usage of asbestos by those societies.

5.43 Hillerdal [20] comments along similar lines:

"... SV40 might be a cofactor to asbestos in some patients with mesothelioma, but the [findings] have not been confirmed and are still disputed. ... In summary, then, as far as is known today, factors other than mineral fibres can only explain a very small proportion of mesotheliomas, and can for practical purposes be disregarded [i.e. when approaching the causation of mesothelioma among large cohorts or populations]. Thus, a malignant mesothelioma can be regarded either as caused by asbestos or belonging to a normal background level — that is a spontaneously occurring tumour." [p 506].

(v) Male: female ratio for mesothelioma

5.44 Asbestos-induced mesothelioma affects males more often than females in a ratio of about 8:1, as a reflection of occupational exposure.

(vi) Anatomical distribution of mesothelioma

5.45 With the exception of one series in which 44 per cent of mesotheliomas were peritoneal [104],

[14]The National Cancer Institute's Surveillance Epidemiology and End Results program.

there is general agreement that primary asbestos-induced mesothelioma affects the pleura more often than the peritoneum, in a ratio of at least 3:1 or even up to = 11:1 [31, 33] (see also AMR 99). In Australia, = 91 per cent of mesotheliomas arise in the pleural cavities, whereas about 7 per cent represent primary peritoneal mesotheliomas and = 1 per cent affect the pericardium or tunica vaginalis testis [33]. This predominance of pleural mesotheliomas in comparison to the peritoneum appears to correlate with gender differences in the frequency of occupational exposure to asbestos (the same high ratio of pleural to peritoneal tumours is also encountered in the United States). In females, a smaller proportion of mesotheliomas arises in the pleura, and in one study of Swedish insulation workers, all seven mesotheliomas arose in the peritoneum [105] (please see following discussion).

5.46 One report [106] that included cases notified to the Australian Mesothelioma Register from 1986 through 1988 gave figures for the anatomical sites affected in men and women: 676 of 723 men had a pleural mesothelioma (93 per cent), whereas 38 were peritoneal tumours (5 per cent) and nine occurred in other sites (1 per cent). In contrast, 84 mesotheliomas in 101 women were pleural in location, whereas 17 per cent had a peritoneal mesothelioma.

5.47 Presumably, this difference in anatomical distribution between sexes is a reflection of different rates of occupational exposure to asbestos. On theoretical grounds, one would expect mesotheliomas entirely unrelated to asbestos to occur with about equal frequency in the pleura and peritoneum, or more often in the peritoneum because of the greater surface area of the peritoneal cavity.

5.48 A partial list of the factors that might explain the higher proportion of peritoneal mesotheliomas in some series and in women includes the following [33]:

- The high proportion of pleural mesotheliomas in men is presumably a reflection of asbestos exposure, with deposition of asbestos fibres in lung tissue, followed by translocation of the fibres to the pleura; on this basis, asbestos inhalation appears to skew the proportional distribution of mesothelioma towards the pleura in comparison to other sites. In contrast, fibres presumably follow a more circuitous route from the lung to the pleura, across the diaphragm and into the peritoneal cavity, to induce peritoneal mesothelioma; higher inhaled doses of asbestos might be necessary for the requisite number of fibres (whatever that is) to reach the peritoneum via the pleura, in order to induce peritoneal mesothelioma.

- The high proportion of peritoneal tumours in some series may be a consequence of patterns of referral for cases that constitute problems in diagnosis, because the diagnosis of peritoneal mesothelioma is, in general, more difficult than for pleural mesotheliomas. This may explain the higher proportion of peritoneal mesotheliomas among cases referred to the US-Canadian Mesothelioma Panel [107], because many of these represented problems in diagnosis, whereas the Australian Mesothelioma Surveillance Program (AMSP) captured all mesotheliomas throughout Australia [48].

- Genuine biological differences in the inhaled dose, deposition or transport of different asbestos fibre types in some groups of workers, notably insulation workers [108] and former Wittenoom workers [109] — as a consequence of heavy occupational exposure — and in women [106, 110].

5.49 In answer to questions posed by the European Communities (Question 3, see Annex II), the following comment is made:

> "... malignant diffuse mesothelioma is a cancer of the mesothelial cells of the pleura, the pericardium and the peritoneum. Furthermore, peritoneal mesothelioma is an even more typical result of exposure to amphiboles than pleural mesothelioma."

5.50 From the preceding discussion on the proportions of mesotheliomas arising in the pleural cavities versus the peritoneum, it is evident that this proposition is not correct: use of the term typical in this context is inappropriate. In reality, pleural mesothelioma is a more typical or usual outcome of asbestos exposure, whereas asbestos-induced peritoneal mesotheliomas are usually associated with more prolonged and heavier exposures than pleural mesotheliomas, so that the proportion of patients with asbestosis is higher than for pleural mesothelioma [111]. It has also been claimed that peritoneal mesotheliomas are almost always a consequence of amphibole exposure (as opposed to chrysotile only) [112]. Nonetheless, although some of the peritoneal mesotheliomas in my own series of cases followed high-dose exposures to asbestos that included one or more of the amphiboles, a few followed lower cumulative exposures, and Neumann *et al.* [111] have reported peritoneal mesotheliomas as a consequence of exposure in the building trades and metal industries, in addition to asbestos industries; Rogers *et al.* [3] recorded peritoneal mesotheliomas in whom only chrysotile fibres were detected on lung fibre analysis (see Table 9, paragraph 5.137).

(vii) Latency intervals (lag-times)

5.51 In all reported studies, mesothelioma is a disease of long latency between exposure to asbestos and the subsequent diagnosis of the mesothelioma. In the AMSP [48], the mean latency interval (lag-time) was 37 years, with a reported range of 4-75 years; the lag-time was reported to be < 10 years in only four of 499 asbestos-associated mesotheliomas (0.8 per cent). Many authorities set a minimum lag-time of 10 years (e.g. The Helsinki Criteria [113]), and for most patients the lag-time is in the range of 20-40 years, When the lag-time is < 10 years, it is likely that the proximate exposure was coincidental, and that there were one or more earlier exposures.

(c) Spontaneous or background mesothelioma: does it exist?

4.52 The rare occurrence of mesothelioma in childhood and even as a congenital malignancy supports the existence of a background of spontaneous mesotheliomas unrelated to asbestos (in addition, mesothelioma has been reported in fish (trout) [114], where inhalation of airborne asbestos fibres cannot be invoked). However, in epidemiological studies on adult populations, it is virtually impossible to separate spontaneous mesotheliomas from those that are arguably attributable to environmental exposure to asbestos [70, 115]. The incidence of mesothelioma in women is sometimes used as an index of the background or spontaneous rate: the crude incidence rate for women in Western Australia is about 2.6 per million person-years at age = 15 years [115]. The incidence rate in other populations is listed in Table 2 (following page).

5.53 In the answer to the WTO Panel's questions to Canada (Question 9, see Annex II), the following statement is made:

> "Recent analyses of Canadian data on mesothelioma in Canada, British Columbia and Quebec all agree that the incidence rate of mesothelioma has been stable among women of all age groups since 1984. The rates are 70% higher in Quebec than in the rest of Canada, presumably as a result of more frequent and more intense exposure in the workplace."

5.54 The statistics for Australia differ on this point (Table 2): mathematical modelling of Western Australian data suggests that the incidence rate in women has risen about two-fold from the 1970s until the 1980s, which might be explicable by increased general environmental exposure to asbestos, plus some occupational exposures among women [70, 115] (please see also AMR 99 — i.e. the graph for the Age Specific-Incidence Rates of Malignant Mesothelioma in Australia Women, 1986-1995, especially for ages 50-64 and 65-79). This increased incidence among women

presumably reflects direct or indirect occupational exposure, domestic exposure or environmental exposure [115]; in this respect, it is worth emphasizing that domestic (household contact) exposure to asbestos — e.g. among wives laundering the dust-laden workclothes of an asbestos-exposed husband — is not necessarily low-level exposure, and analysis of the asbestos fibre content of the lungs in a small number of such patients indicates that this type of exposure can approach occupational levels [116].

TABLE 2: INCIDENCE OF MORTALITY OF MESOTHELIOMA IN VARIOUS COUNTRIES AND AREAS OVER TIME, 1960S TO 1994 (PER MILLION INHABITANTS PER YEAR)

Country or area	Year	Males	Females
United States	1968-81	2.1	0.8
North America	1972	2.8	0.7
Texas	1976-80	5.8	2.1
Selected cities, United States	1970s	4.4-11.1	1.2-3.8
United States	1986	7-13	1-2
Nantes-Saint-Nazaire, France	1956-74	5.2	0.2
Nantes-Saint-Nazaire, France	1975-84	17.2	0.8
Nantes-Saint-Nazaire, France	1985-92	19.4	4.0
Great Britain	1968-71	8.4	2.3
Great Britain	1972-76	12.6	2.8
United Kingdom	1983	17.5	3.2
Great Britain	1968-71	20.7	4.3
Great Britain	1982-86	30.5	4.9
Great Britain	1987-91	44.0	6.4
Australia	1982-88	28.3	3.3
Australia	1994	49.9	4.8
Denmark	1978-80	14.7	7.0
Barcelona, Spain	1983-90	8.3	4.7
Finland	1990-94	10	2.9

Modified from Hillerdal [20].

5.55 The often-cited background or spontaneous rate of mesothelioma of 1-2 per million person-years [10, 117], has in part also been derived from backward extrapolation of the incidence rates in men, to the point where the estimated incidence rates for men and women diverged from each other (i.e. linear extrapolation to the point where the sex ratio = 1:1) [117] . Hillerdal [20] suggests that this incidence probably represents a high estimate and comments in the following terms:

> "... there seems to be a small spontaneous basal or background incidence of the tumour [mesothelioma] ... However, it is of course possible that some of these background cases might in fact be due to occupational, domestic, or even environmental exposure, unknown to (or forgotten by) the patients themselves. ... There are authors who claim that the presumed background levels must be very low, and retrospective searches for the tumour in the medical literature reveal no convincing cases of mesothelioma before 1946, although such negative evidence is of questionable value.[15] McDonald and

[15]Mark and Yokoi [118] have called into question the existence of mesothelioma in the absence of asbestos exposure, pointing out that the early descriptions of pleural tumours may have dealt with localized fibrous tumours of the pleura (four of the five tumours reported by Klemperer and Rabin [119]) or secondary carcinoma. Thus, mesothelioma might represent a new disease consequent upon the industrial use of asbestos (analogous to AIDS) and it may disappear upon withdrawal of the causative asbestos from the environment (analogous to smallpox). In support of this proposition, these authors cited the records of the Massachusetts General Hospital, where no examples of mesothelioma were diagnosed before 1946, in contrast to 100 autopsy cases thereafter, in a total of 47,000 autopsies. They also referred to the Henke-Lubarsch *Handbuch der*

> McDonald, in a recent review, estimated the background level to be 1-2/million per year; they came to this figure by extrapolating backwards from epidemiological studies from various countries. ... It is nevertheless possible that there is a background level of mesothelioma, — that is, that the tumour can occur even in the complete absence of asbestos (or erionite) fibres. However, the data reviewed here indicate that if so, this background level must be very low — probably much < 1 case/million people/year. This figure comes from studies of industrialized countries, where background exposure to asbestos is unavoidable. What the true figure is can only be guessed ... ". [p 507].

5.56 De Klerk [115] and Comin *et al.* [70] have commented that in the absence of specific exposure to asbestos, the final estimated rate for both men and women in Australia is 2.6 per million person-years — higher than the equivalent figure of 1.6 for Los Angeles [115]. This difference may lend some support to the proposition that general environmental exposure to asbestos may have produced an increase in the mesothelioma rate in Western Australia [115]. However, it is difficult or impossible in general to draw firm conclusions from differences between different studies, because of variation in the accuracy of diagnosis and differences in the ways that data are collected.

5.57 In response to questions from the WTO Panel (Question 9, see Annex II) the Canadian document also observes that:

> "The incidence of [mesothelioma] among men levelled off after 1984 in British Columbia ... and seems to have levelled off in Quebec after 1990 ... Finally, analysis of Canadian rates between 1973 and 1992 ... estimates that the risk is four times greater for men born before 1940 than for men born between 1951 and 1955. Those analyses therefore suggest that the incidence of mesothelioma has levelled off in Canada, is declining in British Colombia, and has levelled off in Quebec ..." .

5.58 In response to these observations, I emphasize the following: (i) the incidence of mesothelioma among Australian males shows little evidence of levelling off, and has continued to rise until 1994-1995 and thereafter (please see Table 2 and the 1998 and 1999 Reports for the Australian Mesothelioma Register); (ii) from the recent report by Peto *et al.* [24], it is also evident that the incidence of mesothelioma in Western Europe continues to rise, with particular emphasis on males born between 1945 and 1950 who used asbestos-containing products in the 1960s and the 1970s (and the early 1980s).

(d) Magnitude of the mesothelioma problem

5.59 Malignant mesothelioma continues to represent a major health problem within industrialized societies, and together with lung cancer it represents the most important occupational cancer among so-called blue-collar workers [121-124].

5.60 It has been estimated that across Western Europe, North America and Australia (combined population ~ 800,000,000), around 10,000 mesotheliomas and 20,000 asbestos-induced lung cancers occur annually, related mainly to occupational exposure (about one mesothelioma for every 200 tons of asbestos produced, taking into account the prolonged lag-times) [125]. Steenland *et al.* [126] estimate that approximately 9000-10,000 men and 900-1900 women develop lung cancer each year in

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speziellen pathologischen Anatomie und Histologie, wherein the four pages devoted to tumours of the pleura did not specifically acknowledge the existence of mesothelioma; the Henke-Lubarsch authors concluded that many cases described in the literature as primary pleural neoplasms were cases of lung cancer with spread to the pleura. I find the evidence for Mark and Yokoi's proposition to be underwhelming and unconvincing. The case reported in the 1920 paper by Du Bray and Rosson [120] is, I believe, a clear example of a mesothelioma, as is the fifth case of Klemperer and Rabin [119]. Failure to diagnose a tumour is hardly synonymous with its non-existence, and the pathological features of many tumours have been delineated in quite recent times. Pathological diagnoses follow prevailing evidence and fashions, and because the groundwork for modern concepts of mesothelioma was laid down in 1931 by Klemperer and Rabin, it is hardly surprising that the diagnosis became more widespread only after this time.

the United States because of past exposure to occupational carcinogens, and more than half of these lung cancers are related to asbestos (this overall estimate is considered probably to be conservative). Predictions of asbestos-related diseases in Australia (population ~ 18,000,000) indicate that about 13,000 cases of mesothelioma (range 8000-20,000), about 40,000 cases of lung cancer (range 30,000-76,000) and 1000 cases of asbestosis are likely to occur between the years 1987 and 2020 [70, 127].

5.61 More recently, Peto *et al*. [24] have predicted that about 190,000 mesothelioma deaths are likely to occur throughout Western Europe (Britain, France, Germany, Italy, the Netherlands and Switzerland) over the next 35 years. If one adds in lung cancer at a ratio of one lung cancer for every mesothelioma death, this figure would rise to 380,000 deaths, and if the ratio of lung cancers to mesotheliomas is 2:1 the figure rises to 570,000 deaths.

5.62 Overall, asbestos may have caused approximately 5,000,000 deaths across industrialized societies so far. When future deaths in so-called developing nations are added, the final toll is almost certain to be substantially higher, especially because occupational exposures in those countries are likely to be heavier (e.g. China). Estimates of this magnitude are likely to engender alarm among those who set social policy. Even so, it is important that this problem, like others (e.g. atomic energy), is approached with common sense, rationality and prudence, taking into account population-based risk estimates: it would be irrational to swap one risk for another higher risk if the two risks were equally serious.

(e) Some general observations on approaches to risk assessment on society and on epidemiological studies of asbestos-related cancers

5.63 The documentation supplied to the WTO includes estimates of risks from low-level exposure to chrysotile in proportion to various other risk factors in society: in fact, the relative risk of mesothelioma from low-level exposure to asbestos in place is the focus of considerable controversy. Clearly, detailed analysis of this issue is beyond the scope of this report, but the excess risk of mesothelioma from very low-level exposure to asbestos — e.g. simple occupancy of public buildings or schoolrooms where average asbestos fibre concentrations are about < 0.001-0.02 fibres per litre — appears to be very slight: about = 5.5 mesotheliomas per million lifetimes of 80 years, or < 1 case per 10,000,000 per year.

5.64 The estimated mesothelioma risk for a 10-year exposure to low levels of airborne asbestos in schools (age start 7-8 years; fibre concentration 0.00065-0.001 fibre per ml[16] is in the range 6.6-20 per million lifetimes (0.0825-0.25 per million person-years). Estimates of this type are predicated on linear dose-response models with no threshold, and these have been the subject of argument and criticism. The occupational groups from which they were derived were exposed to mixtures of asbestos types, but one might expect the risk to be even less or "undetectably low" in nations where only chrysotile was used. At this point, it is sufficient to emphasize that - even if one accepts for a moment the no-threshold linear dose-response relationship - calculations indicate that a single asbestos fibre (the so-called "one fibre" hypothesis) would only have a 50-50 chance of producing a single excess mesothelioma among all the humans who have ever inhabited Planet Earth.

5.65 These observations on mesothelioma risk estimates for very low-level exposure to asbestos in buildings do not contradict the earlier suggestion in this report that Western Australian and Australian increases in mesothelioma incidence among females were a possible consequence of general environmental exposure to asbestos: the two-fold rise in incidence in Western Australia could be due to environmental fibre levels higher than those recorded in public buildings elsewhere.

[16]Given in various publications as fibres/ml, fb/ml, f/ml, f/mL and fibres/cm^3.

5.66 If it exists, the risk of mesothelioma from very low-level environmental exposure to asbestos needs to be considered in proportion to other risks of death in society. The lowest death-risk at any age occurs in girls aged 4-14 years and is ~ 100 per million per year, but the risk in late teenage increases by 300-400 per million per year — attributable largely to increased travel by motor vehicle. A 40-year-old man at risk of mesothelioma from low-level exposure to asbestos during childhood (excess mesothelioma risk < 1 per million person-years) has an annual risk of death from all causes of about 2000 per million. In 1990, de Klerk [128] had this to say on the subject:

> "In the US the acceptable (or possible litigation-proof) lifetime risk seems to be one per million. The FDA have this as a set policy; the EPA approximates to it, and other agencies seem to employ similar figures. In the UK the Royal Society has set a higher range with an annual risk of one per million considered negligible, with any form of control unjustified; one per 100,000 considered low ("very few would consider action necessary" — e.g. 16,000 km air or rail travel); one per 10,000 moderate (few would commit their own resources to reduce risk" — e.g. 16,000 km car driving, working as a coalminer); one per 1,000 high (e.g. age 30-39, 16,000 km motor cycling); and one per hundred unacceptable (e.g. age 55-59, smoking 20 cigarettes per day, heavy exposure to crocidolite)."

5.67 Within this context, one might ask what constitutes a negligible as opposed to an acceptable (or unacceptable) risk? In the context of the foregoing discussion, one might argue that a negligible risk is a statistical and scientific concept: a risk so slight that it does not require preventive or remedial steps in comparison to other risks in society (although some might argue about the dividing line between negligibility and unacceptability in this context). With acceptability or unacceptability other factors come into play: they include, for example, social, political and industrial considerations — and the likelihood of litigation over any situation wherein the theoretical or estimated risk is elevated to a background level, no matter how slightly. Accordingly, a risk, though slight or even negligible, might still be considered unacceptable in legal or sociopolitical terms.

5.68 Others may dissent from the acceptability of the Royal Society approach to low, moderate and high risks discussed above. Conclusions about the acceptability or unacceptability of risk will also vary according to the seriousness of the risk (e.g. the approach to a lethal risk such as mesothelioma would be quite different from a factor that caused a large proportion of the population to sneeze once or twice); these assessments will also vary according to the avoidability of the risk, the individuals making the assessment, and the question of informed consent by those at risk.

5.69 Furthermore, society abounds with inconsistencies and contradictions over the relativity of various risks. For example, some societies that regulate or propose a ban on chrysotile asbestos make extensive use of radioactive materials — e.g. in nuclear power stations and the production of radioactive isotopes for medical purposes. Even so, the use of fissionable materials for these purposes may be justified and justifiable within those societies, because: (i) the risk of morbidity or death from well-publicized mishaps at nuclear reactors is still substantially less than the risk of death from alternative energy sources (e.g. higher mortality rates among coalminers); and (ii) because the materials in question can be regulated and controlled so that they are accessible to only a small fraction of society (i.e. workers who can be trained in the controlled use of radioactive substances); and (iii) nuclear power does not contribute significantly to air pollution or greenhouse gas emissions in comparison to the burning of fossil fuels.

5.70 In addition, Nicholson [129] places the problem into the perspective of voluntary versus involuntary risks:

> "Rather than compare asbestos risks with voluntary risks (smoking, school football) or risks that remain high despite expenditures of substantial public and private money (aircraft and highway accidents), it is worthwhile to compare them with other involuntary environmental risks that are controlled by regulatory agencies (pesticide exposures, drinking water contamination). In a review of regulatory actions taken by the FDA ... and the EPA it was found that for estimated population risks exceeding one

> death/year, the individual lifetime risks were usually regulated if they exceed 1/1,000,000 for a lifetime exposure. Only eight of 31 carcinogenic exposure circumstances that exceeded this level were not regulated. They involved saccharin, aflatoxin, formaldehyde and polycyclic organic matter ... " [p 81].

5.71 In fact, it is my view that over-reaction to the low risks produced by asbestos in place may lead to a greater risk — i.e. the carcinogenic risks imposed by asbestos removal programmes. Two mesotheliomas encountered in my own practice during 1999 occurred not in asbestos removal workers, but in others who sustained bystander exposure as a result of this activity: (i) pleural mesothelioma in a lecturer who had walked to and from her classroom at an Australian university each day over a period of weeks, through a building where an asbestos insulation removal programme was being carried out; (ii) pleural mesothelioma in a fireman who attended fires in buildings that contained asbestos-cement products and who participated in clean-up operations thereafter; about once a month for some years, he also attended and examined buildings where fire alarms had been activated by high atmospheric concentrations of asbestos fibres produced by removal programmes. (In addition, a recent survey in Finland found "occasional high fibre concentrations even inside personal protectors during asbestos removal work" [130]).

5.72 Two other important points are worth emphasis. First, because they usually focus on specific cohorts or groups of workers, epidemiological studies may fail to identify a small but real risk, because of low statistical power. In this respect, the documents submitted to the WTO state that it may be impossible to prove a negative (absence of risk), but one can also state that absence of proof does not constitute proof of absence. For example, a number of investigations have failed to identify a statistically significant increase in the relative risk (RR) of cancer among individuals with parietal pleural fibrous plaques. In an extensive review of asbestos and lung cancer, Henderson *et al.* [131] commented along the following lines in relation to pleural plaques and lung cancer:

> "Nurminen and Tossavainen [132] also emphasized the issue of statistical power; they calculated the RR for plaque-associated lung cancer in the general population to be as low as 1.1, given the prevalence of 4.6% among unlikely exposed and 13.0% among probably exposed men with an estimated twofold risk of lung cancer. Detection of this RR at a level of statistical significance would require a population sample of about 300 000." [p 102].

5.73 In a discussion of the Hughes-Weill study [133] on radiological asbestosis and lung cancer in New Orleans asbestos-cement factory workers — one of the three key investigations that proposed an obligate intermediary step of pulmonary fibrosis for the induction of lung cancer by asbestos — Henderson *et al.* [131] also commented in the same review:

> " ... the number of lung cancer cases [in the Hughes-Weill investigation] was small. What number of workers would be required in such a study to detect an increase in risk of, say 1.4, 1.56 or 2.0, as opposed to the risk in workers with chest x-ray opacities? ... person-years of follow-up equivalent to 20-50 expected cases would be required to have any reasonable chance of detecting RRs of 1.4 to 1.6 at a level of significance of 0.05. ... The power level for the actual sample of 420 ... to detect a risk of 1.5 would be about 40%. That is, a true effect would be falsely declared 'non-significant' 60% of the time. ... The low power of the Hughes-Weill study is exemplified by the fact that ... lung cancer risk was not significantly associated with duration of employment or cumulative exposure (there was a fairly restricted range of employment periods) and even the association of lung cancer with fibrosis was only marginally significant." [pp 93-94].

5.74 The point is that a low, non-significant or undetectable risk in a small cohort may nonetheless translate into a substantial body of disease when spread over a large population: e.g. an RR of 1.1 representing an increase in risk of 10 per cent may require a population size of 300,000 to be detectable at a level of statistical significance of 0.05, whereas this 10 per cent increase in a common disease such as lung cancer may amount to a substantial burden of disease when spread across a

population of, say, 1,000,000, 10,000,000, or 100,000,000. (Please see also later discussion on mesothelioma among brake mechanics: answer to Question 2.)

5.75 Another point is that a high frequency of a cancer such as mesothelioma in a small population may be overshadowed in absolute numbers by a lower occurrence rate for the same disorder spread over a large population. For example, among non-smoking former Wittenoom workers, mesothelioma is now the most common cause of death [70] (in most cohorts exposed to amphibole asbestos, < 10 per cent will develop mesothelioma). Nonetheless, mesotheliomas among the Wittenoom cohort contribute only 5-6 per cent of the total burden of mesothelioma across the Australian population [AMR 99]. For example, the 1999 Report for the Register records 189 mesotheliomas among the former Wittenoom population with only a single exposure to asbestos, in comparison to 187 mesotheliomas among carpenters/joiners with only a single exposure to asbestos; the point is that the lower risk of mesothelioma from asbestos exposure among carpenters has produced almost the same number of cases, because carpenters among the Australian workforce constitute a much larger occupational group than the entire Wittenoom cohort of about 6000.

5.76 This observation also applies to the numbers of mesotheliomas among chrysotile miners and millers in Quebec, in proportion to other cases among the general population of Quebec. Bégin *et al.* [134] divided Quebec mesotheliomas into three groups, as shown in the following Table:

TABLE 3: MESOTHELIOMAS IN QUEBEC, 1967-1990

Group	**Type of asbestos exposure**	**Number of cases**	**Average age**	**Average duration of exposure**
1	Chrysotile miners and millers, Thetford and Asbestos, Quebec	49	62 ± 8.1 yrs	30.5 ± 13.7 yrs
2	Manufacturing, industrial insulation, shipbuilding yards of Quebec	50	56.7 ± 8.6 yrs	21.4 ± 14 .5 yrs
3	General construction/ building maintenance industries of Quebec	21	57.7 ± 7.2 yrs	27.7 ± 7.2 yrs

From Bégin *et al*. [134].

5.77 In this study, Bégin *et al.* [134] also commented that "the incidence of pleural mesothelioma in chrysotile miners and millers, although not as high as in the crocidolite workers, is well above the North American male rate". They also observed that "asbestos exposures in Group 3, although difficult to quantify on the basis of the record, appear to be often very low intensity". Bégin *et al.* also commented in the following terms:

> "The present study documents an increasing incidence of malignant mesothelioma in chrysotile miners and millers of the eastern townships of Québec, with 49 cases in the last 23 years and a rate of 2.5 cases per year in the last 10 years in the primary industry, as compared with a rate of 0.3 per year in the years prior to 1969 ... To put these rates into perspective, a comparison of the incidence for the combined population of the Asbestos and Thetford townships of Québec of some 40,000 adult males or the maximal estimated workforce of 10,00-15,000 men [sic; surely this is a typographic error in the original, and it should be 10,000-15,000], 20 years ago and currently at risk, reveals that the incidence of mesothelioma in the chrysotile mining townships of Québec would give an annual incidence rate of 62.5 cases per million per year for the 1980-1990 period, or in chrysotile miners and millers of Québec, would give an incidence rate of 150-250 cases per million per year for the 1980-1990 period. These values are well below the annual incidence rate of the crocidolite mining townships of South Africa, estimated at 542 cases per million per year, and well above the rate for the North American population, estimated at between 2.5 to 13 cases per year per million adult males for the 1970-1980 period, and 14.1 cases per year per million adult males in 1984 and 15 cases per million for 1980 and projected to increase for the 1990s. ...

> Thus, our observations add information of interest to the on-going debate regarding the relative carcinogenicity of different types of asbestos fibers. Our data suggest that some of the cases of malignant mesothelioma in Québec chrysotile miners and millers may not be necessarily attributable to amphibole and could be chrysotile-induced. Lung tissue burden analyses, a better indication of exposure than tumour tissue burden, will be done on these cases to further investigate this point. ....
>
> Finally, our data strengthen the view that a substantial number of malignant mesothelioma cases have a relatively short asbestos exposure, particularly seen in Group 3. In our study, 25% of all cases are in such a category" ... [pp 539-541].

5.78 In one of the documents submitted to the WTO, it is argued that evaluation of, and actions on, risks should be based on probability rather than mere possibility. This proposition is open to dispute. For example, action is often taken to avoid the possibility of harm — by regulation or prohibition — even though the likelihood of injury is remote, because of the seriousness of the potential outcome. In medical ethics, this is the principle of first, do no harm (*primum non nocere*). Two examples follow: (i) the antibiotic chloramphenicol was known to be highly effective in the treatment of various infections, including typhoid fever, but on rare occasions it induced bone marrow aplasia; despite the low likelihood of this side-effect — about 1 in 250,000 — the use of chloramphenicol was restricted to only a few life-threatening infections (e.g. typhoid fever), and it is now almost never used because safer effective alternatives are available; (ii) over recent years, there has been a flood of publicity over global warming and greenhouse gas emissions. A causal or direct relationship between greenhouse gases (such as CO_2 and methane) and climate change is open to argument, and Earth undergoes repeated cycles of natural cooling and warming; in this respect, there is also evidence that melting of the Antarctic ice cap has been going on for some thousands of years, and global warming for over 100 years. Nonetheless, the consequences of inaction over greenhouse gas emissions are potentially so serious that strategies to reduce the release of these gases into the atmosphere are entirely appropriate, despite uncertainty over the link between them and global warming.

(f) General observations on induction of mesothelioma by asbestos, especially the amphibole varieties of asbestos such as crocidolite and amosite

(i) The linkage between the amphibole varieties of asbestos, and commercial chrysotile, and the subsequent development of malignant mesothelioma is well established and is not in dispute.

5.79 This link is generally accepted as causal; in this respect, asbestos fulfils all *The Bradford Hill Criteria* for the establishment of causality [44].

(ii) There is a dose-response relationship between cumulative exposure to asbestos and the subsequent incidence of mesothelioma in asbestos-exposed cohorts or populations; the incidence is also related to time since exposure, so that early exposures are more significant for mesothelioma induction than later exposures, other factors being equal.

5.80 This relationship is expressed by the Peto model and its various modifications:

$$I = K*F*(T^p - [T - D]^p)$$

where I = incidence; K depends on fibre type, mix, size and other site-specific variables; F = intensity of exposure in f/ml; and D = years of exposure. For the purposes of modelling, T can be replaced by (T – 10) to build in a minimum 10-year lag-time, and the cubic power of time (T^3) is often used, so that:

$$I = K*F*([T - 10]^3 - [T - 10 - D]^3)$$

An important aspect of this model is that early exposures are more significant for mesothelioma induction than later equivalent doses.

5.81 Of the variables D, F and K, it is D that is the most accurately measurable, whereas the values for K and F are often unknown, though some estimates of F can be made from the type of work activity. When there are multiple periods of employment for which the type of work is similar for each, one can assume that the value for each of F_1, F_2, F_3 ... F_n remains constant, which also applies to K_1, K_2, K_3 ... K_n, so that:

$$I \propto ([T - 10]^3 - [T - 10 - D]^3)$$

In practice, a simpler equation can be used: $I = ct^k$

where the constant c is dependent on exposure, usually taken as proportional to the intensity of exposure multiplied by its duration (i.e. cumulative exposure), with weightings for different fibre types; the power k remains about 3.5, or 3 for short periods of exposure. As de Klerk and Armstrong [135] state:

> "The model predicts that risk is increased after each increment of exposure by an amount proportional to the level of exposure and the cube of time after that. In terms of the multistage model of cancer, it implies that asbestos acts at the first stage of a 4-stage process. ... The model predicts that incidence is much more dependent on early or low levels of exposure and increases less rapidly as exposure continues to increase, depending mainly on time since first exposed." [p 232].

5.82 When one is faced with multiple exposures to asbestos, the following points emerge, specifically for mesothelioma induction, provided that the characteristics and time for each exposure are appropriate for a biological effect: (i) it is not valid to point to one exposure among the others and incriminate it as the sole cause of a mesothelioma, with exoneration of the other exposures; (ii) it is not valid to point to one exposure among the others and exonerate it from a causative role in the development of a mesothelioma, and to incriminate all the others; (iii) when there are multiple episodes of exposure as a background to a mesothelioma, it is often the case that each exposure in isolation would be sufficient for attribution of the mesothelioma to asbestos, with the provisos mentioned above (characteristics and times of exposures). When each exposure among others is appropriate for mesothelioma induction if the particular exposure occurred alone, it is not logical to state that this exposure — which could have a biological effect in isolation — has no effect when in combination. In such circumstances, it is not the presence or absence of an effect that is in question, but the magnitude of each effect in proportion to the others.

5.83 A dose-response relationship has been observed with both estimates of airborne exposure to asbestos [136], and quantitative and qualitative fibre burden analysis of the asbestos content in human lung tissue of mesothelioma patients [3, 25, 137, 138]: e.g. see Rogers *et al.* [3], and, more recently, Williams *et al.* [138], who noted in 1997 that:

> "It was shown there that while the relative risk of all three diseases [i.e. asbestosis, mesothelioma and lung cancer] increased with increasing exposure, the relative risk of malignant mesothelioma is greater at low levels of exposure when compared with the risk of asbestosis but is lower at very high levels of exposure." [p. 39].

5.84 In their study on the relationship between lung asbestos fibre type and the lung tissue concentration of asbestos versus the relative risk of mesothelioma, Rogers *et al.* [3] made the following comment:

> "Fiber content in the lung depends on both the amount of fiber deposited and the amount cleared. The amount deposited depends on duration and intensity of exposure in the occupational or general

environment. Clearance rate is thought to be dependent on the amount deposited at any point in time, i.e, clearance is exponential. Thus, the same fiber content in the lung at death or time of resection may be achieved from a high initial deposition, followed by absence of deposition and absence of clearance over a long period of time, or by a continuous deposition at a lower level, with or without clearance. Since detailed mechanisms of mesothelioma initiation and progression are not known, 'dose' as estimated by final lung fiber content may not relate to the 'dose' required to produce mesothelioma. It is thus possible that a high lung fiber content in a mesothelioma case may represent continuing accumulation of fibers after a lower level of fibers had produced malignant change. It is more likely, however, that the malignant change did not occur until the fiber content reached a sufficiently high level." [p 1913].

(iii) The dose-response relationship between the amphiboles and mixtures of asbestos types is linear at high exposures [15]

5.85 For example, please see EHC 203 and Table 4.

(iv) This dose-response relationship between asbestos exposure and the risk of mesothelioma has also been detected at low levels of exposure, which overlap with environmental exposures.

TABLE 4: INCIDENCE OF MESOTHELIOMA IN OCCUPATIONALLY EXPOSED GROUPS BY FIBRE TYPE AND TIME SINCE FIRST EMPLOYED

Fibre type	**Industry**	**Years since first employed**	**Rate per million person–years**
Mixed: crocidolite, amosite and chrysotile	Manufacture textiles and insulation	20-24 25-30 30+	1520 1710 3180
Mixed, mainly amosite	Insulation workers	20-24 25-29 30-34 35-39 40-44 45+	290 1550 2760 6300 6330 8110
Mixed: crocidolite and chrysotile	Fibrous cement manufacture	20-24 25-29 30-34	2700 6300 9600
Chrysotile, some crocidolite	Textile manufacture	20-24 25-29 30-34 35-39 40+	108 143 1156 493 1774
Amosite	Insulation manufacture	20-24 25-29 30-34 35+	744 2623 5078 1842
Mixed	Dockyards	20-24 25-29 30-34 35-40 40-44 45-49	120 410 220 370 1240 1510
Crocidolite	Mining and milling	20-24 25-29 30-34 35-39	900 2200 3000 7000

From de Klerk and Armstrong [135].

5.86 A recent case-control study [136] from France on the dose-response relationship between low levels of asbestos exposure and the odds ratio (OR) for mesothelioma showed a clear dose-response relationship between estimated cumulative asbestos exposure and the OR for pleural mesothelioma. In the final paragraph of the article, the authors stated:

> "We found a clear dose-response relation between cumulative exposure to asbestos and pleural mesothelioma in a population-based control study, with retrospective assessment of exposure. A significant excess of mesothelioma was observed for levels of cumulative exposure that were probably far below the limits adopted in many industrial countries during the 1980s." [last sentence of abstract].

Although some concerns have been expressed about this type of investigation [139], it is my opinion that these points were addressed in the original paper [136], and they are common and intrinsic to epidemiological studies of this type — e.g. see Camus *et al.* [140, 141]. This study [136] found an OR for mesothelioma of 4.2 [95 per cent CI 2.0-8.8] at estimated cumulative exposures of 0.5-0.99 fibre-year,[17] with elevation of the OR at about 0.5 fibre-year.

5.87 In a fibre burden study on mesothelioma patients, Rödelsperger [137] observed that:

> "A significantly increased OR [for mesothelioma] is obtained even within the very low concentration range of 0.1-0.2 F/µg [i.e. concentrations in the range of 100,000-200,000 fibres per gram dry lung tissue], which may be expected for about 5% of the population." [p lll] (which also corresponds to an estimated cumulative exposure in the range of about 1-2 fibre-years).

5.88 In a more recent study on mesothelioma cases (N = 66) and controls (N = 66), Rödelsperger *et al.* [25] found an OR for mesothelioma of 4.5 at fibre concentrations of 100,000 to < 200,000 per gram dry lung tissue (for fibres > 5 µm in length; 95 per cent CI 1.1-17.9). These authors also recorded an OR = 2.4 at concentrations of 50,000 to < 100,000 fibres per gram dry lung tissue (95 per cent CI 0.8-7.6). The controls for this study — surgical lung resections mainly for lung cancer — would be expected to bias the OR towards 1.0 (i.e. underestimate the effect) [25], and hence the OR of 2.4 probably represents a genuine doubling of risk or more at these low fibre concentrations.

> "Even within the concentration interval of 0.1-0.2 f/µg dry weight [i.e. 100,000 to 200,000 fibres per gram dry weight], a significantly increased odds ratio of 4.5 was obtained. Previously, the same method of tissue analysis was used to estimate a 95 percentile of the amphibole fibre concentration of 0.1 f/µg dry weight for persons without detectable exposure to asbestos at the workplace. Therefore, within the range of the normal background level [up to 300,000 fibres per gram dry lung in Germany], a positive dose response is observed." [p 191].

This study did not detect an increase in the OR for chrysotile or for other mineral fibres.

5.89 The risk detected by Rödelsperger *et al.* [25] appears to correlate reasonably well with the French case-control study reported by Iwatsubo *et al.* [136], which found an OR for mesothelioma of 4.2 at estimated cumulative exposures of 0.5-0.99 fibre-year, with elevation of the OR at about 0.5 fibre-year.

(v) No lower threshold (minimum) level of asbestos exposure has been delineated, below which there is demonstrably no increase in the risk of mesothelioma.

5.90 This observation is expressed by Hillerdal [20] in the following terms:

> "There is no proof of a threshold value — that is, a minimal lower limit below which asbestos fibres cannot cause the tumour [i.e. mesothelioma] — and thus it is plausible that even such low exposure can

[17] Fibre-year = concentration of airborne asbestos fibres (f/ml) x years of exposure.

> cause mesothelioma (even if the risk is extremely low). Patients with mesothelioma whose lungs show fibre concentrations within the normal range cannot be dismissed as background cases, — that is, not due to asbestos. ... The only way to prove such a hypothesis would be to compare the incidence of mesothelioma in a group with such background exposure with the incidence in a truly non-exposed group. This is not possible, as no such group can be found." [i.e. the lung tissue of virtually all mammals contains some asbestos fibres derived from natural, environmental or occupational sources] [p 510].

5.91 Points worth emphasis in Hillerdal's review [20] include reports of mesothelioma among school teachers (9/487 patients with mesothelioma in one reference), in jewellers, and in individuals exposed to asbestos insulation at home (6/262 patients with mesothelioma according to one reference). Hillerdal [20] also makes the point that low-level asbestos exposure "more often than not contains peak concentrations which can be very high for short periods" (e.g. airborne asbestos fibre concentrations of up to 78 f/ml from sweeping asbestos from the floor [New Caledonia]).

(vi) The dose-response relationship for commercial Canadian chrysotile and mesothelioma incidence is also linear at high levels of exposure.

5.92 For example, among the Quebec chrysotile miners and millers it has been noted that:

> "All the observed 38 cases were pleural with the exception of one of low diagnostic probability, which was pleuro-peritoneal. None occurred in workers exposed for less than two years. There was a clear dose-response relationship, with crude rates of mesotheliomas (cases/thousand person-years) ranging from 0.15 with cumulative exposure < 3530 million particles per m^3 (mpcm)-years (< 100 million particles per cubic foot (mpcf)-years) to 0.97 for those with exposures of more than 10 590 mpcm-years (> 300 mpcf-years)." [EHC 203, p 8].

(vii) So far as I am aware, there are no observational data on dose-response relationships between chrysotile only at low exposure levels and mesothelioma incidence; in this respect, estimates are based on extrapolation of a linear dose-response line from high exposures down to low exposures.

> "Overall, the available toxicological data provide clear evidence that chrysotile fibres can cause fibrogenic and carcinogenic hazard to humans. The data, however, are not adequate for providing quantitative estimates of the risk to humans. This is because there are inadequate exposure-response data from inhalation studies, and there are uncertainties concerning the sensitivities of the animal studies for predicting human risk." [EHC 203, p 7].

5.93 Because of the lack of such data, no definite threshold for chrysotile in relation to mesothelioma and lung cancer has been delineated: According to EHC 203 (p 144):

> "(a) Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks."

5.94 In summary:

TABLE 5: ASBESTOS-RELATED DOSE-RESPONSE RELATIONSHIPS FOR MESOTHELIOMA

	Amphiboles	**Chrysotile**
Heavy exposure	Dose-response effect; linear	Dose-response effect; linear
Low-level exposure	Dose-response effect	No data
Threshold	No threshold delineated	No threshold delineated

(viii) To the best of my knowledge, there are no observational data on the interactive effect of low (or for that matter, high) concentrations of inhaled chrysotile fibres only, when these are

superimposed later and separately upon a pre-existing amphibole ± chrysotile burden within lung tissue (?superimpositional additive or multiplicative effect).

5.95 For example, it has been estimated that up to 15-20 per cent of men in industrialized societies may have sustained occupational exposure to asbestos (chrysotile/amphiboles). Rödelsperger *et al.* [137] indicate that fibre concentrations of 100,000-200,000 amphibole fibres per gram dry weight lung tissue may be expected for about 5 per cent of the population in Germany. We do not know what the effect of subsequent chrysotile fibre inhalation on top of this type of amphibole burden might be.

> "Data were analysed on a case-referent basis, to relate relative risks of mesothelioma to dose of fibre, as measured both by lung content and estimated airborne exposure. Multivariate analysis of cases found a dose response relationship for lung fibre content of crocidolite, amosite and chrysotile and the development of mesothelioma. Either a multiplicative or additive model could be used to fit the relative risk/dose coefficients for the various asbestos types. A progressive increase in relative risk with increasing fibre content was reported for all fibres ... . Tests for trend were highly significant in all cases." [NICNAS 99, p 61].

(ix) There is a long lag-time between asbestos exposure and the subsequent diagnosis of mesothelioma (10 years as a minimum; usually in the range of 20-40 years). It follows that the mesotheliomas encountered in the 1990s and the incidence of mesothelioma in various nations are a consequence of exposures, especially occupational exposures, sustained from the 1940s through to the 1970s and even beyond.

5.96 Exposures from the 1940s through to the 1980s usually involved one or more of the amphibole varieties of asbestos. For example, asbestos-cement building products used in Australia usually contained one or more of the amphibole varieties of asbestos, namely crocidolite or amosite, or both, at different times; in this respect, the use of crocidolite in the products was discontinued in 1966, and amosite in 1984.

5.97 Peto *et al.* [24] make this point in the following terms:

> "The extraordinarily high mesothelioma incidence throughout Western Europe in men born around 1945-50 reflects the extent of asbestos use in the 1960s and 1970s at the beginning of their working lives. Annual raw asbestos imports to European Union countries peaked in the early to mid 1970s and remained above 800 000 tonnes per year until 1980, falling to about 100 000 tonnes by 1993 (European Commission, 1996). Increasingly stringent exposure limits were enforced in the manufacture of asbestos-containing products over this period, but exposure to users of such materials, particularly in the building industry, remained virtually uncontrolled in many countries. Chrysotile asbestos products are still widely used in several European countries, and maintenance or demolition work on older buildings may result in substantial exposure to amphiboles as well as to chrysotile. We have not included men born after 1955 in our projections, but the effects of asbestos exposure during the 1980s and 1990s, although not yet apparent, may prove considerable." [p 670].

(x) Properties of asbestos fibres that determine carcinogenicity

5.98 As indicated in the documents submitted to the WTO Panel, the properties of asbestos fibres implicated for mesothelioma induction (and, possibly, lung cancer and other disorders), can be summarized as the **three Ds**:

5.99 **Dose**: this issue is covered in the preceding sections [(f)(ii)to (vi)].

5.100 **Dimensions**: according to *The Stanton Hypothesis*, the carcinogenicity of asbestos fibres appears to reside primarily in long thin fibres (length > 5 µm and especially > 8 µm, and in the range of 10-20 µm, and diameters < 0.25 µm) — e.g. see Pott [142]. On the other hand, shorter fibres appear to be less carcinogenic, although data indicate that tremolite fibres > 4 µm in length

and < 1.5 µm in diameter produce malignant mesenchymal tumours when implanted into the pleural cavities of rats [2]. On the other hand, very short-length fibres appear to have little carcinogenic activity, although Churg [143] comments on fibre dimensions in the following terms:

> "There has been extensive investigation of the relation of mesothelioma induction and fiber size in experimental models. Using intrapleural inoculation of different types of fibers with different size distributions, Stanton *et al*. concluded that long, thin (i.e., high aspect ratio) fibres were much more powerful mesothelial carcinogens than were short, thick fibers and that fiber type was less important. The exact size of fiber that qualifies as long and thin is unclear: fibers ... longer than 8 µm and widths narrower than 1.5 µm are usually cited from Stanton's work, but the same experiments show that fibers with lengths greater 4 µm and widths less than 0.25 µm were also effective carcinogens. The Stanton hypothesis has been supported by animal inhalation experiments using size-separated fibers: few mesotheliomas were found with either amosite or chrysotile prepared to contain few fibers longer than 5 µm.
>
> Human data on the question of fiber length and mesothelioma are equivocal. The tremolite found as a natural constituent of chrysotile ore is a relatively short, thick fiber compared with commercial amosite or crocidolite, and if one attributes 'chrysotile-induced' mesotheliomas in man to the tremolite component, the differences in mesothelioma do correlate with fiber size. However, attempts to prove this proposition directly have produced equivocal results ... McDonald *et al*. concluded that the number of fibers longer than 8 µm explained most mesotheliomas and that chrysotile played no role. However, Rogers *et al*. found that fibers both longer and shorter than 10 µm, including chrysotile fibers, played a role, although long fibers were generally more important. The problem with both of these studies is that most patients with mesothelioma have had occupational asbestos exposure, and fibers in lungs from those with occupational exposure are always longer than fibers in the general population; thus the same result would have been obtained if the test group were exposed but had no disease or some disease other than mesothelioma. My colleagues and I have attempted to circumvent this problem by comparing fiber sizes in a chrysotile mining and milling cohort and a cohort with heavy amosite exposure, using exposed workers with no disease as the control group. In neither cohort could we show that fibers in mesothelioma cases were significantly longer and thinner than those in the other disease categories or even in the disease-free workers." [p 353].

5.101 In other words, it is possibly the bio-persistence of amphibole fibres that is important for mesothelioma induction, rather than precise fibre dimensions.

5.102 **Durability** (bio-persistence): the greater mesotheliomagenic (mesothelioma-producing) potency of the amphiboles in comparison to chrysotile is widely ascribed to greater persistence of the amphiboles in tissues, with significantly longer half-lives than chrysotile (please see later discussion, Section (g)(v)). On the other hand, it is conceivable that the same effect might be achieved by sustained inhalation of chrysotile over a prolonged time interval or, possibly, shorter, but more intense exposures so that the chrysotile fibres persist despite shorter half-lives than the amphiboles.

(xi) There is general though not universal agreement of a differential potency between the amphiboles versus chrysotile for mesothelioma induction

5.103 In this respect, the amphiboles are substantially more potent, with estimates ranging from 2-4X, to 10X, to 12X on a fibre-for-fibre basis, to 30X, to a 30-60X greater potency, or more (e.g. please see EHC 203). A minority view that the amphiboles in chrysotile have roughly equal mesotheliomagenicity is not supported by the prevailing evidence for humans. Although acknowledging the greater potency of the amphiboles for mesothelioma induction, some argue that chrysotile is of equal or greater importance overall, because chrysotile accounts for > 95 per cent of world asbestos production. According to this perspective, commercial chrysotile is a weaker carcinogen on a fibre-for-fibre basis, but this lesser potency is multiplied across a much greater tonnage, leading to an overall equivalent or greater effect [144].

(xii) Tobacco smoke plays no role in the development of mesothelioma at any anatomical site — unlike the synergy between asbestos and tobacco smoke for the causation of asbestos-related lung cancer (see section (i)(i) below).

(g) Commercial Chrysotile and Mesothelioma Induction

(i) There is general agreement that commercial chrysotile has the capacity to induce mesothelioma in experimental animals and humans

5.104 There is dispute, however, over which fibres in commercial chrysotile are implicated (i.e. the predominant chrysotile or the trace quantities of fibrous tremolite).

(ii) Canadian chrysotile contains trace amounts of tremolite, including fibrous tremolite, as a contaminant [2, 10, 13, 14, 145-148]

5.105 The amount of tremolite appears to vary from one sample to another, but is generally < 1 per cent (please see EHC 203).

(iii) It has been argued that the occurrence of mesotheliomas among the Quebec chrysotile miners and millers is a consequence — not of the chrysotile per se — but of the coexistent trace quantities of tremolite (a non-commercial amphibole).

5.106 Analysis of the asbestos fibre content of lung tissue from this cohort demonstrates disproportionately high concentrations of tremolite in comparison to chrysotile; this appears to represent a bio-accumulation phenomenon whereby chrysotile is cleared from lung tissue more rapidly than the tremolite, so that the tremolite not only persists but increases in proportional concentration. In this respect, the tremolite content of the lung tissue can be used as an index on the past chrysotile exposure and some claim that the incidence of mesotheliomas in the same cohort can be related directly to the tremolite content [13, 14].

(iv) It is known that fibrous tremolite has the capacity for mesothelioma induction

5.107 Mesotheliomas related to the use of tremolite in whitewash or stucco have been reported in Turkey, Greece, Cyprus and Corsica [149-152] (for additional references, see Hillerdal [20]).

> "Tremolite asbestos, a minor component mineral of commercial chrysotile, has also been shown to be carcinogenic and fibrogenic in a single inhalation experiment and an intraperitoneal injection study in rats. Exposure/dose-response data are not available to allow direct comparison of the cancer potency of tremolite and chrysotile." [EHC 203, p 6].

5.108 Tremolite has also been implicated in lung cancer and mesothelioma induction in a group of vermiculite miners in Montana [2, 16, 153, 154]. It appears that these miners were exposed only to tremolite-actinolite fibres. The group was shown to have a:

> " ... very high lung cancer incidence (standard mortality ratio [SMR] 285 ...), as well as four cases of mesothelioma and eight of pneumoconiosis. Examination of sputum samples from all but three (170/173) current workers demonstrated asbestos bodies (AB) in 75%, the numbers showing a close parallel with cumulative exposures in fibre-years." [2] [p 493].

5.109 Case [2] has extensively reviewed the biohazards of tremolite, including epidemiological investigations in humans and experimental data on animal models. In his review, he emphasized the pathogenicity of the tremolite found in Quebec chrysotile samples, especially at Asbestos and in the Thetford mine:

> "Tremolite was not identified in Montreal air, was just detectable (0.2 fibres/l) in Asbestos, and was one order of magnitude higher in Thetford mines (still only 1.5 fibres/l or 0.0015 fibres/cc ...)." [pp 496-497].

5.110 He also favoured the expression "chrysotile/tremolite" for Quebec chrysotile:

> "As to the separate issue of 'chrysotile vs. tremolite', few would dispute the abilities of both to produce lung cancer and asbestosis, again in sufficient exposure dose. The weight of epidemiological, animal, and, especially, lung internal-dose biomarker studies leads to the inevitable conclusion that it is the tremolite 'component' of Quebec chrysotile which causes mesothelioma [but please see later discussion in this report]. It is unfortunate that adequate terminology for tremolite-contaminated chrysotile has not been introduced: I for one would favour the simple compound phrase 'chrysotile/tremolite'." [p 500].

5.111 Case [2] also states:

> " ... it becomes important to know to what degree 'chrysotile-in-place' is really 'chrysotile/tremolite-in-place'. No easy answer can be expected: both bulk analyses and air sampling, even with analytical electron microscopy, can miss very low levels of tremolite. Studies in the Quebec mining district indicate that, at the very least, such low levels (roughly 0.0015 fibres/cc) can induce biological effects (i.e., pleural plaques). Unfortunately, only expensive *in vivo* animal bioaccumulation assay systems can truly answer the question: the alternative is to wait 40 to 50 years for the next wave of asbestos disease — which is likely to occur mainly among present-day asbestos abatement workers and to some degree in custodial personnel and other tradesmen" …. [p. 500].

(v) Clearance of chrysotile from lung tissue

5.112 It is well known that chrysotile fibres are cleared more rapidly than amphiboles, especially in long-term studies [145]. Clearance of amphibole fibres does occur and the clearance mechanisms appear to be more effective for short fibres (for both chrysotile and the amphiboles) so that the mean length of retained fibres increases over time. Churg and Vedal [155] calculated a half-life in lung tissue of about 20 years for amosite. Estimates of the tissue half-life for crocidolite fibres have been somewhat shorter (in the order of 5-10 years) [156-158], and de Klerk *et al*. [158] could find no difference between the clearance rates for long and short fibres. Oberdörster [159] estimates human clearance half-times to be about 90-110 days for chrysotile and 200-1500 days for crocidolite fibres > 16 µm in length, based on extrapolated rat and primate inhalation data.

5.113 It has been claimed that chrysotile is cleared from lung tissue within 28-48 hours of inhalation. This claim seems extraordinary and begs the question: why, if chrysotile is cleared from lung tissue so rapidly, is it still demonstrable in human lung tissue many years or decades after cessation of inhalation of commercial chrysotile (or mixtures of asbestos types)? For example, in one of my recent referral cases — an elderly man with lung cancer who sustained exposure from mixing loose asbestos and sweeping up dried insulation materials — an asbestos fibre analysis carried out on lung tissue resected 16 years after his exposure stopped showed a total asbestos fibre count of 8,440,000 fibres/gram dry lung (> 1 µm in length; aspect ratio = 3:1), made up by 6,250,000 chrysotile fibres + 940,000 tremolite fibres + 940,000 anthophyllite fibres + 310,000 crocidolite fibres (the 24 year lag-time is enough for a carcinogenic effect).

(vi) The Quebec chrysotile cohort

4.114 In an analysis of mesotheliomas among the Quebec chrysotile miners and millers, up to 1997, McDonald *et al*. [13, 14] reported 38 mesotheliomas, and most of these occurred after prolonged and heavy exposure, especially at the mine where the greatest concentrations of trace tremolite occurred (Thetford). For example, these authors [13] recorded the breakdown of the mesotheliomas shown in Table 6 (below).

5.115 McDonald *et al.* [13] identify two main reasons for the low mesothelioma rate from the five smallest mines (1 case only among 6010 person-years, equivalent to 166 cases per million person-years): firstly, workers within this sub-group were younger than the remainder of the cohort; secondly, these mines had been opened recently so that "there were inadequate periods of latency". A single additional mesothelioma shortly after completion of the study would erase the difference in incidence rates between the five smallest mines and the main complex. McDonald *et al.* [13] go on to indicate that the other rates are "reasonably comparable". In comparison to the Thetford main complex, there were relatively few mesotheliomas among workers at the asbestos mine and mill (23 versus 8), despite nearly equivalent person-years of observation; in addition, asbestos fibre analysis on lung tissue demonstrated crocidolite and amosite in five out of the eight cases from the mine and mill at Asbestos and in two out of the five mesotheliomas from the Asbestos factory (Table 7, below). In focussing on the Thetford mines group, it was noted that most of the mesotheliomas came from the five central mines (Area A; Group C) as opposed to the 10 peripheral mines (Area B; Group P), so that the odds ratio for mesothelioma for Group C plus employees who had jobs in both Area A and Area B (Group M) was 2.50 (based on net service; 20 adjusted years), in comparison to an odds ratio of 0.80 for Group P.

TABLE 6: MESOTHELIOMAS AMONG QUEBEC CHRYSOTILE MINERS AND MILLERS, 1997

	Number of mesothelioma deaths	**Subject-years (000s)**	**Rate (per 100,000 subject-years)**
Thetford Mines:			
Main complex and the oldest of the smaller mines	23	65.14	35.3
The five smallest mines	1	6.01	26.6
Asbestos:			
Mine and mill	8	60.64	13.2
Factory	5	10.84	46.2

From McDonald *et al.* [13].

5.116 The clear implication of this complex and sophisticated study is that the risk of mesothelioma was related strongly to years of service in the central area at Thetford where geological factors "in Area A would probably result in tremolite, some in fibrous form, being mined with the ore". In addition, the mesothelioma rate for miners and millers was > 2.5 times higher at Thetford mines (excluding the smallest mines) than at Asbestos, and this difference was also attributed to differences in the amount of fibrous tremolite in the ores. Despite these differences within the cohort for the distribution of mesothelioma related to chrysotile and tremolite (and also to crocidolite and amosite at the Asbestos factory and the Asbestos mine and mill), the results indicate that Quebec chrysotile — on average contaminated by fibrous tremolite in small amounts — is capable of mesothelioma induction: the Abstract describes 25 mesotheliomas from the Thetford mines, representing a mesothelioma rate of 337 per million person-years, which is substantially (almost 20X) higher than the mesothelioma incidence rate of about 17 per million per person-years for men in British Colombia and the USA in 1982 and 1973-1984 respectively, and well above the background rate for spontaneous mesotheliomas of 1-2 per million person-years.

TABLE 7: ASBESTOS FIBRE CONCENTRATIONS IN LUNGS AT AUTOPSY FROM 21 MESOTHELIOMA CASES AMONG QUEBEC CHRYSOTILE MINERS AND MILLERS
(FIBRES PER µg: GEOMETRIC MEANS)

Place of employment	No. of cases	Chrysotile	Tremolite	Crocidolite	Amosite
Mines and mills					
Thetford Mines	14	12.8	104.1	0	0
Asbestos	5	4.3	7.5	1.7	0.3
Factory					
Asbestos	2	2.1	0.5	6.4	0.3

Table from McDonald *et al.* (1997): Table 2 in the original reference. See also Table 1 in the original.
In calculating geometric means, a zero count has been replaced by half the detectable limit.
For crocidolite and amosite, all counts were zero: i.e. below the detectable limit.
For fibre counts/g lung tissue, multiply the figures by 106.

5.117 In the final two paragraphs of the paper, McDonald *et al.* [13] comment as follows:

> "The tremolite hypothesis, if correct, has several important implications. First, it supports the widely but not universally held view that most, if not all, asbestos-related mesotheliomas are caused by amphibole fibres. This in turn points to fibre durability and biopersistence as critical factors in aetiology ... a point of even greater relevance in assessing the safety of man-made mineral fibres. Second, it implies that uncontaminated chrysotile carries very little risk of mesothelioma. In Asbestos, exposures were not to uncontaminated chrysotile, but also to some tremolite and crocidolite, yet among the miners and millers only five deaths from a total of over 3,300 can be confidently attributed to their work.
>
> At present-day levels of dust control the mesothelioma risk must be vanishingly small. Even so, it remains desirable to minimise, perhaps by screening, the contamination of commercial chrysotile by amphibole fibres, however difficult this may be." [p 718].

5.118 Despite the importance of this study by McDonald *et al.* [13], the following comments can also be made:

- The number of mesotheliomas in all groups except for the Thetford main complex was small (1, 8 and 5 mesotheliomas respectively; please see Table 6 above). In this respect, misdiagnosis or misclassification of the mesotheliomas according to the places worked could significantly affect the results, although there is no evidence that this happened; however, the probability for the diagnosis of mesothelioma also varied, with a high probability in 19 cases, moderate probability in 14, and a low probability (though considered more likely than not) in five; of these 38 cases, only 18 had been coded on the death certificate to ICD 163, and the rest to a variety of other diagnostic codes. Furthermore, in analysing the mesotheliomas according to Area A versus Area B at the Thetford mines (groups C, M and P), the numbers were 104 for group C, 69 for group P and 35 for group M; McDonald *et al.* noted that the odds ratio for group P was unstable as shown by the "very wide confidence intervals, and as the point estimate is well below unity it is quite unrealistic ".

- The low incidence of mesotheliomas in the Quebec chrysotile cohort appears to parallel similar low incidence rates for asbestosis and lung cancer for the same cohort

> [160, 161]; the incidence rates for lung cancer and mesothelioma appear to be different in other chrysotile-exposed cohorts.

5.119 For these reasons and because of the different rates of various asbestos diseases (asbestosis, lung cancer and mesothelioma) between the Quebec cohort and other groups of workers, I would be reluctant to recommend national policies from the findings in this cohort in isolation, and I would look for coherence of the evidence across different cohorts and studies.

5.120 In relation to the Quebec cohort, there is an important error in the Canadian reply to Question 4 (see Annex II) from the European Communities, where the following statement is made:

> "Regarding asbestos-related mesothelioma, a number of studies have demonstrated cogently that this type of cancer is almost exclusively linked to exposure to amphiboles. Cases of mesothelioma in chrysotile asbestos miners in Quebec are quite rare — in a cohort of 11,000 workers who were very carefully tracked (in the McDonald study), there were no more than 50 or so cases over several decades. Exhaustive research on their employment history revealed that most of the cases were related to short-term exposure to commercial amphiboles. For example, during World War II, some of the miners with mesothelioma had worked in plants manufacturing products for the Allied Forces and amphiboles imported into Canada had been used to make a variety of products, including gas masks, to assist in the War effort."

5.121 The statement that "most of the cases were related to short-term exposure to commercial amphiboles" is incorrect and misleading. As demonstrated in the study by McDonald *et al.* [13], most of the mesotheliomas occurred among chrysotile miners who worked at the Thetford main complex, without exposure to commercial amphiboles such as crocidolite or amosite. This is clearly shown in Table 7 (above), slightly modified from the paper by McDonald *et al.* [13] where fibre burden analysis on lung tissue from 14 mesothelioma cases from the Thetford mines showed both chrysotile and a high concentration of tremolite, with a zero count for the commercial amphiboles crocidolite and amosite. The point to be emphasized is that the mesotheliomas from the Thetford mines were not related to commercial amphiboles such as crocidolite or amosite, but to chrysotile with its content of fibrous tremolite.

(vii) As discussed earlier, a dose-response relationship between the incidence of mesothelioma and cumulative asbestos exposure has been demonstrated for commercial chrysotile.

5.122 Mesotheliomas have also been produced in experimental animals by implantation and inhalation of chrysotile (presumably also containing trace amounts of tremolite). Mesotheliomas can also be induced in rats by intraperitoneal injection of chrysotile, with evidence of a dose-response effect [1] (see also bibliography for EHC 203).

> "In non-inhalation experiments (intrapleural and intraperitoneal injection studies), dose-response relationships for mesothelioma have been demonstrated for chrysotile fibres." [EHC 203, p 5].

(viii) Chrysotile is also known to be toxic to a variety of cell lines in vitro, with induction of a variety of chromosomal alterations (e.g. please see EHC 203, pp 69-102).

(h) Other Chrysotile-Exposed Cohorts and Studies

5.123 In addition to the Quebec chrysotile miners and millers, mesotheliomas have also been reported among other workforces apparently exposed only to chrysotile, with no significant tremolite.

(i) Russia

5.124 Chrysotile from the Urals region (Uralasbest) in Russia [162, 163] is said to represent pure chrysotile. Although precise figures for the mesothelioma incidence in this area are difficult to procure, Kogan [164] makes the following comment in a recently-published textbook on occupational lung diseases:

> "In the Middle Ural mountains, the main asbestos mining region in Russia, only chrysotile asbestos is produced. In the 50 districts of this region, the mortality from mesothelioma over a 10-year period was six-fold higher than the average rate in the Sverdlovsk region, an area of negligible asbestos mining. Most with mesothelioma had worked at the asbestos mining and milling plants, or had lived in an adjacent town near old and very 'dusty' mills." ... . [p 251].

5.125 Because it is difficult to equate exposure levels in the Russian chrysotile industry with other industries (e.g. the airborne fibre concentrations at Uralasbest are usually expressed as gravimetric measurements), and I have been unable to ascertain the numbers of cases relative to exposure levels, I consider this evidence to be weak in comparison to other studies.

5.126 One might expect data on mesothelioma incidence in Central and Eastern European nations to be of interest, from an assumption that some of these countries would have imported mainly chrysotile from Russia until the breakup of the Soviet Union. Unfortunately, it is difficult to evaluate national mesothelioma statistics, because a number of these nations also imported amphibole asbestos. For example, in Slovenia, the total consumption of asbestos (1947-1995) was 580,000 tonnes, of which crocidolite accounted for 37,133 tons, until its use was stopped in 1992 [165]. Similarly, the annual usage of asbestos in Bulgaria during the 1970s and 1980s reached approximately 32,000 tons of chrysotile (mainly from Russia and Canada), together with about 1000 tons of crocidolite from Africa and 6000-7000 tons of Bulgarian amphibole material (anthophyllite and tremolite) [166]. In Poland, total consumption of asbestos for the manufacture of asbestos-cement products between the end of the Second World War until 1993 was about 1.4 million metric tons which included about 8500 metric tons of amosite and approximately 86,000 metric tons of crocidolite [167].

(ii) Germany

5.127 The former German Democratic Republic (GDR): Sturm *et al.* [5, 7] have published data on asbestos-related diseases and asbestos types in the German State of Saxony-Anhalt. These authors pointed out that:

> "All asbestos-based products were made from raw asbestos which was primarily imported from the former Soviet Union, particularly from the Kiembay mining area in the Ural mountains (said to represent pure chrysotile). Small quantities of long-fibred grades came from Canada (2,990 tonnes in 1989) and were mainly used for the manufacture of asbestos-cement pressure pipes free of amphibole asbestos. This was a share of approximately 7% in total imports. We never obtained any information about the Canadian mines from which the asbestos processed in the former GDR originated. ... However, several analyses carried out by the GDR Central Institute for Industrial Medicine confirmed that both the Canadian and the Russian asbestos were pure chrysotile. In addition to these imports of chrysotile asbestos, smaller quantities of amphibole asbestos were imported. For example, in the period from 1980 to 1985, some 90 tonnes of anthophyllite were imported annually from Mozambique. This anthophyllite was used exclusively by a Berlin manufacturer and was of acid-proof products, similar to the way crocidolite had been used in previous years to produce filters, seals and acid and lye-proof plastic materials. In Saxony-Anhalt, our region of work, these amphibole imports did not have any significance from the point of view of industrial medicine" ... [p 318/173].

5.128 Between 1960 and 1990, a total of 1082 mesotheliomas was recorded in Saxony-Anhalt, and these included 843 "proven asbestos-accepted mesotheliomas"; Table 8 from Sturm *et al.* [5, 7] gives

a breakdown of 812 cases for which adequate data were available: 67 were said to follow exposure to chrysotile only, and 331 were associated with "chrysotile; possible amphiboles".

(iii) Italy

5.129 Two mesotheliomas have now been recorded among more than 900 workers employed at the Balangero mine and mill in Italy [168, 169]. EHC 203 gives the following summary:

> "The cohort of chrysotile production workers employed at the Balangero mine and mill ... was almost exactly one tenth the size of the Quebec cohort. At the end of 1987, when 427 (45%) of the cohort had died, there were two deaths from pleural mesothelioma, both in men employed for more than 20 years with cumulative exposure estimated respectively at 100-400 and > 400 f/ml years. One diagnosis was confirmed histopathologically, and one was based on radiological findings and examination of pleural fluid. Fibrous tremolite was not detected in samples of chrysotile from this mine, but another fibrous silicate (balangeroite), the biological effects of which are not known, was identified in low proportions by mass (0.2-0.5%). At a comparable stage in the evolution of the Quebec cohort, mesothelioma accounted for 10 of 4547 deaths, a lower but not dissimilar proportion." [p 112].

TABLE 8: MESOTHELIOMAS ACCORDING TO TYPES OF EXPOSURES TO ASBESTOS IN SAXONY-ANHALT

	Amphiboles	**Amphiboles and chrysotile**	**Chrysotile; possible amphiboles**	**Chrysotile**	**Mean values**
Age at beginning of exposure	25	28	28	34	28
Duration of exposure	16	21	19	14	19
Lethal period (years)	40	40	41	31	38
Age of person dying of mesothelioma	65	68	69	65	66
Number of mesotheliomas	135	279	331	67	N = 812

Note: All types of application of asbestos with common addition of chrysotile fall under the heading "Chrysotile. Amphiboles possible" when previous admixture of amphiboles cannot be definitely excluded. From Sturm *et al.* [5, 7] .

(iv) China

5.130 At the XV International Scientific Meeting of the International Epidemiological Association (Florence, September 1999), Yano *et al.* [170] presented a paper on lung cancer incidence in a cohort of 515 male asbestos workers heavily exposed to chrysotile containing < 0.001 per cent tremolite, in Chongqin; two mesotheliomas over 11,850 person-years of observation occurred in this cohort (discussion to the paper; assuming this rate to be representative, it would amount to 170 mesotheliomas per million person-years).

5.131 In a retrospective cohort mortality study of 1227 men employed at a chrysotile mine in Hebei Province of China before 1972, Zou *et al.* found three deaths from mesothelioma (please see EHC 203, p 120).

(v) United States

5.132 Two mesotheliomas have also been observed among the cohort of South Carolina chrysotile textile workers — who used Canadian chrysotile — studied by Dement *et al.* [171, 172] (please see EHC 203, p 115).

(vi) Australia

5.133 There is also some indication of an increased frequency of mesothelioma among Australian brake mechanics who were potentially exposed only to chrysotile from grinding of brake blocks that contained Canadian chrysotile (please see later discussion on friction products, and NICNAS 99 and AMR 99).

(vii) Zimbabwe

5.134 One pathologically confirmed case of mesothelioma has been recorded in association with occupational exposures to asbestos in the Zimbabwe mines and/or mills, with one other case said to resemble mesothelioma radiologically (EHC 203, p 121).

(viii) Fibre burden studies on human lung tissue from mesothelioma patients

5.135 Fibre burden analyses also support the notion that some mesotheliomas occur in association with, or as a consequence of, inhalation of pure chrysotile.

5.136 Morinaga *et al.* [173] detected asbestos fibres in 19 of 23 mesothelioma studied; amphibole fibres were found in 13 cases, but six were found to have only chrysotile fibres (five pleural mesotheliomas and one peritoneal mesothelioma). Nonetheless, the methodology for this study seems unimpressive, with relatively small numbers of fibres analysed.

4.137 The 1991 paper by Rogers *et al.* [3] recorded a substantial number of mesothelioma patients in whom the only detectable type of asbestos was chrysotile (Table 9), with evidence of a dose-response effect as reflected in a trend to an increasing odds ratio (OR) at a relatively low fibre concentration of = 10^6 fibres per gram dry lung tissue ($\log_{10}$ = 5.5–6; OR = 8.67).

TABLE 9: DISTRIBUTION OF FIBRE CONCENTRATION: TRANSMISSION ELECTRON MICROSCOPIC ANALYSIS, CHRYSOTILE ONLY (ALL LENGTHS)

		Mesothelioma cases		**Controls**		**Odds ratio**
		No.	Percent	No.	Percent	95% Cornfield Cl
f/g	0-200.000	12	48.0	26	83.9	
$\log_{10}$ (f/g)	5.3-5.5	1	4.0	2	6.5	**1.08** (0-17.95)
	5.5-6	7	28.0	3	9.7	**8.67**(1.77-48.14)
	6-6.5	3	12.0			
	6.5-7	1	4.0			
	7-8	1	4.0	$?^2_1 = 9.80$ (P<0.0005) (trend)		

From Rogers *et al.* [3]. Cl: confidence interval; f/g: fibres per gram of dried lung tissue.

5.138 Finally, fibre burden studies have demonstrated that both chrysotile fibres and amphibole fibres can translocate from lung parenchyma to reach the pleura; EHC 203 summarizes these findings in the following way:

"In a study of asbestos fibres in the lung parenchyma and the parietal pleura of 29 asbestos workers,

> Sebastien *et al*. (1980) found that chrysotile fibres predominated in the pleura and that amphibole fibres could not be detected. A similar result was reported by Dodson *et al*. (1990). Kohyama & Suzuki (1991) found short chrysotile fibres in pleural plaques and in mesothelial tumours. In contrast, Boutin et al. (1993) found 0.21 x 10^6 fibres per g of parietal pleura and 1.96 x 10^6 in samples of lung parenchyma. Fibre concentrations were higher in subjects with a history of asbestos exposure and most of the fibres were amphiboles. Churg (1994) reported detection of chrysotile fibres in the subpleural parenchyma in chrysotile miners and millers." [pp 64-65].

(ix) Other Observations

5.139 Nicholson and Raffn [8] analysed mesothelioma risk over 40 studies for which little or no exposure information was available, using the excess numbers of lung cancers as a measure of exposure and comparing the ratios of mesotheliomas to excess lung cancers across these studies. They suggested that:

> " ... the ratio of mesothelioma to excess lung cancer is the same for exposures to 100% chrysotile (presumably Canadian chrysotile), 97%+ chrysotile, 100% amosite, and mixtures of chrysotile, amosite and crocidolite, within statistical uncertainty. Only 100% crocidolite exposures appear to have a greater ratio, about two to four times that of predominantly chrysotile. This relatively small difference in the potential for crocidolite to produce mesotheliomas compared with other fibre exposure cannot explain the high risk seen in chrysotile exposures accompanied by a very small crocidolite exposure. The data speak strongly that much of the mesothelioma risk in predominantly chrysotile exposures is from the chrysotile." [p 402].

5.140 In other words, these authors appear to argue, like Smith and Wright [144], Stayner *et al*. [11], and Landrigan *et al*. [21] that although chrysotile may be cleared more rapidly from lung tissue than tremolite — and that tremolite can be used as an indicator of past chrysotile — it may not be valid to ascribe all the mesothelioma risk to the tremolite and to ignore the far more numerous chrysotile fibres. Nonetheless, I do not find Nicholson and Raffn's argument to be persuasive, taking into account the K values for different industries.

5.141 Therefore, it is my perception that epidemiological and experimental evidence clearly demonstrates that Canadian chrysotile with its trace amounts of fibrous tremolite has the capacity for mesothelioma induction. Although the tremolite may have a disproportionately large effect, it is my perception that the evidence does not allow one to conclude that the chrysotile has no effect on mesothelioma induction: there is evidence from other cohorts and studies that chrysotile per se can also induce mesothelioma, even when tremolite is undetectable, and in experimental models in animals, chrysotile is as carcinogenic as, and more toxic than, the amphiboles. However, there is also general agreement that in humans, chrysotile is substantially less carcinogenic for the mesothelium than the amphiboles, and my estimate is that it has a potency $1/10^{th}$ – $1/30^{th}$ the carcinogenicity of crocidolite, with amosite being less mesotheliomagenic than crocidolite but more carcinogenic than chrysotile on a fibre-for-fibre basis. Amosite is an important factor in the incidence of mesothelioma in the United States, because of its widespread use in insulation materials from the 1960s [155, 174-176].

(i) Abestos and Lung Cancer

5.142 Still the focus of some controversy, this subject has been reviewed by Henderson *et al.*: (i) Henderson DW, Roggli VL, Shilkin KB *et al.*, *Is Asbestosis an Obligate Precursor for Asbestos-Induced Lung Cancer?* In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases, vol 11*. Charlottesville: Michie; 1995;11:97-168 [177]; (ii) Henderson DW, de Klerk NH, Hammar SP, *et al., Asbestos and Lung Cancer: Is it Attributable to Asbestosis, or to Asbestos Fiber Burden?* In: Corrin B, ed. *Pathology of Lung Tumors*, New York: Churchill Livingstone; 1997:83-118 [131]; (iii) Leigh J, Berry G, de Klerk NH, Henderson DW., *Asbestos-Related Lung Cancer: Apportionment*

of Causation and Damages to Asbestos and Tobacco Smoke, In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases, vol 13*, Charlottesville: Michie; 1996:141-66 [178]; (iv) Multiple authors. *Consensus Report: Asbestos, Asbestosis, and Cancer: the Helsinki Criteria for Diagnosis and Attribution*, Scand. J. Work Environ. Health 1997;23:311-6 [113].

5.143 Some salient features of asbestos-associated lung cancer include the following:

(i) Synergy between asbestos and tobacco smoke

5.144 Historically, most asbestos workers have also been cigarette smokers, and the lung cancer rate in virtually all cohorts is an outcome of the combined and synergistic effects of tobacco smoke and asbestos. Vainio and Boffetta [179] emphasize that asbestos and tobacco smoke are complex carcinogens that can affect multiple steps in the multistage process of cancer evolution, and that the combined effects will depend on the relative magnitude of each carcinogen at each stage; the interactive effect ranges from less than additive to supramultiplicative, but the model for insulation workers approximates a multiplicative effect (reviewed in Henderson *et al.* [131]). If the multistage model of carcinogenesis holds, and asbestos and smoking act at different stages, then a multiplicative relationship follows [180]. Leigh *et al.* [178] have reviewed various models for the apportionment of fractional contributions from cigarette smoke and asbestos towards the development of lung cancer.

(ii) Lung cancer incidence rates for asbestos-associated lung cancer vary greatly from one cohort to another

5.145 Please see following discussion.

(iii) Asbestos fibre type and lung cancer risk

5.146 The greater carcinogenicity of the amphiboles for the mesothelium in comparison to chrysotile appears not to extend to the induction of lung cancer [11]. In this respect, chrysotile is implicated in one of the lowest rates of asbestos-associated lung cancer (in Quebec chrysotile miners and millers), but also the highest rate (in South Carolina asbestos textile workers who used Canadian chrysotile) [171]. The reasons underlying this = 30-fold difference in lung cancer risk remain unknown (reviewed recently by McDonald [161]; please see also EHC 203). The risk of lung cancer in other asbestos-exposed cohorts is intermediate between these two extremes [15].

(iv) Dose-response relationship

5.147 In most studies, there is a direct and linear relationship between the relative risk of lung cancer and cumulative exposure to asbestos, including chrysotile and the amphiboles.

5.148 Accordingly, EHC 203 gives the following account:

> "The slopes of the relationship between cumulative exposure to chrysotile and the relative risk of lung cancer are summarized in Table 23 for those studies that reported this information. These studies all expressed this relationship using the following linear relative risk (RR) model:
>
> RR = 1 + B x E
>
> where B is the slope and E is the cumulative exposure to chrysotile asbestos expressed in f/ml-years.
>
> The slopes from the studies of the mining and milling industries (0.0006 to 0.0017), the latter having been estimated on a subset of the cohort on which the former was based, and the friction production industries (0.0005 to 0.0006) are reasonably similar. Hughes *et al*. (1987) in a study of cement workers (section 7.1.2.1b) reported a similar slope (0.0003) in one plant (plant 1) that only used chrysotile, and

a nearly 20-fold higher slope (0.007) among workers only exposed to chrysotile in another plant (plant 2).

The slopes of 0.01 and 0.03 reported for the two studies of the chrysotile-exposed textile workers conducted on overlapping populations, as well as the slope of 0.007 from one of the two plants (plant 2) of cement workers in the study of Hughes *et al.* (1987), were an order of magnitude greater than those reported for the other cohorts. It should be noted that the two textile cohorts were identified from the same textile facility, but were based on different cohort definitions. Hence, it is not surprising that the slopes from these two studies were similar. The slopes in the studies of chrysotile-exposed textile workers are also remarkably similar to those reported in other studies of textile workers with mixed fibre exposures (Peto, 1980; McDonald *et al*., 1983b; Peto et al., 1985). This similarity in findings provides some support for the validity of the slopes reported in the chrysotile-exposed textile cohorts.

The reason for the much higher slopes observed in studies of textile workers is unknown, although several possible explanations have been suggested. The first is that these differences might be attributed to errors in the classification of exposures in these studies. Particular concern has been raised about errors in the exposure assessment related to conversions from mpcm (mpcf) to fibres/ml that were performed, particularly in the mining and milling studies (Peto, 1989). Sebastien *et al.* (1989) conducted a lung burden study specifically designed to examine whether the differences in lung cancer slopes observed in the Charleston chrysotile textile cohort and the Quebec mining industries could be explained by differences in errors in exposure estimates. Lung fibre concentrations were measured in: (a) 32 paired subjects that were matched on duration of exposure and time since last exposure; and (b) 136 subjects stratified on the same time variables. Both analyses indicated that the Quebec/Charleston ratios of chrysotile fibres in the lungs were even higher than the corresponding ratios of estimated exposures. This finding was interpreted by the author as being clearly inconsistent with the hypothesis that exposure misclassification could explain the large discrepancy in the lung exposure-response relationships observed in the two cohorts." [pp 118-119].

5.149 Boffetta [15] expresses the relationship in the following terms:

"A large number of studies have been conducted on lung cancer risk following asbestos exposure. The interpretation of their results is complicated by several factors: (i) dose, geological type of fibres and industry are all important determinants of risk and are strictly correlated; (ii) the biologically relevant exposures occur 20 or more years before appearance of the disease, and their quantitative assessment is imprecise; and (iii) the role of potential confounders, in particular, tobacco smoking, can hardly be evaluated. In general, the risk of lung cancer is smaller in studies of miners and friction product manufacturers, is intermediate in studies of asbestos-cement and asbestos product manufacturers, and is highest in studies of asbestos textile workers. This likely reflects a stronger carcinogenic effect of individual, long and thin fibres, like those occurring in the textile industry, as compared to grouped, short and coarse fibres, like those occurring in mining.

Several cohort studies provide sufficient details to allow a quantitative evaluation of the risk of lung cancer from cumulative asbestos exposure. In all cohorts, the empirical relationship fits well a linear correlation with no threshold, which can be expressed as:

$RR_1 = 1 + K_1*CE$,

where RR_1 is the relative risk of lung, CE represents cumulative asbestos exposure, expressed as fb/ml-yrs, and K_1 is the industry-specific slope of the relationship (RR for the increase in 1 fb/ml-year of exposure) for lung cancer and varies across cohorts. Similarly, the risk difference (RD_1) can be expressed as

$RD_1 = K_1*CE*Exp$,

where Exp is the number of expected cases of lung cancer. In other words, the number of cases of (or deaths from) lung cancer attributable to asbestos exposure depends on the number of expected cases (deaths), the cumulative exposure, and the intrinsic carcinogenic potential of the exposure

> circumstance. The value K_1 varies from 0.05-0.01 in cohorts of insulation and asbestos textile workers to 0.001-0.0005 in friction manufacturers and miners, while cohorts with mixed exposure have, in most cases, intermediate values. ... While all estimated values of K_1 are positive, the type of asbestos does not seem to be correlated to lung cancer risk.
>
> In the interpretation of these results, however, one should consider several limitations. Most studies are based on a small number of cases or deaths: for example, the risk estimate of 100 fb/ml-yrs for the cohort of asbestos textile workers presented by McDonald and colleagues (RR 2.4) has a 95% confidence interval from 1.7 to 3.8. Another source of uncertainty, and possibly bias, relates to the estimate of cumulative exposure: in the same cohort of asbestos textile workers, the range of RRs based on the extremes of the distribution of possible exposure values is 1.3-6.7. For these reasons, several governmental and scientific committees have suggested to adopt an 'average' value of K_1, independent from fibre type and circumstance of exposure ...: the most widely accepted value is 0.01 which corresponds to an increase of 1% of the risk of lung cancer for each fb/ml-yr of exposure. ...
>
> Tobacco smoking is the main cause of lung cancer, and this applies also to the cohorts of asbestos-exposed workers. Despite the limitations of the available studies, which limit the precision of the estimates of the combined effect of the two carcinogens, the risk from tobacco smoking seems to act synergistically with that of asbestos exposure, according to a multiplicative model. ... The available data are consistent with the most widely accepted model of quantitative dose-response between cumulative exposure to asbestos and lung cancer risk, which assumes a linear relationship with no threshold. Alternative models, however, would also be consistent with the data. In particular, as no precise data are available for cumulative exposures below 1 fb/ml, a model with a threshold at low exposure cannot be rejected." [pp 473-475].

(v) Histological types of lung cancer

5.150 Although some studies have shown a relative excess of adenocarcinomas in proportion to other histological types of lung cancer, all of the major histological types occur among asbestos workers in proportions equivalent to, or only slightly different from, those in the general population [112]. Therefore, the histological type of a lung cancer has no value in ascertaining whether or not asbestos has contributed significantly to the genesis of the cancer (reviewed by Henderson *et al.* [131]).

(vi) Lobar distribution and the central versus peripheral distribution of asbestos-related lung cancer

5.151 Some studies have reported a reversal of the upper lobe:lower lobe ratio for lung cancers in asbestos workers, in comparison to a reference non-exposed population. Recently, Lee *et al.* [181] addressed the lobar distribution of lung cancer in asbestos-exposed individuals and found that the tumours were predominantly located in the upper lobe (i.e. they did not find a reversal of the upper lobe to lower lobe ratio). The lobe of origin for a cancer has no value in ascertaining whether the cancer is likely to be asbestos-related. The distribution of lung cancer between the central versus the peripheral airways does not differ significantly in asbestos workers from a control non-exposed population (please see Henderson *et al.* [131]).

(vii) Asbestos and lung cancer risk

5.152 Cumulative exposure versus fibrosis (asbestosis): as discussed already, most epidemiological studies dealing with lung cancer risk in asbestos workers have reported a direct correlation between the relative risk of lung cancer and cumulative asbestos exposure, although the slope of the dose-response line varies from one cohort to another. Most of the documents submitted to the WTO appear to agree on this relationship, the main area of uncertainty or dispute being the question of whether a threshold exists or not.

5.153 However, Canada's answers to questions from the Panel and the European Communities appear to resurrect the fibrosis-cancer hypothesis, which postulates that asbestos does not induce lung cancer per se, but only through an obligate intermediary step of pulmonary fibrosis (asbestosis), so that fibrosis becomes the determinator of lung cancer risk, not cumulative exposure:

> "1. Canada does not disagree that chrysotile causes lung cancer. However, the way in which exposure to chrysotile asbestos may increase the risk of lung cancer has not yet been fully explained; it could be just an indirect cause. ...
>
> 2. The risk may become detectable in cases of long-term exposure to high levels, but it is by no means certain that chrysotile acts as a direct carcinogen or that it acts in the form of pulmonary fibrosis, which would be a precursor to neoplasia. In other words, exposure must be intense and long enough to induce pulmonary fibrosis, which predisposes the pulmonary parenchyma to a high risk of cancer."

5.154 It is my perception that the fibrosis?cancer hypothesis represents a minority opinion: with some prominent exceptions, most authorities in this area reject the fibrosis?cancer theory and focus instead on the asbestos fibre burden in lung tissue as the main determinator for lung cancer risk, as discussed earlier in this report.

5.155 The fibrosis?cancer hypothesis is predicated upon three key but flawed studies:

- In the investigation reported by Kipen *et al*. [182], there was major problem with case selection (only 138 cases out 450 — 31 per cent — had a tissue specimen with sufficient non-malignant tissue for assessment of fibrosis); in addition, the histological criteria used for the diagnosis of asbestosis are unacceptable to most pathologists — i.e. no asbestos bodies in some cases; fibrosis restricted to the subpleural zone considered to be asbestosis — so that this study seems to have suffered from an over-diagnosis of asbestosis [183, 184].

- As discussed in Section (e) above, the Hughes-Weill study [133] on chest X-ray opacities related to lung cancer mortality in New Orleans asbestos-cement workers had low statistical power, so that it had only a 40 per cent chance of detecting a significant lung cancer standardized mortality ratio (SMR) of 1.5. Other studies based on X-rays have shown an increase in risk or mortality for lung cancer in the absence of radiological asbestosis (e.g. Wilkinson *et al*. [185], Finkelstein [186] and de Klerk *et al*. [187]).

- The autopsy study on South African crocidolite miners reported by Sluis-Cremer and Bezuidenhout [188] was also bedevilled by problems of selection (black people excluded; autopsies on 36.7 per cent of deaths only; autopsies on cases for which compensation was sought). Analysis of the findings indicates that the effect of duration of exposure (the most accurately measurable of the exposure variables) was still significant even after adjustment for the grade of asbestosis and other variables. This indicates that exposure to asbestos still had an independent effect on lung cancer mortality even after adjustment for the grade of asbestosis, as in the study reported by Wilkinson *et al*. [185]. In subsequent correspondence, Sluis-Cremer and Bezuidenhout [189] conceded that when they carried out a logistic regression analysis, allowing for the grade of asbestosis, years of exposure accounted for most of the variation, but the degree of asbestosis still emerged as a highly significant risk factor.

5.156 Recently, Case and Dufresne [190] have commented as follows:

> " ... Hughes and Weill go much further in stating that asbestosis is a prerequisite for lung cancer attribution in those with asbestos exposure. This statement goes beyond the known facts and relies on mechanistic speculation. The authors believe that asbestosis is produced by a mechanism or mechanisms that will also lead to lung cancer. Their hypothesis requires that the mechanism(s) always be intermediate in that lung cancer always follows asbestosis. Finally, the speculation requires that lung cancer occurring without asbestosis can never be caused by asbestos exposure alone (or in synergy with cigarette smoking) regardless of the level of that exposure, and that no mechanism can occur that does not involve intermediate fibrosis. The biological fallacy of this argument has been well documented ... one must remember that lung cancer originates in the large airways, while asbestosis is a disease of the lung parenchyma at and beyond the respiratory bronchioles. ... To ignore our knowledge of indices of exposure other than the simple presence or absence of asbestosis is simplistic and biologically naïve." [p 1118].

5.157 The case-control studies carried out on South Carolina asbestos textile workers by Dement *et al.* [171] clearly undermine the fibrosis?cancer hypothesis and, in this respect, they constitute Popper's *Black Swan* factor:[18] Dement and his colleagues clearly identified a lung cancer SMR > 2.5 at 2.7-6.8 fibre-years of exposure (well below the exposure level necessary for histological asbestosis in the same cohort [191]).

(viii) Non-occupational asbestos exposure in Quebec and lung cancer risk

5.158 The first written submission from Canada also refers to the study reported by Camus *et al.* [140] on non-occupational exposure to chrysotile asbestos in Quebec and the risk of lung cancer:

> "It is also interesting to note the work of Dr Camus *et al.* (see Camus, M., Siemiatycki, J. Meek, B., *Nonoccupational Exposure to Chrysotile Asbestos and the Risk of Lung Cancer*, (1998) 338, New England Journal of Medicine 1565). They published a vast study on women in chrysotile mining communities in Quebec, many of whom were exposed to very high levels of fibres between 1920 and 1975. These women were subjected to exposure of 0.0107 f/ml,[19] higher than the current exposure limits in France, and literally thousands of times higher than the levels measured in public buildings. Nonetheless, no excess in lung cancer was detected in this population. According to the study's authors, this is particularly important in the light of the current French situation. In fact, applying the risk model adopted by France for the exposure studied, results in a forecast of approximately 100 lung cancer deaths, while in reality there are none. Likewise, use of the French risk model would have resulted in estimates of approximately 250 and, at any rate, no less than 50 deaths from mesothelioma, while the preliminary results of the study in question show only 10 cases, some of which may be associated with exposure to amphiboles. Research continues, particularly with an analysis of the work history of each individual in order to determine the exact link, if any, between these cases of mesothelioma and on-the-job exposure, as well as exposure to amphiboles."

---

[18]"The philosopher of science, Sir Karl Popper ... coined the term 'falsification' to express the concept that scientific theories are not proven by repetition of results but rather survive because they successfully withstand refutation (falsification). His example of the black swan makes this point clearly. Suppose you have a hypothesis that all swans are white ... you observe, say, 10,000 swans and they are all white. Another scientist repeats your efforts and observes another 10,000 swans: they too are all white. So far the theory is standing up well. The repetition helped to strengthen it — but if only a single black swan is sighted, this falsifies the theory: it is no longer tenable. Popper asserted that scientific statements have to be formulated in a manner that subjects them to the possibility of falsification. One of the important demarcating criteria between science and nonscience, according to Popper, is this formulation of statements in a manner permitting falsification" [pp 18-19] [44].

[19]This figure is inconsistent with the former limit of 0.1 f/ml in France, and contradicts Case's claim that the Quebec women were exposed at up to 1 f/ml [192]; at a level of 0.0107 f/ml (a figure two orders of magnitude less than 1f/ml), a cumulative exposure of 5 fibre-years would require residence of > 150 years (adjusted for equivalence to an 8-hour working day) and > 750 years to reach 25.0 fibre-years (using the same adjustment).

4.159 In fact, Camus *et al.* [140] investigated the relative risk of death from lung cancer among 2242 deaths between 1970 and 1989 among women = 30 years of age who lived in two chrysotile asbestos-mining areas that comprised eight towns of which three (Thetford mines, Black Lake and Asbestos) contained nearly all the asbestos mines and mills. Eighty percent of the women lived within 4 km of a mine or mill, and all lived within 10 km.

5.160 The estimated average cumulative level of exposure was 25 fibre-years (range 5-125 fibre-years) made up by neighbourhood exposure (16.0 fibre-years), household exposure of 7.8 fibre-years and occupational exposure of 1.2 fibre-years, making a total of 25.0. The authors of this study pointed out that:

> " ... The lower limit of 5 fibre-years per ml corresponds, for example, to 50 years of exposure to asbestos at a level of 0.1 fibre per ml (the actual mean ambient airborne asbestos level in the area in 1974); the upper limit of 125 corresponds for example, to 50 years of exposure to 2.5 fibres per ml — a relatively low exposure level in local asbestos-mining and asbestos-milling industries before 1960." [p 1568].

5.161 This investigation found a standardized mortality ratio of 1.0 in comparison to the reference population (i.e. no observed excess of lung cancer mortality). However, seven deaths from "pleural cancer" were observed (RR = 7.64; $p < 0.05$).

5.162 A few points about this study are worth emphasis:

- The Quebec chrysotile miners and millers have a low risk of lung cancer in comparison to other cohorts, such as the South Carolina chrysotile textile workers, for whom the frequency of lung cancer is at least 30 times higher. Therefore, it is not surprising that the low risk of lung cancer in the chrysotile miners and millers of Quebec extends across residents exposed environmentally to the same ore. In other words, the absence of a detectable increase in lung cancer mortality in female residents of this region of Quebec may not apply to other groups exposed environmentally to asbestos from other asbestos industries.

- The study reported by Camus *et al.* [140] stimulated considerable correspondence in the columns of the same journal (NEJM), and at least two of the correspondents (Churg [193] and Case [192]) emphasized that the seven-fold increase in mesothelioma mortality (seven cases) among the women was probably explicable by occupational exposure to amphiboles from manufacture of gas masks, repair of bags that contained imported asbestos, and, possibly in one case, domestic exposure to "tremolite brought home on miners' clothes".

  In his letter to the editor, Case [192] also pointed out that "[T]hese women were exposed to levels of chrysotile as high as 1 fibre per ml of air as recently as one month in 1984."

- I have some misgivings over the exposure estimates for this female population, and the figure of 25 fibre-years from environmental exposure in the general neighbourhood or vicinity of the Quebec chrysotile industry seems high in comparison to neighbourhood or environmental exposures from other industries. For example, ECH 203 (p 35) reproduces a Table of asbestos fibre concentrations in Quebec chrysotile mining towns, where the fibre concentration in 1984 is in the vicinity of 0.005 fibre/ml and the concentrations in 1973 and 1974 are given as 0.08 fibre/ml. In other words, Case's figure of 1 fibre/ml for one month in 1984 [192] may be doubtful, unless there were some catastrophic event in the industry, with

a burst of asbestos into the general environment. Unless earlier environmental airborne fibre concentrations were substantially above the 1973/1974 concentrations, it is difficult to see how a cumulative exposure of 25 fibre-years would come about; e.g. Camus *et al*. [140] state that residence in the area for 50 years at a mean fibre concentration of 0.1 fibre/ml would lead to the lower estimate of 5 fibre-years.

In addition, the estimate of 25 fibre-years seems high in comparison to data on environmental airborne fibre levels related to the Zimbabwean and Russian chrysotile industries. For example, EHC (p 47) states:

> "There are some data concerning fibre levels in the air close to chrysotile mines. Baloyi (1989) found fibre levels around the Shabani mine (Zimbabwe) to range from below the limit of detection of the method" (less than 0.01 f/ml) to 0.02 f/ml of air, assayed by PCOM. [PCOM = phase contrast optical microscopy].

Scherbakov *et al.* [163] also give a comparable environmental airborne fibre concentration in Asbest City of 0.1 mg/m^3 (comparative data for the same industry [194] suggest that the gravimetric measurement of mg/m^3 is very roughly equivalent to the same number of fibres/ml).

The point is that if the estimate of cumulative asbestos exposure in the Quebec female population is high, this would lead to underestimation of lung cancer risk or mortality. For example, no detectable increase in lung cancer mortality among the 2242 deaths would be expected at the low cumulative estimate of = 5 fibre-years.

- In addition, in their reply to the Letters to the Editor, Camus and Siemiatycki [141] state that "[W]e agree ... that the study had low statistical power to detect small risks; this was conveyed by the wide confidence intervals for our risk estimates ...", although they go on to indicate that the Quebec study should have detected a risk of the magnitude predicted by the Environmental Protection Agency [EPA].

(ix) The Helsinki Criteria

5.163 This set of criteria deals with attribution of lung cancer to asbestos for the individual patient [113]:

> "Because of the high incidence of lung cancer in the general population, it is not possible to prove in precise deterministic terms that asbestos is the causative factor for an *individual* patient, even when asbestosis is present. However, attribution of causation requires *reasonable* medical certainty on a probability basis that the agent (asbestos) has caused or contributed materially to the disease. The likelihood that asbestos exposure has made a substantial contribution increases when the exposure increases. Cumulative exposure, on a probability basis, should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk." [p 314; emphasis in original].

5.164 The Helsinki Criteria set an exposure level of = 25 fibre-years of exposure; however, it should be emphasized that this level of cumulative exposure is required for the individual patient as an index for an asbestos-attributable relative risk of lung cancer of = 2.0 (which, in the individual patient, equates to a probability of causation or material contribution of = 50 per cent — the civil standard of proof). Intended as a criterion for individual compensation, this exercise is clearly different from population-based relative risks relevant to the dispute before the WTO.

5.165 In summary:

TABLE 10: ASBESTOS-RELATED DOSE-RESPONSE RELATIONSHIPS FOR LUNG CANCER

	Chrysotile or Amphiboles
Heavy exposure	Dose-response effect; linear
Low-level exposure	Dose-response effect for South Carolina textile workers (chrysotile)
Threshold	No threshold delineated

(j) Some General Observations on Experimental Models of Asbestos Carcinogenesis, including *in vivo* and *in vitro* Systems

(i) In vivo experimental models

5.166 Although animal models of asbestos carcinogenesis - especially induction of mesotheliomas in animals such as rats - are of value to demonstrate the capacity of different fibres to induce tumours and to elucidate the mechanisms underlying carcinogenesis, they are not strictly comparable to carcinogenesis in humans, for a number of reasons:

- The airborne fibre concentrations to which experimental animals are exposed for inhalation experiments are substantially higher than in workplace or environmental situations for humans.

- The routes of administration of asbestos or other fibres - e.g. injection or direct implantation into the pleura or peritoneum - are not comparable to the human situation, with the exception of inhalation experiments.

- High concentrations of asbestos or other fibres are necessary to reduce latency intervals so that a reasonable yield of mesotheliomas or other cancers is obtainable within the life span of the animal used. In other words, the latency intervals are not comparable to the human model.

- There are known to be marked differences in the susceptibility of different species to asbestos carcinogenesis.

5.167 For example, in a review of asbestos and lung cancer, Henderson *et al.* [131] state the following:

> "The dose of asbestos delivered by inhalation or installation over a short time interval in experimental animals, the lag-times, and the histological spectrum of the tumors also make it difficult or impossible to extrapolate the findings from such models to humans. The exposure to asbestos in positive inhalation experiments seems to have been so high that fibrosis was an unavoidable association with an increased cancer risk (exposure to at least 100 f/ml, > 1,000 f/ml for some groups, 5 x 7 hours per week, up to 12 months or more). Wagner *et al*. remarked on a number of 'surprising' results in their study (e.g. no differences in carcinogenicity or fibrogenicity between chrysotile and the amphiboles). ...
>
> The sensitivity of humans to the carcinogenic effects of asbestos is about 100-fold greater than that of rats. ...
>
> ... Experimental studies of this type address asbestos inhalation in isolation, instead of asbestos combined with tobacco smoke [for the study of lung cancer]. Hence, they are of questionable relevance to most lung cancers in asbestos workers, for which tobacco smoke is an important co-factor.

> For the reasons stated above, we consider that the existing literature on tumorigenesis by inhalation of asbestos in laboratory animals allows no conclusions on the asbestos-asbestosis-lung cancer controversy in humans." [p 96].

5.168 Davis [195] comments in the following terms:

> "In experimental inhalation and injection studies, however, chrysotile has repeatedly produced as many mesotheliomas as other asbestos types. This finding probably indicates that the carcinogenic potential of chrysotile to cells is as high as the other asbestos types, and it is just sufficiently durable to exert its maximum effect in rats, although it is unable to survive long enough to do so in humans." [p 201; but see discussion in this report on chrysotile clearance from lung tissue, Section A.(g)(v)].

(ii) In vitro systems

5.169 It is obvious that the effects of asbestos and other fibre types on isolated cell lines used for *in vitro* studies are not comparable to the induction of mesothelioma or lung cancer in humans. *In vitro* studies of this type are of most value in showing that asbestos and other fibres can induce chromosomal injury, oncogene expression or mutations similar to those induced by other known carcinogens.

5.170 Detailed discussion of the voluminous literature on this topic lies beyond the scope of this report. Henderson *et al.* [131] give some details of the effects of asbestos on cell lines *in vitro*; more extensive reviews are given in EHC 203 (pp 69-102), Both *et al.* [196], and Mossman *et al.* [197-202], and Bielefeldt-Ohlmann [203]. Only a few recent studies on chrysotile follow:

- "In the study by Haugen *et al.* [204], chrysotile was about 10 times more cytotoxic than amosite or crocidolite (as assayed by inhibition of clonal growth rate) and > 100-fold more toxic than glass fibres; epithelial cells were 10-15 times more sensitive to the cytotoxic effects of asbestos fibres than bronchial fibroblasts from the same human. We can find no comparison with mesothelial cells in this paper [204], despite at least one claim to this effect [197] ... " [p 97].

- "Harrison *et al.* demonstrated synergy between the lung carcinogen N-nitrosoheptamethyleneimine (NHMI) and chrysotile in the production of hyperplastic epithelial lesions in the lungs of rats, with a dose-response relationship for NHMI, augmented by chrysotile. Neoplastic lesions (adenoma and adenocarcinoma) were found only in animals treated with both NHMI and asbestos, but the number of such tumours was small (N = 6 among 115 rats studied)" [p. 118; see Henderson *et al.* [131] for references].

- "Hei and Piao reported on malignant transformation of a human papillomavirus-immortalized human bronchial epithelial cell line (BEP2D) by a single 7-day treatment with chrysotile: the cells so treated evolved through a series of sequential steps to become tumorigenic, with the formation of progressively growing tumours in nude mice" [p. 118; see Henderson *et al.* [131] for references].

- In an investigation of the capacity of different asbestos fibre types to induce loss of heterozygosity [LOH] mutations in lymphocytes and diploid mesothelioma cells that were heterozygous for the HLA A2/A3 histocompatibility complex studied (in collaboration with Dr David Turner at the Department of Haematology-Oncology at the Flinders University), it was found that chrysotile was more toxic to the cell lines used so that few viable cells remained, making it difficult to evaluate LOH mutations, in contrast to UICC South African crocidolite.

- More recently, Dr. Turner and I have investigated the effects of UICC South African crocidolite injected into the peritoneal cavity of mice, for the investigation of somatic intrachromosomal recombinational events in mice that are transgenic for the gene that encodes the enzyme ß-galactosidase; using PCR [the polymerase chain reaction], we detected a 5-fold reduction in SIR within only a few days of administration of the crocidolite. This finding parallels the results obtained with other carcinogens (e.g. cytotoxic drugs used for cancer chemotherapy) and may be explicable by a reduction of SIR because the asbestos produces an increase in other classes of mutation (e.g. point mutations or deletions), or because of impairment of DNA repair mechanisms.

5.171 The picture now emerging on asbestos carcinogenesis is a prolonged multistage parametric process [205], in which asbestos fibres may participate in both the initiation and promotion phases [196]. Some classes of mutation potentially inducible by asbestos - e.g. loss of heterozygosity mutations - are implicated in the initiation or progression phases of cancer development in humans, thought to be related to loss of tumour suppressor genes (e.g. retinoblastoma, astrocytoma, and colonic, gastric, prostatic and breast cancer) [206-211].

5.172 Free radicals — generated either from the surface of the fibres themselves [205, 212-215] or via macrophages [213, 216-218] — have been shown to have genotoxic or clastogenic properties [205, 212-214, 217, 219, 220], and are also implicated in asbestos carcinogenesis.

2. Questions by the Panel and Comments by the Scientific Experts

Question 1:

High-density chrysotile products (i.e. products where chrysotile fibres are bound in a matrix, such as chrysotile-cement, as opposed to "friable" products, such as flocking and heat insulation) represent the main use of chrysotile asbestos. The parties to this dispute disagree as to the circumstances of exposure to chrysotile and the risks to human health associated with such products. In this context, various questions arise with respect to the risks to human health associated with the use of high-density chrysotile products, in particular chrysotile-cement (of particular concern are installation, modification, repair, maintenance, demolition and disposal).

1.(a) Canada argues that workers who are at greatest risk of exposure to chrysotile asbestos are, in descending order: (i) chrysotile miners and workers employed in the processing (milling) industry; (ii) workers in the chrysotile textile industry; (iii) workers involved in the production of friction materials (such as brakes, clutches); (iv) workers involved in the manufacturing of chrysotile-cement products; (v) workers involved in the removal of asbestos from buildings; and (vi) workers involved in construction, renovation, maintenance and the heat insulation of buildings. Furthermore, according to Canada, the last two categories are likely to be exposed to amphiboles. On the other hand, the European Communities argues that, in France, the secondary users, which includes installation, maintenance, repair, insulation, waste management and "handyman" type persons, etc. are at the greatest risk of exposure and that they are mainly exposed to chrysotile asbestos, since, for some fifty years, chrysotile has represented about 97 per cent of asbestos consumption in that country. Could you comment on these contrasting views, with a special focus on current uses and products?

Dr. de Klerk:

5.173 This question is rather curious and is either irrelevant or the wrong words have been used: trying to elucidate an ordering of working groups according to their "risk of exposure to chrysotile asbestos". The risk of an event is the probability that it will occur. The event in question here is that a

worker will come into contact with chrysotile asbestos. It is certain that the workers in groups (i) to (v) are exposed to chrysotile so their risks are all the same and equal to 1.0. Workers in group (vi) may not come into contact with chrysotile so their risk of exposure is less. The more relevant question here is: who is likely to receive the most exposure and therefore have the greatest risk of disease? In general, workers in well-regulated industries, where government inspection is mandatory, where there is a long history of efficient industrial hygiene practices, will have less risk of disease than those in the smaller less well-regulated industries. A good example can be found with silicosis: the majority of cases now occurring in both the USA and Australia arising from small unregulated industries with no awareness of risks or hygiene practices. Similar examples from own experience are: witnessing (in 1992) use of Russian asbestos in an asbestos-cement factory in Czechoslovakia (as it was then) where all the warnings on the bags of asbestos were in English; passing by demolition in progress of an old asbestos cement factory building in Sydney last month where no observable precautions of any kind were being taken. (Note added later: It has struck me that the misunderstanding with this question could be due to the relative imprecision of the French language, where "de" means both "of" and "from", words with quite different meanings, especially in this context!)

Dr. Henderson:

5.174 In past historical terms, the Canadian proposition about the classes of workers at risk of exposure to chrysotile is correct — provided that this risk is expressed in terms of a numerical value for the risk per person-years of observation (e.g. per 100,000 or 1 million person-years). However, this situation has changed over recent years, as airborne fibre concentrations have been reduced in the mining and milling industries and during the production of friction products. As one example, NICNAS 99 points out that manufacture of friction products (brake linings and gaskets) in Australia is a completely closed operation, with low airborne fibre concentrations.

5.175 EHC 203 refers to this reduction in airborne fibre concentrations:

> "Based on data mainly from North America, Europe and Japan, in most production sectors workplace exposures in the early 1930s were very high. Levels dropped considerably to the late 1970s and have declined substantially to present day values. In the mining and milling industry in Quebec, the average fibre concentration in air often exceeded 20 fibres/ml (f/ml) in the 1970s, while they are now generally well below 1 f/ml. In the production of asbestos-cement in Japan, typical mean concentrations were 2.5-9.5 f/ml in 1970s, while mean concentration of 0.05-0.45 f/ml were reported in 1992. In asbestos textile manufacture in Japan, mean concentrations were between 2.6 and 12.8 f/ml in the period between 1970 and 1975, and 0.1-0.2 f/ml in the period between 1984 and 1986. Trends have been similar in the production of friction materials: based on data available from the same country, mean concentrations of 10-35 f/ml were measured in the period between 1970 and 1975, while levels 0.2-5.5 f/ml were reported in the period between 1984 and 1986. In a plant in the United Kingdom in which a large mortality study was conducted, concentrations were generally above 20 f/ml in the period before 1931 and generally below 1 f/ml during 1970-1979." [pp 2-3].

5.176 In contrast, the risk per million person-years of observation may be less in building construction, renovation and maintenance workers, but this smaller risk is spread across a substantially larger workforce (i.e. there are many more carpenters/joiners, builder's labourers, electricians, plumbers and other tradespeople in Western societies than the numbers of workers engaged in the mining, milling or production of high-density asbestos-containing materials such as asbestos-cement sheets and pipes or brake blocks).

5.177 According to EHC 203:

> "It should be recognized that although the epidemiological studies of chrysotile-exposed workers have been primarily limited to the mining and milling, and manufacturing sector, there is evidence, based on the historic pattern of disease associated with exposure to mixed fibre types in western countries, that

risks are likely to be greater among workers in construction and possibly other user industries." [EHC 203, p 9].

"Past uncontrolled mixed exposure to chrysotile and amphiboles has caused considerable disease and mortality in Europe and North America. Moreover, historical experience to mixed fibre types in European countries has clearly indicated that a larger proportion of mesotheliomas occurs in the construction trades than in production. Far larger quantities of chrysotile than of other types of asbestos were used in most construction applications. Epidemiological studies that contribute to our understanding of the health effects of chrysotile conducted to date and reviewed in this monograph have been on populations mainly in the mining or manufacturing sectors and not in construction or other user industries. This should be borne in mind when considering potential risks associated with exposure to chrysotile." [EHC 203, p 137].

"Few data on concentrations of fibres associated with the installation and use of chrysotile-containing products were available to the Task Group, although this is easily the most likely place for workers to be exposed." [EHC 203, p 138].

"There is potential for widespread exposure of maintenance personnel to mixed asbestos fibre types due to the large quantities of friable asbestos materials still in place. In buildings where there are control plans, personal exposure of building maintenance personnel in the USA, expressed as 8-h time-weighted averages, was between 0.002 and 0.02 f/ml. These values are the same order of magnitude as exposures reported during telecommunication switch work (0.009 f/ml) and above-ceiling work (0.037 f/ml), although higher concentrations have been reported in utility space work (0.5 f/ml). Concentrations may be considerably higher where control plans have not been introduced. For example, in one case, short-term episodic concentrations ranged from 1.6 f/ml during sweeping to 15.5 f/ml during cleaning (dusting off) of library books in a building with a very friable chrysotile-containing surface formulation. Most other values, presented as 8-h timed-weighted averages, are about two order of magnitude less." [EHC 203, p 139].

5.178 These points are also borne out by the 1999 Report for the Australian Mesothelioma Register [AMR 99], where the broad spread of prior occupations among mesothelioma victims is plain. For example, the number of mesotheliomas from the former Wittenoom blue asbestos industry (189 mesotheliomas related to a single exposure only; 25 additional mesotheliomas as a consequence of multiple exposures; total = 214) is less than the numbers of mesotheliomas as a consequence of asbestos exposure in different occupations (e.g. carpenters/joiners: 187 mesotheliomas from a single exposure; 33 additional mesotheliomas due to multiple exposures; total = 220; for builders/builders' labourers the corresponding numbers are 150 + 27 = 177). In other words, mesotheliomas among the former Wittenoom cohort constitute a relatively small number (214) in comparison to the aggregate numbers of mesotheliomas from asbestos exposures in other occupations (2585 – 214 = 2371 other asbestos-associated mesotheliomas; no exposure data for 717 cases, and no apparent exposure for 443; aggregate total = 3745).

5.179 NICNAS 99 makes the same point (p 59):

"Occupation/industry classification of the mesothelioma cases on the register are based on the Australian Bureau of Statistics 'Industry and Occupation Codes'. The percentage of overall cases of mesothelioma (January 1986 to March 1995) according to exposure category are: repair and maintenance of asbestos material (13%), shipbuilding (3%), asbestos cement production (4%), railways (3%), power stations (3%), boilermaking (3%), mining (Wittenoom) (5%), wharf labour (2%), para-occupational, hobby, environmental (4%) carpentry (4%), building (6%) navy (3%), plumbing (2%) brake linings (manufacture/repair) (2%) and combinations of the above (multiple) (12%) (Leigh *et al*., 1997). Leigh (1994) reported that the pattern of exposure is shifting away from the older traditional industries towards product, domestic and environmental exposure. An analysis of 16 years data in 1996 by Yeung *et al*. (1997) showed more cases (on a number of cases basis) in more recent years in the asbestos user industries and from occupations such as plumbers, carpenters, machinists and car mechanics."

5.180 Similar patterns of exposure — and resultant diseases (lung cancer; mesothelioma) — have been recorded in the United Kingdom (EHC 203, pp 123-124):

> "Based on analyses of mortality of workers with mixed exposures to chrysotile and amphiboles in the United Kingdom, by far the greatest proportion of mesotheliomas occurs in users of asbestos-containing products, rather than those involved in their production. ...
>
> 1. Asbestos exposure caused approximately equal numbers of excess deaths from lung cancer (749 observed, 549 expected) and mesothelioma (183 deaths) within the occupations covered by the 1969 and 1984 Regulations ...
>
> 2. Only a few (5%) of British mesothelioma deaths were among workers in regulated occupations (Peto *et al*., 1995). The majority of deaths occurred in unregulated occupations in which asbestos-containing products are used, particularly in the construction industry. The risk was particularly high among electricians plumbers and carpenters as well as among building workers."

5.181 As shown by the literature cited in this discussion, it is my perception that there is broad agreement among experts on these patterns of exposure.

Dr. Infante:

5.182 The relative exposure categorization of the six job situations mentioned in the question depends on the nature of controls being used in each situation. In general, exposures are more easily controlled in manufacturing and more difficult to control in construction, maintenance, repair, demolition and disposal activities. Today, exposures would be more easily controlled in mining and milling because of awareness of the hazard and the clear identification of the operations as sources of asbestos exposure. Quite often workers involved in maintenance, repair and handyman type activities do not know whether asbestos is present or not. In the absence of such knowledge, workers usually do little, or nothing to protect themselves from exposures to asbestos in these situations. As a result, workers involved in these activities are most likely to be the most heavily exposed in the occupational setting today. These types of activities often result in asbestos being carried home on the workers' clothing. A typical scenario that comes to mind is a situation whereby a worker is in a crawl space and encounters asbestos insulation. There is no active supervision in this situation and the asbestos most likely is not labelled. Thus, the worker cuts through the insulation to get to the area needing to be repaired without knowledge of the hazard and without having the appropriate personal protective equipment. In the latter scenario, even when workers do wear respirators, they are often dust masks, which do not provide a proper face seal and the filter medium is not adequate i.e., HEPA filters are not part of the filtration material on these masks. In the repair trades particularly, it is common practice for workers to use dust masks that do not provide HEPA filtration. As a result, the respirator is inadequate for filtering out the fibres of dimensions that are thought to lead to cancer and other asbestos related diseases. Furthermore, even in situations where the appropriate respirators may be worn, comprehensive respiratory fit-testing programmes may not be included as part of the industrial hygiene programme and as a result, the respirators leak because of the inability to achieve a proper face seal. In situations where workers may be drilling, sawing, crushing, or sanding asbestos cement products the only appropriate respirator may be a supplied air respirator, but it may not be used because it is too cumbersome for the job situation. In my opinion, scenarios (v) and (vi) are usually the most dangerous in current times because the workers are not aware of the presence of asbestos and they are more likely not to have received training and education about the hazards of asbestos exposure.

5.183 The risk of exposure should be considered not only by level of exposure, but also by the extent of the populations exposed to chrysotile asbestos. The large number of mesotheliomas associated with secondary and tertiary users of chrysotile asbestos (maintenance workers, electricians, bystanders, etc.) is a reflection of the large number of individuals in the population exposed in these

situations. Thus, in terms of the risk of disease from chrysotile exposure, one must consider not only the intensity of exposure in the various work situations, but also the extent of the population exposed. One study (Begin *et al.*, 1992) reports that 33 per cent of mesothelioma cases identified among maintenance workers, electricians, bystanders, etc. were the result of exposure for less than five years, and that the incidence of these occasionally exposed cases was increasing more rapidly than in the primary industries (mines and mills), or in the secondary industries (manufacturing, daily handling of asbestos).

Dr. Musk:

5.184 The term "risk of exposure" is taken to mean who is most likely to receive the most exposure and therefore be at the greatest risk of developing asbestos-related disease. This would depend on the nature of the industry in the locality and the type of asbestos being produced or used or otherwise encountered. Those workers likely to receive the most exposure would be those in industries where regulations are most permissive or compliance with them is poorest from absence of supervision or means of personal protection. It would also depend on the conditions of work such as indoor versus outdoor etc. Canada's "argument" could be settled by monitoring of exposure! The "arguments" do not seem to be incompatible.

1.(b) Should we consider that the risk to human health associated with the various uses of chrysotile throughout its life-cycle is a workplace issue or does this risk affect a larger part of the population?

Dr. de Klerk:

5.185 The risk of disease from chrysotile affects everyone. The risk of disease depends on intensity of exposure, the duration of exposure and the time since exposure. The population who do not work with asbestos will still come into contact with it, albeit at a much lower intensity, however this population is much larger and hence the burden of disease may be greater. There are numerous examples of asbestos-related disease arising in people living in the vicinity of asbestos works or living with asbestos workers.

Dr. Henderson:

5.186 From my perspective, this is overwhelmingly a workplace issue (e.g. construction workers). The risk of cancer for the larger general population from exposure to asbestos in place has been discussed in an earlier part of this report (see above section C.1.(e)). Please see also my answer to the preceding question.

5.187 For example, asbestos-cement roofs are common in Germany where corrosion by acid rain represents a potential problem. Measurements carried out by Spurny *et al.* [221-224] on airborne asbestos fibre concentrations in the vicinity of such buildings consistently reveal levels in the order of 0.0002-0.0012 f/ml, in comparison to fibre concentration in other urban environments that range up to 0.1 f/ml (but generally = 0.001 f/ml).

5.188 Measurements have also been made on airborne fibre levels related to asbestos-cement roofing in schools in Western Australia [128] , with only one asbestos fibre detected in each of two schools (air monitoring at 9 sites over 720 hours). Based on the findings, it was estimated that airborne fibre concentrations would be unlikely to exceed 0.002 f/ml and were likely to be < 0.0002 f/ml. These levels were considered to represent a negligible risk to health; the Western Australia Advisory Committee on Hazardous Substances that carried out this investigation considered that a greater risk to health would arise from: (i) unskilled attempts to clean up the

asbestos-cement roofs before application of protective coating; and (ii) trauma to the workers — e.g. falling from or through the roofs.

5.189 It is my perception that there is little or no dispute among experts on this issue.

Dr. Infante:

5.190 In general, workers are at relatively greater risk of exposure to chrysotile and disease, particularly those involved in maintenance, modification, demolition, repair and disposal activities as compared to those exposed in non-occupational situations. A large number of people from the general population, however, also will be exposed to chrysotile and elevated risk of disease when they engage in home repairs that involve manipulating or disturbing asbestos-containing products. (The latter individuals usually have little or no education about the hazards of asbestos, nor of the most appropriate means to handle it with the least amount of exposure.) These types of operations will also create some standby exposures (Ascoli *et al*. 1996). If appropriate controls are not used when handling asbestos insulation in buildings, the building can become contaminated and the occupants will become exposed. Therefore, the major problem with asbestos exposure is related to occupational situations though a much larger population is exposed beyond the occupational setting to relatively lower levels. Reports of cases of mesothelioma among non-occupationally exposed individuals document non-occupational exposures to asbestos causing disease. Family members of workers involved in the asbestos cement industry (Magnani *et al*. 1993) as well as children of miners and millers (McDonald and McDonald 1980) have been diagnosed with mesothelioma.

Dr. Musk:

5.191 It is my opinion that the risks resulting from exposure affects all exposed people and depends on the cumulative level of exposure. It is also my opinion that there is not an exposure threshold below which there is no risk. The risks to people not occupationally exposed to asbestos are likely to be much less than the risks to those with occupational exposure because the degree of exposure is likely to be less (though not necessarily always so). However, while the individual risks may be much less the total burden of disease in the community may not be because it is likely that there are many more people experiencing these risks (albeit lower). For example the burden of disease in the residents of the town of Wittenoom, Western Australia has been significant albeit less than that of the workers. The WA Mesothelioma Registry contains subjects whose only exposure was from neighbourhood industries. Similar cases have been documented in the Quebec areas.

1.(c) Can chrysotile-cement products (for instance in buildings) release fibres, through weathering, corrosion or general degradation, thus presenting a possible risk to human health? Can you quantify this risk?

Dr. de Klerk:

5.192 There is good evidence that both wind and rain cause the release of fibres even from new asbestos cement sheeting. Other possibilities are fires and unwanted demolition. It is hard to quantify the risk which again depends on intensity and duration, but measurements have been made in the vicinity of such buildings which are detectable but low.

Dr. Henderson:

5.193 Please see the preceding answer, and section C.1.1(e). Quantitation of the risk is based on backward extrapolation according to the linear no-threshold model because there are no observational data on the dose-response effects from low-level exposure to chrysotile, and the estimates are,

therefore, open to question and dispute, but the risks to health from very low-level environmental exposure appear to be minuscule or negligible.

Dr. Infante:

5.194 Yes, weathered and corroded asbestos cement products are capable of releasing chrysotile fibres into the environment, and most of the fibre is transported by rainwater though some will be released into the ambient air in low concentrations. One study indicates that chrysotile exposure in such circumstances will generally be less than 1,000 fibres longer than 5 microns per cubic meter of air. The fibres released were shown to have the same carcinogenic potency as "standard" chrysotile fibres (Spurny, 1989). Asbestos fibres also will be released into water from cement water pipes. I have not seen any estimates of risk from this type of asbestos exposure. Although the relative risk of disease is considerably less than that from occupational exposures, the population at risk is considerably larger.

Dr. Musk:

5.195 I understand that asbestos-cement products do release fibres as they weather. Release of fibres occurs from both old and new products. Asbestos fibres can also be released when asbestos-cement products are involved in fires. Quantitative estimates of the risks are theoretically possible as airborne concentrations can be measured and dose-response relationships are known.

1.(d) Can interventions on chrysotile-cement and other high-density chrysotile products release fibres, thus presenting a possible risk to the health of the individual making such interventions or to the public in general? Can you quantify this risk?

Dr. de Klerk:

5.196 It is during interventions such as drilling, sawing, sanding, moving in stacks, loading onto transport etc, that concentrations of fibres are greatest, both for the operators and bystanders. The concentrations associated with such operations have been extensively tabulated in the literature. Exposure response relationships can be used to estimate the risk for any combinations of intensity, duration and time after exposure, as shown in the Table below.

5.197 Lifetime risks (to age 85 years) of mesothelioma after exposure to chrysotile, assuming 0.1 f/ml for 10 years from age 20 with competing causes of death at 1992 Western Australian death rates.

Assumptions	Expected cases per million lifetimes
Health Effects Institute equation	724
Wittenoom crocidolite equation 1/12th potency	210
Wittenoom crocidolite equation , 1/80th potency	32
Background risk (Peto study of "unexposed" Los Angeles population)	112

Dr. Henderson:

5.198 My answer to the first question is YES. Operations such as drilling or sawing asbestos-cement products release fibres and produce elevated airborne fibre concentrations. (i) asbestos-cement sheets can release respirable fibres in the absence of manipulation, even when new (up to 0.001 f/ml; for references, see de Klerk and Armstrong [135]); (ii) a 1938 report in

New South Wales indicated that cutting asbestos-cement products with a power saw could generate 4-5 million particles/cubic foot (roughly equivalent to 12-15 f/ml); cutting with hand saws produced lower concentrations; (iii) as shown in the following Table 11, Sturm *et al.* [5, 7] reported measurable fibre concentrations from various operations on asbestos-containing materials, including asbestos-cement in the former East Germany, as measured by occupational inspectors.

TABLE 11: ASBESTOS FIBRE CONCENTRATIONS AT WORKPLACES, WITHOUT SUCTION DEVICES, DETERMINED BY KONIMETRY (FROM UNPUBLISHED REPORTS PREPARED BY OCCUPATIONAL INSPECTORATES)

Type of Work	Fibre Concentration (f/ml)
Scratching and crushing of asbestos-cement	0.03 to 0.3
Abrasive cutting of asbestos-cement without dust removal by suction	0.3 to 10.0 approx.
Drilling asbestos-cement without dust removal by suction	0.5 to 3.4
Machining of brake linings	0.1 to 13.0
Replacement of gaskets	0.02 to 0.5
Punching of gaskets (rubber asbestos)	0.02 to 1.9
Use of asbestos gloves	0.02 to 0.6
Replacement of clearing layers	0.06 to 0.5
Use of talcum for powdering gloves	0.6 to 20.0
Level limit value (over a whole working day)	1.0

5.199 In 1993, Kumagai *et al.* [4] in Japan reported on dust levels generated by repair work on asbestos-cement pipes, including use of a high-speed disc cutter both inside holes dug in the ground to gain access to the pipes and outside the holes. The concentration of asbestos fibres > 5 µm in length ranged from 48-170 f/ml inside the hole (average = 92 f/ml) and ranged from 1.7-15 f/ml outside the hole. The Abstract for this paper follows:

> "Asbestos cement pipes (ACPs) containing 15 to 20% chrysotile or crocidolite have been used for underground conduits. Even today 16.2% of all conduits in Japan are ACPs, though the production of ACPs was suspended in 1985. When such a conduit is accidentally damaged the workers belonging to the Waterworks Bureau of a local government cut off the damaged conduit using a high-speed disk cutter and replace it with a new conduit. This operation develops a cloud of dust and the workers involved run the risk of asbestos exposure. It was the aim of the present study to estimate asbestos exposure levels among these workers. First, in the experiment, we established the typical working conditions and requested an experienced worker to cut an ACP using a high-speed disc cutter in a hole dug in the ground as he routinely does. The experiment was repeated three times. During a bout of each experiment, dust was sampled at several points both inside and outside the hole. Second, a self-administered questionnaire survey was conducted to obtain information from the workers regarding their working conditions in cutting ACPs. The subjects of the survey were 1,048 men belonging to conduit repair sections of the Waterworks Bureau of 119 local governments. The results obtained can be summarized as follows. (1) Each bout of cutting ACPs required about five minutes. The concentration of asbestos fibers longer than 5 microns with 3:1 aspect ratio ranged from 48 to 170 fibers/ml (92 fibers/ml on an average) inside and 1.7 to 15 fibers/ml outside the hole. The concentration inside the hole exceeded the ceiling limit (10 fibers/ml) recommended for asbestos by the Japanese Association of Industrial Health. A concentration of 92 fibers/ml is equivalent to 0.96 fibers/ml as 8-h time-weighted average. (2) The number of subjects with experience of cutting ACPs was 849 (81.0%). The average length of service in conduit repair section was 14.2 yr. Based on the information obtained from each subject regarding the average working days per yr for each decade from 1946, the cumulative days to date expended in cutting ACPs was estimated to average 235 d, that is, 17 d per yr. Only 18.1% of the subjects used a protective respiratory device."

5.200 EHC 203 also gives the following data (p 40):

> "Weiner *et al*. (1994) reported concentrations in a South African workshop in which chrysotile asbestos-cement sheets were cut into components for insulation. The sheets were cut manually, sanded and subsequently assembled. Initial sampling showed personal sample mean concentration of 1.9 f/ml for assembling, 5.7 f/ml for sweeping, 8.6 f/ml for drilling and 27.5 f/ml for sanding. After improvements and clean-up of the work environment, the concentrations were 0.5-1.7 f/ml.
>
> Nicholson (1978) reported concentrations of 0.33-1.47 f/ml in a room during and after sawing and hammering of an asbestos-cement panel."

5.201 It is my perception that there is no dispute among experts on this issue.

5.202 In relation to the second part of this question, apart from stating that there is a risk because of the generation of airborne asbestos fibres from interventions on asbestos-cement and other high-density asbestos products, it is not possible to quantify the risk in a way that would meet with universal agreement or a broad consensus, because few data are available for the risks for this type of operation on chrysotile-cement products: the risk would be related to cumulative exposure, which would vary according to the types of operation carried out, and their frequency. In addition, risk estimates would be dependent on extrapolation from the linear dose-response model that has been called into question by Canada. Therefore, I would expect disagreement among authorities on the magnitude of the risk.

5.203 Table 12 derived from NICNAS 99 gives risk estimates for lung cancer at airborne chrysotile concentrations of 0.1-1.0 f/ml, according to the National Occupational Health and Safety Commission in Australia (NOHSC) and two US occupational health and safety bodies (OSHA and NIOSH).

TABLE 12: ESTIMATED RISK OF LUNG CANCER AT VARIOUS LEVELS OF EXPOSURE TO CHRYSOTILE

Exposure(yearly average fibre/ml)	**Excess risk (per 100,000 persons exposed)**		
	NOHSC	US OSHA	US NIOSH
1	173	2880	5760
0.5	86	1440	2880
0.1	17	288	576

Excess risk = Risk coefficient x lifetime exposure (yrs) x average exposure level (f/ml) background risk.*
*A cumulative background risk for lung cancer in the male population was used in these calculations (i.e. 7200/100,000 assuming mixed smoking habits).

5.204 However, NICNAS 99 goes on to discuss uncertainties concerning these risk estimates:

> "There are several other reasons why there is considerable uncertainty regarding these risk estimates, which include:
>
> 1. Past occupational exposures have generally involved exposure to a mixture of asbestos fibres. As it appears likely that different types of asbestos have different degrees of hazard, it is difficult to determine the risk attributable to chrysotile per se. In addition, commercial chrysotile often has low levels of tremolite contamination.
>
> 2. Fibre size, such as difference in fibre size between different chrysotile industries, probably influences the degree of hazard and/or potency.
>
> 3. There is a long latency between exposure to asbestos and development of lung cancer. Hence, it is not possible to state definitively what fibre type and level of exposure caused the disease. Consequently, risk estimates are related more to duration of employment rather than intensity of exposure.

4. A linear, non-threshold model may not be an appropriate model as there is some evidence suggesting that lung cancer due to chrysotile exposure may have a threshold for effect.

5. Past exposure estimates (both quantitative and qualitative) are subject to considerable error. For example, conversion of historical results in mpcf units to fibres/mL has inherent uncertainties.

6. There is a high background level of lung cancer in the general population due to smoking. Cases of lung cancer attributable to asbestos cannot be distinguished from those due to smoking. Attribution can only be assessed in terms of excess of lung cancers above a control population, hence the choice of control population is critical.

7. The identification of the disease is dependent on medical diagnosis, however autopsies are not always conducted.

The impact of some of these uncertainties can be accounted for to some extent. For example, it is considered that (1) and (2) are largely accounted for by basing risk estimates on epidemiological studies where exposure was only to chrysotile in the most relevant industry. For the remainder of the above uncertainties it is unclear what influence they have on the risk estimates and how they should be accounted for. For example, recently there has been some debate in the literature as to whether a threshold or non-threshold model should be used when predicting risk due to chrysotile exposure. Meldrum (1996) states that based on balance of toxicological evidence, the linear no-threshold model for chrysotile-induced lung cancer may not be appropriate. ... Epidemiological data alone are not able to clearly distinguish between the possibility of a threshold or a non-threshold model due to the relatively high background rate of lung cancer in the human population. There is at present no consensus with respect to a threshold level of exposure for chrysotile below which there is no risk of disease" [pp 70-71].

5.205 Table 13 gives an estimate of lifetime mesothelioma risk from exposure to low levels of chrysotile (1.0 f/ml and 0.1 f/ml), based on dose-response data for the Wittenoom cohort, and assuming lower potencies for chrysotile than crocidolite (i.e. 1/12th, 1/30th and 1/80th).[20]

TABLE 13: ESTIMATES OF LIKELY MESOTHELIOMAS RELATED TO CHRYSOTILE INHALATION AT AIRBORNE CONCENTRATIONS OF 1.0 AND 0.1 F/ML, ASSUMING A CARCINOGENIC POTENCY 1/12TH, 1/30TH OR 1/80TH THAT OF CROCIDOLITE

	Numbers to age 85 Mesotheliomas/million persons		
Airborne fibre concentration; Potency	**Duration of exposure* (years)**		
	1 yr	**10 yrs**	**40 yrs**
1.0 f/ml -1/12th	282	2101	3530
1.0 f/ml - 1/30th	113	840	1412
0.1 f/ml - 1/12th	28	210	353
0.1 f/ml - 1/30th	11	84	141
0.1 f/ml - 1/80th	4	32	53

*Starting at age 20 yrs.

[20]Estimates calculated at my request by Dr. N.H de Klerk.

Dr. Infante:

5.206 Interventions on chrysotile cement products can result in extremely high atmospheric fibre concentrations (Rodelsperger *et al.*, 1980) and studies of roofers have demonstrated asbestosis from such exposures (Stauder *et al.*, 1982). A study of workers involved in inside finishing work with concrete asbestos containing 30 per cent chrysotile asbestos has also shown that airflow obstruction among workers can be caused by such exposure (Harless *et al.* 1978). Open-air cutting such as that involved in roofing and inside finishing operations also will result in exposure to other workers not directly involved in the manipulation of the asbestos, e.g., standby exposure. Such manipulation of asbestos cement will also expose the general population.

5.207 A study of 404 roofers with long term exposure to cement dust indicated that 14 per cent had significantly increased small irregular opacities with profusion in 13 per cent (Stauder *et al.*, 1982). The prevalence of these abnormalities was significantly greater than that observed in the control group. The study by Harless *et al.* (1978) indicated that approximately 50 per cent of workers exposed for about six months to dust from asbestos cement developed airflow obstruction. The risk of developing lung pathology per unit of asbestos fibre exposure cannot be determined from these studies because of lack of exposure data. The studies do indicate, however, that uncontrolled manipulation of chrysotile cement products can result in a high rate of lung pathology. Lung function can be adversely affected as a result of exposure over a very short period of time.

5.208 A large number of reports indicate mesothelioma related to car mechanics involved in brake repair. General population exposures from such work would be minimal except for those situations where individuals would engage in their own brake repair work.

Dr. Musk:

5.209 Interventions on asbestos-cement products can release fibres therefore a risk of disease exists as in 1(c).

1.(e) Can occasional interventions on high-density chrysotile products, either in occupational circumstances (such as electricians, plumbers, repairers, insulation workers, etc.) or by private individuals ("handyman" type) release fibres, thus presenting a possible risk to the individual making such interventions or to the public in general? Can you quantify this risk?

Dr. de Klerk:

5.210 Yes of course, see (c) and (d).

Dr. Henderson:

5.211 To answer the second question first, I am unable to quantify potential risk, because there are no systematic observational data available for this type of work, to the best of my knowledge (but please see Tables 12 and 13 above in my answer to Question 1(d)

5.212 The first part of the question has been covered in the preceding answer, with the observation that occasional interventions of this type would predictably produce low cumulative exposures, with a lower risk, for the reasons discussed earlier. Please also refer to AMR 99, for data on mesotheliomas among electricians, carpenters, plumbers, insulation workers and so forth (it is acknowledged that most if not all these mesotheliomas are a consequence of exposure to asbestos-containing materials that included a mixture of asbestos types, including chrysotile and one or more of the amphiboles); in drawing attention to AMR 99, my purpose is simply to use mesothelioma rates as a reflection of past exposures and hence evidence that airborne fibre concentrations were produced by these types of

operation, without regard to the fibre types. My own cases of mesothelioma also include a number of individuals whose only exposure to asbestos took the form of maintenance work and renovations carried out on the patient's home, where there were asbestos-cement building materials. Again, in drawing attention to this type of background, it is not my intention to address fibre type, but simply to indicate that mesothelioma as an outcome of this type of exposure indicates that elevated concentrations of respirable airborne fibres were produced.

5.213 EHC 203 gives the following account (pp 122-123):

> "Although the odds ratio for lung cancer associated with exposure to "asbestos" has been estimated in many case-control studies, the studies have not been in general able to distinguish between chrysotile and amphibole exposure, and are therefore less informative for the present evaluation ... In a multisite case-control study from Montreal, Canada, however, exposures to chrysotile and to amphiboles were separated, although exposure to amphiboles was not controlled for in the analysis for exposure to chrysotile (Siemiatycki, 1991). In this study, the occupational history of male cases (age 35-70) of cancer at 20 sites and of 533 population controls was evaluated by a team of industrial hygienists and chemists to assess exposure to 293 agents. Overall, the lifetime prevalence of exposure to chrysotile was 17% and that of exposure to amphiboles 6%. The main occupations involving exposure to chrysotile that were considered were motor vehicle mechanics, welders and flame cutters, and stationary engineers. When lung cancer cases (N = 857) were compared with cases of all other types of cancer, the odds ratio (OR) of any exposure to chrysotile was 1.2 (90% CI = 1.0-1.5; 175 exposed cases), and that of 10 or more years of exposure with at least 5 years of latency ('substantial exposure') was 1.9 (90% CI = 1.1-3.2; 30 exposed cases). Corresponding ORs of exposure to amphiboles were 1.0 and 0.9. The OR of exposure to chrysotile was higher for oat cell carcinoma than for other types of lung cancer. Twelve cases of mesothelioma were included in this study. The OR of any exposure to chrysotile was 4.4 (90% CI = 1.6-11.9; 5 exposed cases) and that of substantial exposure was 14.6 (90% CI = 3.5-60.5; 2 cases). Corresponding ORs of exposure to amphiboles were 7.2 (90% CI = 2.6-19.9; 4 cases) and 51.6 (90% CI = 12.3-99.9; 2 cases). "

5.214 Please see also Tables 5, 9, 10, 11, 12, 13, in EHC 203.

5.215 It is my perception that there is no dispute among experts that such interventions release fibres; disagreement is likely over the magnitude of the risk.

Dr. Infante:

5.216 The worker making the intervention would be the most highly exposed and presented with the greatest risk of asbestos related diseases. The extent of exposure to the worker as well as to those in the surrounding area would depend on the nature of the intervention, e.g., the circumstances under which the chrysotile asbestos product is manipulated in terms of work practices, the controls, or lack of controls in place and the type of personal protective equipment provided to the worker. While data on fibre exposure levels in these situations are sparse, data on mesothelioma indicates association with workers who have jobs that result in occasional interventions to asbestos products. Because these exposures are not routine and the hazard often goes unrecognized, these operations are unlikely to be well controlled, i.e., they are not anticipated so proper training and education about these types of exposures is often lacking.

5.217 It is difficult to quantify this risk because atmospheric measurements are usually not made during these interventions. However, the identification of cases of mesothelioma associated with these interventions in the literature indicate that they are perhaps the most detrimental to human health. Mesothelioma has been identified from these exposure situations because it is a marker cancer related to asbestos exposure. What goes unidentified and unmeasured from these situations is the much larger burden of disease and death from pneumoconiosis and lung cancer. The attributable burden from these latter diseases will be much greater than that from mesothelioma, but they are not usually recognized because lung cancer has a high background rate in the general population and

asbestosis may be diagnosed as another type of pneumoconiosis unrelated to asbestos exposure. Mesothelioma has also been documented among the wives of construction workers, indicating that the family member portion of the public in general is also at risk. These latter cases of mesothelioma are most likely the result of carry-home exposure from contaminated clothing.

5.218 If one considers that the handyman types of exposures are considered as exposures to the general public, then this segment of the general population would also be at an elevated risk of developing asbestos related diseases. Exposures to family members that might result from interventions by home owners would depend on the nature and location of the removal or manipulation of the asbestos. The general public is also exposed through manipulation of asbestos in residential buildings that is not carried out with appropriate controls in place and by carry home exposures from contaminated work clothing.

Dr. Musk:

5.219 Occasional interventions on asbestos-cement products by anyone can release airborne fibres, therefore there is some risk as in Question 1(c).

1.(f) Are chrysotile fibres from chrysotile-cement dust released during interventions (cutting, sawing, etc.) on chrysotile-cement products as dangerous as pure chrysotile fibres? Is the physical and chemical composition of asbestos-cement dust different from pure asbestos dust?

Dr. de Klerk:

5.220 Risk from fibres depends on size, shape and durability (and their quantity). Asbestos cement contains about 10-20 per cent asbestos, so that the dust concentration is going to be less than if the sheets were pure asbestos. However, cement does not form fibres, so that any airborne fibre measurements made would only reflect the asbestos concentration in the air.

Dr. Henderson:

5.221 To answer the second part first, the physico-chemical composition of asbestos-cement dust does differ from pure asbestos dust, because the asbestos in asbestos-cement products is diluted by the cement (asbestos = 10-15 per cent by weight); this being so, one expects the asbestos fibres to be diluted by cement dust, in comparison to equivalent operations on pure asbestos materials.

5.222 To return to the first part of this question: the claim may be made that chrysotile fibres released from asbestos-cement products by high-speed cutting are altered physically or chemically, with a predominance of short-length fibres not implicated in carcinogenesis. For example, in Canada's first oral submission, the following comments are made:

> "The European Community has also advanced the thesis that the ban is necessary because France has no control over trade persons or the 'handyman' who will cut into chrysotile-cement and, in so doing, free some of the chrysotile that was locked into it. Canada is puzzled by France's assertion that la République Française is unable to regulate its handymen. In any event, there are three technical reasons why France's concern is misplaced.
>
> First, this thesis is based on the misconception that cutting high-density locked-in non-friable asbestos-containing materials releases substantial amounts of chrysotile. In fact, even if improper tools such as high-speed saws are used to cut chrysotile-cement, the dust released from such an operation contains only a very small amount of pure, respirable-size chrysotile fibres, if any at all.
>
> Second, science tells us that most of the chrysotile fibres released during high-speed cutting have been chemically altered: the resulting entity is chemically and structurally different, and has a biological

> potential to induce harmful effects, which is different, and less than amphiboles. Similarly, the dust that results from abrading chrysotile-containing resin or plastic reinforced products contains very small amounts of chrysotile fibres. The same is also true of the dust that comes from wear and abrasion of friction materials: analysis of brake shoes shows that almost all of the chrysotile fraction of the finished product is found to be transformed into a totally different, biologically-inactive material called forsterite ...."

5.223 The first paragraph of the Canadian statement is dealt with in later discussion in this report (my answers to Question 5). For the second and third paragraphs of this statement, one can state that in other situations, only a small fraction of airborne asbestos fibres are of respirable size: as one example, about 0.67 per cent of airborne asbestos fibres within indoor air of buildings were longer than 5 µm in length. However, in the Japanese study reported by Kumagai *et al.* [4] on cutting asbestos-cement pipes with a high-speed disc cutter, where airborne fibre concentrations within the hole used to gain access to the pipes averaged 92 fibres/ml (range 48-170 fibres/ml), the study dealt with fibres longer than 5 µm (i.e. dimensions in the range for which carcinogenicity has been reported). Please see also Table 11 in EHC 203 where Rödelsperger *et al.* recorded airborne fibre concentrations of 4-5 f/ml and 5-10 f/ml for fibres longer than 5 µm, from blowing off and grinding brake blocks, including truck brakes. (Please see also Table 11 and my answer to Question 2.)

5.224 Clearly, there is disagreement between the parties to the dispute and their respective experts over the issue of whether chrysotile fibres released from high-density products are dangerous. For the reasons outlined above and in later discussion, it is my perception that at least a small proportion of the fibres has dimensions that are associated with carcinogenicity.

Dr. Infante:

5.225 As long as the interventions result in the release of chrysotile fibres, the exposures should be considered as dangerous as pure chrysotile fibres because respirable-sized asbestos fibres will be released. The study by Spurny (1989) indicates that the fibres released from weathered and corroded chrysotile asbestos cement products have the same carcinogenic potency as "standard" chrysotile fibres. Although fibres released by weathering may be somewhat different from fibres released from cutting or drilling asbestos cement products, the former fibres show a potency similar to that of pure chrysotile fibres. Moreover, because of the potential for cleavage during interventions on asbestos cement products, the dust resulting from cutting, drilling, etc. on asbestos cement may actually contain a greater portion of the asbestos fibres being thinner and more respirable than those that were initially mixed into the cement during the manufacturing process. Therefore, the fibres released from cement during interventions should be considered at least as dangerous as "pure chrysotile fibres." I can find no data to support an opinion that fibres released from interventions on chrysotile asbestos cement products would be less carcinogenic, or less dangerous. Further, asbestos related pathology has resulted from such situations.

5.226 The asbestos cement dust would be somewhat different in physical and chemical composition from pure chrysotile asbestos because the cement dust would contain respirable asbestos fibres, crystalline silica plus other substances added to the cement.

Dr. Musk:

5.227 It is my opinion that in general airborne fibres released from asbestos-cement products pose a risk. This may differ from other sources of chrysotile depending on the characteristics of the fibres. The area of fibre characteristics and their relationship to different sources is not within my area of expertise.

1.(g) What is the risk to human health associated with demolition and removal of high-density chrysotile products, such as chrysotile-cement products? Can you quantify this risk?

Dr. de Klerk:

5.228 See my answers to Questions 1(c) and (d).

Dr. Henderson:

5.229 I am not aware of any studies that have specifically focussed on either of these situations: therefore, no firm data are available, but one would expect the biohazards to be related to cumulative doses of respirable fibres (i.e. airborne fibre concentrations and the frequency of exposure from these types of work). This being so, one would expect the risks to be equivalent to other operations of like frequency that generated similar airborne fibre levels (Tables 12 and 13).

Dr. Infante:

5.230 Exposure to high density chrysotile products through demolition carries with it the potential risk of lung cancer, asbestosis and mesothelioma. Testimony presented at the OSHA hearings related to its Final Asbestos Standard that was promulgated in 1994 indicated that removal of intact chrysotile asbestos "transite" panels that were held in place by screws can result in airborne fibre concentrations that exceeded 1 f/cc. In this situation, the exposed surfaces were wet prior to removal and the operation was done within a negative pressure enclosure. Many transite panels used in interior wall construction consist of rough inner surfaces from which asbestos fibre is readily released into the air. Other testimony (OSHA, 1994) presented evidence that transite panels can be removed in a manner that results in exposure well below 0.1 f/cc when appropriate work practices are followed. Because of concern for the potential release of asbestos fibres into the air from such demolition, the OSHA standard requires that a "competent person" supervise such activities, e.g., make an assessment and determine that the type of controls being used are appropriate for the removal situation and that the required work practices are being followed. Therefore, the extent of the risk during demolition of chrysotile cement products depends upon the compliance with mandated requirements. (See my answer to Question 5(c) regarding compliance with procedures to reduce risk of disease from asbestos exposure.)

Dr. Musk:

5.231 In so much as demolition activities may result in airborne fibres there is a risk (as above).

1.(h) What is the risk to human health associated with high-density chrysotile wastes, such as chrysotile-cement waste? Can you quantify this risk?

Dr. de Klerk:

5.232 This depends on how the waste is treated and stored, depending of course on the chance of any fibres becoming airborne and hence respirable. Otherwise, see my answers to Questions 1(c) and (d).

Dr. Henderson:

5.233 See my response to Question 1(g).

Dr. Infante:

5.234 I have not researched this issue, but I am inclined to believe that there would not be much potential for fibre exposure from the handling of such waste unless a person at a waste site was hauling asbestos cement and not aware of the product he/she was moving.

Dr. Musk:

5.235 The risk posed by waste products will also be dependent on the chances of fibres becoming airborne as above.

1.(i) Can high-density chrysotile products wastes, such as chrysotile-cement wastes, be dealt with so as to eliminate risks to human health?

Dr. de Klerk:

5.236 They can, by following approved methods for disposal which ensure that fibres are sealed from airborne release. There is of course the chance that subsequent work (for example, waste removal) may disturb the waste and release fibres.

Dr. Henderson:

5.237 In theory, YES — once the asbestos-cement or other high-density product has been removed from its in-place location (though few data are available on exposure levels produced by the actual removal). For example, in Australia, imported chrysotile is delivered to production facilities in sealed plastic bags, so that the same procedure for bagging or encapsulation of high-density wastes should also be applicable, and should prevent release of asbestos fibres once the encapsulation or bagging exercise is complete, unless the bags are ruptured for one reason or another.

5.238 According to NICNAS 99 (p 74), in Australia:

> "Waste chrysotile, the polyethylene bags in which it is supplied, and chrysotile containing materials from the manufacturing process, are disposed to landfill by licensed disposal contractors. As chrysotile fibres are unlikely to be mobile in the soil or water table, landfill is not inappropriate from a public health perspective."

Dr. Infante:

5.239 I have not researched this issue.

Dr. Musk:

5.240 These risks may be eliminated if the fibres could be successfully sealed so that they cannot become airborne.

Question 2:

What is the risk to human health associated with other current applications of chrysotile asbestos (in particular, friction materials and textiles)? In occupational circumstances? In non-occupational circumstances?

Dr. de Klerk:

5.241 While the industries themselves may be well regulated, controlled and compliant with standards, the major problem again could occur in "downstream" users: boilermakers, plumbers, brake mechanics etc. Fibres released from friction products have a higher proportion of shorter fibres than those from textiles, which release the highest proportion of longer fibres.

Dr. Henderson:

(i) Friction Products (e.g. brake linings)

5.242 Automotive mechanics and garage workers constitute a large population of workers potentially exposed to chrysotile derived from brake linings. For example, brake blocks and linings used in Australia have contained only Canadian chrysotile for many years, and the materials are either imported as pre-formed brake blocks and linings, or chrysotile is imported into Australia for subsequent manufacture of these products. It has been estimated that this group of mechanics amounts to at least 900,000 workers in the US, and the figure may be even higher if one adds in all those who have ever worked in the automotive repair industry but then moved into other employments, and those who have retired.

5.243 For Australia, the number of persons employed as mechanics in 1991 amounted to 85,155 (84,293 males); for 1996, the corresponding figures are 83,647 (82,827 males), out of a total population of 16,852,256 in 1991 (8,363,677 males); for 1996, the total population was 17,892,423 (8,849,224 males). These figures for Australia include all mechanics, including automotive, brake and engine mechanics, together with supervisors and apprentices; the figures for 1996 also include mechanics' assistants (not included for the 1991 figures).[21] Taking into account the fact that the Australian population is less than $1/10^{th}$ that of the United States population, these statistics appear to be roughly comparable.

5.244 The literature contains anecdotal reports of malignant mesothelioma among automotive and brake mechanics. However, the question that arises is whether these anecdotal reports are explicable as the chance occurrence of spontaneous or background mesotheliomas among a large population of mechanics, or whether this group of workers has sustained other significant exposures to asbestos, including one or more amphiboles. In other words, the question is whether there is a general increase in the incidence of mesothelioma among automotive and brake mechanics with no other exposures to asbestos.

5.245 Brake repair workers are potentially exposed to asbestos during a number of procedures, which include removal of dust from bakes by air hoses, and a variety of other manipulations that include bevelling, grinding and drilling. Clearance of dust from brakes by use of an air hose can create a cloud of visible dust, and airborne fibre concentrations of 2.0 to 29.4 f/ml have been recorded in the immediate vicinity [225, 226]. Please see also Table 11 in EHC 203 (pp 42-43).

5.246 In North America, chrysotile has been used almost exclusively in brake linings since the 1940s; chrysotile is also the type of asbestos used in brake linings in Europe (and also Australia). As I mentioned previously, commercial chrysotile (e.g. Canadian chrysotile) contains on average small quantities of contaminant amphiboles in the form of tremolite (usually = 1%).

5.247 However, the significance of this type of potential exposure among brake repair workers is complicated by a number of factors:

[21] Statistics supplied by the Australian Bureau of Statistics on 12 October 1999.

- During moderate braking of automobiles, temperatures as high as = 500°C can be reached within the brakes, and at this temperature a proportion of the chrysotile undergoes dehydroxylation and recrystallization to form the mineral forsterite, which is not implicated in mesothelioma induction.

> "Heating of chrysotile at 700°C for an hour converts it to an amorphous, anhydrous magnesium silicate material ... Intensive dry grinding also destroys the structure of chrysotile. Analysis of wear debris from brake linings made with asbestos has shown that virtually all of the chrysotile fibre is converted to amorphous material, in association with the mineral forsterite (a recrystallization product). The conversion is explained by localized temperatures above 1000°C at the point of contact between the brake lining and the drum" ... . [EHC 203, p 14].

- Most chrysotile fibres released from brakes comprise short-length fibres < 0.4 µm in length (> 80 per cent of all chrysotile fibres from brakes). However, some fibres > 5 µm in length and even > 10 µm in length appear to survive (e.g. please see Table 11 in EHC 203). The short-length fibres appear to have only questionable or limited carcinogenicity, and this property is thought to reside primarily in fibres > 5 µm in length. In addition, limited fibre burden studies on brake repair workers have shown a low pulmonary asbestos content.

> "The fibres found in the brake wear debris are predominantly (99%) less than 0.4 µm in length … Rödelsperger *et al.*, (1986) found less than 1% of fibres longer than 5 µm." [EHC 203, p 14]

- One also needs to remember that assessment of mesothelioma risk among brake repair workers can be confounded by other occupational exposures to asbestos [227].

5.248 In a review of changing risk groups for malignant mesothelioma, Huncharek [228] gives the follow account for brake mechanics:

> "A major problem with epidemiologic studies of this workforce is the difficulty in tracing a large, non-unionized group of workers. Estimation of disease risk has been impeded by lack of quantitative data on exposure levels among individuals with long-term exposure. ...
>
> In 1976, investigators at the Mount Sinai School of Medicine studied asbestos exposure among brake repair workers in New York city. Both clinical examinations and fibre counts produced by various operations in brake maintenance workers were analyzed. Samples taken at a distance of 3-5 feet from brake drums during periods of blowing dust showed fibre concentrations of 6.6 to 29.4 fibres/ml, with a mean of 15.9 fibres/ml. In addition, ten samples of brake drum dust were analyzed by phase contrast, optical microscopic examination and transmission electron microscopic examination to determine the percentage of short fibres (i.e., 25-500 angstroms x 760 to 3750 angstroms[22]). Eighty-three percent of all chrysotile fibres were in this category, and almost 20% of the total mass of 10 samples was chrysotile (determined by electron diffraction). 'Throughout the examination by electron microscopy, attention was given to the morphology of the fibres. A majority of fibres showed little alteration in the typical chrysotile fibre'.
>
> In an additional report from Mount Sinai, Rohl *et al.* analyzed residual dust recovered from brake linings and made direct measurements of the free asbestos fiber content of 'workroom air' in areas in which brake lining maintenance and brake shoe installation occurred. Airborne asbestos dust concentrations were similar to those cited by Lorimer et al. (i.e., mean airborne fiber concentrations during compressed air blowing of brake drums ranged from 2.6 fibers/ml at a distance of 10-20 feet to 16.0 fibers/ml at 3-5 feet). Samples of brake drum dust showed that the proportion of chrysotile in this material averaged 3% to 6% (both as free fibre and as particulates in pulverized binder).

[22] 10,000 Ångstroms = 1.0 µm.

> Regarding the health effects resulting from the exposures described, Langer and McCaughey published ... a case of mesothelioma in a brake repair worker ... a 55-year-old man who had worked in the used car, tire and car repair business since the age of 19 years. He reported routinely servicing automobiles, including the replacement of brake linings. No other source of asbestos exposure was found.
>
> Analysis of lung tissue showed the presence of chrysotile fibres (no amphibole was found) confirmed by electron diffraction techniques. Ten percent of the fibers found were longer than 10 microns.[23] The authors point out that 'Controversy over the potential of chrysotile to cause mesothelioma has continued despite evidence from asbestos textile fabricators, thought to have used only chrysotile, from workers making brake pads, from chrysotile miners and millers, and from animal studies'. They also state 'The risk of malignant asbestos disease among these workers seems to be low but mortality data have yet to be thoroughly evaluated.'
>
> The most recent report of mesothelioma in a brake mechanic reviews a pleural mesothelioma occurring in a 47-year-old male automobile mechanic whose only known exposure to asbestos was from clutch and brake repair work during an 11-year period. ...
>
> Another case of mesothelioma in a brake repair worker was recently published. In this report, a 56-year-old male elevator mechanic ... reported working as an elevator mechanic for 30 years. He reported exposure primarily from elevator brake linings that he routinely cut, fitted, and removed during elevator installation and maintenance.
>
> Several recent studies from Scandinavia on this topic also deserve mention. Hansen, from the Institute of Community Medicine in Denmark, completed a historical cohort study examining the mortality of car mechanics from ischaemic heart disease and malignant neoplasms. The study cohort was identified using records of a nation-wide census carried out in Denmark in November 1970. Comparison was made with another cohort of skilled male workers who were not exposed to asbestos or 'petrochemical substances'. Of 583 observed deaths, one case of pleural mesothelioma was found.
>
> Likewise, Jarvholm and Brisman, in a 1998 report, used the Swedish death register and the census of 1960 to study the occurrence of asbestos-associated tumors in car mechanics. One hundred and eighty-seven deaths attributable to cancer were observed, whereas 154 were expected. Thirty-nine were caused by lung cancer, whereas only 23 were expected. Again, one death from pleural mesothelioma was found. ...
>
> It has been estimated that 20,000 deaths from asbestos-related cancer will occur during the next 40 years among automotive maintenance workers in the United States. With the many difficulties faced by epidemiologists studying this workforce, it is unclear how accurate this estimate will prove to be. Clearly, what is needed is better information on duration and intensity of exposure to respirable asbestos fibers in this occupational group. Additional study is needed to accurately determine the incidence of mesothelioma among members of this workforce." [pp 2704-2705].

5.249 The situation is further complicated by other reports on garage mechanics and workers involved in friction products manufacture [229, 230]. These studies have been reviewed briefly by Wong [231]: three found no increase in the RR for mesothelioma among garage mechanics (RR = 0.9, 0.65 and 1.0 respectively).

5.250 An analysis of > 13,000 workers at a UK friction products factory showed no detectable excess mortality due to lung cancer or other cancers; 13 mesotheliomas were found but 11 had known exposure to crocidolite [229, 232].

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[23]This relatively high proportion (10 per cent) in comparison to the smaller fraction of airborne fibres of the same size is presumably explicable by preferential clearance of short fibres from lung tissue, with a proportional increase of long fibres over time.

5.251 McDonald *et al.* [230] identified excess mortality from lung cancer among friction products workers, but there were no mesotheliomas:

> "A study by McDonald *et al*. (1984) investigated mortality due to lung cancer, mesothelioma and asbestosis in three US factories manufacturing friction products and packings. The cohort comprised 3641 men employed between 1938-1958. During the 1930s exposures for most processes were 1-5 mpcf (millions of particles per cubic foot) and > 10 mpcf during dry mould mixing. By the 1960s most exposures were < 0.5 mpcf. A significant excess of deaths (reference was to mortality rates for Connecticut) due to respiratory cancer was observed however this was not related to duration of employment. No cases of mesothelioma were reported. There was limited evidence of an increase in risk of lung cancer with increasing exposure. However the SMR for lung cancer was noted in workers with less than one year of service.
>
> A study by Finkelstein, (1989) investigated mortality rates among 1657 employees at two Ontario factories manufacturing chrysotile friction materials. The study population consisted of workers employed for at least 12 months after 1 January 1950. The study showed a significant increase in mortality from laryngeal cancer and lung cancer. No increase in mortality was noted from gastrointestinal cancer or from non-malignant respiratory disease. One or two deaths may have been due to pleural mesothelioma. Case-control analysis demonstrated a lack of association between the risk of death from laryngeal or lung cancer and the duration of employment or employment in departments where chrysotile had been used. The author also noted that cigarette smoking is a risk factor for laryngeal cancer and lung cancer, and therefore, increased risk may be in part attributable to differences in smoking habits." [NICNAS 99, p 65].

5.252 Similarly, Woitowitz and Rödelsperger [227, 233] found that:

> "There is no evidence that car mechanics are exposed to an increased risk of mesothelioma even if they do brake repairs, but asbestos exposure in other employment is an important confounding factor, so that if there is a mesothelioma risk for car mechanics but it was small, it would not be detectable."

5.253 Nonetheless, the 1999 Report for the Australian Mesothelioma Register[24] (AMR 99) records 58 mesotheliomas among brake mechanics with no other exposures to asbestos, during the almost 13-year period between 01 January 1986 and 31 October 1999 (total cases with a stated history of asbestos exposure = 2585). Mechanics who frequently or consistently work on brake linings and brake blocks represent only a sub-fraction of the total workforce of mechanics in Australia. If one takes the 1996 census figure of 82,827 for male mechanic[25], this amounts to 58 mesotheliomas in 1,062,946 person-years (= 54.6 mesotheliomas per million person-years). If one rounds off the workforce to 100,000 male mechanics, the figure becomes 45 mesotheliomas per million person-years. If one then doubles the workforce population to take into account retirees and other workers who moved on to other occupations (although a figure of 200,000 is almost certainly an overestimate because it would include all mechanics, whereas brake mechanics constitute a smaller sub-class), the mesothelioma rate becomes 22.6 per million person-years — well under the rate of 337 mesotheliomas per million person-years for the Quebec chrysotile miners and millers but still substantially above the upper limit of the estimated background rate of 1-2 mesotheliomas per million person-years (about 10-fold). One might suspect that mesotheliomas in brake mechanics will cluster in those involved in the grinding, bevelling and other operations on new brake blocks and brake linings (i.e. brake materials unaltered by heat).

5.254 Using an earlier set of data for Australia, NICNAS 99 came to a similar conclusion:

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[24]The Register is a compilation of all and unselected mesotheliomas throughout Australia.

[25]This over-estimates the number of brake mechanics, because the figure includes all automotive mechanics, engine mechanics, apprentices, and supervisors: Australian Bureau of Statistics, 12 October 1999.

> "Out of 2119 mesothelioma cases registered (with a response to history) for the period 1986-1995, 46 cases were listed for the category 'brake lining - manufacture/repair', 40 of which were recorded in car mechanics, of which 37 were exposed to asbestos in this occupation only ... Overall the numbers indicate a slight increase of around 1-2 cases per year, which is roughly proportional to the growth rate of all mesothelioma cases in Australia" ... [p 66].

5.255 It is apparent that these considerations apply to occupational circumstances.

5.256 Evidence indicates that the general population is exposed to only very low levels of asbestos derived from the braking of passing automobiles, and that most of these fibres represent short-length fibres and heat-altered chrysotile. NICNAS 99 has this to say on the subject:

> "It is claimed that the amount of asbestos found in the dust arising from braking is rarely more than 1% of the wear product (Asbestos Information Committee, 1975). It is not known what quantity of chrysotile is imported in brake linings and other friction materials, but ABS [Australian Bureau of Statistics] data indicates in excess of 750,000 articles (brake linings, pads and clutch facings) being imported in 1997 containing asbestos and therefore possibly containing chrysotile. Assuming each unit weighs 200 g and contains 50% chrysotile, this equates to around 150 tonnes of chrysotile per annum. Assuming a further 1000 tonnes of chrysotile present in friction products manufactured in Australia, it is estimated that (assuming a worst case scenario of 1% release per annum, i.e. all products are completely worn in one year), around 11.5 tonnes of chrysotile will be released per annum countrywide or 32 kg per day spread all around the country. It is acknowledged that this figure may be an overestimate, as studies have shown that some of the chrysotile is degraded to magnesium silicates and forsterite ... In addition, some of the debris will be retained in the brake system and removed and disposed of under controlled conditions." [p 78].

(ii) Exposure from chrysotile textile materials

5.257 Table 14 represents a reproduction of Table 7 in EHC 203 for exposure estimates in the South Carolina chrysotile textile plant (1930-1975) before and after controls on exposure levels. As can be seen from this Table, the application of controls on exposure levels produced a significant reduction of exposure, and currently available control technology allows even lower levels to be attained (EHC 203).

5.258 EHC 203 refers to a study in Japan which reported a geometric mean concentration of 0.1-0.2 f/ml in the period 1984-1986, for asbestos spinning. From published studies, it seems clear that asbestos textile workers are at greater risk for asbestosis (historically) and lung cancer than mesothelioma. EHC 203 also gives the following comments:

TABLE 14: EXPOSURE ESTIMATES IN A CHRYSOTILE TEXTILE PLANT (1930-1975)
(ESTIMATED MEAN EXPOSURE TO FIBRES LONGER THAN 5 µM IN F/ML)

Operation	Without controls	With controls
Fibre preparation	26.2-78.0	5.8-17.2
Carding	10.8-22.1	4.3-9.0
Spinning	4.8-8.2	4.8-6.7
Twisting	24.6-376.0	5.4-7.9
Winding	4.1-20.9	4.1-8.4
Weaving	5.3-30.6	1.4-8.2

From Dement *et al*. (1983)

"Studies that correlate disease prevalence or symptoms with cumulative exposure can under-estimate disease risk due to progression of disease after employment ceases. Although workers were exposed to both chrysotile and crocidolite (the latter being approximate 5% of all asbestos used), results for 379 men employed at least 10 years in the Rochdale asbestos textile plant are informative ... Exposure estimated from work histories range from an average of 2.9 to 14.5 f/ml. Overall, small opacities (> 1/0) were reported in 88/379 (23%) of chest radiographs, with evidence of a gradient seriously confounded by date of first employment and transfer of subjects with suspected asbestosis to less dusty conditions. On the basis of data on incidence, the authors drew conclusions on exposure-response between cumulative exposure and prevalence or incidence of crepitations, possible asbestosis and certified asbestosis - all three depending on clinical opinion and judgement. The authors state that possible asbestosis occurs in no more than 1% of men after 40 years of exposure to concentrations between 0.3 and 1.1 f/ml" [EHC 203, p 105].

5.259 On p. 114, EHC 203 goes on to discuss other consequences of exposures sustained during textile manufacture:

"The health of employees has been studied in any detail in only three asbestos textile plants. These comprise a factory at Rochdale, England, originally studied by Doll (1955) and more recently by Peto *et al*. (1985), another located in Mannheim, Pennsylvania, USA, studied by McDonald et al. (1983b) and a plant in Charleston, South Carolina, USA. Only the study in South Carolina is considered primarily relevant for assessment of the health effects of chrysotile. Although the SMRs for lung cancer in these plants were broadly equivalent, the rates of mesothelioma varied considerably, which may reflect the greater proportions of amphiboles in the Mannheim and Rochdale cohorts.

The textile workers in South Carolina plant have been studied in two separate but overlapping cohorts ... . The only amphibole used in this plant was approximately one tonne of imported crocidolite from the early 1950s until 1972, plus a very small quantity of amosite for experimental purposes briefly in the late 1950s. The crocidolite yarn was processed at a single location only, so Charleston can be considered an almost pure chrysotile operation. Exposure levels for workers at this plant were estimated by Dement *et al*. (1983a) using nearly 6000 exposure measurements covering the period 1930-1975 and taking into account changes in plant processes and engineering controls (Table 7). The conversion of past exposures measured in mpcm (mpcf) to f/ml was based on both paired sample data (100 pairs) and concurrent samples (986 samples) by these two methods collected in plant operations during 1968-1971.

The most recent update of the Charleston study by Dement *et al*. (1994) demonstrated an overall lung cancer SMR of 1.97 (126 observed) and an overall SMR for non-malignant respiratory diseases ... of 3.11 (69 observed). The data for white males, for which data were more complete, demonstrated an overall lung cancer SMR of 2.34 for those achieving at least 15 years of latency. The risk of lung cancer was found to increase rapidly in relation to cumulative exposure. Data for the entire cohort demonstrated an increase in the lung cancer risk of 2-3% for each fibre/ml-year of cumulative chrysotile exposure. Two mesotheliomas were observed among this cohort and an additional mesothelioma was identified among plant workers, occurred after the study follow-up period. Analyses of an overlapping cohort from the same factory ... provided similar results.

... the regression line slopes for relative risks of lung cancer in relation to accumulated exposure in the Charleston plant are all some 30 times steeper than those observed in chrysotile mining and cement product manufacture."

5.260 From the foregoing discussion, it is plain that these risks apply to occupational circumstances, and not to non-occupational situations. It is my perception that there is debate among experts over the carcinogenicity of chrysotile released from or associated with friction products and textiles respectively.

Dr. Infante:

5.261 Exposure to chrysotile asbestos through the manufacturing and downstream manipulation of friction products and textiles carries with it the risks associated with exposure to asbestos, most notably, lung cancer, asbestosis and mesothelioma. This is mostly an occupational issue except for consumers, who do their own brake replacement which would put them at risk of developing these diseases.

5.262 Epidemiological studies of workers involved in the manufacturing of friction products demonstrate an elevated risk of lung cancer (McDonald *et al.* 1994). Other investigators have not observed an excess of lung cancer in the manufacturing of chrysotile and cricidolite containing friction products, but have identified cases of mesothelioma related to both types of asbestos used to produce these products (Berry and Newhouse, 1983). Cases of mesothelioma also have been reported among car mechanics who serviced brakes containing chrysotile fibres only and were exposed to levels estimated to be below 1 f/cc-year cumulative exposure (Woitowitz and Rodelsperger, 1991). Epidemiological study results indicate a high risk of disease related to chrysotile textiles. See my responses to Questions 4(a)-4(c).

Dr. Musk:

5.263 It is my opinion that there is a risk of disease from the release of chrysotile fibres from friction materials (as in brakes and clutches) or textiles (as in asbestos blankets and suits). In general the risk will be dependent on the degree of exposure (as above) and is therefore likely to be greater in people with occupational rather than non-occupational exposure. Fibres released from friction materials may be shorter than from other sources. These fibres may clear more rapidly from the lungs and possibly be related to lower risks but I am not aware of any direct data on this.

Question 3:

The parties disagree as to the relative pathogenicity of amphibole and chrysotile asbestos. Canada argues that, due to chemical and physical differences, a crucial distinction is to be made between amphibole and chrysotile asbestos: the latter is less pathogenic than the former. The European Communities, on the other hand, argues that chrysotile is as dangerous as amphiboles. In answering the four sub-questions below, please specify to what extent your opinion is based on epidemiological data, in vivo or in vitro evidence.

3.(a) For the purpose of assessing the risk to human health arising from exposure to asbestos fibres, should a distinction be made between chrysotile and amphibole asbestos?

Dr. de Klerk:

5.264 In terms of risks to human health, epidemiological evidence is clear that, for a given quantity (intensity and duration) of exposure, chrysotile imparts less risk than amphibole fibres.

Dr. Henderson:

5.265 YES — a clear distinction should be made between chrysotile and the amphibole forms of asbestos. On a fibre-for-fibre basis, amphiboles such as crocidolite and amosite are substantially more carcinogenic for the mesothelium than chrysotile. This potency differential is to some extent confounded by the far greater usage of chrysotile both now and historically (> 95 per cent of world asbestos production). In addition, it is worth reiterating that Canadian chrysotile on average contains trace quantities of fibrous tremolite (an amphibole).

5.266 It is my perception that there is broad agreement among experts that the amphiboles are more potent carcinogens for the mesothelium than chrysotile.

Dr. Infante:

5.267 For purposes of assessing disease risk from exposure to asbestos fibres, I see no basis for making a distinction between chrysotile and amphibole asbestos. Several high quality epidemiological studies of workers exposed to chrysotile asbestos demonstrate an elevated risk of death from lung cancer, asbestosis and mesothelioma. The risk of death from lung cancer and asbestosis related to chrysotile exposure appears to be similar to that from exposure to other forms of asbestos. Although epidemiological study suggests that the risk of death from mesothelioma from chrysotile exposure may be less than the mesothelioma risk from amphibole asbestos, it is somewhat difficult to make this comparison. Many of the studies of workers exposed to amphibole asbestos go back further in time and less information is available on the quantitative aspects of exposure. Thus, the role of error in exposure estimation on the perceived difference is difficult to determine. On the other hand, some experimental inhalation studies demonstrate that chrysotile asbestos may be more potent than other forms of asbestos in the induction of mesothelioma (and lung cancer) in relation to the amount of dust deposited in the lungs (Wagner *et al.,* 1974). In any event, the attributable risk of respiratory diseases from exposure to asbestos is going to be heavily weighted by lung cancer and asbestosis. So, even if exposure to chrysotile asbestos would result in a slightly lower relative risk of death from mesothelioma, the overall risk of the asbestos related diseases combined, i.e., lung cancer, asbestosis, mesothelioma, decrements in pulmonary function, will not be perceptively different for chrysotile as compared to the amphiboles. Thus, a distinction should not be made between chrysotile asbestos and amphibole asbestos. In my opinion, exposure to all forms of asbestos results in a significant disease burden to society.

5.268 I believe that there is a need to distinguish chrysotile asbestos from amphiboles based on the epidemiological data at least.

3.(b) What are the key properties which cause pathogenicity of, respectively, amphiboles and chrysotile fibres for (i) asbestosis, (ii) lung cancer, (iii) mesothelioma, and (iv) other asbestos-related pathologies?

Dr. de Klerk:

5.269 The properties are the same: size, shape and durability in the lung, that is fibres have to be of certain size and shape to be deposited in the lungs and have to stay there long enough to produce a response. Since most of the body's responses to fibres appear to be stochastic in nature, the additional features are of course the intensity and duration of exposure as outlined above. All the types of asbestos differ according to these properties, the main difference with chrysotile is that it is less durable in lung tissue than amphibole fibres: it is more soluble and the fibres tend to break more readily into smaller fibrils, it also tends to be more curly rather than dead straight in shape.

Dr. Henderson:

5.270 As discussed already, the pathogenicity of asbestos appears to reside in the physical properties and biopersistence of the fibres, summarized as the 3 Ds — namely dose, fibre dimensions and durability (bio-persistence). All commercial asbestos has the capacity to induce asbestosis, lung cancer, mesothelioma and other pleural abnormalities (e.g., parietal pleural fibrous plaques, benign asbestos pleuritis with effusion, and diffuse pleural fibrosis). Chrysotile and the amphiboles have different potencies in generating these disorders: for example, the amphibole varieties of asbestos appear to be substantially more pathogenic than chrysotile for the induction of asbestosis and mesothelioma, whereas the differential is not sustained for the induction of lung cancer, for which

chrysotile is associated with one of the lowest lung cancer rates (in Quebec chrysotile miners and millers) and the highest rate of lung cancer (South Carolina asbestos textile workers, who used Canadian chrysotile).

5.271 Asbestosis: There is good evidence that asbestosis is a dose-dependent disorder with a threshold effect. There is widespread agreement that asbestosis in general is a consequence of high intensity exposure (or lower intensity but more prolonged exposure) than mesothelioma not associated with asbestosis, so that the concentration of asbestos bodies and uncoated asbestos fibres within the lung tissue of asbestotics is considerably higher than the concentrations encountered in mesothelioma patients without asbestosis and in individuals with parietal pleural plaques. In addition, some studies have shown that the severity of asbestosis and its liability to progression are related to the asbestos body and fibre concentration in lung tissue. In the past, asbestosis as a consequence of high-dose exposure was a progressive disorder leading to progressive respiratory insufficiency and death, whereas many cases of asbestosis encountered during the 1980s and 1990s represent milder and static forms of the disease.

5.272 Churg [234] points out that:

> " ... a considerably greater burden of chrysotile (with its accompanying tremolite) than of amosite or crocidolite is required to produce any particular diseases. ... For example, the mean burden of chrysotile plus tremolite in the lungs of [Quebec chrysotile] miners and millers with asbestosis is 17 times the amosite burden in the lungs of shipyard workers with asbestosis." [p 294].

5.273 To some extent, this differential in potency may reflect fibre dimensions and the generation of oxidants, but a more likely explanation is the faster clearance of chrysotile from lung tissue than any of the amphiboles. However, Churg's comments seem to apply to Quebec chrysotile miners and millers in particular; in contrast, the study reported by Green *et al.* [191], which reported histological asbestosis at relatively low cumulative exposures was carried out on South Carolina asbestos textile workers who also worked with Canadian chrysotile (please see following discussion).

5.274 There is also evidence that long fibres are implicated in the development of asbestosis, but this may reflect in part the anatomical distribution of long versus short fibres in lung tissue. For example, some studies have shown that short fibres in the vicinity of 1 µm in length have a biological potency similar to that of the longer fibres for initiation of the inflammatory changes implicated in the development of asbestosis, but they do not penetrate the walls of bronchi or bronchioles to the same extent as the longer fibres, to reach the alveolar interstitium. Another study related the development of asbestosis to the total surface area of deposited fibres, rather than to particular lengths of fibres.

5.275 The effect of dose: There is good evidence that the inhaled dose of asbestos affects: (i) the development of the disease itself; (ii) the latency period between exposure and the onset of the disease; and (iii) the severity and progression of disease.

5.276 Fibre burden studies on human lung tissues show that patients with asbestosis in general have higher tissue burdens than patients with asbestos-related diseases other than asbestosis. Accordingly, Mossman and Churg [202] state that:

> " ... asbestosis is clearly a fiber-dose driven disease but, nonetheless, only a fraction of any cohort exposed to a fibrogenic dose of asbestos develops asbestosis. It has been proposed that person-to-person variations in either fiber deposition or fiber clearance may account for this phenomenon." [p 1671].

5.277 The Ontario Royal Commission [235] noted that asbestos exposures < 25 fibre-years would be unlikely to produce clinical asbestosis (about = 1 per cent of individuals exposed at this level may

develop clinical or radiological asbestosis), whereas Browne [236] considered that the minimum dose required to produce clinical asbestosis was in the range of 25-100 fibre-years.

> "Chest X-ray changes among textile and friction product workers in China were reported by Huang (1990). A total of 824 workers employed for at least 3 years in a chrysotile products factory from the start-up of the factory in 1958 until 1980, with follow-through to September 1982, were studied. Chest X-ray changes compatible with asbestosis were assessed using the Chinese standard system for interpretation of X-rays. Cases were defined as Grade I asbestosis (approximately equivalent to ILO = 1/1). Overall, 277 workers were diagnosed with asbestosis during the follow-up period, corresponding to a period prevalence of 31%. Exposure-response analysis, based on gravimetric data converted to fibre counts, predicted a 1% prevalence of Grade I asbestosis at a cumulative exposure of 22 f/ml-years." [EHC 203, p 106]

5.278 In an autopsy study of South Carolina asbestos textile workers who used Canadian chrysotile — the same group studied by Dement *et al.* [171, 172, 237-241] and McDonald et al. [161, 242] — Green et al. [191] showed that histological asbestosis was usually present at = 20 fibre-years of exposure, and a few cases were observed at 10-20 fibre-years. Thimpont and de Vuyst [243] reported that around 50 per cent of specimens of lung tissue removed because of lung cancer showed low-grade airway and interstitial fibrosis with asbestos bodies, when the asbestos body concentration was = 5000 per gram dry lung tissue.

5.279 These different threshold doses are not inconsistent with each other, because they deal with identification of asbestosis by different modalities (i.e. clinical/radiological asbestosis versus histological asbestosis). In this respect, histological examination is generally considered to represent the most sensitive and specific technique for the diagnosis of asbestosis, followed in descending order by high-resolution CT scanning, conventional CT scans and chest X-rays (which will fail to detect asbestosis in about 20 per cent of cases, especially low-grade asbestosis). In other words, early (grade I) asbestosis may be undetectable by clinical investigations.

5.280 Latency interval: there is also evidence that the latency period between first exposure to asbestos and the subsequent diagnosis of asbestosis is roughly inversely proportional to the exposure level, so that short latencies are encountered with high exposures (e.g. the Wittenoom cohort).

5.281 Mossman and Churg [202] also state that:

> "Fibre burden studies also indicate that there is a correlation between pathologic severity of asbestosis and increasing burden of asbestos bodies (which are largely markers of amphibole exposure) or uncoated amosite and crocidolite fibres" ... . [p 1670].

5.282 There are two additional points that are worth emphasis:

- There appears to be considerable variation among individuals in their propensity to develop asbestosis (or variation in the latency intervals for equivalent exposures). For example, I have seen cases where there was a high content of amphibole asbestos in lung tissue (up to 100 million fibres per gram dry lung tissue or more), in the absence of histological asbestosis — at the time when the fibre burden analysis was carried out — whereas other cases had clinical asbestosis with much lower tissue fibre burdens. Of course, one caveat about this situation concerns latency, whereas another relates to the time when the fibre burden analysis was carried out, which is different from the time when the disease was developing.

- In addition to high fibre burdens, the severity and progression of asbestosis can be

influenced by other factors, such as cigarette smoke (though tobacco smoke by itself cannot cause asbestosis, of course).

5.283 Mesothelioma: Please see discussion in Section C.1.(f) to(h).

5.284 Lung cancer: Please see discussion in Section C.1.(i).

5.285 Other asbestos-related pathology: The effect of dose is less clear for the induction of parietal pleural fibrous plaques than for other asbestos-related disorders. There is evidence that the frequency and extent of plaques are dose-related, so that plaques tend to be more extensive with higher exposures. However, plaques can also follow trivial exposures to asbestos and asbestos-like minerals, so that the frequency of asbestos-related pleural plaques appears to correlate more closely with duration since exposure than the level of exposure. Plaques are known to be endemic in Finland, apparently as a consequence of very low-level exposure to asbestos-like fibres in the general environment; on the other hand, in societies where plaques are not endemic — e.g. North America, Western Europe and Australia — about 80-90 per cent of radiologically well-defined plaques are a consequence of occupational exposure to asbestos. In such societies, pleural plaques also represent a useful tissue marker for prior asbestos exposure. At the same time, it is also worth emphasising that parietal pleural plaques by themselves do not predispose to any other asbestos-related disorder, the liability being related to inhaled dose and fibre types.

Dr. Infante:

5.286 The key properties of the pathogenicity of amphiboles and chrysotile fibres, although not known for certain, are thought to be related to the physical characteristics of the fibres, namely the diameter, length, aspect ratio of length to diameter, surface area and perhaps surface charge of the fibres. Toxicological data suggest that long thin asbestos fibres may be relatively more potent than other asbestos fibres in their ability to induce asbestos related diseases. There is also experimental evidence, however, that shorter fibres can produce asbestos related diseases, though not in as great a magnitude. Any manipulation of these fibres that results in their diameter becoming thinner may provide a relatively greater contribution to the associated pathology and a greater toxic response than if the fibres were not manipulated in any way. The issue of solubility has been raised in terms of asbestos fibres and their capability to cause disease. The role of solubility with asbestos fibres and disease potential, however, is not so clear because chrysotile asbestos seems to have the same overall potency as other forms of asbestos, yet chrysotile fibres appear to be relatively more soluble than amphibole fibres.

Dr. Musk:

5.287 It is my opinion that the key characteristics determining the pathogenicity of asbestos for asbestosis, lung cancer, mesothelioma and other diseases are determined by the physical and chemical characteristics of the asbestos. The physical characteristics include length, diameter and "straightness" of the fibres. The chemical properties determine the durability of the fibres.

3.(c) What is the respective capacity of amphiboles and chrysotile to induce (i) asbestosis, (ii) lung cancer, (iii) mesothelioma, and (iv)other asbestos-related pathologies?

Dr. de Klerk:

5.288 Comparisons between chrysotile and the amphiboles in their capacity to produce mesothelioma and lung cancer have been extensive, for the other diseases, much less so. There is some *in vitro* and *in vivo* evidence that amphiboles, particularly crocidolite, are more fibrogenic than chrysotile, but there is no clear epidemiological evidence on this. Pleural plaques appear to be more

common among anthophyllite workers than others while crocidolite workers have more diffuse pleural thickening, and benign asbestos pleurisy also seems to be more common after crocidolite exposure. Historically, asbestosis occurred commonly after heavy exposure to all types of asbestos. For mesothelioma, it is thought that for a given cumulative exposure, chrysotile is between one tenth and one hundredth as potent as crocidolite. There is some controversy over the relative capacity of amosite and crocidolite, but amosite seems to carry about one tenth of the risk of crocidolite. For lung cancer, amosite and crocidolite seem to have similar capacities, with chrysotile weighing in around one tenth to one fiftieth of this.

Dr. Henderson:

5.289 See my response to Question 3(b).

Dr. Infante:

5.290 The quantitative risk of dying from lung cancer as a result of exposure to chrysotile asbestos is at least as great as that from exposure to other forms of asbestos. Quantitative risk assessments based on several epidemiological studies indicate a very high risk of lung cancer (potency) among workers exposed to chrysotile asbestos. The cohort study by Dement et al., first published in 1983 and updated in 1994 contains one of the best estimates of worker exposure to chrysotile accompanied by the estimates of relative risk (RR) for lung cancer and asbestosis. In this study, the investigators determined conversion ratios from the available industrial hygiene data by evaluating sampling results from surveys that used the impinger method to measure dust in million particles per cubic foot (MPPCF), and membrane filter samples that allowed for the counting of fibre concentrations. For early exposure periods, the investigators converted one MPPCF to the equivalent of 3 f/cc > 5 um in length for all areas except for preparation where a conversion factor of 8 fibres for every MPPCF was used. In my opinion, this study is the strongest in its methodology for estimating asbestos exposure among cohort members. Quantitative risk assessment based on data for the entire cohort estimates an increase in the RR of lung cancer ranging from 2-3 per cent for each fibre/cc-year of cumulative chrysotile exposure. To my knowledge, this is the highest estimated risk of lung cancer among asbestos exposed workers that has been corroborated by other investigators, i.e., the same population was also studied by McDonald et al., (1983) and the results are remarkably similar. Studies of lung cancer risk among chrysotile asbestos textile workers from two additional occupational cohorts by McDonald et al., (1982) and Peto et al., (1985) also provide similar results. Using data from the McDonald et al., (1983) study and a conversion factor of 6 fibres per MPPCF, Peto et al., (1985) estimated an increase in the RR of lung cancer among chrysotile textile workers to be 1.25 per cent per fibre/cc-year of exposure. From their own study of Rochdale textile workers, Peto et al., (1985) estimated the excess lung cancer risk to range from 0.5-1.5 per cent per f/cc-year of cumulative exposure depending upon whether the estimate was based on the entire cohort or on those employed in 1951 or later, respectively. These estimates are of surprisingly similar magnitude given that they are from epidemiological studies that incorporated retrospective exposure estimation that had to rely upon the conversion of measurements from particles to fibres per cc. Thus, three separate populations of chrysotile asbestos textile workers demonstrate remarkably similar elevated risks of lung cancer and therefore add to the confidence of the estimates of excess lung cancer risk per unit of fibre exposure that these studies demonstrate.

5.291 Studies of workers exposed to chrysotile in several industries demonstrate a significantly elevated risk of lung cancer. Studies of workers exposed to a combination of chrysotile and cricidolite, or to chrysotile only in cement production (Hughes et al., 1987) indicate a virtually identical excess risk of mortality from lung cancer. In a study of workers exposed to crocidolite in mining, de Klerk et al., (1989) estimate an elevated relative risk of lung cancer of 1 per cent per f/cc-year of exposure. Essentially, the excess relative risk for lung cancer indicated in all of these studies is close to about 1 per cent per fibre per cc-year of exposure (Stayner et al., 1997). The risk of lung

cancer from chrysotile asbestos exposure is at least as great as the risk of lung cancer associated with amphibole asbestos exposure.

5.292 Analyses based on the study by McDonald et al. (1993) indicates a much lower dose response for lung cancer in relation to chrysotile asbestos in mining and milling as compared to the risk estimates from other studies, particularly chrysotile textile workers. I suspect, however, that the fibre exposure from this study may be overestimated, particularly among the portion of the cohort that is comprised of miners and that a fair amount of misclassification took place in the amount of fibre exposure estimated for individual cohort members.

5.293 Gibbs and Lachance (1972) who published the initial exposure estimates for this population of workers stated that their cumulative dust concentrations for the cohort members may have been far from their actual experience. Subsequent dose response analysis of this cohort applied only a single conversion factor to estimate fibre exposures from the dust count data for the entire cohort. In the study of Siberian chrysotile miners and millers (Tossavainen et al., 1999), those involved in mining experienced average exposures of 0.08 f/cc, while those involved in two separate mills experienced average exposures of 3.62 f/cc (ranges of average exposures for different milling operations 0.37-6.21 f/cc) and 0.65 f/cc (range of average exposures 0.20-1.26 f/cc). The gravimetric sampling results indicate a 5-fold difference in average exposure for miners versus millers, while the sampling results for fibre exposures (the exposure of concern) indicate a 45-fold difference in average exposure concentrations between miners and millers. Taking into consideration the variation within mining and milling jobs, the difference between exposures to workers involved in these jobs would be even greater. The large difference in the fibre exposures between miners and millers that was observed in the Tossavainen et al. (1999) study adds further support to my concern that applying a single conversion factor from dust samples in estimating chrysotile fibre exposures for miners and millers is the most likely explanation for the slope of the dose response for lung cancer in the McDonald et al. study being so different from the slope for lung cancer based on the studies of chrysotile textile workers. Stayner et al. (1996) provide a summary of data indicating that the lung cancer risk from exposure to chrysotile in either experimental animals or in humans is similar to the risk from exposure to amphibole asbestos in these species.

5.294 With regard to asbestosis, Stayner et al. (1997) published a dose response analysis based upon the updated study of chrysotile textile workers by Dement et al. (1994). Their analysis indicates an excess risk of 2 deaths from asbestosis per 1000 employees exposed to 0.1 f/cc for an occupational lifetime exposure of 45 years, or 0.2 per cent from a cumulative exposure of 4.5f/cc-years. Two additional studies estimate similar risks of death from asbestosis in relation to cumulative chrysotile exposure. The study by Berry et al. (1979) estimates a risk of 1 per cent for those categorized as "probably having asbestosis" among chrysotile textile workers who were exposed to an estimated range of 0.3 f/cc to 1.1 f/cc for 40 years, or a cumulative exposure of 12-44 f/cc-years. For those categorized as "certified asbestosis" a 1 per cent excess risk of death was associated with 63 f/cc-years of exposure. Huang (1990) estimates a risk of asbestosis of 1 per cent associated with 22 f/cc-years of exposure during the manufacturing of chrysotile textile and friction products. Data from Dement et al. (1994) allow for an estimate of 2 per cent asbestosis associated with 22.5 f/cc-years of exposure. Studies of workers exposed to a mixture of chrysotile and crocidolite asbestos in the manufacture of cement products have demonstrated an elevated the risk of "certified asbestosis" of 1 per cent associated with 10 f/cc-years of exposure (Finkelstein 1982). Finkelstein was of the opinion that he identified a 1 per cent excess risk of asbestosis related to a lower cumulative dose of asbestos exposures than Berry et al. (1979) because of the longer period of follow-up of the cohort which gave more time for the asbestosis to clinically manifest itself.

5.295 I am not aware of evidence that similar cumulative exposures to amphibole forms of asbestos will result in a greater risk of asbestosis. Thus, it is difficult to make any distinction in potency between the amphiboles and chrysotile asbestos in relation to asbestosis.

5.296 Chrysotile exposures related to numerous jobs and occupations has been associated with mesothelioma through epidemiological studies and case reports. In some situations, standby exposure only was associated with mesothelioma. Based on epidemiological studies, the potency of chrysotile to induce mesothelioma may be less than that from other forms of asbestos. However, the rarity of mesothelioma in the general population and the difficulty in determining asbestos exposure levels experienced by cohort members decades before measurements were taken, coupled with conversion of dust counts in particles to fibres per cc, make it difficult to determine differences in potency estimates with regard to the various forms of asbestos and mesothelioma. Based on toxicological study results, in terms of the quantity of dust deposited and retained in the lungs, chrysotile may be more potent than other forms of asbestos in the induction mesothelioma and fibrosis (Wagner et al., 1974). The population attributable risk of contracting mesothelioma from chrysotile, however, will be greater than from other forms of asbestos because of the much greater potential for exposure to chrysotile.

5.297 I have not seen any quantitative data related to decrements in lung function for either chrysotile and amphibole asbestos. On the basis of mortality from asbestosis, I assume there is little difference in lung function related to the various forms of asbestos.

5.298 In summary to this question, in evaluating the epidemiological evidence, I see no basis for concluding that the overall disease potential from exposure to the amphiboles is any different than that from exposure to chrysotile asbestos with the possible exception that amphiboles may be more potent in causing mesothelioma and chrysotile may be more potent in causing lung cancer. Studies in experimental animals demonstrate the ability of chrysotile asbestos as well as the amphiboles to induce fibrosis, lung cancer and mesothelioma. From a public health standpoint, in terms of quantification of disease, it would be extremely difficult to make any distinction between exposure to the amphiboles and chrysotile asbestos fibres.

Dr. Musk:

5.299 Broadly it is my understanding that the relative pathogenicity of the different fibres for the various diseases is different (see Table)

	CROCIDOLITE	**AMOSITE**	**ANTHOPHYLLITE**	**CHRYSOTILE**
Asbestosis	1	1	1	1
Lung cancer	10	10	10	<1
Mesothelioma	100	10	5	1
Benign asbestos pleural effusion/ diffuse pleural thickening	100	10	10	1
Pleural plaques	1	1	10	1

<u>Question 4</u>:

The parties in this dispute disagree as to the risk to human health associated with chrysotile asbestos fibres at low levels of exposure, i.e. either prolonged exposure to low concentrations of fibres or occasional peaks of exposure. The European Communities considers that, because of a lack of data at low levels of exposure, it is appropriate to endorse the linear relationship model to assess risks associated with such low levels of exposure. On the other hand, Canada is of the view that, at such low levels of exposure, empirical evidence suggests that there is a practical threshold below which chrysotile asbestos fibres present no measurable effects on health.

4.(a) Are epidemiological data available for low levels of exposure to chrysotile fibres and what do they show?

Dr. de Klerk:

5.300 There have been several epidemiological studies that have shown no increased risk for low levels of exposure to chrysotile, particularly in friction products industries.

Dr. Henderson:

5.301 So far as I am aware, there are no exposure-response data for such levels of exposure.

5.302 For example, EHC 203 states the following:

> "Few data on concentrations of fibres associated with the installation and use of chrysotile-containing products are available, although this is easily the most likely place for workers to be exposed" [EHC 203, p 3.].
>
> "Overall, the available toxicological data provide clear evidence that chrysotile fibres can cause fibrogenic and carcinogenic hazards to humans. The data, however, are not adequate for providing quantitative estimates of the risk to humans. This is because there are inadequate exposure-response data from inhalation studies, and there are uncertainties concerning the sensitivities of the animal studies for predicting human risk" [EHC 203, p.7].
>
> "There is evidence that fibrous tremolite causes mesothelioma in humans. Since commercial chrysotile may contain fibrous tremolite, it has been hypothesized that the latter may contribute to the induction of mesotheliomas in some populations exposed primarily to chrysotile. The extent to which the observed excesses of mesothelioma might be attributed to the fibrous tremolite content has not been resolved" [EHC 203, p 8-9].
>
> "Epidemiological studies that contribute to our understanding of the health effects of chrysotile conducted to date and reviewed in this monograph have been on populations mainly in the mining or manufacturing sectors and not in construction or other user industries. This should be borne in mind when considering potential risks associated with exposure to chrysotile" [EHC 203 , p 137].

Dr. Infante:

5.303 One means of determining the risk from low exposure levels for carcinogens is to estimate the risk from studies where exposure information is of reasonably good quality and the estimates of risk were made using sound epidemiological principles and methodology. Once these studies have been identified, an appropriate way to determine quantitative risk from low exposure levels is to use all of the available data in a particular study and to estimate dose response. As mentioned in my response to Question 3(c) above, several studies of workers exposed to chrysotile asbestos can be used to estimate risk from low levels of exposure. The studies mentioned above, which represent three separate populations of chrysotile textile workers demonstrate an excess relative risk of lung cancer ranging from 0.5-3 per cent for each fibre/cc-year of exposure. Risk assessment based on the study of South Carolina chrysotile textile workers (Stayner et al., 1997) indicates that individuals exposed to 0.1 f/cc-year for an occupational lifetime of 45 years, e.g., 4.5 f/cc-years cumulative exposure to chrysotile, have an elevated risk of 5 extra deaths from lung cancer and 2 extra deaths from asbestosis per 1000 workers. Dose response for mesothelioma could not be estimated because there were too few deaths from this cause in the study.

5.304 Epidemiological study results and case reports indicate that a large number of jobs which entail occasional peak exposures to chrysotile asbestos have resulted in workers being diagnosed with

mesothelioma. Furthermore, mesothelioma has been diagnosed among household contacts of asbestos cement workers (Magnani et al., 1992; Ascoli et al., 1996), among individuals living near chrysotile mining and milling operations (Began et al., 1992), or living in houses constructed with asbestos cement (Ascoli et al., 1996), or who were exposed to low cumulative amounts of chrysotile asbestos from brake lining work (Woitowitz and Rodelsperger, 1991), or from standby exposure as bakers (Ascoli et al., 1996). This information contributes to the evidence that very low exposure to all forms of asbestos can induce cancer. These observations of mesothelioma should be considered as sentinel events for the pathologies other than mesothelioma that are more difficult to identify among large populations that experience relatively more remote exposure to chrysotile asbestos. In my opinion, these studies constitute high level risk from low levels of exposure to chrysotile asbestos.

Dr. Musk:

5.305 It my understanding there are epidemiological studies which do not show a statistically increased risk of disease from low levels of exposure to chrysotile. However, the absence of demonstrating an increased risk does not mean that there is not some risk as it is not possible to prove a negative and a threshold has not been demonstrated for any carcinogen (nor in my opinion is it biologically likely that one exists).

4.(b) Is there a threshold below which exposure to chrysotile fibres does not induce (i) asbestosis, (ii) lung cancer, (iii) mesothelioma, and (iv) other asbestos-related pathologies, such as pleural plaques? If there is such a threshold, is it a practical one or is it scientifically established?

Dr. de Klerk:

5.306 It is extraordinarily difficult to demonstrate lack of an effect, or a threshold effect, in epidemiological studies because of the ubiquitous problems of bias, confounding and chance. In particular, the smaller effect that needs to be demonstrated, the larger the study needs to be, both in population size and follow-up time and such studies can rarely be done, even with animals.

Dr. Henderson:

(i) Asbestosis

5.307 Please see my answer to Question 3(b).

(ii) Lung Cancer

5.308 Please see discussion in Section C.1.(i). Please see also the statement from EHC 203 below on the question of a threshold for carcinogenesis by asbestos. Some authorities favour a linear no-threshold model, whereas others argue that a threshold probably exists; nonetheless, there is no general agreement on a numerical threshold for asbestos-induced lung cancer. Dement et al. [171] observed odds ratios of > 2.5 at 2.7-6.8 fibre-years of exposure among South Carolina asbestos textile workers.

(iii) Mesothelioma

5.309 As indicated in previous discussion in this report, a linear dose-response model has been identified for mesothelioma induction by the amphiboles, and the dose-response relationship is maintained at low occupational levels of exposure that overlap with environmental exposures: e.g. definite dose-response relationship reported by Rödelsperger et al. [25, 137] at asbestos fibre concentrations in lung tissue of 100,000-200,000 fibres per gram dry lung tissue, with an indication that this relationship is maintained at lower levels of 50,000-100,000 fibres per gram dry lung (the

level of 100,000-200,000 fibres corresponds to a cumulative dose of 1-2 fibre-years). Iwatsubo et al. [136] identified an increase in the relative risk at 0.5-0.99 fibre-year. No threshold has been identified for amphibole-related mesothelioma. For chrysotile exposures, a dose-response relationship has also been identified at high exposures, but to the best of my knowledge, there are no dose-response data for low-level exposures to chrysotile.

5.310 On this point, EHC 203 states that "No threshold has been identified for carcinogenic risks" [for chrysotile; p 144]. At the same time, no increase in risk for mesothelioma has been identified at very low-levels of exposure, of the type associated with well-maintained asbestos in place, in public buildings. However, it is impossible to ascertain whether or not there is an increase in risk at this order of exposure, because no control or reference group can be assembled where there is no asbestos content in lung tissue. If a threshold exists, it must lie somewhere in this area, between no exposure, low-level environmental exposure, and low-level occupational exposure.

5.311 De Klerk [115] has also commented on the difficulty or impossibility of distinguishing between background versus environmental mesotheliomas:

> "There have been increases in the incidence rates of malignant mesothelioma in women, in those without identified exposure to asbestos and, possibly, those younger than 35 years of age in Australia and Western Australia. Although part of the first two increases, at least, may be attributable to specific exposure to asbestos, mathematical modelling of the Western Australia data suggests that there has been about a twofold increase in incidence rates from the 1970s to the 1980s that may be due to increased general environmental exposure to asbestos. ... The excess of 1 per million person years over this presumed 'background' rate is also, coincidentally, the amount that was estimated as possibly caused by exposure of school children to 1 fiber per liter ... , a level that might result from use of asbestos-based insulation or other general contamination of the environment with asbestos.
>
> A final consideration in the use of national trend data for estimating environmental effects is the comparison of the likely extrapolated risk from occupational data with the background risk estimated here or from Peto's Los Angeles data. From the Peto paper ... , the incidence of mesothelioma is related to age in the following way:
>
> $$\text{Incidence} = 1.7 \cdot 10^{-12} \cdot (\text{age})^{3.5}$$
>
> which translates to a lifetime risk to age 80 of just over 100 per million people, which is much greater than any of the estimated environmental risks described earlier. An equivalent risk from the adjusted Western Australian data is about 160 per million lifetimes. The question remains as to how these background risks and environmental risks interact. Is the postulated environmental incidence already included in the background incidence, or should the risks be added or even multiplied together? This question is almost certainly unanswerable using epidemiological methods. ...
>
> It is doubtful whether epidemiological methods ... could ever be definitive in deciding whether there is an appreciable hazard from general environmental exposure to asbestos ... or, more importantly, whether the hazard is large enough to justify specific remedial action ..." [pp 29-31].

5.312 Clearly, this issue is one major focus of dispute between experts; from the preceding discussions, it is my perception that — with the exception of asbestosis — no threshold has been delineated, and that even those who claim that a practical threshold must exist cannot delineate such a threshold in precise numerical terms (in this respect, I do not know what the expression "practical threshold" really means).

Dr. Infante:

5.313 Thresholds have not been demonstrated for any substance known to cause cancer and there is no theoretical basis to assume a threshold for the diseases related to chrysotile, or other forms of

asbestos, particularly when the mechanisms involved in the pathology are not fully understood. Furthermore, it is not possible to determine thresholds from epidemiological studies because of lack of statistical power to distinguish that the risk is virtually zero. [Note: At times, some investigators state that a single point estimate from the lowest dose evaluated in an epidemiological study that does not demonstrate a significant elevation in cancer risk constitutes a threshold level for the carcinogen. Such a conclusion is scientifically invalid. When estimating dose-response, one has more confidence in the risk related to a particular dose level by using all of the data available in the study. Using only a single point estimate results in more instability in the estimate of risk for that data point in contrast to using all of the available data in the study.]

5.314 Dose response analyses and modeling specifically for chrysotile asbestos exposure and lung cancer and asbestosis have been conducted recently by Stayner et al. (1997) using data from the Dement et al. (1994) study. Alternative exposure-response models were evaluated as part of the study. A model designed to evaluate evidence of a threshold also was fitted for asbestos exposure in relation to lung cancer and asbestosis. There was no significant evidence for a threshold in models pertaining to either lung cancer or asbestosis.

5.315 With regard to mesothelioma, and other asbestos-related diseases, I am not aware of any evidence of a threshold pertaining to either chrysotile, or other forms of asbestos. Furthermore, from a practical standpoint, even if there were a threshold for the chrysotile related diseases, the exposures that workers will routinely encounter in the future through continued use of chrysotile in commerce, will expose them to concentrations of asbestos that have already been related to pathology in humans. In other words, continued use of asbestos will continue to expose individuals to levels and exposure circumstances that have already been related to disease. The threshold question, therefore, seems moot.

Dr. Musk:

5.316 It is my understanding that a threshold for disease has not been scientifically established.

4.(c) Is the linear relationship model an appropriate method for assessing the risk to human health posed by exposure to chrysotile asbestos at low levels of exposure?

Dr. de Klerk:

5.317 The linear relationship model is generally used as a so-called "conservative" estimate, that is, if it is incorrect, it is more likely to err on the side of safety. In some ways, how one extrapolates risk assessment outside the range of available data is more of a societal decision than a scientific one. Biological plausibility could probably be given to any model.

Dr. Henderson:

5.318 In the absence of alternatives because of unavailability of data on exposure-response relationships at low levels of exposure to chrysotile, the linear relationship model is widely employed. Under these circumstances, this may be an appropriate method for risk assessment at low levels of exposure. Whether or not it is a valid method is unknown.

5.319 NICNAS 99 (p. 72) observes that:

> "There are many problems associated with low-dose risk extrapolation, such as the assumption of a linear relationship. However, as insufficient data exist to indicate a threshold exposure for effect, the linear extrapolation methodology provides a conservative worst-case scenario estimate of risk. Other

> confounding factors in estimating risks from epidemiological data are possible contamination by other fibre types and inaccurate estimates of historical exposures."

5.320 Clearly, this too is one major focus of dispute between experts.

Dr. Infante:

5.321 A linear relationship model is appropriate for determining dose response for chrysotile exposure and lung cancer, and perhaps asbestosis and mesothelioma as well, but the most reasonable model for the latter two diseases are less clear than for lung cancer. With regard to this issue, Stayner et al. (1997) evaluated exposure-response relationships for chrysotile asbestos, and lung cancer and asbestosis by applying several alternate models. The exposure-response relation for asbestos and lung cancer gave the best fit when using a linear model. This observation is consistent with the conclusions of other investigators, who have evaluated dose response for chrysotile asbestos textile workers, or other asbestos workers and mortality from lung cancer (McDonald et al., 1983; Peto et al., 1985; Enterline, Hartley & Henderson, 1987). Furthermore, there appears to be a linear relationship between asbestos exposure and lung cancer over a wide range of exposures where such data are available. Therefore, it seems reasonable to accept a linear relationship for lung cancer when extrapolating risks to exposures below the ranges that have been evaluated in epidemiological studies. Moreover, I am not aware of any literature that convincingly proves that the dose response for asbestos and lung cancer is non-linear. Thus, in my opinion, the linear model is the most appropriate model for estimating dose-response for chrysotile exposure and lung cancer.

5.322 A linear relationship might also be used for chrysotile asbestos exposure and asbestosis although one might make the argument that a non-linear model is also appropriate for asbestosis. Stayner et al. (1997) evaluated this issue using data from the Dement et al. (1994) study and concluded that the association between chrysotile exposure and asbestosis appeared to be non-linear. Stayner et al. (1997) used a non-threshold, non-linear model and the estimates of asbestosis predicted from the model seem to fit very closely with point estimates for asbestosis from other studies of chrysotile exposed populations as mentioned in my responses to Questions 3(c) and 4(a).

5.323 An analysis by Peto et al. (1985) of chrysotile asbestos textile workers shows that a linear model fits the data for mesothelioma with the cube of time since first exposure. In this non-threshold model, the response is linear with dose of asbestos, but exponential with time since initial exposure. The predicted number of mesotheliomas by dose and time since first exposure was in reasonable agreement with the observed number. According to the authors, however, there were too few cases to test the model stringently and they did not attempt to fit other models to their data. Nevertheless, given the consistent observations of the long latency period between initial exposure to various forms of asbestos and the clinical manifestation of mesothelioma, it seems reasonable to use a model that is linear with exposure and exponential with time from initial exposure for chrysotile asbestos and mesothelioma.

Dr. Musk:

5.324 It is my opinion that the linear relationship model is the most appropriate one.

4.(d) Are there scientifically acceptable methods other than the linear relationship model which could be used to assess the risk to human health at low levels of exposure? What results do they suggest?

Dr. de Klerk:

5.325 While a threshold model suggests a lack of risk below a fixed level, it is unlikely that this risk would be completely zero, so that if applied to a much larger population, such a risk could lead to cases of disease.

Dr. Henderson:

5.326 I am not aware of any other methods that have met with broad scientific acceptance or a consensus. It has been suggested that an S-shaped curve might be more appropriate, but I have not seen any data on what the form of the S-curve might be; in other words, the S-shaped model appears to presuppose the existence of a threshold, but no such threshold has been established to the best of my knowledge.

5.327 The problem with arguing that there exists a practical threshold level for lung cancer and mesothelioma induction is that it is impossible to delineate such a threshold in numerical terms, because of a lack of observational data. (Please see also my answer to Q.5(c)).

Dr. Infante:

5.328 From the public health perspective, it has been the convention to use non-threshold linear models for estimating cancer risk to humans. This is particularly the case for substances known to cause cancer in humans. One might deviate from this concept if the mechanism(s) by which the substance causes the cancer were known. This is not the case with chrysotile, or any other form of asbestos. In the particular case of chrysotile asbestos, Stayner et al. (1997) selected several Poisson regression models to explore the shape of the exposure-response relationship between chrysotile asbestos exposure and risk of death from lung cancer. The models were capable of reflecting a wide range of exposure response patterns, including linear, sublinear and supralinear relationships. They also considered a threshold model to determine whether there was evidence that exposures below a certain exposure concentration were equivalent to zero, i.e., that a threshold was present. As mentioned above, for lung cancer, a linear model gave the best fit; for asbestosis, the response preferred was that based on a non-linear, non-threshold model. In both cases, the models did not provide any support for the existence of a threshold. Thus, in my opinion, these models are appropriate to assess risk for these diseases as a result of occupational exposure to chrysotile asbestos. With regard to lung cancer and asbestos, I am not aware of any public health organization, or governmental agency that has ever used a non-linear model to estimate risk. During the hearings held by the U.S. Occupational Safety and Health Administration (OSHA) as part of its rulemaking related to the standard promulgated for asbestos in 1994, numerous scientists were of the opinion that a non-threshold linear model was the preferred model to use for estimating the relationship between asbestos exposure and lung cancer. It is my opinion that a non-linear model is not an acceptable model to use in estimating dose response for asbestos exposure and risk of death from lung cancer. For mesothelioma, I would favor a non-threshold model that incorporates a linear relationship with exposure.

Dr. Musk:

5.329 I believe that there is no reason to discard the linear model as no threshold for any carcinogen is known to exist.

4.(e) To what concentration of chrysotile fibres and for how long must a person be exposed in order to be considered at risk of developing a chrysotile asbestos-related disease (lung cancer, mesothelioma or other asbestos-related pathology)?

Dr. de Klerk:

5.330 A person is "at risk" of developing a chrysotile asbestos-related disease after any exposure to chrysotile asbestos, the lower the amount of exposure, the lower the risk. For example, it could be estimated that there was a 50-50 chance that exposure to 1 fibre of crocidolite could cause 1 case of mesothelioma among the whole population of the world (including all those who have ever lived), i.e. a very small probability, but still greater than zero.

Dr. Henderson:

5.331 This question iterates the issue of a threshold exposure. The answer is essentially the same as for Questions 4(a)-4(d) in the absence of exposure-response data at low levels of exposure.

Dr. Infante:

5.332 The answer to this question depends upon the amount of risk that is considered unacceptable by a particular country. It is a matter of health policy. In the United States, the Environmental Protection Agency (EPA) regulates risk to a level below one extra death in a population of 100,000 people over its entire lifetime. I have already provided estimates for excess risk of death from lung cancer and asbestosis from chrysotile exposure. These risk estimates, however, are average risks to a group of individuals that are based on maximum likelihood estimates (MLEs) and they do not incorporate statistical uncertainty in terms of variability, e.g., they are not based on upper 95 per cent confidence limits as is usually the health policy when estimating adverse health effects to a group of individuals at risk from exposure to an environmental insult. In addition, the risk estimates may only be appropriate to workers' health risks. They are derived from a group of healthy adults, who were able to pass a physical in order to gain employment. They are not representative of individuals in the general population who may be exposed to asbestos and have a compromised immune system, or be exposed to other conditions which may exacerbate their risk of contracting the various diseases related to chrysotile asbestos as estimated from a healthy worker population. There will always be some risk from exposure to asbestos and the degree of that risk will depend upon the amount of asbestos exposure in relation to the susceptibility of the individuals exposed in terms of their health status and other factors that interact to produce clinical manifestation of disease.

5.333 It is noteworthy, as mentioned in my response to Question 1(a) above, that the population surrounding the Quebec mining and milling operation (Begin et al., 1992) that was exposed to background levels of chrysotile asbestos, developed mesothelioma at an incidence of 62.5 cases per million population per year, or 0.625 per 10,000 per year. This risk level translates to 0.5 per cent of the population developing mesothelioma over an 80-year lifetime from this background exposure. This estimate may well represent an underestimate of risk since the identification of cases was based on a workman's compensation board review, and additionally, out-migration of inhabitants would also result in loss of some cases. In the same report, it was pointed out that the largest increase in the mesothelioma rate in Quebec was among individuals that had occupations where their exposure would be occasional only, and that 33 per cent of these cases were exposed for less than a 5-year period. When one adds to this information to additional cases of mesothelioma reported in the literature that are associated with standby exposure to chrysotile, it leads one to the conclusion that occasional exposure for a short period of time, or constant low level exposure to chrysotile asbestos leads to death from mesothelioma (and lung cancer and asbestosis). [Note: while an excess of lung cancer was not identified in the Camus et al. (1998) study of women in the same Quebec population, the study had limited power to detect an excess of lung cancer; it did, however, demonstrate an excess of asbestosis and mesothelioma even though out-migration may have resulted in the loss of all three of these diseases in the study.]

Dr. Musk:

5.334 In my opinion any level of exposure to chrysotile (or other form of asbestos) constitutes some risk and that the level of "acceptable risk" is not a scientific issue but an issue for society to debate and determine at different times according to the evidence as they perceive it.

Question 5:

Canada states that, with controlled use, "health risks associated with occupational exposure throughout the life-cycle of chrysotile asbestos can be reduced to acceptable levels already recognized as such by competent international organizations. The European Communities questions this assertion and says that "les données scientifiques disponibles montrent que l'utilisation dite "sécuritaire" de l'amiante chrysotile ne permet pas d'empêcher un grand nombre de cas d'exposition entraînant des pathologies mortelles". ["available scientific evidence shows that so-called "controlled" use of chrysotile asbestos does not make it possible to prevent many cases of exposure causing fatal pathologies"]

5.(a) Is there a generally agreed methodology applicable to any use of chrysotile-cement products and other high-density chrysotile products throughout their life-cycle that can be referred to as "controlled use"? Is it embodied in international standards?

5.335 This is rather outside my areas of expertise. It does however appear theoretically feasible but practically very unlikely given the problems with "downstream" use described above.

Dr. Henderson:

5.336 In principle, regulation and control of chrysotile and high-density chrysotile products is feasible at some points of the life-cycle (manufacture and disposal), but in reality not others (please see following discussion).

5.337 The manufacture of high-density products is usually carried out under closed conditions with dust extraction. As one example, the manufacture of chrysotile friction materials in Australia involves the following processes: following transfer of other ingredients required for the product mix, unopened 50 kg plastic bags of raw chrysotile are placed in the mixer and opened under dust extraction. The empty bag is then delivered into a second plastic bag attached to the mixer. When full, this second bag is sealed and taken to a controlled disposal site. Mixing is a closed process. After mixing, the material is emptied under dust extraction before decanting into smaller buckets for weighing and use in moulding and finishing processes. The moulding is a hot process and when complete, the moulded product undergoes finishing processes that include grinding, grooving and drilling — all carried out under dust extraction. The finished disc pads and commercial vehicle brake blocks and linings are then wrapped and packed into sealed containers.

4.338 The potential for exposure includes opening and emptying chrysotile bags into the mixer, the moulding and finishing processes, and handling of damaged bags containing raw chrysotile. The workforce amounts to a few hundred workers, and the maximum exposures per employee vary from minimal, to the largest group involved in the processing operations. Airborne asbestos fibre levels are assessed by personal monitoring. About 84 per cent of 461 samples between 1992 and 1997 were < 0.1 f/ml; 10 per cent = 0.01–= 0.2 f/ml; 6 per cent were = 0.02–< 0.5 f/ml, and < 1 per cent were = 0.5 f/ml. The manufacture of compressed asbestos fibre sheeting — most for export and the remainder processed into finished cut gaskets for industrial applications is also a closed process carried out under similar conditions to the manufacture of friction products (please see NICNAS 99, pp 32-34). A total of 232 personal samples between 1991 and 1996 showed similar low airborne fibre

concentration (58 per cent < 0.1 f/ml and only one sample = 0.5 f/ml). Static samples recorded during guillotine and trimming activities were all = 0.05 f/ml.

5.339 Although controlled use of this type is feasible for these manufacturing processes, and for disposal of materials left over (e.g. empty polythene bags), it is my perception that, historically and in reality, it is almost impossible to extend analogous controlled and regulated use to the end-users of asbestos products such as workers involved in building construction and demolition (e.g. builders' labourers, carpenters, electricians, painters, plasterers and plumbers), or to individuals who carry out maintenance or renovation work on their own homes, or to brake mechanics (please see AMR 99). This is because these groups add up to a large population of disparate and varied workers; many such individuals work for small businesses or are self-employed, so that it is difficult or impossible to extend controlled use or training to all of them.

5.340 Some of the supporting documentation submitted to the WTO refers to ILO recommendations, but it is also worth emphasizing that prohibition or regulation of asbestos-containing products varies from one nation to another, with different upper limits of airborne fibre concentrations (e.g. < 1 f/ml or < 0.1 f/ml). These are summarized in Tables 27 and 28 and Appendix 7 in NICNAS 99.

5.341 From the literature cited throughout this report and the reasons discussed, it is my perception that broad agreement exists among experts that controlled use of chrysotile (or other varieties of asbestos) is not a feasible option in the real world for certain worker groups, notably those involved in construction trades (e.g. see EHC 203).

Dr. Infante:

5.342 I am not familiar with any "agreed upon" methodology applicable to chrysotile-cement products and other high-density chrysotile products related to "controlled use" in the sense that these products could be used without harm to human health. Perhaps the consensus is that when using asbestos, the exposure should be controlled, and various countries have developed programmes or standards that recommended, or require, specific engineering controls, work practices, training and education and personal protective equipment to control exposures to asbestos to the extent feasible. This, to me, is different from the concept of "controlled use" in the dialogue being used by Canada, which seems to imply that using or manipulating asbestos or asbestos containing products can be done in such a manner that people are not exposed, or that the risk from such exposure is *de minimus*.

5.343 I also am not aware of international standards related to "controlled use" of asbestos products. What you may be referring to here are recommendations from international organizations, or recommendations or regulations (in the context of being enforceable by law) from various countries. These documents, however, should not be considered as international standards that lead to the "controlled use" of asbestos. For example, in 1994, the United States, promulgated a new standard for occupational exposure to asbestos which required the permissible exposure limit (PEL) to be no higher than 0.1 f/cc as an 8-hour time-weighted- average (TWA). In the opinion of OSHA, workers exposed to this PEL over an occupational lifetime (45 years) are still at a significant risk of developing asbestos related diseases. Therefore, the Agency included in the standard several provisions ancillary to the PEL. One could argue that the PEL plus the ancillary provisions constitutes one of the best examples of the concept of "controlled use" of asbestos as I understand it in the concept being brought forward by Canada. Yet, in the United States, the PEL for asbestos as well as the ancillary provisions that include training and education about the health hazards of exposure, work practices, requirements for personal protective equipment, medical surveillance, etc. are not complied with for various reasons in a large number of workplaces. Based on the observations of violations of several provisions of the asbestos standard in the United States, discussions with occupational safety and health personnel from other countries and my review of the literature, it is my opinion that a controlled use concept for chrysotile asbestos is not realistic in workplace situations. It would be much less realistic as applied

to non-occupational situations where individuals would make repairs involving the manipulation of asbestos products in their homes.

5.344 I am not aware of any international standard that embodies "controlled use" of asbestos in the context that the manipulation of asbestos, or asbestos containing products will result in exposures that will not result in harm to many of those exposed. Furthermore, it should be recognized that programmes to control asbestos in many countries are "agreements" and as such, they are not enforceable by law. Even more disconcerting is the observation that countries like the United States that promulgated stringent requirements to control asbestos find that their standards are often violated. A general downfall with the concept of "controlled use" is that it relies upon human behaviour, which cannot be controlled in too many situations. Hence, it is unreliable.

Dr. Musk:

5.345 In my opinion "controlled use" of asbestos is theoretically possible but not practically feasible. The second half of the question is not within my area of expertise.

5.(b) To what extent is controlled use feasible in terms of the training of those involved in such use, implementing process changes, monitoring, etc.? Have there been studies conducted in this regard and what do they show?

Dr. de Klerk:

5.346 This is definitely outside my area of expertise.

Dr. Henderson:

5.347 In theory, training of specific workers (e.g. products manufacture) and some other personnel in the controlled use of chrysotile ought to be feasible (as for other potentially hazardous materials such as radioactive materials used in nuclear reactors). As a matter of common sense, training is most likely to be effective when there is a small and cohesive workforce using materials that are not accessible to most other workers who have limited or no training, and when the workers have a clear understanding of the hazards and risks of the materials handled.

5.348 However, training of this type becomes less feasible or impossible in practice when there is a large and non-cohesive workforce and when there is general accessibility to the materials in question (e.g. builders' labourers, carpenters, electricians, plumbers and so forth at building sites, and brake mechanics).

5.349 Even so, I am sceptical about the consistent and universal effectiveness of training programmes in the real world, even when the workforce is small and cohesive, and involved in the handling of materials that present a clear hazard (e.g. radioactive isotopes). For example, following the recent mishap at a nuclear reactor in Japan, a BBC report broadcast by ABC News Radio on Sunday 31 October 1999 pointed out that the workers at the Japanese nuclear plant had been poorly trained, with a poor understanding of the risks.

5.350 An analogous report was printed in *The Guardian Weekly* (October 28-03 November 1999, p. 9):

> "Britain's key nuclear warheads factory this week admitted to more than 100 breaches of safety in the past year but ... the Director of Communications at the Atomic Weapons Establishment in Aldermarston ... branded claims that only luck had prevented an accident worse than that in Japan as 'irresponsible scaremongering'. ... The Plant's emphatic denials came after The Observer newspaper

> published details of a leaked report highlighting more than 100 dangerous incidents since September last year. ... Among these were eight breaches of the 'criticality' rules ... and eight instances of environmental contamination outside the site. There were also eight occasions when materials — including plutonium — were incorrectly packaged or labelled, and 19 highly serious health and safety incidents, including all fire-fighting pumping appliances being unfit to service. ... The lead-in of the catalogue of breaches comes as the environment agency prepares to decide whether to prosecute the privatised Aldermarston for dumping tritium, a radioactive substance, in a stream from which Reading's drinking water is sourced ... it also follows the imposition of a £22,000 fine on the company after two workers breathed in radioactive particles from plutonium that had escaped from a laboratory in August last year. ... The establishment insisted it was 'regulated up to the eyeballs' by external agencies and said it was penalised for its 'openness and transparency' in reporting breaches ...".

Dr. Infante:

5.351 Training is beneficial in reducing worker exposure to toxic substances in some circumstances, but minimizing exposure to asbestos to the extent that a significant amount of disease will not occur, is not one of these circumstances because of the extent of the training provisions necessary to reduce exposures, the lack of ability to reach all of the potentially exposed populations, and the wide-spread use of chrysotile asbestos. Training would be relatively better achieved in the manufacturing sector where the employed population is relatively more stable as compared to the construction sector wherein many workers are transient employees. Because of the transient nature of the workforce and the cost involved to train workers, there is a tendency to not train those who will only be employed for short periods of time.

5.352 In terms of studies related to the feasibility of using chrysotile asbestos in a "controlled manner" OSHA health compliance data may offer some insight. The United States has an asbestos standard that includes training requirements that are enforceable by law, and violations of which are punishable by monetary penalties. Yet, improper work practices (presumably a reflection of lack of training) and violations of the permissible exposure limit continue to be identified. Since 1980, OSHA compliance officers have identified almost 14,000 violations among establishments for failure to comply with provisions of its asbestos standard. During the recent 3-year period of 1996-98, over 4,000 violations have been cited. Because of the small number of OSHA compliance staff in relation to the number of facilities in the United States, it has been estimated that compliance officers are able to visit industrial workplaces an estimated one time in every 84 years. Thus, the non-compliance with provisions of the United States asbestos standard as identified by its compliance officers, represents a "tip of the iceberg" in identifying non-compliance with provisions of the standard that are thought necessary to control asbestos exposure in the workplace. As difficult as this situation is in the occupational setting, I am inclined to believe that training leading to "controlled use" of chrysotile would be even more difficult to achieve outside of the occupational setting.

Dr. Musk:

5.353 As above in my opinion controlled use of chrysotile is theoretically feasible but probably not practically possible. The second question part of the question is not in my area of expertise.

5.(c) Can controlled use, when properly applied, reduce exposure levels to chrysotile fibres to below 0,1 f/ml[26]? Can controlled use provide the assurance that there will be no peaks above this figure for any type of use of high-density chrysotile products? For workers or other persons exposed to chrysotile asbestos at this level, can you quantify the risk?

[26]Maximum exposure limit which was authorized in France before the ban.

Dr. de Klerk:

5.354 See Table in paragraph 5.197 above.

Dr. Henderson:

5.355 When properly applied, controlled use in specific situations (e.g. friction products manufacture) can reduce airborne chrysotile concentrations to < 0.1 f/ml for most of the time: e.g. as mentioned in my answer to Question 5(a), at the Australian friction products factory in Victoria, 84 per cent of 461 samples (1992-1997) showed an airborne fibre concentration < 0.1 f/ml; however, the remainder showed airborne fibre levels above this figure, although the concentrations were still low. This also demonstrates that one cannot always guarantee that fibre concentrations will never exceed 0.1 f/ml, even in highly regulated circumstances such as the manufacture of high-density chrysotile products. For the reasons discussed in preceding sections of this report, it is not possible to quantify the risks (e.g. lung cancer or mesothelioma) from occasional peak exposures > 0.1 f/ml, because these risks necessarily depend upon fibre type, the intensity of the exposure, and the frequency and duration of exposure; in addition, quantification of the risk necessarily involves extrapolation from a linear dose-response model, which is the subject of dispute, because there are no observational data on risks from low-level chrysotile exposure.

5.356 Tables 12 and 13 above (see my response to Question 1(d)) give some estimates of lung cancer and mesothelioma risk at various levels of exposure to chrysotile.

5.357 Finally, occasional bursts or peak concentrations of fibres can be released from buildings by catastrophic and uncontrollable events that include, for example, destruction by fire [244-247], earthquake, or explosions [246], including war (e.g. bombing of cities). Although disasters like these are infrequent — and the fall-out from fires is probably of little consequence to nearby residents or the public in general [247] — they pose a potential health risk for some occupational groups likely to encounter asbestos fall-out and debris more often, such as fire fighters and those involved in clean-up operations.

Dr. Infante:

5.358 Since I believe that "controlled use" is a misnomer in relation to potential for exposure to asbestos, I prefer to answer this question in the context of whether a stringent standard for the control of asbestos in the workplace can reduce exposures to below 0.1 f/cc. In many situations, when standards are properly applied, or adhered to, it is possible to maintain exposures below 0.1 f/cc. However, in many situations today, even in the presence of a stringent standard to control exposure, the 0.1 f/cc level for asbestos is exceeded, specified work practices and housekeeping provisions that are required to reduce exposures are not adhered to, communication of hazards is not provided, and appropriate personal protective equipment is not used. As a result, both average exposures and peak exposures above 0.1 f/cc will occur. With regard to high density chrysotile products specifically, over 2,000 violations of the OSHA asbestos standard have been identified in standard industrial classification (SIC) sectors where potential for exposure to chrysotile asbestos would occur, namely: wrecking and demolition work, asbestos products manufacturing, roofing, siding and sheet metal work, manufacturing of gasket, packing and sealing devices and automotive repair shops.

5.359 In addition to the above examples, in my opinion, many work practices are simply not good enough in some situations to reduce exposures below 0.1 f/cc. For example, even though asbestos cement products may be "pre-sized" in manufacturing, cement sheets need to be modified, drilled, cut and ground to fit them into specific areas. Although wetting may be used in some of these situations for cutting asbestos cement, the material dries and the dust becomes airborne resulting in uncontrolled asbestos dust being released into the work environment. Even in such situations where workers are

provided with respirators to prevent exposure, the respirators supplied for the situation are often the wrong ones. In addition, respirator effectiveness depends upon how well the masks fit the face, how often they are replaced, cleaned and repaired and how well the employees are trained to select, clean and repair their respirators. In spite of rules requiring good respirator practices, employers often simply do not comply with the regulations.

5.360 In a more general sense, non-compliance with regulations that could assist in the control of asbestos to exposure levels below 0.1 f/cc is often the result of human error, (not recognizing that asbestos is present, not understanding the requirements to protect workers from exposure in specific situations), wilful non-compliance, poor judgement, accidents, inability to adequately reach the potentially exposed population with proper training and education of the health hazard, etc. Based on my experience in occupational health and the results of OSHA compliance data and the literature, it is my opinion that it is impossible to use asbestos and assure that exposures can be kept below 0.1 f/cc in a great number of situations.

5.361 Regarding health risks from chrysotile exposure to 0.1 f/cc, as mentioned in my answer to Question 3, the risk assessment conducted by Stayner et al. (1997) based on the study by Dement et al. (1994) provides a good estimates of disease risks from exposure to chrysotile asbestos which are not much different from those provided from other studies. The analyses estimate that exposure to 0.1 f/cc of chrysotile asbestos for an occupational lifetime of 45 years, e.g., 4.5 f/cc-years of cumulative exposure, results in five extra deaths from lung cancer and two extra deaths from asbestosis per 1000 workers. The analysis did not include mesothelioma because there were too few deaths from this cause in the study to provide estimates of risk. I have not seen risk assessments for mesothelioma based on chrysotile exposure. Thus, I cannot provide a quantitative estimate of risk for this cause of death.

5.362 Of these diseases, the risk of developing asbestosis is most likely underestimated. This underestimation is a reflection of the fact that most risk assessments are based on mortality studies and individuals diagnosed with asbestosis often die from other causes. For example, in the study by Finkelstein (1982), of 24 individuals with certified asbestosis, who had died, 14 (58 per cent) died from lung cancer or mesothelioma and three (12.5 per cent) died from ischemic heart disease. Thus, 70 per cent of the individuals with asbestosis who died would not have been identified from a mortality study of this population of exposed workers. Asbestosis may also be underestimated in a study because it may be confused clinically with other non-malignant lung diseases. Therefore, the risk of death from asbestosis based on mortality studies may usually be underestimated. I am not familiar with quantitative estimates of risk from other pathologies related to chrysotile asbestos.

Dr. Musk:

5.363 The question is not within my area of expertise except that risks from exposure could be calculated if exposure levels are known.

5.(d) Is it possible to control the risks to human health presented by exposure to high-density chrysotile products, in particular chrysotile-cement, throughout the life-cycle of the product? Is controlled use a feasible and practicable option in everyday life for workers who are exposed occasionally to potentially high levels ("peaks of exposure") of chrysotile asbestos (such as plumbers, electricians, maintenance, repair, insulation, demolition, waste management and "handyman" type persons)?

Dr. de Klerk:

5.364 See my response to Question 5(a).

Dr. Henderson:

5.365 From my perspective, these questions are crucial to the dispute before the WTO. In my opinion, the answer to both questions is NO. I do not see how asbestos in place — or existing chrysotile products — can be controlled at every point of end-use. For example, EHC 203 indicates repeatedly that exposure is most likely to affect workers involved in building construction or demolition; this is because of the large number of these workers involved in myriad different tasks; in addition, these various workers represent a non-cohesive workforce, many self-employed or working as part of "small business".

5.366 My perception is also based on studies such as that reported by Kumagai et al. [4] on Japanese workers involved in the repair of asbestos-cement pipes, where fibre concentration for fibres > 5 µm in length ranged from 92 f/ml inside the hole where this work took place (range 48-170 f/ml) and up to 15 f/ml outside the hole; the final sentence of the Abstract for this Report indicates that only about 18 per cent of the workers used a protective respiratory device. In addition, a survey in Finland found occasional high fibre concentrations inside personal protectors during asbestos removal work, suggesting that these devices are not always effective.

5.367 Another factor that merits consideration in some societies such as Australia, is poor worker compliance with controls. For example, I am aware of non-compliance in the use of protective equipment (despite penalties), because respiratory protective devices may be cumbersome and uncomfortable — especially in hot climates like Australia — with skin irritation from sweat accumulating within them.

5.368 From the literature cited throughout this report and the reasons discussed, it seems clear that there is a broad consensus among experts that controlled use of chrysotile (or other varieties of asbestos) is not feasible in practice for certain worker groups, notably those involved in construction trades (e.g. see EHC 203).

Dr. Infante:

5.369 As mentioned in my responses to several previous questions, in my opinion, it is not possible to control the risk to human health posed by exposure to chrysotile asbestos throughout its life cycle. "Controlled use" is not a feasible and practical option in everyday life for workers. While "controlled use" is relatively more achievable in the manufacturing sector, violations of regulations enforceable by monetary fines still occur. In the construction sector, control of exposure to asbestos is much more difficult to achieve as compared to manufacturing. Workers who have jobs as plumbers, electricians, maintenance personnel, repairmen, insulators, demolition, waste management and handymen will most likely experience intermittent peak exposures to asbestos. These exposures result from lack of awareness of the hazard, lack of recognition of the hazard, lack of personal protective equipment, lack of training on the maintenance of the protective equipment, etc. as mentioned in responses to other questions above.

Dr. Musk:

5.370 In my opinion controlled use is probably not practically possible but this is not an area of my expertise.

5.(e) Is it possible to control the risks to human health presented by exposure to high-density chrysotile products, in particular chrysotile-cement, in non-occupational circumstances, such as intervention on these products by private individuals (cutting, sawing, removal, etc.)? Is controlled use a feasible and practicable option for this category of the population?

Dr. de Klerk:

5.371 See my response to Question 5(a).

Dr. Henderson:

5.372 As a follow-on to my answer to the preceding question, my answer to both of these questions is also NO. However, the risks from occasional or infrequent interventions on chrysotile-only products (e.g. by home "handymen") - although not quantifiable because of absence of data - must be very small for lung cancer and mesothelioma, and non-existent for asbestosis.

Dr. Infante:

5.373 As difficult as it is to control exposure to chrysotile asbestos during intervention with cement products in the occupational setting, it is much more so in non-occupational circumstances because there is no effective means of identifying the potential population at risk. As a result, there is a lack of awareness of the hazard, lack of recognition of the hazard, lack of personal protective equipment, lack of training on the maintenance of the protective equipment, etc. as mentioned in responses to other questions above. Therefore, in my opinion, it is not possible to limit exposure in such circumstances.

Dr. Musk:

5.374 Controlled use is probably not practically possible, in my inexpert opinion.

Question 6:

The parties disagree as to the relative pathogenicity of chrysotile fibres vs. substitute fibres, in particular cellulose fibres, para-aramid fibres, glass fibres and polyvinyl alcohol (PVA) fibres. Canada considers that, overall, substitute fibres have not been demonstrated to be less toxic than chrysotile fibres, and that, by banning chrysotile, France has replaced the "much studied but nonetheless undetectable risk associated with modern uses of chrysotile with the unknown, and perhaps greater risk associated with the use of substitute fibres" [Premier exposé oral du Canada, paragraph 90]. On the other hand, the European Communities argues that none of the substitute products -fibrous or non-fibrous- for chrysotile, and in particular none of the substitutes for chrysotile-cement, has been classified as a proven carcinogen to humans; hence, overall, substitute products present less of a risk to human health than chrysotile asbestos [see Deuxième soumission écrite, pp. 10-15].

6.(a) Is it correct to argue that non-fibrous substitutes are safe or less hazardous than chrysotile and that concern over potential health risks should be focused on fibrous ones? In this context, could you elaborate upon the "effet fibre" ["fibre effect"] of substitute fibres? What general conclusions can be drawn as to the respirability and biopersistence of substitute fibres?

Dr. de Klerk:

5.375 As outlined above, the pathogenicity of fibres is related to their size, shape, durability and quantity. Thus, all the parts to this question can be answered in the same way. The argument here is whether it is safer to stick with the well-studied chrysotile that has a semi-quantifiable and definite carcinogenic risk, than to use other substances which have the potential to increase risk in an unquantifiable way, ie. the "better the devil you know" principle. For example, para-amid fibres have recently been classified by IARC in Group 3, that is, 'not classifiable as to its carcinogenicity'.

5.376 Substitutes need to be compared to chrysotile in terms of the parameters listed above, namely, size, shape, durability and quantity. These are all properties of fibres and therefore "concern should be focused on fibrous substitutes". Substitute fibres can then be compared with chrysotile on the four parameters. I am inexpert in commenting on the "extent to which hazardous concentrations can be controlled" but it is my understanding that all four substitutes mentioned involve less dusty operations than equivalent ones involving chrysotile. As far as the other three parameters are concerned: all four substitutes except glass fibre produce a larger proportion of non-respirable fibres than chrysotile does, but respirable fibres are similar for all substances and glass fibre is the least durable; all four except cellulose are less durable than chrysotile, but cellulose is much less dusty and has also been in use for a long while without evidence of ill effect.

5.377 On balance, the substitute fibres appear less likely to cause adverse effects (from their fibres) than chrysotile.

Dr. Henderson:

5.378 Current thinking on this issue indicates that the bio-hazards — specifically the carcinogenic risks - of all fibres are determined by the three Ds: dose, fibre dimensions and durability (bio-persistence) [248-250]. Therefore, substitute materials that have engendered most concern are fibrous materials as opposed to non-fibrous substances (non-fibrous materials may or may not show different effects in terms of toxicology, but this discussion focusses on carcinogenic risks). For example, refractory ceramic fibres (RCF) are a cause for concern [251] because they may have dimensions similar to those of the amphibole varieties of asbestos and RCF have been reported to induce mesothelioma in experimental animals.

5.379 In a 1995 review, de Vuyst et al. [248] concluded that:

> "The group of man-made mineral or vitreous fibres (MMMFs or MMVFs) includes glass wool, rock wool, slag wool, glass filaments and microfibres, and refractory ceramic fibres (RCFs). Experimental observations have provided evidence that some types of MMVF are bioactive under certain conditions. The critical role of size parameters has been demonstrated in cellular and animal experiments, when intact fibres are in direct contact with the target cells. It is, however, difficult to extrapolate the results from these studies to humans since they bypass inhalation, deposition, clearance and translocation mechanisms. Inhalation studies are more realistic, but show differences between animal species regarding their sensibility to tumour induction by fibres. Fibre biopersistence is an important factor, as suggested by recent inhalation studies, which demonstrate positive results with RCF for fibrosis, lung tumours and mesothelioma. There is no firm evidence that exposure to glass-, rock- and slag wool is associated with lung fibrosis, pleural lesions, or nonspecific respiratory disease in humans. Exposure to RCF could enhance the effects of smoking in causing airways obstruction. An elevated standard mortality ratio for lung cancer has been demonstrated in cohorts of workers exposed to MMVF, especially in the early technological phase of mineral (rock slag) wool production. During that period, several carcinogenic agents (arsenic, asbestos, polycyclic aromatic hydrocarbons (PAH)) were also present at the workplace and quantitative data about smoking and fibre levels are lacking. It is not possible from these data to determine whether the risk of lung cancer is due to the MMVFs themselves. No increased risk of mesothelioma has been demonstrated in the cohorts of workers exposed to glass-, slag- or rock wool. There are in fact insufficient epidemiological data available concerning neoplastic diseases in RCF production workers because of the small size of the workforce and the relatively recent industrial production" [abstract].

5.380 In a 1999 review published in French, Boillat et al. [250] came to similar conclusions:

> "The group of man-made mineral fibres includes slagwool, glasswool, rockwool, glass filaments and microfibres, as well as refractory ceramic fibres. The toxicity of mineral fibres is determined by several factors such as the diameter (< or = 3-3.5 microns) and the length of the fibres (< 100 microns), their biopersistence, which is much shorter for man-made mineral fibres than for asbestos fibres, their

> physicochemical structure and surface properties, and the exposure level. The chemical composition of the various types of man-made mineral fibres depends directly on the raw material used to manufacture them. While naturally occurring fibres are crystalline in structure, most man-made mineral fibres are amorphous silicates combined with various metal oxides and additives. Observations using intracavitary administration have provided evidence that some types of man-made mineral fibres are bioactive in cellular and animal experiments and may induce lung tumours and mesothelioma. It is difficult to extrapolate these results to humans since they bypass inhalation, deposition, clearance and translocation mechanisms. Inhalation studies show more realistic results but differences are observed between animal species regarding their sensibility to tumours. There is no firm evidence that exposure to various wools is associated with lung fibrosis, pleural lesions or nonspecific respiratory disease in humans. A possible exception may be mentioned for refractory ceramic fibres. A slightly elevated standard mortality ratio for lung cancer has been documented in large cohorts of workers (USA, Europe and Canada) exposed to man-made mineral fibres, especially in the early technological phase. It is not possible to determine from these data whether the risk of lung cancer is due to the man-made mineral fibres themselves, in particular due to the lack of data on smoking habits. No increased risk of mesothelioma has been demonstrated in these cohorts. Epidemiological data are insufficient at this time concerning neoplastic diseases in refractory ceramic fibres" [abstract].

5.381 In one study on RCF, Glass et al. [252] reported that:

> "In recent inhalation experiments conducted with both rats and hamsters ... at the highest dose tested ... there was an increased incidence of tumours in both species. Lower doses were only examined in the rat and at these doses there was no significant excess of lung tumours. Epidemiological investigations of workers engaged in the manufacture of ceramic fibres have shown a small excess of pleural plaques. This phenomenon is being further investigated but could be due to confounding exposures. The populations available for study are small and their exposures fairly short, but it is considered prudent that they should remain under surveillance for some time to come. This is despite the fact that present exposures in the ceramic fibre industry are low (< 1 f/ml) and are being reduced" [abstract].

5.382 Okayasu et al. [253] also found that RCF-1 fibres were less cytotoxic and mutagenic than chrysotile:

> "Cytotoxicity and mutagenicity of tremolite, erionite and the man-made ceramic (RCF-1) fibre were studied using the human-hamster hybrid A(L) cells. Results from these fibres were compared with those of UICC Rhodesian chrysotile fibres. The A(L) cell mutation assay, based on the S1 gene marker located on human chromosome 11, the only human chromosome contained in the hybrid cell, has been shown to be more sensitive than conventional assays in detecting deletion mutations. Tremolite, erionite and RCF-1 fibres were significantly less cytotoxic to A(L) cells than chrysotile. Mutagenesis studies at the HPRT locus revealed no significant mutant yield with any of these fibres. In contrast, both erionite and tremolite induced dose-dependent S1- mutations in fibre-exposed cells, with the former inducing a significantly higher mutant yield than the latter fibre type. On the other hand, RCF-1 fibres were largely non-mutagenic. At equitoxic doses (cell survival at approximately 0.7), erionite was found to be the most potent mutagen among the three fibres tested and at a level comparable to that of chrysotile fibres. These results indicate that RCF-1 fibres are non-genotoxic under the conditions used in the studies and suggest that the high mesothelioma incidence previously observed in hamster may either be a result of selective sensitivity of hamster pleura to fibre-induced chronic irritation or as a result of prolonged fibre treatment. Furthermore, the relatively high mutagenic potential for erionite is consistent with its documented carcinogenicity" [abstract].

5.383 An important consideration is that fibre dimensions for some substitute materials (e.g. fibreglass) can be varied according to the manufacturing processes employed, so that they can be designed to have fibre characteristics and dimensions different from asbestos, or similar to asbestos: as one example, the dimensions of fibreglass can be varied and when implanted into experimental animals, fibres of the "right" size can induce mesothelioma.

5.384 For this reason, testing of substitute materials with fibre dimensions similar to those of asbestos should be carried out before these materials are used in products available to the general

public (e.g. testing for toxicology, clastogenicity, DNA strand breaks, mutagenicity and free radical generation using *in vitro* systems and/or testing *in vivo* — such as the intraperitoneal test in rats) [248, 249, 251-256].

5.385 Nonetheless, it is my perception that lumping all substitute fibres together is as erroneous as lumping amphibole and chrysotile fibres into the same category. For example, RCF are the subject of continuing concern, but other substitute fibres such as cellulose fibres, para-aramid fibres and polyvinyl alcohol (PVA) fibres appear to be different from chrysotile, in terms of fibre dimensions and especially bio-persistence.

5.386 NICNAS 99 summarizes these considerations in the following terms:

> "Any substitution of chrysotile should be with a less hazardous substance. There has been ongoing debate regarding the health effects of alternatives, such as synthetic mineral fibres (SMF), natural organic fibres and synthetic organic fibres.
>
> In general, less data on health effects of alternative materials (in comparison to asbestiform fibres) are available and because of this, it is difficult to make an assessment of the pathogenicity and potential carcinogenicity of many substitutes.
>
> Although not the only determinant of potential pathogenicity, fibre dimensions (length, width and aspect ratio) are considered to be [some] of the most important factors associated with carcinogenic (lung cancer and mesothelioma) potential ... The commonly accepted 'peak hazard' dimensions ... are > 5 µm long (length) and < 3 µm wide (diameter).
>
> The most commonly used alternatives in Australia (and overseas) for friction materials are aramid fibres, attapulgite, fibreglass, refractory ceramic fibres (RCF), semi-metallics, mineral wool, steel wool, cellulose, titanate fibres and wollastonite, and for gaskets are glass fibre, carbon fibre and aramid fibre.
>
> ... It should also be noted that ... differences in fibre length, diameter and surface properties may lead to entirely different toxicological profiles.
>
> A recent report by EC concludes that the available data are generally supportive of the conclusion that PVA, cellulose, p-aramid, glass wool and slag wool are likely to be safer in use than chrysotile. However, RCFs are the subject of ongoing concern ..." [p 125].

Dr. Infante:

5.387 I have not seen any information that indicates that non-fibrous substitutes for chrysotile are carcinogenic, or cause non-malignant lung diseases. I would focus attention on the fibrous substitutes in terms of their ability to reach lung tissue (respirability) and their known toxicity. Clearly, if the substitute fibres are not respirable, there is little concern for their "potential" to cause lung diseases. (Attention would then focus on adverse effects from exposure to the skin and eyes.) If the substitute fibres are respirable, then attention needs to focus on their toxicity relative to that of chrysotile in their ability to cause lung cancer, non-malignant lung diseases and mesothelioma.

5.388 The data I have reviewed in this area of investigation appear to indicate that polyvinyl alcohol fibres (PVA) are mostly in the range of 10-16 microns in diameter and hence are too large to respirable and thus cause lung disease. In terms of biopersistence, if they were respirable, they would degrade very slowly. Para-aramid fibres are also generally 10-12 microns in diameter and they also would have little chance of being respired. These fibres, however, contain fibrils of about 0.2 microns in diameter that can be liberated with high energy input and they would be respirable. P-aramid fibrils greater than 5 microns in length are less biopersistent than chrysotile fibres greater than 5 microns in length (Searl, 1997). Data for dimensions of cellulose fibres show a median length and diameter of about 7.5 and 1.50 microns, respectively, which indicates that they are in the respirable range (Muhle

et al. 1997). In terms of biopersistence, cellulose fibres had a mass half time in the rat lung of 72 days and bioaccumulated in the lungs. Data on the distribution of glass fibres indicates that the majority are in the respirable range, but the fibre size distribution of glass filaments indicates that a small portion are in the respirable range. Glass fibres are less biopersistent than chrysotile fibres. In general, in terms of the combination of respirability and biopersistence, with the exception of cellulose fibres, it appears that the substitute fibres would have less bioaccumulation in the lung than chrysotile fibres because they are either less respirable, or they are not as biopersistent.

5.389 The role of biopersistence in relation to toxicity is complicated. Chrysotile fibres are less biopersistent than amphibole fibres, yet experimental data demonstrate a similar potency for lung cancer, mesothelioma and fibrosis.

Dr. Musk:

5.390 I agree with the Canadian argument philosophically. However there is no evidence that I know of carcinogenicity of substitutes in animal studies and only rockwool has been associated with increased lung cancer risk in epidemiological studies.

6.(b) To what extent do physical characteristics and chemical properties of substitute fibres determine their toxicity? Is it correct to say that man-made fibre substitutes are superior to natural fibre ones in terms of the extent to which exposure to hazardous concentrations can be controlled during the various stages of production? Is your opinion based on one or more of the following evidence: (i) chemical/physical characteristics of the substitute fibres, (ii) epidemiological data, (ii) in vitro evidence, (iv) in vivo evidence?

Dr. de Klerk:

5.391 See my response to Question 6(a).

Dr. Henderson:

5.392 These questions are covered to a large extent in my answer to the preceding question. Again, dose, fibre dimensions (including surface chemistry) and bio-persistence appear to represent the properties that determine the toxicity and carcinogenicity of fibres of any type. The issue of controllability during various stages of production is an engineering and industrial question, and falls outside my expertise.

5.393 My opinions concerning the potential bio-hazards of these fibres are based on the physical characteristics of the substitute fibres, *in vivo* evidence (tumour induction in experimental animals) and *in vitro* studies (mutagenicity analogous that reported for other known carcinogens). To the best of my knowledge, there are no large-scale epidemiological studies on cellulose fibres, para-aramid fibres or PVA fibres; two large epidemiological investigations on slag wool fibres in both Europe and the United States did show an increase in the relative risk for lung cancer among the production workers, but this effect may have been explicable by other confounding factors involved in the manufacture of these materials.

Dr. Infante:

5.394 As a matter of general toxicology, I would focus concern on the "potential" for adverse health effects from any fibrous material of dimensions and aerodynamic diameter that will result in its being respirable. This is discussed in my response to Question 6(a) above. The toxicity of the substitute fibres and whether that information was determined on the basis of epidemiological or toxicological evidence is discussed in Question 6(c). The role of the chemical properties of fibres to induce cancer

is not clear to me. The nature of the production process makes the substitute fibres more amenable to control than asbestos fibres.

Dr. Musk:

5.395 I do not know: but it is my broad understanding that the physical and chemical properties of the substitute fibres suggests less risk of disease.

6.(c) The parties focus part of their arguments on cellulose fibres, para-aramid fibres, glass fibres and polyvinyl alcohol (PVA) fibres. What evidence exists with respect to the toxicity and health risks of these substitutes? Does the existing evidence suggest that these products are less/equally/more toxic than chrysotile asbestos fibres?

Dr. de Klerk:

5.396 See my response to Question 6(a).

Dr. Henderson:

5.397 In experimental studies on para-aramid fibres in comparison to chrysotile, Warheit et al. [12, 257] found that p-aramid is bio-degradable in the lungs of exposed rats, with faster clearance than long chrysotile fibres which showed greater bio-persistence. In their 1996 study, these authors [12] found that:

> " ... p-aramid is biodegradable in the lungs of exposed rats; in contrast, the clearance of long chrysotile fibres was slow or insignificant, resulting in a pulmonary retention of long chrysotile asbestos fibres. The dimensional changes of asbestos fibres as well as the pulmonary cell labelling data indicate that chrysotile asbestos fibres may produce greater long-term pulmonary effects when compared to inhaled para-aramid fibrils" [abstract].

5.398 The present status of knowledge has been summarized by Harrison et al. [19] in a recent review of the comparative hazards of chrysotile and its substitutes:

> "There are now practicable substitutes for the major remaining uses of chrysotile. Although lack of a full health and toxicological data set precludes a comprehensive assessment of the safety of substitute fibers, the application of basic principles of fiber toxicology enables a pragmatic decision to be made on the relative safety of potential substitutes. Our judgement is based on relative considerations of the intrinsic properties of fibers, on the pathogenicity of chrysotile in comparison with that of substitute fibers, and on the potential for uncontrollable exposures. The three parameters of dose, dimension (especially diameter), and durability are key to determining the differential hazards. Due consideration of these factors leads us to the following conclusions regarding chrysotile and its main substitutes.
>
> Chrysotile per se can cause lung cancer and asbestosis; it is less clear that chrysotile alone can cause mesothelioma in humans, and indeed it may not, whereas tremolite and other amphiboles certainly can do so. There is no definitive evidence for a threshold exposure level for lung cancer induction, although some studies suggest that a threshold does exist.
>
> The intrinsic hazardous properties of chrysotile can never be 'engineered out', and the potential for harm will always remain. Prevention of ill health will thus always rely on the control of exposure, something that history has shown cannot be guaranteed.
>
> Unlike chrysotile, substitute fibres can often be designed or selected to have particular characteristics. Criteria for the substitution of asbestos by other fibers include a) the substitute fibers are not in the respirable range, do not readily fibrillate, and/or are less durable than chrysotile; b) other materials that must be incorporated into the replacement product do not, in combination with the replacement fiber,

produce more harm overall than chrysotile alone; c) the replacement product has an equivalent or acceptable performance; and d) substitution would result in overall lower fiber exposures during manufacture and use and disposal, taking into account likely exposures. The same general principle can be applied to substitute fibers others than those considered here.

We judge that PVA fibers will pose less risk than chrysotile because they are generally too large to be respirable, do not fibrillate, and the parent material causes little or no tissue reaction. Aramid fibers have a reduced potential for exposure when compared to chrysotile because they are generally of high diameter and the production of respirable fibrils is energy intensive. The fibrils are less pathogenic than chrysotile, are less biopersistent, and are biodegradable. Cellulose has the benefit of long experience of use in a variety of industries without having raised significant concern. The potential for the generation of respirable fibers seems to be less than is the case for chrysotile, although fibrillation is possible. Cellulose is durable in the lung, and its biological properties should therefore be investigated further. However, exposure levels for current uses are low, and it is biodegradable in the environment.

We believe that the continued use of chrysotile in asbestos-cement products is not justifiable in the face of available and technically adequate substitutes. Likewise, there seems to be no justification for the continued residual use of chrysotile in friction materials" [pp 610-611].

5.399 From known past uses of asbestos and surveys of current uses in Australia, it is evident that alternatives have replaced chrysotile to a large extent for the following products [NICNAS 99, p 111]:

"Products where chrysotile use has been completely replaced:

- Cement sheeting, tubes and piping.
- Roofing tiles.
- Textiles.
- Fibre insulation.
- Railway brake blocks.
- Brake disc pads in new automotive vehicles (only 1 new vehicle model was identified as being supplied with asbestos pads in Australia).

Products where a major proportion of chrysotile use has been replaced:

- Clutch facings (in automotive vehicles and industrial machinery e.g. tractors, centrifuge drives).
- Brake disc pads (in older taxi and courier vehicles, and industrial machinery).
- Gaskets, such as spiral wound and head gaskets.
- Washers.
- Packing material.
- Rotor blades (e.g. in high vacuum pumps)".

5.400 It is notable that chrysotile is no longer used for brake linings in new passenger cars produced in Australia by most manufacturers, having been replaced by substitute materials: NICNAS 99 comments that:

"Out of 26 companies, 25 stated that they are using non-asbestos original equipment in all current models. One company (Ford Motor Australia) reported that they are still using asbestos parts in two current models: asbestos head gaskets for the Econovan and asbestos rear brake linings for the Ford utility. Ford Australia introduced non-asbestos components for their most popular models (e.g. Laser, Falcon and Fairlane) between 1989 and 1995. Other current models manufactured by Ford have been asbestos-free since their introduction. ... Asbestos parts are imported by 6 of the 26 companies (BMW, Ford, Mazda, Mitsubishi, Nissan and Toyota) with five companies using asbestos parts for superseded vehicles and one company (Ford Australia) using asbestos parts in superseded and current models ... the majority of the vehicle manufacturing companies stated that they have had policies in place in regard to not using asbestos components in new vehicles for the last 5 to 10 years" [p 22].

5.401 This trend to use of brake linings free of asbestos is shown in the following Table 15 — in comparison to the usage of asbestos brake linings — between 1994 and 1998 (asbestos-containing brake linings appear to be used primarily on older and superseded vehicle models).

TABLE 15: IMPORTS OF ASBESTOS AND NON-ASBESTOS BRAKE LININGS INTO AUSTRALIA, 1994-1998

	Number of Articles				
Import	**1994**	**1995**	**1996**	**1997**	**1998 (Jan-Aug)**
Asbestos brake linings, passenger cars	492,295	47,735	43,087	771,182	(548,692)
Non-asbestos brake linings, passenger cars	70,109	321,472	485,812	2,084,963	(4,057,143)

Source: NICNAS 99.

Dr. Infante:

5.402 There is no information to my knowledge that cellulose fibres, para-aramid fibres or ploy vinyl alcohol (PVA) fibres are carcinogenic. Cellulose fibres have not been studied experimentally for carcinogenicity. It is noteworthy, however, that cellulose has been used in the paper industry for hundreds of years and to date an elevated risk of death from lung cancer and mesothelioma has not been observed. Excess incidences of pharyngeal and/or laryngeal cancers were reported in two studies, but these observations have not been corroborated in other studies (IARC, 1987). Wood dust is associated with sino-nasal cancer, but not with lung cancer or mesothelioma. A relatively greater risk appears to be associated with hard woods as compared to soft woods, which suggests that the cellulose may not be the primary factor in the induction of these cancers. Workers exposed to cotton dust also do not demonstrate an excess of lung cancer or mesothelioma even though they develop byssinosis. The debate as to whether this disease is due to cotton dust per se, or to contaminants of the cotton fibre, however, is not resolved.

5.403 Para-aramid fibrils have been studied for carcinogenicity in experimental animals by inhalation and by intra-peritoneal injection. No cancer response was observed. As concluded by IARC (1997), there is inadequate evidence for the carcinogenicity of para-aramid fibrils in experimental animals. The carcinogenicity of para-aramid fibrils has not been evaluated in humans. Likewise, IARC (1987) concluded on the basis of its review of animal cancer tests that there is no evidence for the carcinogenicity of PVA fibres. PVA fibres have not been evaluated for carcinogenicity in humans. It is also my opinion that there is no evidence that these fibres present any risk of cancer to humans.

5.404 With regard to glass fibres, IARC (1988) concluded there was sufficient evidence for the carcinogenicity of glass wool in experimental animals and that there was inadequate evidence for the carcinogenicity of glass wool to humans. Subsequent to the IARC (1988) review, my colleagues and I have reviewed the toxicological and epidemiological studies related to exposure to glass fibres. In our opinion, there is conclusive evidence from implantation and inhalation studies that glass fibres are carcinogenic in experimental animals (Infante et al. 1994). Studies of workers exposed to glass fibres also demonstrate a significantly elevated risk of death from lung cancer. It is our interpretation of these studies that employment in the manufacturing of glass fibres carries with it an elevated risk of death from lung cancer. Is it proven beyond a doubt through epidemiological study that fibrous glass is a human carcinogen? In my opinion, it is not. However, given the positive animal cancer test results, knowledge that these fibres can be inhaled and retained in the lungs, evidence that workers

employed in the manufacturing of these fibres die at a significantly elevated rate of lung cancer, it is my opinion that it is more likely than not that glass fibres are carcinogenic to humans and that employment in this industry carries with it an elevated risk of death from lung cancer.

5.405 It is also my opinion that glass fibres are not as potent as chrysotile asbestos in causing disease. With regard to the capability of glass fibres to cause lung cancer, I have previously published the opinion that on a fibre-per-fibre basis, glass fibres may be as potent or even more potent than asbestos in causing lung cancer. This opinion was based on epidemiological studies which generally demonstrated 10 per cent to 20 per cent elevation in the relative risk of lung cancer (the 1987 study of Canadian workers by Shannon demonstrated a 2-fold risk) as a result of exposures to glass fibres that were reported to be fairly low. Within the past year, however, I have had the opportunity to discuss occupational exposures during glass fibre manufacturing with workers formerly employed at the Canadian facility that manufactured fibrous glass. According to several workers, they were also exposed to crystalline silica many times over the permissible limit through the dumping of sand into the hopper that was used to feed the furnace for melt down. They were exposed to asbestos that lined the furnace when they removed the insulation from the oven doors by hand, or chiselled it away in the absence of respiratory protection; they would then add water to asbestos fibre to make "asbestos mud" that was applied to the oven doors by hand, or by trowel. Workers were so uninformed of the hazard that they sometimes would throw "asbestos mud balls" at each other. The workers at this facility were also exposed to phenol formaldehyde resin that was used as a binder for the glass fibres; they were also exposed to tar that was applied to paper that was then applied to the glass fibre pack. Exposures to glass fibres only is mentioned in the study of these workers that was published by Shannon (1987).

5.406 Furthermore, there is less evidence from epidemiological studies that exposure to glass fibres is associated with a pneumoconiosis as compared to the data for chrysotile asbestos exposure and asbestosis. There is no evidence that exposure to glass fibres is associated with mesothelioma. For the few cases of mesothelioma that have been identified among workers exposed to glass fibres to date, there is claim that they also had been exposed to asbestos fibres. Therefore, it is difficult to attribute these cases of mesothelioma to the glass fibre exposures. Therefore, the totality of disease related to chrysotile asbestos exposure would be greater than that related to a similar amount of exposure to glass fibres.

5.407 In conclusion, only one of the fibres (glass fibres) that may play any significant role in substitution for chrysotile asbestos demonstrates evidence of being carcinogenic. Data for the total toxicity related to these fibres, however, is less than that for chrysotile asbestos fibres. Cellulose fibres have not been tested for carcinogenicity in experimental animals, but epidemiological studies of workers exposed to cellulose in three separate industries, i.e., furniture manufacturing, cotton textile manufacturing and the paper products industry, have not demonstrated an elevated risk of contracting lung cancer or mesothelioma. Regarding non-malignant lung disease among cotton dust exposed workers, it is not known whether the cotton dust per se, or contaminants of the cotton fibre, are responsible for the byssinosis observed in these workers.

Dr. Musk:

5.408 I understand that substitutes have been shown to be less toxic in animals.

3. Summary Comments by Dr. Henderson

5.409 In-place asbestos is widely distributed in industrialized societies and much includes mixtures of chrysotile and amphiboles — although chrysotile has been the predominant type of asbestos used throughout Western Europe for many years (about 94-97%).

5.410 Lung cancer and mesothelioma are the most important bio-hazards from asbestos in place and the continued use of asbestos.

5.411 Because of the prolonged lag-time between exposure and the subsequent development of either lung cancer or mesothelioma, most mesotheliomas in the 1990s and beyond can be attributed to exposures sustained decades before; the mesothelioma "epidemic" predicted for Europe over the next three decades can be attributed to exposures before, during and after the 1960s and 1970s, especially to one or more of the amphibole varieties.

5.412 For the amphibole forms of asbestos and mixtures of asbestos types, a linear dose-response relationship has been found at high levels of exposure; a dose-response relationship with an increase of the relative risk of mesothelioma to > 2.0 has also been observed at low levels of exposure, in the order of 0.5-1.0 fibre-year (which overlaps with non-occupational environmental exposures). No lower threshold dose for mesothelioma induction has been delineated for the amphiboles.

5.413 Chrysotile also has the capacity to induce mesothelioma, although it is less mesotheliomagenic than the amphiboles (my estimate is $1/10^{th}$-$1/30^{th}$).

5.414 Commercial Canadian chrysotile on average contains trace quantities of tremolite, including fibrous tremolite (< 1%).

5.415 Tremolite — a non-commercial amphibole — also has the capacity to induce mesothelioma.

5.416 The carcinogenicity of Canadian chrysotile may be attributable to the trace tremolite content, but it is not possible to separate the dose-response effects for the chrysotile and the tremolite.

5.417 At high levels of exposure to Canadian chrysotile, a linear dose-response relationship has been observed.

5.418 To the best of my knowledge, there are no epidemiological or observational data on dose-response effects of chrysotile only at low levels of exposure.

5.419 No lower threshold dose for the carcinogenic effects of chrysotile has been identified (EHC 203).

5.420 To the best of my knowledge, there are no observational data on the potential carcinogenic effects of inhaled chrysotile when superimposed upon a pre-existing burden of amphiboles ± chrysotile in lung tissue.

5.421 Although the amphiboles are far more potent than chrysotile for mesothelioma induction, this differential in carcinogenicity may be less obvious or absent for lung cancer induction, but this is still the subject of some dispute; chrysotile is associated with a low risk of lung cancer among Canadian chrysotile miners and millers, but the highest risk for lung cancer induction has been observed for South Carolina asbestos textile workers who used Canadian chrysotile almost exclusively.

5.422 A linear dose-response relationship has also been observed for the risk of lung cancer versus cumulative asbestos exposure. Although some authorities favour a linear no-threshold model for lung cancer induction, others suggest that a threshold may exist, but this has not been delineated in numerical terms.

5.423 In contrast, asbestosis is a dose-dependent non-cancerous disorder, with clear evidence of a threshold effect, although the threshold may be lower than previously supposed, at least for

histological asbestosis; there is no risk of asbestosis at low levels of chrysotile exposure.

5.424 Although reduction of airborne asbestos fibre concentrations in the mining and manufacturing industries has been achieved, it is too early to evaluate the effects of these reduced exposures, because no epidemiological data are available; however, with reduction of cumulative exposures, a reduction in the incidence of both asbestos-related mesothelioma and asbestos-related lung cancer can be expected.

5.425 The risks from low-level occupational exposure to chrysotile, or from occasional peak concentrations, have not been delineated but are predictably small.

5.426 Carcinogenic hazards from ultra-low levels of atmospheric chrysotile fibres (e.g. simple occupancy of public buildings) appear to be minuscule, negligible or undetectable.

5.427 Therefore, health concerns over chrysotile dust exposure narrow down to a workplace issue.

5.428 There is evidence of an increased incidence of mesothelioma among, say, brake mechanics in Australia exposed to chrysotile derived from brake blocks and lining.

5.429 With the reductions of airborne fibre concentrations in the asbestos mining, milling and manufacturing industries, construction trades workers constitute the group of workers at greatest risk from exposure to asbestos-cement products (e.g. builders, builders' labourers, carpenters, electricians, plumbers and roofing workers). This group constitutes a large, disparate and non-cohesive workforce for which controlled use of asbestos is not achievable, for the reasons discussed earlier in this report.

5.430 Therefore, chrysotile asbestos should not be used in building materials, because of the hazards imposed by installation, maintenance and removal operations (EHC 203); these risks may be compounded for some groups by catastrophic events affecting buildings — e.g. fires (with a burst of asbestos fibres into the atmosphere and the necessity for clean-up operations), and other disasters.

5.431 Substitutes for chrysotile are available for many applications (e.g. cellulose fibres, para-aramid fibres and polyvinyl alcohol); evidence indicates that these fibres are less bio-persistent than chrysotile and, therefore, national health authorities (EHC 203, NICNAS 99) have recommended phasing out or prohibition of chrysotile whenever safer substitute materials are available.

5.432 Therefore, from a perspective of caution and prudence for occupational health and safety, it follows that chrysotile should either:

(a) Be restricted to only a few and well-defined applications so that it is inaccessible to the great majority of workers and is available for use by only small and cohesive specialized worker groups that can be trained effectively in its controlled use (e.g. analogous to nuclear fuels); in effect, this means that chrysotile should not be used in building products (e.g. high-density fibro-cement materials such as asbestos-cement sheets) or friction products.

OR

(b) It should be made inaccessible to everyone, by prohibition, unless the alternatives pose equal or greater hazards and equal or greater problems with control.

5.433 These views are also expressed in EHC 203, wherein it is stated:

"a) Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks.
b) Where safer substitute materials for chrysotile are available, they should be considered for use.
c) Some asbestos-containing products pose particular concern and chrysotile use in these circumstances is not recommended These uses include friable products with high exposure potential. Construction materials are of particular concern for several reasons. The construction industry workforce is large and measures to control asbestos are difficult to institute. In-place building materials may also pose risk to those carrying out alterations, maintenance and demolition" … [p 144]
d) The combined effects of chrysotile and other insoluble respirable particles needs further study.
e) More epidemiological data are needed concerning cancer risks for populations exposed to fibre levels below 1 f/ml, as well as continued surveillance of asbestos-populations" … [p 145]

5.434 NICNAS 99 sets out a similar set of recommendations:

- "Chrysotile is a known human carcinogen.
- Prudent OHS [occupational health & safety] policy and public health policy favours the elimination of chrysotile wherever possible and practicable.
- The main exposure to Australian workers arises from manufacture, processing and removal of friction products and gaskets. Home mechanics are also exposed during 'do-it-yourself' replacement of brake pads/shoes. ... . In Australia, chrysotile is no longer used in high density materials such as chrysotile-cement.
- Current overseas experience with the phasing out of chrysotile products indicates that a range of alternatives is available to suit the majority of uses. Good OHS practice dictates that use of chrysotile products should be restricted to those uses where suitable substitutes are not available, and alternatives should continue to be sought for remaining uses".

5.435 Whether the objective of removal of chrysotile from the workplace and the general environment is achievable by enforcement of controlled use for a few restricted applications — or by prohibition — is essentially a societal question and a public health policy issue. For the reasons discussed in this report, a complete ban is more certain to accomplish this objective (paragraph 5.432(b)). Therefore, as a cautious and prudent approach to national occupational health policy, a complete ban is neither unreasoned nor unreasonable; on the balance of prevailing scientific evidence and uncertainties discussed in this report, such a policy seems defensible and, arguably, justifiable as a national health measure. Perhaps it is best to let Bradford Hill have the last words:

"All scientific work is incomplete — whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone action that it appears to demand at a given time."

4. Endnote by Dr. Henderson

5.436 Wishing to add two further pertinent references after completing his Report, Dr. Henderson attached the following Endnote. These references[27] deal with the following:

5.437 Clearance of chrysotile fibres from human lung tissue: In the past, the kinetics of chrysotile clearance from lung tissue have been investigated mainly in experimental models using rodents. In an autopsy study published in 1999, Finkelstein and Dufresne [1] investigated clearance of chrysotile

[27]For complete references, see Annex III to this Panel Report.

from the lung tissue of 72 Quebec chrysotile miners and millers in comparison to 49 control subjects, using regression analyses, with the following findings:

- There was a significant association between the duration of occupational exposure and the tissue burdens of chrysotile and tremolite.

- The concentration of chrysotile decreased with time after exposure ceased but the concentration of tremolite did not.

- The clearance rate varied inversely with the length of chrysotile fibres. For fibres > 10 µm in length - i.e. fibre lengths in the reported range for carcinogenicity - the clearance half-time was estimated to be eight years. In other words, the tissue bio-persistence of chrysotile fibres in this study seems substantially more prolonged than in rodent experiments, and presumably corresponds to persistent high chrysotile fibre concentrations for many years after cessation of occupational exposure in humans, as discussed in paragraphs 5.112 - 5.113. It is also notable that the concentration of 6,250,000 chrysotile fibres mentioned in those paragraphs (for an individual but by no means unusual patient) is probably above the level at which Rogers et al. [2] identified an odds ratio for mesothelioma of > 8.5 (even allowing for differences in fibre size between the two different laboratories), and even the duration of 16 years after exposure stopped (as opposed to its commencement: 24 years) falls into the lag-time range lung cancer induction by asbestos.

- Studies like this suggest that clearance mechanisms can be overwhelmed and break down at occupational levels of exposure in humans, with the existence of a long-term sequestered fraction of chrysotile fibres.

5.438 Mesothelioma rates in men and women in Sweden: attached to this Endnote is a recent paper by Jarvholm et al. [3] on trends in mesothelioma incidence in Sweden, which re-emphasizes some of the points made earlier in this report.

D. COMMENTS BY THE PARTIES ON THE RESPONSES FROM THE EXPERTS

1. Canada

5.439 Canada is pleased that the experts agree with Canada on certain crucial aspects of the debate in this case. Most importantly they opine that:

- Chrysotile is significantly safer than amphibole asbestos (three of the four experts agree);

- there is no risk to the public from low-level environmental exposure to chrysotile or from exposure in buildings that contain chrysotile (all of the experts agree);

- there is no risk to workers in mines or factories where use of chrysotile is controlled (three of the four experts agree); and

- there is no risk to "handymen" or "do-it-yourselfers" who disturb chrysotile products, because their exposure is intermittent and thus inconsequential (three of the four experts appear to agree).

5.440 In short, although the experts agree on the inadequacy of a data (statistical limitation to support a threshold), their findings are consistent with the view that low levels of exposure to

chrysotile asbestos create no detectable health risks. Indeed, the only population that the experts view as having problematic exposure is tradesmen, e.g., plumbers, electricians and mechanics, who disturb or modify chrysotile cement and friction products. At this point, the experts and Canada diverge; also, at this point, the experts stray beyond their specialities (as several admit). Canada maintains that adequate controls for these exposures can be developed and applied and has set forth such controls in Comments to Experts' Answers to Question 5.

5.441 Several other aspects of the experts' answers require comment. Some of the responses of the experts appear not to distinguish between chrysotile and amphibole exposure, and between modern uses (e.g., chrysotile friction and cement products) and historical uses (e.g., insulation containing amphiboles). In many places, for example, the experts appear to draw conclusions regarding chrysotile based in part or in whole on data from individuals exposed to amphiboles and/or amphiboles and chrysotile. This is of greatest concern to Canada regarding the experts' conclusions on tradesmen; as the experts no doubt agree, the greatest risk to tradesmen is not exposure to modern chrysotile products, but the disturbance of flocking or insulation containing amphiboles. Similarly, the experts do not always distinguish peak and cumulative exposures. For the purposes of defining health risks, the cumulative measure, not the peak measure, is key.

5.442 It is crucial that this proceeding forms on the pertinent issues. The key issue is whether exposure to modern uses of chrysotile can be controlled to ensure worker safety or if a total ban is required to achieve an equivalent level of safety. The experts' answers help to focus the proceeding on tradesmen exposure.

Question 1(a)

5.443 Canada believes that the Panel should take note of the clarifications to this question proposed by Drs. de Klerk and Musk. The former writes that "the more relevant question here is: who is likely to receive the most exposure and therefore have the greatest risk of disease". Dr. Musk rephrases the question in almost identical terms, taking the expression "risk of exposure" to mean "who is most likely to receive the most exposure and therefore be at the greatest risk of developing asbestos-related disease". In Canada's view, because the evidence suggests different risk per unit fibre exposure in different sectors, it is the combination of level of exposure, duration of exposure and risk per unit fibre exposure that is important.

5.444 The experts have confirmed that any risk from exposure to chrysotile will depend on the nature of an individual's specific occupational setting and the risk per unit fibre exposure in that setting, certain sectors being the subject of more stringent controls than others. For example, the experts echo the Parties' agreement to the effect that the mining and manufacturing sectors have successfully controlled the risks to which their workers had previously been exposed. Certain settings pose lower risk per unit fibre exposure than others do.

5.445 Canada does not disagree with the statement that the so-called secondary user sector is the most diverse. Canada nevertheless understands that the experts do not believe that the diversity of this particular workforce is to be considered the only factor that may contribute to a greater likelihood of exposure; rather, as Dr. Musk puts it, "the risk of developing asbestos-related disease [...] (also) depend(s) on [...] the type of asbestos being produced or used or otherwise encountered. It would also depend on the conditions of work such as indoors versus outdoor etc." As the Panel also knows, the specific uses or products also entail more or less risk.

5.446 Canada does not believe that the diversity of this workforce precludes effective control. The diversity of a specific workforce is not indicative of the quality of the work practices actually observed by the members of that workforce. A typical construction site offers numerous examples of sound safety practices: from hard hats to proper footwear, from the use of common sense to following

trade-specific work practises, measures are taken to insure safety and avoid trauma.

5.447 Canada notes that the experts have not commented on the assertion by the European Communities that there is a correlation between the amount of chrysotile used by France and the incidence of asbestos-related disease. Clearly no such correlation can be made, in logic or in fact. The logic on which the European Communities purports to base this assertion is a sophism, and should be dismissed accordingly. From a factual point of view, the following factors suggest that the correlation is false: the relative difference in potency and in biopersistence of amphiboles and chrysotile, the historical uses of each fibre type, and the differences in risk per unit fibre exposure in different sectors.

5.448 Canada notes that Dr. Infante assimilates friable amphibole or mixed fibre type exposure circumstances to those of high-density chrysotile products, thereby answering the wrong question. He correctly identifies worker contact with insulation as being the "typical scenario" in which exposure to asbestos will occur. But most insulation is friable, as opposed to high-density, and most friable insulation products contained amphiboles or mixed fibre types. It is not clear how this answer based on friable mixed asbestos products responds to a question relating solely to safety of high-density chrysotile products.

Question 1(b)

5.449 Canada takes note that the experts have indicated that the risk of human health associated with the various uses of chrysotile throughout its life cycle is overwhelmingly a workplace issue, and therefore not related to the "handyman".[28]

Question 1(c)

5.450 The answers given by the experts indicate that, on their own, chrysotile cement products do not pose a health risk because of their normal weathering, erosion or general degradation, and that "there is little or no dispute among experts on this issue".[29]

5.451 Canada wishes to draw the attention of the Panel to the results of the investigation carried out by the Western Australia Advisory Committee on Hazardous Substances (WAACHS), cited by Dr. Henderson.[30] This report contains different sections describing asbestos cement products, their production and use and their health effects, as well as surveys of schools and other relevant measurements of asbestos concentrations. In addition to pertinent recommendations, the report contains several appendices, including one on the *Effects of Asbestos Cement Products – A Review of the Literature* and another on *Acceptable Air Concentrations of Asbestos Fibres in the General Environment*, both prepared by one of the experts to this Panel, Dr. de Klerk.

5.452 On low level air concentrations, Dr. de Klerk writes: "[M]ost of these estimates are on or below the level of what the Royal Society would consider acceptable [...] The 1986 IPCS report did not even bother to estimate such risks and summarised the risk exposure unrelated to occupation as being undetectably low".[31] Indeed, the executive summary of the WAACHS report indicates: "[...] [T]he level of risk is low enough to be considered to be negligible relative to these other risks in our

[28]Canada notes that, according to Dr. Henderson: "[f]rom my perspective, this is overwhelmingly a workplace issue […]".

[29]Henderson, answer to Question 1(d).

[30]Henderson, p. 54, citing Multiple Authors, *Asbestos Cement Products. Report by the Western Australia Advisory Committee on Hazardous Substances*, Perth, 1990, hereinafter the WAACHS Report.

[31]De Klerk, N., *Acceptable Air Concentrations of Asbestos Fibres in the General Environment. A Review of Scientific Evidence and Opinion* in the WAACHS Report, Appendix 3, p. 10.

society".[32] Similarly, in his report to the Panel, Dr. Henderson underlines that compared to the fibre concentrations observed in the vicinity of asbestos-cement roofing, "a greater risk to health would arise from [workers] falling from or through the roofs".[33]

5.453 High-density chrysotile on buildings has been extensively studied. Indeed Teichert found the following: "the study of emission conducted on coated and uncoated roofing materials revealed low asbestos fibre concentrations, even though severe corrosion was observed on uncoated asbestos cement roofs and a considerable quantity of material containing asbestos could be removed by blowing or suction. The asbestos fibre concentrations that were measured in populated areas are well below the level considered acceptable by the Health Authorities of the Federal Republic of Germany, i.e. clearly below 1000 fibres/m^3 (or 0.001 f/ml)".[34] Felbermayer and Ussar, for their part, write: "a comparison of the asbestos fibre concentrations in those areas with and without asbestos-cement roofing (...) lead to the conclusion that there is no statistical significant connection between the use of asbestos-cement materials and the asbestos fibre concentrations found in the various measurement areas."[35]

5.454 Finally, Canada would like to bring to the Panel's attention the following recommendation of the WAACHS report, which is: "[A]n asbestos cement roof, which has not deteriorated to an extent where physical safety or structural integrity is of concern, should not be replaced. In addition, an asbestos cement roof should not be treated with a coating on the basis of risk to health. Other asbestos cement products are generally less prone to deterioration and do not require attention for health purposes".[36] Nonetheless, many chrysotile-cement products are coated with protective sealant agents.

Question 1(d)

5.455 The experts agree that the degree of risk to the health of workers intervening on high-density chrysotile cement products will depend on the manner in which an intervention is carried out. As noted by Dr. Infante in his response to question 1(e), "the extent of the exposure to the worker (...) would depend on the nature of the intervention, e.g., the circumstances under which the chrysotile asbestos product is manipulated in terms of work practices, the controls, or lack of controls in place and the type of personal protective equipment provided to the worker". Dr. Henderson illustrates this proposition when he writes that "cutting (chrysotile-cement) with hand saws produced lower concentrations."

5.456 Canada accepts that abrasion and cutting of high-density chrysotile products can release materials. However, the degree of exposure, if any, will depend on the methods and controls used. Canada notes that the experts disagree as to the exact composition of the materials that would be released by such interventions (see question 1 (f)), although there is apparent agreement that cutting chrysotile cement releases crystalline silica, an IARC Class 1 carcinogen.[37] Cutting chrysotile cement using simple work practices such as those outlined in ISO Standard 7337 will therefore provide protection from any potentially harmful material contained in such a product. Wetting the product before cutting and/or using commonly found suction attachments when sawing are techniques that can be used as added, but perhaps unnecessary, precautions. A final safety barrier would be for the worker to wear a facemask: this step would render it virtually impossible for the worker to inhale dust.

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[32]WAACHS Report, p. 2.

[33]Henderson, answer to Question 1(b).

[34]Teichert, U., *Immissionen durch Asbestzement-Produkte,* (1986) Teil 1 Stub Reinhaltung der Luft, Vol. 46, No. 10, pp. 432-434.

[35]Felbermayer, W., Ussar, M. B., *Research Report: Airborne Asbestos Fibres Eroded from Asbestos Cement Sheets*, (1980) Institut für Umweltschutz und Emissionesfragen, Leoben, Austria.

[36]WAACHS Report, p. 4.

[37]Infante, answer to Question 1(f).

5.457 Neither the European Communities nor the experts have demonstrated that such practices would subject workers to cumulative exposures presenting health risks. An American survey estimated that a worker would spend less than $1/16^{th}$ of his work time on tasks that would involve aggressive interventions on chrysotile-cement of the type susceptible of releasing any substantial amount of dust.[38] Canada submits that the European Communities have not identified any population of workers that would be subject to a detectable risk because of professional contact with high-density chrysotile cement. The European Communities' contentions *vis-à-vis* the "handyman" are therefore even less convincing (see next answer).

Question 1(e)

5.458 Canada agrees with Dr. Henderson's conclusion that "occasional interventions (...) would predictably produce low cumulative exposures, with a lower risk (...)". Dr. Henderson also affirms that for "electricians, carpenters, plumbers, insulation workers and so forth", "it is acknowledged that most if not all these mesotheliomas are a consequence of exposure to (...) a mixture of asbestos types, including chrysotile and one or more of the amphiboles."

5.459 The Panel has not been presented with evidence that contradicts Canada's assertion that occasional interventions do not pose a risk that is significantly different from zero (statistically). Therefore, the experts have not validated the EC's claim that an alleged risk for workers or the "handyman" is something more than undetectable.

5.460 Nor has the Panel been presented with evidence or expert opinion that supports the European Communities' claim with respect to the "handyman". Given that cohorts exposed to relatively high concentrations of chrysotile over entire occupational lifetimes show no increase of disease, it is unlikely that occasional interventions by a "handyman" would produce more than an equally undetectable risk. Obviously the "handyman" or *bricoleur du dimanche* will not encounter high-density chrysotile-cement products on a daily basis, nor devote his "handiwork" exclusively or principally to cutting such products. Rather, the typical "handyman" will rarely, if ever, come into contact with chrysotile cement products, let alone be sawing them.

5.461 The Panel should note that no evidence has been presented that shows any fatality in workers, let alone in "handymen", who would have been subject to any form of exposure, high or low, from contact with chrysotile cement products; the argument presented by the European Communities has been based entirely on hypothetical scenarios.[39]

Question 1(f)

5.462 There is debate in the scientific community and among the experts appointed by the Panel as to the exact physical and chemical composition of what is contained in dust from certain interventions on chrysotile cement products. Dr. Infante writes, however, that this dust (indeed, all cement dust) will contain "crystalline silica", a known IARC Class 1 carcinogen found in all cement.

5.463 A 1992 IARC publication determined that "in asbestos-cement products, the asbestos fibres usually represent 10-15 per cent of the total weight and are embedded in the cement. Therefore, it is not certain a priori that dust generated from asbestos-cement products will have the same effect as dust from pure chrysotile. [I]n asbestos-cement dust most of the asbestos fibres form aggregates with cement particles ... [t]hose which do not form aggregates ... appear to be coated with a calcium-containing layer. In absorption experiments, the asbestos-cement dust behaves more like cement dust

[38]CONSAD Research Corporation, 1990, No. 8282.
[39]See Canada's Comments to Questions 5(c) and (e).

than like asbestos dust."[40] Because the surface properties of asbestos fibres are altered by certain heating, pH, and abrasion conditions[41], it can be deduced that the composition and effect of the final aerosol would be different than that suggested by studies of concentrations of fibres alone. And again, controlled use procedures limit release, and proper breathing equipment precludes exposure.

Question 1(g)

5.464 Canada believes that the Panel was not presented with any quantification of this risk, or indeed its existence. Dr. Infante describes how the removal of chrysotile cement panels can be accomplished with negligible release of respirable fibres. Most other chrysotile cement products are found in the form of underground water pipes. Studies show that these products remain intact for decades after installation.[42] Hence, very little of this product will need to be disturbed. Moreover, the excavation and removal of pipes is not executed by manual labour, the bulk of any removal being done by heavy machinery.

5.465 The Panel should also note that the removal of chrysotile cement products does not generally entail crushing. Rather, if and when necessary, chrysotile cement products can be removed, transported, and disposed of by means that do not constitute a detectable risk to human health. The French Circulaire 97-15 accomplishes this goal for the high-density products at issue in this proceeding.[43] Also, if France is ensuring the safe removal and disposal of friable asbestos materials[44] known to contain amphiboles or mixed fibre types, the Panel should conclude that the removal and disposal of high-density chrysotile cement products can be accomplished even more safely, since high-density materials are indisputably recognised, even by France, as much easier to manage than anything in friable form.[45]

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[40]*Characterization and Proprieties of Asbestos-Cement Dust*, in Biological Effects of Mineral Fibres, Vol. 1, IARC Scientific Publications No. 30, Lyon, 1980, pp. 43, 49 and 50.

[41]Henderson, answer to Question 2.

[42]Canada notes that a study of 15 water supply systems in the State of Illinois U.S.A., where some asbestos-cement pipes were up to 40 years old, and where the water was non-aggressive to moderately aggressive, shows no significant differences in the water before and after passing through the asbestos-cement pipe network: Hallenbeck, W. H., et al., *Is Chrysotile Asbestos Released from Asbestos Cement Pipe into Drinking Water*, (1978) Journal of the American Water Works Association 70 (2): 97-102.

[43]Circulaire no. 97-15 du 9 janvier 1997 *relative à l'élimination des déchets d'amiante-ciment générés lors des travaux de réhabilitation et de démolition du bâtiment et des travaux publics, des produits d'amiante-ciment retirés de la vente et provenant des industries de fabrication d'amiante-ciment et des points de vente ainsi que tous autres stock*.

[44]Circulaire no. 96-60 du 19 juillet 1996 *relative à l'élimination des déchets générés lors des travaux relatifs aux flocages et aux calorifugeages contenant de l'amiante dans le bâtiment*.

[45]Canada notes that French regulations indeed recognize a difference in disposal proscriptions between "les matériaux friables" and "l'amiante liée" – see Note DPPR/SDPD/BGTD/LT/LT no. 97-320 du 12 mars 1997 relative aux conséquences de l'interdiction de l'amiante et à l'élimination des déchets, which is as follows:

> *"III. – Quelles sont les fillières d'élimination des déchets contenant de l'amiante?*
>
> *"Deux circulaires ont été diffusées, l'une le 19 juillet 1996 pour les déchets issus des travaux relatifs aux flocages et aux calorifugeages, l'autre le 9 janvier 1997 pour les déchets d'amiante-ciment.*
>
> *"Les fillières d'élimination des déchets contenant de l'amiante autres que ceux qui ont fait l'objet des deux cricularies précitées peuvent être déterminées par analogie aux prescriptions de ces deux circulaires:*
>
> - *Les matériaux friables, c'est-à-dire les matériaux susceptibles d'émettre des fibres sous l'effet de chocs, de vibrations ou de mouvements d'air, sont assimilables aux flocages et aux calorifugeages. Ils devront être éliminés dans des installations de stockage des déchets industriels spéciaux ou dans l'unité de vitrification;*
> - *pour les déchets contenant de l'amiante liée, trois cas sont envisageables:*

Question 1(h)

5.466 See comments on previous question.

Question 1(i)

5.467 Canada wishes to add the following comments on the answers to this question. Once removed from a building, a chrysotile cement panel, even if broken into several pieces, remains as intact as when it formed part of that building. Studies referred to above indicate that chrysotile cement roofing does not contribute (< 0.001 f/ml) to the levels of chrysotile occurring naturally in the environment. Likewise, chrysotile cement piping is generally found below ground, and therefore does not contribute to the levels of chrysotile naturally occurring in the atmosphere. If removed from roofing, or if excavated and removed from a water system, chrysotile cement products are transported to a landfill and buried anew beneath a layer of earth. Consequently, Canada is of the view that used chrysotile cement products can be eliminated safely.

5.468 Canada also notes that recent technology has enabled safe (in some cases, on-site) disposal of chrysotile products. For example, chrysotile can be treated with chemicals and/or subjected to high temperatures so as to render the end product entirely harmless and, in fact, suitable for enhancing the quality of soils. For example, in the United States, a foam has been developed that eliminates the risk associated with removing asbestos from buildings; when this product is sprayed onto asbestos fireproofing, the fibres turn into harmless globs of magnesium silica. A U.S. building contractor recycles asbestos by subjecting it to a chemical bath and high temperatures resulting in a totally inert end-product suitable for soil improvement. A Japanese company, responding to a government law mandating pollution-free disposal of asbestos, melts asbestos into harmless glass.[46]

Question 2

5.469 Canada has advocated the use of chrysotile in high-density products only; textiles are not of that category, and had been banned in France prior to the adoption of the measure that is the subject of this dispute. Friction materials using chrysotile have not been shown to constitute a risk to human health.[47] Indeed, the contrary is probably true: lesser braking action of linings manufactured without

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- *Si les déchets sont composés d'amiante associée uniquement avec des matériaux inertes, ceux-ci pourront être éliminés conformément à la circulaire du 9 janvier 1997 relativement à l'élimination des déchets d'amiante-ciment;*
- *si l'amiante est associée avec des matériaux, qui lorsqu'ils deviennent des déchets, sont classés déchets ménagers et assimilés, c'est par exemple le cas des dalles vinyl-amiante, ils pourront être éliminés dans des installations de stockage de déchets ménagers et assimilés;*
- *si l'amiante est associée avec des matériaux, qui lorsqu'ils deviennent des déchets, sont classés déchets industriels spéciaux, ils devront être éliminés soit dans des installations de stockage de déchets industriels spéciaux, soit dans l'unité de vitrification.*

"Dans tous les cas, l'industriel ou l'entreprise devra fournir des éléments permettant de caractériser les déchets afin de déterminer les filières d'élimination adaptées."

[46]*Acid v. Asbestos,* Discover, Information Access Company, No. 7, Vol. 20, July 1, 1999, p. 102.; *Contractor Recycles Asbestos for Re-Use in Construction*, Air Conditioning, Heating & Refrigeration News, Business News Publishing Company, Vol. 194, No. 2, January 9, 1995, p.1*; Kent Firm Fires up New Asbestos-Disposal System*, Puget Sound Business Journal, Vol. 13, No. 14, August 21, 1992, p. 9; *Japanese Plant Turns Asbestos into Glass*, American Metal Market, Vol. 100, No. 145, July 28, 1992, p. 4.

[47]Appendix A on Control Use in the Friction Industry, Canada's Comments to Question 5(a), contained in Annex IV to this Report.

chrysotile is cited by France as the safety concern for which it exempted certain military vehicles from the purview of the Decree.[48]

Question 3(a)

5.470 Three of the four experts concur with the position of Canada and the WHO that a clear distinction must be made between chrysotile and amphiboles. Dr. Musk believes that "there is a need to distinguish chrysotile asbestos from amphiboles based on the epidemiological data at least" and that the relative pathogenicity of some amphiboles to chrysotile may, in some cases such as mesothelioma, be 100 to 1. Dr. de Klerk affirms that the "epidemiological evidence is clear that, for a given quantity (intensity and duration) of exposure, chrysotile imparts less risk than amphibole fibres." The difference in pathogenicity is, according to Dr. de Klerk, up to 50-fold in the case of lung cancer and up to 100-fold for mesothelioma. Dr. Henderson concludes that: "a clear distinction should be made between chrysotile and the amphibole forms of asbestos."

5.471 Domestic legislation and international standards have long recognized the relative pathogenicity of different asbestos fibre types by permitting higher exposures to chrysotile than to amphiboles. In the European Communities in 1998, for example, the maximum exposure level for amphiboles was 0.3 f/ml, whereas it was 0.6 f/ml for chrysotile. In Canada (Quebec), it is 0.2 f/ml for crocidolite and 1 f/ml for chrysotile. Similarly, international instruments such as the ILO's *Convention 162* and *Recommendation 172* advocate an outright ban on crocidolite, while recommending replacing chrysotile if and only if safer substitutes exist.

5.472 Dr. Infante acknowledges epidemiological data to the effect that chrysotile is less dangerous than amphiboles, but sees no basis for distinguishing between asbestos fibre types. Dr. Infante's dissident view to the question of relative pathogenicity between asbestos fibres – one which echoes the European Communities' argument but simply begs the question – is that because amphiboles and chrysotile are both classified as carcinogens, no distinction should be made.

5.473 In 1998, the WHO affirmed that a distinction should be made between chrysotile and amphiboles because using data from exposures to amphiboles "contribute[s] less to our understanding of the effects of chrysotile, due to concomitant exposure to amphiboles."[49] The distinction between chrysotile and amphiboles is crucial in this instance since the current problem of asbestos in France is due to past uses of friable materials, high-level exposures, and the use of amphibole fibres. The distinction between chrysotile and amphibole asbestos is also important because the extrapolations made by INSERM to assess the risks associated with chrysotile are based on exposures to amphibole fibres in proportions of up to 100 per cent in circumstances which have nothing to do with the current uses of chrysotile.[50]

Question 3(b)

5.474 Physical properties, as well as chemical properties that determine biopersistence, are identified as relevant factors of pathogenicity by Drs. Musk, Henderson and de Klerk and by the WHO.[51]

5.475 Dr. de Klerk, for example, has written that:

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[48]Article Ier 2a) *Arrêté du 17 mars 1998 relatif aux exceptions à l'interdiction de l'amiante*.

[49]WHO, *IPCS Health Criteria 203 on Chrysotile*, WHO, Geneva, 1998, p. 107.

[50]See notably INSERM Report, p. 213.

[51]WHO, *IPCS Health Criteria 203 on Chrysotile*, WHO, Geneva, 1998, p. 51: "[I]t is considered that the potential respiratory health effects related to [...] airborne concentrations, patterns of exposure, fibre shape, diameter and length (which affect lung deposition and clearance) and biopersistence."

> "[T]he important carcinogenic properties of asbestos are related to the physical properties of size and shape of the fibers, and to their quantity. To cause any harm, fibers must be able to reach the target organs [...]." [...]
>
> "[I]n all occupationally exposed series of mesotheliomas, none have occurred in cohorts where amphibole asbestos has never been used or detected. Chrysotile asbestos has not been directly implicated in any case of peritoneal mesothelioma. [...] The main differences between the effects of chrysotile and amphibole fibers are:
>
> 1. Industries using a mixture of asbestos types have higher rates of disease than similar industries using only chrysotile.
> 2. Chrysotile fibers are eliminated more readily from the lungs than are amphibole fibers.
> 3. Much smaller doses of amphibole fibers than chrysotile fibers can induce mesothelioma."[52]

5.476 All four experts recognize the lower biopersistence of chrysotile. INSERM, citing numerous studies, also acknowledges the lower biopersistence of chrysotile:

> "Les études expérimentales ont montré que la biopersistance des fibres de chrysotile était inférieure à celle des amphiboles (Wagner et al., 1974; Davis et al.; Davis and Jones, 1988, Churg et al., 1989; Churg, 1994)."[53]

5.477 Dr. Infante identifies the physical characteristics as also relevant to the relative pathogenicity of asbestos fibre types, but, unlike the three other experts and the WHO, believes that the role of biopersistence, through the element of solubility, "is not so clear."

5.478 Chrysotile fibres are "curly" and downy while amphibole fibres are straight and rigid like needles.[54] Drs. de Klerk and Musk both specifically address the "straightness" element. The WHO has observed that:

> "Inhalation of respirable straight fibres [amphiboles] is reported to be associated with greater penetration to the terminal bronchioles than in the case of 'curly' fibres [chrysotile]."[55]

5.479 Once they have entered the respiratory tract, chrysotile asbestos fibres, because of their curly shape, are more easily cleared by the mucociliary process than are straight and rigid amphibole fibres.[56] Dr. Henderson writes: "[I]t is well known that chrysotile fibres are cleared more rapidly than amphiboles, especially in long-term studies (Churg, 1994)."[57] This is confirmed by a 1994 European study by Dr. Albin: "[A]dverse effects are associated rather with the fibres retained (amphiboles), than with the ones being cleared (largely chrysotile)."[58]

5.480 For chrysotile fibres that do nonetheless manage to become lodged in the lungs, the solubility of the fibres and the action of macrophages come into play to make chrysotile a much less potent fibre. First, as the WHO recognizes, chrysotile has a lower resistance than amphiboles in acidic

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[52]de Klerk, N.H. and Armstrong, B.K., *The Epidemiology of Asbestos and Mesothelioma* in Malignant Mesothelioma, Henderson, D.W. et al., eds. Hemisphere Publishing, New York, 1992, 223 at p. 230.

[53]INSERM Report, p. 92.

[54]WHO, IPCS Health Criteria 203 on Chrysotile, WHO, Geneva, 1998, p. 11.

[55]WHO, IPCS Health Criteria 203 on Chrysotile, WHO, Geneva, 1998, p. 11.

[56]Kumar, V., Cotran, R. and Robbins, S., *Basic Pathology*, 6th Ed., London, Saunders Co., 1997, p. 228.

[57]Henderson, see above para. 5.112.

[58]Albin, M., *et al., Retention Patterns of Asbestos Fibres in Lung Tissue Among Asbestos Cement Workers* (1994) 51 J. of Occupational Environmental Medicine 205.

environments such as the lungs.[59] Second, the macrophages responsible for eliminating fibres from the lungs are able to deal more easily with chrysotile fibres than with amphibole fibres. A 1997 report of the French Government (G2SAT) referred to by the European Communities, recognizes that as a result of the chemical dissolution process that takes place in the lungs, carcinogen activity is subsequently practically nil:

> "Il a été démontré que le chrysotile est nettement plus facilement éliminé du poumon humain que les autres formes [amphiboles]. Par ailleurs, il ne présente pratiquement plus d'activité cancérogène (par injection intra-cavitaire) après attaque acide, laquelle dissout la majorité du magnésium."[60]

5.481 Dr. Wagner, in his 1988 study of asbestos-related diseases, concluded:

> "Chrysotile is the least harmful form of asbestos in every respect and [...] more emphasis should be laid on the different biological effects of amphibole and serpentine asbestos fibre."[61]

5.482 It should also be noted that gravimetric comparisons between amphiboles and chrysotile – widely used in the past in experimental work – tend to grossly misrepresent the relative pathogenicity of the fibres. According to the WHO, chrysotile "may contain more than 10 times more fibres per unit weight."[62] Recent studies that use both the fibre mass and the number of fibres as dose units confirm that, on a per fibre basis, amphiboles are far more pathogenic than chrysotile.[63]

Question 3(c)

(i) Asbestosis

5.483 Dr. Henderson asserts that: "[T]he amphibole varieties of asbestos appear to be substantially more pathogenic than chrysotile for the induction of asbestosis and mesothelioma." According to Dr. Henderson, "[A]sbestosis is a dose-dependent disorder with a threshold effect [...] There is widespread agreement that asbestosis in general is a consequence of high intensity exposure (or lower intensity but more prolonged exposure)."

5.484 INSERM also supports the existence of a threshold for asbestosis,[64] and according to INSERM, current low-level exposures to chrysotile pose no threat of asbestosis: "les expositions actuellement relevées dans les industries directement utilisatrices d'amiante devraient conduire à la disparition des cas d'asbestose confirmée (Doll et Peto, 1985)."[65] It is clear, therefore, that asbestosis is not relevant to this dispute.

(ii) Lung Cancer

5.485 Dr. Musk believes that lung cancer risks are more than ten times greater in the case of amphiboles than in the case of chrysotile asbestos. Dr. de Klerk suggests the difference may be up to 50-fold.

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[59]WHO, *IPCS Health Criteria 203 on Chrysotile*, WHO, Geneva, 1998, p. 4. Kumar, V., Cotran, R. and Robbins, S., *Basic Pathology*, 6th ed., London, Saunders Co., 1997, pp. 227; INSERM Report, p. 396.

[60]INRS, *Rapport du Groupe scientifique pour la surveillance des atmosphères de travail (G2SAT)*, 1997, p. 47.

[61]Wagner, J.C. et al., *Correlation between Fibre Content of the Lung and Disease in East London Asbestos Factory Workers*, (1988) 45 British Journal of Industrial Medicine 305.

[62]WHO, IPCS Health Criteria 203 on Chrysotile, WHO, Geneva, 1998, p. 69.

[63]See WHO, *IPCS Health Criteria 203 on Chrysotile*, WHO, Geneva, 1998, p. 69 and 81; INSERM Report, Table 2, p. 196; EPA, Integrated Risk Information System, *Asbestos*, Document No. CASRN 1332-21-4 on-line: EPA, <http://www.epa.gov/ngispgm3/iris/subst/0371.htm> (access date: June 10, 1999).

[64]INSERM Report, p. 327.

[65]INSERM Report, p. 327.

5.486 Dr. Henderson states that the "greater carcinogenicity of the amphiboles [...] appears not to extend to the induction of lung cancer"[66] but he admits that "chrysotile is implicated in one of the lowest rates of asbestos-associated lung cancer (in Quebec chrysotile miners and millers)."[67] Dr. Henderson's reluctance to conclude the greater carcinogenicity of amphiboles seems to be caused by the results of Dr. Dement's study of the Charleston, South Carolina asbestos textile industry.[68]

5.487 The Charleston data has recently been revisited by Bruce Case, André Dufresne, A.D. McDonald, J.C. McDonald and Patrick Sébastien in a study released in Maastricht in October 1999 at the *VII*th *International Symposium on Inhaled Particles*, a symposium attended by some of the world's leading experts. This study shows that a significant amount of crocidolite and amosite fibres was found in the textile workers' lungs. This analysis sheds new light on the issue and explains the extreme results of the original study by Dr. Dement[69] and the subsequent study by Dr. Stayner.[70] These studies of textile workers exposed to crocidolite and amosite can thereby no longer be used to demonstrate the risks associated with chrysotile fibres.

5.488 The seminal findings of Case et al may cause Dr. Infante to reconsider his view – based principally on the studies by Dement and by Stayner – that "chrysotile may be more potent in causing lung cancer."

(iii) Mesothelioma

5.489 On the relative risks of mesothelioma, Dr. Henderson observes that: "[T]here is general though not universal agreement of a differential potency between the amphiboles versus [chrysotile] for mesothelioma induction." He believes amphiboles may be greater than 60 times more likely than chrysotile to induce mesothelioma.[71] Drs. Musk and de Klerk estimate that the potency of amphiboles may be 100 times greater. And although Dr. Infante also concedes that "amphiboles may be more potent in causing mesothelioma", he fails to conclude from this that a distinction exists between chrysotile and amphibole fibres.

5.490 This distinction is also emphasized in pathology medical reference books:

> "It is important to make the distinction between various forms of amphiboles and serpentines, because amphiboles, even though less prevalent, are more pathogenic than the serpentine chrysotile, particularly with respect to induction of malignant pleural tumors (mesotheliomas). Indeed, some studies have shown the link is almost invariably to amphibole exposure."[72]

(iv) Other Diseases

5.491 Dr. de Klerk links other asbestos-related diseases such as pleural plaques and pleural thickening more with amphiboles than with chrysotile: "[P]leural plaques appear to be more common among anthophyllite workers than others while crocidolite workers have more diffuse pleural

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[66]Henderson, see above paragraph 5.146.

[67]*Ibid*.

[68]*Ibid*.

[69]Dement, J.M., Brown, D.P. and Okun, A. , *Follow-Up Study of Chrysotile Asbestos Textile Workers: Cohort Mortality and Case-Control Analyses*, (1994) 26 American J. of Industrial Medicine 431.

[70]Stayner, L., Smith, R., Bailer, J., Gilbert, S., Steenland, K., Dement, J., Brown, D., Lemen, R., *Exposure-Response Analysis of Risk of Respiratory Disease Associated with Occupational Exposure to Chrysotile Asbestos*, (1997) 54 Occupational Environmental Medicine 646.

[71]Henderson, see above para.5.103.

[72]See Kumar, V., Cotran, R. et Robbins, S., *Basic Pathology*, 6th Ed., London, Saunders Co., 1997 at pp. 227-28.

thickening, and benign asbestos pleurisy also seems to be more common after crocidolite exposure." Dr. Henderson also raises the issue of types of fibres in dealing with parietal pleural plaques.

Question 4(a)

5.492 Drs. de Klerk and Musk agree that the existing epidemiological data show no excess health risks at low-level chrysotile exposures. Dr. Henderson is not aware of exposure-response data for low-level exposures. Dr. Infante again relies heavily on Stayner's study, a study on one single cohort of textile workers now known to be based on textile workers exposed to amphiboles as well as to chrysotile.[73] Newhouse and Sullivan studied exposures to chrysotile in the manufacturing setting: "[I]t is concluded that with good environmental control, chrysotile asbestos may be used in manufacture without excess mortality."[74]

5.493 Thomas et al. concluded similarly for an asbestos cement factory: "[T]hus the general results of this mortality survey suggest that the population of the chrysotile-cement factory studied are not at any excess risk in terms of total mortality, all cancer mortality, cancers of the lung and bronchus, or gastrointestinal cancers."[75]

5.494 There is clearly no increased risk of lung cancer in the friction products manufacturing industry at levels below 356 f/ml-years. This means that there was no chrysotile-related increase in lung cancer risk for persons exposed to the equivalent of up to 8.9 f/ml for 40 years. Even if we allowed a 10-fold protection factor this would be 0.9 f/ml for 40 years for lung cancer.[76] More recently in 1997, McDonald et al. concluded from the analysis of a cohort of 10,000 asbestos workers with average exposures to 45 f/ml over 20 years that: "[...] from the point of view of mortality [...] exposure in this industry to less than 300 mpcf.years [approximately 45 f/ml over 20 years] has been essentially innocuous."[77] This unequivocal data comes from the longest term study of the largest group of chrysotile workers ever conducted. A review of eight studies of cohorts exposed to chrysotile only led its authors to conclude: "[T]he evidence for chrysotile shows that for lung cancer and mesothelioma there exist levels of exposure below which risks are for practical purposes zero."[78]

Question 4(b)

5.495 According to Dr. Henderson, whether a threshold exists generally is a much-debated issue. For the case at hand, i.e. low-level exposure to chrysotile, Dr. Henderson states that: "[I]f a threshold exists, it must lie somewhere in this area, between no exposure, low-level environmental exposure, and low-level occupational exposure." He also points out that, although no threshold has been identified, '[a]t the same time, no increase in risk of mesothelioma has been identified at very low-levels of exposures." Drs. Musk and de Klerk agree that the epidemiological data show an absence of risk at low exposure levels, but are unwilling to commit to the existence of a threshold. If there is

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[73]See Canada's Comments to Question 3.

[74]Newhouse, M.L. and Sullivan, K.R., *A Mortality Study of Workers Manufacturing Friction Materials,* (1989) 46:3 British Journal of Industrial Medicine 176, p. 176.

[75]Thomas, H.F., Benjamin, I.T., Elwood, P.C. and Sweetnam, P.M., *Further Follow-Up Study of Workers From an Asbestos Cement Factory,* (1982) 39:3 British J. of Industrial Medicine 273, p. 275.

[76]Berry, G. and Newhouse, M.I., *Mortality of Workers Manufacturing Friction Materials Using Asbestos,* (1983) 40 British Journal of Industrial Medicine 1 at 6, p. 6.

[77]Liddell, F.D.K., McDonald, A.D. and McDonald, J.C., *The 1891-1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Development from 1904 and Mortality to 1992*, (1997) 41 Annals of Occupational Hygiene 13, p. 13.

[78]Browne, K. and Gibbs, G., "Chrysotile Asbestos – Thresholds of Risk" in Chiotany, K., Hosoda, Y., Aizawa, Y., eds., *Advances in the Prevention of Occupational Respiratory Diseases,* Elsevier, Amsterdam, 1998 at p. 306.

agreement that low level exposures show no increased health risk, admitting the existence of a threshold is academic.

5.496 The extreme difficulty of proving a threshold scientifically is echoed by the European Communities' DG XXIV Report:

> "In fact, a threshold implies the demonstration that an effect does not occur at or under a given dose level. The unequivocal demonstration (i.e. identification) of a 'negative' is tantamount to impossible."[79]

5.497 The corollary to the proof of a threshold is the proof of the absence of a threshold. The proof that no threshold exists would need to explain the absence of an excess risk of lung cancer or mesothelioma in chrysotile-only cohorts, as well as the lack of any chrysotile-related increase in lung cancer mortality in workers exposed to less than 900 f/ml-years in the 10,000 miners and millers studied in Quebec.[80] Dr. Henderson does acknowledge the existence of a threshold for asbestosis in his answer to Question 3: "Asbestosis is a lung dependent disorder with a threshold effect [...] There is widespread agreement that asbestosis in general is a consequence of high intensity exposure (or lower intensity but more prolonged exposure)." INSERM also supports the existence of a threshold for asbestosis:

> "La plupart des données épidémiologiques recueillies dans des populations professionnelles exposées suggèrent que l'asbestose cliniquement et/ou radiologiquement caractérisée n'apparaît qu'à partir d'expositions suffisamment élevées [...] un seuil minimal de 25 f/ml-années a ainsi été avancé (Doll et Peto, 1985)."[81]

5.498 Why could there not be a threshold for other asbestos-related diseases? Dr. de Klerk asserts that:

> "[I]t is now widely believed that the risk for chrysotile workers in fibrous cement and friction product manufacturing is so slight as to be undetectable. It is widely held that this kind of negligible risk level 'threshold' exists at different levels for all types of asbestos for all relevant diseases."[82]

5.499 Some experts advising the EC believe there is a threshold for diseases other than asbestosis:

> "It is very likely that there is a practical level of exposure below which it will be impossible to detect any excess mortality or morbidity due to asbestos. [...] Thus, it is possible that there is a level of exposure (perhaps already achieved in the general public) where the risk is negligibly small."[83]

5.500 This links to Dr. de Klerk's observation that: "[T]he smaller the effect that needs to be demonstrated, the larger the study needs to be." Dr. Infante, who dismisses the Panel's question as "moot", points out that "it is not possible to determine thresholds from epidemiological studies because of the lack of statistical power to distinguish that the risk is virtually zero." Canada argues –

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[79]DG XXIV, *Opinion on a Study Commissioned by Directorate General III (Industry) of the European Commission on "Recent Assessments of the Hazards and Risks Posed by Asbestos and Substitute Fibres, and Recent Regulation on Fibres World-Wide"*, Environmental Resources Management, Oxford (opinion expressed on 9 February 1998).

[80]Liddell, F.D.K., McDonald, A.D. and McDonald, J.C., *The 1891-1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Development from 1904 and Mortality to 1992*,(1997) 41 Annals of Occupational Hygiene 13.

[81]INSERM Report, p. 327.

[82]de Klerk, N.H. and Armstrong, B.K., *The Epidemiology of Asbestos and Mesothelioma*, in Malignant Mesothelioma, Henderson, D.W. et al., eds. Hemisphere Publishing, New York, 1992, 223 at pp. 230-31.

[83]CEC, *Report of the Working Group of Experts to the Commission of the European Communities: Public Health Risks of Exposure to Asbestos*, Oxford, Pergamon Press, 1977 cited in: WHO, *Environmental Health Criteria 53 for Asbestos and Other Mineral Fibres*, WHO, Geneva, 1986, p. 43.

epidemiological data in hand – just that low-level exposures to chrysotile pose a risk that is "virtually zero": "un risque indétectable". Dr. Infante uses Stayner's data once again to claim that the chrysotile data fit with a linear no-threshold model. With the new analysis on the Charleston cohort data discussed above, this argument does not hold.[84]

Question 4(c)

5.501 Drs. de Klerk and Musk agree that there is epidemiological data indicating no increased risk at low-level exposures, but the experts believe the linear model may be appropriate. However, "[W]hether or not it is a valid method is unknown."[85] According to international experts from the Health Effects Institute-Asbestos Review (HEI-AR), such as Julian Peto, David G. Hoel and W. Nicholson, the linear model is not used for its validity, but precisely because it tends to overestimate risk.[86] Dr. de Klerk shares this view and states that the model provides a "conservative estimate."

5.502 The limits of the linear model and the conditions under which extrapolations are made must be clearly set out. Extrapolations from high-level exposures and exposures to amphiboles should not be taken at face value to ban chrysotile in today's context of low-level chrysotile-only exposures. Canada's critical view of the linear model is supported by a 1999 report by the Australian National Industrial Chemicals Notifications and Assessments Scheme (NICNAS) cited by Dr. Henderson:

> "There are many problems associated with low-dose risk extrapolation, such as the assumption of a linear relationship. However, as insufficient data exist to indicate threshold exposure for effect, the linear extrapolation methodology provides a conservative worst-case scenario estimate of risk. Other confounding factors in estimating risks from epidemiological data are possible contamination by other fibre types and inaccurate estimates of historical exposures."[87]

5.503 Not only does the linear model provide a worst-case scenario, it provides a grossly exaggerated estimate of risk when "confounding factors", as Dr. Henderson calls them, are so clearly present. INSERM made extrapolations from high-level amphibole exposures to mixed fibre type exposures, as well as from exposures in the textile industry and during the installation of low-density products such as flocking.[88] Amphiboles are much more potent than chrysotile, and the risks in the textile industry cannot be compared with the risks in the high-density chrysotile products, as Dr. Henderson points out in citing Boffetta: "[I]n general, the risk of lung cancer ... is highest in studies of asbestos textile workers."[89]

5.504 Another important consideration is the human biological defence mechanisms that are naturally much more effective at low-levels of exposure, i.e. clearance, biopersistence and DNA repair mechanisms.[90] Given these mechanisms, the reasoning behind the threshold model is both intuitively

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[84]See Canada's comments to Question 3.

[85]Henderson, answer to question 4(c).

[86]Health Effects Institute-Asbestos Research, Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge (Executive Summary), Cambridge, 1991, p. 6-62.

[87]Australia National Industrial Chemicals Notifications and Assessments Scheme (NICNAS), *Chrysotile Asbestos: Priority Existing Chemical No. 9 (Full Public Report)*, February 1999 at p. 72, cited by Henderson in his answer to question 4(c).

[88]INSERM Report, p. 213.

[89]Henderson, paragraph 5.149 above, citing: Boffetta, P., *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment*, (1998) 89 Med. Lav. 471.

[90]Voir Holland CD, Sielken RLJ., *Quantitative Cancer Modeling and Risk Assessment*. Englewood Cliffs, New Jersey: Prentice Hall, 1993; Sielken RL, Jr., Bretzlaff RS, Stevenson DE., *Incorporating Additional Biological Phenomena into Two-Stage Cancer Models* in: Spitzer HL, Slaga TJ, Greenlee WF, McClain M, eds. *Receptor-Mediated Biological Processes: Implications for Evaluating Carcinogenesis*. New York: Wiley-Liss, 1994;237-60. Stevenson DE, Sielken Jr. RL, Bretzlaff RS., *Challenges to Low-Dose Linearity in Carcinogenesis*

and scientifically sound, as well as epidemiologically validated. To illustrate this, consider the following illustration: the effect of 50 fibres in the lungs will be more than five times the effect of ten fibres.

5.505 According to Sir Richard Doll, who first demonstrated the link between asbestos and lung cancer (as well as between smoking and lung cancer), "[W]e have no real ground for postulating that a linear relationship for lung cancer can be extrapolated back to the levels of dose with which we are concerned in non-occupational settings."[91] Ames and Gold are of the same view: "[l]inear extrapolation from the maximum tolerated dose in rodents to low-level exposure in humans has led to grossly exaggerated forecasts in mortality." [92] Fournier and Efthymiou are even more categorical: "[L]inear extrapolation to zero is an unscientific methodology whose social consequences are so immense that it warrants unconditional elimination."[93] INSERM acknowledges the limits of the linear model's application when it states that it provides nothing more than food for thought: "cette extrapolation ne crée pas une information scientifiquement certaine, elle représente une aide à la réflexion en matière de maîtrise de risque."[94]

5.506 As Dr. de Klerk points out, "how one extrapolates risk assessment outside the range of available data is more of a societal decision than a scientific one."

Question 4(d)

5.507 Situations where there is no increased risk at low levels of exposure have been used by Stayner et al. to establish NOAELs [i.e. no observable adverse effect levels] for silica. A similar model is used for asbestosis. Canada believes that the use of such a model is warranted for other asbestos-related diseases, particularly since it has been acknowledged by Dr. Musk and Dr. de Klerk that epidemiological data exists to justify such an approach.

Question 4(e)

5.508 We concur with Dr. Henderson's view that "[t]his question iterates the issue of a threshold exposure." Canada nonetheless notes the use by Dr. Infante of a 1992 study by Bégin et al. to demonstrate the risks related to "background levels" is erroneous. As has been pointed out by Canada in its factual arguments,[95] this study is based on exposures to a mix of chrysotile and amphiboles in the manufacturing and construction industry, and therefore is not relevant to exposures from the current uses of chrysotile.

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from Interactions among Mechanistic Components as Exemplified by the Concept of 'Invaders' and 'Defenders'. BELLE Newsletter 1994;3(2):1-8. Stevenson DE., *Dose-Response Studies of Genotoxic Rodent Carcinogens: Thresholds, Hockey Sticks, Hormesis or Straight Lines? - Comment on the Kitchin and Brown paper*, BELLE Newsletter 1995;3(3):14-15.

[91]Doll, R., *Mineral Fibres in the Non-Occupational Environment: Concluding Remarks,* in Bignon, J., Peto, J. and Saracci R., eds., Non-Occupational Exposure to Mineral Fibres, IARC Scientific Publication N° 90, 1989, pp. 516-17.

[92]Ames, B.N. et Swirsky Gold, L., *Causes and Prevention of Cancer: Gaining Perspectives on the Management of Risk,* in Risks, Costs, and Lives Saved: Getting Better Results From Regulation?, New York, OUP, 1996, p.6.

[93]Fournier, E. and Efthymiou, M.-L., *Problems with Very Low Dose Risk Evaluation: The Case of Asbestos*, in What Risk?, p.49.

[94]INSERM Report, p. 239 and 414.

[95] See above Section III.A.5.

Question 5(a)

5.509 Clearly the answers given by the four experts are based on their concept of what is meant by controlled use. It is also evident that the controlled use concept as espoused by Canada was not the approach that resulted in their answers. We must therefore respectfully disagree with the answers given by the experts in respect of controlled use of chrysotile and high-density chrysotile containing products. The fact that they agreed that controlled use of chrysotile and high-density chrysotile products is feasible at some points of the life cycle, but not in others, suggests that they are not far from the view of Canada. The only difference is that Canada believes that the experts misunderstand the controlled use principle and that, as properly understood and implemented, use can be controlled throughout the full life cycle of high-density chrysotile containing products. The basis for our view, with supporting evidence, is set out below.[96]

(i) Canada's understanding of the "Controlled use" principle

5.510 The Canadian government's review of the experts' reports and answers to the questions posed by the Panel reveals that there is one crucial issue, which seems to override all other issues. This is the question of whether the application of the controlled use principle is feasible and credible in all stages in the life cycle of a product. While there is a reasonably high degree of agreement among experts that controlled use can be a reality in the mining and manufacturing sectors, serious doubts are expressed that controlled use can be applied in a few sectors of use – installation, maintenance and demolition. However, the basis for this view is not documented, except by Dr. Infante and Dr. Henderson.

5.511 By "controlled use", the Canadian government means "stewardship" based on the total life cycle. This is outlined in the document *The Mineral and Metals Policy of the Government of Canada: Partnerships for Sustainable Development*.[97] With regard to asbestos, this "controlled use" is based on the following general principles:

- Only the chrysotile variety is used;
- only a limited number of well-defined product applications, where it has been demonstrated that they can be handled safely, are allowed (i.e. where the fibres are encapsulated in a matrix such as cement, bitumen, plastic, resin, etc.);[98] and
- new product applications may be introduced only after a strict evaluation to ensure that a certain level of fibre release is not exceeded during its life cycle.

[96]Canada notes that the "controlled-use" approach has been endorsed by the WHO in its 1998 *Environmental Health Criteria 203: Chrysotile Asbestos*, p. 144. "Control measures, including engineering controls and work practices, should be used in circumstances where occupational exposure to chrysotile can occur. Data from industries where control technologies have been applied have demonstrated the feasibility of controlling exposure to levels generally below 0.5 fibres/ml. Personal protective equipment can further reduce individual exposure where engineering controls and work practices prove insufficient."

[97]NRCAN, The Minerals and Metals Policy of the Government of Canada: *Partnership for Sustainable Development*, Public Works Canada, 1996. Canada notes that the "controlled-use" approach to regulating chrysotile asbestos is well researched as evidenced in the studies and conclusions referred to by Canada in its factual arguments (see above Section III.A.6).

[98]To illustrate this point, examples of "controlled use" of friction products and asbestos-cement are detailed in Appendices A and B respectively to these comments. (These Appendices can be found in Annex IV to this Report).

5.512 With regard to the downstream use sectors, "controlled use" implies that all distributors/manufacturers of asbestos will be required to have an import permit. This permit will be withdrawn if the company does not meet the following commitments:

- To distribute its products only to companies (users) licensed to purchase these products. Those companies must have workers trained and licensed to install products, and must be in compliance with regulations. Approved users shall not resell to third parties, and any unused materials must be returned to the manufacturer;

- to provide a list of users of products to the responsible government agency;

- to provide products cut to specification and to establish centres equipped to cut the products to size, and where persons cutting the products are trained and are licensed to work with asbestos; and

- to police the downstream users in co-operation with the government. The product manufacturer visits, monitors and reports on the performance of the downstream users at regular intervals. There are penalties for failing to provide this product stewardship.

5.513 While high-density products in most countries are not considered to pose any occupational or environmental health risk, disposal should only be undertaken by approved and appropriately trained persons.

5.514 Dr. Infante's description of the permissible exposure limit for chrysotile asbestos, as well as programmes or standards that recommend or require specific engineering control, work practices, training and education and personal protective equipment to control exposures to asbestos corresponds, to some extent, to Canada's approach. Dr. Infante seems to suggest that because some workers do not comply with standards and regulations on controlled use in the United States, controlled use is not feasible. As explained in Appendix A on friction material and Appendix B on asbestos cement, the controlled use approach can minimize, if not eliminate, workers' non-compliance.[99]

5.515 Canada does not propose that any chrysotile products produced, sold or used without the implementation and enforcement of very stringent control procedures. Taking into account the types of products being manufactured and used in France at the time of the ban, Canada does not advocate re-introducing any product that cannot be handled according to the safety criteria outlined above. Canada is not advocating the introduction anywhere in the world of manufacturing facilities of products for which the technology does not exist to protect workers from exposure to chrysotile at levels where risks would be above epidemiologically based practical thresholds.

5.516 The experts have indicated that the level of exposure is such that they are not concerned about asbestos-related disease for persons living in buildings containing chrysotile asbestos products, including friable insulation. As none of the chrysotile products that will be used in the future are friable, this conclusion would be further reinforced. If the procedures envisaged under the "controlled use" policy are followed by licensed practitioners, the public will not be placed under any practically determinable increased risk of disease as a result of the manufacture and use of chrysotile containing products. Unlike friable insulation products where janitorial staff, electricians, carpenters, and others may be required to work regularly in an environment where exposures to asbestos would occur, the nature of the high-density products will ensure that exposures are a much rarer event.

[99]Appendices A and B can be found in Annex IV to this Report.

5.517 Canada recognizes that the clock cannot be turned back. Friable mixed products produced in the past are now in place, and trades such as electricians or telephone engineers face situations where the potential health risk from exposure is considerably greater than any additional risk that new high-density chrysotile products would present. It is evident that the protection of workers who come into contact with friable products must be assured by the responsible jurisdictions through training in trade schools, appropriate information programmes by unions, and by governments and employers ensuring that the appropriate equipment and tools are made available to workers.[100]

5.518 Regarding high-density products, Canada believes that no less stringent measures should be required, even though the evidence shows that the risk from exposure to high-density chrysotile products is minuscule compared to the risk from friable products, in many cases containing mixtures of chrysotile and amphibole fibres. Furthermore, in the absence of sound scientific data to the contrary, the same criteria should be applied to the handling of all products in which respirable fibres, including asbestos substitutes, may be released.

(ii) International Standards

5.519 None of the experts acknowledges that controlled-use of chrysotile asbestos cement products and other high-density chrysotile products stems from international standards. Dr. Infante even denies the existence of international standards on controlled-use of high-density chrysotile products. Canada wishes to remind the Panel that international standards, as the term is defined in the Agreement on Technical Barriers to Trade, do exist. Regulatory developments on asbestos fibres have been guided by ILO Convention 162 concerning Safety in the Use of Asbestos.[101] ILO convention 162 provides for: (i) the prescription of adequate engineering controls and work practices; (ii) the prescription of special rules and procedures for the use of asbestos or certain types of asbestos or products containing asbestos or for certain work processes; (iii) where necessary to protect the health of workers and technically practicable, the replacement of asbestos or of certain types of asbestos by other materials or the use of alternative technology scientifically evaluated by the competent authorities as harmless or less harmful; and (iv) total or partial prohibition of the use of asbestos or of certain types of asbestos in certain work processes.[102]

5.520 The Code of Practice on Safety in the Use of Asbestos of the International Labour Office referred to by Canada in all its submissions is another international standard on controlled-use.[103] The objects of the Code are: (i) to prevent the risk of exposure to asbestos dust at work; (ii) to prevent harmful effects on the health of workers arising from exposure to asbestos dust; and (iii) to provide reasonably practicable control procedures and practices for minimising occupational exposure to asbestos dust. To do so, the Code gives detailed guidance on the limitation of exposure in respect of asbestos cement and friction materials. Finally, Canada has referred the Panel to International Standard ISO 7337: Asbestos Reinforced Cement Products – Guidelines for On-Site Work Practices.[104] This international standard gives guidelines for tools and working methods to be used on site with a view to maintaining the dust emission at the lowest practicable level. It applies to asbestos-cement products.

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[100]See Section III.A.5 of this Report and Camus M., *L'amiante et les risques pour la santé*, April 1999.

[101]Conférence internationale du travail, Convention concernant la sécurité dans l'utilisation de l'amiante (Convention 162), adoptée le 24 juin 1986, and Recommandation concernant la sécurité dans l'utilisation de l'amiante (Recommandation 172), adoptée le 24 juin 1986.

[102]According to Canada, the emphasis of ILO Convention 162 is on controlled-use and not on product prohibitions. The Convention calls for two specific prohibitions: crocidolite and all products containing crocidolite, and sprayed-on applications of asbestos.

[103]Recueil de directives pratiques du BIT sur la sécurité dans l'utilisation de l'amiante, Organisation internationale du travail, Genève, 1984.

[104]ISO, standard ISO-7337 1984.

5.521 The ILO Convention 162 and the Code of Practice on Safety in the use of chrysotile should be supplemented by a national policy on responsible use based on the recognition and acceptance of the principles that both international standards set forth.[105] As explained above, the objective of responsible use is to limit the handling of chrysotile to companies that comply with the national regulations or that have submitted action plans and formal commitments in writing with a view to bringing their activities into line with these regulations.

Question 5(b)

5.522 The experts recognize that training could be achieved in the manufacturing sector, where there is a small and cohesive workforce, but assert without support that it cannot be achieved in the construction sector, where there is a large and non-cohesive workforce. Dr. Infante wrongly equates non-compliance with regulated training requirements to non-feasibility of training for controlled-use of chrysotile asbestos.[106]

5.523 In Europe, as in other countries, there are now requirements for training workers. In Canada, both levels of government require training at all workplaces. It is possible for training to be made available by industry. In fact, information and training is one of the most important elements of a company's preventive control programme. In line with the controls suggested at paragraphs 5.511 and 5.512, France could require through legislation that all construction workers handling asbestos products attend training sessions. France could also require that only designated, properly trained workers be allowed to work with those asbestos products that need to fall under a controlled regime.

5.524 During manufacture, controls such as wet processes and exhaust ventilation, essentially eliminate all exposure. On the work site, process changes are reduced by the industry manufacturing products requiring no, or virtually no, modifications on site. The controlled use approach includes the use of pre-cut and pre-drilled asbestos cement products, and provides for designated locations where chrysotile asbestos cement sheets or pipes are cut and drilled and where the appropriate controls are in place. The monitoring process is similar to that for other workplaces: all complaints are submitted to governmental inspectors for evaluation. The supplier has the responsibility for ensuring that all companies to which they supply have in place the proper equipment and training to ensure safe use of the product throughout its life cycle. Finally, the removal of high-density chrysotile products is carried out in accordance with government codes.

Question 5(c)

5.525 Both Dr. Henderson and Dr. Infante agree that, in many situations, when standards are properly applied, it is possible to maintain exposure below 0.1 f/ml. Also, as explained in Appendix A on the friction industry and Appendix B[107] on the asbestos cement industry, experience shows that a level below 0.1 f/ml can be achieved because the technology and work practices exist to control exposure during manufacture. No guarantee can be offered that there would never be a situation in which 0.1 f/ml might be exceeded as a peak exposure. However, there is no evidence that occasional peak exposures increase the risk of lung cancer or mesothelioma in chrysotile exposed workers. For example, the health experience of brake mechanics, i.e. no evidence of an increased risk of mesothelioma or lung cancer, is based on exposures that involved peak exposures, such as occurred during the blowing out of brake wear debris and the occasional grinding of brake linings. These operations involved short exposures above 0.1 f/ml. The actual concentrations associated with various

[105]*Memorandum of Understanding between the Government of Canada and the Asbestos Industry on Responsible-Use of Chrysotile Asbestos*, 1997.

[106]Canada notes that it should be recalled that his basis for risk assessment is based on the textile industry.

[107]See Annex IV to this Report.

tasks have been reported by Kauppinen and Korhonen,[108] and by Rödelsperger.[109] In spite of these short-term peak exposures, the average exposure of auto mechanics was less than 0.05 f/ml.

5.526 A person repairing their own brakes periodically nowadays (using disc brake pads mainly) would have extremely low cumulative exposures compared to full time auto mechanics and there is no reason for them to have any, even short term, exposures exceeding 0.1 f/ml. The risks associated with cumulative exposure to chrysotile at these levels would not be epidemiologically detectable for handymen handling friction or asbestos cement products.

5.527 Rödelsperger[110] made dust measurements on about 40 buildings sites in Germany. He reported peak exposures of more than 100 f/ml in the vicinity of a grinding machine used to cut asbestos cement sheets. However, when he used the standardized work histories of 61 roofers, who had a mean duration of exposure of 16 years, he found that their mean cumulative exposure was 1.6 fibre-years/ml. These measurements were made 20 or more years ago, with the products and technology available then and for regular construction workers. It is evident that even under these circumstances, lifetime cumulative exposures were low. Thus, a handyman, even if he did not take proper precautions would still have a low cumulative fibre exposure because peak exposures are of short duration and he would be at a very low, undetectable risk of health effects.

5.528 It is generally agreed that at the levels of exposure associated with the use of the modern high-density products, they would not even put a full-time worker at increased risk of asbestosis and, therefore, this would not be of concern for a handyman working occasionally with the product. It has been amply demonstrated that the risk of lung cancer increases with increasing cumulative lifetime exposure that combines duration and level of exposure. A person exposed at 0.1 f/ml for 40 years has a cumulative lifetime exposure of 4 f/ml-years. If that person worked on a project only once each week for four hours for 40 years, he would not achieve the same lifetime exposure unless he was exposed to 1 f/ml continuously for the four hours of exposure every time he was exposed for 40 years. Thus occasional peak exposures of a few minutes contribute very little to cumulative lifetime exposure which is important in evaluating the risk of chronic diseases such as lung cancer or mesothelioma.

5.529 Gardner[111] found no increased risk of lung cancer or other asbestos-related disease in a chrysotile asbestos cement plant where exposures were less than 1 f/ml. This was in a cohort of workers employed between 1941 and 1983. It is evident that any risk would have been well below the detection limit at 0.1 f/ml. A study of chrysotile cement production workers by Thomas[112] and Neuberger & Kundi[113] identified no chrysotile-related increased risk of lung cancer and Weill,[114] while reporting an increased risk of lung cancer in asbestos cement workers, found the increased risk only in

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[108]Kauppinen, T. and Korhonen, K., *Exposure to Asbestos During Brake Maintenance Of Automotive Vehicles by Different Methods*, (1987) 48 Am. Industr. Hyg. Assoc. J, pp. 499-504.

[109]Rödelsperger, K. et al., *Asbestos Dust Exposure During Brake Repair*, (1986) 10 American Journal of Industrial Medicine, pp. 63-72.

[110]Rödelsperger, K., Woitowitz, H.J. and Krieger, H.G., *Estimation of Exposure to Asbestos-Cement Dust on Building Sites*, in Biological Effects of Mineral Fibres, Vol. 2, J.C. Wagner Editor, 1980, International Agency for Research on Cancer: Lyon, pp. 845-853.

[111]Gardner, M.J., Winter, P.D., Pannett, B. and Powell, C.A., *Follow-Up Study of Workers Manufacturing Chrysotile Asbestos Cement Products*, (1986) 43 British J. of Industrial Medicine, pp. 726-732.

[112]Thomas, H.F., Benjamin, I.T., Elwood, P.C. and Sweetman, P.M., *Further Follow-Up Study of Workers from an Asbestos-Cement Factory*, (1982) 39 British Journal of Industrial Medicine, pp. 273-276.

[113]Neuberger, M. and Kundi, M., *Individual Asbestos Exposure: Smoking and Mortality - A Cohort Study in the Asbestos-Cement Industry*, (1990) 47 British Journal of Industrial Medicine, pp. 615-620.

[114]Weill, H., *Biological Effects: Asbestos-Cement Manufacturing*, (1994) 41 Ann. Occup. Hyg., pp. 533-538.

those with asbestosis. In this study, there was little evidence of asbestosis below 30-40 f/ml-years of exposure. This is about 0,75-1 f/ml continuous exposure for 40 years. Thus, there is little evidence to support a detectable increase in risk of lung cancer in workers with a 40 years cumulative lifetime exposure at 4 f/ml-years.

5.530 Any risk estimates obtained by linear extrapolation from high exposures to such low exposures are somewhat hypothetical and both Lash[115] and Camus[116] have shown that the risk estimates made by the U.S. Government have overestimated lung cancer risks.

Question 5(d)

5.531 Canada disagrees with the views of Dr. Henderson[117] and Dr. Infante that controlled use of chrysotile asbestos is not feasible for workers involved in the construction trade and that service and maintenance workers such as carpenters, plumbers, and electricians will experience peaks of exposures to asbestos that place them at risk. The nature of high-density chrysotile asbestos products is such that few of the trades listed above will ever need to work on the products, with the possible exception of demolition workers. Again, there is evidence that during demolition exposure concentrations associated with chrysotile asbestos cement products is very low.[118] Today, with chrysotile cement products and controlled use procedures, health risks become insignificant.

5.532 Recommended installation methods can eliminate the need to cut or drill into chrysotile-based products at construction sites, since those products are distributed in a variety of pre-cut and pre-drilled sizes, according to buyers' specifications. In fact, many asbestos cement products are pre-formed ready for use. They are factory-made to the correct size and shape including holes so that a minimum of on-site preparations is needed. Once installed, chrysotile asbestos cement pipes are below ground and pose no risk to workers. Even if dug up, they pose no risks unless comminuted, ground or sawed, and, when this is necessary, the use of appropriate tools and controls will keep the release of dust and exposure well within the level considered safe by the WHO. Chrysotile asbestos cement sheets are used for roofing and exterior building walls. Once installed, there is no need to modify the roof until the life of the product is over. Similarly, there is no need to modify chrysotile asbestos sheets used as walls once they have been installed. The product can be painted without fibre release.

5.533 Chrysotile cement products are unlikely to release fibres into the environment or breathing zones of workers such as janitors, plumbers, electricians, repair men, etc., unless these workers have to actually cut or drill the product. Unlike insulation products, there will rarely be a need for anyone to perforate, saw, or grind installed chrysotile cement products. Where cutting or drilling is required, hand tools and low speed power tools are recommended in combination with wetting to keep dust levels to a minimum. Dust levels for various types of on-site working have been measured both in laboratories and in the field and these facts showed that risks could be maintained below detection limit.

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[115]Lash, T.L., Crouch, E.A.C. and Green, L.C., *A Meta-Analysis of the Relation between Cumulative Exposure to Asbestos and Relative Risk of Lung Cancer*, (1997) 54 Occupational and Environmental Medicine, pp. 254-263.

[116]Camus, M., Siemiatycki, J. and Meek, B., *Nonoccupational Exposure to Chrysotile Asbestos and the Risk of Lung Cancer*, (1998) 338 N. Eng. J. Med., 1565.

[117]Henderson, answers to Questions 5(a) to (d).

[118]Hoskins J.A., *Chrysotile in the 21st Century*, UK, 1999, p.12.

Question 5(e)

5.534 Dr. de Klerk and Dr. Musk wrote that efficiency of controlled-use in the case of home handymen is outside their area of expertise. However, both Dr. Henderson and Dr. Infante have concluded that it is not possible to control exposure to chrysotile asbestos high-density products in non-occupational circumstances (occasional interventions by home handymen). Neither bases his conclusion on data. Dr. Henderson adds to his answer that although such risks are not quantifiable because of absence of data, these risks must be very small for lung cancer and mesothelioma, and non-existent for asbestosis.

5.535 Controlled use will reduce and even eliminate risks. The risk of chrysotile-related health effects is tied to cumulative exposure, that is, duration and level of exposure. Rarely will an individual under non-occupational circumstances achieve the exposure of a full-time worker. Occasional uncontrolled exposures for a handyman would not result in appreciable cumulative exposure. Data published by Brown[119] showed time-weighted average (TWA) levels during demolition of weathered asbestos-cement roofing between 0.3 and 0.6 f/ml. One can likely guess that a handyman would not practice such an activity more than 40 hours in 25 years. This would average out to a TWA of 0.015 f/ml for the year of this activity, and a TWA of 0.0006 f/ml each year of the worker's adult life. This is 1 million times less than past asbestos workers are. It is equivalent to exposure levels in schools containing ACM.[120]

5.536 Based on INSERM[121] and HEI-AR risk tables, which are based on mixed asbestos exposures, the resulting lifetime cancer risk would be between 10 and 20 in a million depending on the time occurrence of this exposure scenario. More accurately however, the lifetime risk would be near zero per million, based on chrysotile friction workers who were exposed to similar fibres (species and dimension wise), and about 1 in a million, based on the risks of past chrysotile miners and millers. The casual user of a high density product, even if the product were weathered, is not likely to be at any increased risk of an asbestos related disease. If the supplier follows through on the requirements of controlled use, the casual purchase of chrysotile asbestos-cement products by the handyman will not be possible. However, there is probably no way of stopping any individual from doing something to any product if they can obtain it. This is a problem that exists for any product many of which pose serious health risks if abused.

Question 6(a)

5.537 Canada respectfully disputes the conclusions of the experts regarding the risk from substitute fibres, and with respect to one expert, the ability of substitutes to serve as suitable replacements to chrysotile. Canada notes that the treatment of the issue by two of the experts is terse, comprising only several sentences. To their credit, Drs. de Klerk and Musk indicate that the use and control of substitute fibres is not within their areas of special expertise. They offer some responses nonetheless. Canada is concerned, in particular, by their lack of familiarity with the relevant studies and actual modes of production, use and disposal of substitute fibres. For example, they apparently are unaware of research conclusively demonstrating the significant health risks from exposure to refractory ceramic fibres (RCF), which are discussed below.

5.538 This concern applies to Dr. Infante as well. Dr. Infante further appears unaware of (or ignores) recent research demonstrating that chrysotile is less biopersistent than many substitute fibres.

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[119]Brown, S.K., *Asbestos Exposure During Renovation and Demolition of Asbestos-Cement Clad Buildings*, (1987) 48 Amer. Ind. Hyg. J., pp. 478-486.

[120]Health Effects Institute – Asbestos Research, *Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge (Executive Summary)*, Cambridge, 1991, pp. 1-11.

[121]INSERM Report.

Dr. Infante also ignores the population the experts agree are most at risk from exposure to any fibre – tradesmen – when he concludes (without support or, even, explanation, Canada notes) that the "nature of the production process makes substitutes more amenable to control than asbestos fibres." Assuming he is not again conflating chrysotile with amphiboles when referring to "asbestos," his point, were it true, would be irrelevant. The experts agree that chrysotile and chrysotile products can be safely mined and produced. The key is exposure to tradesmen. And, for that exposure, no rationale exists suggesting that the ability to impose effective controls differs based on the type of the fibre.

5.539 Dr. Henderson, for his part, recognises that, as with all fibres, the pathogenicity of substitutes is defined by the "3Ds" (dimension, dose, durability). He seems also to understand that, due to the (lack of) historical use of substitutes, we cannot fully know the risks of using them.[122] However, he then seems to ignore the importance of these facts.

5.540 All of the experts fail to take into account several very important factors. First, the chrysotile products at issue in this proceeding are quite few. Second, the exposure levels during the manufacture, use and disposal of these products are extremely low. Third, the data demonstrate that these few products have been and can be used without detectable health effects in humans. Moreover, in order to assess whether a substitute is safer to use than chrysotile in a product: (i) it is fundamental that the characteristics of the fibres being compared be those of the fibres as they are used in the product or as they are released from the product throughout the product's lifecycle; (ii) it is essential that data on at least the key parameters (exposure, biopersistence and dimensions) be available to make this assessment. Unfortunately, the experts have not addressed these topics. In short, the experts have based their opinions on very limited, if any, data. While the experts reach conclusions that various substitutes (PVA fibres, glass fibres, cellulose and para-aramid fibres) are safer to use than chrysotile, they provide no systematic comparison of risks and very limited, questionable scientific data in support of their opinions.

5.541 Canada presents below a survey of the studies and concepts that the experts ignored. These studies give a picture of risks from substitute fibres starkly different from that suggested by the Panel's experts. As demonstrated below, the situation concerning risk from substitute materials is as Canada set out in its factual arguments.[123]

(i) The Fibres to Compare

5.542 Experimental data for a wide range of fibres have shown that the physical characteristics (diameters, lengths, density) of fibres are important in determining their respirability, when they are deposited in the respiratory system, and their capacity to induce fibrosis and cancers. Further, the risk of effects also depends on dose (exposure). Thus, differences in the risk of disease in various industrial sectors would be expected to occur because of differences in these, as well as other factors. As the characteristics of chrysotile and any substitute fibres are likely to be dictated by the product in which they are used, it is not appropriate to assess the risks associated with friction products or asbestos cement products using data from other industrial sectors. The data that should be available and used for the purpose of comparing risks should be those for the fibres as used in the specific products under review. Canada's presentation proceeds on this basis.

5.543 Davis[124] pointed out that while materials like wool, cellulose and other fibres have in some cases been used for many years, they are now being used in quite different applications, about which

[122]Henderson, response to Question 6(b).
[123]See above Section III.A.6.
[124]Davis, J.M.G., *The Toxicity of Wool and Cellulose*, (1996) 12 J. Occ. Health and Safety Australia and New Zealand, pp. 341-344.

knowledge is very limited. As a consequence, the characteristics of the fibres used in the newer applications may not be the same as those in the conventional products manufactured in the past. Such changes can modify the respirability and biological activity of the materials. There is a further complication for substitutes that is not addressed by the experts. This is the fact that substitution does not always involve replacement of chrysotile by a single fibre, but often by several different materials or substitute fibres. For example, cocktails of fibres are needed to meet technical requirements in friction products. In addition, when substituting for chrysotile, other materials such as silica or other fibres, fire retardants or biocides must often be added. These agents may themselves be toxic or carcinogenic, and may act synergistically.

(ii) The Outcomes to be Measured

5.544 While it is reasonable to compare the risks of lung cancer and mesothelioma between the various fibre types, it must be remembered that different sized fibres may lead to fibre deposition at different locations in the respiratory system. For example, if more fibres of one material than another are likely to be deposited in the nasal passages, one should consider the possibility of an increased frequency of nasal cancer in evaluating the substitute. Dr. Infante mentioned the increased risk of nasal cancer in woodworkers, which has been well established.[125] This might raise a question concerning the sources of the cellulose used as a substitute and whether controls are in place to avoid exposure to cellulose from woods that have caused such cancers. Also, some materials may cause dangerous allergic responses. Certain glass fibres cause skin irritation. Harrison[126] notes that there are indications of an accumulation of oligomers in the kidney in some circumstances, so that attention should be given to the molecular weight of PVA used "especially if a smaller diameter material were to be produced."

5.545 In considering risks, the composition of the dusts and fibres to which workers are exposed when handling the "raw substitute" materials, manufacturing the product, cutting, grinding, manipulating or disposing of the product also must be considered. For example, it is important to know whether the fibres of para-aramid, PVA or cellulose are opened (fibrillised) or comminuted during preparation or manufacture of the product? Does manipulation, sawing or drilling of the product give rise to narrower diameter respirable "fibres" such as result with polyester fibres during weaving? Do these fibre fragments have biological significance? What are actual use concentrations? It must be remembered throughout that there exists a substantial body of information concerning chrysotile. Unfortunately, in the case of substitutes, there are rarely any human epidemiological data available and even experimental data are limited. Perhaps this fact led Dr. de Klerk to conclude that "[G]iven the comparative lack of knowledge about the health effects of substitute materials, the continued use of chrysotile under [controlled] circumstances seems sensible.[127]

(iii) The Essential Data

5.546 The data that need to be compared in an evaluation of the relative safety of chrysotile and substitutes include the following:

- Epidemiological data that provide direct evidence of the risks associated with the products.

[125]Infante, answer to Question 6(c).

[126]Harrison, T.W., Levy, W.S., Patrick, G., Pigott, G.H. and Smith, L.L., *Comparative Hazards of Chrysotile Asbestos and its Substitutes: A European perspective*, (1999) Environmental Health Perspective, 107.

[127]de Klerk, N.H. and Armstrong, B.K., *The Epidemiology of Asbestos and Mesothelioma", in Malignant Mesothelioma*, Henderson, D.W. et al., eds Hemisphere Publishing, New York, 1982, p. 231.

- Experimental data by inoculation of fibres or by inhalation experiments in experimental animals.
- Dimensions of fibres in the respirable airborne dust during the manufacture of the product.
- Dimensions of fibres in the respirable airborne dust during the use of products containing the fibre.
- Dimensions of fibres in the lungs of workers engaged in the manufacture of products containing the fibre.
- Dimensions of fibres in the lungs of persons exposed during the use of products containing the fibre.
- Dimensions of fibres in the respirable airborne dust and in the lungs following exposure during the disposal of the fibre or products.
- The biopersistence of the fibres in humans and animals.
- The cumulative exposure (i.e.: concentration x time) of workers engaged in all phases of manufacture, use and disposal of the product.
- Data on alterations or modification of the fibres chemically, physically and biologically during their life cycle that might affect their potential to cause health effects.

5.547 Even if one were to narrow the requirements to a smaller number of key parameters such as fibre dimensions, biopersistence and exposure-response, the available data are still inadequate to provide a credible basis for an adequate comparison. Thus, the unqualified wholesale affirmation that "substitutes are safer than chrysotile" is not well founded and potentially very dangerous. For example, prior to the finding of a very high risk of mesothelioma for persons exposed to very low concentrations of fibrous zeolite erionite in Turkey, there had been no indications world-wide that such fibres might after 30 years from first exposure at such very low levels produce such a high rate of mesotheliomas in humans. In South Africa, crocidolite had been used for about 60 years before Wagner[128] reported that mesotheliomas were associated with crocidolite exposure. In humans, mesotheliomas do not occur until 40-60 years after first exposure. Thus, caution is needed in the absence of data regarding substitute fibres. As one expert stated: "better the devil you know" than the devil you do not know.[129]

Question 6(b)

(i) Dimensions

5.548 The experts present no data that show that the dimensions of all fibrous substitutes are outside the respirable size range during the substitute product's lifecycle. This is because no such data exist.

[128]Wagner, J.C., Newhouse, M.L., Corrin, B., Possister, C.E. and Griffiths, D.M., *Correlation between Fibre Content of the Lung and Disease in East London Asbestos Factory Workers*, (1988) 45 British J. of industrial Medicine, 305.

[129]de Klerk, answer to Question 6(a).

(ii) PVA & Aramid Fibres

5.549 Views from the experts appear to be mixed. Dr. Henderson quotes Harrison's review stating that PVA and aramid fibres are too large to be respirable. Dr. de Klerk states that all substitutes except glass (cellulose, aramid, PVA) produce a larger proportion of non-respirable fibres than chrysotile but respirable fibres are similar for all substances. Dr. Musk offers no opinion. Dr. Infante states that PVA fibres are "mostly" in the size range 10-16 µm and aramid fibres 10-12 µm. However, he notes, quite correctly, that, as mentioned below, the aramid fibres can and do split into fibrils of about 0.2 µm in diameter.

5.550 In assessing fibre respirability, none of the experts accounts for the fact that respirability depends on density as well as fibre diameter. The densities of PVA and para-aramid fibres are both considerably less that that of chrysotile. This means that much larger diameter substitute fibres would be respirable. In fact, the upper limits of diameters that are respirable for these fibres, as reported by Harrison[130], are approximately 7 µm and 6-7 µm respectively. The equivalent upper level diameter for chrysotile is about 3-3.5 µm. Thus, fibres of much greater diameter can penetrate into the alveolar region of the lung. A review of the available information in the literature is that there is a general opinion without data that the respirable fraction of PVA fibres is small. However, there do not appear to be any data on the dimensions of airborne fibres during mixing with cement or other materials, or as released from the products during processing and use.

(iii) Glass and Cellulose Fibres

5.551 As far as cellulose and glass fibres are concerned, none of the experts provided any actual measurement data on the sizes or respirability of the fibres. Also, the dimensions of fibres at various stages of the processing, use and disposal of cellulose have not been reported. The actual dimensions of fibres in the airborne dust will depend on the specific glass fibres used and how they were prepared.

(iv) Biopersistence

5.552 It is well known that biopersistence is a key parameter. Indeed, the human evidence for chrysotile indicates that it is likely to be one of the main reasons why chrysotile is less dangerous than the amphiboles in respect to mesothelioma risk. This is clearly recognized by three of the four experts, as well as by INSERM.[131]

(v) Cellulose

5.553 Drs. Infante, Henderson and de Klerk recognise that cellulose is durable in the lung. In fact, the data show that some cellulose fibres have half lives of about 1000 days in the lung, which are many times longer than even those published data for amphibole fibres, much less chrysotile fibres.[132]

(vi) PVA

5.554 Dr. Musk and Dr. Henderson had no comment on PVA durability. Dr. de Klerk presented no data, but expressed the view that PVA was less durable than chrysotile. Davis, in a review in 1998, found no published data on the biopersistence of PVA fibres. Data was not published until 1999,

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[130]Harrison, T.W., Levy, W.S., Patrick, G., Pigott, G.H. and Smith, L.L., *Comparative Hazards of Chrysotile Asbestos and its Substitutes: A European perspective*, (1999) Environmental Health Perspective, 107.

[131]See Canada's comments on Question 3.

[132]Muhle, H., Ernst, H. and Bellman, B., *Investigation of the Durability of Cellulose Fibres in the Rat Lungs*, (1997) 41 Ann. Occup. Hyg., pp. 184-188.

when Harrison [1999] reported that PVA "will degrade very slowly, if at all in the lung."[133] There do not appear to have been any systematic studies of the biopersistence of PVA fibres, a crucial parameter in assessing the hazard associated with PVA fibres.

(vii) Para-aramid Fibres

5.555 Based on a study by Searl,[134] who compared chrysotile and para-aramid fibres, the general view of the experts is that para-aramid fibres are less biopersistent. However, Searl failed to check the lung tissue to confirm that the retained fibres were chrysotile. Based on studies using a standard protocol, Dr. David Bernstein has found that the biopersistence of chrysotile is in fact less than that of para-aramid fibres.[135]

(viii) Glass Fibres

5.556 Drs. Musk, de Klerk and Henderson presented no data on the biopersistence of glass fibres. Dr. Infante, without identifying the specific glass fibres, reports that glass fibres are less biopersistent than chrysotile. In fact, the recent work by Dr. Bernstein, in which the same protocol was used as for synthetic fibres, found that long [i.e.: > 20um] pure chrysotile fibres are removed from the lung faster than most, if not all, of the glass fibres reported in the published literature.[136]

(ix) Chrysotile

5.557 As far as chrysotile is concerned, it is well accepted that chrysotile is readily removed from the lung. This is why the lungs of chrysotile millers and miners, exposed to chrysotile, have been found at autopsy to contain more tremolite (an amphibole asbestos mineral) than chrysotile.[137] The chrysotile cleared, but the tremolite fibres remained in the lung because of their much greater biopersistence. There are various estimates of the half time for chrysotile clearance. Oberdöerster[138] studied baboons and estimated a 90-110 day half time for chrysotile fibres. The study by Searl was mentioned above. The estimates by Dr. Bernstein are even shorter (< 10 days).[139] For direct comparison purposes, the clearance rates for fibres in the same ranges of dimensions must be studied. In addition, it is crucial that the fibres be tested using the same methodology. The studies by Bernstein best fit these criteria and show that, size for size, chrysotile has a very short half-life.

Question 6(c)

(i) Exposure-Response

5.558 In the absence of exposure-response data, it is not possible to quantify the risks associated with the various fibres. The question to be addressed is not: is one material more dusty than another? Nor is it: is the concentration higher when working with one material compared to another? Rather,

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[133]Davis, J.M.G., *The Biological Effects of Fibres Proposed as Substitutes for Chrysotile Asbestos: Current State of Knowledge*, 1998.

[134]Searl, A., *Clearance of Respirable Para-Aramid from Rat Lungs: Possible Role of Enzymatic Degradation of Para-Aramid Fibrils*, (1997) 41 Ann. Occup. Hyg., pp. 148-153.

[135]Bernstein, D.M., Graph on Biopersistence of p-Aramid Fibres.

[136]Bernstein, D.M., *Summary of the Final Reports on the Chrysotile Biopersistence Study*, Geneva, 1998, document submitted to the Panel by Brazil as a Third Party (see above Section IV).

[137]Rowlands, N., Gibbs, G.W. and McDonald, A.D., *Asbestos Fibres in the Lungs of Chrysotile Miners and Millers - A Preliminary Report*, (1982) 26 Ann. Occup. Hyg., pp. 411-415.

[138]Oberdörster, G., *Macrophage-Associated Responses to Chrysotile*, (1994), 38 Ann. Occup. Hyg., pp. 601-615.

[139]Bernstein, D.M., *Summary of the Final Reports on the Chrysotile Biopersistence Study*, Geneva, 1998, document submitted to the Panel by Brazil as a Third Party (see above Section IV).

the question that must be asked is: what is the risk for workers manufacturing or using the product? The decision on which fibre is safer has to be made on the basis of an assessment of the risk of disease for workers when manufacturing and using a product containing chrysotile compared to that when manufacturing and using the same product containing the substitute when subjected to the same or equivalent handling.

5.559 Three sources of data might be considered: experimental animal studies; human (epidemiological) studies; and *in vitro* studies. The latter (*in vitro*) are of little value for estimating risk as they only involve tests of, for example, biological activity in cells isolated from the processes which occur in a complete organism. Thus, they are an inadequate basis of comparison to the effects of inhalation on animals, much less humans.

(ii) Animal Studies

5.560 The first approach involves the exposure of animals to fibres of well-defined characteristics and concentrations by inhalation and following them for their lifetimes. Such studies have been done for a wide range of synthetic mineral fibres. Problems with this approach are many, as has been demonstrated in the considerable work done in recent years on synthetic mineral fibres. First, the animal species may have a limit on the size of fibre that it can inspire. Second, there are marked differences in the sensitivity of different animal species. For example, a refractory ceramic fibre (RCF) which produced one or two mesotheliomas in rats, produced mesotheliomas in 40 per cent of the hamsters exposed. Third, the lifetime of rats is about two years. In order to produce an effect within the lifetime of the animals they are subject to enormous exposures. Such exposures can produce the abnormal situation of lung overload so that the real reason for any biological effect is not clear. Fourth, an animal must produce an effect within two years (before it dies of natural causes). If biopersistence is important, fibres that are readily removed in humans over the course of a human's life are not removed from the lung of an experimental animal because of the high exposures and shorter life. Fifth, the interpretation of much of the experimental work must be done with caution, because until recently, fibre exposures were reported on mass not number basis. As the materials tested can have quite different dimensions, the same mass can lead to exposures involving considerably different concentrations of fibres on a number basis.

(iii) PVA

5.561 There are no studies relating the long-term effects of exposure to PVA fibres.

(iv) Cellulose

5.562 Studies that have been done with cellulose have shown that it initiates a severe inflammatory response[140] and fibrosis.[141] Unfortunately, no chronic exposure data have been published.

(v) Glass Fibres

5.563 While there have been many studies of glass fibres, the only study in which the same methodology was applied to the study of synthetic mineral fibres and chrysotile asbestos is that of Hesterberg.[142] He found that while there is an increased risk of lung cancer identified at high

[140]Hadley, J.G., Kotin, P. and Bernstein, D.M., *Subacute (28 Day) Repeated Dose Inhalation of Cellulose Building Insulation in the Rat*, (1992) The Toxicologist, 225 (abstract).

[141]Muhle, H., Ernst, H. and Bellman, B., *Investigation of the Durability of Cellulose Fibres in the Rat Lungs*, (1997) 41 Ann. Occup. Hyg., pp. 184-188.

[142]Hesterberg, T.W., Miller, W.C., Theveney, Ph. and Anderson, R., *Comparative Inhalation Studies of Man-Made Vitreous Fibres: Characterization of Fibres in the Exposure Aerosol and Lungs*, (1995) 39 Ann. Occup. Hyg., pp. 637-653. Hesterberg exposed rats to a concentration of 10,000 WHO fibres/ml of chrysotile,

concentrations (as for glass and other fibres) the animal data suggest that at low level exposure, the risk associated with the chrysotile exposure is considerably less than that associated with the synthetic mineral fibres tested.

(vi) Para-aramid Fibres

5.564 While in recent years the information base concerning aramid fibres has increased greatly, there remain several issues. The only data are those derived from experimental studies in animals. While studies of biopersistence suggest that long fibres are shortened by enzymes in the lungs of animal experiments (Searl) and hence removed from the lung, the situation in humans is not known. Two researchers (Davis[143] and Pott[144]) have produced mesotheliomas by intra-peritoneal injection of these fibres, so their potential to produce mesotheliomas cannot be dismissed. The interpretation of "proliferative keratin cysts" observed during inhalation experiments remains unclear.[145] Minty et al., in a criteria document for an occupational exposure limit (OEL) in the UK, summarized what was known about the para-aramid fibres at that time and drew several parallels with chrysotile. For example they state that "[T]he balance of evidence suggests that respirable aramid fibres possess a low potential to produce mesothelioma which is likely to be at least as low as with chrysotile.[146]

5.565 Referring to chrysotile, they conclude that mesothelioma "would only be detectable following very heavy and prolonged exposures." The recent evidence that the mesothelioma risk for chrysotile miners and millers is associated with tremolite, will render the threshold of mesothelioma for downstream workers even more remote. These authors considered a clear no-effect level of 2.5 f/ml for pulmonary toxicity and a recommended OEL of 0.5 f/ml to allow for "uncertainties in interspecies differences."

(vii) Epidemiological Data

(i) PVA

5.566 Drs. Musk, de Klerk, Henderson and Infante did not identify any epidemiological studies of PVA fibre workers. In fact, there is one study involving a small number of PVA fibre production workers (about 400 exposed employees).[147] Even though the length of exposure thus far is quite short, already two lung cancer deaths have occurred in the cohort to date. Clearly a much longer follow-up is needed. Regarding mesothelioma, it must be noted that with such a small population, even if half were dead and there were one mesothelioma, the risk would be 0.5 per cent which is more than the risk of mesothelioma found in Quebec miners and millers exposed to tremolite contaminated

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which resulted in 18.9 per cent lung tumours. Rats exposed to 232 f/ml of one type of glass fibre resulted in 5.9 per cent lung tumours, with 4.4 per cent lung tumours reported with other man-made vitreous fibres and 13 per cent with a RCF sample. The air control resulted in 1-3 per cent tumours (At 1000 f/ml, the risk of lung tumours would be just under 2 per cent which is well within the rate of tumours in the control animals).

[143]Davis, J.M.G., *Carcinogenicity of Kevlar Aramid Pulp Following Intraperitoneal Injection into Rats*, (1987) Technical Memorandum No. TM/87/12 Published by the Institute of Occupational Medicine, Edinburgh, Scotland.

[144]Pott, F., Roller, M., Ziem, U., Reiffer, F.J., Bellman, B., Rosenbruch, M. and Huth, F., *Carcinogenicity Studies on Natural and Man-Made Fibres with Intraperitoneal Tests in Rats,* (1989) In: Non-Occupational Exposure to Mineral Fibres. J. Bignon, J. Peto, K. Saracci eds. IARC Scientific Publication No. 90 Publ. International Agency for Research on Cancer, Lyon, pp. 173-179.

[145]IARC International Agency for Research on Cancer (1997), Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol. 68.

[146]Minty, C.A., Meldrum, M., Phillips, A.M. and Ogden, T.L., *P-aramid Respirable Fibres Criteria Documents for an Occupational Exposure Limit*, HMSO (1995).

[147]Morinaga, K., Nakamura, K., Koyama, N. and Kishimoto, T., *A Retrospective Cohort Study of Male Workers Exposed to PVA Fibres*, (1999) 37 J. Industr. Health, pp. 18-21.

chrysotile. (Also, the Panel should note that there were no mesotheliomas among 1267 deaths in chrysotile exposed friction product manufacturing workers exposed to chrysotile.) Thus, this study cannot detect either mesothelioma or lung cancer risks as low as at that already known for chrysotile. Clearly, there are no human data on which to assess risk to conclude that the risk is less than it is for chrysotile either per f/ml of exposure or globally from work with products manufactured using PVA fibres.

(ii) Cellulose

5.567 Dr. Infante states that there are three studies in which cellulose exposures have been investigated, but he does not identify them. The other two experts do not suggest any epidemiological data. Studies in which there is no overall increase in mortality from lung cancer are not adequate to investigate the risk of exposure. To assess this risk, the relationship between lung cancer and cellulose fibre exposures on a per fibre basis must be, but has not been, examined.

(iii) Para-aramid Fibres

5.568 None of the experts reported any epidemiological data. Clearly, para-aramid fibres can be inhaled, as experimental animals inhaled them. However, because para-aramid fibres have been used for such a short time, there are no data on the relationship between levels of fibre exposure and the risks of lung cancer, mesothelioma or other adverse effects for persons working with this substitute or products manufactured using it.

(iv) Glass Fibres

5.569 There have been several studies of workers exposed to glass fibres during fibre manufacture. Studies have also included rock [stone] and slag wool exposures. The latter were associated with an increased risk of lung cancer even at very low levels of exposure. Doll[148] concluded that the risks from such exposures were greater than those associated with chrysotile asbestos. Doll summarized the situation as follows: "an occupational hazard of lung cancer has been demonstrated in the rock and slag wool section of the industry and possibly the glass wool section." The human evidence since that time has not dispelled concern about the risks associated with these fibres. This question is still not resolved.

5.570 Dr. Infante[149] and co-workers once reached the same conclusion for glass fibre (although he has changed his opinion in his current report). In his report, Dr. Infante mentions that after speaking with workers, he now thinks that there was asbestos exposure at the plant studied by Shannon in Ontario, Canada, where a high level of risk of lung cancer was found in glass fibre workers. A recent discussion with Dr. Harry Shannon about his study of glass fibre workers reveals that to his recollection, no one had raised the question of asbestos as a potential confounder in his study. He noted that, as the study was published many years ago, it seems unlikely that this issue – if it actually existed – would not have been raised and studied, especially by the glass fibre industry.[150] Clearly, no new analyses have been done, so the impact of supposed asbestos exposure, if it took place, is not known. Dr. Infante's reversal of opinion does not seem justified, as no new data are presented. For example, it is not known whether the "asbestos exposed workers" had high or low glass fibre exposure. If they had low glass fibre exposure, then the risk associated with the glass fibre exposures

[148]Doll, R., *Mineral Fibres in the Non-Occupational Environment: Concluding Remarks*, in Bignon, J., Peto, J. and Saracci, R., eds., Non-Occupational Exposure to Mineral Fibres, IARC Scientific Publication No. 90, 1989, pp. 511-518.

[149]Infante, P.F. et al., *Fibrous Glass and Cancer*, (1994) 26 Am. J. Industrl. Med., pp. 559-584.

[150]Gibbs, G., Phone Communication.

might increase. Thus, without additional analyses, the best estimates at present are the original analyses by Shannon.[151]

5.571 It was noted earlier that exposure levels during production may not be the same as those during product use. While it has not been possible to find data on the use of glass fibres in chrysotile cement or friction products, there has been an estimate of the risk associated with glass fibres as installed in homes. In this study, Wilson[152] used animal data to derive lung cancer risk estimates for glass fibre exposure. They assumed an exposure of 1f/ml for one year based on available data and estimated that the lung cancer risk in smokers associated with blown glass wool without binder in a smoker without a respirator would be 2.4×10^{-4}. If one uses the same methodology as applied by them to derive a chrysotile estimate (based on epidemiological data), but for friction product manufacture, the risk would be very much lower: $0.12 \times 0.00058 = 0.00007$ or 7×10^{-5}. This is a lower risk than calculated for glass fibres. In fact, there is no demonstrated increased risk of lung cancer in the friction industry, so even this chrysotile risk is hypothetical and certainly an overestimate. Wilson acknowledges this in their paper.

5.572 In this light, it is safer to work with chrysotile in friction products than to work with glass fibres. While it might be argued that there has been no report of an increased risk of mesothelioma in humans as a result of manufacturing glass fibres, in the case of chrysotile, there is greater confidence concerning this lack of risk because there is no evidence of an increased risk of mesothelioma associated with friction products throughout their lifecycle, and the studies are far more voluminous and varied in approach. There are no systematically gathered data available concerning downstream risks for glass fibres as used as a substitute in cement or friction products. Similarly, with regard to the asbestos cement industry, Harrison[153] reports that most studies have not found an increase in mesothelioma; certainly this is true for chrysotile asbestos cement plants. Thus, it is evident that there are clear no epidemiological or experimental data to conclude that "glass fibres" are safer than chrysotile, indeed, there is evidence to suggest the contrary.

5.573 In summary, the experts have based their opinions on very limited, if any, data. The data that do exist suggest that the conclusions of the Panel's experts concerning the relative safety of the substitutes and chrysotile at low concentrations are incorrect.

2. The European Communities

(i) Introduction

5.574 Each of the four scientific experts appointed by the Panel has recently responded to the points which the Panel wished to clarify. The European Communities note that the four experts consulted unanimously and unambiguously corroborate the analysis that led France to adopt the Decree 96-1133 banning asbestos. This analysis was communicated to the Panel in the two written submissions of the European Communities of 21 May and 30 June 1999 and is based on the following points:

(a) All forms of asbestos, including chrysotile asbestos, are carcinogens, and there is no scientifically established threshold below which exposure to asbestos would be without risk for humans;

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[151]Shannon, H.S. et al., *Mortality Experience of Ontario Glass Fibre Workers - Extended Follow-Up*, (1987) 31 Ann. Occup. Hyg., pp. 657-662.

[152]Wilson, R., Langer, A.M. and Nolan, R.P., *A Risk Assessment for Exposure to Glass Wool*, 30 Regulatory Toxicology and Pharmacology, pp. 96-109.

[153]Harrison, T.W., Levy, W.S., Patrick, G., Pigott, G.H. and Smith, L.L., *Comparative Hazards of Chrysotile Asbestos and its Substitutes: A European Perspective*, (1999), Environmental Health Perspective, 107.

(b) exposure to asbestos, including chrysotile asbestos, is the cause of many cancers, the vast majority of which affect secondary users, particularly workers coming into contact with materials containing asbestos, including asbestos cement;

(c) so-called "controlled" use of asbestos is in fact impossible in practice;

(d) there are asbestos substitutes which are far less dangerous for human health.

5.575 In this document, the European Communities do not wish to make systematic and detailed comments on all the replies by the four experts consulted, but will simply refer to the main conclusions and give a summary of their replies in the annex.[154]

(ii) The four experts consulted agree that all types of asbestos, including chrysotile, are carcinogens and that there is no established threshold under which exposure to asbestos is without risk for humans

5.576 The four scientific experts unanimously consider that chrysotile asbestos, as well as amphiboles, can cause mesothelioma and lung cancer *inter alia*.

5.577 The four experts also unanimously agree that there is no scientifically established threshold below which exposure would not pose any risk of cancer for humans. All the experts state that the risk of cancer is proportional to the cumulative level of exposure and all consider that the non-threshold linear model is the most scientifically appropriate model for guaranteeing the level of health protection decided upon by France in this particular case. This explains and confirms that the non-threshold linear model has always been used, without exception, by the authorities in all those countries that have so far carried out scientific assessment of the cancer risk.

(iii) The four experts consider that exposure to asbestos, including chrysotile asbestos, is the cause of many cancers that mainly affect secondary users, particularly workers in contact with materials containing asbestos, including asbestos cement

5.578 The four experts consider that the vast majority of the risks concern so-called "secondary" users[155], in other words, workers making interventions (building workers, electricians, plumbers, maintenance workers, handymen, etc.) because of their large number and the nature of their activities, even if the individual risks are sometimes lower.

5.579 For example, most cases of mesothelioma now affect this category of workers in all the industrialized countries, including Canada (Quebec) and Australia, countries which produce asbestos. The four experts point out that the levels of exposure in the course of occasional contacts with asbestos cement products are very high, much higher than the levels at which a risk of cancer has been definitely and scientifically established.

(iv) The four experts consider that the so-called "controlled" use of asbestos is not practically possible

5.580 The four experts unanimously agree that so-called "controlled" use aimed at ensuring a constantly low level of release of the fibres into the atmosphere is absolutely impracticable in the vast majority of work situations where workers have to deal with friable or non-friable materials containing asbestos.

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[154]See Annex V to this Report.

[155] See factual arguments by the EC, Section III.A.4.

5.581 The four experts consider that it might be possible in very special situations where a small number of workers carry out a very precise task. They also indicate that interventions on materials such as asbestos cement can release very large quantities of asbestos fibres; that protective equipment is not or not always effective and not always used; that the recommended procedures are rarely or incorrectly followed in small enterprises such as those in the building sector; that it is quite impossible to apply them to non-professionals (for example, handymen, etc.).

E. SUPPLEMENTARY REMARKS FROM DR. HENDERSON[156]

1. Concerning the comments from the European Communities

5.582 The comments from the European Communities are very brief, occupying only four pages in English translation, so that only a short comment is needed. The tabular summary of the four experts' reports appears to represent a fair précis of my conclusions and opinions, if an oversimplification. In para. 5.579, the European response refers to Canada (Quebec) and Australia as countries that produce asbestos. As pointed out in para 5.27, Australia is no longer an asbestos producer.

2. Concerning the comments from Canada

5.583 At 62 pages and with over 50 Annexes, the comments from Canada are far lengthier than the response from the European Communities; the Canadian documents include new information, necessitating more extensive discussion. Some general comments follow; other issues are discussed later under specific sub-headings.

5.584 In para. 5.441, the comment is made that some of the answers from the experts "appear not to distinguish between chrysotile and amphibole exposure" or between "modern uses ... and historical uses ... ." Throughout my own Report, I tried to make this distinction wherever appropriate, and my answers to the Panel's questions deal almost exclusively with chrysotile (like EHC 203 [1]) -e.g. my discussion of the risks to brake mechanics[157] and the tabulation of risk estimates for lung cancer and mesothelioma (Tables 12 and 13 in paras. 5.203 and 5.205. At the same time, it seems worth reiterating that commercial chrysotile from Canada on average contains variable trace amounts (about <1 per cent) of tremolite (fibrous tremolite is a non-commercial amphibole; e.g. please see EHC 203). In relation to Canada's concerns about the "experts' conclusions on tradesmen" (e.g. building construction workers), my perspective seems to concur with the IPCS/WHO monograph on chrysotile (EHC 203):

> "... (c) Some asbestos-containing products pose particular concern and chrysotile use in these circumstances is not recommended These uses include friable products with high exposure potential. Construction materials are of particular concern for several reasons. The construction industry workforce is large and measures to control asbestos are difficult to institute. In-place building materials may also pose risk to those carrying out alterations, maintenance and demolition" ... [p 144].

5.585 My recognition that chrysotile is substantially less potent than the amphiboles on a fibre-for-fibre basis for mesothelioma induction - and that present exposures overall are substantially less than in the past - explains why my Report dwelt mainly on workplace exposures (e.g. to building materials and friction products). Non-workplace exposures (e.g. non-occupational exposures including household contact or neighbourhood exposures [2-4]) - and exposures to friable insulation materials - received less attention and were included mainly to put the present situation into historical perspective.

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[156]For complete references of the documents quoted in this Section, see Annex III to this Panel Report.

[157]For decades, brake blocks and brake linings used in Australia have contained Canadian chrysotile asbestos only, with no added amphiboles.

5.586 In para. 5.489, the Canadian comments state that: "... He [Henderson] believes amphiboles may be greater than 60 times more likely than chrysotile to induce mesothelioma ... ." In fact, in para. 5.103, I had stated that:

> "There is general though not universal agreement of a differential potency between the amphiboles versus chrysotile for mesothelioma induction; in this respect, the amphiboles are substantially more potent, with estimates ranging from 2-4X, to 10X, to 12X on a fibre-for-fibre basis, to 30X, to a 30-60X greater potency, or more (e.g. please see EHC 203)."

5.587 Later in my Report (paras. 5.141 and 5.413), I gave my estimate that chrysotile has a potency $1/10^{th}$-$1/30^{th}$ the carcinogenicity of crocidolite *for the mesothelium.* This estimate has not changed.

5.588 Some clarification of my answer to Question 1(b)[158] from the Panel seems necessary, taking into account Canada's quotation of my view and the comment that: "Canada takes note that ... [this is] ... overwhelmingly a workplace issue, and therefore not related to the 'handyman'." In my answer, I took "workplace" to refer to any situation where work of any type is carried out (e.g. cutting, sawing, drilling, grinding, rasping or sanding of asbestos-cement building products), whereas I interpreted the expression "a larger part of the population" to refer to general environmental exposure to asbestos (e.g. simple occupancy of buildings, or potential exposure of urban dwellers in general to asbestos derived from the brakes of passing vehicles). Obviously, any risks to handymen who carry out maintenance on homes only occasionally will be much less than the risks to professional tradesmen such as carpenters, who work day in, day out at building construction sites - because the frequency and duration of exposures for the handyman will be less (with lower cumulative exposures), assuming the types of asbestos to be the same. However, this may not always be so. For example, I know that some "handymen" in Australia specialize in buying and living in dilapidated houses, to carry out extensive renovations on these dwellings (e.g. throughout each weekend or more often) before selling them a year or more later; because the house qualifies as a principal place of residence, the profit is not taxable. The handyman then repeats this exercise on another "handyman special" house, and so on. Many such handymen also regularly carry out maintenance and renovation work on other homes, so that their exposures may approach those of professional tradesmen, but they style themselves as "home handymen". The activities of such handymen are virtually unregulated because they are self-employed and a number work on a "strictly cash" basis.

5.589 In commenting on the experts' responses to Questions 1(b), Canada reiterates the proposition that "chrysotile is readily removed from the lung", and estimates of the half-life of chrysotile are given as 90-110 days, and even a shorter estimate of < 10 days from Dr. David Bernstein. Canada goes on to state that "size for size, chrysotile has a very short half-life." I again draw attention to the 1999 study by Finkelstein and Dufresne [5], who found a lung tissue half-life of eight years for chrysotile fibres > 10 µm in length; this investigation was discussed briefly in the Endnote to my report (see Section V.C.4):

> " ... in the past, the kinetics of chrysotile clearance from lung tissue have been investigated mainly in experimental models using rodents. In an autopsy study published in 1999, Finkelstein and Dufresne [5] ... investigated clearance of chrysotile from the lung tissue of 72 Quebec chrysotile miners and millers in comparison to 49 control subjects, using regression analyses, with the following findings:
>
> There was a significant association between the duration of occupational exposure and the tissue burdens of chrysotile and tremolite.
>
> The concentration of chrysotile decreased with time after exposure ceased but the concentration of tremolite did not.

[158] Please see also my answers to Questions 1(e) and 5(a).

> The clearance rate varied inversely with the length of chrysotile fibres. For fibres > 10 µm in length - i.e. fibre lengths in the reported range for carcinogenicity - the clearance half-time was estimated to be eight years. In other words, the tissue bio-persistence of chrysotile fibres in this study seems substantially more prolonged than in rodent experiments, and presumably corresponds to persistent high chrysotile fibre concentrations for many years after cessation of occupational exposure in humans, as discussed on p 31. It is also notable that the concentration of 6,250,000 chrysotile fibres mentioned on p 31 (for an individual but by no means unusual patient) is probably above the level at which Rogers et al. [6] identified an odds ratio for mesothelioma of > 8.5 (even allowing for differences in fibre size in the counts by the two different laboratories), and even the duration of 16 years after exposure stopped (as opposed to its commencement: 24 years) falls into the lag-time range for lung cancer induction by asbestos.
>
> Studies like this suggest that clearance mechanisms can be overwhelmed and break down at occupational levels of exposure in humans, with the existence of a long-term sequestered fraction of chrysotile fibres."

5.590 This study seems to be of particular significance for the tissue bio-persistence of chrysotile fibres in comparison to substitute materials (please see below, paras. 5.642 to 5.652).

5.591 I also emphasize that some of the estimates given in my Report were conservative, with potential under-estimation of effects. For example, after discussing the incidence rate for spontaneous mesothelioma unrelated to asbestos as being in the range of 1-2 mesotheliomas per million person-years - whereas the likely true figure is probably less than one [4] - I nonetheless used the upper figure of two cases/million for comparison with mesothelioma incidence in some occupational groups (e.g. the incidence of mesothelioma among male automobile/brake mechanics in Australia; please see para. 5.253). In a similar way, I referred to a 30-fold differential rate for lung cancer among the South Carolina (Charleston) chrysotile textile workers in comparison to the Quebec chrysotile miners and millers, whereas others give the differential as up to "about 50 times higher in Charleston" [7].

5.592 I also draw attention to the occurrence of mesothelioma among various cohorts and studies other than the Quebec chrysotile miners/millers, as set out in paragraphs 5.124-5.141, and to the incidence of mesothelioma among mechanics in Australia as shown in the 1999 Report for the Australian Mesothelioma Register [AMR 99] and in NICNAS 99 (see my answer to Question 2).

5.593 In my Report, I discussed the limitations or deficiencies of those studies which reported an increased risk of lung cancer only among workers with pre-existing asbestosis (e.g. the Hughes-Weill study [8]) - and the uncertainties of the study by Camus et al. [9] on lung cancer risk from non-occupational exposure to chrysotile among females in Quebec[159] - but the comments from Canada (para. 5.529) reiterate the Hughes-Weill conclusion that an increased risk of lung cancer occurs "only in those with asbestosis." (Please see paras. 5.73-5.74 and 5.152-5.162 above; this subject was reviewed extensively by Henderson et al. [13] in 1997. Lung cancer risk among the South Carolina chrysotile textile workers versus the Quebec chrysotile miners/millers is also discussed in paras. 5.596 to 5.620 below).

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[159]Canada's comments also refer to the meta-analysis of lung cancer risk reported by Lash et al., [10], which identified a low risk. Meta-analysis is a field that lies outside my expertise, but I understand that there are various models for meta-analysis and problems with this approach (e.g. see Blettner et al. [11] who state that "... Meta-analyses from published data are in general insufficient to calculate a pooled estimate since published estimates are based on heterogeneous populations, different study designs and mainly different statistical models [Abstract] ... Meta-analyses using published data are, therefore, restricted and seldom useful to produce a valid quantitative estimate or to investigate exposure relations such as dose-response [p 8] ..."). In a meta-analysis of 69 asbestos-exposed occupational cohorts, Goodman et al. [12] identified "... meta-SMRs of 163 and 148 [for lung cancer] with and without latency, with significant heterogeneity of results ..."

5.594 At various points (paragraphs 5.475, 5.498 and 5.545), the comments from Canada quote de Klerk and Armstrong [14], in a chapter on *The Epidemiology of Asbestos and Mesothelioma*, in the book *Malignant Mesothelioma,* for which I was the senior editor and a co-author. I shall leave it to Dr. de Klerk to respond.

5.595 In passing, I point out that *Malignant Mesothelioma* was published in 1992; the text for those chapters which I wrote was current up to September 1990, and the manuscript was sent to the publisher shortly thereafter. Much new information on asbestos-related diseases has accumulated since that time (e.g. references 15, 16, 113, 125, 126, 131-133, 140, 141, 170-172, 177-179, 181, 185-187, and 190-194 in my Report, to list but a few). My views on many aspects of asbestos-related disorders have changed very substantially since *Malignant Mesothelioma* was published (e.g. my views on asbestos and lung cancer - please see references [13, 15-18] in these Supplementary Remarks).

(a) Lung cancer rate among South Carolina (Charleston) chrysotile textile workers versus the Quebec chrysotile miners/millers

5.596 With respect to this question, Canada states (see paras. 5.485-5.486):

> "Dr Henderson states that the "greater carcinogenicity of the amphiboles [...] appears not to extend to the induction of lung cancer [p 40], but he admits that 'chrysotile is implicated in one of the lowest rates of asbestos-associated lung cancer (in Quebec chrysotile miners and millers)' [where I also stated that chrysotile is also implicated in the highest lung cancer rate]. Dr Henderson's reluctance to conclude the greater carcinogenicity of amphiboles seems to be caused by the results of Dr Dement's study of the Charleston, South Carolina asbestos textile industry [...]".
>
> "The Charleston data has [*sic*] recently been revisited by Bruce Case, André Dufresne, A.D. McDonald, J.C. McDonald and Patrick Sébastien in a study released in Maastricht in October 1999 at the *VIIth International Symposium on Inhaled Particles,* a symposium attended by some of the world's leading experts. This study shows that a significant amount of crocidolite and amosite fibres was found in the textile workers' lungs. This analysis sheds new light on the issue and explains the extreme results of the original study by Dr Dement [...] and the subsequent study by Dr Stayner [...]. These studies of textile workers exposed to crocidolite and amosite can thereby no longer be used to demonstrate the risks associated with chrysotile fibres."

5.597 Subsequently, the manuscript for a paper by Case et al. [19] entitled *Asbestos Fibre Type and Length in Lungs of Chrysotile Textile and Production Workers: A Preliminary Report* arrived by facsimile transmission. I offer the following comments on this document (and, later, on the Abstract for the corresponding presentation at the Maastricht meeting [20]):

5.598 A disclaimer beneath the title [19] indicates that this is a "DRAFT DOCUMENT: SUBJECT TO REVISION - NOT TO BE CITED". It is cited nonetheless. There is no indication that this document has gone through a process of peer review and been accepted for publication.

5.599 This study revisits the study reported in 1989 by Sébastien et al. [7], and the draft manuscript indicates that the same grids were examined (but fewer cases). The main difference between this investigation and the earlier study by Sébastien et al. [7] is that Case et al. [19, 20] analysed long fibres > 18 µm in length, whereas Sébastien et al. [7] studied fibres > 5 µm in length, with an aspect ratio > 3:1. (It is common practice for fibre burden analyses to focus on fibres = 5 µm in length and there is no evidence that the carcinogenicity of asbestos fibres - in terms of lung cancer induction - is restricted only to fibres about 20 µm in length or more.)

5.600 Another study on the lung fibre content of the Charleston chrysotile textile workers was reported in 1997 by Green et al. [21]; this investigation studied all fibres resolvable by electron

microscopy and with an aspect ratio > 3:1. For this study, lung tissue was analysed from 39 textile workers versus 31 comparable controls matched closely for age (median age at death for the asbestos workers was 56.0 years, versus 59.0 years for the controls).

5.601 In the Green et al. [21] study, the Charleston chrysotile workers had a higher lung content of chrysotile in comparison to the controls (geometric mean = 33,450,000 versus 6,710,000 f/g dry lung), with a higher content of tremolite (3,560,000 *vs*. 260,000); the asbestos workers also had a slightly elevated mean amosite/crocidolite content of 470,000 fibres *vs*. 210,000 for the controls (please see Table 1).

TABLE 1: MINERAL FIBRE CONTENT OF LUNG TISSUE, SOUTH CAROLINA ASBESTOS TEXTILE WORKERS *VS*. CONTROLS (ALL COUNTS = FIBRES X 10^6 / G DRY LUNG)*

	Textile workers	**Controls**
Age at death (median; years)	56.0 (M); 57.0 (F)	59.0 (M); 62.5 (F)
Year of death (median)	1971 (M and F)	1972 (M); 1971 (F)
Chrysotile (fibres x 10^6/ g dry lung)	33.45	6.71
Tremolite	3.56	0.26
Amosite/crocidolite	0.47	0.21
Anthophyllite	0.16	0.13
Mullite	1.63	4.01
Other	1.02	1.9
All fibres	52.46	16.02

*Modified from Tables 1 and 3 in Green et al. [21]; M = men; F= females.

5.602 In the discussion section, Green et al. [21] commented that:

> "The population was exposed almost exclusively to chrysotile asbestos from Quebec. The native ore contained about 1% tremolite asbestos. The high concentrations of chrysotile and tremolite asbestos found in the lungs of the asbestos textile workers are also consistent with their exposure histories. Our finding on enrichment of tremolite relative to chrysotile in the lungs of asbestos workers is consistent with previous reports. The presence of crocidolite in some of the lungs of the asbestos workers is in keeping with the use of small quantities of crocidolite between 1950 and 1975, but the values were only slightly greater than those found in the control population. ... The increased risk of lung cancer in the asbestos textile workers is also unlikely to be due to differences in exposure to tremolite asbestos, as Sebastien et al. have shown that the textile workers had less tremolite asbestos in their lungs than miners and millers of the original ore after matching for exposure intensity. Differences in exposure to other commercial amphiboles (crocidolite and amosite) may have played a small part based on our own data ... and on the data of Sebastien et al., which showed a small excess of these amphiboles in the lungs of the textile workers compared with the miners; however, it is very unlikely that this is the whole explanation as commercial amphiboles formed a very small proportion of the total amphiboles in both studies. Moreover, review of the 10 cases with lung cancer in this study on whom lung fibre analyses were made, showed only one case with substantially increased (> 1 x 10^6 fibre/g dry lung) crocidolite or amosite".

5.603 In this study, it is also notable that the lung cancer cases on which fibre burden analysis was carried out were not representative of the cohort as a whole: e.g. autopsies were carried out on only about 10 per cent of all deaths in the cohort, and the mean lifetime cumulative exposure for the ten lung cancer cases was 94.6 fibre-years in comparison to 67 fibre-years for male lung cancer cases across the whole cohort [21, 22].

5.604 There are even greater concerns about the representativeness of the cases on which fibre burden analysis was carried out by Sébastien et al. [7]. For example, this study was confined to tissue from 72 autopsies among 857 deaths (8.4 per cent) among the Charleston cohort, and there were only seven lung cancer cases out of 66, whereas Case et al. [19] list 126 lung cancers, so that the fibre burden data reported by Case et al. [19] appear to deal with no more than 5.56 per cent of the Charleston lung cancers. It is also notable that the mean age at death in the Charleston group was about a decade younger than the age at death for the Thetford group which formed the basis for comparison in the 1989 study by Sébastien et al. [7][160]

5.605 In addition, as reported by Sébastien et al. (see Table 3 in reference [7]), those cases from the Thetford group that came to autopsy showed an over-representation of asbestos-related diseases (lung cancer, mesothelioma and pneumoconiosis) than the Thetford cohort overall - so that cases of lung cancer + mesothelioma + pneumoconiosis added up to 37 out of 89 autopsies (42 per cent), in comparison to 306 out of 4463 deaths across the whole cohort (7 per cent) [7]. For the Charleston cohort, the figures were more comparable, so that lung cancer + mesothelioma + pneumoconiosis cases added up to 13 out of 72 autopsies (18 per cent) in comparison to 10 per cent across the cohort [7].

5.606 In the more recent study from Case et al. [19], there is a further point on which the two study groups (Thetford versus Charleston) are not comparable: the time following cessation of exposure was a median of eight years for the Thetford group, in comparison to 20 years for the Charleston cohort (please see Table 2). Therefore, it is clear that those lung cancer cases on which fibre burden analysis was carried out from each cohort were not representative of each cohort, and that there were also substantial differences between the two cohorts for the same types of case. Finally, the manuscript from Case et al. [19] does not include a control group against which the two cohorts can be compared (the only one of the three investigations that does is the Green study [21]).

TABLE 2: COMPARISON OF THE SOUTH CAROLINA AND QUEBEC CHRYSOTILE WORKER COHORTS*

	South Carolina textile workers	**Quebec miners/millers**
Cohort number	3022	10,918
Cohort deaths	1258	8009
Age at death (years)	67 ± 10 (??)**	56 ± 6 (??)**
Lung cancers in cohort	126 [SMR 197]	657 [SMR 137]
Mesotheliomas in cohort	2	38
Years since cessation of exposure (median)	20	8
Geometric mean exposure (mpcfy)***	3.63	186
Subjects studied	64	43
Lung cancer cases studied	? (7/72 autopsies in ref [7])	? ("random" selection of 43 cases from 89 original cases that included 22 lung cancer cases - ref [7])
Chrysotile (fibres x 10^6/ g dry lung)	0.054	0.231
Tremolite	0.027	0.325

[160]There is a discrepancy between the ages at death in the original paper by Sébastien et al. [7] (i.e. mean = 55.8 ± 9.7 for the Charleston cohort vs 67.5 ± 9.7 for the Thetford group) and the follow-up study by Case et al. [19] (Table 1A, where the ages are reversed: 67 ± 10 for the Charleston group vs 56 ± 6 for Thetford). Clearly, one or the other must be wrong.

	South Carolina textile workers	Quebec miners/millers
Amosite/crocidolite	0.037	0.024
Total amphibole (tremolite + amosite/crocidolite)	0.064	0.349

*Modified from Case et al. [19]. Fibre counts represent geometric means; all expressed as fibres x 10^6/ g dry lung; **see footnote 160; ***mpcfy = millions of particles per cubic foot-years.

5.607 From the above Table, it is evident that the amosite/crocidolite content of lung tissue from the textile workers is slightly (< 2-fold) higher than the amosite/crocidolite in the lung tissue from the Quebec miners and millers (37,000 fibres > 18 µm in length versus 24,000). This difference in concentration seems to be insufficient to explain the "huge" [19] risk difference (about 30-fold) in the slope of the lung cancer dose-response line between the two groups. In addition, it is noteworthy that the tremolite content of lung tissue was higher in the Quebec miners and millers than the Charleston textile workers (325,000 versus 27,000 for fibres with a mean fibre length of 21.7 versus 21.9 µm). The point is that the total amphibole content (tremolite + amosite + crocidolite) is higher in the Quebec miners and millers at 349,000 f/g dry lung in comparison to a total amphibole content of 64,000 among the Charleston textile workers. In this respect, there is no evidence that tremolite is substantially less potent than the other amphiboles for lung cancer induction, as shown by the high lung cancer incidence (SMR = 285) among Montana vermiculite miners exposed only to tremolite/actinolite (please see paragraph 5.107-5.111).

5.608 From these studies, it appears that the amosite/crocidolite content of lung tissue among the Charleston textile workers may in part be a reflection of low level exposure to the small amount of crocidolite (< 1000 kg total) used in the plant from 1950-1975 to make an asbestos tape or braided packing. The material was received at the plant as a yarn ready for weaving, and no fibre preparation, carding, spinning or twisting was done using crocidolite. Packing workers were not included among the textile worker cohort, and analysis of lung cancer risk by operation in the plant shows all operations to be at about the same lung cancer risk after controlling for chrysotile exposure in a logistic model (Dement, personal communication, 1999).

5.609 A portion of the amosite/crocidolite content may also be explicable by general environmental (non-occupational) exposure, taking into account the small differences between the amphibole content in the textile workers versus the controls in the study reported by Green et al. [21]. In this respect, amphibole concentrations of up to 100,000-200,000 fibres per gram (f/g) dry lung tissue can be expected for about 5 per cent of the population in Germany [23]. Therefore, it seems that the amosite/crocidolite cannot explain the risk of lung cancer in the Charleston cohort in comparison to either matched controls (also matched for smoking) versus the Thetford miners and millers.

5.610 If major significance is to be assigned to the small difference in amosite/crocidolite content of lung tissue between the Charleston workers versus the Thetford miners/millers for lung cancer induction, a question that immediately arises is: *where are the mesotheliomas among the Charleston workers?* Case et al. [19] suggest that misclassification of mesotheliomas as lung cancers among the Charleston workers could have produced under-estimation of the true number of mesotheliomas "while having virtually no effect on the lung cancer excess or lung cancer exposure-disease slope of risk." No evidence in support of this proposition is adduced, and Case et al. [19] state that this is "speculation." The larger number of mesotheliomas in the Quebec cohort may be explicable in part by the higher mean total amphibole content for this group, but this still leaves unexplained the disproportionately larger numbers of lung cancers in the Charleston group (e.g. the ratio of lung cancers to mesotheliomas in the Thetford group is 657/38 = about 17:1, whereas the ratio for the Charleston group is 126/2 = 63:1).

5.611 Case et al. [19] are also rather more cautious in their interpretation than the propositions put forward in Canada's responses to the reports from the experts. For example, on the last page of text they state:

> "... comparison of groups of individuals using this technique is valid only insofar as those studied are representative of the larger groups ... from which they are derived. We cannot be certain to what degree our groups of chrysotile miners/millers and textile workers are representative of the cohorts from which they are derived[161] ... the two groups are not directly comparable in some ways: not only was exposure much higher in the miners/millers, but the interval between cessation of employment and death was shorter .... . Our results closely parallel those reported by Sebastien et al.. Any other result would be surprising since the subjects were drawn from the latter study. ... Caution remains in interpretation. ... One continuing mystery, given the apparent non-trivial long-fibre commercial amphibole exposures is the low level of reported mesotheliomas in this cohort ...".

5.612 Given the data on fibre lengths across the cohorts, in comparison to the data in Sébastien et al. [7], the difference in lung cancer rates between the two groups cannot be explained by differences in fibre length. This is stated explicitly by Case et al. [19].

5.613 However, on looking at the data, it seems that the differences between the two cohorts might be explicable in part by the exposure estimates. Differences in exposure assessment are not refuted by the "new" study reported in draft form by Case et al. [19] or by the earlier study reported by Sébastien et al. [7]: e.g. the difference between 20 years (Charleston) and eight years (Quebec) for clearance after exposure ceased could have a large effect. One can calculate the final exposure (end of exposure) (N_0) from the final fibre content in lung tissue at death (N), from the equation

$$N/N_0 = e^{-?t}$$

where ? represents a clearance coefficient (? = 0.693 ÷ $T_{1/2}$) and t = half-life in tissue ($T_{1/2}$). For $T_{1/2}$ = 8 years [5], ? = 0.693/8, so that for the chrysotile miners/millers, where N = 0.231, N_0 = 0.462. For the Charleston textile workers, where N = 0.054, N_0 = 0.306.

5.614 If $T_{1/2}$ is shorter (e.g. one year), then N_0 for the miners/millers = 59.2 and the corresponding N_0 for the textile workers = 56456.

5.615 Therefore, for a half-life of eight years, one would expect the ratios of exposure (exposure miners/millers ÷ exposure textile workers) to be 0.462/0.306 = 1.5. For a half-life of one year the ratio becomes (exposure miners/millers ÷ exposure textile workers) 59.2/56465 = 0.001. (For tissue half-lives of 90-110 days or < 10 days, the differences would be even more drastic.) However, the ratio of the estimated exposures ($mpcfy_{Quebec}$/$mpcfy_{Charleston}$) is 186/3.63 = 50, suggesting that one or other particle count estimate is incorrect.

5.616 In this respect, it might be argued that the exposure estimates for the Charleston cohort represented an under-estimation of exposure, but this suggestion is not supported by the low tremolite content in the lung tissue of the Charleston workers, and is explicitly rejected by Sébastien et al. [7], who state (p. 187):

> "The hypothesis of a systematic underestimation of exposures to asbestos in Charleston, which would have accounted for the difference in risk, must therefore be rejected and other explanations sought."

5.617 Given that contamination of the Charleston chrysotile by mineral oils has now been excluded, one possibility that remains is over-estimation of the exposures for the Quebec chrysotile

[161]Clearly, from the data in Table 2 and the discussion in paras. 5.604 to 5.609, they are not representative.

miners/millers (with under-estimation of risk). If this explanation is unsustainable, it follows that the paradox remains, it remains unexplained, and seems likely to remain so.

5.618 Finally, I draw to the attention of the Panel the following comment by Case and Dufresne [20] in the Abstract for their presentation at the Maastricht meeting:

> "Risk assessment for asbestos exposure is based on lung cancer risk for textile workers, rather than miners/millers."

5.619 In the draft manuscript, Case et al. [19] state only that:

> "... suggestions that the textile worker mortality data [are] suitable for chrysotile risk assessment [for lung cancer] should be re-evaluated ... ."

5.620 Therefore, even if one accepts this proposition for the moment, the claim that the South Carolina cohort can "thereby no longer be used to demonstrate the risks associated with chrysotile fibres" goes beyond the data in this study. For the reasons discussed in this section, I conclude that the data in Sébastien et al. [7] and in Case et al. [19] do not detract from the conclusions drawn by myself and other authorities from the investigations carried out the South Carolina cohort by Dr. Dement and his colleagues [22, 24].

(b) The question of a threshold for the carcinogenicity of chrysotile (lung cancer and mesothelioma

5.621 On this question, I simply reiterate EHC 203:

> "Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks" [p. 144].

5.622 In the absence of a threshold or an agreed alternative (non-linear) exposure-response model, the linear relationship model is widely employed for risk assessment at low levels of exposure.

5.623 As indicated, the precision or validity of this model is not known at low levels of exposure and, as stated by Dr. de Klerk, the model provides a "conservative estimate". This is the point: in the absence of direct observational data or credible alternative models, the linear model errs - if it does err - on the side of safety, which is appropriate for risk assessment as a prelude to the formulation of occupational health and safety and public health policy. The principle is: if there is doubt, play safe (i.e. first, do no harm; *primum non nocere).*

5.624 In relation to prudent approaches to occupational and public health policy, *The Minerals and Metals Policy of the Government of Canada*[162] states the following (p 7):

> "The precautionary principle is an important factor when the Government needs to make a decision in the face of scientific uncertainties about cause and effect, and when the potential environmental consequences are generally considered to be serious or irreversible. This principle was enunciated clearly as Principle 15 in the 1992 *Rio Declaration on Environment and Development* (the Rio Declaration) of the United Nations Conference on Environment and Development (UNCED), to which Canada is a signatory:
>
> 'Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.'

[162]NRCAN, The Minerals and Metals Policy of the Government of Canada: *Partnership for Sustainable Development*, Public Works of Canada, 1996.

> The principle complements science-based approaches for the management of risks. Its use is premised on the recognition that our scientific understanding of the potential magnitude and consequence of impacts on human health and the environment of the production and uses of some minerals and metals may be incomplete. While there is a need to work toward closing such gaps in our understanding, there is also a requirement, where potential impacts are 'serious or irreversible', to consider a cost-effective precautionary approach."

5.625 Later, on page 12, the same *Minerals and Metals Policy* document states:

> "... It is generally accepted that, in some cases, the risks associated with certain products or product uses cannot be properly controlled or managed. Consequently, where such a situation exists, the Government [of Canada] will either discontinue or prohibit the specific product or product use."

5.626 Three additional points are worth iteration:

- Exposure to commercial Canadian chrysotile is not "chrysotile-only", but usually chrysotile + trace tremolite exposure, although evidence indicates that chrysotile when uncontaminated by tremolite also has the capacity for the induction of lung cancer and mesothelioma.

- Risk estimates for lung cancer and mesothelioma for low levels of chrysotile exposure were set out in Tables 12 and 13 (see my response to Question 1(d)).

- As stated in section C.1(f)(viii) and in paragraph 5.95, there are no observational data on the interactive effects of inhaled commercial chrysotile fibres when these are superimposed separately and later upon a pre-existing amphibole ± chrysotile burden within lung tissue (?superimpositional additive or multiplicative carcinogenic effect). In my Report, I emphasized that it has been estimated that up to 15-20 per cent of men in industrialized societies may have sustained occupational exposures to asbestos (chrysotile/amphiboles), and Rödelsperger et al. [23] indicate that fibre concentrations of 100,000-200,000 amphibole f/g dry lung tissue may be expected for about 5 per cent of the population in Germany. Rödelsperger et al. [23] have also identified a dose-response relationship for mesothelioma induction at these low fibre concentrations. We do not know what the effect of subsequent chrysotile fibre inhalation superimposed upon an existing amphibole burden of this order might be, but NICNAS 99 states the following (p. 61):

> "... multivariate analysis of cases found a dose-response relationship for lung fibre content of crocidolite, amosite and chrysotile and the development of mesothelioma. Either a multiplicative or additive model could be used to fit the relative risk/dose coefficients for the various asbestos types. A progressive increase in relative risk with increasing fibre content was reported for all fibres ... ."

5.627 Because the risks of both lung cancer and mesothelioma show a dose-response effect related to total cumulative exposure levels, it may be expected that later superimpositional inhalation of chrysotile ± tremolite fibres would aggravate the overall consequences of a pre-existing asbestos burden (i.e. increase the risk further).

(c) The feasibility *in practice* of "controlled use" of chrysotile asbestos

5.628 In para. 5.510, Canada identifies the feasibility in practice of "controlled use" of chrysotile asbestos as "one crucial issue, which seems to override all other issues" (i.e. the question of whether the application of the "controlled use principle" is feasible and credible at all stages in the life cycle of chrysotile asbestos).

5.629 As already indicated (see my reply to Question 5), I agree that this proposition is crucial to the dispute before the WTO. However, for the reasons discussed in my Report, I see no requirement to resile from my perception that - although regulation and control of chrysotile and high-density chrysotile products may be achievable at some points of the life-cycle (e.g. the manufacture of friction and high-density products) - "controlled use" of this type is not feasible in reality or in practice at others (e.g. in building construction and other points of end-use).

5.630 No airborne fibre measurements are available for the overwhelming majority of asbestos-related diseases encountered in my everyday practice, even for exposures throughout the 1970s and in some cases extending into the late 1980s. Among my series of asbestos-associated lung cancers and mesotheliomas, I cannot recollect ever seeing actual airborne dust and fibre measurements at points of end-use (e.g. at building construction sites or shipyards).

5.631 The concentration of total asbestos fibres in lung tissue from one of my cases of asbestos-associated lung cancer was up to 125,000,000 f/g dry lung (up to 108,000,000 amosite + crocidolite fibres), for a worker who had been employed at a major asbestos-cement manufacturing facility for about 2-3 years (lag-time = 28 years) [15]. I also note Dr. de Klerk's comment about demolition of an old asbestos-cement factory in Sydney in the latter half of 1999 (probably the same factory) "where no observable precautions of any kind were being taken"[163] (Dr. de Klerk, response to Question 1(a)).

5.632 Other interventions on high-density asbestos-cement materials that can lead to high fibre concentrations are discussed in my first Report (e.g. Kumagai et al. [25]; my answer to question 1(d); please see also the 1980 report by Rödelsperger et al. [26] on exposure to asbestos-cement dust at building sites, which refers to a daily mean airborne fibre concentration of 0.6 f/ml for fibres > 5µm in length, and "peak concentrations of more than 100 fibres/ml").

5.633 In para. 5.532, the comments from Canada include the statement that chrysotile "can be painted without fibre release" (presumably including building products). However, painting of such products can cover warning notices and disguise the true nature of the product, so that workers who later carry out maintenance or renovation work on the same product - and those who recycle the same material - may be unaware of its true nature. In my own series of mesotheliomas, it is not uncommon to encounter cases for which the patient was unaware or unsure that he (or less often she) had worked in the past with an asbestos-containing product.

5.634 In one recent case, the patient worked (1973-1988) at a factory where tins and pails were produced. In about 1979 she had worked for several months at a conveyor belt that carried the tins and pails into a fan-forced oven, which appears in retrospect to have been lined by asbestos-containing insulation. The patient was present when maintenance work on the oven was carried out, and she recalled hot air continually blowing from the oven into her face as she worked on the conveyor. After diagnosis of her mesothelioma and its treatment in the late 1990s by radical pneuropneumonectomy, an asbestos body and fibre analysis on her lung tissue revealed a count of 1640 asbestos bodies per gram dry lung, and a total asbestos fibre count of 34,120,000 f/g dry lung (30,770,000 chrysotile fibres[164] + 3,350,000 crocidolite fibres). This was the only history of exposure that was obtainable on exhaustive questioning.

[163]The potential for misuse of asbestos-containing materials remains, as shown by some prosecutions (e.g. please see the UK Health & Safety Executive (HSE) press releases E198:98 and E079:99; http://www.hse.gov.uk/press/e98198.htm and http://www.hse.gov.uk/press/e99079.htm), but cases that come before the courts almost certainly represent only a small fraction of the misuses, most passing unnoticed by regulatory agencies.

[164]Please note the high chrysotile count almost a decade after the patient's employment ended.

5.635 A similar history was also obtained in another mesothelioma case seen on referral in 1999, where a radio assembly worker had used asbestos-containing cloths used to clean soldering irons, together with a history of about one fibre-hour of exposure to four asbestos-cement building sheets used for maintenance work on his home; only later did I discover that during his work at the radio factory, he often entered a walk-in fan-forced oven apparently lined by insulation bricks.

5.636 Again, please see the spread of occupations in AMR 99 attached to my Report; a similar spread of occupations is listed by Hodgson et al. [27] in a 1997 report on mesothelioma mortality in Britain[165] - e.g. see Table 1 and Fig 1 in the original reference. In footnote 96 to the comments from Canada, it is stated that:

> "The 'controlled use' approach has been endorsed by the WHO in its 1998 Environmental Health Criteria 203: Chrysotile Asbestos, p. 144. 'Control measures, including engineering controls and work practices, should be used in circumstances where the occupational exposure to chrysotile can occur. Data from industries where control technologies have been applied have demonstrated the feasibility of controlling exposure to levels generally below 0.5 fibres/ml. Personal protective equipment can further reduce individual exposure where engineering controls and work practices prove insufficient'."

5.637 I interpret this passage from EHC 203 differently when it is taken in the context of the preceding paragraphs; apart from the heading, the complete text on page 144 of EHC 203 is:

> a) Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks.
> b) Where safer substitute materials for chrysotile are available, they should be considered for use.
> c) Some asbestos-containing products pose particular concern and chrysotile use in these circumstances is not recommended. These uses include friable products with high exposure potential. Construction materials are of particular concern for several reasons. The construction industry workforce is large and measures to control asbestos are difficult to institute. In-place building materials may also pose risk to those carrying out alterations, maintenance and demolition. Minerals in place have the potential to deteriorate and create exposures.
> d) Control measures, including engineering controls and work practices, should be used in circumstances where occupational exposure to chrysotile can occur. Data from industries where control technologies have been applied have demonstrated the feasibility of controlling exposure to levels generally below 0.5 fibres/ml. Personal protective equipment can further reduce individual exposure where engineering controls and work practices prove insufficient.[166]
> e) Asbestos exposure and cigarette smoking have been shown to interact to increase greatly the risk of lung cancer. Those who have been exposed to asbestos can substantially reduce their lung cancer risk by avoiding smoking."

5.638 When seen in the context of para. c), I take d) to mean that in those situations where exposure is likely or unavoidable, exposure can be reduced or minimized by certain procedures appropriate to the circumstances (e.g. engineering controls in manufacture/production or best work practices), but EHC 203 has already identified friable products and building products as materials of "particular concern" and their use is "not recommended", in part because of difficulties of control in the construction industry. I do not see paragraph d) as an endorsement of on-going "controlled use".

5.639 A similar sentiment is expressed in NICNAS 99:

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[165]Hodgson, J.T., Peto, J., Jones, J.R., and Matthews, F.E., *Mesothelioma Mortality in Britain: Patterns by Birth Cohort and Occupation,* (1997), 41 Ann. Occup. Hyg., 129-133.

[166]Omitted from my original Report because I did not - and do not - take this to be an endorsement of "controlled use", and also because the figure of up to 0.5 f/ml is up to five times higher than the level of 0.1 f/ml mentioned in Question 5(c) from the WTO Panel.

"Prudent OHS [occupational health and safety] policy and public health policy favours the elimination of chrysotile wherever possible and practicable [p 139] ...
Best practice must be implemented to minimise occupational and public exposure, and to minimise environmental impact, over the remaining period(s) of use [p 140].
A risk reduction strategy using all available and appropriate measures is required to ensure that the risks posed by chrysotile are continually reduced and eliminated wherever possible" [p 140].

5.640 NICNAS 99 also goes on to state (p 140):

"In achieving this it is further recommended that:

a) Specific phase-out periods should be set, with stages (over the shortest possible period of time) to encourage and reflect the availability and suitability of alternatives [to chrysotile].
b) Action is taken in the immediate future to prohibit the replacement of worn non-chrysotile original equipment with chrysotile products, as alternatives are now available.
c) No new uses of chrysotile or chrysotile products should be introduced (i.e., an immediate prohibition on new uses).
d) Occupational health and safety authorities take the lead role in considering this recommendation and specific strategies to implement it as worker health is identified as the major concern."

5.641 As stated in the paper by Jarvholm et al. [28] attached to the Endnote (Section V.C.4) for my report:

"... The first regulation of asbestos [in Sweden] was introduced in the early 1960s and subjects who started their occupational career in the 1960s should have been exposed to lower doses on average, than those who started earlier. On the other hand, by the 1960s asbestos was being used more extensively so the number of people exposed to asbestos may have increased. ... More stringent regulations of asbestos were introduced in the mid-1970s, which led to the sharp decrease in its use. People who have only worked under such conditions were born from 1955 onwards. They have not yet reached a sufficient latency time for possible mesotheliomas to have developed so the number of cases [is] few. However, the first indication is that they may have a decreased risk compared with earlier birth cohorts. A more certain conclusion can probably not be drawn for another ten years. Thus, the preventive measures of the mid-1970s can probably not be evaluated with reasonable precision until around 2005, 30 years later.

The present situation in Sweden, that mortality from mesothelioma due to early use of asbestos is of a similar size to the total number of fatal occupational accidents, is caused by a situation in which at least 90% of the asbestos used was chrysotile. However, we have no information about the type of exposure to asbestos among the cases of mesothelioma - whether they had an exposure to crocidolite or amosite. There is some pressure from the asbestos industry world-wide to change the asbestos regulations to allow the use of chrysotile. To evaluate such an experiment would take at least another 30 years. Even if the major cause of mesothelioma in Sweden was from types of asbestos other than chrysotile, it is difficult to see how the benefits from an increased use of asbestos in Sweden could outweigh the uncertainty of the risks. A similar prudent approach would also be appropriate in other European countries ..."

(d) Are substitute fibres safer than chrysotile?

5.642 In para. 5.539, Canada states:

"Dr. Henderson, for his part, recognises that, as with all fibres, the pathogenicity of substitutes is defined by the "3 Ds" (dimension, dose, durability). He seems also to understand that, due to the (lack of) historical use of substitutes, we cannot fully know the risks of using them. However, he then seems to ignore the importance of these facts."

5.643 My comments on the safety or potential biohazards of substitute fibres were based on the following:

- The dimensions and respirability of substitute fibres. For example, it appears that synthetic fibres can be engineered to be either shorter than the lengths of asbestos fibres that have been associated particularly with carcinogenicity, or to be predominantly non-respirable. In contrast, according to Harrison et al. [29]:

"The intrinsic hazardous properties of chrysotile can never be "engineered out", and the potential for harm will always remain. Prevention of ill-health will thus always rely on the control of exposure, something that history has shown cannot be guaranteed. ... Unlike chrysotile, substitute fibers can often be designed or selected to have particular characteristics."

- Dose: reported airborne fibre concentrations from the manufacture or use of substitute (e.g. synthetic) fibres are low - comparable to or lower than the airborne fibre concentrations produced by the manufacture or subsequent use of chrysotile-containing materials. This being so, my conclusions about the relative safety of chrysotile versus substitute fibres are based primarily on fibre dimensions (discussed above) and biopersistence (discussed below).

- Durability (biopersistence): in para. 5.552, Canada states the following:

"It is well known that biopersistence is a key parameter. Indeed, the human evidence for chrysotile indicates that it is likely to be one of the main reasons why chrysotile is less dangerous than the amphiboles in respect to mesothelioma risk. This is clearly recognised by three of the four experts, as well as by INSERM."

Canada then emphasizes the rapidity of clearance of chrysotile from lung tissue, with reference to a 90-110 day half-life for chrysotile in lung tissue, and an even shorter estimate of < 10 days. Again, I draw attention to the recent study from Finkelstein and Dufresne [5] who estimated a lung tissue half-life of eight years for chrysotile fibres > 10 µm in length in Quebec chrysotile miners and millers. Accordingly, in my survey of the literature, I placed particular emphasis on the biopersistence of substitute fibres in comparison to chrysotile.

- The relative potency of substitute fibres or chrysotile fibres to produce pathological changes (e.g. genotoxicity/mutagenicity and the capacity for tumour induction).

5.644 Warheit et al. [30] claim that p-aramid fibres are biodegradable in the lungs of exposed rats, with faster clearance times than long chrysotile fibres, which showed greater biopersistence.

" ... p-aramid is biodegradable in the lungs of exposed rats; in contrast, the clearance of long chrysotile fibres was slow or insignificant, resulting in a pulmonary retention of long chrysotile asbestos fibres. The dimensional changes of asbestos fibres as well as the pulmonary cell labelling data indicate that chrysotile asbestos fibres may produce greater long-term pulmonary effects when compared to inhaled para-aramid fibrils" [Abstract].

5.645 In 1993, Hesterberg et al. [31] compared the effects of size-separated respirable fractions of fibrous glass (FG) with refractory ceramic fibres (RCF) and chrysotile fibres. They found that:

"Exposure to chrysotile asbestos (10 mg/m^3) and to a lesser extent RCF (30 mg/m3) resulted in pulmonary fibrosis as well as mesothelioma and significant increases in lung tumours. FG [fibreglass designated MMVF10 and MMVD11] exposure was associated with a non-specific inflammatory response (macrophage response) in the lungs that did not appear to progress after 6-12 months of exposure. The cellular changes are reversible and are similar to the effects observed after inhalation of an inert dust. No lung fibrosis was observed in the FG-exposed animals. Further, FG exposure resulted

> in no mesotheliomas and no statistically significant increase in lung tumour incidence when compared to that of the negative control group. These findings, along with previous inhalation studies, suggest that respirable fibrous glass does not represent a significant hazard for fibrotic or neoplastic lung disease in humans" [Abstract].

5.646 In a later (1995) study, Hesterberg et al. [32] found that exposure of rats to crocidolite and chrysotile asbestos and to RCF by inhalation induced pulmonary fibrosis, lung tumours and mesotheliomas (41 per cent of hamsters exposed to RCF developed mesothelioma[167]); fibreglasses MMVF10 and MMVF11, slagwool (MMVF22) and stonewool (MMVF21) did not produce a significant increase in lung tumours or mesotheliomas[168]

5.647 In a further study published in 1998, Hesterberg et al. [33] investigated the biopersistence of synthetic vitreous fibres and amosite in the rat lung, together with refractory ceramic fibres (RCF1A). They found that "the very biopersistent fibres were carcinogenic" (amosite, crocidolite, RCF1 and two relatively durable special application fibreglasses designated MMVF32 and MMVF33), whereas "the more rapidly clearing fibres were not" (including rock [stone] wool designated MMVF21, HT stonewool designated MMVF34, slag wool, and insulation fibreglasses designated MMVF10 and MMVF11).[169]

5.648 An Annex from Canada[170] also includes a 1995 document on p-aramid fibres from the Health & Safety Executive (HSE) in the United Kingdom. In a summary statement (p. 22) the HSE document states that:

> "The balance of evidence suggests that aramid fibres possess a low potential to produce mesothelioma, which is likely to be at least as low as for chrysotile asbestos. While chrysotile is thought to present a hazard with respect to mesothelioma development, current knowledge indicates that the risks for human exposure are low, and would only be detectable following very heavy and prolonged exposure. Thus, if in terms of mesothelioma production, aramid fibres are equally, or less hazardous than chrysotile, it can be concluded that the risks at occupationally relevant levels of exposure would be extremely low."

5.649 The HSE then set an exposure limit of 2.5 f/ml, but in a subsequent document on *Substitutes for Chrysotile (White) Asbestos,* the HSE[171] commented that:

> "There are many long-established alternatives to chrysotile which do not rely on fibre technology. For example, corrugated polyvinylchloride (PVC) and steel sheeting can be used instead of asbestos cement sheets.

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[167]The hamster seems to show a propensity for mesothelioma induction in some circumstances (e.g. SV40 inoculation) but not others; in some studies (Research and Consulting Company) chrysotile did not induce mesothelioma or lung in hamsters but in rats it produced pulmonary fibrosis, lung tumours and mesotheliomas, so that the rat has been advocated as the most appropriate model for assessment of the human risk from fibre inhalation [32].

[168]Hesterberg, T.W., Miller, W.C., Thevenez, Ph. and Anderson, R., *Chronic Inhalation of Man-made Vitreous Fibres: Characterization of Fibres in the Exposure Aerosol and Lungs*, (1995) 39 Ann. Occup. Health, pp. 637-653.

[169]Wilson et al. [34] estimate that fibreglass is 5-10 times less "risky" than chrysotile and they state that "... no one has found any cancer attributable to the manufacture or installation of glass wool fibers ... ." In their estimates of lung cancer risk from chrysotile, they use the unit carcinogenicity factor of 0.01 (K; used before them by the US EPA), and they calculate an absolute excess lung cancer risk of 1.2×10^{-3} for smokers, and 1.4×10^{-4} for non-smokers; for 40 yrs exposure at 1.0 f/ml, these estimates equate to 4.8×10^{-2} (smokers) and 5.6×10^{-3} (non-smokers) - i.e. about 5 per cent and 0.5 per cent respectively, both of which can be considered quite "high". (Wilson, R., Langer, A.M. and Nolan, R.O., *A Risk Assessment for Exposure to Glass Wool,* (1999) 30 Regulatory Toxicology and Pharmacology, pp. 96-109.

[170]Minty, C.A., Meldrum M., Phillips, A.M., and Ogden, T.L., *P-aramid Respirable Fibres Criteria Documents for an Occupational Exposure Limit,* HMSO (1995).

[171]HSE document: http://www.hse.gov.uk/pubns/misc155.htm.

> Several types of non-asbestos fibres can also be substituted for asbestos; they have been developed for use in a wide range of products. The main non-asbestos fibres in current use are polyvinyl alcohol (PVA), aramid and cellulose. A considered scientific view on their safety has recently become available. In July 1998, the UK's Department of Health Committee on Carcinogenicity (CoC) concluded that these three asbestos substitutes (PVA, cellulose and aramid) are safer than chrysotile. This view was endorsed by the European Commission Scientific Committee on Toxicity, Ecotoxicity and the Environment in September 1998."

5.650 More recently, a press release[172] from the UK Health and Safety Commission (HSC/HSE) announced a prohibition on the importation, supply or use of chrysotile in Great Britain, effective from 24 November 1999.

5.651 I also re-emphasize the comments in the reviews quoted in my original Report (answer to Question 6), including the review by Harrison et al. [29] who comment along the following lines:

> "The diameter of PVA [polyvinyl alcohol] fibres, as manufactured, is well above the respirable limit and most of them are not inhalable. ... the fibres are mostly in the range of 10-16 µm diameter. There is evidence that they do not fibrillate (split lengthwise). Many of the particles seen in the atmosphere are non-fibrous. ... Although the published toxicologic information on PVA is relatively sparse, the parent material has been used extensively in surgery and has food contact clearance, presumably based on unpublished studies. Indications of an accumulation of oligomers in the kidney in some circumstance[173] ... mean that the spectrum of molecular weight of material in the fibres as used should be considered, especially if a smaller diameter material were to be produced. The material will degrade only slowly, if at all, in the lungs. ... Thus, substitution of PVA for asbestos fibers in products such as asbestos-cement should result in reduced exposures. This prediction has been confirmed in industrial applications where very low fibre counts have been experienced. Misuse of installed material would not result in significant exposure.
>
> ... On balance, the use of aramid fibers should result in reduced levels of fiber exposure as compared to chrysotile asbestos and the fibrils released will be no more toxic and will be less biopersistent. The predicted reduction in absolute exposure levels has been achieved in industrial practice. Misuse of installed material would not be expected to give significant exposures.
>
> ... On balance, the coarse fiber structure and the long experience in use indicate that substitution of cellulose fiber for chrysotile asbestos should result in reduced occupational exposures to fiber and lower levels of deposition in the lung. The apparent biopersistence of cellulose in the lung would be a possible cause for concern if the potential for limited lung damage is confirmed.
>
> ... We believe that the continued use of chrysotile in asbestos-cement products is not justifiable in the face of available and technically and adequate substitutes. Likewise, there seems to be no justification for the continued residual use of chrysotile in friction materials."

5.652 These comments also coincide with one of the recommendations in NICNAS 99:

> "... Current overseas experience with the phasing out of chrysotile products indicates that a range of alternatives is available to suit the majority of uses. Good OHS practice dictates that use of chrysotile should be restricted to those uses where suitable substitutes are not available, and alternatives should continue to be sought for remaining uses" [p 139].

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[172]HSC press Release C054:99: http://www.hse.gov.uk/press/c99054.htm.
[173]Referred to in the comments from Canada.

(e) Summary

5.653 It is my perception that the conclusions in my Report submitted already to the WTO concur with mainstream thinking and approaches to occupational and public health policy from national and international health authorities; these include, *inter alia:*

- The National Occupational Health & Safety Commission in Australia (WorkSafe Australia). (Please see NICNAS 99.)
- The World Health Organization (EHC 203).
- INSERM (France).
- The National Health & Safety Commission/Health & Safety Executive (HSC/HSE) in Great Britain.
- Medical Research Council (MRC) Institute for Environment and Health at the University of Leicester (UK).
- National health authorities in other European Nations.
- The Collegium Ramazzini.

5.654 This being so, it is my perception that the dispute before the WTO is, to some extent, focused upon inappropriate issues. There has been on-going argument among scientists on the health hazards of chrysotile asbestos *(the chrysotilophiles versus the chrysotilophobes).* Given the extent and complexity of the scientific literature - with contradictory observations on some important issues and with uncertainties related to gaps in observational data - it is almost inconceivable that this controversy can be resolved by the WTO Panel, or, indeed, that it will be resolved in the foreseeable future (partly because no control group free from asbestos exposure can be assembled to ascertain the true spontaneous mesothelioma rate).

5.655 The point to be emphasized is that there exists a substantial body of independent scientific and medical opinion - embodied in national and international health authorities - that chrysotile is carcinogenic with no delineated threshold; that it cannot be controlled at all points of end use; and that existing scientific evidence indicates that safer substitute materials are available.

5.656 To me, this body of opinion is no tendentious artifice designed only to secure a commercial advantage. From my perspective, this is perhaps the crucial issue, from the so-called precautionary principle, given that neither side is likely to concede that the other has proven its case at a high order of scientific probability. In other words, the question is not so much whether there exists a proven health risk or virtually no risk from the continued use of chrysotile, but whether there exists a body of independent and reputable opinion that the possible risks or uncertainties about risk justify a policy of highly restricted use or non-use.

5.657 From this perspective, restriction of chrysotile to only a very few special applications - or its prohibition - is a reasonable and defensible measure designed as a cautious and prudent approach to public and occupational health policy.

5.658 Therefore, I re-affirm the conclusions set out in my original Report (paragraph 5.431) that chrysotile should either:

(a) Be restricted to only a few and well-defined applications[174] so that it is inaccessible to the great majority of workers and is available for use by only small and cohesive specialized worker groups that can be trained effectively in its controlled use (e.g. analogous to nuclear fuels); this means that chrysotile should not be used in building products (e.g. high-density fibro-cement materials such as asbestos-cement sheets) or friction products.

OR

(b) It should be made inaccessible to everyone, by prohibition, unless the alternatives pose equal or greater hazards and equal or greater problems with control.

5.659 In this latter circumstance, the principle is that minimization of exposure is more certain when no new chrysotile-containing products are introduced into the workplace or the general environment, so that the total amount of asbestos in-place will diminish over time; the problem then becomes primarily one of minimization of exposure to existing asbestos products during maintenance, repair, removal, demolition and disposal.

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[174]In the UK HSC Press Release C054:99 announcing implementation of a policy of prohibition of chrysotile from 24 November 1999, the following specific uses are allowable until 2001-2005:

- The use of compressed asbestos fibre (CAF) in gaskets for use with saturated and superheated steam, and with certain flammable, toxic and corrosive chemicals until 1 January 2001;
- The use of CAF in gaskets for use with chlorine until 1 January 2003;
- The use of any sheet which, when in a dry state, has a density greater than 1900 kilograms per cubic metre and is used at temperatures at or above 500°C until 1 January 2003;
- The use of asbestos components in aeroplanes and helicopters where this is crucial for their safe operation until 1 January 2004;
- The use of any product consisting of a mixture of asbestos with a phenol formaldehyde or with a cresylic formaldehyde resin in vanes for rotary vacuum pumps, vanes for rotary compressors, any bearing or its housing or for split-face seals used to prevent water leakage from hydro-electric power generation turbines or from cooling water pumps in power stations until 1 January 2004;
- The use of asbestos in pre-formed joints made from proofed asbestos cloth for sealing the doors of steam boilers until 1 January 2004;

The use of asbestos in personal protective clothing when used in very high temperatures (500°C or more) until 1 January 2005.

VI. SUBMISSIONS FROM NON-GOVERNMENTAL ORGANIZATIONS

6.1 The Panel received four *amicus briefs* from the following non-governmental organizations:

- *Collegium Ramazzini*, dated 7 May 1999
- *Ban Asbestos Network*, dated 22 July 1999
- *Instituto Mexicano de Fibro-Industrias A.C.*, dated 26 July 1999
- *American Federation of Labor and Congress of Industrial Organizations*, dated 28 July 1999

6.2 These *amicus briefs* were transmitted to the parties for their information. In their written rebuttals of 30 June 1999, the EC incorporated by reference the submission of the *Collegium Ramazzini.* In a letter dated 18 August 1999, Canada notified the Panel that, bearing in mind the general nature of the opinions expressed by the non-governmental organizations in those submissions, they would not be useful to the Panel at this advanced stage of the proceedings. Should the Panel nonetheless accept the submissions as *amicus briefs*, Canada believed that the parties should be given the possibility to respond to the factual and legal arguments set out in them. In a letter dated 3 November 1999, the EC informed the Panel that it was incorporating by reference the *amicus brief* submitted by the *American Federation of Labor and Congress of Industrial Organizations*, as that body supported the EC's scientific and legal arguments in this dispute. The EC also proposed to the Panel that it reject the submissions from the *Ban Asbestos Network* and the *Insituto Mexicano de Fibro-Industrias A.C.*, as those documents contained no information of relevance to the dispute. In a letter dated 10 November 1999, Canada again urged the Panel to reject the four *amicus briefs* as it was inappropriate to admit them at this stage in the proceedings. Should the Panel nevertheless consider these submissions, Canada considered that, for the sake of procedural fairness, the parties should have an opportunity to comment on their content.

6.3 In a letter dated 12 November 1999, the Panel informed the parties that, in the light of the EC's decision to incorporate into its own submissions the *amicus briefs* submitted by the *Collegium Ramazzini* and the *American Federation of Labor and Congress of Industrial Organizations*, the Panel would consider these two documents on the same basis as the other documents furnished by the EC in this dispute. It was also on that basis that the Panel submitted those two submissions to the scientific experts for their information. At the second substantive meeting of the Panel with the parties, the Panel gave Canada the opportunity to reply, in writing or orally, to the arguments set forth in these two *amicus briefs*. At that same meeting, the Panel also informed the parties that it had decided not to take into consideration the *amcius briefs* submitted by the *Ban Asbestos Network* and by the *Instituto Mexicano de Fibro-Industrias A.C.*

6.4 On 27 June 2000, the Panel received a written brief from the non-governmental organization *ONE ("Only Nature Endures")* situated in Mumbai, India. The Panel considered that this brief had been submitted at a stage in the procedure when it could no longer be taken into account. It therefore decided not to accept the request of *ONE* and informed the organization accordingly. The Panel transmitted a copy of the documents received from *ONE* to the parties for information and notified them of the decision it had taken. At the same time, it also informed the parties that the same decision would apply to any briefs received from non-governmental organizations between that point and the end of the procedure.