

ANNEX IIIPublications and Documents Referred to by the Experts (Section V)**Dr. Henderson**

Three key references on chrysotile asbestos – published in 1998 and 1999 respectively – are quoted or cited frequently throughout Dr. Henderson's report in abbreviated form:

- EHC 203: Multiple authors, *Environmental Health Criteria 203: Chrysotile Asbestos*, Inter-organization Programme for the Sound Management of Chemicals (IPCS), Geneva, World Health Organization, 1998.
- NICNAS 99: *Full public report: Chrysotile Asbestos – Priority Existing Chemical n° 9*, National Industrial Chemicals Notification and Assessment Scheme (NICNAS), National Occupational Health and Safety Commission (NOHSC), Sydney, Commonwealth of Australia, February 1999.
- AMR 99: Leigh J., Hendrie L., Berry D., *The Incidence of Mesothelioma in Australia 1994 to 1996*, Australian Mesothelioma Register (AMR) Report, 1999, Sidney, NOSHC, 1999.

1. Documents referred to in Introductory Comments and Comments to the Questions by the Panel (Section V.C.1-2)

1. Pott F, Roller M, Ziem U, et al. Carcinogenicity studies on natural and man-made fibres with the intraperitoneal test in rats. IARC Scientific Publication no 90. In: Bignon J, Peto J, Saracci R, eds. Non-occupational exposure to mineral fibres. Lyon: International Agency for Research on Cancer (IARC); 1989:173-9.
2. Case BW. Health effects of tremolite. Now and in the future. *Ann NY Acad Sci* 1991;643:491-504.
3. Rogers AJ, Leigh J, Berry G, et al. Relationship between lung asbestos fiber type and concentration and relative risk of mesothelioma: a case-control study. *Cancer* 1991;67:1912-20.
4. Kumagai S, Nakachi S, N. K, et al. Estimation of asbestos exposure among workers repairing asbestos cement pipes used for conduits [Japanese]. *Sangkyo Igaku* 1993;35:178-87.
5. Sturm W, Menze B, Krause J, Thriene B. Use of asbestos, health risks and induced occupational diseases in the former East Germany. *Toxicol Lett* 1994;72:317-24.
6. Rösler JA, Weitowitz HJ. Recent data on cancer due to asbestos in Germany. *Med Lav* 1995;86:440-8.
7. Sturm W, Menze B, Krause J, Thriene B. Asbestos-related diseases and asbestos types used in the former GDR. *Exp Toxicol Pathol* 1995;47:173-8.
8. Nicholson WJ, Raffn E. Recent data on cancer due to asbestos in the U.S.A. and Denmark. *Med Lav* 1995;86:393-410.
9. Warheit DB, Driscoll KE, Oberdoerster G, et al. Contemporary issues in fiber toxicology. *Fundam Appl Toxicol* 1995;25:171-83.
10. McDonald JC, McDonald AD. The epidemiology of mesothelioma in historical context. *Eur Respir J* 1996;9:1932-42.
11. Stayner LT, Dankovic DA, Lemen RA. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. *Am J Public Health* 1996;86:179-86.

12. Warheit DB, Hartsky MA, Frame SR. Pulmonary effects in rats inhaling size-separated chrysotile asbestos fibres or p-aramid fibrils: differences in cellular proliferative responses. *Toxicol Lett* 1996;88:287-92.
13. McDonald AD, Case BW, Churg A, et al. Mesothelioma in Quebec chrysotile miners and millers: epidemiology and aetiology. *Ann Occup Hyg* 1997;41:707-19.
14. McDonald JC, McDonald AD. Chrysotile, tremolite and carcinogenicity. *Ann Occup Hyg* 1997;41:699-705.
15. Boffetta P. Health effects of asbestos exposure in humans: a quantitative assessment. *Med Lav* 1998;89:471-80.
16. Multiple authors. Environmental Health Criteria 203: Chrysotile Asbestos. International Programme on Chemical Safety (IPCS). Geneva: World Health Organization; 1998.
17. Rödelsperger K, Woitowitz H-J, Brückel B, Arhelger R. Non asbestos mineral fibres in human lungs. *Eur J Oncol* 1998;3:221-9.
18. Anonymous. Full public report: Chrysotile Asbestos — Priority Existing Chemical no. 9. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). National Occupational Health and Safety Commission (NOHSC). Sydney: Commonwealth of Australia, February 1999.
19. Harrison PT, Levy LS, Patrick G, et al. Comparative hazards of chrysotile asbestos and its substitutes: a European perspective. *Environ Health Perspect* 1999;107:607-11.
20. Hillerdal G. Mesothelioma: cases associated with non-occupational and low dose exposures. *Occup Environ Med* 1999;56:505-13.
21. Landrigan PJ, Nicholson WJ, Suzuki Y, Ladou J. The hazards of chrysotile asbestos: a critical review. *Indust Health* 1999;37:271-80.
22. Leigh J, Hendrie L, Berry D. The incidence of mesothelioma in Australia 1994 to 1996. Australian Mesothelioma Register (AMR) Report, 1999. Sydney: NOHSC, 1998.
23. Leigh J, Hendrie L, Berry D. The incidence of mesothelioma in Australia 1994 to 1996. Australian Mesothelioma Register (AMR) Report, 1999. Sydney: NOHSC, 1999.
24. Peto J, Decarli A, La Vecchia C, et al. The European mesothelioma epidemic. *Br J Cancer* 1999;79:666-72.
25. Rödelsperger K, Woitowitz HJ, Brückel B, et al. Dose-response relationship between amphibole fiber lung burden and mesothelioma. *Cancer Detection & Prevention* 1999;23:183-93.
26. Churg A, Green FHY, eds. *Pathology of Occupational Lung Disease*. New York: Igaku-Shoin; 1988.
27. Henderson DW, Shilkin KB, Langlois SL, Whitaker D, eds. *Malignant Mesothelioma*. New York: Hemisphere; 1992.
28. Roggli VL, Greenberg SD, Pratt PC, eds. *Pathology of Asbestos-Associated Diseases*. Boston: Little, Brown; 1992.
29. Jaurand M-C, Bignon J, eds. *The Mesothelial Cell and Mesothelioma*. New York: Marcel Dekker; 1994.
30. Churg A, Green FHY. *Pathology of Occupational Lung Disease*, 2nd edn. Baltimore: Williams & Wilkins; 1998.
31. Henderson DW, Shilkin KB, Whitaker D, et al. The pathology of mesothelioma, including immunohistology and ultrastructure. In: Henderson DW, Shilkin KB, Langlois SL, Whitaker D, eds. *Malignant Mesothelioma*. New York: Hemisphere; 1992:69-139.

32. Henderson DW, Shilkin KB, Whitaker D, et al. Unusual histological types and anatomic sites of mesothelioma. In: Henderson DW, Shilkin KB, Langlois SL, Whitaker D, eds. *Malignant Mesothelioma*. New York: Hemisphere; 1992:140-66.
33. Henderson DW, Comin CE, Hammar SP, et al. Malignant mesothelioma of the pleura: current surgical pathology. In: Corrin B, ed. *Pathology of Lung Tumors*. New York: Churchill Livingstone; 1997:241-80.
34. Nomori H, Horio H, Kobayashi R, Morinaga S. Long survival after extrapleural pneumonectomy for pleural malignant mesothelioma with metastasis to infradiaphragmatic lymph node. *Scand Cardiovasc J* 1997;31:237-9.
35. Turler A, Monig SP, Raab M. Problems in diagnosis and therapy of malignant pleural mesothelioma [German]. *Med Klin* 1997;92:101-5.
36. Sugarbaker DJ, Norberto JJ, Swanson SJ. Extrapleural pneumonectomy in the setting of multimodality therapy for diffuse malignant pleural mesothelioma. *Semin Thorac Cardiovasc Surg* 1997;9:373-82.
37. Sugarbaker DJ, Richards WG, Garcia JP. Extrapleural pneumonectomy for malignant mesothelioma. *Adv Surg* 1997;31:253-71.
38. Sugarbaker DJ, Garcia JP. Multimodality therapy for malignant pleural mesothelioma. *Chest* 1997;112:272S-275S.
39. Kamiya I, Umeda T, Kako T. A case of panpleuropneumectomy for diffuse pleural mesothelioma [Japanese]. *Kyobu Geka* 1998;51:793-6.
40. Sugarbaker DJ, Norberto JJ. Multimodality management of malignant pleural mesothelioma. *Chest* 1998;113:61S-65S.
41. Pass HI, Temeck BK, Kranda K, et al. Preoperative tumor volume is associated with outcome in malignant pleural mesothelioma. *J Thorac Cardiovasc Surg* 1998;115:310-7.
42. Daly BD. Late results. *Chest Surg Clin Nth Amer* 1999;9:675-93.
43. Sugarbaker DJ, Flores RM, Jaklitsch MT, et al. Resection margins, extrapleural nodal status, and cell type determine postoperative long-term survival in trimodality therapy of malignant pleural mesothelioma: results in 183 patients. *J Thorac Cardiovasc Surg* 1999;117:54-63.
44. Stolley PD, Lasky T. *Investigating Disease Patterns: The Science of Epidemiology*. New York: Scientific American; 1998.
45. Newhouse ML, Thompson H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br J Ind Med* 1965;22:261-9.
46. Newhouse ML, Thompson H. Epidemiology of mesothelial tumors in the London area. *Ann NY Acad Sci* 1965;132:579-88.
47. Churg J, Selikoff IJ. Geographic pathology of pleural mesothelioma. In: Liebow AA, Smith DE, eds. *The Lung*. International Academy of Pathology Monograph No. 8. Baltimore: Williams & Wilkins; 1968:284-97.
48. Ferguson DA, Berry G, Jelihovsky T, et al. The Australian mesothelioma surveillance program 1979-1985. *Med J Aust* 1987;147:166-72.
49. Leigh J, Corvalan C, Copland P. Malignant mesothelioma incidence in Australia 1982-1992. In: *Proceedings of the International Congress on Applied Mineralogy*; 1993: 28-30.
50. Antman KH, Ruxer RL, Aisner J, Vawter G. Mesothelioma following Wilms' tumor in childhood. *Cancer* 1984;54:367-9.
51. Peterson JT, Greenberg SD, Buffler PA. Non-asbestos-related malignant mesothelioma. A review. *Cancer* 1984;54:951-60.

52. Anderson KA, Hurley WC, Hurley BT, Ohrt DW. Malignant pleural mesothelioma following radiotherapy in a 16-year-old boy. *Cancer* 1985;56:273-6.
53. Austin MB, Fechner RE, Roggli VL. Pleural malignant mesothelioma following Wilms' tumor. *Am J Clin Pathol* 1986;86:227-30.
54. Horie A, Hiraoka K, Yamamoto O, et al. An autopsy case of peritoneal malignant mesothelioma in a radiation technologist. *Acta Pathol Jpn* 1990;40:57-62.
55. Pappo AS, Santana VM, Furman WL, et al. Post-irradiation malignant mesothelioma. *Cancer* 1997;79:192-3.
56. de la Pena A, Lucas I. Malignant peritoneal mesothelioma as late complication of radiotherapy for Hodgkin's disease [Spanish]. *An Med Intern* 1997;14:319.
57. Andersson M, Wallin H, Jonsson M, et al. Lung carcinoma and malignant mesothelioma in patients exposed to Thorotrast: incidence, histology and p53 status. *Int J Cancer* 1995;63:330-6.
58. van Kaick G, Wesch H, Lührs H, et al. Epidemiological results and dosimetric calculations - an update of the German Thorotrast study. In: van Kaick G, Karaoglou A, Kellerer AM, eds. *Health effects of Internally Deposited Radionuclides: Emphasis on Radium and Thorium*. Singapore, New Jersey: World Scientific; 1995:171-75.
59. Ishikawa Y, Mori T, Machinami R. Lack of apparent excess of malignant mesothelioma but increased overall malignancies of peritoneal cavity in Japanese autopsies with Thorotrast injection into blood vessels. *J Cancer Res Clin Oncol* 1995;121:567-70.
60. Neugut AI, Ahsan H, Antman KH. Incidence of malignant pleural mesothelioma after thoracic radiotherapy. *Cancer* 1997;80:948-50.
61. Behling CA, Wolf PL, Haghghi P. AIDS and malignant mesothelioma — is there a connection? *Chest* 1993;103:1268-9.
62. Roggli VL, McGavran MH, Subach J, et al. Pulmonary asbestos body counts and electron probe analysis of asbestos body cores in patients with mesothelioma: a study of 25 cases. *Cancer* 1982;50:2423-32.
63. Hillerdal G, Berg J. Malignant mesothelioma secondary to chronic inflammation and old scar: two new cases and review of the literature. *Cancer* 1985;55:1868-1972.
64. Chahinian AP, Pajak TF, Holland JF, et al. Diffuse malignant mesothelioma. Prospective evaluation of 69 patients. *Ann Intern Med* 1982;96:746-55.
65. Gentiloni N, Febbraro S, Barone C, et al. Peritoneal mesothelioma in recurrent familial peritonitis. *J Clin Gastroenterol* 1997;24:276-9.
66. Belange G, Gompel H, Chaouat Y, Chaouat D. Malignant peritoneal mesothelioma occurring in periodic disease: apropos of a case [French]. *Rev Med Interne* 1998;19:427-30.
67. Livneh A, Langevitz P, Pras M. Pulmonary associations in familial Mediterranean fever. *Curr Opin Pulm Med* 1999;5:326-31.
68. Hillerdal G. Non-malignant pleural disease. *Thorax* 1981;36:669-75.
69. Greenberg SD. Benign asbestos-related pleural diseases. In: Roggli VL, Greenberg SD, Pratt PC, eds. *Pathology of Asbestos-Associated Diseases*. Boston: Little, Brown; 1992:165-87.
70. Comin CE, de Klerk NH, Henderson DW. Malignant mesothelioma: current conundrums over risk estimates, and whither electron microscopy for diagnosis? *Ultrastruct Pathol* 1997;21:315-320.
71. Gold B, Kathren RL. Causes of death in a cohort of 260 plutonium workers. *Health Phys* 1998;75:236-40.

72. Baris I, Simonato L, Artvinli M, et al. Epidemiological and environmental evidence of the health effects of exposure to erionite fibres: a four-year study in the Cappadocian region of Turkey. *Int J Cancer* 1987;39:10-17.
73. Baris YI, Simonato L, Saracci R, Winkelmann R. The epidemic of respiratory cancer associated with erionite fibres in the Cappadocian region of Turkey. In: Elliott P, Cuzick J, English D, Stern R, eds. *Geographical and Environmental Epidemiology: Methods for Small-Area Studies*. Oxford: Oxford University Press; 1992:310-22.
74. Metintas M, Hillerdal G, Metintas S. Malignant mesothelioma due to environmental exposure to erionite: follow-up of a Turkish emigrant cohort. *Eur Respir J* 1999;13:523-6.
75. Roggli VL, Brody AR. Experimental models of asbestos-related diseases. In: Roggli VL, Greenberg SD, Pratt PC, eds. *Pathology of Asbestos-Associated Diseases*. Boston: Little, Brown; 1992:257-97.
76. Carbone M, Pass HI, Rizzo P, et al. Simian virus 40-like DNA sequences in human pleural mesothelioma. *Oncogene* 1994;9:1781-90.
77. Cristaudo A, Vivaldi A, Sensales G, et al. Molecular biology studies on mesothelioma tumor samples: preliminary data on H-ras, p21, and SV40. *J Environ Pathol Toxicol Oncol* 1995;14:29-34.
78. Pass HI, Kennedy RC, Carbone M. Evidence for and implications of SV40-like sequences in human mesotheliomas. *Important Adv Oncol* 1996;:89-108.
79. Pepper C, Jasani B, Navabi H, et al. Simian virus 40 large T antigen (SV40LTA) primer specific DNA amplification in human pleural mesothelioma tissue. *Thorax* 1996;51:1074-6.
80. De Luca A, Baldi A, Esposito V, et al. The retinoblastoma gene family pRb/p105, p107, pRb2/p130 and simian virus-40 large T-antigen in human mesotheliomas. *Nat Med* 1997;3:913-6.
81. Stenton SC. Asbestos, Simian virus 40 and malignant mesothelioma. *Thorax* 1997;52, suppl 3:S52-7.
82. Carbone M, Rizzo P, Grimley PM, et al. Simian virus-40 large-T antigen binds p53 in human mesotheliomas. *Nat Med* 1997;3:908-12.
83. Carbone M, Rizzo P, Pass HI. Simian virus 40, poliovaccines and human tumors: a review of recent developments. *Oncogene* 1997;15:1877-88.
84. Kuska B. SV40: working the bugs out of the polio vaccine. *J Natl Cancer Inst* 1997;89:283-4.
85. Gibbs AR, Jasani B, Pepper C, et al. SV40 DNA sequences in mesotheliomas. *Dev Biol Stand* 1998;94:41-5.
86. Griffiths DJ, Nicholson AG, Weiss RA. Detection of SV40 sequences in human mesothelioma. *Dev Biol Stand* 1998;94:127-36.
87. Carbone M, Fisher S, Powers A, et al. New molecular and epidemiological issues in mesothelioma: role of SV40. *J Cell Physiol* 1999;180:167-72.
88. Pacini F, Vivaldi A, Santoro M, et al. Simian virus 40-like DNA sequences in human papillary thyroid carcinomas. *Oncogene* 1998;16:665-9.
89. Carbone M, Rizzo P, Procopio A, et al. SV40-like sequences in human bone tumors. *Oncogene* 1996;13:527-35.
90. Butel JS, Lednicky JA, Stewart AR, et al. SV40 and human brain tumors. *J Neurovirol* 1997;3, suppl 1:S78-9.
91. Huang H, Reis R, Yonekawa Y, et al. Identification in human brain tumors of DNA sequences specific for SV40 large T antigen. *Brain Pathol* 1999;9:33-42.

92. Cicala C, Pompetti F, Carbone M. SV40 induces mesotheliomas in hamsters. *Am J Pathol* 1993;142:1524-33.
93. Matker CM, Rizzo P, Pass HI, et al. The biological activities of simian virus 40 large-T antigen and its possible oncogenic effects in humans. *Monaldi Arch Chest Dis* 1998;53:193-7.
94. Murthy SS, Testa JR. Asbestos, chromosomal deletions, and tumor suppressor gene alterations in human malignant mesothelioma. *J Cell Physiol* 1999;180:150-7.
95. Mutti L, Carbone M, Giordano GG, Giordano A. Simian virus 40 and human cancer. *Monaldi Arch Chest Dis* 1998;53:198-201.
96. Mayall FG, Jacobson G, Wilkins R. Mutations of p53 gene and SV40 sequences in asbestos associated and non-asbestos-associated mesotheliomas. *J Clin Pathol* 1999;52:291-3.
97. Strickler HD, Goedert JJ, Fleming M, et al. Simian virus 40 and pleural mesothelioma in humans. *Cancer Epidemiol Biomarkers Prev* 1996;5:473-5.
98. Dhaene K, Verhulst A, Van Marck E. SV40 large T-antigen and human pleural mesothelioma. Screening by polymerase chain reaction and tyramine-amplified immunohistochemistry. *Virchows Archiv* 1999;435:1-7.
99. Mulatero C, Suretheran T, Breuer J, Rudd RM. Simian virus 40 and human pleural mesothelioma. *Thorax* 1999;54:60-1.
100. Galateau-Sallé F, Bidet P, Iwatsubo Y, et al. SV40-like DNA sequences in pleural mesothelioma, bronchopulmonary carcinoma, and non-malignant pulmonary diseases. *J Pathol* 1998;184:252-7.
101. Olin P, Giesecke J. Potential exposure to SV40 in polio vaccines used in Sweden during 1957: no impact on cancer incidence rates 1960 to 1993. *Dev Biol Stand* 1998;94:227-33.
102. Strickler HD, Rosenberg PS, Devesa SS, et al. Contamination of poliovirus vaccines with simian virus 40 (1955-1963) and subsequent cancer rates. *JAMA* 1998;279:292-5.
103. Fisher SG, Weber L, Carbone M. Cancer risk associated with simian virus 40 contaminated polio vaccine. *Anticancer Res* 1999;19:2173-80.
104. Kannerstein M, Churg J. Peritoneal mesothelioma. *Hum Pathol* 1977;8:83-94.
105. Jarvholm B, Sanden A. Lung cancer and mesothelioma in the pleura and peritoneum among Swedish insulation workers. *Occup Environ Med* 1998;55:766-70.
106. Leigh J, Corvalán CF, Grimwood A, et al. The incidence of malignant mesothelioma in Australia 1982-1988. *Am J Ind Med* 1991;20:643-55.
107. McCaughey WTE, Colby TV, Battifora H, et al. Diagnosis of diffuse malignant mesothelioma: experience of a US/Canadian mesothelioma panel. *Mod Pathol* 1991;4:342-53.
108. Malker HSR, McLaughlin JK, Weiner JA, et al. Peritoneal mesothelioma in the construction industry in Sweden. *J Occup Med* 1987;29:979-80.
109. Musk AW, de Klerk NH, Eccles JL, et al. Wittenoom, Western Australia: a modern industrial disaster. *Am J Ind Med* 1992;21:735-47.
110. Daya D, McCaughey WTE. Pathology of the peritoneum: a review of selected topics. *Semin Diagn Pathol* 1991;8:277-89.
111. Neumann V, Muller KM, Fischer M. Peritoneal mesothelioma — incidence and etiology [German]. *Pathologe* 1999;20:169-76.
112. Churg A. Neoplastic asbestos-induced diseases. In: Churg A, Green FHY, eds. *Pathology of Occupational Lung Disease*. New York: Igaku-Shoin; 1988:279-325.

113. Multiple authors. Consensus report: asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. *Scand J Work Environ Health* 1997;23:311-6.
114. Herman RL. Mesothelioma in rainbow trout, *Salmo gairdneri* Richardson. *J Fish Dis* 1985;8:373-6.
115. de Klerk N. Environmental mesothelioma. In: Jaurand M-C, Bignon J, eds. *The Mesothelial Cell and Mesothelioma. Lung Biology in Health and Disease*, vol 78. New York: Marcel Dekker; 1994:19-35.
116. Roggli VL. Mineral fiber content of lung tissue in patients with malignant mesothelioma. In: Henderson DW, Shilkin KB, Langlois SLP, Whitaker D, eds. *Malignant Mesothelioma*. New York: Hemisphere; 1992:201-22.
117. McDonald JC, McDonald AD. Mesothelioma: is there a background? In: Jaurand M-C, Bignon J, eds. *The Mesothelial Cell and Mesothelioma. Lung Biology in Health and Disease*, vol 78. New York: Marcel Dekker; 1994:37-45.
118. Mark EJ, Yokoi T. Absence of evidence for a significant background incidence of diffuse malignant mesothelioma apart from asbestos exposure. *Ann NY Acad Sci* 1991;643:196-204.
119. Klemperer P, Rabin CB. Primary neoplasms of the pleura: a report of five cases. *Arch Pathol* 1931;11:385-412.
120. Du Bray ES, Rosson FB. Primary mesothelioma of the pleura: a clinical and pathologic contribution to pleural malignancy, with report of a case. *Arch Intern Med* 1920;26:715-37.
121. Albin M, Magnani C, Krstev S, et al. Asbestos and cancer: a n overview of current trends in Europe. *Environ Health Perspect* 1999;107, suppl 2:289-98.
122. Bruske-Hohlfeld I. Occupational cancer in Germany. *Environ Health Perspect* 1999;107, suppl 2:253-8.
123. Coggon D. Occupational cancer in the United Kingdom. *Environ Health Perspect* 1999;107, suppl 2:239-44.
124. Merler E, Vineis P, Alhaique D, Miligi L. Occupational cancer in Italy. *Environ Health Perspect* 1999;107, suppl 2:259-71.
125. Tossavainen A. Asbestos, asbestosis and cancer. Exposure criteria for clinical diagnosis. *People and Work Research Reports* 14. Helsinki: Finnish Institute of Occupational Health; 1997:8-27.
126. Steenland K, Loomis D, Shy C, Simonsen N. Review of occupational lung carcinogens. *Am J Ind Med* 1996;29:474-90.
127. Leigh J. Predicting future numbers of cases of asbestos-related disease in Australia. In: *Asbestos-related Diseases: Setting the National Research Agenda 1996 to 2006*. Sydney, June 1996.
128. Multiple authors. Asbestos cement products. Report by The Western Australian Advisory Committee on Hazardous Substances. Perth; 1990.
129. Nicholson WJ. Comparative dose-response relationships of asbestos fiber types: magnitudes and uncertainties. *Ann NY Acad Sci* 1991;643:74-84.
130. Karjalainen A. Asbestos — a continuing concern. *Scand J Work Environ Health* 1997;23:81-2.
131. 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.

132. Nurminen M, Tossavainen A. Is there an association between pleural plaques and lung cancer without asbestosis? *Scand J Work Environ Health* 1994;20:62-4.
133. Hughes JM, Weill H. Asbestosis as a precursor of asbestos related lung cancer: results of a prospective mortality study. *Br J Ind Med* 1991;48:229-233.
134. Bégin R, Gauthier J-J, Desmeules M, Ostiguy G. Work-related mesothelioma in Québec, 1967-1990. *Am J Ind Med* 1992;22:531-42.
135. de Klerk NH, Armstrong BK. The epidemiology of asbestos and mesothelioma. In: Henderson DW, Shilkin KB, Langlois SL, Whitaker D, eds. *Malignant Mesothelioma*. New York: Hemisphere; 1992:223-50.
136. Iwatsubo Y, Paireon JC, Boutin C, et al. Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study. *Am J Epidemiol* 1998;148:133-42.
137. Rödelsperger K. Anorganische Fasern im menschlichen Lungengewebe. Lungenstaubfaseranalytik zur Epidemiologie der Risikofaktoren des diffusen malignen Mesothelioms (DMM). [Inorganic fibres in human lung tissue. Epidemiology of the risk factors for diffuse malignant mesothelioma (DMM) based on lung dust fibre analysis]. Schriftenreihe des Bundesanstalt für Arbeitsmedizin: Forschung Fb 01 HK 076. Berlin: Bundesanstalt für Arbeitsmedizin; 1996.
138. Williams VM, de Klerk NH, Musk AW, et al. Measurement of lung tissue content of asbestos (an example from Western Australia). In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases*, vol 15. Charlottesville: Lexis; 1997;15:17-46.
139. Siemiatycki J, Boffetta P. Invited commentary: is it possible to investigate the quantitative relation between asbestos and mesothelioma in a community-based study? *Am J Epidemiol* 1998;148:143-7.
140. Camus M, Siemiatycki J, Meek B. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 1998;338:1565-71.
141. Camus M, Siemiatycki J. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 1998;339:1001-2.
142. Pott F. Neoplastic findings in experimental asbestos studies and conclusions for fiber carcinogenesis in humans. *Ann NY Acad Sci* 1991;643:205-18.
143. Churg A. Neoplastic asbestos-induced disease. In: Churg A, Green FHY, eds. *Pathology of Occupational Lung disease*, 2nd edn. Baltimore: Williams & Wilkins; 1998:339-91.
144. Smith AH, Wright CC. Chrysotile asbestos is the main cause of pleural mesothelioma. *Am J Ind Med* 1996;30:252-66.
145. Churg A. Deposition and clearance of chrysotile asbestos. *Ann Occup Hyg* 1994;38:625-33, 424-5.
146. Dufresne A, Harrigan M, Masse S, Bégin R. Fibers in lung tissues of mesothelioma cases among miners and millers of the township of Asbestos, Quebec. *Am J Ind Med* 1995;27:581-92.
147. McDonald JC, McDonald AD. Chrysotile, tremolite, and mesothelioma. *Science* 1995;267:776-7.
148. Liddell FD, McDonald AD, McDonald JC. Dust exposure and lung cancer in Quebec chrysotile miners and millers. *Ann Occup Hyg* 1998;42:7-20.
149. Coplu L, Dumortier P, Demir AU, et al. An epidemiological study in an Anatolian village in Turkey environmentally exposed to tremolite asbestos. *J Environ Pathol Toxicol Oncol* 1996;15:177-82.

150. Sakellariou K, Malamou-Mitsi V, Haritou A, et al. Malignant pleural mesothelioma from nonoccupational asbestos exposure in Metsovo (north-west Greece): slow end of an epidemic? *Eur Respir J* 1996;9:1206-10.
151. Dumortier P, Coplu L, de Maertelaer V, et al. Assessment of environmental asbestos exposure in Turkey by bronchoalveolar lavage. *Am J Respir Crit Care Med* 1998;158:1815-24.
152. Metintas M, Ozdemir N, Hillerdal G, et al. Environmental asbestos exposure and malignant pleural mesothelioma. *Respir Med* 1999;93:349-55.
153. McDonald JC, McDonald AD, Armstrong B, Sebastien P. Cohort study of mortality of vermiculite miners exposed to tremolite. *Br J Ind Med* 1986;43:436-44.
154. Amandus HE, Wheeler R. The morbidity and mortality of vermiculite miners and millers exposed to tremolite-actinolite: part II. Mortality. *Am J Ind Med* 1987;11:15-26.
155. Churg A, Vedal S. Fiber burden and patterns of asbestos-related disease in workers with heavy mixed amosite and chrysotile exposure. *Am J Respir Crit Care Med* 1994;150:663-9.
156. Berry G, Rogers AJ, Pooley FD. Mesotheliomas — asbestos exposure and lung burden. IARC Scientific Publications 1989;90:486-96.
157. Du Toit RS. An estimate of the rate at which crocidolite asbestos fibres are cleared from the lung. *Ann Occup Hyg* 1991;35:433-438.
158. de Klerk NH, Musk AW, Williams V, et al. Comparison of measures of exposure to asbestos in former crocidolite workers from Wittenoom Gorge, W. Australia. *Am J Ind Med* 1996;30:579-87.
159. Oberdörster G. Macrophage-associated responses to chrysotile. *Ann Occup Hyg* 1994;38:601-15.
160. Liddell D. Cancer mortality in chrysotile mining and milling: exposure-response. *Ann Occup Hyg* 1994;38:519-23.
161. McDonald JC. Unfinished business: the asbestos textiles mystery. *Ann Occup Hyg* 1998;42:3-5.
162. Kashansky SV, Scherbakov SV, Kogan FM. Dust levels in workplace air (a retrospective view of “Uralasbest”). In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases*, vol 15. Charlottesville: Lexis; 1997;15:337-54.
163. Scherbakov SV, Dommin SG, Kashansky SV. Dust levels in workplace air of the mines and mills of Uralasbest Company. In: Lehtinen S, Tossavainen A, Rantanen J, eds. *Proceedings of the Asbestos Symposium for the Countries of Central and Eastern Europe*. Budapest, December 1997. *People and Work Research Reports* 19. Helsinki: Finnish Institute of Occupational Health; 1998:104-8.
164. Kogan FM. Asbestos-related diseases in Russia. In: Banks DE, Parker JE, eds. *Occupational Lung Disease: An International Perspective*. London: Chapman & Hall; 1998:247-53.
165. Vudrag M, Krajnc K. Asbestos in the Republic of Slovenia. In: Lehtinen S, Tossavainen A, Rantanen J, ed. *Proceedings of the Asbestos Symposium for the Countries of Central and Eastern Europe*. Budapest, December 1997. *People and Work Research Reports* 19. Helsinki: Finnish Institute of Occupational Health; 1998:79-84.
166. Tcherneva-Jalova P, Lukanova R, Demirova M. Asbestos in Bulgaria. In: Lehtinen S, Tossavainen A, Rantanen J, eds. *Proceedings of the Asbestos Symposium for the Countries of Central and Eastern Europe*. Budapest, December 1997. *People and Work Research Reports* 19. Helsinki: Finnish Institute of Occupational Health; 1998:33-8.
167. Indulski J, Szeszenia-Dabrowska N. Asbestos in Poland. In: Lehtinen S, Tossavainen A, Rantanen J, eds. *Proceedings of the Asbestos Symposium for the Countries of Central and Eastern*

Europe. Budapest, December 1997. People and Work Research Reports 19. Helsinki: Finnish Institute of Occupational Health; 1998:55-62.

168. Rubino GF, Piolatto G, Newhouse ML, et al. Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. *Br J Ind Med* 1979;36:187-94.

169. Piolatto G, Negri E, La Vecchia C, et al. An update of cancer mortality among chrysotile asbestos miners in Balangero, northern Italy. *Br J Ind Med* 1990;47:810-4.

170. Yano E, Wang ZM, Wang XR, et al. Does exposure to chrysotile asbestos without amphibole cause lung Cancer? In: *Epidemiology for Sustainable Health: The XV International Scientific Meeting of the International Epidemiological Association*, Florence, September 1999: 209.

171. Dement JM, Brown DP, Okun A. Follow-up study of chrysotile asbestos textile workers: cohort mortality and case-control analyses. *Am J Ind Med* 1994;26:431-47.

172. Dement JM, Brown DP. Lung cancer mortality among asbestos textile workers: a review and update. *Ann Occup Hyg* 1994;38:525-32.

173. Morinaga K, Kohyama N, Yokoyama K, et al. Asbestos fibre content of lungs with mesotheliomas in Osaka, Japan: a preliminary report. *IARC Sci Publ* 1989;:438-43.

174. Dodson RF, O'Sullivan M, Corn CJ, et al. Analysis of asbestos fiber burden in lung tissue from mesothelioma patients. *Ultrastruct Pathol* 1997;21:321-36.

175. Langer AM, Nolan RP. Asbestos in the lungs of persons exposed in the USA. *Monaldi Arch Chest Dis* 1998;53:168-80.

176. Levin JL, McLarty JW, Hurst GA, et al. Tyler asbestos workers: mortality experience in a cohort exposed to amosite. *Occup Environ Med* 1998;55:155-60.

177. 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.

178. 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;13:141-166.

179. Vainio H, Boffetta P. Mechanisms of the combined effect of asbestos and smoking in the etiology of lung cancer. *Scand J Work Environ Health* 1994;20:235-42.

180. Day NE, Brown CC. Multistage models and primary prevention of cancer. *J Natl Cancer Inst* 1980;64:977-89.

181. Lee BW, Wain JC, Kelsey KT, et al. Association of cigarette smoking and asbestos exposure with location and histology of lung cancer. *Am J Respir Crit Care Med* 1998;157:748-55.

182. Kipen HM, Lilis R, Suzuki Y, et al. Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. *Br J Ind Med* 1987;44:96-100.

183. Rudd RM. Pulmonary fibrosis in asbestos insulation workers with lung cancer. *Br J Ind Med* 1987;44:428-9.

184. Suzuki Y, Kipen H, Lilis R, Selikoff IJ. Pulmonary fibrosis in asbestos insulation workers with lung cancer. *Br J Ind Med* 1987;44:719-20.

185. Wilkinson P, Hansell DM, Janssens J, et al. Is lung cancer associated with asbestos exposure without small opacities on the chest radiograph? *Lancet* 1995;345:1074-8.

186. Finkelstein MM. Radiographic asbestosis is not a prerequisite for asbestos-associated lung cancer in Ontario asbestos-cement workers. *Am J Ind Med* 1997; 32:341-8.

187. de Klerk NH, Musk AW, Glancy JJ, et al. Crocidolite, radiographic asbestosis and subsequent lung cancer. *Ann Occup Hyg* 1997;41, suppl 1:134-6.
188. Sluis-Cremer GK, Bezuidenhout BN. Relation between asbestosis and bronchial cancer in amphibole asbestos miners. *Br J Ind Med* 1989;46:537-40.
189. Sluis-Cremer GK, Bezuidenhout BN. Relation between asbestosis and bronchial cancer in amphibole asbestos miners. *Br J Ind Med* 1990;47:215-6.
190. Case BW, Dufresne A. Asbestos, asbestosis, and lung cancer: observations in Quebec chrysotile workers. *Environ Health Perspect* 1997;105, suppl 5:1113-9.
191. Green FH, Harley R, Vallyathan V, et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. *Occup Environ Med* 1997;54:549-59.
192. Case BW. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 1998;339:1001.
193. Churg A. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *N Engl J Med* 1998;339:999.
194. Tossavainen A. Health and exposure surveillance of Siberian asbestos miners: a joint Finnish-American-Russian project. In: Lehtinen S, Tossavainen A, Rantanen J, eds. *Proceedings of the Asbestos Symposium for the Countries of Central and Eastern Europe*. Budapest, December 1997. *People and Work Research Reports* 19. Helsinki: Finnish Institute of Occupational Health; 1998:89-91.
195. Davis JMG. Experimental and spontaneous mesotheliomas. In: Jaurand M-C, Bignon J, eds. *The Mesothelial Cell and Mesothelioma*. Lung Biology in Health and Disease, vol 78. New York: Marcel Dekker; 1994:187-206.
196. Both K, Henderson DW, Turner DR. Asbestos-induced aberrations and mutations in cells. In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases*, vol 10. Salem: Butterworths; 1994:1-55.
197. Mossman BT, Bignon J, Corn M, et al. Asbestos: scientific developments and implications for public policy. *Science* 1990;247:294-301.
198. Mossman BT. Mechanisms of asbestos carcinogenesis and toxicity: the amphibole hypothesis revisited. *Br J Ind Med* 1993;50:673-676.
199. Mossman BT. Carcinogenesis and related cell and tissue responses to asbestos: a review. *Ann Occup Hyg* 1994;38:617-24.
200. Mossman BT, Kamp DW, Weitzman SA. Mechanisms of carcinogenesis and clinical features of asbestos-associated cancers. *Cancer Invest* 1996;14:466-80.
201. Alleman JE, Mossman BT. Asbestos revisited. *Sci Am* 1997;277:54-7.
202. Mossman BT, Churg A. Mechanisms in the pathogenesis of asbestosis and silicosis. *Am J Respir Crit Care Med* 1998;157:1666-80.
203. Bielefeldt-Ohlmann H, Jarnicki AG, Fitzpatrick DR. Molecular pathobiology and immunology of malignant mesothelioma. *J Pathol* 1996;178:369-78.
204. Haugen A, Schafer PW, Lechner JF, et al. Cellular ingestion, toxic effects, and lesions observed in human bronchial epithelial tissue and cells cultured with asbestos and glass fibers. *Int J Cancer* 1982;30:265-72.
205. Jaurand MC. Observations on the carcinogenicity of asbestos fibers. *Ann NY Acad Sci* 1991;643:258-70.
206. Janatipour M, Trainor KJ, Kutlaca R, et al. Mutations in human lymphocytes studied by an HLA selection system. *Mutat Res* 1988;198:221-26.

207. Turner DR, Grist SA, Janatipour M, Morley AA. Mutations in human lymphocytes commonly involve gene duplication and resemble those seen in cancer cells. *Proc Nat Acad Sci USA* 1988;85:3189-92.
208. Both K. The nature of mutations induced by asbestos and erionite in human cells [PhD Thesis]. Flinders University of South Australia; 1994.
209. Both K, Turner DR, Henderson DW. Loss of heterozygosity in asbestos-induced mutations in a human mesothelioma cell line. *Environ Molec Mutagen* 1995;26:67-71.
210. Fan K, Dao DD, Schutz M, Fink LM. Loss of heterozygosity and overexpression of p53 gene in human primary prostatic adenocarcinoma. *Diagn Molec Pathol* 1994;3:265-70.
211. Cavenee WK, White RL. The genetic basis of cancer. *Sci Am* 1995;272:50-57.
212. Emerit I, Jaurand MC, Saint-Etienne L, Levy A. Formation of a clastogenic factor by asbestos-treated rat pleural mesothelial cells. *Agents Actions* 1991;34:410-15.
213. Walker C, Everitt J, Barrett JC. Possible cellular and molecular mechanisms for asbestos carcinogenicity. *Am J Ind Med* 1992;21:253-73.
214. Marczynski B, Kerenyi T, Marek W, Baur X. Induction of DNA - damage after rats exposure to crocidolite asbestos fibers. In: Davis JMG, Jaurand M-C, eds. *Cellular and Molecular Effects of Mineral and Synthetic Dusts and Fibres*. NATO ASI series, vol. H85. Berlin: Springer; 1994:227-32.
215. Rahman Q, Mahmood N, Khan SG, Athar M. Augmentation in the differential oxidative DNA-damage by asbestos in presence of H₂O₂ and organic peroxide/hydroperoxide. In: Davis JMG, Jaurand M-C, eds. *Cellular and Molecular Effects of Mineral and Synthetic Dusts and Fibres*. NATO ASI series, vol. H85. Berlin: Springer; 1994:171-81.
216. Soodaeva SK, Korkina LG, Velichovskii BT, Klegeris AM. Formation of active forms of oxygen by rat peritoneal macrophages under the effect of cytotoxic dust [Russian]. *Biull Eksp Biol Med* 1991;112:252-54.
217. Korkina LG, Durnev AD, Suslova TB, et al. Oxygen radical-mediated mutagenic effect of asbestos on human lymphocytes: Suppression by oxygen radical scavengers. *Mutat Res* 1992;265:245-53.
218. Vallyathan V, Mega JF, Shi X, Dalal NS. Enhanced generation of free radicals from phagocytes induced by mineral dusts. *Am J Respir Cell Molec Biol* 1992;6:404-13.
219. Jackson JH, Schraufstatter IU, Hyslop PA, et al. Role of oxidants in DNA damage: hydroxyl radical mediates the synergistic DNA damaging effects of asbestos and cigarette smoke. *J Clin Invest* 1987;80:1090-5.
220. Kamp DW, Israbian VA, Preusen SE, et al. Asbestos causes DNA strand breaks in cultured pulmonary epithelial cells: role of iron-catalyzed free radicals. *Am J Physiol* 1995;268:L471-80.
221. Spurny K, Marfel H, Boose C, et al. Fiber concentration in the vicinity of objects and buildings with asbestos-containing building materials. *Zentralb Bakteriol Mikrobiol Hyg B* 1988;187:136-41.
222. Spurny KR. Asbestos fibre release by corroded and weathered asbestos-cement products. *IARC Sci Publ* 1989;90:367-71.
223. Spurny KR. On the release of asbestos fibers from weathered and corroded asbestos cement products. *Environ Res* 1989;48:100-16.
224. Spurny KR, Marfel H, Boose C, et al. Fiber emission from weathered asbestos cement products. 1. Fiber release in ambient air. *Zentralb Hyg Umweltmed* 1989;188:127-43.

225. Lorimer WV, Rohl AN, Miller A, et al. Asbestos exposure of brake repair workers in the United States. *Mt Sinai J Med* 1976;43:207-18.
226. Rohl AN, Langer AM, Wolff MS, Weisman I. Asbestos exposure during brake lining maintenance and repair. *Environ Res* 1976;12:110-28.
227. Weitowitz HJ, Rödelsperger K. Mesothelioma among car mechanics? *Ann Occup Hyg* 1994;38:635-8.
228. Huncharek M. Changing risk groups for malignant mesothelioma. *Cancer* 1992;69:2704-11.
229. Berry G, Newhouse ML. Mortality of workers manufacturing friction materials using asbestos. *Br J Ind Med* 1983;40:1-7.
230. McDonald AD, Fry JS, Wooley AJ, McDonald JC. Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med* 1984;41:151-7.
231. Wong O. Chrysotile asbestos, mesothelioma, and garage mechanics. *Am J Ind Med* 1992;21:449-51.
232. Newhouse ML, Sullivan KR. A mortality study of workers manufacturing friction materials: 1941-1986. *Br J Ind Med* 1989;46:176-9.
233. Weitowitz H-J, Rödelsperger K. Chrysotile asbestos, mesothelioma and garage mechanics: response to Dr. Wong. *Am J Ind Med* 1992;21:453-5.
234. Churg A. Nonneoplastic disease caused by asbestos. In: Churg A, Green FHY, eds. *Pathology of Occupational Lung Disease*, 2nd edn. Baltimore: Williams & Wilkins; 1998:277-338.
235. Dupres JS, Mustard JF, Uffen RJ. Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario (2 vols). Toronto: Ontario Ministry of Government Services: Queen's Printer for Ontario; 1984.
236. Browne K. A threshold for asbestos-related lung cancer. *Br J Ind Med* 1986;43:556-8.
237. Dement JM, Harris RL, Jr., Symons MJ, Shy C. Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. *Ann Occup Hyg* 1982;26:869-87.
238. Dement JM, Harris RL, Jr., Symons MJ, Shy CM. Exposures and mortality among chrysotile asbestos workers. Part II: mortality. *Am J Ind Med* 1983;4:421-33.
239. Dement JM, Harris RL, Jr., Symons MJ, Shy CM. Exposures and mortality among chrysotile asbestos workers. Part I: exposure estimates. *Am J Ind Med* 1983;4:399-419.
240. Dement JM. Carcinogenicity of chrysotile asbestos: a case control study of textile workers. *Cell Biol Toxicol* 1991;7:59-65.
241. Dement JM. Carcinogenicity of chrysotile asbestos: evidence from cohort studies. *Ann NY Acad Sci* 1991;643:15-23.
242. McDonald AD, Fry JS, Wooley AJ, McDonald JC. Dust exposure and mortality in an American chrysotile asbestos textile plant. *Br J Ind Med* 1983;40:361-7.
243. Thimpont J, De Vuyst P. Occupational asbestos-related diseases in Belgium (epidemiological data and compensation criteria). In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases*; vol 17. Charlottesville: Lexis; 1998;17:311-28.
244. Lewis NJ, Curtis MF. Occupational health and hygiene following a fire in a warehouse with an asbestos cement roof [see comments]. *J Soc Occup Med* 1990;40:53-4.
245. Markowitz SB, Garibaldi K, Lillis R, Landrigan PJ. Asbestos exposure and fire fighting. *Ann NY Acad Sci* 1991;643:573-7.

246. Hoskins JA, Brown RC. Contamination of the air with mineral fibers following the explosive destruction of buildings and fire.. *Drug Metab Rev* 1994;26:663-73.
247. Bridgman SA. Lessons learnt from a factory fire with asbestos-containing fallout. *J Pub Health Med* 1999;21:158-65.
248. De Vuyst P, Dumortier P, Swaen GM, et al. Respiratory health effects of man-made vitreous (mineral) fibres. *Eur Respir J* 1995;8:2149-73.
249. Foa V, Basilico S. Chemical and physical characteristics and toxicology of man-made mineral fibers [Italian]. *Med Lav* 1999;90:10-52.
250. Boillat MA. Synthetic mineral fibers [French]. *Schweiz Med Woch/ J Suisse Med* 1999;129:468-74.
251. Steenland K, Stayner L. Silica, asbestos, man-made mineral fibers, and cancer. *Cancer Causes Control* 1997;8:491-503.
252. Glass LR, Brown RC, Hoskins JA. Health effects of refractory ceramic fibres: scientific issues and policy considerations. *Occup Environ Med* 1995;52:433-40.
253. Okayasu R, Wu L, Hei TK. Biological effects of naturally occurring and man-made fibres: in vitro cytotoxicity and mutagenesis in mammalian cells. *Br J Cancer* 1999;79:1319-24.
254. Hesterberg TW, Mast R, McConnell EE, et al. Chronic inhalation toxicity of refractory ceramic fibers in Syrian hamsters. In: Brown RC, Hoskins JA, Johnson NF, eds. *Mechanisms of Fiber Carcinogenesis*. NATO ASI Series A; vol 223. Berlin: Springer; 1992;223:519-39.
255. Dopp E, Schiffmann D. Analysis of chromosomal alterations induced by asbestos and ceramic fibers. *Toxicol Lett* 1998;96-97:155-62.
256. Hesterberg TW, Hart GA, Chevalier J, et al. The importance of fiber biopersistence and lung dose in determining the chronic inhalation effects of X607, RCF1, and chrysotile asbestos in rats. *Toxicol Appl Pharmacol* 1998;153:68-82.
257. Warheit DB, Snajdr SI, Hartsy MA, Frame SR. Lung proliferative and clearance responses to inhaled para-aramid RFP in exposed hamsters and rats: comparisons with chrysotile asbestos fibers. *Environ Health Perspect* 1997;105, suppl 5:1219-22.

2. Documents referred to in the Endnote (Section V.C.3)

1. Finkelstein MM, Dufresne A. Inferences on the Kinetics of Asbestos Deposition and Clearance among Chrysotile Miners and Millers. *Am. J. Ind. Med.*, 1999; 35:401-12.
2. Rogers AJ, Leigh J, Berry G, et al.. Relationship between Lung Asbestos Fiber Type and Concentration and Relative Risk of Mesothelioma: A Case-Control Study. *Cancer* 1991; 67:1912-20.
3. Jarvholm B, Englund A, Albin M. Pleural Mesothelioma in Sweden: An Analysis of the Incidence According to the Use of Asbestos. *Occup. Environ. Med.* 1999; 56:110-3.

3. Documents referred to in Supplementary Comments (Section V.F)

1. Multiple authors. Environmental Health Criteria 203: Chrysotile Asbestos. International Programme on Chemical Safety (IPCS). Geneva: World Health Organization; 1998.
2. Constantopoulos SH, Sakellariou K. Non-Occupational Mesothelioma: Epidemiological Considerations. In: Peters GA, Peters BJ, eds. *Sourcebook on Asbestos Diseases*, vol. 17. Charlottesville: Lexis; 1998;17 :71-97.

3. Schneider J, Weitowitz H-J. Asbestos-Related Non-Occupational Malignant Mesothelioma. In: Peters GA, Peters BJ, eds. Sourcebook on Asbestos Diseases, vol. 17. Charlottesville: Lexis; 1998;17:43-69.
4. Hillerdal G. Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures. *Occup. Environ. Med.* 1999;56:505-13.
5. Finkelstein MM, Dufresne A. Inferences on the Kinetics of Asbestos Deposition and Clearance Among Chrysotile Miners and Millers. *Am. J. Ind. Med.* 1999;35:401-12.
6. Rogers AJ, Leigh J, Berry G, et al. Relationship between Lung Asbestos Fiber Type and Concentration and Relative Risk of Mesothelioma: a Case-Control Study. *Cancer* 1991;67:1912-20.
7. Sebastien P, McDonald JC, McDonald AD. Respiratory Cancer in Chrysotile Textile and Mining Industries: Exposure Inferences from Lung Analysis. *Br. J. Ind. Med.* 1989;46:180-7.
8. Hughes JM, Weill H. Asbestosis as a Precursor of Asbestos Related Lung Cancer: Results of a Prospective Mortality Study. *Br. J. Ind. Med.* 1991;48:229-33.
9. Camus M, Siemiatycki J, Meek B. Nonoccupational Exposure to Chrysotile Asbestos and the Risk of Lung Cancer. *N. Engl. J. Med.* 1998;338:1565-71.
10. Lash TL, Crouch EA, Green LC. A Meta-Analysis of the Relation Between Cumulative Exposure to Asbestos and Relative Risk of Lung Cancer. *Occup. Environ. Med.* 1997;54:254-63.
11. Blettner M, Sauerbrei W, Schlehofer B, et al. Traditional Reviews, Meta-Analyses and Pooled Analyses in Epidemiology. *Int. J. Epidemiol.* 1999;28:1-9.
12. Goodman M, Morgan RW, Ray R, et al. Cancer in Asbestos-Exposed Occupational Cohorts: a Meta-Analysis. *Cancer Causes Control* 1999;10:453-65.
13. 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.
14. de Klerk NH, Armstrong BK. The Epidemiology of Asbestos and Mesothelioma. In: Henderson DW, Shilkin KB, Langlois SL, Whitaker D, eds. *Malignant Mesothelioma*. New York: Hemisphere; 1992:223-50.
15. 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.
16. 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;13:141-66.
17. Hillerdal G, Henderson DW. Asbestos, Asbestosis, Pleural Plaques and Lung Cancer. *Scand J. Work Environ. Health* 1997;23:93-103.
18. Multiple authors. Consensus Report: Asbestos, Asbestosis, and Cancer: the Helsinki Criteria for Diagnosis and Attribution. *Scand. J. Work Environ. Health* 1997;23:311-6.

19. Case BW, Dufresne A, McDonald AD, et al. Asbestos Fibre Type and Length in Lungs of Chrysotile Textile and Production Workers: A Preliminary Report. Unpublished draft manuscript 1999.
20. Case BW, Dufresne A. Asbestos Fibre Type and Length in Lungs of Chrysotile Textile and Production Workers: a Preliminary Report. In: VII International Symposium on Inhaled Particles. Maastricht, October 1999.
21. Green FH, Harley R, Vallyathan V, et al. Exposure and Mineralogical Correlates of Pulmonary Fibrosis in Chrysotile Asbestos Workers. *Occup. Environ. Med.* 1997;54:549-59.
22. Dement JM, Brown DP, Okun A. Follow-Up Study of Chrysotile Asbestos Textile Workers: Cohort Mortality and Case-Control Analyses. *Am. J. Ind. Med.* 1994;26:431-47.
23. Rödelsperger K, Weitowitz HJ, Bruckel B, et al. Dose-Response Relationship between Amphibole Fiber Lung Burden and Mesothelioma. *Cancer Detect. Prevent.* 1999;23:183-93.
24. Dement JM, Brown DP. Lung Cancer Mortality among Asbestos Textile Workers: A Review and Update. *Ann. Occup. Hyg.* 1994;38:525-32, 412.
25. Kumagai S, Nakachi S, Kurumatani N, et al. Estimation of Asbestos Exposure among Workers Repairing Asbestos Cement Pipes Used for Conduits [Japanese]. *Sangkyo Igaku* 1993;35:178-87.
26. Rödelsperger K, Weitowitz H-J, Krieger HG. Estimation of Exposure to Asbestos-Cement Dust on Building Sites. In: Wagner JC, ed. *Biological effects of Mineral Fibres*, vol. 2. Lyon: IARC; 1980:845-53.
27. Hodgson JT, Peto J, Jones JR, Matthews FE. Mesothelioma Mortality in Britain: Patterns by Birth Cohort and Occupation. *Ann. Occup. Hyg.* 1997;41, suppl. 1:129-33.
28. Jarvholm B, Englund A, Albin M. Pleural Mesothelioma in Sweden: An Analysis of the Incidence According to the Use of Asbestos. *Occup. Environ. Med.* 1999;56:110-3.
29. Harrison PT, Levy LS, Patrick G, et al. Comparative Hazards of Chrysotile Asbestos and its Substitutes: a European Perspective. *Environ. Health. Perspect.* 1999;107:607-11.
30. Warheit DB, Hartsky MA, Frame SR. Pulmonary Effects in Rats Inhaling Size-Separated chrysotile Asbestos Fibres or p-Aramid Fibrils: Differences in Cellular Proliferative Responses. *Toxicol. Lett.* 1996;88:287-92.
31. Hesterberg TW, Miller WC, McConnell EE, et al. Chronic Inhalation Toxicity of Size-Separated Glass Fibers in Fischer 344 rats. *Fundament. Appl. Toxicol.* 1993;20:464-76.
32. Hesterberg TW, Miller WC, Thevenaz P, Anderson R. Chronic Inhalation Studies of Man-Made Vitreous Fibres: Characterization of Fibres in the Exposure Aerosol and Lungs. *Ann. Occup. Hyg.* 1995;39:637-53.
33. Hesterberg TW, Chase G, Axten C, et al. Biopersistence of Synthetic Vitreous Fibers and Amosite Asbestos in the Rat Lung Following Inhalation [published erratum appears in *Toxicol. Appl. Pharmacol.* 1999;155:292]. *Toxicol. Appl. Pharmacol.* 1998;151:262-75.
34. Wilson R, Langer AM, Nolan RP. A Risk Assessment for Exposure to Glass Wool. *Regul. Toxicol. Pharmacol.* 1999;30:96-109.

Dr. Infante:

Ascoli V. et al. Malignant mesothelioma in Rome, Italy 1980-1995. A retrospective study of 79 patients. *Tumori* 82:526-532, 1996.

Baeten J. et al. Nature, structure, and properties of asbestos cement dust. *Br. J. Industr. Med.* 37:33-41, 1980.

Berry G. and Newhouse ML. Mortality of workers manufacturing friction materials using asbestos. *Br. J. Industr. Med.* 40:1-7, 1983.

Begin R. et al. Work-related mesothelioma in Quebec, 1967-1990. *Am. J. Industr. Med.* 22:531-542, 1992.

Berry G. et al. Asbestosis: a study of dose-response relationships in an asbestos textile factory. *Br. J. Industr. Med.* 36:98-112, 1979.

Camus M. et al. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *New Eng. J. Med.* 338:1565-1571, 1998.

DeKlerk NH. et al. Cancer mortality in relation to measures of occupational exposure to crocidolite at Wittenoom Gorge in Western Australia. *Br. J. Industr. Med.* 46:529-536, 1989.

Dement JM. et al. Exposures and mortality among chrysotile asbestos workers. Part I: Exposure estimates. *Am. J. Industr. Med.* 4:399-419, 1983.

Dement JM. et al. Follow-up study of chrysotile asbestos textile workers: Cohort mortality and case-control analyses. *Am. J. Industr. Med.* 26:431-447, 1994.

Enterline PE. Et al. Asbestos and cancer: a cohort followed up to death. *Br. J. Industr. Med.* 44:396-401, 1987.

Finkelstein MM. Asbestosis in long-term employees of an Ontario asbestos-cement factory. *Am. Rev. Resp. Dis.* 126:496-501, 1982.

Gibbs GW and Lachance M. Dust exposure in the chrysotile asbestos mines and mills of Quebec. *Arch. Environ. Health* 24:189-197, 1972.

Harless KW. Et al. The acute effects of chrysotile asbestos exposure on lung function. *Environ. Res.* 16:360-372, 1978.

Hughes JM. et al. Mortality of workers employed in two asbestos cement manufacturing plants. *Br. J. Industr. Med.* 44:161-174, 1987.

Infante PF. et al. Fibrous glass and cancer. *Am. J. Industr. Med.* 26:559-584, 1994.

International Agency for Research on Cancer. IARC Monograph Vol. 19:341-359, 1979.

International Agency for Research on Cancer. IARC Monograph on the Evaluation of Carcinogenic Risk to Humans. Vol. 68: Silica, Some Silicates, Coal Dust and Para-aramid Fibrils. IARC, Lyon, 1997.

International Agency for Research on Cancer. IARC Monograph on the Evaluation of Carcinogenic Risk to Humans. Vol. 43: Man-made Mineral Fibres and Radon. IARC, Lyon, 1988.

International Agency for Research on Cancer. IARC Monograph on the Evaluation of Carcinogenic Risk to Humans. Overall Evaluations of the Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, Suppl. 7, Lyon, 1987

Iwatsubo Y. et al. Pleural mesothelioma: Dose-response relation at low levels of asbestos exposure in a French population-based case-control study. *Am. J. Epid.* 148:133-142, 1998.

Magnani C. et al. A cohort study on mortality among wives of workers in the asbestos cement industry in Casale Monferrato, Italy. *Br. J. Industrl. Med.* 50:779-784, 1993.

McDonald AD. et al. Epidemiology of primary malignant mesothelioma tumors in Canada. (In *Pneumoconiosis Proceedings of the International Conference Johannesburg 1969*, Shapiro, HA ed.) Cape Town, Oxford University Press, 1970. pp.197-200.

McDonald AD and McDonald JC. Malignant mesothelioma in North America. *Cancer* 46:1650-1656, 1980.

McDonald AD. et al. Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. *Br. J. Industrl. Med.* 39:368-374, 1982.

McDonald AD. et al. Dust exposure and mortality in an American chrysotile textile plant. *Br. J. Industrl. Med.* 40:361-367, 1983.

McDonald JC. et al. The 1891-1920 birth cohort of Quebec chrysotile miners and millers: mortality 1976-88, 1993.

Muhle H. et al. Investigation of the durability of cellulose fibres in rat lungs. *Ann. Occup. Hyg.* 41: (Suppl 1)184-188, 1997.

Occupational Safety and Health Administration. 29 CFR Parts 1910, et al. Occupational Exposure to Asbestos: Final Rule. *Federal Register* 59, No. 153:40964-41158, 1994.

Peto J. et al. Continuing increase in mesothelioma in Britain. *The Lancet* 345:535-539, 1995.

Peto J. et al. Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. *Ann. Occup. Hyg.* 29:305-355, 1985.

Rodelsperger K. et al. Estimation of exposure to asbestos-cement dust on building sites. (In *Biological Effects of Mineral Fibres*, Wagner, JC, ed.) IARC Scientific Pub. No. 30, International Agency for Research on Cancer, Lyon, 1980. pp. 845-853.

Rohl AN. et al. Asbestos exposure during brake lining maintenance and repair. *Environ. Res.* 12: 110-128, 1976.

Searl A. Clearance of respirable para-aramid from rat lungs: Possible role of enzymatic degradation of para-aramid fibrils. *Ann. Occup. Hyg.* 41 (Suppl 1):148-153, 1997.

Shannon HS. Et al. Mortality experience of Ontario glass fibre workers—Extended follow-up. *Ann. Occup. Hyg.* 31:657-662, 1987.

Stauder B. et al. X-ray results in roofers after exposure to dust from working with asbestos-cement for many years. *trans.* 1982.

Stayner LT. et al. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. *Am. J. Pub. Health* 86:179-186, 1996.

Stayner LT. et al. Exposure-response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. *Occup. Environ. Med.* 54:646-652, 1997.

Spurny KR. On the release of asbestos fibers from weathered and corroded asbestos cement products. *Environ. Res.* 48:100-116, 1989.

Teta MJ. Et al. Mesothelioma in Connecticut, 1955-1997. Occupational and geographic associations. *J. Occup. Mrd.* 25:749-756, 1983.

Tossavainen A. et al. Health and exposure surveillance of Siberian asbestos miners: A joint Finnish-American-Russian project. *Am. J. Industrl. Med.* (Suppl 1):142-144, 1999.

Wagner JC. et al. The effects in the inhalation of asbestos in rats. *Br. J. Cancer* 29:252-269, 1974.

Warheit DP. Et al. Pulmonary effects in rats inhaling size-separated chrysotile asbestos fibres or *p*-aramid fibrils: differences in cellular proliferative responses. *Toxicol. Letters* 88:287-292, 1996.

Woitowitz H. and Rodelsperger K. Chrysotile asbestos and mesothelioma. *Am. J. Industrl. Med.* 19:551-553, 1991.

ANNEX IV

Canada's Comments on the Experts' Responses to the Questions from the Panel

APPENDIX A

EXAMPLE OF THE APPLICATION OF A CONTROLLED USE POLICY IN THE FRICTION INDUSTRY

The following example applies to friction product manufacture and use. In fact, for this sector, data indicate that even with past work practices, the risk, if any, for friction product manufacturing workers and mechanics has been extremely low. Canada presents it as a manner of achieving France's desired level of safety, which is less trade restrictive than the ban. Company M wishes to manufacture brake linings, brake disc pads and dry clutches using chrysotile asbestos. The asbestos will be purchased from a producer of chrysotile P. Manufacturer will sell friction products through a distributor D to automobile manufacturers and to automobile service centres G.

The steps that would be followed under the proposed controlled use programme are as follows:

1. Company M requests a permit from the competent government authority to import chrysotile asbestos.

The government authority grants permit only if:

- (a) Company M has in place the equipment, training programmes and work practices to protect workers from chrysotile exposure throughout manufacture and disposal of any waste materials.
 - (b) Producer P would inspect the plant to ensure that all the regulated fibre-handling processes are in place to eliminate/minimize any potential for exposure.
 - (c) Company M would provide the results of periodic measurements of the exposure of workers to the producer.
2. Once Company M has the import permit, the chrysotile producer will supply chrysotile to it, on the understanding that shipments will cease immediately if Company M fails to meet or exceed all applicable standards.
 3. The chrysotile will be shipped in sealed containers to preclude exposure of workers, transportation personnel or the public during chrysotile shipment.
 4. On arrival at the plant, the transfer of fibres to the process will be such as to eliminate workers opening bags of asbestos [e.g. automatic, sealed bag handling].

Risks of Lung Cancer and Mesothelioma for Friction Manufacturing Workers

A study of some 13,000 friction manufacturing workers in the United Kingdom in which lifetime estimated exposure to chrysotile ranged up to 356 f/ml-years [i.e. equivalent to 40 years exposure at just below 9f/ml] found no chrysotile-related increased risk of lung cancer or mesothelioma.

In this study the authors concluded: "with good environment control, chrysotile asbestos may be used in manufacture without causing excess mortality" [Berry & Newhouse 1983, Newhouse &

Sullivan 1989, Berry 1994]. This study involved workers exposed up to 50-60 years ago, so controls were poor relative to present day standards.

In the USA, another study found no mesothelioma among 1630 deaths in persons manufacturing friction products [McDonald et al 1984].

Exposure Levels

The concentrations to which persons in the cohort studied by Berry and Newhouse were exposed were considerably higher than those reported in the Australian friction product plant by Dr. Henderson, even including peaks above the standard.

Control Feasibility

The technology and work practices to control exposures during manufacture exist and the experts appear to agree that exposures during product manufacture can be well controlled.

5. After manufacture under controlled conditions, the products will be shipped to the distributor in sealed packages. The manufacturer will ensure that all distributors have the knowledge and have a proper place to store the products without removing them from the original packaging.

Exposure Levels

As the product is composed of chrysotile in a high-density matrix and as it is sent in sealed containers [e.g. boxes], no persons at the distributor have potential for exposure.

6. On request, the distributor will deliver the products to the automobile manufacturers in the sealed containers. The manufactured products will be ready-mounted linings and pads and clutches which require no modification by the installer.

Exposure Levels

The brake linings and brake disc pads consists of chrysotile embedded in resin. Measurements have shown that exposure from the handling of these products is, at most, minuscule.

7. On request, the distributor will deliver the products in the sealed containers to automotive service centres. The range of sizes of brake linings (e.g. oversize) already exists so that when brake drums are turned (to eliminate scoring etc.) the lining of appropriate thickness is available for installation without modification. If it becomes necessary to have linings "ground to size" this would only be permitted at "authorized centres", equipped with the appropriate exhaust ventilation systems. These centres would be identified to the manufacturer and competent authority. They would be at the same locations as those undertaking the turning of brake drums so that the linings could be fitted under controlled conditions and it would not be necessary to modify them when they are returned to the automotive repair centre.

Exposure Levels

The technology exists to do this work with virtually no exposure. [See NIOSH Report]

8. The distributor will provide the manufacturer and competent authority with a list of the names and addresses of purchasers. The purchaser will be informed that this list has been provided to the manufacturer and competent authority. In this way, the competent authority can readily target those locations where chrysotile-containing products are used. If the distributor, manufacturer or competent

authority has reason to believe that safe work practices are not being followed, the supply of friction products would be discontinued.

9. The removal and installation of brake shoes, brake disc pads and clutches will be carried out according to precise codes of practice. This would include clean up and disposal requirements.

Risks of Lung Cancer and Mesothelioma for Brake/Clutch Repair Mechanics

The requirements for controlled use here go well beyond those necessary to protect worker's health.

The following studies demonstrate that workers carrying out brake repair work are not at an increased risk of lung cancer or mesothelioma.

Hansen, ES [1989] Mortality of Auto Mechanics. A Ten Year Follow-Up. Scand J Work Environ. Health 15 43-46 1989.

McDonald AD, Fry JS, Woolley AJ & McDonald JC [1984]. Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Product Plant. Brit. Industr. Med. 41 151-157.

Berry G & Newhouse ML [1983]. Mortality of Workers Manufacturing Friction Materials Using Asbestos. Brit. J. Industr. Med. 40 1-7.

Newhouse ML & Sullivan KR [1989]. A Mortality Study of Workers Manufacturing Friction Materials; 1941-86 Brit. J. Indust. Med. 46 176-179.

Berry G [1994] Mortality and Cancer Incidence of Workers Exposed to Chrysotile Asbestos in the Friction Products. Ann. Occup. Hyg. 38 539-546.

McDonald AD & McDonald JC [1980] Malignant Mesothelioma in North America. Cancer 46 1650-1656.

Teta MJ, Lewinsohn HC, Meigs JW, Vidone A, Mowad LZ & Flannery JT, [1983] Mesothelioma in Connecticut. JOM 15 749-756. Ann. Occup. Hyg. 38 539-546.

Woitowitz H.-J and Rodelsperger K [1994] Mesothelioma among Car Mechanics? Ann. Occup. Hyg. 38 635-638.

Jarvholm B & Brisman J [1988] Asbestos Associated Tumours in Car Mechanics. Brit. J. Industr. Med. 45 645-646.

Malker HS, McLaughlin JK, Malker BK, Stone BJ, Weiner JA, Erickson JL & Blot WJ [1985]. Occupational Risks for Pleural Mesothelioma in Sweden. J. Natl. Cancer. Inst.. 74 61-566.

Hodgson JT, Peto J, Jones JR and Matthews FE [1997] Mesothelioma Mortality in Britain: Patterns by Birth Cohort and Occupation. Ann. Occup. Hyg. 41 129-133.

Hutchings S, Jones J, Hodgson J Asbestos-Related Diseases. In F Drever [ed] Occupational Health, Decennial Supplement, HSE London, 1996 127-152.

Teschke et al, [1997] Mesothelioma Surveillance to Locate Sources of Exposure to Asbestos. Can. J. Publ. Health. 88 163-168.

The fact that there is no increased risk of lung cancer during manufacture of friction products shows that at exposure levels well above those of brake mechanics, there is no chrysotile related increased risk of lung cancer or mesothelioma.

Exposure Levels

The requirement and work practices exist and have been shown under field use conditions to reduce workers' exposure during brake repair work to well below 0.01 f/ml. [See NIOSH reports].

In the 1980s the average concentrations to which brake repair mechanics were reported to be exposed in Finland were less than 0.05 f/ml for automobile brake mechanics and less than 0.1 f/ml for truck and bus brake mechanics. [Kauppinnen & Korhonen]. Similar results were found in Germany where the lifetime exposure of brake mechanics after more than 20 years of full time brake work was less than 14 f/ml-years. These exposures took into account grinding, bevelling, sanding and otherwise modifying the brake linings as well as using compressed air to remove brake wear debris from brake drums.

The exposure of workers from work on clutches in the past was even lower than that associated with brakes [Lynch (1968), Kauppinnen & Korhonen (1987), Jacko & Ducharme 1973].

10. On removal of brake shoes, brake discs and clutches from the vehicles, these will be placed in containers provided by the distributor and returned through the distributor to the manufacturer.

11. As the worn brake shoes are returned to the manufacturer, any re-lining by unauthorised companies/persons is precluded. Any re-lining of brakes will be done as subcontracts by the brake lining manufacturer and with equipment and work practices that are no less stringent than those required of the manufacturer. There will be no brake lining material sold to other "re-lining companies".

12. Disposal of any used brake lining, clutch facing or brake disc pad will be done according to jurisdictional requirements.

Environmental Releases and Public Health Risks

Data show that during braking or use as a friction product, chrysotile is altered to non-asbestos mineral or amorphous silicates. Thus the bulk of the material to which workers are exposed from used brakes is not asbestos as mentioned by one of the experts. Also, almost all residual fibres are very short [e.g. > 80% of fibres are less than 0.4µm in length].

Because of the mineralogical and particle size alterations, the environmental release of chrysotile fibres greater than 5 µm from the use of chrysotile containing friction products is extremely low in the case of brakes and essentially nil in the case of clutches. [Lynch, JR (1968) Brake Lining Decomposition Products. J. Air Pollution Control Assoc. 18: 824-826]. Concentrations of chrysotile fibres measured at street level have also been very low. The data obtained in the United Kingdom under situations of very heavy vehicular traffic indicate that the use of asbestos in brake linings does not measurably contribute to atmospheric asbestos concentrations in the urban environment. Even at two heavily used intersections in the London metropolitan area, concentrations vary from 0.0002 to 0.0004 f/ml. Jaffrey, S (1990) Environmental Asbestos Fibre Release from Brake and Clutch Linings in Vehicular Traffic. Ann. Occup. Hyg. 34:529-534.

As there is no indication of an increased risk of lung cancer or mesothelioma in friction product workers or brake mechanics exposed at many orders of magnitude above the general public, the actual risk for the public at their levels of exposure will be epidemiologically undetectable.

APPENDIX B**EXAMPLE OF THE APPLICATION OF A CONTROLLED USE POLICY
IN THE ASBESTOS -CEMENT INDUSTRY**

The major portion of current chrysotile cement products is for outdoor applications, such as roofing, exterior wall cladding, rain gutters, pipes, etc. Chrysotile fibres are transported from fibre suppliers to asbestos cement plants, packed in sealed 50 kg plastic bags piled and "stretched-wrapped" on pallets, and are delivered to the plant premises in closed containers. Thus the possibility of dust emissions during transport is practically nil. Fibres are delivered to asbestos cement plants that comply with a "Controlled use" code of practices. This includes prohibition of reselling of unused fibre inventories to third parties by the asbestos cement plant manufacturers. Suppliers of chrysotile asbestos from Canada, Brazil, Zimbabwe and Swaziland have signed and endorsed a *Memorandum of Understanding on Responsible-Use of Chrysotile Asbestos*, whereby the signatories agree in particular that they will "provide a written commitment to appropriate national authorities indicating that chrysotile asbestos will be supplied directly to chrysotile asbestos-product manufacturing facilities on condition that chrysotile asbestos not be resold upon delivery ..."

Typical flow of the main steps from manufacture to disposal

1. In-plant handling of chrysotile fibres through the different steps (wet process) leading to the finished product:
 - (a) Bag opening inside hoods under negative pressure. Operators must wear protective equipment;
 - (b) wet-processing of the fibre cement mix, shaping of the product, wet-curing as the case may be, and wet practices for final shaping and cutting of the various products.

Comment

All work must be carried out according to safe work practices such as those described in the ILO Code of Practice "Safety in the Use of Asbestos", chapter 13, and under engineering controls that have been shown to reduce occupational air concentrations to levels presenting a negligible, undetectably low health risk, as shown by the following published data:

Thomas, H.F., Benjamin, I.T., Elwood, P.C. and Sweetnam, P.M. (1982). Further Follow-Up Study of Workers from an Asbestos Cement Factory. British Journal of Industrial Medicine 39(3):273-276.

In an asbestos cement factory using chrysotile only, 1,970 workers were traced, and their mortality experience was examined. There was no appreciably raised standardised mortality ratio (SMR) for the causes of death investigated, including all causes, all neoplasms, cancer of the lung and pleura, and cancers of the gastrointestinal tract. The authors indicate: "Thus the general results of this mortality survey suggest that the population of the chrysotile asbestos 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."

Weil, H., Hughes, J. and Waggenpack, C. (1979). Influence of Dose and Fibre Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing. American Review of Respiratory Disease 120(2): 345-354.

An investigation of 5,645 asbestos cement manufacturing workers, showing no raised mortality resulting from exposure for 20 years to chrysotile asbestos at exposure levels equal to or less than 100 MPPC.years (corresponding to approximately 15 fibres/ml.years). The authors state:

"... However, the demonstration that low cumulative and short-term exposures did not produce a detectable excess risk for respiratory malignancy may be of assistance in the development of regulatory policy, because a scientifically defensible position based on these data is that there are low degrees of exposure not associated with a demonstrable excess risk".

Ohlson, C.-G. and Hogstedt, C. (1985). Lung cancer among asbestos cement workers. A Swedish cohort study and a review. British Journal of Industrial Medicine 42(6):397-402.

A cohort study of 1,176 asbestos cement workers in a Swedish plant using chrysotile asbestos showing no excess related mortality at exposures of about 10-20 fibres/ml.years.

Gardner, M.J., Winter, P.D., Pannett, B. and Powell, C.A. (1986). Follow-Up Study of Workers Manufacturing Chrysotile Asbestos Cement Products. British Journal of Industrial Medicine 43:726-732.

A cohort study carried out on 2,167 subjects employed between 1941 and 1983. No excess of lung cancers or other asbestos-related excess death is reported, at mean fibre concentrations below 1 f/ml, although higher levels had probably occurred in certain areas of the asbestos cement factory.

2. Delivery of pre-cut, pre-drilled chrysotile cement products (according to client's specifications) to licensed contractors, with notification to government authorities.

3. Installation on the work site of the pre-sized, pre-drilled chrysotile cement product must be performed by workers who have received an approved training programme, which includes mandated work practices and working tools, such as those described in the above-mentioned ILO Code of Practice, chapter 13.4, and also in "Catalogue of Tools for Working with Asbestos Cement Products on Site" (AIA Recommended Control Procedure No. 2A). This will ensure that fibre emissions are kept at levels where measurable health risk is unlikely, and undetectably low.

Comment

Evidence published in 1980 by Rödelsperger et al (IARC Sci. Pub. No. 30, pp. 845-853) shows high levels of exposure of up to 100 f/ml peak exposure for workers installing roofing shingles when using high-speed grinding power tools. This is clearly not a situation where "controlled use" was observed. However, more recently available data show that installation of asbestos cement roofing shingles will not result in workers' personal exposures to levels associated with detectable risk. All sample results of measurements are below 0,1 f/ml.

Bonacci et al (1987) "Report of Industrial Hygiene Survey at J. Alloca Residence, Florham, NJ". SSM Analytical Laboratory, Reading, PA, USA.

The same observations (below 0,1 f/ml) were made during removal of old roofing asbestos cement shingles. Complete data is found in:

Bonacci et al (1998) "Report of Industrial Hygiene Survey at 10233 Norton Road, Potomac, MD". SSM Analytical Laboratory, Reading, PA, USA.

Similar low exposure levels have also been measured during various operations on old weathered asbestos cement sheets (water jet cleaning or painting, demolition by removal of whole sheets from roofs and walls). This data can be found in:

Brown SK (1987) Asbestos exposure during renovation and demolition of asbestos-cement clad buildings. Amer. Ind. Hyg. Assoc. J. 48:478-486.

In case of asbestos cement pipes, when improper tools and work practices, such as using high speed, abrasive disks for cutting pipes for instance, exposure as high as 35 f/ml may result. However, operations such as using a manual or power lathe for cutting sewer pipes, and using the adequate power hole cutter (about 15-20 min duration) will result in exposures in the 0,1-0,2 f/ml range. Such data can be found in:

Noble et al (1977) "Asbestos exposures during the cutting and machining of asbestos cement pipes". Report prepared by Equitable Environment Health, Inc., Berkeley, CA.

As mentioned earlier, chrysotile cement products are essentially found in outdoor applications or in underground pipes, thereby not very likely to be subject to interventions by tradesmen such as plumbers or electricians, after their installation. If interventions are required, use of appropriate tools and work practices as described in many international standards (ref. previous comments on question 5(a)) will prove sufficient to manage any potential risk, if any.

4. During the normal service life of the asbestos cement product, emissions from in-place asbestos cement do not result in measurable increases above the average, naturally occurring environmental air concentrations.

Comment

Evidence supporting this can be found in the following published data:

W. Felbermayer and M.B. Ussar (1980) Research Report: "Airborne Asbestos Fibres Eroded from Asbestos Cement Sheets", Institut für Umweltschutz und Emissionsfragen, Leoben, Austria.

... "A comparison of the asbestos fibre concentrations in those areas with and without A/C roofing ... lead to the conclusion that there is no statistically significant connection between the use of asbestos cement materials and the asbestos fibre concentrations found in the various measurement areas".

Ullrich Teichert (1986) Immissionen durch Asbestzement-Produkte, Teil 1. Staub Reinhaltung der Luft, Vol. 46, No. 10, pp. 432-434.

... "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 1000f/m³ (length =5 µm)". (translation)

5. Disposal of asbestos cement plant and demolition waste must be carried out according to well-known waste management practices approved by national authorities.

Comment

Proper disposal site management practices have shown that there is no measurable additional burden to the naturally occurring environmental fibre concentrations, as is illustrated in the following example:

Marfels et al (1988) Staub Reinhaltung der Luft, 48: 463-464

This report is about a survey of air concentrations at disposal sites in Germany, showing the following data:

- *directly over disposal sites: 0,0005 to 0,003 f/ml*
- *vicinity of disposal sites: 0,0001 to 0,0009 f/ml*

ANNEX VComments of the European Communities on the Replies by the Scientific Experts
to the Questions from the Panel

SUMMARY OF REPLIES BY THE EXPERTS

QUESTION NO.	P. INFANTE	N. H. DE KLERK	D. W. HENDERSON	A. W. MUSK
1(a) Main categories of workers at risk	Principally secondary users (building industry, intervention, maintenance, ...)	Principally secondary users (building industry, intervention, maintenance, ...)	Principally secondary users (building industry, intervention, maintenance, ...)	Principally secondary users (building industry, intervention, maintenance, ...)
1(b) Mainly an occupational or an environmental risk	Mainly an occupational or occupation-related risk	Mainly an occupational or occupation-related risk, but also environmental	Mainly an occupational or occupation-related risk	Mainly an occupational or occupation-related risk, but also environmental
1(c) Release of fibres through degradation of asbestos cement	Fibre release; small, non-quantifiable risk	Fibre release; small, non-quantifiable risk	Fibre release; small, non-quantifiable risk	Fibre release; small, non-quantifiable risk
1(d) Fibre release during intervention on asbestos cement	Release of large quantities of fibre; established risk	Release of large quantities of fibre; established risk	Release of large quantities of fibre; established risk	Release of large quantities of fibre; established risk
1(e) Release of fibres during intervention on non-friable products containing chrysotile	Release of fibres; established risk for workers and handymen	Release of fibres; established risk for workers and handymen	Release of fibres; established (non-quantifiable) risk for workers and handymen	Release of fibres; established risk for workers and handymen
1(f) Danger of the fibres released by asbestos cement	Dangerous fibres	Dangerous fibres	Dangerous fibres (at least some of them)	Dangerous fibres
1(g) Risk during demolition and removal of asbestos	Established risk	Established risk	Probable risk	Established risk
1(h) Risk of wastes	Theoretical risk, probably low	Theoretical risk (if not handled properly)	Theoretical risk (if not handled properly)	Theoretical risk (if not handled properly)
1(h) Safety when disposing of the waste	No opinion	Theoretical risk (if not handled properly)	Theoretical risk (if not handled properly)	Theoretical risk (if not handled properly)
2. Risk associated with other applications of chrysotile	Established risk	Established risk	Established risk for most applications; likely for others	Established risk
3(a)(b)(c): Relative pathogenicity of chrysotile/amphibole	Chrysotile and amphiboles are carcinogens for lung cancer and mesothelioma Comparable chrysotile carcinogenicity for lung cancer; not likely to be so high for mesothelioma The physical and chemical characteristics play a decisive role	Chrysotile and amphiboles are carcinogens for lung cancer and mesothelioma Higher amphibole carcinogenicity (specific response not according to type of cancer) The physical and chemical characteristics play a decisive role	Chrysotile and amphiboles are carcinogens for lung cancer and mesothelioma Lower chrysotile carcinogenicity for mesothelioma; probably comparable for lung cancer The physical and chemical characteristics play a decisive role	Chrysotile and amphiboles are carcinogens for lung cancer and mesothelioma Amphibole carcinogenicity higher for mesothelioma and lung cancer The physical and chemical characteristics play a decisive role
4(a): Epidemiological data for low levels of exposure to chrysotile	Established risk for many professions	Some studies do not show a high risk	No data quantifying the exposure-effect relationship	Some studies do not show a high risk
4(b): Safety threshold	No threshold for any disease	Impossible to demonstrate the existence of a threshold	No threshold (except for asbestosis)	No threshold for any disease

QUESTION NO.	P. INFANTE	N. H. DE KLERK	D. W. HENDERSON	A. W. MUSK
4(c)(d): Linear model	The linear model is the most appropriate; no credible alternative model	The linear model is the most appropriate; no credible alternative model	The linear model is the most appropriate; no credible alternative model	The linear model is the most appropriate; no credible alternative model
4(e): Concentration/duration of exposure to chrysotile	No lower level of exposure without risk	No lower level of exposure without risk	No lower level of exposure without risk	No lower level of exposure without risk
5(a)(b)(c)(d)(e) "Controlled" use	Impossible in practice in the vast majority of situations	Impossible in practice in the vast majority of situations	Impossible in practice in the vast majority of situations	Impossible in practice in the vast majority of situations
6(a): Risks of non-fibrous substitutes	Not carcinogenic	No direct reply: only fibrous substitutes should be taken into account for cancer risk	No direct reply: only fibrous substitutes should be taken into account for cancer risk	No direct reply: only fibrous substitutes should be taken into account for cancer risk
6(b)(c): Physical and chemical characteristics of substitute fibres; relative risk in comparison with chrysotile	<p>Dimensions and form (whether or not they can be inhaled) and durability are associated with toxicity</p> <p>All substitute fibres are less dangerous than chrysotile</p>	<p>Dimensions and form (whether or not they can be inhaled) and durability are associated with toxicity</p> <p>All substitute fibres are less dangerous than chrysotile</p>	<p>Dimensions and form (whether or not they can be inhaled) and durability are associated with toxicity</p> <p>All substitute fibres are less dangerous than chrysotile (doubts concerning refractory ceramic fibres)</p>	<p>Dimensions and form (whether or not they can be inhaled) and durability are associated with toxicity</p> <p>All substitute fibres are less dangerous than chrysotile</p>

ANNEX VI**Meeting with Experts – 17 January 2000***Transcript***Chairman**

1. I would like to welcome the four scientific experts and the delegations of Canada and the European Communities. I should like to introduce the Panel members and the Secretariat staff especially for the benefit of anyone who wasn't at previous meetings. My name is Adrian Macey, on my right is Mr. Lindén and his right Mr. William Ehlers. In the Secretariat staff, the Secretary to the Panel is Ms. Mireille Cossy and Assistant Secretary Ms. Doaa Abdel-Motaal. The Legal Officers are Mr. Yves Renouf and Ms. Kerry Allbeury. I would like to remind everybody that we have simultaneous interpretation in French and English. Secondly, the proceedings will be recorded and subsequently transcribed. The verbatim transcript will become an integral part of the final report. I would like now to invite the experts to introduce themselves, going in alphabetical order.

Dr. de Klerk

2. My name is Nick de Klerk, I work as an epidemiologist in asbestos-related diseases in Western Australia.

Dr. Henderson

3. My name is Douglas Henderson. I am the Professor of Pathology at the Flinders University of South Australia and the Flinders Medical Centre. I have been pursuing an interest in asbestos-related diseases for some 32 years.

Dr. Infante

4. Peter Infante. I am an epidemiologist and I am with the United States Occupational Safety and Health Administration.

Dr. Musk

5. I am Bill Musk, a clinical professor of medicine and public health of the University of Western Australia.

Chairman

6. We have received lists of the two delegations, Canada and the European Communities. Could we ask the delegations' leaders to indicate who is who amongst your delegations? Can I ask firstly Canada to introduce themselves?

Mr. Hankey (Canada)

7. Thank you, Chairman. I am Blair Hankey, Associate General Counsel at the Department of Foreign Affairs and International Trade. I have on my right Maître Thomas-Louis Fortin who is Legal Counsel at the Ministry, Eric Wildhaber, who is also Legal Counsel and Sebastien Beaulieu, also Legal Counsel at the Department of Foreign Affairs and International Trade. Also opposite me is André Dulude, who is Director of the Regulation and Technical Barriers Division at the Department,

and Pierre Desmarais from the same Division. Behind me, I have Louis Perron from the Canadian Ministry of Natural Resources, and on his left, Gilles Mahoney, who is Director of the mineral industry for the "Ministère des ressources naturelles" of the Government du Québec. Then, on my left, I have Professor Corbett McDonald, as scientific adviser to our delegation, and Professor Alison McDonald. On my right, I have Dr. Graham Gibbs, who is also an expert, and behind me, Dr. Jacques Dunnigan and Dr. Michel Camus, also experts. I would also like to add that the Professors McDonald are serving as honorary members of the delegation and have declined to accept any compensation from Her Majesty in order that both their independence and the appearance thereof may be guarded.

Chairman

8. Can I now ask the representative of the European Communities to briefly introduce their delegation?

Mr. Christoforou (European Communities)

9. Thank you, Mr. Chairman. My name is Theofanis Christoforou, and I am a Legal Adviser of the European Commission in Brussels. We have a big delegation, composed partly of Commission officials and French representatives. We have scientific experts and members of the Member states' delegations which are based here in Geneva. As the presence is quite long, I would rather leave it to each member to present himself or herself shortly.

10. Jean-Jacques Boufflet, Legal Adviser of the Delegation in Geneva of the Commission; Hubert van Vliet, member of the Legal Service of the Commission in Brussels; Dr. A. Tossavinen, Scientific Adviser; Marcel Goldberg, Scientific Adviser; Maud Valat-Taddei, responsable de la réglementation concernant l'amiante, Ministry of Employment and Solidarity, France; Sophie Chaillet, Ministry of Health, France; Marie-Christine Poncin, Ministry of Economy, Finance and Industry, France; Pierre Monnier, Legal Adviser, Permanent Delegation of France to the WTO, Geneva; Christian Forwick, Permanent Mission of Germany in Geneva; Mr. H. Rieck, Permanent Mission of Germany in Geneva; Mr. M. Nielsen, Permanent Mission of Denmark in Geneva; Sergio da Gama, Legal Adviser, Portuguese Mission in Geneva; Jacques Bourrinet, Professor à l'Université d'Aix-Marseille; Mrs. A. Bensch, DG Trade, EC Commission, Brussels; Dr. B. Terracini, Professor, Scientific Adviser; Dr. P. Huré, Scientific Expert; Mr. B. Castleman, Scientific Adviser; Mrs. Mchanetzki, Ministry of Economy, Finance and Industry, France.

Chairman

11. Thank you very much. I would like to explain how the Panel intends to organize its work for today. I would like to thank the four experts for having agreed to serve as advisers on the Panel and for the very hard work that they have managed to perform over such a short period of time. We do operate under significant time constraints in the WTO dispute settlement system and we have to produce reports within certain deadlines. This does put pressure on everybody involved. The purpose of this meeting is essentially to allow the experts to expand on the written responses that they have already given us to the Panel's questions. Obviously, these documents are substantial; we have all received them and it is not a matter of repeating what is already before us. The experts will initially be given an opportunity to make any general comments they may have, and reactions to their own colleagues' reports, as well as on the written comments received from the parties. The parties will be given the opportunity during the meeting to seek clarification on the experts' reports, to express their views on them. The focus, I would stress, is on the experts and on questions to them. So we would ask the parties to limit their interventions to questions and comments directly related to the issues that the experts have raised, either in the existing written comments or orally during the meeting. The experts will, of course, have the invitation to react as they wish to what is being said by the parties. Let us come back again to the point that the priority of the meeting which is to hear the experts. I

hope that the meeting will give us a full opportunity for an exchange of views between the experts, the parties and the Panel, so that, at the end of the day, the Panel can be as fully informed as possible about the scientific and technical issues involved in the case. It is not the purpose of the meeting to hear new evidence which the parties have not previously submitted and we will have to reserve our right to disregard any argument or evidence which is not directly related to statements made by the experts or the other party.

12. Firstly, I need to remind everyone that the proceedings, according to the rules of dispute settlement, are confidential. We will handle the meeting as follows. Firstly, we would like to invite opening statements or comments by each of the experts, which we will do in alphabetical order. It may be that following their brief introductory comments, one or other or more of the experts might like to take up additional points, which they are welcome to do, but when this initial introduction is concluded, it is over to the parties to put their questions and comments. We would like to focus this main part of the meeting question by question, i.e. the questions that we sent to the experts fall under six main headings – there are essentially six main questions with a number of sub-questions under each one. For the clarity and the good order of the meeting, we will try to focus our discussion question by question. So that means that after any opening comments by the parties, we will, in the case of each question, invite first Canada and then the European Communities to raise comments and questions that they have under the first question, and then we will go on to the second question until we get to question six. I would like to invite the parties to be selective in the items that they comment or ask questions on, so that they can be sure that the key points they see as relevant to the dispute can be covered in the time available. We will of course be flexible as the meeting develops, and the Panel will do its best to make sure that we assist you in a smooth progression of the discussion and ensure that we don't leave any important issues aside.

13. There is limited time available, so we can now proceed to the first stage of our meeting, which is to invite each of the experts to make any opening comments that they might wish to do, beginning with Dr. de Klerk. When we have had each of your opening comments, I might return to the experts again and ask if there are any points which you may want to follow up.

Dr. de Klerk

14. I think the six questions fall into three main ones. The first one is whether chrysotile is dangerous at all, and I think the generally accepted view is that it is. A subset of that question is which dose-response relationships are appropriate and whether you should use linear extrapolation or not. A corollary of that is which equation one actually uses, which dose-response, which group of studies one uses to do that, and that seems to be open to a bit of argument. The second question, and perhaps the more easy to answer, is the question about controlled use. It is clear, just by observation in Australia, that you can't control the use of a dangerous product like chrysotile all the way down the chain of use. It is possible that you can do it in the factories that produce it, but certainly down towards end-users it seems almost impossible, and in fact most cases of asbestos-related disease have arisen through such downstream use, probably mainly of other forms of asbestos, but certainly that sort of use. The third question is whether substitutes for asbestos are known to be safer – I would say that the evidence at present is that they are. I think that that is a summary of how I see the issues.

Dr. Henderson

15. At the outset, I would express my great appreciation to the WTO and to the Panel and the Chairman, Dr. Macey, for asking me to act in my capacity as an adviser to the Panel. I would also like to thank the Secretariat and in particular Ms. Mireille Cossy for her consistent helpfulness at all times in responding to my questions and requests. My introductory remarks can be given quite briefly. My full opinion is set out in quite extensive reports already submitted to the WTO, both my original report of November last year with an attached Endnote and, following submission of new information, I did prepare a 26-page summary of additional Remarks which clarify and amplify some

of my opinions and conclusions. Broadly speaking, I see the issues in a way which is very similar to that of my colleague, Dr. de Klerk. The three key issues as I see them are: firstly, is chrysotile carcinogenic for the lung and for the mesothelium? My answer to this is that the evidence is strongly in favour of the fact that it is, and that it is capable of inducing both lung cancer and mesothelioma at reasonably low levels of exposure; for example, low levels of exposure, such as occurred in the South Carolina asbestos textile workers in the studies carried out by Dr. Dement and his colleagues, led to a greater than two-fold increase in the standardized mortality ratio at quite low levels of exposure for white males, in the order of 2.7 to 6.8 fibre-year. And the additional information submitted in this case concerning the possible significance of amphiboles in the lung tissue of those workers does not, in my opinion, detract from the significance of that observation, and the reasons why are set out in my Supplementary Remarks to the Panel.

16. The second point, which I think is a crucial point, is whether or not the use of chrysotile can be controlled at all points of use. Again, I would be in close agreement with my colleague, Dr. de Klerk, that it cannot. My own series of mesotheliomas, amounting to in excess of 2,000 cases, indicates that by far the greatest number of mesotheliomas that I see occur – not in miners and millers nor in products manufacture – but they occur in those exposed to asbestos at the multiple points of end-use. In the Australian Mesothelioma Register, which represents a systematic compilation of all mesotheliomas found in Australia, there is good evidence that the greatest number of mesotheliomas that we see occurs among carpenters, builders' labourers, plumbers, plasterers, painters and all others involved in building construction in particular. In Australia, this group represents a very large workforce; it is usually employed by small business, or the workers are self-employed. In the past, it has not been possible to extend controlled use of asbestos to this group of workers, and to the best of my knowledge, this situation continues today, although the use of chrysotile in building construction materials in Australia was phased out in 1987 or 1989.

17. The third issue that I believe is of significance here, is whether alternative substitute materials for chrysotile are safer than chrysotile. Again from my survey of the literature, I would be in close agreement with my colleague Dr. de Klerk that the evidence available to me indicates that substitute fibres are – according to national and international health authorities – safer for end-use than the use of chrysotile. And these are, I believe, the three key issues for resolution by this Panel.

Chairman

18. Thank you. Dr. Infante, please.

Dr. Infante

19. Thank you for asking me to participate. First, I would like to state what it is that I feel that all of us experts agree upon. That is that chrysotile presents a high risk of cancer to society, to exposed individuals. It is unlikely to ever be controlled enough to use safely. Substitutes appear available, and there is no evidence that they are as harmful as chrysotile asbestos. Regarding some particular studies - I did express this in writing, I want to reiterate it - the Dement study which has been reviewed, analysed and critiqued, the study of chrysotile textile workers, shows one of the highest risks of lung cancers ever observed among any asbestos-exposed population on a fibre-per-fibre basis. The increase in the relative risk from this study is 2 to 3 per cent per fibre per c.c. year. There are two additional studies of chrysotile textile workers; the Rochdale chrysotile workers which shows a risk of 0.5 to 1.5 per cent. There is a risk assessment based on a study by McDonald et al. which shows a relative risk of about 1.25 per cent increase per fibre per c.c. year. There has been a lot of discussion about the McDonald study of miners and millers; this study shows a significant excess of lung cancer but the dose response is about 30 times lower than the 2 per cent relative risk from the Dement study and about 16 times lower, if one assumes that the relative risk is 1 per cent per fibre per c.c. year of exposure. I suspect, in this study, that there is a fair amount of misclassification of exposure. Because, when you have misclassification of exposure, you are going to dampen the dose response

and be biased towards a flat dose response curve. For mesothelioma, I think the recent analysis by Landrigan and Nicholson et al., which concludes that chrysotile is only one half to one quarter as potent for causing mesothelioma as crocidolite asbestos, I think that is a reasonable analysis. There may be certainly other reasonable analysis as well. But, even if chrysotile asbestos did not cause mesothelioma, which in my opinion it does, there is still enough risk from lung cancer alone, that there should be intervention to substitute for chrysotile asbestos. There is a recent paper that was sent out after we completed our initial reports from Case and Dufresne. It was stated that I might change my opinion regarding the Dement study after I had reviewed this study, so I want to comment on that. I would add that this is an unpublished study and that the authors are much more restrained in their interpretation of the study than is Canada's submission about this study. The authors state that they can't determine to what degree the findings of fibre content in the lungs examined are representative of the entire cohort; their lung tissue fibre analysis only represents what is retained in the lungs by the time of death and there is a tremendous difference here between the miners and the millers and the textile workers between the time from cessation of exposure to death. Therefore you would expect a lot more chrysotile clearance from the textile workers' lungs. Dr. Henderson has done an analysis based on assuming various half-lives of chrysotile fibres in the lungs which I think is a reasonable analysis, which indicates that there would be much more chrysotile in the lungs of the textile workers. But also on the basis of this new report, or furthermore, if the lung cancer in the Dement cohort study of textile workers was related to amphibole exposure, one would expect more than two mesotheliomas in this cohort. So I think that is striking also. Furthermore, Dr. Dement has done an analysis in response to this paper, which he has provided to me and which I would be happy to share with the Panel. What this study shows is that, regardless of when you analyse your data, - because Green had found that only one of 39 workers hired in the 1940s or later had significant amphiboles in their lungs - these were the chrysotile textile workers - only one of 39 that were hired after 1940 had significant amphiboles in their lungs, Dement did a new analysis where he looked at the entire group of 126 lung cancers in his study and he gets the same dose response whether he looks at total employment or employees who were first employed after 1940, or who were employed before 1940 or 1950. So what it shows is the same dose response accounting for different periods of employment. So I thought that was impressive.

20. Regarding controlled use, it is my opinion that it may be theoretically possible, but is highly unlikely that chrysotile can be controlled in commerce. My point from my written submission was, that while it may be possible, in the United States alone, we have had over 4,000 violations of our asbestos standard in the last three years. In the United States there are monetary penalties that go along with these violations and yet, if we have this large amount of non-compliance in the United States in the presence of monetary penalties, and also in some cases there can be criminal penalties, then what does this bode for other countries that might not have this stringent requirement or penalties. Canada's document was criticising, I believe, that I didn't understand their controlled use programme. It seems to me that from recent articles that I have seen in countries where Canada appears to, or is importing, its chrysotile asbestos, in Morocco, Brazil and India, recent reports just came out indicating that asbestos is not controlled according to its controlled use programme. Therefore, in my opinion, the programme has little credibility to me. My point is that if it can't be handled in the United States, I suspect that it is going to be even more difficult to control its use in other countries. Regarding substitutes, I feel that the substitutes do not present the cancer risk that chrysotile asbestos does. Three have been studied experimentally, two of the substitute fibres have been negative in animal cancer studies; fibreglass has been positive. I did not mention refractory ceramic fibres because the question was not specifically asked about refractory ceramic fibres. Refractory ceramic fibres are carcinogenic in experimental animals. I definitely think that there should be various serious concerns to humans exposed, but these fibres are limited to special high heat applications and I don't believe that these fibres would be substitutes for chrysotile in most current applications of chrysotile asbestos.

21. It was commented in the last submission by Canada that I had a different opinion about the carcinogenicity of fibreglass compared to asbestos than what I had published in 1994. Looking

further at data, I feel that there is not sufficient evidence in humans that fibreglass is carcinogenic, but I think that one should presume that these glass fibres are carcinogenic to humans; that doesn't mean that it is proven, but I think that there is enough evidence that we should be concerned about that. But I don't feel that they are as potent as chrysotile asbestos. As I indicated, I recently spoke with several workers who are employees of the fibreglass manufacturing facility that showed a two-fold risk of lung cancer. Those workers explained to me that there were other known human carcinogens to which they were exposed at that facility which had not been mentioned in the report, namely, they were exposed to asbestos and to crystalline silica, along with several others which I mentioned in my report. Because of that, I feel that one cannot look at that study in terms of the fibreglass/fibre count in relation to the elevated risk of lung cancer, there is confounding from other known carcinogens or highly suspected human carcinogens in that population that are not accounted for.

22. On page 49 of the Canadian response, it states that there are three studies in which cellulose exposures have been investigated but that I did not identify them. Cellulose has not been studied for carcinogenicity in experimental animals. What I had indicated was that there are three industries where there is cellulose exposure, namely the paper industry and this study in this industry does not indicate any elevated risk of lung cancer or mesothelioma. I didn't identify the literature, there is an entire IARC monograph on the paper industry. The same with wood dust. I didn't cite any particular specific studies of workers exposed to wood dust which contains cellulose. IARC has an entire monograph on the furniture manufacturing industry and a more recent monograph on wood dust. There is no indication of any excessive risk of lung cancer or mesothelioma. Cotton dust, I didn't cite any particular study but there is a tremendous literature on workers exposed to cotton dust. The Occupational Safety and Health Administration in the United States issued a new regulation for cotton dust a number of years ago, and cancer was never an issue that was raised as a health concern; it was byssinosis from workers exposed to cotton dust. Regarding the cotton dust exposure and the byssinosis, it was never proved whether it was the cotton fibres *per se* or the contaminants that were related to the byssinosis. In any event there is no indication of lung cancer or mesothelioma from cotton dust exposure. So while I didn't cite those, there is a tremendous literature on those.

23. Finally, I would conclude by saying that, once it is known that these fibres are carcinogenic, one should not need to demonstrate their carcinogenicity in ever sector where blue-collar workers come in contact. Once you have identified the hazard, it is not convincing to say a particular study does not show an excess, it is the exposure that we are concerned about. We already know that exposure to these fibres are dangerous. This is an industrial health problem of abating the hazard, not continuing to identify the hazard in new populations that have not heretofore been studied. There have been epidemiological studies, that is, not of controlled environments like the laboratory setting, there are always errors and misdiagnosis of disease, there is incorrect recording of disease on death certificates, there is misclassification of exposure. All of these factors, particularly the misclassification of exposure, lead to flattening the dose response, so that you don't find any dose response. So I think that we have identified the hazard, and it is my opinion that since there are substitutes available for it, I would recommend a substitute for asbestos. Thank you.

Chairman

24. Thank you very much. Dr. Musk, would you like to make some introductory comments?

Dr. Musk

25. Thank you. I would like to echo my fellow panelists' gratitude for inviting me. My analysis and distillation of evidence from my own work and from the literature is that all forms of asbestos may cause disease. The main diseases being well known are: asbestosis, lung cancer, malignant mesothelioma and pleural plaques. The issues of main concern are the malignant diseases that may arise from chrysotile. The approximate relative potency of the different forms of asbestos to produce the different outcomes is summarized and tabulated in my submission. I want to stress that it is obvious that these are very ballpark estimates. I hope that we don't argue too much about the numbers themselves. The outcomes from exposure to asbestos appear to be determined by the dose of exposure, the dimensions of the particles, their durability and chemical properties – least well understood. Not all the data is consistent, particularly regarding the effects of chrysotile and mesothelioma, thresholds of exposure may exist, especially for asbestosis, but there is no direct evidence for them that I am aware of and I believe that it is unlikely that there are thresholds for carcinogens. Control of asbestos-related diseases is dependent upon control of exposure in the people who handle it from mining onwards or are otherwise exposed to it in the chain of events to ultimate or penultimate disposal. Controlled use of asbestos in production and manufacturing may be feasible. But controlled use does not seem feasible when extended to subsequent use or handling or incidental exposures. Finally, substitutes are probably safer, in my assessment, than chrysotile.

Chairman

26. Do any of the experts, having heard what their colleagues have just said, want to comment further? No. Well, we will move into the part of the meeting that gives parties and the Panel, of course, the opportunity to ask any additional questions. Before I give the floor to Canada, I think it would be helpful if delegations could limit general statements they may wish to make, if they could be quite concise. I would like to give each delegation the opportunity, should they wish, to begin with any general comment they might have. Could I ask Canada first if you would like to begin with the general comments, or whether you would like to go straight into addressing question to the experts?

Mr. Hankey (Canada)

27. I would like to thank the experts for the hard work they have done. We look forward to discussing their views with them today with the object of shedding more light on this complicated scientific question, insofar as it is relevant to the issues before the tribunal. In its response to comments by the experts, Canada referred without citation to an unpublished study by Case, Dufresne, Sebastien, and two distinguished members of our delegation, Professors A. D. and J. C. McDonald. In the response to the comments by the experts we indicated that this study had been the object of a presentation at a conference held in Maastricht this last fall. When asked by the Panel to provide a text of this study, we submitted it to the Panel's Secretary in its draft form which was found on the Internet. This Internet version was marked "Not for citation". It should be pointed out that this version of the study had yet to be seen or approved by either Dr. A. D. or Dr. J. C. McDonald. They acknowledge that there were statistical errors in this text which have been pointed out to Dr. Case both by the McDonalds and by Dr. Henderson in his Supplementary Remarks. These errors have been corrected in the version which is to be submitted for publication. Finally, I note that the statistical errors noted by Dr. Henderson in his Supplementary Comments do not detract from the essential findings of this study including that amphiboles are present in textile workers' lungs. I have one other question. Mr. Infante has just made reference to a text by Mr. Dement which I believe has not been filed before the tribunal. I wonder, you had said that it was not your intention to allow the admission of new evidence at this time. Does that apply only to the parties and not to the experts? Because we have not seen that study.

Chairman

28. Could I come back to that point later, and ask the European Communities whether they would wish to make any introductory comment.

Mr. Christoforou (European Communities)

29. Can I join you and my Canadian colleagues in thanking the four experts for indeed the hard work and the time they have taken to provide so detailed and pertinent replies to the questions we are facing here. I would request the four experts, when they reply to the questions by Canada – this is not a polemic comment – to try to identify each time what their views are. We say this because we have the feeling that in Canada's comments, sent to the Panel on 13 December, there is an underlying attempt by Canada to somehow confuse by grouping all the scientists together – saying "the four scientists" or the scientists - whereas in some of the cases probably only one of the scientists had said something. So I would appreciate it if the experts tried each time to identify what their personal views are on the specific questions. It is very important for the record to show what the views of each of the experts are and not leave with generalizations. One procedural issue, Mr. Chairman: I did not hear anything about the time that is allowed to the parties, especially I did not hear the word "equal time" to the parties. So I would assume since we are second under ... **[END OF TAPE]** ... otherwise we may end up here today with us having no time to ask questions to the experts.

Chairman

30. Thank you. To take the latter point first. I said that the Panel would make sure that we are able to do our work as efficiently as we can and I think that we will have to see how the meeting goes, but we are not yet at the point of having to allocate time to each party as we are not yet at a point when we are under any pressure for time. But we will need to see how the discussion goes but obviously, yes, we will make sure that both parties have a fair opportunity to have their points heard.

31. On the other point of evidence, we did not obviously limit the evidence that the experts might supply for the amplification of their views. And the parties today have the chance to comment on that evidence. I think that the point was in terms of the due process of the Panel's case itself that we were not, on the part of the parties, we were not in a position to be able to look at completely new issues that had not been raised before. We think we are at the point where we can begin with the questions to the experts. I would invite you as far as possible to follow the path we suggested to take the issues question by question. Obviously there may be some overlap in the questions, especially in questions 1 to 4 which are all concerning chrysotile, question 5 was concerning controlled use and question 6 concerning substitute fibres. For the good order of the meeting and to be able to monitor our progress through the issues as we go, it would be helpful if the parties can as much as possible try to address questions to the experts under each of the headings of our questions 1-6. I give the floor to Canada on issues concerning question 1. Are you ready to begin or do you want a few minutes to consult among yourselves, in the light of what the experts said in their introductory comments?

Mr. Hankey (Canada)

32. No, Sir. I am ready to begin. I just want to come back to that procedural issue I raised at the beginning, that is to say the admission of evidence. I take it that inasmuch as the experts have admitted or led new evidence either in their submissions today or for example in the extra procedural filing by Mr. Henderson a week ago, that we would have the opportunity to lead evidence that we may need to rebut or to respond to the new evidence brought forward by the experts.

Chairman

33. I would just like to make the point that we are not here to put the experts on trial.

Mr. Hankey (Canada)

34. The experts are leading evidence which is obviously material to the question put to them. Mr. Infante has just referred to a study by Dr. Dement, which he has used to rebut points that we have made. I think it important that he file that paper here so that we have access to it and that if we see fit, we be given the opportunity to file paper over the coming weeks in response thereto. That would be normal.

Chairman

35. I think that it was made clear that we have at our disposal a limited amount of time for this expert phase of the Panel process. That phase essentially concludes at the end of today. I think that rather than delay our proceedings in any further discussion of a procedural nature, it would be very helpful if we could begin straightaway on the questions themselves. So I would invite Canada to begin. What I would suggest is that we can alternate questions between Canada and the EU. Canada, would you present your question on question 1 first, and then we can follow with the EU.

Mr. Hankey (Canada)

36. I do just want to signal to you at this time that we agree to proceed with this part of the process as you suggest. But on Thursday I will be raising what we consider to be serious procedural problems with the way the expert consultations have taken place. But let's not bother with that now.

37. This question is directed to all of the experts. A majority of you have identified construction workers as being the population at greatest risk. Who do you include in the definition of construction workers? Do you include, for example, skilled workers such as electricians and plumbers?

Chairman

38. The parties are free to ask their questions either to an individual expert or to the experts as a group and in cases such as this one, where questions are being asked to the experts as a group, we will leave it to the experts themselves as to which question they wish to respond. I would just like to give the floor briefly to Mr. Christoforou.

Mr. Christoforou (European Communities)

39. I really regret having to intervene, but I would suggest – Canada is free of course to ask and to term the question the way it wishes – but I would make a second plea to avoid words like the "majority" without knowing who of the four scientists had said what. I would request Canada to identify which of the scientists had said what, words like the "majority" or "most of you", it is our suggestion that they should be avoided. We need to know who said what instead of referring to the majority of the scientists. Thank you.

Chairman

40. Thank you. Take note of that, please.

Mr. Hankey (Canada)

41. Thank you, Mr. Christoforou. I could rephrase the question if it is helpful, either to say "some of you have identified construction workers etc. etc." And those of you who wish to respond may do so. I don't insist, I am not in a position to insist, that anyone responds who doesn't think the question pertinent.

Chairman

42. I pass the floor to whoever wants to respond to that question. Mr. Hankey, would you mind repeating the question?

Mr. Hankey (Canada)

43. Some of you have identified construction workers as being the population at greatest risk. I suppose I can address the question to those of you who have done so. Perhaps you haven't all done so and perhaps the majority of you haven't done so, and perhaps we don't count so well. Who do you include in the definition of "construction workers"? Do you, for example, include skilled workers, such as electricians and plumbers?

Dr. de Klerk

44. Speaking for myself, I was talking about people in the construction industry, so that would include electricians, plumbers, carpenters, ladders, boiler makers, anyone in any form of construction. It's basically the group of workers who form the largest part of people who come down with mesothelioma. And where regulations are going to be hardest to police.

Chairman

45. Dr. Henderson was going to make a point.

Dr. Henderson

46. My inclusion amongst construction workers would include a large and disparate workforce which includes both skilled and unskilled workers involved largely in building construction and building maintenance and so forth. If one looks at mesothelioma as an index tumour for asbestos exposure and you go to the attachment I gave to my first report of the professions or workers included in the Australian Mesothelioma Register, they do include, going down them alphabetically: people who carry out maintenance on asbestos dwellings, fences, they include builders, brickworkers, builders' labourers, carpenters, joiners, construction workers, civil engineer, demolition worker, electrical engineer, electrical fitter, electrical mechanic, electrician. Going further down the list, labourer, locksmiths, machine fitters, maintenance carpenters, maintenance electricians, maintenance fitters, mechanics (they're not involved in building construction, of course, they are a different group). They do include painters, plasterers, plumbers. Together I think it adds up to a fairly large and disparate workforce which is very poorly regulated in Australia.

Chairman

47. Thank you. Any expert wishes to add anything?

Dr. Infante

48. I would agree with that. It is both skilled and unskilled in the rubric of construction workers.

Dr. Musk

49. That would fit in with my ideas. We might argue whether construction and demolition aren't opposite processes, but there is so much overlap in the sort of tasks that people in the construction industry undertake, that we could probably include demolition with construction.

Chairman

50. May I invite any further comments or issues that parties might like to raise in connection with this question? No, in that case can we turn to the European Communities for their first question or comment.

Mr. Christoforou (European Communities)

51. This question is addressed to all the scientists, in particular, to Dr. Infante and Dr. Henderson. In your reply to question 1(e) of the Panel, where you are discussing occasional interventions on asbestos, (for example Dr. Infante states 'mesothelioma has been identified from these exposure situations because it is a marker cancer related to asbestos exposure'). We would appreciate it if you could expand on this, and whether you think there are data from the mesothelioma registers which support this, and what is the part of the population which is at most risk. And therefore the question of public health concern.

Dr. Henderson

52. Dealing with this group of workers, and in particular, the occasional workers, I think that it is fair to say that the risk of mesothelioma and of lung cancer will be related to the frequency and to the cumulative exposures that these individuals sustain, because professional workers, for example professional carpenters, will be working most consistently and regularly with asbestos-containing building materials. It is they who will sustain the highest cumulative exposures, and therefore suffer the greatest risk of both mesothelioma and lung cancer. For the occasional worker, the risks will be substantially less because the cumulative exposure will be less. But in my own series of mesotheliomas in Australia, I have a number of cases of individuals who simply dwelt in asbestos cement houses and who carried out maintenance and renovation on the houses. It so happens that most of those individuals would also have sustained exposure to the amphiboles. Given the relative potency differential between the amphiboles and chrysotile, I would expect the risks of the occasional worker with pure chrysotile cement materials to be substantially less than those exposed to mixed asbestos cement materials. However, I would also point out that in Australia there are individuals who style themselves as "home handymen" and they make a career of buying dilapidated houses, often asbestos cement houses, and they live in them for a year while carrying out extensive renovations and maintenance work. They then sell these houses a year later and because they have dwelt in the house for a year, the profit that they make is not subject to taxation. These individuals call themselves "home handymen". The houses they buy are often called "handyman specials", because they require maintenance and renovation. These individuals will move through a succession of houses at yearly intervals. Now, it so happens that if you look at their cumulative exposure, they may approach the types of cumulative exposure one would expect for a professional carpenter. So I would have to say that the risks would be related to the frequency and the duration of the exposure, and its intensity, and therefore to the total cumulative exposure.

Chairman

53. Dr. Infante, is there anything that you wish to add?

Dr. Infante

54. In these situations there is not good information about exposure but rather as the scenario type of exposures. It's this intermittent exposure that you really don't know how much fibre these individuals are exposed to. But the fact that you see some mesotheliomas, it indicates that without being able to add up the cumulative dose of their fibre exposure, it indicates that it was enough in some situations to induce mesothelioma. We don't have specific information on dose-response from these operations because we don't have information directly on dose or fibre counts over time. The point I was trying to make was that if you identify mesothelioma from these types of exposure, there will also be the unidentified risk from lung cancer from those types of exposures. Lung cancer is more difficult to identify because it has got a high background level and there are other factors that relate to lung cancer in addition to asbestos. So it is difficult to identify the lung cancer cases.

Chairman

55. Professor Henderson?

Dr. Henderson

56. Could I perhaps just elaborate on Dr. Infante's reply? I agree entirely with his proposition that mesothelioma is a tumour which is an index tumour for asbestos exposure. In Australia, the data we have ... – because we collect every mesothelioma in the country, with good exposure data, and then they are entered into a Central Mesothelioma Register – we have very good data for mesothelioma. The situation changes dramatically for lung cancer. In fact, we have very poor data for lung cancer. There are indications from some countries, e.g. the United States, that about 3-5 per cent of lung cancers will have asbestos as a co-factor, usually with tobacco smoke, but the estimates range from less than 3 per cent to 20 per cent in different countries. In Australia, when we look at the Mesothelioma Register and the Dust Diseases Register for New South Wales, the data for mesothelioma are very good. But the data for lung cancer are very poor. It has been suggested that normally one sees about somewhere between one lung cancer for every mesothelioma to up to 10 lung cancers for every mesothelioma. In New South Wales, despite the adequacy of mesothelioma data, the data we have for lung cancer are very poor, so that if you look at compensated cases for lung cancer in New South Wales, we see a reversal of the lung cancer to mesothelioma ratio. So, we see ten mesotheliomas compensated for every lung cancer. The situation probably is that most of these asbestos-related lung cancers are passing unrecognized by the medical attendants, because the patient is a cigarette smoker – that is explanation enough and no further explanation is sought. And even the cases that come before the Register, a large number of them are rejected on the basis that the data on exposure don't suggest that there is sufficient exposure for the individual case to be compensated. But if you approach this on a population basis it seems that a large number of our lung cancers have a contribution from asbestos exposure, passing unnoticed by the national health authorities and regulatory authorities.

Chairman

57. Any additional comments on this point? Canada.

Mr. Hankey (Canada)

58. Dr. Henderson, I wonder if we may get back to the question that was actually asked, which was I believe about your Register study of mesotheliomas. How many of the mesotheliomas deaths do you consider to be attributable to chrysotile only?

Dr. Henderson

59. That is a very difficult question to answer. I am afraid I cannot give a precise answer because many of the individuals will have sustained mixed exposures or they have sustained exposures for which we have no precise data as to fibre type. However, if you look amongst the Australian Mesothelioma Register data, there is a figure of 58 mesotheliomas among automobile and brake mechanics whose only exposure was to brake linings, and brake blocks themselves. For decades in Australia, brake blocks and brake linings have only contained Canadian chrysotile in a bonding matrix, so there have been no amphiboles in that material for some decades.

Mr. Hankey (Canada)

60. What sort of controls did you have in place for this study?

Dr. Henderson

61. This is not a study. These are figures from the National Mesothelioma Register where the occupational histories are quite good. Again, it is one of those things that one is reliant upon the data supplied to the Register. But the increase in incidence has been worked out by comparison with the Australian census figures for the total number of automobile mechanics, including all types of mechanics, and the number of mesotheliomas occurring among them over a particular period of time. The same type of figure is also given in the NICNAS document which I submitted to the Panel as an annexure to my original report.

Mr. Hankey (Canada)

62. Thank you. Dr. de Klerk, what value would you attribute to a register study that is conducted without controls? What probative value would you consider that it has?

Dr. de Klerk

63. Mesothelioma register studies are widely used. Basically, the problem being that you cannot compare, generally, the rates with other groups if you haven't got any population basis. But I think Professor Henderson said that somebody actually looked at this in relation to the population in that occupational group. So obviously if the occupational group, you know you have the total population, you can ascribe a rate of disease in that group and you can compare that with the overall rate in the population. In terms of the proof scenario, people always put case series down at the bottom of the list but it is often in medical history that the case series come up with the sort of proof, well not the proof, but come up with the first idea as to something being a risk factor. You only have to look at all the nickel workers in Wales, the chimney sweeps and all those kind of things. They were all first observed purely through case series. So I think the case series is a very valuable epidemiological tool.

Mr. Hankey (Canada)

64. So, do I understand you to say that in terms of probative value in scientific rigour, you would place it near the bottom or at the bottom of methodologies that are used to determine relative rates of disease in one occupational group as opposed to another?

Dr. de Klerk

65. In standard epidemiology texts, they always start off by saying that the best thing for showing an effect is the randomized control trial. You can't really do a randomized control trial in this situation. So the next thing you do is a cohort study and because these are all people in disparate industries, you can't do a cohort study. Then you could do a case control study, but the exposure is

fairly rare, so it is not very good to do a case-control study. So you end up with the case series. I can see your point, but at the same time, if you have got this number of cases with only this exposure, it has got to carry a fair amount of weight in terms of if you choose to stand next to somebody blowing out dust from their brake drums, if you see what I mean?

Mr. Hankey (Canada)

66. You say you attribute a fair bit of weight, to what? What conclusions would you draw from the study?

Dr. de Klerk

67. Well there are a lot more brake mechanics getting mesothelioma, I mean the rate in brake mechanics is a lot higher than the rate in other groups of the population. Therefore one would attribute a fair amount of weight to that study.

Mr. Hankey (Canada)

68. Are you aware that there have been four case controlled studies of garage mechanics, two in the United States. (McDonald and McDonald, Teta et al.), one in Canada (Teschke) and another in Germany (Woitowitz and Rödelsperger). They have all shown no increased risk of mesothelioma for garage and brake mechanics. Do you accept these data?

Dr. de Klerk

69. If those studies are there and that is what they show.

Mr. Hankey (Canada)

70. And what would you consider to be the more scientifically rigorous methodology and which would have in a court of law the greatest probative value: the register analysis that has been done by Dr. Henderson or these kinds of case control studies?

Dr. de Klerk

71. I think that you would have to look at them on an individual basis. The problem with case control studies is that it is very easy to do a bad case control study, where you have a sort of register in place that is sort of collecting data as fully as it possibly can, one might make the point that the register might be better. At the same time, in the case control study, there is a problem with sample size: I mean to show no increase in risk is not the same as showing that there is no risk. It is just showing that the study doesn't have sufficient power to detect an increase if it is there, and I make that point somewhere else in my document, the standard case control case is bedevilled by small sample size problems. I wouldn't like to generalize too far, there may be heterogeneity in the cases in the study, there may be different work practices in the different countries. It is just that certainly in Australia, there seems to be good evidence that the brake mechanics do have an increased risk of mesothelioma.

Mr. Hankey (Canada)

72. I don't think that you got the point of my question. I am not asking you to attack the method by which Dr. Henderson conducted his study, as related to the rigour of the four case control studies on garage mechanics to which I have referred. But rather I am asking you: *grosso modo*, as a form of analysis, as a form of enquiry, which is generally considered to be the more reliable in terms of producing hard results?

Dr. de Klerk

73. Well, the case control study.

Mr. Hankey (Canada)

74. Yes, all right, good. Now, Dr. Henderson, are you aware that a proportional mortality study of mesotheliomas in England and Wales covering the period 1979-1980 and 1982 to 1990 showed no evidence, and I repeat no evidence, of an increased risk of a mesothelioma in motor vehicles? That is the study by Hodgson et al.

Dr. Henderson

75. Yes, I am aware of the studies that have shown negative findings with no detectable increase in risk. I would simply amplify the comments that Dr. de Klerk has already made. (And I don't regard myself as an expert epidemiologist). I would simply say that if you are looking at a small effect in a small population, you may not detect an effect. When you deal with national populations, yes, the quality of the information and the controls may diminish, but you are not looking at the same issue in many respects, and we are not looking here – when we look at the Australian incidence of mesothelioma among automobile mechanics – to provide proof in a court of law. We are looking for an indication of an effect that might be used for the formulation of national occupational health and safety policy, which I think is an entirely different exercise. But yes, I am aware of those negative studies and I have to counterbalance those with the indications that we have – not only from my own looking at the Register – but from the National Occupational Health and Safety Commission looking at the Register, to say that there is an indication of an increased frequency of mesothelioma amongst brake mechanics in Australia and that the increase is in the order of 1 to 2 per cent increase per year, which is roughly comparable to the overall growth of mesotheliomas in the Australian population. But when we look at that type of effect, we need to ask what group are we going to compare them against. And if you look at the background rate of so-called spontaneous mesothelioma of 1 to 2 cases per million of the population per year, we do have an indication of an increased effect in making that comparison.

Mr. Hankey (Canada)

76. I just want to point out that, although you pointed out a difference in the kind of evidence that you seem to think is appropriate for setting policy and the kind that is appropriate in the court of law, but I think that, in both circumstances you would admit that what is important is the probative value of the evidence studied. In both cases, people are trying to draw conclusions to complex and difficult questions. But I just want to conclude by saying, of the four studies that I cited earlier in my question to Dr. de Klerk, these studies of garage mechanics by McDonald and McDonald, Teta et al., and Teschke, Weitowitz and Rödelsperger, of these four studies, one of them was a strictly controlled series of mesothelioma study by McDonald in 1980. The mesothelioma study considered all 344 cases of mesothelioma reported by pathologists throughout North America during the reference period. These were compared with 344 strictly matched controls. Of these 344 cases, 12 cases had been garage workers. This perfectly matched the 12 controls who were garage workers, indicating that the rate of mesothelioma among garage workers is the same as in the general population. Do you accept these data?

Dr. Henderson

77. Well, you are going into some highly specific details, amongst thousands and thousands of pages of information that I have tried to digest in preparation for this meeting. But yes, I agree with the general conclusions, and the simple fact that I would draw attention to is that with so many studies on asbestos-related diseases, one is dealing with contradictory sets of data. The question arises as to what weightings one places upon one set of data as opposed to another and what significance one gives to a particular set of data when trying to set national occupational health and safety policy.

[Coffee break]

Chairman

78. ... **[Not recorded]** Dr. Infante said that he wished to intervene on the previous question we were discussing.

Dr. Infante

79. My comment relates to which is a better study, a case control study or using the mesothelioma registry in Australia to estimate the risk of mesothelioma. In a case control study you are sampling your controls, hoping that they represent the universe. The extent to which they do or not, you don't know, but you use certain matching criteria and hope that they do. The extent to which they do, may affect your findings. On the other hand, looking at the mesothelioma registry for the entire country of Australia, you don't need to sample the universe, because the denominator is already the universe. So you don't have any sampling error that you have to be concerned about. Then Dr. Henderson estimated then what the incidence of mesothelioma would be in the general population of Australia, based on the cases that were reported to the registry. In my opinion, he overestimated the denominator, by making certain assumptions. But nevertheless, he had quite a high incidence of mesothelioma per million population from his analysis. So, in my opinion, in this particular case, I feel that the registry is a very good source and in fact may be superior to using a case control study where you are trying to estimate what the incidence is and the relative risk compared to the universe which you are presuming from your controls. And also it is like we are talking about asbestos exposure in mesothelioma here, it's not that we are looking for some new disease related to asbestos. It is a disease that has already been indicated as being associated with asbestos. So, I feel that using the registry, where we have the entire data based on the entire country, may in fact be preferable to a case control study where you are trying to sample or estimate what the frequency is and the comparison population.

Chairman

80. Thank you. Canada wishes to further comment on this question?

Mr. Hankey (Canada)

81. I would like to ask Professor Corbett McDonald, who conducted the largest cohort of asbestos workers for the longest period of time of any study that has ever been conducted, I would like him to comment on the relative merits of the various forms of studies that are being discussed here.

Dr. C. McDonald (Canada)

82. I suppose my question would be to Dr. Infante, as an epidemiologist, to ask whether you really are in a controlled study trying to sample the universe. Are you not trying to sample the part of the universe that is comparable to your cases? That is people from the same place, of the same age and sex, with questions about their occupation which are comparable and which are analysed blind,

without any preconceived idea of an association. Is not that the object of a properly designed case control study? And secondly, is it not true that in a register study, of which I have done many, the issue of asking information about occupations is almost certainly biased by concepts of what you believe or what the people believe to be the truth? Is not in Australia, the biggest producer at one time of crocidolite, liable to get questions which suggest that lots of occupations may be due to mesothelioma? Is not the object of an objective, scientific epidemiological study to remove sources of sampling bias and information bias?

Dr. Infante

83. Yes, when you are sampling, you are trying to eliminate the bias in your study design and the extent to which you do that depends on the success of selecting your controls. If you do a study and you are looking at mesothelioma in North America then you are really trying to sample, as you said, the individuals that match closely the cases, and the cases are coming from the entire North America. So in my opinion, you are still trying to estimate the universe in that particular type of a case control study. In terms of bias from a registry source, you can have bias from a case control study, you can have bias from the registry. It depends on how the questions are asked.

Chairman

84. Canada?

Mr. Hankey (Canada)

85. I have one final point. It is to Dr. Henderson and Dr. Infante and it is simply this. If we have a registry study, such as that conducted by Dr. Henderson and we have ..., I forget what number of garage mechanics he found, but let's say that it was 50, how does he determine, without controls, whether that is a large number or a small number, or a number that is more or less in proportion to what you would find in the general population. What is the significance of that number? What does it tell us if there are 50 garage mechanics, if among mesothelioma victims there are 50 who are garage mechanics?

Dr. Henderson

86. The figure in the 1999 Register was 58 mesotheliomas in individuals designated as brake mechanics for which there was only exposure to asbestos derived from brake lining and brake block materials. This was a group separate from the individuals who had multiple other exposures to asbestos. In this respect, most of the history data for the Australian Mesothelioma Register, are fairly accurate, as much as one can ever achieve with a population-based set of statistics, in that the occupational histories in Western Australia and New South Wales are taken by professionals that are asking histories, for example, the New South Wales Dust Diseases Board. We don't know exactly how many brake mechanics there are in Australia, but the 1996 Australian census figures came up with a figure of approximately 87,000 male automobile mechanics, that is mechanics of all descriptions [among whom brake mechanics would constitute a smaller] ... [END OF TAPE] ... number of individuals; what I then did in calculating the statistics was to round it off at 100,000 or 200,000 to take into account the number of mechanics who might have left the industry and retired. I compared that simply with the estimated background rate of mesothelioma for the general population as being one to two cases per million per year. Using the upper figure of two cases per million per year, I still came up with an increased number of mesotheliomas above what I would have expected for purely spontaneous or background mesotheliomas. Now, this was no systematic study on my part, it was a set of calculations. But the interesting thing was that the figure I came up with was roughly comparable to the figures given from the National Occupational Health and Safety Commission in Australia, which had found an increased incidence of mesotheliomas, and that the rate of increase is roughly proportional to the rate of increase of mesotheliomas amongst the rest of the population. I

don't pretend that the statistics are anything more than that, but to me they are an indicator in terms of approaching a problem at a national occupational health and safety level of indicating a possible effect and therefore the need for a cautious and prudent approach.

Mr. Hankey (Canada)

87. Sir, it sounds to me like you are saying that your study, the probative value of your study is your intuition as to what the percentage of garage mechanics is in Australia because the kind of calculations you have just suggested do not strike me as the kind of scientific rigour that would be required in order to produce a study which would have probative value in any court of law.

88. But I have another question, Dr. Henderson, well this is for Dr. Henderson, yes, again. Are you aware that in about 1990, Dr. Woitowitz and Dr. Rödelsperger published a short report noting that they had collected a number of cases of mesothelioma in men who had been automotive mechanics and they were concerned that, because these men had low exposure to chrysotile during brake work that their mesotheliomas would indicate that low exposures to chrysotile were causing mesothelioma. In 1994, they reported that they had carried out a case-control study with two different types of controls and that both showed that there was no association between work as a mechanic or brake repair work and mesothelioma. Now, in view of these findings and the overwhelming evidence for no association between friction products and mesothelioma, wouldn't you agree that no conclusion should be drawn from the Australian Registry regarding the relationship between mesothelioma and brake repair work as no controlled study has been done?

Dr. Henderson

89. Well, I am no epidemiologist, but I would not draw the conclusion that no conclusion can be drawn from the Register figures. We have figures for a number of different occupations, from the Register, who have mesothelioma and the fact that we don't have precise data on all of those other occupations represented on the Register, does not mean that we can draw no conclusions from them. I am aware of the study carried out by Dr. Woitowitz, (in fact, if you read my report initially submitted to the WTO, you'll find that I did discuss that report), but I will also point out that at the conclusion of their revised report, Dr. Woitowitz pointed out that some of their cases had in fact sustained amphibole exposure as well, and that, taking that into account, they could not identify an excess risk. But they also indicated that their study had low power to detect small risks and they indicated that, if the risk of mesothelioma was small, their study would not have detected it. Now, all I am saying is that when we are dealing with a national population of 18 or 19 million individuals and we look at all of the mechanics amongst that population, we are dealing with a larger population, and although it may not have the precise rigour of a case-control analysis, the figures nonetheless do indicate that there is an increased risk of mesothelioma among brake mechanics who are exposed only to chrysotile asbestos from grinding brake blocks. The other point that I would make is – when I looked at the figure for the mechanics across Australia and I compared it to the figures also given for North America – the figures were that the number of estimated brake mechanics for the two populations were surprisingly similar. So one of the points that I would emphasize is that if my figures are inaccurate – and they may well be inaccurate – they are inaccurate on the side of conservatism, in that I have overestimated the total number of brake mechanics and, therefore, perhaps underestimated the effect.

Chairman

90. I think we are keen to move on through the list of questions and I have just consulted briefly with my colleagues, and we feel that on this present question plus set of supplementary questions, we have gained a great deal of clarification of the experts' views. So I would invite Canada maybe to make one last comment or raise one last issue concerning this group of questions before we move on.

Thank you. Before that, we invite Mr. Christoforou to take the floor who has been seeking to make a comment for some time.

Mr. Christoforou (European Communities)

91. Thank you Mr. Chairman. I would like to ask a question on this because I'm afraid that the way we proceed – it is up to you Mr. Chairman - but it will take us quite the entire day probably. We will not finish, and on this point we run the risk of trying to see an individual tree and would lose the entire picture of the entire wood in this case. So I would like to come back to this question with one and then I still have another question to ask, as I pointed out.

Chairman

92. I am sorry I didn't quite understand what precisely you wanted to come back on.

Mr. Christoforou (European Communities)

93. I want to ask a subquestion on this point and then ask the other question I have. I announced two questions on this first point.

Chairman

94. OK. I did invite Mr. Hankey to make a final comment on this particular set of issues. Please go ahead.

Mr. Hankey (Canada)

95. My question is: taking account of the definition of construction workers, of those categories of workers that you each identified as construction workers in response to my first question, over a one-year period, is a construction worker at greater risk from exposure to low-density asbestos products in place, or from exposure to products at issue in this case, that is, high-density chrysotile-cement or friction products? That question is to each of the experts, thank you.

Chairman

96. Would you mind repeating the question, I think the experts are not clear exactly what the question was?

Mr. Hankey (Canada)

97. Of course, Sir. I said that, taking into account the definitions you gave earlier, or rather the list of workers, the universe of workers, that you consider to fall under the general rubric "construction workers", over a one-year period, is a construction worker at greater risk from exposure to low-density in place asbestos products or from exposure to the products at issue in this case, that is to say high-density chrysotile-cement or friction products?

Dr. Henderson

98. The question is a little bit like asking how long is a piece of string. It depends on so many different variables that the answer will vary according to those variables. It depends on what the worker is doing with low-density asbestos-containing insulation materials, how often he or she is doing it, the frequency with which this is happening and so forth. I would believe that for an individual who works consistently with low-density friable insulation materials, either applying them or removing them, the exposure levels are likely to be consistently high and that person would be at

higher risk for both mesothelioma and lung cancer. When one is dealing with a high-density product such as asbestos-cement, it is very difficult to make direct comparisons. But if you look at individuals cutting asbestos-cement building products with a power saw for example, that can generate very high airborne fibre concentrations and again the effect in terms of mesothelioma and lung cancer induction will depend on the levels, the frequency and duration of the exposures and therefore the total cumulative dose. But even if one takes into account the fact that, or one concludes that, the worker dealing consistently with friable insulation materials is at greater risk, the point that I would make is that in Australia, most of the building construction workers we come across give a history of consistently working with high-density asbestos-cement building products. Although their risks might be less than the corresponding insulation worker, there are many more such individuals engaged in that type of activity, manipulating high-density products, and therefore a lower risk needs to be multiplied against a greater number of workers, so that the total effect we see in terms of mesothelioma incidence is greatest for example, among carpenters in the Australian building industry, who consistently cut high-density asbestos-cement building products.

Chairman

99. Thank you. I would now like to give Mr. Christoforou the opportunity to make his comment and also to ask the second aspect of the original question.

Mr. Christoforou (European Communities)

100. Yes, Mr. Chairman, thank you. Before I ask the question of clarification. Canada says the products in this case are high-density cement asbestos products, but that is not the case. Here, Canada exports chrysotile, period. We are not dealing here only with asbestos-cement containing products. I don't see what is this limitation referred to by Canada. Canada exports chrysotile. A substantial part of it may go to the production of asbestos-cement containing products, but this is not the only use, so we cannot really reduce the entire issue to this product and try to probably confuse everybody around this table. My question, or rather subquestion, I wanted to ask to the previous issue we have been discussing, was the following: I guess everybody agrees - and the scientists here have already said it so - international institutions like the International Agency for Research of Cancer, have since a long time classified all forms of asbestos, including chrysotile, as a proven human carcinogen. I guess there are good scientific reasons before an international institution does so. The scientists here have all agreed on this. Now, we also - given the previous definition of the large, wide category of skilled and unskilled workers which are involved in the everyday handling in their jobs of these substances, and also outside non-skilled, non-workers, like the handyman-type situation, which we also discussed in the papers - given this entire category of people that come in contact with chrysotile, how reasonable it is, or does one really think one can explain only by the data referred to by Canada on brake mechanics, that we can cast doubt on the evidence we hear from so many other sources? Mr. Henderson has referred to inputs from a number of countries in his reply, first set of replies. I refer to pages 35 and 37 of his report on inputs from Russia, the former German Democratic Republic, Italy, from China, and so on, which involved only inputs of chrysotile exclusively, nearly exclusively.¹ And we also know that France has been importing over the last 50 years, exclusively chrysotile, 95 per cent or even more. And still we see so many cases, more than 1,000 cases per year in France of mesothelioma lung cancer cases. Do you really think it is possible to attribute all these cases of asbestos-related diseases which we see by the small percentage, infinite percentage of other types of asbestos like amphiboles, crocidolite or everything else in explaining these cases we see. Can we really use the example of [...], if there is any doubt - we think that there is not - from the brake mechanics to question the entire evidence which has lead international institutions to classify all forms of asbestos as a proven human carcinogen? It is addressed to all scientists, in particular, Dr. Henderson. Thank you.

¹See Section V. C. 1(b) of this Report.

Dr. Henderson

101. Well, I would be in broad agreement. I think the argument would be made that most of the mesotheliomas – and some would argue that the lung cancers that we see – are not so much due to the chrysotile, but due to co-existent amphiboles in place and encountered by building construction workers. Certainly most of the mesotheliomas, but not all, that I see occur among workers who have had a history of mixed exposure to asbestos cement building products that contained chrysotile and varying amounts of amosite and crocidolite, or both at different times. However, as I have indicated, I have seen mesotheliomas among brake mechanics who only had exposure to chrysotile. So I think that this becomes an argument, as to whether one says that the chrysotile has no effect whatsoever – and that all the effects we are seeing are due to the amphibole content – or that one is looking at a mixed response to amphiboles plus the biological effects of chrysotile. I suppose that one of the concerns I have about the continued use of chrysotile, particularly in situations where it cannot be controlled, is that many of the workers who will be handling that type of material may have a pre-existent amphibole and chrysotile content in their lung tissue and we have few, if any data, on the additive or multiplicative superimpositional effect of extra chrysotile exposure on top of a pre-existing amphibole burden. Although I can't quantify the effect, one suspects that it would not be a negative effect and that it would contribute both to mesothelioma and lung cancer incidence. But perhaps the others might prefer to elaborate upon that.

Dr. Infante

102. Yes, I think if I understood your question, how I interpreted your question was, that if you have an individual who is diagnosed with mesothelioma and they have been exposed to amphibole and chrysotile, can you dismiss the component of the chrysotile exposure as contributing to that mesothelioma? My answer to that question is no. We know that chrysotile is capable of inducing mesothelioma, so just because individuals have mixed exposure to amphiboles and to chrysotile, you can't exclude that individual's chrysotile exposure as contributing to the development of mesothelioma.

Chairman

103. Would either Dr. de Klerk or Dr. Musk like to add anything to what has just been said? Canada, please.

Mr. Hankey (Canada)

104. Sir, I have a follow-up question to that question. This is directed to Dr. Infante. Dr. Infante, can you identify for us any controlled studies of cement or friction product workers showing that the risk of lung cancer to chrysotile cement or friction product workers is as great as the risk of lung cancer to amphibole workers?

Dr. Infante

105. Yes, the 1987 study by Hughes et al. They analyse their data by workers exposed to chrysotile only versus workers exposed to chrysotile and crocidolite. The dose response for lung cancer in that study is similar.

Mr. Hankey (Canada)

106. Could I ask Professor McDonald to comment on that please?

Dr. C. McDonald (Canada)

107. Dr. Infante, I haven't got the paper of Hughes in front of me, but I am very familiar with it, and in fact are you correct? I am fairly certain that the slope was appreciably higher in the factory which had the amphibole workers. Moreover, they had more mesotheliomas.

Dr. Infante

108. I was answering the question related to lung cancer and I believe that if you look at table 10 in that report, they did an analysis, for lung cancer, looking at individuals exposed to chrysotile only and then they did an analysis looking at individuals manufacturing cement products exposed to chrysotile plus crocidolite, and the dose response is similar for lung cancer.

Mr. Christoforou (European Communities)

109. Mr. Chairman, on this question, we know the Environmental Health Criteria 203, which has been cited by Dr. Henderson with his reply to question 1(e) on page 59, (he quotes a long passage from the WHO Environmental Health Criteria Report, it is already filed with the Panel). I interpret this citation as Dr. Henderson agrees with this study which is cited there, where even in Canada there was a study, where exposure to chrysotile and amphibole was separated and there was exposure to several types of situations. The study here has identified a double increase almost 2 per cent increase of chrysotile, of mesothelioma and lung cancer. Would you think that this is one of the studies which is searched after and Canada would like you to point out because it is used already by the Environmental Health Criteria Report?

Dr. Henderson

110. Well yes. I am familiar with that passage. I took it from the WHO book, Environmental Health Criteria 203, and as far as I can see, that's an accurate quotation. I really cannot elaborate further.

Chairman

111. Canada, please?

Mr. Hankey (Canada)

112. I would like to come back to that later Sir, because the name of the study wasn't identified. But I just want to ask a follow-up to Dr. Infante. Dr. Infante, isn't it the case that the Hughes study contained large numbers of temporary workers and that, when those temporary workers were abstracted out of the study and the figures were calculated only for the permanent workers, that permanent workers exposed only to amphiboles had 25 times more cancers than permanent workers exposed only to chrysotile at the same dose.

Dr. Infante

113. Are you referring to lung cancer or are you referring to mesotheliomas?

Mr. Hankey (Canada)

114. I meant to say lung cancer.

Dr. Infante

115. I can't recall the particulars of that study right now, but I do recall table 10, like I said, which showed a similar dose response. If your question to me is that, well, if they then removed the short-term workers, were the results different? I don't recall the data that well to answer that question at the moment. But the authors in the study pointed out that the dose response was similar for the two groups as I had mentioned earlier. And besides if there is some difference because of short-term workers in a study and if in a particular study short-term workers demonstrate an excess of cancer than other workers, or even a greater risk than other workers, you have to find out in that study what the particulars are among those short-term workers. Were they exposed to – do they have higher levels of exposure? You have to explore it. Quite often, short-term workers have the dirtiest jobs and so they have the highest levels of exposure, but I can't recall the particulars from the Hughes study.

Mr. Hankey (Canada)

116. We have them here. I'd like Dr. McDonald to read them into the record and we will, now that the study has been cited, if it is not already annexed, we will annex it. Thank you.

Dr. C. McDonald (Canada)

117. I have the slopes published, the exposure response slopes for lung cancer from this study. And, in plant 1 which was the one of chrysotile only, the slope of risk per fibre millilitre year was 0.0003. In the plant 2, which also entailed exposure to amphiboles, the slope was 0.0076. On the point of short-term workers, again, I am sure that Dr. Infante, as an experienced epidemiologist, will be aware that virtually every cohort study of any material shows high risk of lung cancer in short-term workers. Sorry, do I make it clear? A higher risk of lung cancer in short-term workers, whatever the material.

Chairman

118. Thank you. Is there any further comment from the experts on this point? If not, by my recollection, all that previous discussion was dealing with the two issues raised by the European Commission, so if we could return to Canada to ask its next question.

Mr. Hankey (Canada)

119. I'd now like to turn to asbestos products, I'm sorry, chrysotile-cement products because, as we know, most Canadian chrysotile is exported to France and to other places, the great majority of it is used for chrysotile-cement products. Gentlemen, Canada wishes you to consider available scientific and properly controlled evidence on the probable risks associated with the manufacture and use of chrysotile cement products. For chrysotile-cement products, we know of four cohorts: Thomas, (1982); Ohlson, (1985); Gardner, (1986) and Hughes, (1986). In total 6,843 men were studied with 1,432 deaths. 118 of the deaths were from lung cancer, that's an SMR of greater than one, that is to say the mortality rate is greater than one relative to the general population. I'm sorry, mortality rate less than one, did I say greater? So the mortality rates overall were less than those in the general population from lung cancer. This means that overall there were fewer deaths from lung cancer than expected in the general population and these are cohort studies of 6,843 men. I would like you to comment on those deaths, Sir. This question is addressed to all four, but perhaps I might address it first to Dr. Musk.

Dr. Musk

120. I'm afraid I'd need those studies in front of me to address that. I'm not familiar enough with them, although I have read them.

Dr. de Klerk

121. I think, because we are looking at asbestos-cement and, therefore, because Canada is saying that most of the products that they want to export to the European Union, presumably are used in asbestos cement, that therefore one should ignore all the other evidence about chrysotile apart from that from asbestos-cement workers, I think that it is a bit of a ..., I can't think of the word, but anyway I think you know what I mean.

Mr. Hankey (Canada)

122. I don't know what you mean. I would be grateful if you would elaborate on that.

Dr. de Klerk

123. Well it's a ..., I can't think of the word, I will explain what I mean. It's, you are sort of ignoring a lot of the fact that chrysotile will be completely different in its actions and effects; because it's in a cement product, ignores the facts about the fibre clouds produced by asbestos-cement products and the evidence from other forms of use of chrysotile. You know, even when you add these four studies together, they are in fact quite small, if you look at the numbers of deaths and to show that they show no effect is, I doubt very much whether you could actually rule out what could be quite an appreciable effect. So I think that you have to look at all the studies about chrysotile together, rather than just concentrating on asbestos-cement products. I think it is a bit of a ... it will come to me.

Chairman

124. We will let Dr. de Klerk come back on that when he finds the exact word. Thank you.

Mr. Hankey (Canada)

125. I have a follow-up question to Dr. de Klerk, but would you like Sir, that the other Doctors ...

Chairman

126. Maybe give the other experts an opportunity to speak first.

Dr. Infante

127. I am looking at the summary in the document 203 of the studies of asbestos-cement production. As I had mentioned earlier, we already know the toxicity of chrysotile-asbestos and the toxicity of these fibres does not have to be demonstrated in every occupational situation where it occurs. What we need to do now, is to control the hazard that's recognized. Having said that, when you look at the four studies, the numbers of the largest study, the Hughes study for chrysotile only demonstrates a significant excess of lung cancer. The Hughes study of chrysotile had included crocidolite and amosite exposure, there is a 17 per cent excess, that's not statistically significant. The Gardner study, the SMR is 92, that's not significant elevation or deficit. In the Hogstedt and Ohlson study, there is a 58 per cent excess and that's not statistically significant, and in the Thomas study it isn't. But we already know the hazards of these fibres and, also, if you look at the upper 95 per cent confidence limit, some of these studies were only 95 per cent confident that the risk, for example in

the Ohlson study is not as high as three-fold for lung cancer. So I think that you have to look at not only the SMRs, but the confidence intervals around these studies.

Chairman

128. Professor Henderson, would you like to add anything on this point?

Dr. Henderson

129. I couldn't add anything unless I have the particular reference in front of me. It is part of a large volume of material and I can't remember the precise details. In general, though, I would point out that certainly in the manufacture of high-density chrysotile products, at least in Australia, where it is almost a totally closed operation and the airborne fibre concentrations are extremely low with a predictably low risk for that particular cohort. My major concerns about the use of these products is in the end-users who manipulate, saw, drill, rasp, grind or otherwise handle these materials and that one knows that some of the fibres released from these operations will produce elevated airborne concentrations of fibres which are in the dimension range known to be associated with carcinogenicity, even though in some circumstances it might be a relatively small proportion of the total fibres released.

Chairman

130. Thank you. Canada, please.

Mr. Hankey (Canada)

131. I think we have really gotten to the heart of the matter, because it is incontestable that in these four studies - which are the only cohort studies of persons working with chrysotile cement, the only control studies - they all show together, collectively, that there are fewer deaths from lung cancer than expected in the general population. I don't think those data, Sir, can be swept under the carpet. Dr. de Klerk, I think really does identify what is the issue here. He says that we should look at data for other industries and apply it to the cement industry or the friction industry, because asbestos is a known carcinogen. So it seems to me that Dr. de Klerk proposes that we compare apples with oranges. Now, in my business, Sir, as a lawyer, when we deal with evidence it is always a requirement that we compare like to like. There are many, many rules of jurisprudence that require that. So I would like to ask, now, this question generally because it is on the very same point to each of the experts who wish to take it up: given that each chrysotile industry sector has its own particularities, that is to say, wet or dry processes, open door and closed processes, different fibre lengths and the possibility for oil treatments, doesn't it make sense to base risk assessments as much as possible in one given sector on the particular experiences of the workers in that sector and not in workers in a completely different sector?

Dr. de Klerk

132. Well, it would if there were data available. But I think, as Professor Henderson has pointed out and we all agreed on in our reports, I think the main risks that people are worried about are in the downstream user, the builder, the construction workers etc., and there aren't any data for those. So what one does is extrapolate from other studies where fibres of the relevant size, shape and density are available so that, as Professor Henderson said, the asbestos cement industry itself has been well controlled for a long time. The worry is not the asbestos-cement industry, it is the asbestos-cement user, I think.

Chairman

133. Does any other expert wish to comment?

Dr. Henderson

134. Really I can't elaborate beyond what my colleague, Dr. de Klerk, has said. One of the problems we are dealing with trying to assess the risks to different groups is that we're faced sometimes with conflicting or contradictory data for areas for which we have no direct observational data. Therefore we need to proceed in an area where there is some uncertainty as to the exact risks for a particular population. This is one of the reasons why one tends to use extrapolation models as Dr. de Klerk has said, and also use other investigations. For example, the South Carolina chrysotile-textile workers [are used] almost as a worst-case scenario in order to formulate prudent approaches to population safety.

Mr. Hankey (Canada)

135. I'm still following up on this question. This question I could direct to both Dr. Henderson and Dr. Infante because Dr. Infante is obviously very keen on Charleston, as am I, it is a wonderful city. My question is, if indeed there are no applicable studies to the use of asbestos cement in construction, then evidently we have to find a surrogate study, something that is closest to it and it seems to us, and that is why we put the question, that the other sector where asbestos cement is being handled and being used, that is to say in its manufacture, that that would constitute the best surrogate. The results of those studies incontestably demonstrate that there is no increased risk of mesothelioma or lung cancer, there is no dispute about that. So instead, the surrogate that Dr. Infante takes us to, and I gather Dr. Henderson, is the textile industry in Charleston. Now, I must say that it is a little difficult for me to figure out exactly what the relevance is of textile workers to asbestos cement workers. First of all, Canadian asbestos is not used, cannot be used these days in the manufacture of textiles, because nowhere do we know are these textiles being now manufactured. Certainly not in the European Union or in North America. It is our view that the Charleston data are very unreliable for a number of reasons. Some suggest that data from the Charleston textile cohort is relevant to this proceeding. Unlike asbestos cement in friction material processes, friction material production processes, the processes used in producing textiles differ enormously from those used to produce chrysotile cement and friction products. For example, the Charleston textile cohort was exposed to crocidolite and amosite amphiboles. Chrysotile during carding, a dry process in which the fibres are split and torn apart into numerous fibrils without substantial controls. (I know people who have visited that factory and the stuff was spinning around and it was hanging from the ceiling like cobwebs) This is totally unlike the conditions in a chrysotile-cement factory, or on a construction site, using chrysotile-cement. Finally, carcinogenic oil was sprayed on the fibres at the start of the production processes and it is doubtful that a valuable allowance can be made for oil fibre or oil-smoking interactions. Given these quite substantial and different exposures, why do you think it makes sense to extrapolate from textile cohorts to asbestos cement and friction product cohorts?

Chairman

136. Thank you. I guess the question was addressed to Dr. Infante and to Professor Henderson, so I will give them both the opportunity to respond to that. Then Mr. Christoforou also wanted to make a comment.

Dr. Infante

137. Thank you very much. First of all, in your question, you had an incorrect factual statement. That is if you look at asbestos cement production, the Hughes study, which shows a statistically significant excess, furthermore demonstrates a dose response from exposure to chrysotile-cement

production only and lung cancer. In fact the potency estimate is 0.7 per cent which is just a little bit less than the 1 per cent estimate per fibre per c. c. year which has been identified in two other studies of workers exposed to chrysotile textiles and a little bit lower than the estimate from the Dement study. But there is really not a great deal of difference in my opinion, between 0.7, 1 or 2 per cent. So in fact you do have dose response and when you demonstrate dose response in an epidemiological study, that is a very powerful tool. So there is evidence, even though some of the other studies don't show an excess of risk, they didn't find it for whatever reason, but I would submit that the Hughes study does demonstrate an excess and does demonstrate dose response. In terms of the abuse of the Dement study to estimate risk, it's the study that has been the most thoroughly evaluated, the exposure estimates in that study, in my opinion, are superior to any other study because they took simultaneous samples doing particle counts and counting fibres. They used different correction factors depending on which operation was being used in that study. So the Dement study is very good in terms of characterising exposure. There was some amphibole exposure to that cohort, but when you bring in some amphibole exposure to a cohort for a certain period of time and then it is no longer there, that in fact may dampen your dose response, when you are looking at fibre exposure, if your lung cancer would only be related to the amphibole. So, and furthermore, Dement has done analyses, which I had mentioned earlier, where he identified his cohort, depending on whether it was the entire group, his case control analysis for a dose response, or people who began after 1940, or began before 1940 or 1950 and he has the same dose response. So, in my opinion, that is a very strong study and it should be used to estimate risk of lung cancer from exposure to chrysotile asbestos. And it is not dissimilar from the dose response from the Rochdale chrysotile workers or the Pennsylvania plant in the US.

Chairman

138. Thank you. Professor Henderson, please.

Dr. Henderson

139. I basically agree with the comments made by my colleague Dr. Infante. Yes, on the face of it, the South Carolina, the Charleston chrysotile-textile workers appear to be different from asbestos-cement manufacture. I think the differences are partly explicable by the fact that, as I have said, in Australia asbestos-cement manufacture is a completely closed operation so that there are very low airborne fibre concentrations. But one knows that for downstream users, the operations carried out on those high-density products will produce elevated levels of airborne respirable asbestos fibres. Some of those fibres will have the dimensions that are known to be associated with carcinogenicity.

140. The point about the asbestos textile industry - and I agree with Dr. Infante that this is one study which has been regarded as a classical study and a very rigorous one in its methodology - is that for white males in the Charleston factory, there was a greater than two-fold increase in the standardized mortality ratio for lung cancer at quite low airborne fibre concentrations. That was quite different from the Quebec chrysotile miners and millers. But for many other studies we don't have direct observational data and one needs to try and take into account the fact that some of the downstream uses of asbestos-cement products may generate airborne fibre concentrations which at a cumulative inhaled dose may approach the levels of carcinogenic fibres that have been reported for the Charleston cohort. It might be argued that we don't know that the response from those workers will be the same. But equally we don't know that it will not be the same, because we don't have any data. In relation to the Charleston cohort, it was claimed that these workers had crocidolite and amosite in their lung tissue. I guess some of them did, but one of the problems about this and particularly the Case-study, is that those fibre concentrations were not linked to lung cancer as an outcome. It was simply a study done on this particular group of workers and in fact, when you look at the lung cancers that were studied in this group, and particularly the lung cancers amongst the Quebec chrysotile workers, there are substantial differences which indicate that the two groups simply are not comparable. For example, the interval following cessation of exposure until death, when the fibre

burden analysis was carried out. But there is another factor which needs to be taken in to account, is that it was argued that the Charleston workers had [commercial amphiboles, crocidolite and amosite, in their lung tissues, but if one looks at the total amphibole content]... [END OF TAPE] ... that is tremolite plus amosite plus crocidolite, it was higher for the Quebec group. So that if the lung cancer effect is related to amphiboles, why aren't there more lung cancers and a higher lung cancer risk and mortality rate amongst the Quebec workers? When you look through the evidence, I don't think that this idea of commercial amphiboles being present withstands serious analysis. The other point that was raised was that the Charleston workers may have used carcinogenic oils. Again, I think that there is no evidence for this proposition. All of the reasons for the difference between the Quebec chrysotile workers and the Charleston workers have been explored and, at the moment, there is no convincing explanation for the many-fold difference in lung cancer risk between the two groups. When there is no obvious explanation, my policy is to proceed on a approach of caution, to make sure that people are not exposed to a substantial lung cancer risk. The only other thing that I would say is that it was said that we can't use the Charleston chrysotile workers as a paradigm for lung cancer risk assessment. But this is exactly what Drs. Case and others said in the Abstract that they submitted to the Maastricht meeting. In the Abstract they said that risk assessment for asbestos exposure is based on lung cancer risk for textile workers rather than the miners and millers.

Chairman

141. Thank you. Mr. Christoforou asked for the floor a while ago. Would you like to make a comment now?

Mr. Christoforou (European Communities)

142. In terms of the population that is potentially at risk, as we have identified the people we are concerned, especially the case was of France, when it adopted the Decree in question. You have identified a large segment of the population, both skilled and unskilled and you have said that all these people are at risk. Canada is asking the question and trying to limit the issue on high-density chrysotile-containing cement products and their manufacture. But they export chrysotile as a product. Now, if I may ask, especially Dr. Infante for your experience in the regulatory approach to these questions: do you think it is reasonable, knowing the potential large segment of the population that deals with not only the manufacturing but subsequently during the lifetime of the product that remains in the place, is it reasonable, knowing the evidence we have, to take measures that prohibit the use of asbestos rather than leave it and allow it to be imported and be faced with all its potential effects on all these categories of people. Thank you.

Chairman

143. I'll give the floor to Canada next, if you want to follow up any more specific aspects on the responses just given, and then invite the experts to respond to the point made by Mr. Christoforou.

Mr. Hankey (Canada)

144. I would like Dr. Henderson to recite again, because I didn't perfectly understand, why he feels that the Charleston textile study is a better paradigm or a better surrogate for exposures and risks in the chrysotile-cement construction industry than is the chrysotile-cement manufacturing industry or the friction manufacturing industry or brake mechanics who are exposed to chrysotile. Sir, I think it is incontestable that the Charleston study is an outlying study, it's a study whose results do not conform to nearly all the other studies we have on asbestos. I am not aware, but there may be and I am evidently a layman, I'm not aware of any other study that could be said to corroborate the results of the Charleston study. Yet we have cited many today even, and in our pleadings, many studies that show no increased risk of cancer from exposures to chrysotile cement and to friction products. None of the experts have contested these assertions. Yet you picked this study from Charleston which has

figures which are radically at odds with all the other studies and I am still ... knowing what I do know about the conditions in that plant which are well documented. By the way, it is not factually correct to say that it is not known whether oil was used; oil was used and that is recorded in the studies and used frequently and used consistently, so we know that oil was used and we know that oil is a carcinogenic product. It seems to me that one cannot at all exclude that as a valid hypothesis for the difference. Even apart from the oil, how and why would you justify using that textile mill, where conditions were so different to those in the chrysotile-cement industry, a plant that was clearly negligently controlled by the South Carolina authorities, why would you use such a surrogate for the modern up-to-date chrysotile-cement industry?

Dr. Henderson

145. Well if the question is asked why do I use it? The answer is many others do also. In relation to the comment about oil, I may not have expressed myself with clarity. I was not saying that oil was not used – what I am saying is that studies on the carcinogenicity of the oil have yielded negative results and cannot explain the difference between the two worker cohorts. I think this point was made by Professor McDonald himself in a recent editorial on this issue, that really the difference for the dose-response line for the two groups for lung cancer remains basically unexplained. Because we do not know the explanation, it is difficult then to control for whatever that unknown factor is. In approaching national policy for occupational health and safety, one often adopts a prudent approach, using a conservative or worst-case scenario on the "first do no harm" principle. When it comes to the fact that the two groups are different from, for example, asbestos-cement manufacture, I don't dispute for a moment that asbestos and friction product manufacture nowadays carries a very low risk because there is a low airborne fibre concentration with low cumulative exposures. The reasons why I use the Charleston group as an approach is that it identified a high lung cancer risk at low exposure and that the types of fibre released during that operation can be released during end-work, that is machining asbestos-cement products. If this can produce comparable cumulative exposures, one needs to assume that we have not proven that those exposures will have no effect. Therefore it is basically an approach of safety and prudence for the formulation of the national health policy.

146. The other thing is, I'll just reiterate, that if you go to the Abstract for the paper by Drs. Case and others, they themselves said that the risk assessment for asbestos exposure for lung cancer is based on lung cancer risk for the textile workers rather than the miners and the millers. The question is which is the outlier – do you take the Charleston textile group as the outlier and ignore it or do you say, well, perhaps there is something peculiar about the Quebec chrysotile miners and millers and, in terms of their exposure and dose response, then they are an outlier in that they showed a very low slope to the lung cancer dose-response line. Other studies have showed an intermediate risk. So the question is which one do you adopt for formulation of national health policy?

Chairman

147. As we conclude the morning sessions, could I perhaps make a couple of comments which might help us into the afternoon? Just looking at the questions and the discussion so far, it seems to me that we have had some quite good coverage of the first broad subject which is chrysotile itself. From the Panel's point of view, it will be important that we also have some time to discuss the two other broad topics of controlled use and substitute fibres. So I'd reiterate my request or my suggestion to the parties to be selective in their questions and comments. It obviously is not going to be possible to deal with all the issues in an exhaustive manner. So what we will do, I think, is see how we progress in the afternoon. The Panel will want to make sure that there is time for those other two issues I mentioned and we may have to make time for those issues and then come back to the first set of issues concerning chrysotile at the end if we have time. I'd also ask the parties, when you are asking your questions, could you please as far as possible refer directly to either the Panel's original questions or to the parts in the experts' reports where they have addressed these issues so that it is easier for the experts to respond and see exactly what you are each referring to, especially when we

have cases of some of the studies that have been cited. They are usually there in the material somewhere, but perhaps sometimes a page reference would be helpful to enable the experts to respond more quickly. We will reconvene at 3 p.m. Thank you very much.

[Lunch break]

17 January 2000, p.m.

Chairman

148. We were part way through the discussion of one question. I just checked with Mr. Hankey as to how far through the list of those questions relating to the first four questions submitted by the Panel Canada was and I understand that they've made very good progress through them. So that being the case, I think we will continue on with this current question where there may be one or two comments still to be heard, then proceed on through the remaining one or two questions concerning the first broad heading of chrysotile asbestos itself. That would then give us adequate time to work through controlled use and the question of substitute fibres. I would really express the hope that by 3.30 we should be beginning our discussion of controlled use. If that is acceptable, we now open the floor. We were in the midst of a discussion, there was a point that had been made by Mr. Christoforou which had not yet been responded to. I think Canada also had one or two additional elements that they wanted to mention. So unless the experts feel that they need to add any points to the responses they have given so far, I could pass the floor back to Canada if you were following up on the question currently under discussion. Then we can ask the experts to respond to Mr. Christoforou's point.

Mr. Hankey (Canada)

149. Thank you Chairman. Our follow-up just has to do with this issue that in the absence of much direct data on the use of chrysotile-cement products in the construction industry, what would constitute a good paradigm or surrogate among the various studies that do exist. We know that there are close to sixty studies about the use of chrysotile asbestos and we have had a lot of reference to the Charleston study: Charleston is wonderful for the jazz festival but I am not so sure it is very relevant for the issue before the tribunal. So Dr. McDonald would address the issue of what he thinks might be a more appropriate paradigm or surrogate to examine the issue of risk exposures in the use of chrysotile-cement. Thank you, Dr. McDonald.

Dr. McDonald (Canada)

150. I'll try to be as brief as I can. The first point being that, of course, the Charleston cohort of textile workers is not unique in textile cohorts. There have in fact been three: one, Charleston, is almost entirely chrysotile but there are two others which have been mentioned briefly, in which there were substantial amounts of crocidolite used. I only want to make the point that all three of these textile cohorts show this anomalous high level risk of lung cancer, whether it was chrysotile or whether there were amphiboles, whereas so far as mesotheliomas are concerned, the presence of crocidolite clearly correlated with the incidence of mesothelioma. In other words, there was no excess of mesothelioma in the Charleston cohort, any more than there is in asbestos mining and milling. Whereas in the other two textile cohorts, there were substantial numbers of mesotheliomas. Therefore, those of us who have been trying to understand why the textiles are different conclude, I think, that there is something funny about textiles. It is precisely that point that makes me say I would be personally wondering why you would choose something anomalous rather than something that is in line with the rest. I refer now to the fact that by far the biggest scientific study of chrysotile workers is the one of chrysotile miners and millers in Quebec which has been of some 11,000 men studied continuously for 35 years and now 80 per cent of them are dead. So we have one of the most complete pictures of mortality in chrysotile workers almost ... than you can imagine. There is nothing comparable. These men were exposed in the 1930s and 1940s to astronomically high levels

of chrysotile exposure. Now the issue of exposure levels has been questioned, if you like, by Dr. Infante who said that the methods used in the Quebec cohort were different from those in Charleston. I would like to point out that it is not so. We also estimated exposures individually in relation to fibre conversion and in fact published a detailed report in 1980, showing that the risk estimate based on individual estimations by fibre gave us exactly the same estimate risk as using the average. I can give the reference for that: it was published in an international meeting in Lyon by the IARC and I am sure Dr. Infante is familiar with it. I hope that that would reassure him that there is no reason to think that the exposure estimates in Quebec were any better or any worse, shall I say, than in Charleston. In fact, they were based on a very much larger amount of data, a very much larger, with parallel counts by fibre and dust, as in Charleston. There was no difference. So we are left with the fact that Charleston is an anomaly. Now I don't want to go into the Quebec result in detail but we have 8,000 deaths which is, I suppose, as big as there is in any study. What everybody, I think, is familiar with, is the fact that it showed a very modest risk of lung cancer, except at quite high levels. Indeed, the cohort mentioned as asbestos cement workers in that part by Hughes, in the part of the study which was chrysotile only, gave a slope almost identical with the miners and millers. I would also point out that the type of work in mining and milling, in which you sort out the fibres, is very similar to that in cement workers and in friction product workers, very similar and quite different from that in textile workers. And what that showed, as I say, was a very modest increase at low levels, a substantial increase of lung cancer at high levels, a substantial one, but at levels below about 25 fibres per c.c. for forty years' work, we could not detect an increase in lung cancer. It doesn't mean there wasn't one. We're quite prepared to accept the concept of a linear relationship but the fact remains that for people below that level, we couldn't detect any increase. This is a very big cohort. Equally, yes, there were mesotheliomas in this cohort but not very many in relation. There were 33 deaths from mesothelioma in miners and millers: not a single one in a man who had worked for less than two years and only one in a man who had worked for less than twenty years. Surely that suggests that the risk of exposure in that very large cohort was quite modest and that exposures at modern levels of say one fibre per c.c. wouldn't possibly be detectable. But I would submit to you that surely this experience is much more in line with asbestos cement and friction product workers than textile workers, the explanation of which we really don't know.

Chairman

151. Thank you, Dr. Infante.

Dr. Infante

152. Dr. McDonald, you made some comment that the Hughes study gave a slope identical to ... I didn't understand then which group you were referring to?

Dr. McDonald (Canada)

153. You will recall there were two plants in the Hughes study, one of which was thought to be essentially chrysotile only and gave a slope of 0.0003 if I remember, which is when I say identical, almost identical with the slope in Quebec. That was one. Not the crocidolite one which was something like 25 times higher.

Chairman

154. Thank you. Perhaps we should now pass to the question or element of question which Mr. Christoforou asked us shortly before we concluded. I think it might be helpful if we asked Mr. Christoforou to repeat the question so that the experts can respond. Thank you.

Mr. Christoforou (European Communities)

155. Thank you, Mr. Chairman. What I said was, following up on what Canada was saying about high density chrysotile-containing cement products and I was confining the argument about the manufacture of such products - and the argument was whether there was any evidence that these may have effect and what was the level, whether there were any worries about the level of exposure and the consequent asbestos-related diseases. I said we want to somehow reposition this argument and request Dr. Infante from his experience and also Professor Henderson: given the fact that even Canada does not dispute that all forms of chrysotile have been classified by international agencies, like the International Agency for Research on Cancer. There is a proven human carcinogen - I don't think anyone in this room would dispute it. And given the fact that the four scientists have defined very broadly the population most at risk to include both skilled and non-skilled workers, not only those dealing in the manufacture of cement, high-density cement and products containing asbestos. The question was then addressed to the experts was from the regulatory point of view, and Dr. Infante has such experience, is it really reasonable to believe that a country like France, which has been importing for the last fifty years, more than 95 per cent of chrysotile asbestos, and we see so many cases of asbestos-related diseases, is it really reasonable to attribute these cases to chrysotile, is it reasonable to confine the argument to cement products, which anyhow Canada does not export - Canada exports asbestos as a product.

Chairman

156. Thank you. I pass the floor to the experts. Anyone who may wish to respond?

Mr. Christoforou (European Communities)

157. Mr. Chairman, if you wish, because there is an element of controlled use here which can probably lead us to the next question. If we speak about cement asbestos, high density cement products containing chrysotile, and we know from the comments Canada has sent on 13 December, they speak about pre-sized, ready-made, ready-tailored cement products which are delivered to the construction industry and the question we would like to ask Dr. Infante, because he does make comments on this aspect in his replies: is it really reasonable to believe that even in the construction industry, these cement-containing products will never be required to be modified, cut and changed so as to fit for the purpose of construction? Can we really compare only this type of situation with the other possible parts of the workers that are exposed to chrysotile products, just to make the comparison between strictly cement construction with the rest of the population who later on will come into contact with chrysotile in the different types of activities they are involved: plumbers, electricians, insulation workers and so forth?

Chairman

158. Thank you. Does that clarify the question for you? Dr. Infante?

Dr. Infante

159. Let me see if I understand the question. Is the question: is it possible to control exposure to chrysotile in the construction sector of industry, outside of, talking about manufacturing? Is that essentially the question?

Mr. Christoforou (European Communities)

160. Yes, yes. I can give you an exact reference. It is on page 19 of your replies² where you talk about pre-sized ... and the need to modify these products and whether it is realistic to argue, as Canada does, that these high-density cement products will never be changed so that the risk will be, as Canada argues, in this type of situation very low levels of exposure.

Dr. Infante

161. It's my opinion, that I stated here, that I don't think you can have chrysotile asbestos cement products in commerce without presenting risks to individuals who may need to manipulate those products. Even if they are pre-sized, they periodically have to be cut, those that are in place sometimes have to be cut into to get into the contents inside pipes that are carrying whatever they happen to be carrying. For example, I know, in the United States, if you take chrysotile-cement to a dump, you are charged by the dump for the volume that you take to that dump site. So, for example, if you were to take a large chrysotile pipe to the dump site, you are charged for the entire volume. So it's beneficial to the construction worker to chop the cement up into pieces which then adds to the fibre exposure because, one, it's easier to remove it in pieces and two, it's cheaper when you deliver it to the dump site. I don't know what policies are in other countries or how they do business but that's how it is in the United States and that creates exposure. I feel that, and I think I've said, if you cannot control exposure in the occupational setting, in the United States particularly where even in manufacturing you can't control it, how are you going to control it in the construction sector? There are just too many variables that you can't control. People don't get educated well enough, people don't wear the appropriate respirators, there just are not programmes that can extend that far in my opinion to protect those workers. Even in the manufacturing sector, just this past October, we fined an asbestos brake manufacturer \$125,000 for being over the permissible exposure limit, for not providing respirators, for doing dry sweeping. That's in the United States where we've had an asbestos standard in place for a number of years. So, my point is that it may be theoretically possible but it's not practical to think that you can control exposure to asbestos even in the example I gave in manufacturing and it's certainly less practical to begin to control it in construction.

Chairman

162. Thank you. We appear to have made a seamless transition to controlled use at the moment. I invite new responses by Canada on that.

Mr. Hankey (Canada)

163. Before you get too excited about controlled use, I'd like to bring us back to the question ... There was a premise in Dr. Henderson's answer which I think needs to be examined. He said "even if we can't control exposure to asbestos in the manufacturing industry ... ". But Sir, the only evidence you've cited or any of the experts here or the European Communities have cited that indicates maybe we can't control it in the manufacturing sector, if I'm not mistaken, relates to textiles. As we have demonstrated, or at least argued I think quite coherently, this is an entirely different sector and one in which asbestos is not used and has not been used for many years in the European Union, certainly not in France. We have data relating to some fifty studies of the use of asbestos in the manufacturing of

²See Part V.C.2 of this report, answer to Question 5(c).

cement and friction products. We know of no instance that indicates that, in these current manufacturing facilities, there are levels of exposure, cumulative levels of exposure, to asbestos that cause danger to human health. If you or your colleagues or the European Union can put evidence on the table that indicates otherwise, I'd be happy to see it but it seems to me that the premise you base your conclusions about the use of asbestos in the construction industry is simply not viable.

Dr. Infante

164. I gave one example in manufacturing that was surprising to me because one would think, in manufacturing, you can control and you should control. It was just a shock to me to see this company clearly manufacturing asbestos brakes in the United States last fall. They were way above the exposure limit and not doing anything about it. The basis for my opinion in the construction sector is, what I indicated in my written response, that in the last three-year period there have been over 3,000 violations to our standard. A large portion of those are in the construction sector.

Mr. Hankey (Canada)

165. Tell me, Mr. Infante, how many of those violations concern excess exposure limits?

Dr. Infante

166. No, I can't do that because we don't have the data to break out that way. I'm not talking about exposures above the permissible exposure limit because quite often in construction we don't take atmospheric samples. The reason we don't take atmospheric samples in construction is that by the time you would get the sample result back, they're onto the next job. So, rather than taking atmospheric samples in construction what we do is look for other violations of the standards and proper use of respirators or no respirators, improper hazard communication to the workers that are involved, not having a person who has expertise in the hazards of asbestos responsible for the job. It's those kinds of violations that I'm referring to. I was not referring to levels over the permissible exposure limit because in construction we don't take a lot of samples.

Mr. Hankey (Canada)

167. Sir, if you would recall, the question I brought you to, or sought to bring you to, because you do keep on moving around - you're very agile - was your premise that even if we can't control exposures in the manufacturing industry, so let's forget about construction sites for a minute. As I understand it, the example you referred to was not a study. You simply found a violation, that is to say an excess of exposure limits in a single manufacturing facility. Do you have any notion or any data concerning the health effects of that high exposure?

Dr. Infante

168. Do you mean that particular exposure?

Mr. Hankey (Canada)

169. Yes, that particular exposure.

Dr. Infante

170. I don't think anyone could answer that particular question. They were above a permissible exposure limit that is already considered by the United States to present a significant risk of health hazard. The day that the compliance officer was there, they were exposed above the permissible limit which is 0.1 fibre per c.c. and you're asking me what are the health consequences of that day of

exposure. Well, I don't think anyone can answer the question to that. Canada is arguing controlled use and my point is that that's something to aim for, but because you aim for, or have a policy, doesn't mean that it gets implemented. I'm giving that as one example.

Chairman

171. I'll just interrupt you for a moment. As we're taking an *ad verbatim* transcript here, it's probably better if we go through the Chair so that I can then announce clearly who is speaking each time. So I give the floor to Mr. Hankey.

Mr. Hankey (Canada)

172. Thank you Sir. Insofar as the risks of exposures in the friction products industry, I cited you considerable data earlier which you did not contest. These data indicate no excess risk of lung cancer or mesothelioma to workers in the friction manufacture industry as compared to the general population. Speaking of, for example, Berry and Newhouse, McDonald, Teta, Teschke,

Dr. Infante

173. Can I respond? I thought that our discussion earlier had to do with asbestos-cement production, not friction products.

Mr. Hankey (Canada)

174. Am I mistaken? Did you not raise the issue of exposure levels in a facility that was manufacturing friction products. Am I mistaken?

Dr. Infante

175. Just now I did, yes. But earlier you said I didn't challenge something you had on friction products and my point is I was responding earlier to your comments on asbestos-cement production and that's why I cited the Hughes study. That's a study in asbestos-cement production. I wasn't talking about friction products earlier. I just now gave that as an example where an inspection was made and the company was above the permissible exposure limit and had other violations as well. I commented on friction materials studies.

Mr. Hankey (Canada)

176. Perhaps could I then go through the friction material studies, Mr. Chairman, with Dr. Infante. Because I did recite them all before lunch but perhaps he was not engaged in that particular discussion, I don't recall. I could certainly lead him through the evidence and we could see whether he's familiar with it and concurs with it or differs with it because he has raised, Sir, the issue of manufacturing of friction products. I thought we had demonstrated conclusively that there is no excess risk of disease from the manufacture of chrysotile-containing friction products.

Chairman

177. I think I would, on that particular point just raised, give the opportunity to any of the experts who wish to make a brief comment. It does seem to me that we've spent a considerable amount of time on this particular point. It would be, from the Panel's point of view, useful to move as much as possible toward the various issues concerning controlled use. But let us perhaps invite a brief comment from the experts and if Canada still wants to come back on that one you may. Dr. Infante, please.

Dr. Infante

178. If I look at the document 203, on page 109 and table 23, they list several studies on friction materials production. The study overall by Newhouse and Sullivan does not show any excess like the SMR is 93, the study by McDonald et al. 94, (we're talking lung cancer now), shows a statistically significant excess. Then there are mixed products in friction materials, several of those, in fact all of them, show a significant excess of lung cancer. Granted that these are mixed products, but nevertheless they show an excess and you can't totally discount, in my opinion, the chrysotile contribution. The study by McDonald et al. in 1984 shows a significant excess in lung cancer and the majority of that excess, not all, was in short-term workers. That's noteworthy in that study. So you say, well what does that relate to? I think you have to know something about the short-term workers to know why you have the excess in short-term workers, It's not the first study: workers exposed to beryllium, they were exposed for a short time, show a significant excess of lung cancer and it was all initially in the short-term workers. We know that beryllium is a human lung carcinogen. What the study shows in terms of dose-response is that there is not much potency. One of the problems in doing dose-response in the study is that you have this excess in the short-term workers. So you are not going to expect in that study to find a dose-response because presumably the short-term workers had low exposure and that was the majority of individuals in the study. You are not going to expect to be able to find a dose-response and there is not a lot of statistical power in that study when you go beyond the short-term workers. You can have a U-shaped curve in terms of the dose-response; you have a high risk in the low-exposed group, you have a slightly lower risk in the medium and you have a high risk in the highest exposed group. I don't know what you can say about dose-response in that study given that you have got some kind of observation that you need to try and understand, in my opinion, before you do dose-response.

Chairman

179. Would any of the other experts want to add anything to the point made by Dr. Infante?
Dr. de Klerk.

Dr. de Klerk

180. Can I just semi-respond to Corbett McDonald's points earlier on. I just think that they need some kind of reponse because his basic conclusion, I thought, was that because the textile industries were different, because they had higher risks of lung cancer, then we should ignore them in terms of setting health standards. I suggest that's not really the way you should go about setting health standards. The one thing they all have in common is they're textiles but they are also chrysotile. Therefore, as Professor Henderson has been saying, in terms of setting prudent health policies, if you've got some evidence that a substance is dangerous and then it's going to be used by a lot of people where the properties are unknown and, I thought we'd agreed earlier on that the majority of people we are concerned about are not friction product manufacturers, asbestos-cement manufacturers, we're worried about the people using the products later on and we don't know what characterizes their exposure, only that they will be exposed to chrysotile. We have some evidence that chrysotile is dangerous. We have a lot of evidence that it's dangerous and that we are not in a position to control that exposure. So, to say that we should ignore evidence that it is dangerous, I think is imprudent at best.

Chairman

181. Thank you. Professor Henderson.

Dr. Henderson

182. In relation to my colleague, Dr. de Klerk's, observations, I would have to agree with him. I was struck in Professor McDonald's comments that he pointed to the consistency of the high lung cancer risk among textile cohorts. He also indicated that the explanation for this difference between the textile workers and other groups of workers still awaits elucidation. We have no clear explanation for this difference. In the absence of something which we cannot explain and therefore take measures to control, prudence should lead us to take the position of maximal caution because we don't know that the extremely low risk of lung cancer found in the Quebec chrysotile miners and millers will be translated across other cohorts. In this respect, it's what I said in one of my earlier reports that when in doubt, or there are uncertainties or lack of observational data in comparison with cohorts, one adopts a principle of "first do no harm" or when in doubt play it safe for the setting of national occupational health policy. I was also heartened to hear Professor McDonald basically say that there is a modest risk of lung cancer at low levels, that he did endorse the linear relationship model and he did state that the explanation for these differences is not clearly known. Because of these uncertainties concerning risk, I would adopt the same policy as Dr. de Klerk and argue that one takes a conservative scenario in order to avoid a risk of harm – here we're talking about cancers with close to a 100 per cent mortality rate – for the benefits of the average population.

Chairman

183. Thank you. Dr. Infante wanted to come back on a point.

Dr. Infante

184. I wanted to comment on what Dr. McDonald had said earlier. I think his point was that why would one rely on Dement's study or the other studies of chrysotile-textile workers when the results seem so different from the results from the miners and millers study that he conducted. He also indicated that the Hughes study of asbestos-cement production workers gave a slope closer to the slope of the miners and millers study. Is that ... , you are shaking your head? Yes, that's right. But as I look at the data from the Hughes study, it gives a slope closer to the textile workers study and he just said that the cement production would be closer to miners and millers. When you look at the slope from the Hughes study, if you look on page 168 of that study, they indicate that for the chrysotile group only the slope, this is per unit of fibre, the slope is 0.01 for the chrysotile group and 0.016 for the mixed fibre groups. So it appears to me that that slope is closer; that's close to what the slope is from the study of the textile workers based on McDonald's Pennsylvania cohort and the Rochdale study done by Peto which is about 1 per cent, and it's a little lower than that based on the Dement study which shows 2 to 3 per cent.

Chairman

185. Professor McDonald.

Dr. McDonald (Canada)

186. I would like to say that the slope in the textiles is of the order of 0.1. The slope in the Hughes chrysotile plant was 0.0003. That is indeed approximately similar to the Quebec chrysotile miners and millers. We agree entirely that the textile plants are out of line with that by an approximately a fifty-fold difference. All I can say is that the Quebec plant is not isolated. The thing that is isolated are the textile workers. The Quebec miners and millers are similar to the cohorts of the chrysotile cement workers and similar to the cohorts of friction product workers. Indeed, there are only something like eight studies where anybody has measured the exposure at all. And seven of the eight agree with the miners and millers and only the textile workers don't. I would agree that if we had to

decide about the continuation of textile work, we would be absolutely right to say let's be careful about it. But that's seems to me a rather historical question.

Chairman

187. Another comment from Dr. Infante.

Dr. Infante

188. I just have one point of clarification. The risk of 0.0003 that you indicated for asbestos-cement production according to the document 203, that is the potency estimate for use at plant 1 which was chrysotile, crocidolite and amosite. The risk level for plant 2, which I understand is chrysotile only, was 0.007 and so that's 0.7 per cent.

Dr. McDonald (Canada)

189. It's the other way round, but I'd say we really ought to discuss this somewhere else.

Chairman

190. Well, could I suggest that we should now try to focus ourselves solidly on controlled use, given that most of the discussion so far in this afternoon session has tended to follow on from really the same issues as the morning session. As I say, if we have time at the end of our meeting after we've managed to deal with both controlled use and some aspects of substitute fibres, maybe we can come back and continue some of the discussion covering the first four broad questions of the Panel. Are the parties ready now to address issues specifically concerning controlled-use? I think probably the floor is to Canada for the next main question.

Mr. Hankey (Canada)

191. Sir, my first question has to do with the construction industry. You may regard it as preambular to the issue of controlled use because it really, I think, gives rise to what kind of a controlled-use may be appropriate in that industry. I refer to the 1980 paper by Rödelsperger et al., entitled *Estimation of Exposure to Asbestos Cement Dust on Building Sites*. In that paper the office observed that for past uncontrolled use/uncontrolled conditions, exposure levels reached 10 fibres per mm. during the sawing, cutting and grinding of chrysotile cement sheets and calculated time-weighted average exposure levels of 0.6 to 1.2 fibres per millilitre for the installation operation. Because such operations occurred only one day out of six the resulting average exposures were 0.1 to 0.2 fibres per millimetre, which is one or two orders of magnitude, that means up to one hundred times, lower than the exposure levels for past mining, milling, asbestos-cement and friction product workers. I'm wondering what you make of this data and its relevance to the subject before us since it is data from the very sector we're talking about i.e. the use of cement products in the construction industry.

Chairman

192. I must say it's seems to be a question of exposure rather than controlled use, but given that we have had a question already from the European Communities on controlled use, perhaps we could ask the experts to give us a brief response after which I will again pass the floor to the European Communities.

Dr. de Klerk

193. I thought that in that paper the levels went up to as high as a 100 and 120, as I recall, and not 10. The averages are based on specific jobs and if you are getting levels of a 120 next to somebody cutting an asbestos sheet, it depends on the structure of your job how much you would get over a week and over a year, it just happened that the average was over that particular job. There would be other jobs where you would be doing that all day long, I would have thought.

Chairman

194. Canada please, Mr. Hankey.

Mr. Hankey (Canada)

195. Perhaps I'm in error but I have the impression that, generally speaking, in the construction industry exposures would tend to be intermittent and therefore it was the accumulative factor of peak exposures which was the relevant measure of what would constitute risk. Perhaps I'm not right about that.

Dr. de Klerk

196. If you're putting up asbestos-cement fences, you would be exposed to that kind of level all the time.

Mr. Hankey (Canada)

197. If you're putting up asbestos-cement fences as a sort of a full-time occupation, wouldn't that be a little like working in a nuclear field? If indeed you understand it to be a highly dangerous job, wouldn't it be the sort of job in which presumably controlled-use should, ought to be, I would hope, is enforced and properly administered.

Dr. de Klerk

198. Yes, you would hope so in theory, but it's the kind of thing that doesn't happen in practice and that's I think one of the crucial issues: there's been asbestos regulations in place for over a hundred years and there is ample evidence that in very few places have those regulations ever been adhered to by the people using it.

Chairman

199. Any additional comments from the experts? Professor Henderson.

Dr. Henderson

200. Again I noticed the estimate of the peak airborne fibre concentration cited from the Rödelsperger paper and again my recollection was that the peak concentrations were up to 100 fibres per millilitre of air, so it was a stated underestimate. Yes, one would hope that the use of these products in the building construction industry, in particular [could be "controlled" by best work practices or, alternatively, the use of chrysotile restricted to a few special applications, analogous to nuclear fuels, but even in this latter situation] ... [END OF TAPE] ... a recent episode in Tokaimura, Japan, indicated that not even that is achievable. But the problem we have in Australia, in particular, with asbestos-cement building products is that they are so widely distributed in dwellings and buildings throughout the country, so that the largest group of mesotheliomas and lung cancers that I see related to asbestos comes not from the Wittenoom cohort - which although the exposures were

high and the risks of mesotheliomas were high, was a relatively small workforce - the greatest number of mesotheliomas that I see comes from carpenters who give a history that day in and day out they cut asbestos-cement building products with handsaws, power saws, they used power sanders, they used angle grinders, electric drills and the like. We know that all of those operations can produce substantial elevations of the airborne fibre concentrations. If we are going to use mesothelioma as an index of exposure, the fact that we have such a large number of mesotheliomas among carpenters and building construction workers indicates that exposure did occur. Now, certainly many of those workers, perhaps the majority of them, also sustained exposure to the amphiboles. But here I'm using mesothelioma simply as an index to a marker for the fact that significant exposure did occur. The simple fact is that, among the many, many cases of mesothelioma that I see, a consistent theme amongst the workers is that they were not told by the employers that the materials they were dealing with were dangerous, there were never any airborne fibre concentrations measured in their working environment, only late in history were they provided with face masks, usually in the form of a surgical paper mask or a plastic mask, and we know that even more substantial respiratory protections are sometimes ineffective. So that, from my perspective in Australia, historically, we have never seen controlled use of asbestos and the very fact that no measurements or estimates of the risk were carried out indicates that controlled use has not been in place historically in Australia and so far as I am aware, it still isn't. In fact, it was dealt with by phasing out chrysotile from asbestos-cement building products in 1987 or 1989 so that they are no longer used in this particular application. In this respect I'd have to harp back to the WHO document Environmental Health Criteria 203, which indicated that construction workers pose particular concerns because of the large and diverse nature of the workforce so that it is very difficult to disseminate information to all the individuals concerned in these types of operation. That document indicated that chrysotile use in that situation is not recommended.

Chairman

201. Thank you. Dr. Musk wanted to comment.

Dr. Musk

202. I'd just like to reinforce that. We've been arguing about which is the best sort of model of exposure in industry where it has been measured what the exposures are. But in the construction industry it hasn't been measured and can't be measured regularly, so it isn't really controllable.

Chairman

203. Thank you. I give the floor now to the European Communities.

Mr. Christoforou (European Communities)

204. Thank you Mr. Chairman. I would request the four experts, if they can take a minute, to have a look at page 28 and page 29 of Canada's comments of 13 December. Page 28 please.³ This is a document dated December 13th called "Canada's Comments on the Experts' Responses to the Questions from the Panel".

Mr. Christoforou (European Communities)

205. Page 28, and especially paragraph 6, where there are four bullet points which go over to page 29, where Canada describes what is in its view the so-called controlled use. Canada, I would like to remind you in case you have not read all the documentation, has been changing position constantly since we started this dispute about what is controlled use and progressively moves and tries

³ See Section V. D. 1, Canada's comments to Question 5 (a).

to restrict more and more what in its view is controlled use it has in mind. Now, I would like to request you to read these four bullet points and would appreciate if you could tell me if this type of situation described here, that is: to distribute products only to companies 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 material 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 at established centres equipped to cut the products to size and where persons cutting the products are trained and are licensed to work with asbestos; and, fourth point, to police the downstream users in cooperation 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. The question is, from your own experience in dealing with these questions in your profession, do you think this is a feasible and realistic scenario taking into account the type of population exposed as you have defined it previously? Thank you.

Chairman

206. Thank you. Let's give the experts a moment to decide who might want to respond first on that point or whether you want to take up aspects of it, as it's quite a broad issue, individually. Dr. Infante.

Dr. Infante

207. I feel that this stewardship programme, when I read this, I feel that it's not a reality; it's a possibility but it's unlikely and definitely not likely to occur in construction. With regard to point 6 about controlled use,⁴ that "this permit will be withdrawn if the company does not meet the following commitments", what went through my mind when I read that was: withdrawn by whom? Who enforces this? The first bullet point about "those companies must have workers trained and licensed to install the product", well who oversees that training? It's not clear to me who would do that in countries that would be working with the asbestos? And bullet point 3: "to provide products cut to specification". I think that's good to do that but then there are always adjustments that have to be made, so even though products may be cut to specification, there are places where you have to trim or the pipe or something is too long and you have to make some adjustments, and the concern is when those adjustments are made, that proper precautions aren't taken. Then, in the last bullet there are penalties for failing to provide this product's stewardship. As I read this, I wondered what are these penalties and how many have been issued to date. This, to me, seems good in theory but it doesn't seem real to me. Then when I just recently read an article about asbestos, chrysotile-asbestos exposure in Morocco which imports Canadian chrysotile and I see these photographs in this article just published this year – I have a copy of the article – and it shows that asbestos is just all over the place. So I'm wondering if the Canadian Government, if it has this partnership for a sustainable development, why are there countries like Morocco, Brazil and India that seem not to be following what's required by this stewardship and the controlled use?

Chairman

208. Thank you. I think perhaps any further comments from the experts before we get into discussion on this item. Dr. Musk, please.

⁴See Section V.D.1 of this Report, Canada's comments to Question 5(a).

Dr. Musk

209. This sort of regulation would require a new system for enforcement which hasn't previously existed anywhere that I know of. Secondly, it doesn't take into account people working with products that are already installed, modifying and installing pipes, electricians, plumbers and the like. So it certainly wouldn't cover all the opportunities for exposure.

Chairman

210. Professor Henderson, please.

Dr. Henderson

211. I'd have to agree with my two Panel colleagues that, as I've indicated, so far as I'm aware, controlled use for the stewardship-type of arrangement has never been used in Australia in relation to asbestos products of any type. As I've also indicated we don't really have detailed dust measurements in almost all workplaces including asbestos manufacture; or where they have been done, their count seems to be artificially low in comparison to the fibre count seen in the lung tissue of the workers. So that historically in Australia I cannot see that this has ever been applied and as Dr. Musk said I don't think that it is enforceable in law. It would require a whole new infrastructure in industry and legislation to bring into effect. Just as a common sense observation, as far as I can see for a products manufacturer to police the after-sales uses of its products would introduce a new dimension in Australia. I'm mindful, for example, of the fact that automobile manufacturers who sell cars, yes, they may sell them only to people who hold a driver's license, and the government authorities do have a list of the license holders and the registration numbers; but for an automobile manufacturer to try and police dangerous driving, excessive speed or driving under the influence of alcohol, to monitor drivers on the road and then report them to the police, would produce an entire new dimension into Australian society at least. It's one that I would think would create an immediate conflict of interest between sales and profitability on the one hand, and the policing and regulatory function on the other. But I think it's fine in principle, but I suspect that's it's unworkable in practice in Australia, at least unenforceable at law.

Chairman

212. Dr. de Klerk, do you want to add anything?

Dr. de Klerk

213. I was just curious as to whether there was any sort of precedent for the system that they put into that document. I can't imagine anything like that working anywhere with anything. But presumably there may be some precedent somewhere for that kind of system?

Chairman

214. Thank you. Does either party or Panel members want to comment on those responses? OK. If that's not the case, then perhaps I could give the floor to Canada for their next question.

Mr. Hankey (Canada)

215. You all obviously have some doubts about the efficacy of controlled use: I guess it's chiefly among construction workers, although perhaps your remarks aren't entirely limited to that sector. But, I wonder which of the following aspects of control do you consider key to safeguarding health of construction workers using high-density chrysotile products? There are chrysotile products, low density chrysotile products in place, so evidently a certain amount of due care is required by people in

the construction industry. I'm wondering what measures you would consider to be particularly necessary. I'll list a number and perhaps you could indicate whether you think they are key, whether you think they are useful to making controls work or work better. We could start with hazard risk and risk assessment. Do you think that proper hazard and risk assessment assist in safeguarding health of construction workers using high-density chrysotile products?

Chairman

216. Would it help if you listed all of them?

Mr. Hankey (Canada)

217. I'm quite happy to do that. I'll go slowly though because usually what happens when I read one of these long sentences, I'm asked to repeat it so I was going to take it one by one: Hazard/risk assessment; information; education; training of workers; registration of tradesmen; hazard control; personal protection; licensing for specific potential dangerous risks; sale of products to registered users only; and finally the point which has, I think, just been mentioned: removal of license to purchase chrysotile and chrysotile products if users are not in compliance with regulations. I don't know which of you feel that you have expertise in this area of industrial hygiene, please feel free to answer the question.

Chairman

218. Let's give the experts a moment or two to think about that and let's decide who might want to answer.

Dr. Infante

219. Could you just quickly go over. I missed a couple of them as I was writing them down.

Mr. Hankey (Canada)

220. Sorry, I have hazard/risk assessment; information, education, training; registration of tradesmen; hazard control; personal protection; licensing for specific potential dangerous tasks; sale of products to registered users only; and finally, removal of license to purchase chrysotile or chrysotile products if not in compliance with regulations. And my question is, which of these following aspects of control do you consider key to safeguarding the health of construction workers using high-density chrysotile products? Perhaps I should also frame it slightly differently, that would make a material difference, a significant difference.

Chairman

221. Dr. de Klerk?

Dr. de Klerk

222. I can make a few points. It's probably outside my area of expertise most of the latter ones. The only thing that I would consider myself vaguely expert in is in terms of risk assessment. It's already been mentioned that, in fact, we don't have risk assessment information for most of the downstream users, so obviously that's important. I've been involved in studies where we've tried, using information in education and training - this is outside the asbestos area, this is to prevent accidents in industry and sometimes it works and sometimes it doesn't. The fourth one, hazard control, obviously if you reduce the exposure you reduce the risk of disease, I assume that's what that means. The thing that strikes me about all of them is the fact that, in essence, all of these steps were

part of the asbestos regulations, certainly in force in Australia, at say, for example, the Wittenoom mine and mill and they weren't really of any help at all in preventing disease occurring from there. There was risk assessment in the sense that people knew that heavy exposures caused asbestosis but those levels weren't kept. There was information provided on the notice board that the mine was a registered mine, there were attempts made to reduce the dust but they didn't reduce it, they just spread it around; people were encouraged to use face masks but in the heat they couldn't wear them; the mine had a license which the government was supposed to supervise and it didn't. When they broke the rules it didn't remove the license. So it 's an example of where, although you've got something in theory that should work, in practice it won't.

Chairman

223. Do other experts want to add to that response? Dr. Infante, please.

Dr. Infante

224. I would agree that they would all be helpful, assuming that the hazards/risk assessments have already been done or we wouldn't be here today. As far as information, education and training, yes, that's important; registration of tradesmen, that's important; hazard control, of course these are all important; personal protective equipment is important. They are all important but some of the problems are, you have personal protective equipment, what does that mean? Let's take respirators, for example, when do you wear a respirator? Our standard requires a competent person who has to know about where asbestos may be, whether or not the product may contain asbestos. It's not simply having a respirator available, but do you have a respirator fit-testing programme to assure that the worker who wears a respirator is getting the protection they should have; do you have a programme that cleans the respirator? Do you have different types of respirators that are available depending on what the exposures might be? So a respirator programme requires a fair amount of training in itself and knowledge on the part of a competent person. Then, one of the problems is that in the United States there is a tendency not to train short-term workers in the construction sector because it costs to train workers and you know they're only going to be there short-term and they're going to be moving on to another job where there isn't asbestos exposure. Since they're going to be gone shortly there's a tendency to try to save money and not to train workers that would be there for a short period of time. So, all of these are good: the problem is implementing such a programme in reality, I think is difficult.

Dr. Henderson

225. Again, I would reinforce the comments from my two colleagues. In Australia, the use of respirators in the building construction or any other industry poses particular problems, despite penalties, in the form of fines, and even three breaches of the regulations and the worker is dismissed. The simple fact is that compliance is poor because in a hot, dry environment, where temperatures regularly go over 30°C and sometimes above 40°C, the thermal consequences of wearing a respirator create such discomfort to the worker that they will often discard the respirator, irrespective of penalties of work without them and so will their fellow work mates. In relation to regulation of the various practices outlined, the simple fact is that government regulatory agencies in Australia, increasingly have a diminishing capacity to regulate work hazards. For example, after the Conservative Government was elected in Australia, the National Occupational Health and Safety Commission was downsized and approximately one half of its workforce was made redundant, so they no longer have the capacity to supervise all points of end-use of asbestos or any other product at all times. As I've said, the building construction industry is of particular concern, simply because of the spectrum of different occupations represented in that group who have asbestos-related diseases and many of these individuals simply go straight into the building construction industry with minimal training or no training: they simply leave school and suddenly appear as an unskilled worker in the building industry and acquire their training on-site. Those who employ them are often individuals or

very small businesses which do not themselves have a background and depth to provide training in the correct application of safe work practices. So, we're dealing basically with a very large, diverse, often poorly trained workforce who have a very poor appreciation of the risks to which they're exposed and a common theme amongst the cases I see is that the worker didn't really know that it was asbestos or if he did, he didn't know it was dangerous, didn't know that the operations he was carrying out would in fact generate dangerous levels of airborne dust, and therefore the individuals are unaware of the risks they've been running. In many of the cases I see, for example mesothelioma, we really have to use the tumour as an index of exposure in order to uncover some pattern of asbestos-exposure which even the worker has been unaware of and I cited a couple of examples in the supplementary remarks to my report. The other point I would emphasize is that, from my perspective, controls are most certain when there is a minimization of the total amount of asbestos introduced into society, into the workplace and into the general environment. And if you don't introduce any more, then hopefully, provided you can try and implement reasonably safe work practices, you can minimize exposures to those products that remain, but their total amount will diminish over time. But one must recognize that because of the diversity of this group, that the training programmes will not always be followed, there may be poor worker compliance, and that many of the programmes are not always effective in any case.

Chairman

226. Thank you. Dr. Musk, would you like to add something?

Dr. Musk

227. Once again, this sort of programme would ignore the people handling asbestos that is already *in situ*. I think it would certainly act as a deterrent to using asbestos at all because it would be pretty unwieldy to implement, if it was implemented properly, so people would probably look for other products to substitute but it does ignore the asbestos that's already there.

Chairman

228. European Communities, please.

Mr. Christoforou (European Communities)

229. If I can continue with a follow-up question on this point. One can then legitimately pause for a moment and ask the question: all these requirements indicated by Canada, all these steps one has to go through, where do they come from? How did Canada come up with this list of steps to go through before applying controlled use? And I would like to ask the experts, because I see Dr Infante says on page 17 of his replies⁵ that he is not aware of any international standard that would prescribe a controlled use, let alone a controlled use in the sense of containing all the steps indicated by Canada. So where do they come, all these requirements? Is there any international standard that would require such steps? Thank you.

Chairman

230. Thank you. On the point of the existence or otherwise of an international standard, Dr. de Klerk, first.

⁵See Section V.C.2, reply to Question 5(a).

Dr. de Klerk

231. If I have understood correctly, and if this is the same question I asked earlier about whether there was any precedent for such a system, is that what you're saying. Because I don't know of one and that's why I was asking the Canadians.

Chairman

232. Do other experts wish to address the question just raised by Mr. Christoforou? Dr. Musk, please.

Dr. Musk

233. I'd be interested to hear Canada's response to the question because I don't know where they came from and I'm not aware of them existing elsewhere.

Chairman

234. Does Canada wish to comment on that?

Mr. Hankey (Canada)

235. No. I have a question for Dr. Musk actually.

Chairman

236. I think perhaps Mr. Christoforou would like to clarify his question.

Mr. Christoforou (European Communities)

237. Mr. Chairman, this is partly scientific in the strict sense and partly relevant for the discussions we'll have later on. But I ask this question because of the express sentence in the replies of Dr. Infante on page 17 in the fifth middle paragraph.⁶ Just to indicate that Canada has been portraying until now that one ILO convention, International Labor Organization Convention 162, prescribes something which can be applied and achieve controlled use, which will achieve a level of exposure below a level threshold that is not dangerous. Just to say that the type of controlled use, just now indicated by Canada, exists nowhere and I am glad to see that the scientists confirm they have no knowledge whatsoever of any of this type of controlled use applied anywhere.

Chairman

238. I'll give the floor to Mr. Hankey to ask the question you were going to ask Dr. Musk.

Mr. Hankey (Canada)

239. I just want to clarify your last statement but one. You said something about, in response to my question about which of these aspects of control would be key to safeguarding the health of construction workers, you said something about in-place asbestos. Could you just repeat that? I want to fully understand the import of your remark.

⁶See Section V. C. 2, reply to Question 5(a).

Dr. Musk

240. I was saying, I don't think any of the measures addresses the handling of asbestos that's already in place to the extent that, if the measures were put into place, there would still be asbestos going into construction. That asbestos would then be there for plumbers, electricians and anyone else coming along later. It doesn't address their exposure.

Mr. Hankey (Canada)

241. What, Sir, would you propose to do about asbestos already in place?

Dr. Musk

242. I think, as a general principle, one needs to minimize the exposure to it and work practices are important in that area. Once it's there, as far as I'm concerned, it ought to stay there until there's a good reason to remove it. And then when one does remove it, it should be removed with due care.

Mr. Hankey

243. If I understand you correctly, you would say that asbestos which is already in place should be left there and that due care should be taken with its use. By due care, could controlled use be another way of expressing the same idea or not?

Dr. Musk

244. The notion of controlled use I interpret from these measures relates to providing new asbestos products for the construction industry, not to protecting workers against asbestos that is already in place.

Mr. Hankey (Canada)

245. What measures would you propose? What kind of due care measures, perhaps you wouldn't call them controlled use, I don't know what appellation you would give them, but what kind of measures would you propose to deal with asbestos already in place? Because Sir, I don't know what the situation is in Australia, but I do assure you that in France, which is the country that's at issue here, there are vast amounts of asbestos in place, including very great amounts of low-density asbestos, much of it of mixed fibre, although we have some dispute with the European Union as to how much of it is mixed fibre, certainly a substantial amount of it is. It's incontestable, as a matter of social history, that the very issues that gave rise to the ban that's currently in place, which is the very subject of this dispute, is in-place asbestos, old uses, high density, low density asbestos in places like Jussieu and there are vast amounts of in place in France, so the issue of how to deal with that in-place asbestos strikes me as extremely relevant. So I would like to know, Sir, you seem to think that these measures that I have proposed or put out for comment are not applicable to in-place ... and you also propose that it should be not removed. Now I think everyone would know that it's a real and present danger, these old uses of asbestos, vast amounts of which still exist in France, how would you propose to deal with it if you were a policy maker?

Dr. Musk

246. I'm not a policy maker and this isn't my area of expertise, but I would say that when the time comes that it's required to be removed in buildings where it's past its use-by date, or the insulation is deteriorating or the asbestos-cement products are cracked and broken, the roofs and there's a lot of asbestos-cement roofs, where I come from, have deteriorated to the extent they're not doing their job, then the people permitted to remove them need to be policed to use methods for removal that will not

expose the worker. There are in Australia licensed asbestos removers and they are required to have air-supply respirators and they do the major jobs for removing asbestos from buildings. But the most exposed people are the small businesses or the handyman who does it himself and nobody gets to know that it's happened till it's passed. So it's relatively unregulated.

Chairman

247. Thank you. That's a last point before we break for coffee.

Mr. Hankey (Canada)

248. I am very glad to hear that in Australia you are able to exercise control, it seems, when necessary to remove this stuff. I'm really, though, very interested in what goes on when the stuff is there because it may not be removed, I don't know, for twenty, thirty or forty years, you haven't given me any indication but you say you're not involved in the business of policy-making, but fortunately we have at least a couple of people on the Panel of experts who are ... Mr. Henderson, for example, in his paper, in his summary of conclusions, prescribes indeed the remedy which this Panel should provide in this case, so he is clearly in the business of making policy, or at least he has a very great interest in it. And I wonder, Sir, what remedy you might propose, relative to the vast amounts of asbestos, including very much low density products of asbestos in place in France, much of it containing mixed fibres. What would you do about it, Sir?

Chairman

249. I'll give Professor Henderson the opportunity to respond to this point, and then we will have a coffee break of 15 minutes. Professor Henderson.

Dr. Henderson

250. The question is based on a false premise. I'm not involved in setting public policy on this issue: this is done by others, and particularly, the National Occupational Health and Safety Commission. My comments on the disposal of existing asbestos products in place are similar to those of Dr. Musk. I think that some of the procedures that you've outlined should be implemented, as a matter of common sense, to try and minimize exposures to existing products. As Dr. Musk says, there are licensed asbestos removal organizations in Australia, which are meant to carry out these operations under controlled conditions and at minimal risk to the asbestos-removal workers and to the general public. However, just in the last six months, I've come across two mesotheliomas that have been a direct consequence of asbestos-removal programmes because it appears that those procedures were not followed. One of them was a fireman who was regularly called to buildings which had been incinerated by fire and where fire alarms were set off by high airborne dust fibre concentration as a result of asbestos-removal programmes. This fireman visited these buildings at least once a month to check them through and was, we believe, exposed to elevated airborne fibre concentrations. Another one concerned a university lecturer who for a period of weeks had to walk to and fro through a building where an asbestos-removal programme was being carried out. Although the removalist was supposed to encapsulate the material and seal it in polythene bags, it appears that they left it lying on the ground in an unprotected state and this person, the lecturer, walked past this asbestos material quite regularly over a period of some weeks. So, I agree that best work practices should be aimed at, in order to try and minimize exposures, but my concern is one of caution and prudence, to realize that not everybody is going to implement these procedures at maximal efficiency all the time and that exposures will occur. I'd agree with Dr. Musk that probably the best thing to do with existing asbestos in place is to encapsulate it until such time as the building is demolished or unless it can be shown that elevated airborne fibre concentrations exist in the building, and again I've got other mesotheliomas which have occurred simply from individuals who worked in department stores where there is friable asbestos insulation with elevated airborne fibre concentrations. So, I think you need to

balance the risks of removal against the risks of the asbestos continuing in place until the time of demolition. But implementation of best work practices should minimize exposures but ultimately exposures will be best minimized when there is no new introduction of asbestos materials into the workplace where they can remain for 20, 30 or 40 years and be subject to periodic and sometimes regular maintenance and renovations.

Chairman

251. Thank you Professor Henderson. We'll obviously be continuing the discussion on controlled use after the break, so we'll have a coffee break now, and return at 16h50.

[Coffee break]

Chairman

252. We left for coffee just after Dr. Henderson has responded to a question from Canada and that in itself was part of the discussion on the question Mr. Hankey initially raised regarding the various precautionary measures to take in the case of prevention of exposure to asbestos. Could I ask if there are further comments or follow-up questions on the remarks by Professor Henderson? Canada, please.

Mr. Hankey (Canada)

253. In the overriding interest of not missing my evening cocktail, I was going to desist from further discussion on controlled use. So, if we are moving on the substitutes, I would have no more to say. But if we are going to be staying with controlled use, then, yes, I would have ...

Chairman

254. The Panel's view was that we could perhaps continue for another ten to fifteen minutes on controlled use, if you wish, and, by 5.10-15, we should be into the discussion on substitute fibres. Mr. Christoforou, you would like the floor as well?

Mr. Christoforou (European Communities)

255. Yes, I would like to ask one more question on controlled use.

Chairman

256. Yes, please. We'll continue the discussion for another ten to fifteen minutes, and then we'll move to substitute fibres.

Mr. Christoforou (European Communities)

257. Thank you. The question is addressed to all scientists. It is prompted by a comment made by Drs. Infante and Henderson, that controlled use is even much more difficult to be applied in non-occupational circumstances. I think it is an obvious statement, but I would like you to comment on this and say if you know whether the equipment suggested to be used (the mask and all the other equipment) and the proceedings to follow, will always, constantly, achieve, in occupational and non-occupational circumstances, a level of exposure which is below 0.1 per cent of fibre per mml. Do you think that it will always be achieved below that threshold? Thank you.

Chairman

258. Dr. Infante, please.

Dr. Infante

259. The point of my written comments was that, I don't think controlled use is likely to occur in the occupational setting and so that in the non-occupational circumstances, it would be even much more difficult, because there is no..., you don't really have the potential here for training that you do with the occupational setting, or even the construction sector, where training quite often doesn't take place nor any of the other programmes related to controlled use and exposure to asbestos. If you are asking, well, in non-occupational circumstances, could fibres exposure exceed 0.1 fibre per ml, it depends on what the individual would be doing. I would say, yes, that's possible to occur. For example, I know that with using a Transite, which is an asbestos-cement product, when individuals tear this off a wall, it usually breaks apart, because it's either nailed or screwed on, and it's much faster to just simply pull it off the wall and quite often, it will pull the nails out with it. So, it is a lot faster to remove it that way. But quite often, as is usually the case, it breaks into pieces and when that occurs, you can generate fibre levels above 0.1. That's one example. It just depends on what's being done in a non-occupational circumstance and what product is being manipulated.

Chairman

260. If there is no further comment on that point, I'll pass the floor to Mr. Hankey.

Mr. Hankey (Canada)

261. My question is in relation to the products that we are discussing, in particular those used in construction, that is to say chrysotile-cement products, do you consider the greatest risk to be at the point of installation, or maintenance – that is to say interventions after it's put in, by electricians, carpenters, plumbers and so on and so forth – or its demolition and removal? Where, at which point would you consider the risk to be greatest? And I wonder if each of you could answer that question. Thank you.

Chairman

262. Dr. de Klerk first.

Dr. de Klerk

263. It depends on the exposure, really. I know we said it before but it's obviously where the greatest amount of dust is generated, it's going to be the process that gives the greatest risk in terms of existing measurements made around operations that are available – obviously demolition and removal have the highest exposure levels. But in some ways the people in demolition and removal may experience less exposure because there is more of a likelihood that precautions will be taken. ... **[END OF TAPE]** ... But again, even within this sort of full-face respirators there is a measurable level of asbestos found. So obviously they are all at risk and it depends how well the operations are done. I don't think you can be sort of make hard and fast rules about it, but I mean historically people installing have not taken precautions and have experienced great risks and as we can see from them being the group with very high levels of mesothelioma on all registers. Maintenance, you've got the people who again tend not to historically take any precautions, and again there are groups of people, plumbers, electricians who, using Professor Henderson's point about using mesothelioma as an index of exposure, there are people there who have obviously been exposed. Obviously, though, I'd like to say that they are all at risk in one form or another and it depends on the level of exposure and the precautions that are taken.

Dr. Infante

264. I don't think that, as a general statement, you can say that one has a greater risk than the other. I think they all carry, you know, a great risk depending on how the installation, or the maintenance or the demolition is carried out. That is what relates to the fibre exposure.

Dr. Henderson

265. Again I'd agree with the comments from my two colleagues. I see cases of mesothelioma related to all of these types of activity using mesothelioma as an index-marker of exposure. Again, it's my belief that the risks will be dependent on the frequency of the operation, the types of operation carried out, the airborne fibre concentrations generated and the duration or the type of work. I see mesotheliomas resulting from all of these activities, for example, among carpenters, and for example the handyman who regularly carries out maintenance and renovations on houses, where he might use a power saw to cut a new doorway through an asbestos-cement clad wall, will generate fibre concentrations equivalent to the carpenter carrying out this type of work day after day. It's just the frequency with which he does this type of operation, may be less. The same can also apply to demolition, particularly of small dwellings, if precautions are not carried out during building demolition and disposal of the asbestos-cement product. So I'd have to say that I couldn't give a figure for the risks to each of these groups because they would vary according to the variables I've already mentioned, but I do see cases of mesothelioma resulting from all of these types of activity.

Chairman

266. Thank you. Dr. Musk, would you wish to add anything to those three comments?

Dr. Musk

267. I'd agree with the three previous speakers. I'd suggest that people involved with maintenance, being the least regulated group, and least easily regulated group may be at greater risk but like Dr. Henderson I see cases of mesothelioma from people involved in all those activities.

Chairman

268. Thank you. Mr. Hankey.

Mr. Hankey (Canada)

269. Thank you. If I could just try to make a synopsis of what I just heard. I think each of you said essentially, although Dr. Henderson's answer was I think more complex than the others, but certainly each of you said really it all depends on what precautions are taken. Dr. de Klerk said precisely that, and as did Dr. Infante, Dr. Henderson did say that but along with a number of other things, and finally, Dr. Musk said exactly that and added to that he thought that perhaps maintenance was perhaps the biggest problem because it was the most unregulated. So, if I understand you correctly, then the issue at each point, that is to say installation, maintenance - and by maintenance I mean interventions once it is already there by tradesmen such as plumbers, carpenters and electricians and so on and so forth. And then the removal -you consider you can't distinguish between these risks, you say it all depends on what precautions are taken at each point. That's what each of you said. Now, I'm wondering still if we could come back to this problem about the asbestos in place because we all recognize - and I don't think there is any issue about this - that the asbestos in place, if you like, fibre for fibre and man for man in terms of the exposure to it represents still the greatest risk. I concede that we don't know what the risk will be perhaps 100 years or 200 years from now, that's another question. But currently, I recall, Dr. Henderson said early this morning that, when I asked which he thought was the greatest risk, he indicated, if I understand correctly, that, yes indeed, the

greatest risk from an exposure at a given level, or for the same amount of exposure time, I think was really the point, but you can correct me if I've got it wrong, to low-density products which may contain mixed fibres. You thought that would be greater - sort of intervention for intervention - than interventions in these high-density chrysotile-only products. You said you had difficulty calculating the overall risk because indeed, you felt there were more interventions; more people were perhaps coming into contact with chrysotile-cement products than with these old kinds of products. Is that a correct statement, Sir, of what you have said this morning? I haven't finished my question, but I'm basing it partly on what you've already said. I want to make sure that I've got that right.

Dr. Henderson

270. Well that is not quite correct. What I was trying to say this morning is that the risks of lung cancer and mesothelioma will be dependent on the type of operation carried out, and therefore the airborne fibre concentration, the frequencies with which those operations are carried out, and their durations – that you are looking at a risk related to cumulative exposure levels; and the point that I was trying to make this morning was that, if you take a cohort, for example, the Wittenoom cohort in Western Australia, those individuals have a very high risk of mesothelioma and yet, the cohort, which numbered about 7000 individuals, was relatively small. Although, if you are looking then at a lower risk in a larger group of workers, for example, carpenters, because there are many, many more carpenters in Australian society than Wittenoom workers, then the total number of mesotheliomas you will see in this larger group at lower risk will be equivalent to those you see from the Wittenoom cohort or even larger in terms of absolute numbers. When I took that figure I took the figure for carpenters only, but if you add in plumbers, plasterers, other building workers, it adds up to a very large group, and probably one of the largest groups represented in the Australian Mesothelioma Register.

Mr. Hankey (Canada)

271. I'm a little sceptical about that thesis, because it is a bit like saying, if each of us here, if we have a keg of beer that is brought in and each of us gets a glass of it, that's as much risk as if I drink the whole amount and then go out and drive my car. I rather expect the authorities who are regulating drinking and driving probably wouldn't concur with that approach to the matter. In any case, let me proceed with the real point of my question. If it all depends really on what precautions you take and if the dangers are the same at installation and interventions when it is installed and then removal, I still don't think I have received from any of you any kind of satisfactory answer to what we do about the in-place old uses of asbestos which, I still have to insist that if we look at the social commentary on why France introduced the ban, the ban was introduced precisely in order to remedy those problems. That, or at least I shouldn't say precisely, is certainly what led to the political pressure to introduce the ban, and a study of the French media at the time definitely would prove that; so, if controlled use doesn't work for these new products which, I must say I think most commentators would agree, are less, product per product, dangerous than the old ones and the low density ones that contain mixed fibres and amphiboles, ... what is the political, the social remedy to that stuff in place, if indeed controls don't work because when I ask you what happens to remove any type of product, whether it is the old one or new one, you tell me that really it all depends on what precautions you take, which seems to me that you are saying it all depends on what controlled-use mechanisms are put in place. So I am still at a loss as to what we are going to do about this huge danger facing society with the in-place old products.

Chairman

272. I'll give the experts the opportunity to respond on this point and then I think we need to move on to substitute fibres and then immediately after one or other of the experts has responded on this current question, I'll give the floor to the European Communities. Or did you want to make a point, Mr. Christoforou?

Mr. Christoforou (European Communities)

273. I would like to hear the follow-up question after I hear the replies of the experts on this point, Mr. Chairman, please.

Chairman

274. OK. Fair enough. You may do so. Professor Henderson first, please.

Dr. Henderson

275. Well, in reply to my comment about the workers at risk, I can only reiterate my comment, it is not so much on the controls in place, although hopefully by disseminating information one can try and implement best work practices to minimize exposures to those products that remain in place. When you disputed the estimates I gave for a lower risk among carpenters in comparison to the Wittenoom cohort producing a larger aggregate number of mesotheliomas, your doubts are not supported by the figures from the 1999 Report for the Australian Mesothelioma Register, which records, among carpenters and joiners, 187 mesotheliomas due to single exposures only, 33 additional mesotheliomas from workers with multiple exposures, making a total of 220 cases. Whereas the Wittenoom cohort accounted for 189 mesotheliomas (single exposure) and an additional 25 (multiple exposures), making 214 cases. So, although the risk of mesothelioma is high in the Wittenoom cohort and among non-smoking survivors, mesothelioma is now the most common cause of death, the numbers in aggregate are slightly less than the number of mesotheliomas in absolute numbers we see among carpenters, simply because – although the carpenters are at lower risk – there are many many more carpenters in Australian society than there were Wittenoom workers. So, that low risk needs to be multiplied against a larger population. That is the point that I was making.

276. As for the problem of asbestos in place, I agree entirely that this is a major problem. What do we do about the asbestos which is in place, and how do we minimize exposures? Some of the strategies that you've indicated, in terms of informing people, trying to implement these best work practices, will hopefully minimize the exposures but so far as I am concerned this is an ongoing problem for which we have no easy solution, taking into account that many of the people who carry out interventions on those products, by way of building maintenance and renovation, are almost completely unregulated. Although it is very regrettable, despite our best efforts, I believe that we are going to continue to see mesotheliomas from that type of exposure. But having pointed out the difficulties of minimizing exposure to asbestos in place, that does not by itself, from my perspective, represent a justification for the introduction of more asbestos into the environment whereby the total quantity will become greater and the scope for people to be exposed, even at lower levels, will be translated into an ongoing population over time.

Chairman

277. Thank you, Professor Henderson. I will give the floor briefly to Mr. Christoforou for the follow-up question he wanted to ask. Could I ask that you do make it brief and hopefully the reply could be brief so that we don't lose any more time before getting on to the substitute fibre questions. Thank you.

Mr. Christoforou (European Communities)

278. Mr. Chairman, I renounce to ask the question because the reply of Dr. Henderson covered my point. Thank you

Chairman

279. Well, in that case, I would give the floor to the European Communities, if they wish to ask a question concerning substitute fibres.

Mr. Christoforou (European Communities)

280. Yes, Mr. Chairman, thank you. We would like to request all experts to elaborate on your replies concerning alternatives products which are non-fibrous and whether, in their knowledge and experience, such non-fibrous alternative products have been classified as proven human carcinogens, as is the case with chrysotile asbestos. I highlight the word non-fibrous alternative products.

Chairman

281. Yes. Dr. de Klerk.

Dr. de Klerk

282. I'd just like to answer fairly briefly. The question, as it was asked before, was really asking about alternative fibres but when you look at non-fibrous products, as far as I am aware, anyway, it is the fibre quality of asbestos that makes it dangerous and if you've got a product that isn't fibrous then it doesn't have those qualities and therefore is unlikely to be risky in that same kind of way.

Chairman

283. Thank you. Question six did concern substitute fibres. It was not specifically asked about non-fibrous substitutes. If there are no further comments on that point, could I now pass the floor to Canada on the fibrous substitutes issue.

Mr. Hankey (Canada)

284. You may indeed. I mean, I do have a comment about that question but perhaps if you rule the question at the border, perhaps I need not comment.

Chairman

285. Well I think, as I see it, the issue that was concerning the Panel was the question of fibrous substitutes particularly.

Mr. Hankey (Canada)

286. My question is to any of the experts who really cares to answer, but I'd perhaps suggest that I would like Dr. Infante, among others, to answer because I believe he has considerable expertise in this area. Basically my question is that "Do you agree that the information base regarding human exposure to substitutes is meagre compared to what we know about chrysotile?"

Chairman

287. Dr. Infante.

Dr. Infante

288. I think that compared to what we know about chrysotile asbestos, the data on most toxic substances is meagre in comparison.

Mr. Hankey (Canada)

289. I just wonder then if Dr. Henderson and Dr. de Klerk and Dr. Musk would agree with that statement.

Chairman

290. Dr. Henderson please.

Dr. Henderson

291. I would agree with that statement in broad terms. So far as I am aware, except for a couple of large cohort studies on man-made mineral fibres there are virtually no epidemiological investigations of human populations for the majority of the substitute fibrous materials. Evaluation of their effects is based basically on fibre characteristics and experimental models.

Chairman

292. Dr. Infante would like to add something.

Dr. Infante

293. I just want to elaborate. I think that for workers exposed to fibreglass, there has been considerable epidemiological study, but there has not been epidemiological study to my knowledge with the polyvinyl alcohol, the para-aramid fibres or refractory ceramic fibres. But there is experimental data for those substances and I think I mentioned earlier today what some of the findings were.

Chairman

294. Thank you. Any additional comments from the experts or further follow-up from the parties? Mr. Christoforou, please.

Mr. Christoforou (European Communities)

295. Mr. Chairman, this is a question that addresses partly the previous question on non-fibres and also on this question on the fibres raised by Canada. I would like to request the experts whether, in their view, when asbestos-containing products are replaced in their use are they normally replaced in the majority, if not exclusively, or can they be replaced almost exclusively in their use by products which are non-fibrous? If you allow me to somehow rephrase the question: the European Communities have been arguing that there are - and I can give the example of cast-iron pipe, high-density polyurethane pipe, concrete pipe, metal roofing sheets, clay roofing tiles, plasterboards and so on, which can substitute asbestos contained in products in nearly all of its uses. Are you aware of this fact? Thank you.

Chairman

296. Could I just perhaps reiterate that we did not specifically ask the experts to address the questions of non-fibrous substitutes. The interest of the Panel in the scientific aspects of this were especially concerning the qualities, properties of fibrous substitutes. I could perhaps invite the parties and the experts to concentrate as far as possible on the specific issues that were asked under Question 6, which is really concerning the fibrous substitutes.

Mr. Christoforou (European Communities)

297. Mr. Chairman, with due respect, we don't think this is the situation. Question 6 refers to both fibrous and non-fibrous and we would suggest that it is even more relevant, because, as we suggest here and as we have been making in our submissions, there are numerous non-fibrous products which can substitute asbestos for nearly all of its uses. So the question is very relevant to see the magnitude of the problem, of whether there is a problem posed by fibres, which will come later on.

Chairman

298. Having re-read the question very carefully, I can say there were one or two references to non-fibrous substitutes. I would invite the experts to respond on that point.

Dr. de Klerk

299. I'll just chip in a couple of points. In terms of, in Australia anyway, I mean I haven't really looked into this because I sort of assumed it was fibrous but, for asbestos-cement the main manufacturer uses cellulose instead of asbestos. I think in brakes it's para-amid fibres, so that in fact, as a general rule, most of the substitutes are fibrous, well certainly in Australia. I would also like to add that most of the comments that I made in terms of this, because it is probably outside my area of expertise in a way, were based on a good review by Harrison et al., which I think everyone has probably read. I think that sort of summarizes the extent of knowledge at this time. I haven't found anyone who disagreed with that at all.

Chairman

300. Thank you. Any amplification or further comment? Professor Henderson?

Dr. Henderson

301. Well again, like Dr. de Klerk, I focused on fibrous substitutes because so far as we know the agents implicated in the causation of mesothelioma are almost always fibrous materials namely, amphibole asbestos, chrysotile asbestos, or the naturally occurring mineral erionite. There are some concerns about refractory ceramic fibres; I am not aware that there are any data in humans but there are some experimental models which cause reason for concern. So when we are dealing with mesothelioma I think we are dealing with substitute fibrous materials as opposed to non-fibrous materials. Of course, the non-fibrous materials may have toxic effects which are different, but so far as we know they are not implicated in mesothelioma induction; so I focused my answer on the substitute fibres like my colleague Dr. de Klerk.

Chairman

302. Thank you. Perhaps if there is no further follow-up on that one I could now give the floor to Canada for further question or comment on fibrous substitutes.

Mr. Hankey (Canada)

303. Yes. My next question is: do you believe that fibres used as substitutes for chrysotile in cement and friction products, for example, glass fibres, cellulose fibres, para-aramid fibres, PVA and RCFs, such as potassium octotinate should be used without controls? Perhaps Dr. Infante, you could start, and I would like the others to answer as well.

Dr. Infante

304. If you could perhaps refine your question. What do you mean if they can be used without controls. What do you mean by that?

Mr. Hankey (Canada)

305. Well, for example, would you suggest that workers who are installing them or removing materials made with, that contain these substances, any of them, should work without masks, for example, that they should saw it with high-speed saws. That would be two questions. I would have to really, I'm afraid, ask my experts to propose other answers, or help me formulate other questions. I suppose – I may be wrong – that for each of these they present somewhat different risks, and that therefore the measures you would impose would perhaps be different for each of them. Another thing might be exposure limits for example, would you say there would be a need for exposure limits for any of the materials I've indicated, and if so, which ones?

Chairman

306. Dr. Infante, are you able to answer on the basis of that?

Dr. Infante

307. I think, as a matter of industrial hygiene you should reduce exposures to the extent that you can in the occupational setting. Now by saying that that, doesn't mean that these fibres carry the same risk as chrysotile. I don't think that any of them do, but as a matter of proper industrial hygiene, we should try to reduce exposure levels or use good work practices. You can get some of these things perhaps in your eyes, from sawing them, so perhaps you would want to wear goggles, for example. I always think you should handle substances in the workplace appropriately. Should you be concerned about the same risk of exposure to these substitute fibres, as you should be concerned about asbestos fibres? I guess what I would say is that I don't see the evidence that these fibres are as harmful; but yes, you should try to control them to the extent that you can.

308. You have to look at what some of the information is here. If you look at refractory ceramic fibres, for example, I think that they are hazardous, and that, if you are working with these fibres, yes, you should take precautions with them and you should wear appropriate protective equipment if you are exposed to these. But it is my understanding that the refractory ceramic fibres would not be a substitute for chrysotile on any large basis. That does not mean that they are not toxic. Is there evidence that they are carcinogenic in humans? – No. But there is evidence in experimental animals, and on the basis of that I would take all of the precautions that I could. With the polyvinyl alcohol fibres, there have been some implantation studies that have been conducted on experimental animals and IARC concluded that there is insufficient evidence of carcinogenicity for those fibres. It is my understanding that their size is such that with a large diameter, it is unlikely that they would be respirable. So I think that it is good that that's the case. So I don't think there would be much biopersistence then if they are not able to get into the lungs. With the para-aramid fibres there has been, I believe, inhalation study and intra-peritoneal injection studies that IARC reviewed and they concluded that there was no evidence of carcinogenicity for the para-aramid fibrils. In terms of biopersistence, I think that I cited the study by Searl that indicated that these fibres greater than five

microns in length are less biopersistent than chrysotile fibres greater than five microns in length. The para-aramid fibres, it is my understanding, are like somewhere between ten and twelve microns in diameter, so they would not be in the respirable range. However, there is the potential for some fibrils to break off from these fibres that are smaller. What hazard there is from these has not been studied in humans, but on the basis of the para-aramid fibrils from experimental studies, IARC concluded that there is no evidence of carcinogenicity. I am concerned about any exposure, but when you talk about what's the potential disease risk I think you are dealing with a known factor with chrysotile asbestos; the studies that have been done don't indicate any cancer response for these. So if it were me, I would, if I were in occupational health, I would prefer to see para-aramid fibres substituted for chrysotile in the appropriate applications. With cellulose fibres, they have not been studied in experimental animals or in humans. With glass fibres, in my opinion, there is evidence in experimental animals that these are carcinogenic. I think it is more likely than not that respirable glass fibres may be carcinogenic to humans, it is more likely than not. Does that mean it has been proven? – No it doesn't. But I would exercise caution with those. And what I am talking about here is, you know, there may be a risk of lung cancer in humans. I have not seen any information that mesothelioma is associated with glass fibres – and I think I mentioned that in my report. As I had mentioned earlier, I, at one time, thought that this high risk in the Canadian study was specifically related to low fibres on the basis of the data that were available. But I have other information now about that, the exposure to those cohort members to other known human carcinogens, including asbestos. So I've qualified my comments about the potency of glass fibres compared to chrysotile asbestos. I mean at least we have standards for these in the United States, as nuisance dusts, which is like a fifteen mg. per cubic metre limit. So we do have some regulation of them, and I suppose as you have, if you have more information available that indicates that there should be better control of these and if they are not being controlled – and I think you need to implement that knowledge. But exposure to fibreglass in the United States, at least in manufacturing, has always been quite low. Going back to the 1940s, I think average exposures are 0.04 fibres per millilitre and that has always been low exposure. These fibres are used for blown-in construction and these fibres can get up to – I think the highest levels I ever saw was 7 fibres per ml, that's the peak, the highest I've ever seen. Usually they are below, certainly below 1 fibre but I feel that these glass fibres aren't as potent as chrysotile asbestos. And I have already commented on the ceramic fibres.

Chairman

309. Thank you Dr. Infante. Any point which the other three members would like to add?

Dr. Musk

310. I might just add to that that any particulate would be considered a nuisance dust unless it has specific properties and that is because, partly at least, of the possibility of occupational airway disease, so-called industrial bronchitis and airway narrowing. I mean that's an entity that seems non-specific just related to particulate content irrespective of the nature of the particulate.

Chairman

311. Thank you. Are there any further comments to add. Yes, Professor Henderson.

Dr. Henderson

312. Again, my comments would closely mirror those of my colleagues. From the point of the potential carcinogenicity of substitute fibres, as indicated in my report and my supplementary remarks, the key factors seem to be the dimensions of the fibres, their persistence in lung tissue, and in various studies, their capacity to cause disease. Based on the review by Harrison and a number of the articles submitted, including annexes from Canada, and the recent press release from the Health and Safety Executive in the United Kingdom, there seems to be a growing body of opinion that

the substitute fibres are safer in general, with the exceptions already indicated by Dr. Infante. Importantly that they are less biopersistent in lung tissue, so that presumably their capacity for carcinogenesis is proportionately less than chrysotile.

Chairman

313. Mr. Christoforou, please.

Mr. Christoforou (European Communities)

314. Will you allow me a follow-up question on this point?

Chairman

315. Yes. Go ahead.

Mr. Christoforou (European Communities)

316. The follow-up is what Dr. Henderson said, with a few exceptions mentioned by his colleague, and I think he referred to the statement by Dr. Infante. Dr. Infante has identified ceramic fibres and glass fibres as possible, probably, dangerous substitutes. The question I would like to ask is the following: I don't know if you know of any country which has banned asbestos from use – all uses of asbestos – and it has substituted by glass fibres entirely all previous uses in which asbestos was used and employed. In other words, I wish Dr. Infante to expand on what he said on a large basis. Is it really true that these suspected - these two possibly suspected products – the glass fibres and the ceramic fibres, are a realistic substitute for all uses made of asbestos previously? Is there any country who has? Is there any knowledge about this? Can we really argue, as Canada is implying, that these are possibly dangerous and so because they are too dangerous, we should not ban asbestos? Thank you.

Chairman

317. Thank you. Dr. Infante.

Dr. Infante

318. No, I wasn't implying that they were the substitutes. We were asked to address the toxicity of a number of fibres and I stated my view on the fact, I said that I didn't – from what I understand it – you know ... refractory ceramic fibres are limited to very special high heat applications and it would not be a general substitute for chrysotile – certainly not with a chrysotile-cement products. It is my understanding that I didn't realize that fibreglass was put into asbestos-cement, but I think you would have to ask someone else about that, it was more like the polyvinyl alcohol fibres that I believe that I had read in some submission might be one of the substitutes; and if that is the case, that would probably be good since they are of such dimension that they are unlikely to be respirable. Now knowing the toxicity of them, as the data that are available for the polyvinyl alcohol fibres don't indicate any carcinogenic response. But I'm saying that you don't have to worry about that if they don't get into the lungs at all, whether or not they might be.

Chairman

319. Thank you. Canada, please.

Mr. Hankey (Canada)

320. I have a follow-up question.

Chairman

321. Certainly, please.

Mr. Hankey (Canada)

322. Dr. Henderson, when you said it all depends on the characteristics of a fibre – and I think you were speaking of any fibre, whether it is a natural fibre like chrysotile or an artificial or man-made fibre. Would that be the case that these elements that you identify would be the same for any fibre as being the criteria by which you would determine its carcinogenicity?

Chairman

323. Professor Henderson.

Dr. Henderson

324. In broad terms the answer is yes, and the characteristic that I would focus on is the dose to which the individuals are exposed, the dimensions - are the fibres similar in character to those of chrysotile or amphibole fibres? - and the biopersistence of those fibres in tissues. Finally, in experimental systems, have those fibres shown a carcinogenic effect or not? They would be the four key parameters on which I would base assessment of substitute fibres.

Mr. Hankey (Canada)

325. I gather two of these factors have to do with the quality of the fibre itself, that's to say dimension and biopersistence. They have something to do with the way the fibre is made, I take it, whether it is made naturally by nature or whether it is made by man. The other two - it was dose and you said - what was the fourth - I'm sorry?

Dr. Henderson

326. Dose, dimension, and durability.

Mr. Hankey (Canada)

327. Certainly, dimension and durability would be objective criteria by which any fibre could be measured. And presumably chrysotile is used for certain purposes precisely because it possesses certain characteristics of dimension and biopersistence. Or was biopersistence the correct idea? I mean certainly that it lasts - I presume - that it has a certain durability. Now if that is the case - perhaps it isn't - but let me finish my question and you can demolish it at any point of the logic you see fit. If that's the case, then won't manufacturers be rather inclined to create artificial man-made fibres, substitute fibres as it were, that have similar characteristics to chrysotile; if indeed they tend to use it for the same purposes; in friction products and/or in cement products.

Chairman

328. Thank you. Professor Henderson will respond on that point.

Dr. Henderson

329. That really is an engineering question which is starting to fall outside my area of expertise. My understanding is that chrysotile fibres are chrysotile fibres, and that you can't start engineering them to be of vastly different dimensions from what they already are, whereas for some of the substitute fibres they can be engineered or reproduced in such a way, that the fibres are either not respirable or that they do not have the dimensions that are normally associated with carcinogenesis from asbestos. That is the only point that I would make. I would suggest caution about any substitute fibre which had fibre characteristics which were very similar, for example, to those of the amphiboles and refractory ceramic fibres would be one example. I would treat refractory ceramic fibres with great caution but my understanding is that from the other features that I have mentioned, the fibres are either less biopersistent than chrysotile, or they have different fibre dimensions, or they have not been shown to cause cancers in experimental animals.

Chairman

330. Thank you. Mr. Hankey please.

Mr. Hankey (Canada)

331. You did say just now that artificial fibres can be engineered to produce more or less whatever characteristics the manufacturer wishes, including characteristics that would be similar to chrysotile. I think it is not a stretch to think that, if chrysotile is useful because it possesses such and such characteristics, dimension, length, endurance, there will be a certain incentive on the part of manufacturers to produce similar products, artificial products, to perform similar functions. That being the case, I wonder if you could tell me what controls are in place in Australia to ensure that any new fibres put on the market by manufacturers, or used within an entity, a manufacturing entity - you know, produced by one branch of a corporation and used in another branch of the same corporation - what controls are in place in Australia to ensure that any new fibres brought, created, engineered to replace chrysotile are not carcinogens and generally not as dangerous as chrysotile?

Chairman

332. Professor Henderson.

Dr. Henderson

333. Again, the point that I would make is that your concerns seem to be that the substitute fibres might be manufactured to have fibre properties similar to those of chrysotile. My perception is that there would be a pressure on a manufacturer to produce a substitute material with similar thermal characteristics and stability in the environment - but with fibre characteristics which are clearly dissimilar. It is a matter of deciding on - if you like - the properties of the material to be used versus the fibre characteristics. This is really an engineering question that falls outside my area of expertise and I really cannot comment on any of the engineering processes. In Australia, with the exception of fibreglass, as far as I am aware, the substitute materials are imported rather than manufactured on site; but certainly I do know that the National Health Authorities have recommended substitution of chrysotile in virtually all uses, provided that the alternative material is not more harmful and clearly is not as effective, and there may be certain restrictions for which chrysotile can still be used. For example, there is still some production of brake linings and asbestos-containing gaskets in Australia; but the simple fact is that in new automobiles, manufacturers have largely replaced the use of chrysotile by substitute materials. Now apart from this, I don't know what particular controls have been implemented in Australia and what recommendations ... [END OF TAPE] ...

Mr. Hankey (Canada)

334. I was just wondering, and if I could paraphrase your answer, it seems to be that you are not aware of any controls that are in place to ensure that substitute fibres are not carcinogenic, or otherwise dangerous to human health, but that you think that manufacturers would be decent enough not to produce them. That was more or less what I got from your answer. Would that be a fair restatement of it?

Chairman

335. Professor Henderson.

Dr. Henderson

336. It is not quite correct. The National Occupational Health and Safety Commission has concluded, largely on the basis of overseas investigations, that the substitute fibres are safer, and that there is some allowance for importation of small amounts of chrysotile into Australia, in the order of about a thousand tonnes per year, for the use of production of chrysotile-containing friction products materials and chrysotile-containing gaskets. But certainly, the Occupational Health and Safety Commission has recommended that there should be no introduction of any new material, that a material that has been already replaced by chrysotile should not in future be replaced by a chrysotile-containing material. Certainly the vehicle manufacturers in Australia, with one or two exceptions for older vehicles, have replaced the use of chrysotile in those vehicles. This is monitored to some extent by the National Occupational Health and Safety Commission, but the details of its controls, if there are controls in place, are not known to me.

Mr. Hankey (Canada)

337. Then if I understand, your answer is that the substitutes being used are believed to be more safe than chrysotile, based on experiments carried out and their use in foreign countries. I think that was what you said.

Chairman

338. Professor Henderson.

Dr. Henderson

339. Yes, this is contained in the document I refer to continually as NICNAS99, where the National Occupational Health and Safety Commission has concluded on the basis of overseas expert bodies that the substitute fibres are safer than chrysotile, and for that reason they have recommended that chrysotile be phased out over the shortest time interval possible, its use for the remaining periods of time be restricted only to a few applications, and that substitute fibres be implemented. I would add though, that if one is talking about, if you like, fibres released from brake linings of passing vehicles, apart from the use of those chrysotile-containing materials, to the best of my knowledge there are no controls in the general environment so that the substitute fibres are treated in that respect no differently from those that still contain chrysotile.

Chairman

340. Thank you. As time is moving on, perhaps I could just explain how we would tend to conclude the meeting. The meeting was supposed to finish at 6.00 p.m. It is possible that we can go on to 6.15 p.m. I do want to give the opportunity, but not the obligation, to the experts or any of the experts who wish to make any concluding comments. After they have done that, I would like to

address one or two procedural questions, including the question raised by Canada earlier this morning about the two pages of comments submitted by Dr. Infante. So I think there would be time for perhaps one further point or question from either of the parties. Or maybe one quick one from each of the parties if you have a burning need to ask one or two more questions. Mr. Christoforou.

Mr. Christoforou (European Communities)

341. Mr. Chairman, I would like, as a last point on the issue of substitutes ... I am sure the scientists know that for example the Environmental Health Criteria 203 has recommended the substitution of products of asbestos, all types of asbestos, by other products because they are safer. On the basis of the existing knowledge of which you are aware, with the possible exception of tile fibres and glass fibres, do you think the substitutes which are used are safer than asbestos-containing products?

Chairman

342. I think Dr. Infante will respond.

Dr. Infante

343. There is no evidence that they are harmful. We are talking about the polyvinyl alcohol, para-aramid fibres and fibrils, and cellulose fibres. There is no evidence that any of those potential substitutes are carcinogenic – there is no information at all on that. As scientists and as people involved in public health, we do exercise caution in using these fibrous materials. That is different than saying that they have met the same standard of toxicity as asbestos fibres because they haven't. We just exercise caution. But I would, you know, recommend that, as the document says, chrysotile fibres certainly be substituted for.

Chairman

344. Thank you. It's a good place to give the opportunity to Canada for a last comment or question.

Mr. Hankey (Canada)

345. I just have one short comment and then a question, and my comment is simply ..., and it really relates to the debate I was having with Dr. Henderson. You kept referring to the "substitutes" for chrysotile as if those substitutes were a fixed universe. Now it is my impression, I may be wrong, but they are not in fact a fixed universe, that, given the fact that chrysotile has been banned rather recently in many jurisdictions, new products indeed, new substitutes are invented and come on to the market from time to time, and what I was really asking you was whether you were aware of any controls in place to assess and ensure the safety of these new products, before they were put on the market. I understood your answer to be "no". Now, I'd like now to move on to my last question and it is simply this: a November 1999 report of an independent committee organized by INSERM⁷ states that "No significant excess risk of cancer has been ever detected from exposures to asbestos at the same exposure levels used to evaluate the carcinogenicity of substitutes." Now if you like I could read from the original French but I believe that's the best translation we can give to that. So would you wish that I read it in the French language or not? Well that is my question. My question is do you agree with this statement? And perhaps I might start with Dr. de Klerk.

⁷INSERM, *Effets sur la santé des fibres de substitution à l'amiante*, Paris, 1999, p. 181.

Chairman

346. Well I think we will leave it to the experts as to who wants to answer first. But could I ask the experts to answer this one briefly so that we do have time to go on to any concluding comments you wish to make.

Mr. Hankey (Canada)

347. I would Sir, ask each of the experts to answer because each of the experts did indicate in one way or another that they thought that substitutes were probably safer than asbestos, than chrysotile. So I think it is a fair question.

Chairman

348. So we'll have a first response and then leave it to the others as to how they indicate their views. Dr. Infante.

Dr. Infante

349. Excuse me. I just wondered, rather than repeating it in French could you just repeat it in English? I want to make sure that I understand the question.

Mr. Hankey (Canada)

350. Yes, of course. It says the INSERM Committee stated that no significant excess risk of cancer has ever been detected from exposures to asbestos at the same exposure levels used to evaluate the carcinogenicity of substitutes. That is the INSERM A voluminous report they have just published on the issue of substitutes.

Chairman

351. Dr. Infante. Have you managed to absorb the question? Or maybe anyone else, I could give the floor to anyone.

Dr. Infante

352. Can you repeat it? No significant increased risk of cancer from exposure to asbestos ...

Mr. Hankey (Canada)

353. ... has ever been detected at the same exposure levels used to evaluate the carcinogenicity – I have a lot of problems with that word - of substitutes. Je peux le dire en français.

Chairman

354. While Dr. Infante is pondering his response, perhaps I could hear a comment from Mr. Christoforou. But we really do need to be brief here.

Mr. Christoforou (European Communities)

355. Yes, Mr. Chairman. Thank you. While the scientists are thinking, we have here the author of this report and he can probably put this citation into context because he has written this phrase and he can explain what it is meant and then the scientists give their reply.

Chairman

356. Well, I am happy for the gentleman to do that provided he too can be brief.

Mr. Christoforou (European Communities)

357. Yes, he will be very brief.

Chairman

358. Then we will ask a brief response from the experts.

Dr. Goldberg (European Communities)

359. Merci Monsieur le Président. Je suis Marcel Goldberg et je suis effectivement un des auteurs de ce rapport, et notamment, je suis le responsable de cette partie. Nous avons effectivement écrit la phrase qui a été citée, mais une fois de plus, je crois que la citation est extraite de son contexte. Il est vrai que nous avons écrit cela, mais c'est une discussion dans la partie qui traite uniquement des données épidémiologiques, et il faut rappeler que le rapport complet fait quelque chose comme 450 pages, et que nous avons pris en compte l'ensemble de toutes les données disponibles, y compris les données expérimentales, et que la conclusion de l'ensemble de tout nous a permis de conclure que, très vraisemblablement, le risque de cancer attaché à ce type de fibre était largement inférieur à celui du chrysotile. Merci.⁸

Chairman

360. Thank you. I take it the translation has finished coming through. We will now ask the experts, do they wish to make any comment. Dr. de Klerk.

Dr. de Klerk

361. Does that mean, therefore, that the substitutes are at least as safe as chrysotile? Is that what you mean, is that why you asked the question? That therefore means that all the substitutes are at least as safe as chrysotile, is that what you are saying?

Mr. Hankey (Canada)

362. Yes, I think that could be a fair conclusion – yes. To the same level of exposure.

Chairman

363. Well I think that Professor Henderson wants to make a comment. I was about to conclude that the response from the experts had already been made, but please.

⁸ [Thank you, Mr. Chairman. My name is Marcel Goldberg and I am one of the authors of this report and I am in charge of this part of the report indeed. We have drafted this sentence that has been quoted, but, once again, this quotation is out of context. It is true that we said that, but of course this is one sentence in the part dealing with epidemiological data and the whole report has more or less 450 pages, and we took into account all the data available, including experimental data, and the conclusion of this whole work has enabled us to conclude that, in all probability, the risk of cancer linked to this kind of fibre was largely under that of chrysotile. Thank you.]

Dr. Henderson

364. I was a little bit surprised by the question as put because it didn't distinguish between mesothelioma or lung cancer and amphibole versus chrysotile asbestos. But now that the translation has been given, clearly it refers to epidemiological investigations and I must admit I was a little bit surprised because animal experimental studies usually involve exposure to fibres of quite high levels - this is simply because the lifespan of an experimental animal is sufficiently short in comparison to the humans that you need to expose these animals to very high fibre concentrations or through a peculiar route whereby dust deposition in lung and translocation does not occur. That is you'd use either an implantation or a high-dose inhalation model. Again I'd would draw the same conclusion as Dr. de Klerk that the experimental investigations indicate that, if anything, the substitute fibres are likely to be safer than chrysotile and that even if one takes that question at face value, it indicates that none of them is more hazardous than chrysotile.

Chairman

365. Thank you. I think I would just ask the other experts if they want to indicate a view that differs in any way or adds in any way to the comment Professor Henderson has just made. Yes, Dr. Musk.

Dr. Musk

366. In practice then, and I don't know the answer to this, but one of the issues might be how easy it is to control exposure in the asbestos industry versus the substitute fibre industry.

Chairman

367. Thank you. Well it seems we have exhausted the comments. I would like to thank everybody very much for their participation in this meeting. I did say that I would offer the floor to the experts to give them the opportunity, if not the obligation, to make any concluding comments. We have about five minutes left so, and I would like a couple of minutes myself to deal with the issues that are raised concerning procedure. So I think I will quickly invite any of you who do wish to make any brief comment to do so, otherwise I will continue. Professor Henderson.

Dr. Henderson

368. One point that I would raise as a final issue which has not been covered in the proceedings today is the clearance half-life of chrysotile from lung tissue, because it is stated in a number of the submissions that chrysotile has an extraordinarily short half-life in lung tissue and I think a figure of 28 – 48 hours has been mentioned and a figure of less than ten days. When I read the figure of ten days I was reminded of a story I was told in my childhood of a wise man who performed a service for a Persian king, he was asked as his reward what he would like and he suggested a little thing, Sire, I'd like a grain of rice on the first square of a chess board and for you to double it on each successive square. Now if you go back to the paper by – I might add that he ended up owning all the rice in the kingdom – but if you go back to the study by Green and others on the Charleston textile workers at a median interval after sixteen years following cessation of exposure to asbestos, they still had a mean concentration of over 33 million fibres per gram dry lung tissue. If you then go back and say that the half-life is only ten days then you need to double that count for every ten days that you go back in time, 36.5 doublings per year for sixteen years

Mr. Hankey (Canada)

369. Excuse me, I would like to raise a point of order, Sir. I want to know, Sir, am I to have an opportunity to respond to the experts' closing statements?

Chairman

370. These are closing statements and we cannot offer the opportunity to respond in this ...

Mr. Hankey (Canada)

371. In that case, Chairman, will you please request that the experts not raise new issues in their closing statements. Dr. Henderson has just said that he is raising an issue which has not been discussed today. I think it is not really due process, if I might say so Sir, that the experts raise at the end of the day issues not discussed today to which I shall not have an opportunity to respond. So let's either give the parties a fair opportunity to respond or else let's keep the summaries to issues that have already been covered today.

Chairman

372. As I say, there is no room for any further debate on these issues today, but we are also under extreme time pressure which eliminates the possibility for raising any new issues. Can I just invite Professor Henderson to wrap up his remarks briefly?

Dr. Henderson

373. Well, this is not a new issue, it was covered in my Endnote to my original report and it was covered in the supplementary remarks I made. What I questioned is ...

Mr. Hankey (Canada)

374. Point of order, Mr. Chairman. Shall I have an opportunity to respond to Professor Henderson's Endnote?

Chairman

375. I think that ... Excuse me. We can't get into discussion at this point on whether something was or wasn't a new issue and as the clock is also ticking, I think we noted the point that was made. I think I would like to ask Professor Henderson not to keep addressing this issue but to wrap up his conclusion in the next thirty seconds if he can.

Dr. Henderson

376. OK. I shan't pursue this issue.

Mr. Christoforou (European Communities)

377. Sorry. I really object to this. The experts are free to express their views on what they have written in their reports. I don't understand the objection of my colleague. There is no rule which allows the experts to express their views on what they have written in their report. If Canada didn't feel necessary to raise this issue with [...] because the thing was clear.

Chairman

378. Mr. Christoforou, I have invited Professor Henderson to conclude his remarks in a manner that we can complete our work on time, and he is in the process of doing that. Please continue.

Dr. Henderson

379. Without pursuing the particular story further I would point out that I have already cited a paper by Finkelstein and Dufresne, published in 1999, which for the Quebec chrysotile miners and millers pointed to a half-life in human lung tissue of eight years for fibres greater than 10 micrometres in length, which suggests that chrysotile is far more biopersistent in tissues than many people realize. In fact we need to recognize that there is a short-term rapid clearance of chrysotile and then the remaining fibres that are deposited will remain persistent in lung tissue for a period of years thereafter – long enough for a carcinogenic effect.

Chairman

380. Thank you. Do any other of the experts wish to say anything briefly in conclusion? I say there is no obligation to do so and the shorter the better. Dr. Infante.

Dr. Infante

381. Yes. I would just like to sum up maybe where I began today. That is that, in my opinion, chrysotile asbestos is a very potent carcinogen, controlled use in my opinion is not realistic and the substitute fibres do not demonstrate – none of them demonstrate - carcinogenicity in humans and because of that I feel that in terms of public health it would be beneficial to substitute.

Chairman

382. Thank you. Dr. Musk.

Dr. Musk

383. I have nothing to add to that except to say that I agree with it.

Chairman

384. Dr. de Klerk.

Dr. de Klerk

385. I think I agree with that too.

Chairman

386. Well could I, on behalf of the Panel and the parties, thank our four expert very much, firstly for the very hard work that they put in before this meeting and their forbearance with us all during this meeting, being bombarded with questions and comments on these very complex issues. We are very confident that your work in the service of the Panel will be of great assistance, both to the parties and to the Panel members and to the Secretariat as we move towards the conclusion of our work. I did say that I would address at the end of the meeting the various procedural issues. Concerning the point raised by Canada earlier today following the couple of pages of comments which Dr. Infante circulated, the Panel discussed this in the lunchtime and I reiterate the distinction I made between the rules that were made for the parties and the arrangements which were made for the experts. The

parties were given clear deadlines to submit material, comments on the experts' reports. The experts themselves kept to their deadline for the submission of their own reports. We did not place any restriction on what the experts might do between all those submissions of reports and comments and what happened at this meeting. We certainly viewed the paper put together by Dr. Henderson, I think, it was dated 10 January, as he explained as a contribution to this meeting. I think the Panel would view Dr. Infante's note in a similar light, as a contribution to this meeting, to the content of the discussion at this meeting. So it is not our intention to allow any further submission of evidence in relation to the papers submitted by, or the comments submitted by Dr. Infante, and we would note that Dr. Infante does not really depart at all or vary from the opinion that he expressed in his initial written reply. However, I would remind the parties that the purpose of having the gap of two days between this meeting concerning the scientific issues and the next formal meeting (the second formal meeting of the parties) was precisely so that the parties had time to comment, if you like, in the course of the second formal meeting on some of the discussion of the scientific issues that had taken place here. So, in the Panel's view there was adequate opportunity for the parties to incorporate any comment that they wish in the course of the second formal meeting later this week.

387. Just also to set this proceeding a little bit in the broader context, I could just explain what will happen in the coming weeks. It is probably very familiar to the parties. As I said, following today's meeting and the second substantive meeting which will take place on Thursday and Friday, the Panel will proceed to prepare its report. The first part of the report will be a summary of the facts of the case and the arguments of the parties, and will be provided in draft form to the parties for their comments. The responses of the experts to the Panel's questions will also be included in the report. The experts will all receive a draft of the relevant section and will be given the opportunity to make any necessary corrections. Subsequently we need to provide a first and interim Panel report to the parties, including findings and conclusions. Then the parties have an opportunity to comment on that and we then submit a final report. As stated at the beginning of the meeting, there will be a verbatim transcript of today's meeting which will be included as an annex to the final report, and both the parties and the experts will receive a draft of the transcript of today's proceedings for information and corrections as necessary, because the draft is taken straight off the tape. So we would ask – this one final task – we would ask of our experts, will be to check through the record of their remarks. So that, I hope, is a clear explanation of how we intend to proceed. Mr. Hankey please.

Mr. Hankey (Canada)

388. Thank you Sir. I just wondered, Sir, if we might expect any further contributions from the experts as part of their contributions to the meeting today or whether their contribution is now definitively closed, apart from the checking of the record that you had referred to.

Chairman

389. Thank you. Yes, as I said before the meeting started. We basically have a finite resource, a finite time and it was as set out in our programme and schedule, that this is the final point of the expert process with the exception of the checking of the record of the transcript. So as of now the Panel's expert consultation has been concluded. Yes, Mr. Hankey.

Mr. Hankey (Canada)

390. When, Sir, might the transcript be available to the parties?

Chairman

391. I'll ask the Secretariat to check that. It will be judged on how long it is going to be.

Secretariat

392. Yes it's technically rather long. I don't expect the transcript to be ready before mid-February, before the descriptive part is provided, if not slightly after. Not this week anyway.

Chairman

393. Can I thank all the delegates, the parties and the experts, my own colleagues on the Panel and the Secretariat staff very much once again for your cooperation and enabling us to get through our work in the very short time. And I should also like to express a special thank you to the interpreters who went on a little bit beyond the call of duty. Thank you very much.
