DATABASE:EAP
*** Reference number- 17785
:NO 900932.013
:TL Summary of Substance of Expert Evidence of Dr David Douglas in Robert Culkin v. CSR.
:SO AUTHOR Douglas D
OCCUPATION Doctor
ORGANISATION Australian Occupational Health Management
PLACE Sydney, NSW
REFERENCE WA Supreme Court Action No. 2110 of 1990
:TY SUBEXPEV
:LO JMcD CDR102 PC
:CA THIS DOCUMENT IS PRIVILEGED
:AD CASE NAME: Culkin, Robert
:TT

SUMMARY OF SUBSTANCE OF EXPERT EVIDENCE OF DR. DAVID DOUGLAS

1. Statement by G V Coles

Coles' success in 1958 in obtaining a court order for the institution of practicable control measures for asbestos exposures was in relation to an asbestos factory, not a factory in which a fitter was intermittently exposed to asbestos insulation material. Coles and I were academic colleagues in the Institute of Occupational Health at London University in the 1970s, and he should know that even then, the UK Factory Inspectorate only applied the provisions of the 1969 Asbestos Regulations if it was considered that employees were likely to be exposed to the then TLV of 2 f/mL time weighted average for chrysotilc. The work that Culkin did would never have been regarded in the 1970s (as before) as being subject to the Asbestos Regulations. Page 3 of Coles' statement suggests that occasional exposures for short periods of 2 f/cc (the same as 2f/mL) or more constituted a breach of statutory requirements. Even if fibre levels occasionally reached 2 f/mL or more (a level thought unlikely by G Major, though not measured at the refinery) the TWA would be well below 2f/mL and therefore not in breach of any recommendation.

2. Report prepared by W Musk

attachment 2 - none of the tables from Selikoff and Lee, "*Asbestos and Disease*", refer to the exposures of fitters in contact with insulation material in a factory such as refinery. They refer to the work of insulators.

attachment 10 - the 1956 Annual Report of the Chief Inspector of Factories refers to "one very hazardous process, to which the Asbestos Regulations do not always apply, is the removal of old heat, insulation lagging." The full quote (p 142) concludes "the persons who do it are regularly engaged on it and are constantly exposed to risks". The Report was clearly not referring to the work of a maintenance fitter.

attachment 16 - the NH MRC model asbestos regulations published in October 1975 apply to exposures to "asbestos dust to an extent which is liable to cause damage to the health of persons". Sections 7(1) and 8(1) refer. They were not to apply to any asbestos exposure.

3. Statements by others

From the various other statements it seems clear that Culkin worked only occasionally on insulation material and was never involved in the major insulation work required in the boiler house. Importantly, not all the insulation contained asbestos and some of the most dusty material would have been calcium silicate. It seems clear that, apart from some special gaskets made of crocidolite, all the asbestos insulation was chrysotile.

4. Selikoff paper

Selikoff Churg and Hammond. Asbestos exposure and neoplasia. JAMA 1964.
This papers' abstract states that "building trades insulation workers have relatively light, intermittent exposure to asbestos." But on further reading, it is clear that the paper refers to full-time asbestos insulation workers, not maintenance fitters occasionally exposed to a variety of insulation materials.

In addition, the SMRs for lung cancer were so much higher than those now found in the Wittenoom cohort that one must question the methodology of the research, eg the choice of reference population.

4. Significant recent publications

Sluis-Cremer GK and Bezuidenhout BN
Relation between asbestosis and bronchial cancer in amphibole asbestos miners BJIM 1989; 46; 537-540

In the absence of asbestosis at necropsy, a bronchial cancer in a man exposed to asbestos is unlikely to be due to asbestos.

Neuberger M Kundi M Friedl HP Haider M
Exposure to asbestos, smoking and lung cancer (German) Proceedings of the Annual Meeting of the Austrian Society of Occupational Medicine, Baumgartner E (Ed), Gentner Verlag Stuttgart 1988: 223-226.

The authors calculated that in exposures to chrysotile below 50 fibre-years in the asbestos-cement industry, there is no measurable excess risk of lung cancer.

Kipen HM Lilis R Szuki Y Valcinkas JA Selikoff IJ
Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation BJIM 1987; 44: 96-100

There was histological evidence of parenchynol lung fibrosis in all of the 138 lung tissue specimens studied histologically.

C 3850

Weiss W Smoking and pulmonary fibrosis
J of Occup Med 1988; 30: 33-39

These studies support the hypothesis that smoking causes diffuse interstital pulmonary fibrosis which is radiologically visible with low profusion in low prevalence. The x-ray manifestations may be confused with early

but:

Blanc PD, Gamsu G. Cigarette smoking and pneumoconiosis: structuring the debate.

Amer J Indust Med 1989; 16: 1-4.

The factions in this controversy are aligned into two opposing camps. One group holds that cigarette smoking alone causes radiographic opacities indistinguishable from those caused by inorganic dust inhalation (Weiss 1984, 1988; Morgan 1985). A second group accepts an association between cigarette smoking and dust inhalation on radiographic abnormalities but n to that cigarette smoking is independently capable of producing radiographic opacities that will be interpreted as compatible with pneumoconiosis when employed the International Labour Organisation (1980) ILO classification (Kilburn 1984, 1986; Castellan 1985).

Peto-J Problems in Dose-Response and Risk Assessment: The Example of Asbestos Risk Quantitation and Regulatory Policy. 19 Banbury Report, Hoel, D.G.R.A. Merrill, and F.P. Perera, Editors; Cold Spring Harbour, New York, Cold Spring Harbor Laboratory, pages 89-101,
11 references 1985.

Direct observations show that about 20 years exposure to an average 10 fibers/ml causes a substantial risk, and a lifetime exposure to 2 fibers/ml might cause a risk of one in ten in an asbestos factory. Studies in other working environments suggest lower dose specific risks.

Gardner-MJ; Winter-PD; Pannett-B; Powell-CA Follow Up Study of Workers Manufacturing Chrysotile Asbestos Cement Products BJIM, Vol. 43, No. 11, pages 726-732, 17 references. 1986

A mortality study of workers manufacturing chrysotile-asbestos cement products was conducted. Employee records of an asbestos cement facility (SIC-3292) in England for the period 1941 to 1983 were reviewed. A total of 2167 subjects were identified as having worked at the facility during that period. Company air monitoring data for 1968 to 1982 were reviewed. Each person identified in factory records was traced where possible in the National Health Service Central Registry. Vital status of the cohort, as of December 31, 1984, was determined. Death certificates were reviewed. Company monitoring data showed that since 1970 airborne asbestos concentrations were generally below 1 fiber/milliliter (f/mL; most were under 0.5 f/mL. Only a few exposures above 2f/mL were recorded. Overall mortality was less than expected, although a trend of slightly increasing mortality with increasing time of employment was noted. No excess of lung cancer was observed when compared to local or national rates. A slight correlation between lung cancer mortality and fiber concentrations existed, but the number of lung cancer deaths, 41, was too small to draw any definite conclusions. One death from mesothelioma and one listing asbestosis as an associated condition were seen; however, these occurred in subjects who worked only a short time at

elevated risk. The authors note that their results are consistent with those of two other studies showing no excess cancer risk for workers exposed to low concentration of chrysotile.

Hughes-J; Weill-H lung Cancer Risk Associated With Manufacture of Asbestos- Cement Products Biological Effects of Mineral Fibres, IARC Scientific Publication No.30, International Agency of Research on Cancer, pages 627-635, 7 references, 1980.

The authors conclude that excess risk of respiratory cancer was detected among workers of moderate and heavy total dust exposure but none was seen in workers exposed to less than 100 million particles epr cubic foot per year.

Ohlson-C-G; Hogstedt-C Lung Cancer Among Asbestos Cement Workers. A Swedish Cohort Study and a Review BMIM, Vo.42, No.6, pages 397-402, 22 references. 1985

The average cumulative exposure was estimated to be 18 fiber/year and the cohort exposure was 26,931 person/years. The authors conclude that no excess lung cancer mortality had occurred among the asbestos cement workers. This may be due to the relatively low exposure, the fact that mostly chrysotile was used in production, and the fact that the cohort contained very few cigarette smokers.

M Neuberger, M Kundi Individual asbestos exposure: smoking and mortality - a cohort study in the asbestos cement industry. BJIM 1990; 47:615-620

Deaths from lung cancer in asbestos cement workers were higher (standard mortality ratio (SMR) 1.7), but after adjustment for age and sex specific smoking habits this was not significant (SMR 1.04).

HEALTH EFFECTS OF ASBESTOS

REFERENCES:

MOSSMAN AND GEE, "ASBESTOS RELATED DISEASES", NEW ENGLAND *JOURNAL OF MEDICINE* 320(26): 1721-1730 1989

MURRAY, "ASBESTOS: A CHRONOLOGY OF ITS ORIGINS AND HEALTH EFFECTS", BRITISH JOURNAL OF INDUSTRIAL MEDICINE 47: 361-365 1990

ASBESTOS IS A FAMILY OF CRYSTALLINE HYDRATED SILICATES WITH A FIBROUS GEOMETRY

THE TERM ASBESTOS CAN BE APPLIED TO SIX CHEMICALLY AND PHYSICALLY DISTINCT TYPES OF MINERALS -

CHRYSOTILE, CROCIDOLITE, AMOSITE, ANTHOPHYLLITE, TREMOLITE AND ACTINOLITE

C 3852 CHRYSOTILE - SERPENTINE

OTHERS - AMPHIBOLE

HEALTH HAZARDS SINCE EARLY 1900s:

ASBESTOSIS RECOGNISED FIRST, BUT IS NOW LESS IMPORTANT THAN MESOTHELIOMA AND LUNG CANCER

ASBESTOSIS:

HISTORY OF SIGNIFICANT EXPOSURE - EG - 1 F/ML FOR - 10 YEARS RESULTS IN PULMONARY FIBROSIS CAUSING DYSPNOEA, COUGH, BASAL CREPITATIONS, FINGER CLUBBING, CYANOSIS, RIGHT HEART FAILURE

XRAY CHANGES MAY BE SIMILAR TO OTHER FIBROSES

LUNG FUNCTION TESTS ARE ESSENTIAL

OFTEN COMPLICATED BY SMOKING

NO TREATMENT - BUT ....

INCIDENCE OF ASBESTOSIS HIGH 1930s-1960s

- NOW RARE
- NON-OCCUPATIONAL DOES NOT OCCUR

XRAY CHANGES CHARACTERISTIC OF ASBESTOSIS ARE MORE EVIDENT IN ASBESTOS WORKERS WHO SMOKE

MESOTHELIOMA

AFFECTS THE PLEURA AND PERITONEUM

IN MEN, 49 - 70% PLEURAL MESOTHELIOMA ARE ASSOCIATED WITH POTENTIAL OCCUPATIONAL ASBESTOS EXPOSURE

VARIOUS STUDIES DEMONSTRATE DOSE - DEPENDENCY

VERY UNCOMMON DISEASE IN GENERAL POPULATION - ANNUALLY

US	130,000 LUNG CANCER AND 1500 MESOTHELIOMA
AUSTRALIA	6,600 LUNG CANCER AND 200 MESOTHELIOMA

ASYMPTOMATIC AT FIRST

MAY HAVE PLEURAL THICKENING OR INTERSTITIAL FIBROSIS

DIFFICULT TO DIAGNOSE

RANGE IN TYPE - EPITHELIAL TO SARCOMATOUS

CAN LOOK LIKE METASTATIC ADENOCARCINOMA

OFTEN, ONLY THORACOTOMY ALLOWS ACCURATE DIAGNOSIS

PROGNOSIS IS GRIM - 1 YEAR TO 3 YEARS

RADIOTHERAPY AND CHEMOTHERAPY MAY HELP

LATENCY IS 35-40 YEARS FROM FIRST EXPOSURE, REGARDLESS OF AGE AT FIRST EXPOSURE

MECHANISMS - ASBESTOS MAY BE A COMPLETE CARCINOGEN IN RODENTS

THE SEVERITY OF THE ASSOCIATION BETWEEN LUNG CANCER AND ASBESTOS EXPOSURE VARIES WITH TYPE, FIBRE MORPHOLOGY, DOSE, DURATION, SMOKING AND OTHER CO-FACTORS

LUNG CANCER IS REARE AMONG ASBESTOS WORKERS WHO DO NOT SMOKE

INTERACTION OF SMOKING AND ASBESTOS DIFFERS IN DIFFERENT OCCUPATIONAL GROUPS

ROLE OF ASBESTOSIS AND LUNG CANCER - IN WEST GERMANY AND UK, LUNG CANCER IS ATTRIBUTED TO ASBESTOS ONLY IF THERE IS EVIDENCE OF ASBESTOSIS

STILL RELY ON THE CLINICAL EVIDENCE OF ASBESTOSIS IN ASSIGNING A STATISTAICAL PROBABILITY TO THE RELATIVE CONTRIBUTION OF ASBESTOS AND SMOKING

BENIGN PLEURAL DISEASE

EFFUSIONS, PLAQUES, FIBROSIS AND ROUNDED ATELECTASIS

PLAQUES ARE CURRENTLY THE MOST COMMON MANIFESTATION OF EXPOSURE TO ASBESTOS

VARIATIONS IN PREVALENCE OF BENIGN PLEURAL REACTIONS MAY REFLECT MINERALOGIC OR MORPHOLOGIC DIFFERENCES AMONG FIBRES

IMPORTANT CHARACTERISTICS

GROWIN BODY OF INFORMATION SUGGEST THE CONCEPT THAT THEY AMPHIBOLES, CROCIDOLITE, AMOSITE AND TREMOLITE, ARE LARGELY IF NOT ENTIRELY THE CAUSE OF MALIGNANT MESOTHELIOMA IN HUMANS

LUNG CANCER AND ASBESTOSIS MAY ALSO BE ASSOCIATED MORE COMMONLY WITH THE AMPHIBOLES THAN WITH CHRYSOTILE

TEXTILE WORKERS HAVE HIGHER RISK OF LUNG CANCER THAN OTHER CHRYSOTILE COHORTS - CO-FACTORS SUCH AS SOLVENTS AND OIL SPRAYS MAY BE IMPORTANT

FIBRE SIZE - LONG THIN FIBRES MOST IMPORTANT - 8 UM LONG AND - 0.25 UM DIAMETER

NON-OCCUPATIONAL RISKS

ESTIMATING THE RISK DUE TO ASBESTOS FIBRE IS MORE COMPLICATED THAN ESTIMATING RISK FROM CHEMICAL CARCINOGENS

TYPES AND SIZE OF FIBRES ARE IMPORTANT

LINEAR DOSE-RESPONSE MODEL IS QUESTIONABLE

IMPORTANT CONSIDERATIONS ARE:

RISK OF RESPIRABILITY

AIRBORNE CONCENTRATIONS

FRIABILITY

FIBRE SIZE

TYPE OF ASBESTOS

FOR PREVENTION OFMESOTHELIOMA - CONCENTRATE ON AMPHIBOLES

FOR PREVENTION OF LUNG CANCER - CONCENTRATE ON SMOKING

RISK ESTIMATES FOR VARIOUS CAUSES - US

CAUSES	ANNUAL MORTALITY RATE (NO PER MILLION)
ASBESTOS IN SCHOOLS	0.02 - 0.37
FLOODS	2
AIRCRAFT ACCIDENTS	6
DROWNING (AGES 5-14)	27
HOME ACCIDENTS (AGES 1-14)	60
LONG TERM SMOKING	1200

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