Written evidence from Robin Howie Associates (ASB0048)

I have only recently managed to find the Transcript for the evidence presented to the Select Committee on 2nd February 2020 by Minister Chloe Smith; Sarah Albon, Chief Executive of the HSE; and, Professor Curran, Chief Scientific Adviser to the HSE.

I fully appreciate that neither the Minister nor the Chief Executive of the HSE are experts in the asbestos field, and therefore do not expect them to be au fait with the detailed technicalities in this field other than as briefed by the HSE. However, as HSE's Chief Scientific Adviser, I would expect Professor Curran to be a leading expect in this field.

The critical question is whether Prof. Curran addressed your Committee as a scientist; or as a spokesman endorsing the HSE's approaches to the control of stripping work.

Before examining Prof. Curran's evidence, I would stress that the Chief Executive's statement under Q131 that "Because of the nature of asbestos and the fact that asbestos fibres are quite heavy and they settle very quickly" is factually incorrect. This was not the fault of the Chief Executive but of those who misinformed her.

If we take amosite fibres as being the "heaviest" of the three main commercial types of asbestos fibres used in the UK and of fibrous tremolite from some products containing Quebec chrysotile, amosite fibres would be the type of asbestos fibres that will most quickly settle in still air.

From Walton (1982) about 67% of amosite product fibres longer than 5 um (i.e. fibres that would be counted by optical microscopy) have diameters <0.5 um. As the terminal falling velocity of asbestos fibres is about 3 times as great as for water droplets of the same diameter as the asbestos fibres, 67% of regulated amosite fibres will have terminal falling velocities <0.07 mm per second.

That is, such amosite fibres will remain airborne in affected buildings for a long period until diluted by air infusion into the buildings and can constitute a health risk for extended periods after being rendered airborne. Likely sedimentation rates from generally smaller diameter crocidolite and fibrous tremolite fibres will be even lower than for amosite; and the corresponding risks will be higher.

In his response to Q152 Prof. Curran commented that the Europeans based their proposed standard on the basis of exposures "where you are exposed at high levels all day for eight hours a day for 30 years continuously".

I presume that Prof. Curran was suggesting that such long-term exposures are unlikely and that the risk models should be based on shorter periods than 30 years: and at higher concentrations than proposed in the ECHA document.

The critical mesothelioma risk model used today in the UK is that of Hodgson and Darnton (2000), (H&D).

The H&D model was predicated on exposures starting at age 30 and with likely normal life expectancy to age 80. Note that exposures starting at age 30 was based on the epidemiological data on which the H&D model was predicated.

In written evidence I submitted to the Committee on Carcinogenicity (CoC) in 2012 I extended H&D's Table 9 down to first exposure at age 0 and up to survival to ages 90 and 100 to take account of current predictions from the Office of National Statistics (ONS), CoC (2013).

The CoC accepted my extrapolations down to age 0 and up to age 90 and noted that my age-related risk estimates agreed with other published data. However, I understand that HSE objected to extension down to age 0 and considered that the ONS life expectancy model was faulty. CoC therefore accepted the risk estimate down to age 5 and with life expectancy up to age 80.

As presented to CoC, I have extended Table 9 from H&D below down to first exposure at age 0 and up to starting age 45; with life expectancy to age 80:

Starting age	**0**	**5**	**10**	**15**	**20**	**25**	**30**	**35**	**40**	**45**
Age factor	**7.0**	**5.3**	**4.0**	**3.0**	**2.2**	**1.5**	**1**	**0.6**	**0.4**	**0.2**

Note, figures in red are as shown in H&D.

From the above it can be seen that a 5-year exposure starting at age 5 will generate about 5 times the risk from a equal exposure starting at age 30 and that a 5 year exposure starting at age 45 will generate about 1/5th the risk from an equal exposure starting at age 20.

Note that the risk from exposures between ages 20-24 will generate about 40% of the risk of exposures between ages 20-49 and that the risk from exposures between ages 20 and 29 will generate about 70% of the risk from exposures between 20-49.

Of even greater concern should be that pre-school and school age exposures will generate significant lifetime mesothelioma risk: even from short exposure periods. For example, a 5 year exposure to a given level between ages 5-9 generates over 5 times the risk generated the same exposure between ages 30-34 and about 25 times higher than from between ages 45-49.

That is, Prof. Curran's concern that the draft European Standard is predicated on a 30 year exposure is irrelevant, particularly so for early-in-life exposures.

In his response to Q153 Prof. Curran seems to be adamant that PMR as currently derived is that such index identified those occupations at increased risk. However, he failed to address

the validity of PMR for those in occupations with low level exposures. He comments that finding an unexposed population to do your comparisons against is very hard.

I suggest that the technique I used in the update of my REHIS paper where I used HSE data (undated), which covered mesothelioma deaths by Local Authority Areas/Districts over the period 1976-1991; a period during which diagnosis of mesothelioma should have been relatively secure and during which mesotheliomas due to low-level exposures during the post-war period would have been relatively unlikely, there were 91 Local Authority Areas with a total female population of about 3.9 million in which no mesothelioma deaths had been observed. Note that due to boundary changes between that period and today and due to some changes of District Names: particularly in Wales, the above figure of about 3.9 million females may be slightly incorrect.

For these 91 Local Authority Areas the female mesothelioma rate between 1976-1991 would have been less than about 0.02 per million per year: that is, very much lower than the figure of about 1 per million per year calculated by Tan and Warren (2011).

As all mesotheliomas reported in that document for other Districts for both male and females would probably have resulted from exposures to asbestos 20-40 years prior to diagnosis, I suggest that HSE does have early data on background mesothelioma rates if makes two assumptions: 1- that background rates in males not exposed to asbestos should be similar to background rates in females not exposed to asbestos, and, 2 - that zero mesothelioma deaths in about 3.9 million women over a period of about 16 years would reflect similarly low mesothelioma deaths in a similar number of males with either zero or very low-level exposures to asbestos.

I consider that Prof. Curran is duty-bound to explain why the above data from an HSE publication cannot be used to assess likely "background" mesothelioma rated in GB.

Prof. Curran also comments that "Mr. Howie also believes that mesothelioma takes longer to develop when the exposures are lower. …We do not agree with his position".

Prof. Curran is perfectly entitled to that position. However, I would expect him to have checked to see if that opinion is in line with the published data: including the historical data such as Newhouse and Thompson (1965).

Bianchi and Bianchi (2007) commented that "Some data indicate a reverse relationship between intensity of exposure to asbestos and length of the latency period" and that the Trieste-Monfalcone study insulation workers had 28-32 years latency, mean 29.6 years; for dock

workers latency was 25-69 years, mean 36.2 years; for shipyard workers, latency was 14-72 years, mean 49.1 years; seafarers' latency was 35-75 years, mean 55.9 years; and, among women with domestic exposure latency was 27-62 years, mean 51.4 years. These authors also cited data for the Devonport study that noted that "the trades with more heavy exposure had a mean latency period significantly shorter than trades less heavily exposed".

In addition, from the historic Newhouse and Thompson (1965) paper on mesothelioma deaths it was considered relevant to note that the interval between type (i.e. severity) of exposure and length of interval before death was shortest in 23 asbestos factory workers, 29.4 years, and longest in 11 persons with no other exposure than from living in the vicinity of the asbestos factory, 48.6 years.

Did Prof. Curran assess the validity of his above conclusion in the context of the literature?

We may disagree the literature; but we have to justify doing so.

In any event, given that the Doll and Peto (1985) report indicated that mesothelioma risk increases to the time since first exposure to the power 4, and further given that Dr. Darnton's spreadsheet for quantifying mesothelioma risk using the H&D model adopted a time to the power 4 relationship, such model implicitly gives longer latent periods for lower cumulative exposures: and vice versa.

In addition, as the Doll and Peto (1985) report can still be downloaded from the HSE website, as I have done today, I presume that HSE still accepts the validity of the Doll and Peto (1985) risk model.

Therefore, from both the literature, and indirectly from the Doll and Peto (1985) mesothelioma risk model, I consider it likely that there is generally an inverse relationship between severity of exposure and mesothelioma latent periods.

If you wish copies of any of the references or to question any of the above, please do not hesitate to contact me.

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