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Reply: Comment on 'Estimating the asbestos-related lung cancer burden from mesothelioma mortality'

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Sir,

In response to the comments of Lemen *et al* (2013) on our article (McCormack *et al*, 2012), we welcome the opportunity to endorse the original article and to demonstrate that none of the concerns raised are substantiated.

Our research was designed specifically to address the relationship between mesothelioma and asbestos-related lung cancer (ARLC) mortality, primarily in the form of a ARLC:mesothelioma ratio. This point is critical to interpreting our design and results.

Lemen *et al* (2013) express concerns pertaining to four issues: (i) studies included and omitted; (ii) a lack of consideration of further factors that affect asbestos-related cancer risks; (iii) discussions of the carcinogenicity of chrysotile; and (iv) risk mitigation. We address each of these in turn.

Our study included 68 risk estimates drawn from 55 studies. To estimate the ARLC:mesothelioma ratio, each study was required to have examined both cancer outcomes during the same follow-up

period (see inclusion criteria). Thus, the recent update of the Balangero cohort (Mirabelli *et al*, 2008) was intentionally omitted having assessed only one of the two cancer end points. We are not aware of any studies that were incorrectly omitted; all eligible studies referenced by the two Hodgson and Darnton (2000, 2010) articles were included, including the North Carolina cohort (Loomis *et al*, 2009) that prompted the risk updates. It is not appropriate to compare the studies we included to those included in a meta-analysis with a completely different aim. In our analysis, excess cancer deaths were calculated for each cohort based on observed minus expected deaths, the latter based on national/regional age- and sex-specific rates. Thus, neither the number of excess deaths nor the ratio for each cohort, as a whole, is influenced by the quality or even availability of exposure data. Hence, we had no reasons to exclude the Quebec cohort (Liddell *et al*, 1997).

Our paper emphasises that the estimated fibre-specific ratios 'characterise the overall ARLC–mesothelioma relationship across

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exposure circumstances and over a long period of time, and do not serve to precisely quantify lung cancer excess in a short time period.' Such ratios are also the most relevant when applied externally to estimate ARLCs from observed mesotheliomas, as the latter usually arise from a combination of different, often unknown, exposure histories. As pointed out by Lemen *et al* (2013) and in the devoted Discussion section ('Heterogeneity in ratio estimates within and between cohorts'), variations in the ARLC:mesothelioma ratios between cohorts or between subsets of workers within cohorts may indeed occur due to outcome misclassification, latency, exposure levels, potential confounding. Nevertheless, the best estimates of the average ratios across exposure circumstances are the ones we presented, being based on the most complete evidence-base possible.

On the carcinogenicity of chrysotile, our article clearly shows that there are both excesses of mesothelioma (four mesothelioma deaths per 1000 deaths) and lung cancer (SMR 1.7, table 3) associated with chrysotile. This is entirely consistent with the IARC classification of chrysotile as a Group 1 carcinogen to humans (IARC, 2012). At no point do we conclude that 'mesothelioma occurring in chrysotile-exposed cohorts is due to other asbestos types'; rather we considered it valid to discuss that when multiple carcinogenic fibres are present, the relevant contribution of each is more difficult to disentangle. This is particularly the case for chrysotile in the presence of amphiboles because, as concluded by the most recent meeting of the IARC Monographs, the latter appears to have a greater potency for the induction of mesothelioma than does chrysotile (IARC, 2012).

Lemen *et al* (2013) misinterpret our paper suggesting that it 'minimises the health risks posed by chrysotile'. On the contrary, we concluded the paper by emphasising the cancer risks posed by this asbestos fibre, risks that are often overlooked because they are lung cancers typically occurring in smokers. Finally, on the potential for the reduction of asbestos-related cancers, we focussed on relevant actions in two exposure groups. In currently exposed workers, removing exposure is a priority, which is consistent with WHO's position that the most efficient way to eliminate asbestos-related diseases is to stop the use of all types of asbestos (World Health Organization, 2010). Because this is not an option for formerly exposed workers, we highlighted the benefits of smoking cessation for this group. Unquestionably smoking cessation has multiple benefits for all smokers, regardless of their

current or past asbestos exposure, and at no point do we suggest otherwise.

We trust that the concerns of Lemen *et al* (2013) are sufficiently addressed herein and that the important public health message of the extent of both the mesothelioma and lung cancer burdens due to all types of asbestos fibres is clear.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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