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Lung cancer and mesothelioma risk assessment for a population environmentally exposed to asbestos

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ABSTRACT

Asbestos-related cancer risk is usually a concern restricted to occupational settings. However, recent published data on asbestos environmental concentrations in Thetford Mines, a mining city in Quebec, Canada, provided an opportunity to undertake a prospective cancer risk assessment in the general population exposed to these concentrations. Using an updated Berman and Crump dose–response model for asbestos exposure, we selected population-specific potency factors for lung cancer and mesothelioma. These factors were evaluated on the basis of population-specific cancer data attributed to the studied area's past environmental levels of asbestos. We also used more recent population-specific mortality data along with the validated potency factors to generate corresponding inhalation unit risks. These unit risks were then combined with recent environmental measurements made in the mining town to calculate estimated lifetime risk of asbestos-induced lung cancer and mesothelioma. Depending on the chosen potency factors, the lifetime mortality risks varied between 0.7 and 2.6 per 100,000 for lung cancer and between 0.7 and 2.3 per 100,000 for mesothelioma. In conclusion, the estimated lifetime cancer risk for both cancers combined is close to Health Canada's threshold for "negligible" lifetime cancer risks. However, the risks estimated are subject to several uncertainties and should be confirmed by future mortality rates attributed to present day asbestos exposure.

Introduction

Asbestos is a group of natural fibrous minerals composed of silicates that exhibit particularly interesting physicochemical properties such as flexibility and resistance to traction, heat, and chemical reactions (U.S. EPA, 1988). Because of these properties, asbestos is used commercially and incorporated into numerous products such as cement, asphalt, and brake pads (ATSDR, 2001; Lajoie et al., 2003). Asbestos fibers are divided into two large mineralogical groups: amphiboles, which include crocidolite, amosite, tremolite, actinolite and anthophyllite; and serpentines, which include only chrysotile (WHO, 2006).

In humans, inhalation is the predominant exposure route for asbestos, and the resulting adverse health effects are primarily associated with the respiratory system. These effects have been demonstrated mainly in workers and in laboratory animals (Berman and Crump, 2003; Lajoie et al., 2003; Nicholson, 1986), but they have also been reported in populations non-occupationally exposed to asbestos (Marinaccio et al., 2012; Rake et al., 2009; Vinikoor et al., 2010). Exposure to all types of asbestos fibers is associated with benign pleural diseases, asbestosis (in occupational settings only), lung, larynx and ovary cancer; and mesothelioma of the pleura and peritoneum (IARC, 2012; Roach et al., 2002).

For over 25 years, efforts have been made to provide valid and reliable information about asbestos-related lung cancer and mesothelioma risk in the general population exposed through outdoor and indoor air (Silverstein et al., 2009). A study relying mostly on indirect exposure measurements was carried out in Quebec's asbestos mining area (comprising the towns of Thetford Mines, Black Lake, and Asbestos) where the mortality by lung cancer and mesothelioma were estimated for women in the general population (Camus et al., 1998, 2002). To do so, these authors used an approach developed by Nicholson (1986) for the U.S. EPA. Briefly, Nicholson (1986) characterized the risks of these asbestos-related cancers from epidemiological studies done among workers. Then, a linear dose–response relationship was assumed for lung cancer and mesothelioma respectively, and the corresponding slopes were defined as potency factors: K_L for lung cancer and K_M for mesothelioma. These potency factors were estimated independently of the type of asbestos fiber and could be applied to assess risks in asbestos-exposed populations. Since no direct airborne ambient data were available for the time period of interest (1900–1975), Camus et al. (1998, 2002) estimated the

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past cumulative exposures based on past production volumes of asbestos in the area, aerosol dispersion modeling, recent ambient air data, visible pollution recounts, residential histories and an international exposure expert panel. Then, the predicted excess of mortality by lung cancer and mesothelioma were estimated using Nicholson's K_L and K_M, and compared to the corresponding observed excess of mortality among women from the mining area. Their results suggested that the model overestimate the absolute risk of lung cancer (10-fold) (Camus et al., 1998) and mesothelioma (50-fold) (Camus et al., 2002).

Subsequently, Berman and Crump (2003, 2008a) updated Nicholson's model by integrating results from more recent epidemiological studies and updates of studies that were initially used by Nicholson. They also introduced an additional parameter in the exposure-risk model which accounts for the differences in background lung cancer rates between cases and controls. Moreover, their work enabled them to propose fiber-specific potency factors, i.e. for chrysotile and amphibole separately, while accounting for differences in fiber dimensions (Berman and Crump, 2003). This resulted in an improvement of Nicholson's model given the evidence suggesting differences in the magnitude of the cancer-related toxicity between chrysotile and amphibole, particularly for mesothelioma (Berman and Crump, 2008a,b).

The Canadian mining of chrysotile asbestos is concentrated in Thetford Mines and Asbestos regions, in the province of Quebec. It is assumed that populations living in these towns are exposed to asbestos fibers released in the environment from the mining site and tailings (Lajoie et al., 2003) and, in the case of Thetford Mines, from ore residues used throughout the town for landscaping purposes (Marier et al., 2007). In 2007, Bisson and Couture published data on concentrations of asbestos fibers in outdoor air in the town of Thetford Mines (Bisson and Couture, 2007), and Marier et al. (2007) published data on indoor asbestos levels in the same town.

The present study was prompted by the availability of recent asbestos exposure data in the town of Thetford Mines. The objectives of this study were to: (1) select and evaluate relevant potency factors from Berman and Crump (2008a); (2) estimate asbestos exposure for the general population of Thetford Mines; and (3) use the potency factors and Berman and Crump general dose–response model to assess the lifetime cancer risk for lung cancer and mesothelioma of the population of Thetford Mines.

Methods

The general approach followed involved the determination of the relevant dose–response relationship. This required the selection and evaluation, for both lung cancer and mesothelioma, of potency factors that are applicable to an asbestos risk assessment in the town of Thetford Mines, as well as the determination of corresponding lifetime inhalation unit risk. We then estimated an average lifetime exposure concentration from published data on environmental exposure to asbestos. Finally, the calculated lifetime unit risk was combined with the average lifetime exposure concentration to estimate the population's mesothelioma and lung cancer risks.

Determination of the relevant dose–response relationship

Selection of specific potency factors

When making population-specific asbestos risk assessments, an international expert panel recommended applying potency factor estimates specific to the target exposure setting, rather than general (chrysotile) potency factors estimated across several cohorts pooled together (Health Canada, 2008). Thus, the potency factors we selected are those specifically calculated by Berman and Crump (2008a) for the Quebec mining and milling cohort (Asbestos town and Thetford Mines).

For lung cancer, Berman and Crump (2008a) determined the K_L for the Quebec cohort based on data collected in asbestos mining workers from both Asbestos and Thetford Mines. We retained the best estimate (BE) as well as the upper bound (UB) of the uncertainty interval on this K_L (0.00029 and 0.0011 per unit exposure – i.e. $(\text{f/ml*year})^{-1}$). Since amphiboles have a potency to induce mesothelioma several hundred times greater than that of chrysotile (Berman and Crump, 2008a,b; Hodgson and Darnton, 2000), the potency factor for mesothelioma is very sensitive to the number of amphiboles in the total asbestos fibers counts, which is not the case for lung cancer. Since the contamination by tremolite (an amphibole) of the chrysotile ore is greater in Thetford Mines compared to Asbestos (Berman and Crump, 2008a), the BE and UB values of K_M that we retained (respectively 0.021 and 0.065×10^{-8} $(\text{f/ml*year})^{-1}$) reflect Berman and Crump (2008a)'s data for the Thetford Mines workers only.

Predictive validity of the potency factors

To evaluate the predictive validity of the selected potency factors, we followed the approach used by Camus et al. (1998, 2002) to evaluate Nicholson's K_L and K_M. Thus, we relied on the model's predictions of the number of cancer-related deaths (P) presumably attributable to non-occupational asbestos exposure in the female population of Quebec's asbestos mining area (towns of Thetford Mines and Asbestos). To compute P, we used past cumulative exposure estimates developed by Camus et al. (1998) for best exposure estimate (25 f/ml*year), as well as subjective plausible upper range (125 f/ml*year) and lower range (5 f/ml*year) values. Indeed, Camus et al. (1998) proposed this subjective plausible range to take into account the possible errors in their estimation of past environmental and household exposure. We also used the observed (O) and expected (E) number of cancer-related deaths in that same population for the time period of interest (1970–1989) (Camus et al., 1998, 2002). In his study, Camus et al. (1998) derived the expected number of cancer-related deaths from two different indicators namely, the standardized mortality ratio (SMR) and the standardized proportionate mortality ratio (SPMR). For the purpose of the current study, the latter was selected because it generates a more conservative risk assessment.

Hence, for lung cancer, the predicted relative risk (RR) for women in the studied population was calculated as follows:

$$\text{RR} = 1 + K_L \times X \quad (1)$$

where K_L is the retained potency factor for lung cancer and X is the cumulative exposure estimated by Camus et al. (1998).

Similarly to Camus et al. (1998), the K_L was multiplied by a factor of 4.2 to account for the difference in exposure duration between workers (40 h/week) and the general population (168 h/week). P was then computed as the product of the resulting RR and E, i.e. 64.5 according to Camus et al. (1998). Finally, we compared the predicted excess of deaths ($P - E$) with the excess mortality observed ($O - E$) in that same population, i.e. 6.5 as reported by Camus et al. (1998).

For mesothelioma, the outcome considered in the dose–response model refers to an absolute number of deaths. Indeed, we assumed that every case of mesothelioma leads to death and that the background incidence in an unexposed population is nil. Thus, we compared P, herein being calculated as a predicted number of incident cases of mesothelioma (I_M), with O, where O equals 10 incidental cases (Camus et al., 2002). All things being equal, we computed I_M proportionally as a function of the I_M