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COMMENT

Comment on 'Premature deaths attributed to source-specific BC emissions in six urban US regions'

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Turner *et al* [1] estimated the number of premature deaths that may be attributed to source-specific black carbon (BC) emissions in six urban US regions. Quantitative results as those reported in figure 1 or table 1 can be challenged because the findings rely on formula (1), see the section on materials and methods in [1].

The term in parentheses in formula (1) is equivalent to (RR-1)/RR where $RR = \exp(\beta C_{av,i})$ is the relative rate (risk ratio) allocated to the annual average BC concentration. Robins and Greenland [2] proved that transformations of RRs into numbers of premature deaths ('etiologic deaths') based on the attributable fraction (RR-1)/RR are potentially biased. Indeed, (RR-1)/RR has to be replaced by a factor F. Robins and Greenland [2] showed that F cannot be estimated from epidemiological data alone and that F is bounded by $(RR-1)/[RR^{RR/(RR-1)}]$ and 1 (see equation (11) in [2]). According to Turner et al [3] the RR applied is 1.06, probably an estimate for an increase of cardiopulmonary mortality per 10 μ g m⁻³ PM_{2.5} (1979–1983) taken from table 3 in Krewski et al [4]. Given RR = 1.06 the bounds for F are 0.021 and 1, whereas (RR-1)/RR = 0.057. It follows that the point estimates of the number of premature deaths published in Turner et al [1] suffer from a potential relative bias between -94% and +164%. We note that F may vary in this wide range across regions or with covariates. Imprecision as estimated by confidence intervals is an independent source of uncertainty that has to be considered additionally.

To illustrate the potential bias of -94% in a simple setting we assume two exchangeable cohorts, exposed and unexposed, that are studied epidemiologically without any distortions and we also assume that follow-up of both cohorts is complete. Thus, all subjects under study have died at the end of mortality follow-up. In this scenario RR = 1.06 means that the ratio of average life spans in exposed versus unexposed is 1/

1.06 = 0.943. This epidemiological finding can be generated by an accelerated failure time model that reduces the life span of every exposed person by 5.7%. In this scenario all exposed subjects are affected by exposure so that F = 100%, although (RR-1)/RR = 5.7%. The relative bias is (5.7-100)/100 = -94.3%. Detailed examples are given in Beyea and Greenland [5].

Therefore, although the 'number of premature deaths due to exposure' may appear to be straightforward and easily communicated to political and other stakeholders, it is potentially biased. To answer public health questions, 'years of life lost due to exposure' could be a more appropriate metric. Respective details are provided in Greenland and Robins [6] and Morfeld [7].

Such analytical problems are widespread and affect other publications of burden of disease projects, see Erren and Morfeld [8]. Please note that Greenland already argued that this methodological error has become a social problem [9]. Ignoring possible impacts of identified errors may impede appropriate interpretations of important research regarding environmental epidemiology and public health.

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