Environmental Health

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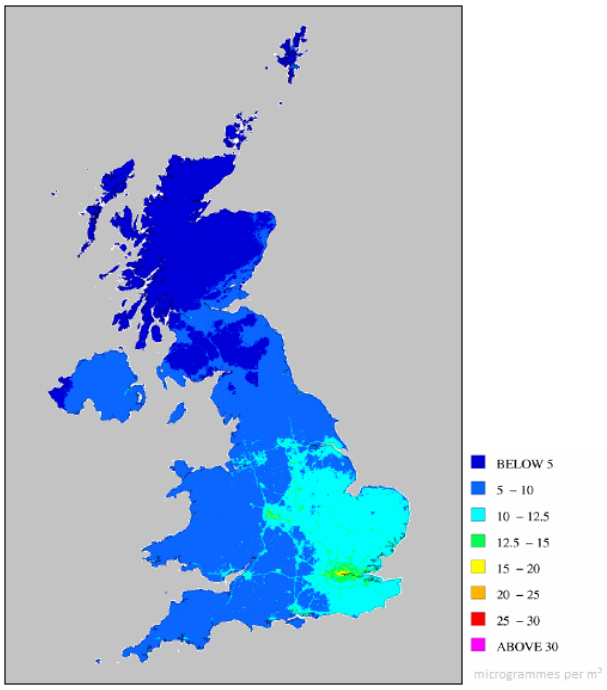
The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom

Evidence from environmental health research could eventually lead to liability exposure, based on material contribution. Of more immediate concern is the support for setting an occupational exposure standard; which would include nanoparticles.

The report is solely concerned with the effects of inhaling very small dust particles. The size range is referred to as $PM_{2.5}$ indicating that the diameter is below 2.5 micrometers. This will include a large proportion as nanoparticles. Obviously dusts can vary very significantly in composition but a broad brush measurement of mass per m^3 is used for risk estimation.

In a previous report, COMEAP estimated that for every 10 μgm^{-3} increase in $PM_{2.5}$ the lifetime mortality risk increases 6%. The average man made dust exposure in the UK is 8.9 μgm^{-3} but this varies very significantly with location:



Exposure levels in 2008

Obviously, much of the low exposure territory is thinly populated. Weighted according to population density the average UK value is 10.39 μgm^{-3} Values in London are around 14 μgm^{-3}; 90% man-made. Manchester, Birmingham, Glasgow etc. would be similar.

COMPEAM estimate that for every 1 μgm^{-3} decrease in exposure, average life expectancy (for those born in 2008) would increase 20 days. If all anthropogenic exposure was prevented, life expectancy would increase by 200 days, or by 9 months in London. For comparison, smokers; the effect on lifespan is an average reduction of life expectancy of 10 years.

Attributable deaths in the UK are currently 29 thousand (5%, out of 570 thousand) a year. Obviously people who are not carried off by air pollution will die eventually of something else; the estimate is therefore crude. The authors estimate that of those attributed to $PM_{2.5}$ air pollution on average each has died 11 years early.

For the UK, a total of about 36.5 million life-years could be gained across the population as a whole, over a period of little more than 100 years if all anthropogenic $PM_{2.5}$ is eliminated, for comparison the removal of all motor-vehicle traffic accidents would lead to a gain of about 8.1 million life-years and the elimination of the mortality risks of passive smoking to a gain of about 13.2 million life-years.

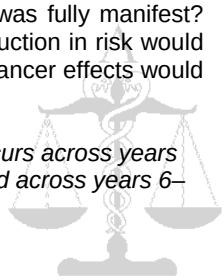
Which disease endpoints are most significant?

> *Evidence suggests that long-term exposure to particulate air pollution principally affects mortality from non-malignant cardio-respiratory causes and from lung cancer.*

Latency?

If pollution was eliminated tomorrow, how long would it take before risk reduction was fully manifest? The authors have adopted the following rationale: [for cardiovascular effects the reduction in risk would be immediate with the most pronounced effect on those with very poor health, lung cancer effects would take longer.]

> *30% of the risk reduction occurs in the first year after pollution reduction, 50% occurs across years 2–5 (i.e. 12.5% per year) and the remaining 20% of the risk reduction is distributed across years 6–20 with smoothed annual values.*



Comment
$PM_{2.5}$ exposure is diffuse in nature and as such a specific insured would be hard to identify. Even if they were identified, the likelihood is that the pollution is not sudden and accidental. However, given the developing theory of liability for diffuse effects (climate change, biodiversity) it is conceivable that liability for $PM_{2.5}$ exposure could one day be an issue for heart disease and stroke.

The work here will undoubtedly add weight to arguments about the cumulative nature of disease processes. That harm can be attributed to $PM_{2.5}$ exposure seems well established, but is the harm limited to people who are at risk for other reasons e.g. smoking, poor diet, lack of aerobic fitness? Studies of the effects of $PM_{2.5}$ may not have been fully adjusted for all known risk factors.

In the absence of specific data on individuals, courts often rely on population estimates such as those presented here. If in the alternative the court decides that quantification of risk is impossible they could assign liability on the basis of any material contribution.

It would perhaps be a vain hope that environmental $PM_{2.5}$ exposure be regarded as an independent alternative cause when considering a claim for cardiovascular disease. Studies of smoking have consistently emphasised cumulative disease mechanisms, there are very few opportunities to argue that dust would be an independent cause of disease.

Exposure at 10 μgm^{-3} has a detectable effect at the whole population level. For respirable dust ($PM_{2.5}$ is 100% respirable), occupational exposure limits are typically 2 to 4 milligrammes per m^3; i.e. several orders of magnitude higher. The chances that environmental exposure would be considered significant when compared to such occupational exposures are very small. A *de minimis* argument would be worth attempting, provided the courts accepted a rational definition of *de minimis*.

Demand for an OEL for $PM_{2.5}$ would seem to be increasingly likely as a result of this work. This would include nanoparticles of course. In the past HSE have set such exposure standards on the basis of preventing a few % lifetime risk i.e. close to the risk currently presented by ambient exposure. However, in order that it be possible to control exposure, the prescribed occupational level must be greater than ambient. Given the wide variation across the country a single standard of greater than 30 μgm^{-3} would seem to be implied.

Strategy
Insurers should engage with HSE if they are tasked with considering an OEL specific to $PM_{2.5}$.

A rational definition of *de minimis* is needed.

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