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Chapter 5

New Directions: Reducing the toxicity of vehicle exhaust

R.L. Maynard

Department of Health, Skipton House, 80 London Road, London, SE1 6LW, UK

Many studies have demonstrated associations between daily average concentrations of particles and the number of deaths and hospital admissions occurring each day. It is likely that these associations are causal. Annual average concentrations of fine particles ($PM_{2.5}$) are associated with a reduction in life expectancy (Department of Health, *Non Biological Particles and Health,* London, HMSO, 1995).

Accepting, then, that exposure to current ambient aerosols has adverse effects on health, it is reasonable to ask what can be done to reduce these effects. A first answer might be to argue simply for a reduction in ambient particle concentrations. This has been the approach taken by countries, including the UK, that have set particle standards and objectives in terms of the metrics used to monitor particle concentrations; PM_{10} for example. If it is accepted that the relationship between, say, PM_{10} and daily deaths is known and is immutable, reductions in PM_{10} will necessarily be accompanied by a predictable reduction in deaths. (The term "deaths" is used here in the sense in which the phenomenon is studied in time-series studies, i.e. deaths brought forward by an (at present) unknown period.)

Whether it is actually inevitable that a reduction in levels of a pollutant will be accompanied by a predicted reduction in effects has been questioned. The reasons for this doubt include difficulties in identifying when the benefits of reducing levels may become apparent, and the difficulties of predicting the impact of competing risks when one is reduced. Should the toxicity of the ambient aerosol change, then we should expect the relationship between particle concentrations and effects on health to also change. That this may be the case is an excellent reason for conducting further studies and for *not* assuming that a reduction in particles will be associated with a reduction in effects.

It is clearly necessary to examine closely the relationships that have been reported between daily levels of particles and effects on health. During the coal smoke smogs of London in the 1950s and early 1960s the curve relating daily concentrations of particles to health effects was less steep than curves

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describing current relationships in the UK, US and Europe. How can this be explained? It is possible that people took evasive action on high pollution days, and thus actual exposure on such days was less than predicted by concentrations recorded at monitoring sites. Also, the very high levels seen in episodes of pollution that lasted up to a week might have killed a proportion of vulnerable subjects in the first days of the episode, leaving fewer to respond on the following days. Thus, when all the data were collected there would be a surprisingly low number of deaths on many high pollution days. But it is also possible that the toxicity of the ambient aerosol was lower in the 1950s than it is today.

One possible explanation for such a change in toxicity might be that the size of particles in the ambient aerosol has decreased. This is difficult to prove. Early studies showed that the number median diameter of the London aerosol in the early 1960s was 90 nm and many small particles were present. Aggregation of very small particles may have been aided by high concentrations of larger particles during intense episodes of air pollution.

The slope of the lines relating daily concentrations of particles and daily deaths in the US tend to be steeper than those reported in European studies. Again the possibility exists that US ambient aerosol is more toxic than that in Europe. It is also possible that in the eastern US, at least, the high acidity of the aerosol plays an important role. It has been reported that the slope of the line relating $PM_{2.5}$ to effects on health is steeper than that for PM_{10}, and that the association between $PM_{10-2.5}$ and health effects is less strong than the association between $PM_{2.5}$ and health effects. This suggests that some of the larger particles making up PM_{10} are acting as a more-or-less inert diluent, and that we should be searching in the $PM_{2.5}$ fraction for the biologically active components of the ambient aerosol.

Despite the convincing epidemiological studies, there is the nagging suspicion that the mass of particles deposited in the lung per day is too small to explain the reported effects. This could mean that the mass concentration of particles in the air is acting as a surrogate for something else that is toxicologically active. An obvious suggestion is some pollutant gas that happens to vary with particle concentration. Several gases have been looked at, and for none is the evidence fully convincing. (Carbon monoxide is a possible exception with regard to effects on the heart.) An alternative explanation is that effects are related to the number of very small particles present (Seaton et al., Lancet 345, 176–178, 1995). Should this number vary linearly with mass measurements of aerosol, e.g., PM_{10}, then clearly PM_{10} would appear to be related to effects.

Evidence to support the idea that very small particles may be playing a role has recently appeared. Peters et al. (*American Journal of Respiratory and Critical Care Medicine* 155, 1376–1383, 1997) have shown that some indices of

ill-health may be related to the number concentration of very small particles. Toxicological studies of compounds such as TiO_2, $A1_2O_3$, and carbon black show that such compounds are much more active on a "per unit mass" basis when presented to animals and in vitro (e.g., cellular) test systems as ultrafine particles, than as particles of 250–500 nm diameter. Ultrafine particles deposit efficiently in the deep (alveolar) region of the lung. Alveolar deposition peaks at about 60% of inhaled particles of about 20–50 nm diameter. Particles of <20 nm are mainly deposited in the upper airways and do not reach the alveoli. On a "per unit mass" basis the total surface area of ultrafine particles is much greater than that of an aerosol of larger particles, i.e. the specific surface area is greater.

Motor vehicles make a large contribution to the primary aerosol in urban areas. Let us accept for the moment that automobile emissions of particles are contributing significantly to that component of the ambient aerosol that is linked with effects on health. Reducing the mass of particles emitted is an obvious first step towards reducing effects. This approach rests on the assumption that the toxicologically active components of the emissions will fall in line with the mass. More importantly, we should ask how the toxicity of the contribution made by motor vehicles to the ambient aerosol could be reduced. We may argue that to exert an effect the particles must find their way deep into the lung and be deposited there. It is possible that comparatively large particles depositing in the upper airways have an effect, perhaps by a reflex producing secondary effects deeper in the lung, but this will not be pursued here. Reduction of deposition of particles in the deep lung could be achieved in two ways. Firstly, if the particles were exceedingly small (<20 nm), and did not agglomerate, they would exhibit peak deposition in the upper airways. Secondly, if the particles could be made larger this would reduce deposition in the deep lung.

The first option seems to me likely to be very difficult to achieve. The second, however, seems to offer some chance of success. The deposition efficiency of 700 nm particles in the alveolar part of the lung is only about 10%, as compared with 40% for 50 nm particles. Thus, shifting the size distribution of the particles could significantly reduce the mass of particles deposited in the lung. Packaging the same mass of particles as 700 nm particles as compared with 50 nm particles would also greatly reduce the total number of particles deposited. It is clearly also important to examine the specific toxicity of various size fractions of the particles emitted. No data on the differential toxicity of various size fractions of vehicle exhaust particles are currently available. If different size fractions of motor vehicle-generated particles could be prepared then this would enable such studies. Particle composition may also be an important factor.

In conclusion, a properly designed study of the toxicity of different size fractions of vehicle-generated particles, and of their chemical components, would aid in the rational planning of strategies to reduce the effects of such particles on health. Reducing the total mass emitted, and increasing the size of the particles, are obvious first steps, but should be supported by experimental studies.

The views expressed in this paper are those of the author and should not be taken as those of the UK Department of Health.