

EDITORIAL

Asthma and urban air pollution

The paper by Wong *et al.* [1] published in this issue of the journal adds to the weight of epidemiological evidence that shows that present day levels of air pollution significantly affect patients suffering from asthma. To many people, the idea that such effects would occur seems an obvious one—indeed, almost self-evident. Despite this, the subject has generated much debate; we might ask why.

It is important to distinguish between the possible effects of air pollutants on the prevalence of asthma and on the clinical condition of patients suffering from the disease. The evidence for the latter is stronger than that for the former. It is also important to recall that ambient air contains a range of pollutants, including gases such as ozone and sulphur dioxide and particles of biological and non-biological origin. The composition of the mixture varies qualitatively and quantitatively with time and with location and thus it would be unwise, for example, to infer from data showing that an air pollution episode in Donora in the USA in 1948 [2] had a significant effect on asthmatic patients, that current day air pollution in, say, Edinburgh, also has such an effect. Or, to take a more recent example, to use data collected in Ontario [3–5] to predict effects in Vancouver [6]: in Ontario an association between daily concentrations of ozone and admissions to hospital for treatment of asthma was found; in Vancouver no such association was recorded. In Ontario, ozone may not have been the air pollutant that actually contributed to the day-to-day fluctuations in admissions in hospital: acidic sulphates are, perhaps, a more likely candidate. Studies producing evidence of associations between variations in daily concentrations of air pollutants and the clinical state of asthma sufferers are plentiful, but so are studies that do not show such associations [7]. Why is this? It may be that, in some areas, concentrations of pollutants are too low to produce effects, or it may be that the key pollutant or pollutants are absent from the pollution mixture. Also it may be that we are dealing with a small effect and uncontrolled or insufficiently controlled confounding factors disguise that effect. Accepting an individual study as providing definitive evidence for or against an effect of air pollutants on asthma is unwise. In London, for example, a study conducted during one period showed a statistically significant association between daily concentrations of

ozone and daily mortality, whilst a study conducted in another period did not [8,9]. Results that cannot be generalized are of limited scientific value and this has led some workers to turn back to well controlled laboratory studies in an attempt to understand more clearly the effects of air pollutants on patients with asthma.

Here the findings are clearer and more reproducible. Patients with asthma are, unsurprisingly, more sensitive to air pollutants that have an irritant effect on the airways than normal individuals [10–12]. Of the common air pollutants this increased sensitivity is usually said to be most clearly seen in the case of sulphur dioxide [13,14] and acidic aerosols, though in fact, data from normal individuals overlap with those collected from asthmatic subjects in the case of acid aerosols [11]. Koenig has shown that asthmatic adolescents respond to low ($100 \mu\text{g}/\text{m}^3$) concentrations of sulphuric acid aerosol [15]. It is interesting to note that in a similar study, Aris failed to show any effects at $3000 \mu\text{g}/\text{m}^3$ sulphuric acid vapour [16]. The former finding was stressed by Amdur in support of her conjecture, based on studies in guinea pigs, that acidic aerosols were an important active component of the US air pollution mixture [17]. The idea that acid may play a leading role in producing at least some of the effects attributed to air pollution has also been stressed by Thurston *et al.* [18]. If we accept that sulphur dioxide and acid have significant effects on asthmatic subjects we should expect asthmatics to have suffered badly during the London air pollution episode of 1952. However, there is little in the official report [19] to show that this was the case and there is some evidence to show that asthmatics suffered few ill effects [20]. The finding of sparing of children suffering from asthma was repeated in 1962 [21]. How odd! Peak hourly sulphur dioxide levels exceeded $5000 \mu\text{g}/\text{m}^3$ in 1952: at such levels marked bronchoconstriction amongst many asthmatics would be expected. Even the limited epidemiological techniques used at the time should have shown effects. Laboratory studies fail to show any effects on asthmatic subjects at concentrations of sulphur dioxide commonly recorded in the UK and many other countries today. Despite this, time-series studies such as that by Wong *et al.* [1] show associations between indices of the clinical status of asthmatics and low daily average concentrations of sulphur dioxide. This, again, is odd, and though explanations focusing on peak vs. average concentrations, personal exposure vs. monitored ambient

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concentrations, local concentration hot spots and the wider range of sensitivity amongst the asthmatic population than amongst groups studied in the laboratory can be produced, a nagging sense of doubt persists. The questions: 'why did the high concentrations of sulphur dioxide in the 1950s produce so few effects?' and 'why do the comparatively very low concentrations of today produce any effects?' remain. Despite these difficulties, an association between concentrations of at least some air pollutants and the clinical status of asthmatic patients remains plausible.

When we turn to the effect of air pollutants on the prevalence of asthma the picture is less clear. If we set to one side biological air pollutants such as pollen fragments and particles of dust mite faeces which contain allergens to which atopic individuals may become sensitized, we might postulate a possible adjuvant effect of air pollutants. Such an effect is inherently plausible and is supported by animal studies [22,23]. That such an effect might occur in man is supported by studies showing an increased prevalence of sensitivity to cedar pollen near busy highways in Japan [24,25] and by the studies comparing the prevalence of allergic diseases between cities and towns in what were East and West Germany [26–30], and by the recent study by Wyler *et al.* [31]. Interestingly, the German studies showed a greater differential with regard to hay fever than asthma. The inference that effects were due to traffic-generated air pollution was drawn. A number of studies have sought to show that the prevalence of asthma, reflected as wheezing, is related to the proximity of peoples' residence to roads [32–38]. Evidence against an effect, at least against a major effect, also exists: asthma prevalence is notably low in some highly polluted regions and high in some comparatively unpolluted areas [39]. The evidence linking asthma prevalence and proximity to traffic is difficult to reconcile with the lack of a clear urban–rural gradient in asthma (wheezing illness) [40,41]. All inferences regarding the possible effect of air pollutants on the prevalence of asthma have to be drawn against the background of the rapid increase in asthma prevalence that has occurred during the past 20–30 years: that this increase is largely due to air pollution seems unlikely.

Where then does the evidence leave us? As regards provocation of a worsening clinical state the evidence seems fairly clear: air pollutants can play a role. As regards induction of asthma it seems at least plausible that air pollutants can play a part as adjuvants. This effect may be related more closely to traffic-generated pollutants than to pollutants produced by the combustion of coal, oil and coke. It is not at all clear that reducing levels of air pollutants will cause the prevalence of asthma to fall, though a small improvement in the clinical status of asthma sufferers in areas previously heavily polluted by traffic may occur. Further research on the interaction between exposure

to air pollutants, dietary factors and sensitization to allergens may allow better advice to be provided to individuals at increased risk, due to genetic factors, of developing asthma.

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