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EDITORIAL

Asthma, rhinitis and air pollution: is traffic to blame?

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This issue of the *European Respiratory Journal* contains two papers that contribute to a growing body of evidence incriminating traffic fumes in respiratory disease. NICOLAI *et al.* [1] report a cross-sectional study that finds significant associations between traffic counts and exposure to traffic-related air pollution on the one hand and current asthma, wheeze and cough on the other. In a sub-group of children exposed to environmental tobacco smoke, traffic counts are also related to allergic sensitisation. LEE *et al.* [2] report a large questionnaire survey from Taiwan, in which a composite measure of exposure to traffic-related air pollution is found to be associated with physician-diagnosed allergic rhinitis. A parallel analysis reported previously [3] found a similar association between traffic-related air pollution and physician-diagnosed as well as questionnaire-reported asthma.

How do these studies relate to earlier work on air pollution and childhood respiratory illness? How do they relate to earlier work on traffic-related pollution?

Large-scale, cross-sectional studies on air pollution and respiratory outcomes in children have been reported from the USA [4], Switzerland [5], Canada [6] and Austria [7]. The US and Swiss studies found associations between some air pollutants (especially fine particles) and cough and bronchitis symptoms, the Canadian study found no relationships and the Austrian study documented associations between nitrogen dioxide (NO_2; described as a marker for traffic-related air pollution) and asthma, wheeze and cough. East/West comparisons have generally highlighted high bronchitis and cough prevalence in the East, which were ascribed to "classical" pollution, consisting of sulphur dioxide (SO_2) and particles [8], with higher rhinitis in the West. A recent study from California, USA, found an association between wheeze prevalence and the air pollution components, acid and NO_2. Taken together, these findings suggest that society is witnessing a transition from classical pollution, dominated by SO_2 and particles generated by coal and oil combustion, with the effects primarily on cough and bronchitis, to pollution mixtures dominated by traffic exhausts represented by NO_2, with effects on wheeze and perhaps asthma prevalence.

It is important to look at what precisely is the definition of asthma in some of these studies. The paper by NICOLAI *et al.* [1] defines asthma as a report by parents that a doctor has diagnosed asthma at least once or that a doctor has diagnosed asthmatic, spastic or obstructive bronchitis more than once. This definition therefore includes, to some extent, symptoms that may be bronchitic rather than asthmatic. "Current asthma" is then defined as a combination of asthma and wheeze symptoms occurring in the past year. The study performed by GUO *et al.* [3] in Taiwan used two definitions: the first was the parental report of a doctor's diagnosis of asthma at any point throughout life; the second reported dyspnoea and nocturnal dyspnoea associated with wheezing and/or attacks of dyspnoea with wheezing and/or physician-diagnosed asthma. Although both studies used the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire to collect symptom and diagnosis data, the construction of the asthma variables used in the analysis was different, so that a direct comparison becomes difficult. The study by NICOLAI *et al.* [1] is one of the few in the literature that has investigated bronchial hyperresponsiveness in relation to air pollution, only to find that there was no association. Another ISAAC study, conducted in France, found that "wheeze in the last 12 months" and "asthma ever" were related to ozone but not SO_2 and NO_2 in a simple regression analysis. However, all associations disappeared after adjustment for a family history of asthma, early childhood respiratory disease and socioeconomical status [9]. Surely, the worldwide variation in the prevalence of asthma is so large that it seems unlikely that traffic-related air pollution is a major determinant of this variation [10]. Nevertheless, it seems the respiratory arena is gradually seeing more data suggesting that asthma prevalence may, to some extent, be determined by air pollution, especially traffic-related air pollution, and that it may not just be a factor triggering attacks in patients with developed asthma. A recent longitudinal report suggesting that children exercising in a high ozone area developed more asthma is another piece of evidence that makes the respiratory field more hesitant to say that air pollution does not induce new asthma cases [11].

The new studies from Taiwan add further observations on air pollution and allergic rhinitis to previous findings. Surprisingly few air pollution studies have addressed allergic rhinitis as an endpoint. A report from Leipzig, Germany [12], investigated upper respiratory symptoms, including runny nose, cough and hoarseness and found these to be related to high SO_2 levels and intermediate particulate matter (PM) and NO_x ($NO+NO_2$) levels. The French ISAAC study found no relationship whatsoever between air pollution and allergic rhinitis [9]. Other reports show that rhinoconjunctivitis symptoms are increased with higher concentrations of ozone and NO_2, and, to a lesser extent, PM10 [13], and that daily consultations, with a general practitioner for allergic rhinitis, increases with ozone and SO_2 [14]. It is clear that more studies are needed on air pollution and allergic rhinitis.

How could traffic-related air pollution influence asthma and allergic rhinitis? Experimental evidence obtained in studies on human volunteers, animals and *in vitro* test systems, suggest that diesel exhaust particles have the capability to: 1) enhance immunological responses to allergens; and 2) elicit inflammatory reactions in the airways at relatively low concentrations and short exposure durations [15–22]. The promoting role of NO_2 in the allergen response has also been reported, but only in a few studies on asthmatics [23]. It is

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difficult, in observational studies, to separate effects of pollutants from different types of vehicles. Questionnaire studies do suggest that perhaps heavy traffic powered by diesel engines is more harmful when compared with light traffic powered by gasoline engines [24–26]. Work undertaken in the Netherlands that was able to use objective traffic counts as exposure metrics suggested the same [27–29].

The use of geographical information systems to obtain more accurate measures of exposure to traffic-related air pollution, as in the study by NICOLAI *et al.* [1], has increased. The power of such systems was well illustrated by two subsequent analyses from Nottingham, UK. The first found no relationship between traffic activity and wheeze in school children when analysing traffic activity in the living area in tertiles [30]. When the same material was analysed for children living within short distances of major roads, a clear relationship with wheeze was observed [31]. Similarly, the use of data on home location with respect to roads and traffic density on those roads resulted in observations of significant relationship with: 1) respiratory hospital admission rates in Toronto, Canada [32]; 2) rates of childhood asthma hospitalisation in New York, USA [33]; and 3) childhood asthma medical care visits in San Diego County, USA [34]. These and other studies suggest that improvement of accuracy and precision of exposure classification helps to detect associations between adverse respiratory outcomes in children and, in a few studies, adults.

The Taiwan studies [2, 3] have used factor analysis to develop one indicator variable to characterise traffic-related air pollution. Not surprisingly, the primary pollutants, carbon monoxide and NO_x, contribute strongly and positively to this factor. However, at the same time, ozone has a negative loading, most likely related to the well-known fact that ozone concentrations are low in areas where primary emission concentrations are high. The interpretation then becomes complicated. Surely the associations found should not be interpreted as showing a protective effect of ozone but rather as suggesting an important role for primary combustion products from traffic. There are only a few other studies that have used pollution factors, rather than single components, as exposure variables. In one example, factor analysis was used to estimate the contribution of various sources to ambient $PM_{2.5}$ concentrations [35]. $PM_{2.5}$ from motor vehicles and coal combustion, but not from crustal sources, was found to be related to daily mortality in that particular case.

The two studies published in this issue of the *European Respiratory Journal* not only contribute to the present knowledge of the effects of traffic-related pollution, but also show new direction for exposure assessment methods that may help to improve traffic studies in the future.

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