

*WHO Monograph<sup>1</sup>*

## **The effects of air pollution on children's health and development: a review of the evidence**

*Authors:*

Blanka Binková, Martin Bobak, Anwesh Chatterjee, Anoop J. Chauhan, Jan Dejmek, Douglas W. Dockery, Mark Everard, Francesco Forastiere, Frank Gilliland, Stephen Holgate, Sebastian Johnston, Michal Krzyzanowski, Birgit Kuna-Dibbert, Robert Maynard, Ole Raaschou-Nielsen, Jonathan Samet, Juergen Schneider, Patrick J. Skerrett, Radim J. Šrám, Dafydd Walters, Stephan K Weiland, Gerhard Winneke

*Contributors and reviewers:*

Hugh Ross Anderson, Tom Bellander, Joseph D. Brain, Bert Brunekreef, Erik Dybing, Tony Fletcher, Klea Katsouyanni, Bernd Seifert, Peter van den Hazel

### **Executive Summary**

The accumulated evidence indicates that children's health is adversely affected by the air pollution levels currently experienced in Europe. This report reviews and summarizes the results of the most recent research and presents an assessment and evaluation of the strength of evidence for different health outcomes.

This review has been conducted by the World Health Organization (WHO) as part of the "Systematic review of health aspects of air pollution in Europe" project, in support of air pollution policy development in Europe, and in particular of the European Commission's Clean Air for Europe (CAFE) programme. WHO invited experts to prepare synthesis papers of the epidemiological and toxicological literature published, for the most part, during the last decade. These were externally reviewed and subsequently discussed at a Working Group meeting. The meeting provided a consensus assessment of the strength of the evidence concerning the links between various health outcomes and air pollution. The review considered factors affecting children's susceptibility to air pollution, the effects on pregnancy outcomes, infant and childhood mortality, lung function development, asthma and allergies, neurobehavioral development and childhood cancer. The authors were asked to provide conclusions as to the likely causality of observed associations with air pollution and a multilevel scale was given: 1) evidence sufficient to infer causality; 2)

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<sup>1</sup> The monograph will be published in the Autumn 2004. For further information please contact M. Krzyzanowski, WHO/ECEH Bonn Office, [info@ecehbonn.euro.who.int](mailto:info@ecehbonn.euro.who.int)

evidence suggestive of causality; 3) evidence insufficient to infer causality; and 4) evidence showing no association.

The special vulnerability and susceptibility of children in respect to air pollution exposure are related to several differences between children and adults: the ongoing process of lung growth and development, incomplete metabolic systems, immature host defences and high rates of infection by respiratory pathogens. Furthermore, activity patterns specific to children can lead to higher exposure to air pollution and higher doses of pollutants reaching the lungs. The efficiency of detoxification systems exhibits a time-dependent pattern during pre- and post-natal lung development that in part accounts for increased susceptibility of young children to pollutants at critical time points.

The review highlights concern about the longer-term implications of lung injury during childhood. Exposure of the developing lung to air pollution reduces the maximal functional capacity achieved as the child enters adulthood, and thus reduces the functional reserve. This could lead to enhanced susceptibility during adult years to the effects of ageing and infection as well as to other pollutants, such as tobacco smoke and occupational exposures.

Some children are more susceptible than others. Individuals with underlying chronic lung disease, particularly asthma, are potentially at greater risk than children not having such conditions. Polymorphic variation in genes involved in protecting against tissue injury or regulating tissue repair may explain some of the variation in individual susceptibility to the adverse health effects of pollutants. Furthermore, patterns of exposure to indoor pollutants vary among children and those receiving higher exposures indoors, for example from tobacco smoke, are at greater risk of being affected by outdoor pollutants.

There is now substantial evidence concerning the adverse effects of air pollution on different pregnancy outcomes and infant health. The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the post-neonatal period. For the association of birth weight with air pollution, the evidence is suggestive of causality although further studies are needed. For pre-term births and intrauterine growth retardation, the current evidence is insufficient to infer a causal relationship. Molecular epidemiological studies suggest possible biological mechanisms for the effect on birth weight, premature birth and intrauterine growth retardation, and support the view that the relation between pollution and these pregnancy outcomes is genuine. For birth defects, the evidence so far is insufficient to draw firm conclusions. In terms of exposure to specific pollutants, evidence is strongest for the relationships between particulates with infant deaths. Otherwise, the existing evidence does not allow precise identification of the specific pollutants and the timing of exposure that can result in adverse pregnancy outcomes.

Evidence is sufficient to infer a causal relationship between exposure to ambient air pollutants and adverse effects on lung function development. Both reversible deficits of lung function as well as chronically decreased lung growth rates and lower lung function levels are associated with the exposure to air pollution, with clearer relationships for particulates and traffic related air pollution (indicated by nitrogen dioxide). Findings of various population-based studies are supported by animal exposure studies, indicating that intrauterine as well as postnatal exposures to pollutants can lead to impaired lung growth.

The available evidence is also sufficient to assume a causal relationship between air pollution exposure and aggravation of asthma (mainly due to exposure to particulate matter and ozone) as well as a causal link between increased prevalence and incidence of cough and bronchitis due to particulate exposure. There is little evidence for a causal association between asthma prevalence/incidence and air pollution in general, though the evidence is suggestive for a causal association between the prevalence/incidence of asthma symptoms and living in close proximity to traffic.

A significant body of evidence supports the explanation that much of the morbidity and mortality related to air pollution in children occurs via interactions with respiratory infections, which are very frequent among children. Evidence suggests a causal relationship between exposure to ambient air pollution and increased incidence of upper and lower respiratory symptoms (much of which are likely to be symptoms of infections).

Recent studies suggest that pollutants can enhance allergic sensitization in those genetically at risk, lending plausibility to the role of potential injurious effects of ambient air pollutants in the causation of paediatric lung disease including asthma. The suggested mechanisms of these effects need further research.

There is evidence of adverse effects of environmental contaminants such as certain heavy metals or persistent organic pollutants (POPs) on the development of the nervous system and behaviour in children. There is sufficient evidence for a causal relationship between exposure to lead indicated by blood-lead levels of 100 µg/l and below, and neurobehavioral deficits in children. There is evidence suggestive of a causal link between adverse health effects and exposure to mercury and to polychlorinated biphenyls (PCBs)/dioxins at current background levels in industrialised European countries. Concerning the effects of manganese, more studies are needed before reaching firm conclusions. Although inhalation is typically not the main exposure route to these contaminants, the emission of these pollutants to the air and their atmospheric transport is an important source of these pollutants.

Accumulated epidemiological evidence is insufficient to infer a causal link between childhood cancer and levels of outdoor air pollution as typically found in Europe. However, the number of available studies is limited and their results are not fully consistent. Future studies, considering exposure during different time windows from the time of child conception to the time of disease diagnosis, may help to support a clearer conclusion about the role of childhood exposures to air pollution in causation of cancers, both in child- and adulthood.

There are as yet, relatively few studies evaluating the effects of reductions in air pollution on children's health, but those that exist have shown that reduced exposure to air pollutants can lead to a decrease in hospital admissions due to respiratory causes, lower prevalence of bronchitis and respiratory infections and improvements of impaired lung function growth rates. Their results provide some direct evidence that decreasing exposures to air pollution will improve children's health.

Relative risk estimates for the health outcomes reviewed, are generally small. However, due to the widespread nature of the exposure and relatively high incidence of many of the relevant health outcomes in the population, attributable risks are high i.e. the amount of

ill-health attributable to air pollution among European children is high. More research studies are needed to disentangle the role of the specific air pollutants on health effects in children as well as these pollutants' interactions with other environmental insults such as respiratory virus infection or allergen exposure, with specific genetic factors affecting susceptibility and with diet. The studies require a careful monitoring of the environment allowing more precise exposure assessment as well as better understanding and consideration of host susceptibility.

While recognizing the need for further research, current knowledge about the health effects of air pollution is sufficient for a strong recommendation to reduce children's current exposure to air pollutants, in particular to the pollutants related to traffic. The experts who conducted this review consider that such reductions in levels of air pollution will lead to considerable children's health benefits.