# International Conference on Asthma, Allergy and the Environment

- Selected presentations

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The International Network on Children's Health, Environment and Safety, INCHES

The International Network on Children's Health, Environment and Safety, INCHES, has initiated projects that focus especially on the effects of the environment on children. INCHES web-site: http://www.inchesnetwork.org

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# Introduction

The external environment seems to have serious consequences for people with asthma and allergy. The external environment in relation to asthma and allergy is ambient air pollution, chemical substances and products. Road traffic contributes with the major part of the hazardous air pollution. In order to highlight the role and effects of the external environment on asthma and allergy, and with a special view on its effects on the vulnerable child, The Ecological Council, Denmark and The International Network on Children's Health, Environment and Safety, INCHES, decided to organise the conference on Asthma, Allergy and the Environment.

The Conference took place 25 January 2000, in Copenhagen, Denmark, and attracted 100 participants, including representatives from Croatia, Germany, Hungary, Poland, Sweden, The Russian Federation and Ukraine.

Four international speakers brought the participants up to date on the effects of air pollution on asthma and allergy, on the relative role of the environment, and on the characteristics of ambient air, including small particles. A section of the conference was devoted to analysis of policy development and mechanisms, especially for preventing asthma and allergy by improving legislation regulating the external environment. Representatives of interest groups presented proposals for policy interventions in the harmful role of external factors on asthma and allergy and on desired directions of policy development.

The role of products and especially cosmetics and other perfumed products were addressed and the mechanisms for safety regulation of these products in Europe were analysed. The role of tobacco smoke and house dust mites was not addressed at the Conference, as these themes seem to be conveniently addressed at other occasions.

The organisers have chosen to produce this report from the conference by printing the presentations by the four international Speakers: Dr. G. Bylin, Sweden, Dr. F. Horak, Austria, Dr. V. Howard, the UK and Dr. Y. Mizernitski, the Russian Federation. The presentations from these speakers have been transcribed from tapes recorded during the conference. The presenters approved the edited version of the text. Moreover we have added a revised text from September 2000, which covers the presentation made at the seminar by Mr. Christian Ege from The Danish Ecological Council.

A special publication includes the majority of the overheads (Named OH in this paper) presented by the four speakers at the conference. It has not been possible to place each overhead in the accurate place in the text, but it is our hope that the figures and tables will provide information and inspiration in addition to the texts.

It is our hope, that the views and conclusions appearing in this report will contribute to increased activity to reduce the harmful role of the external environment on people with asthma and allergy and especially with regards to protecting children's health.

Please find more information about the Ecological Council on the Internet at: http:// www.ecocouncil.dk.

The mission of the International Network on Children's Health, Environment and Safety, INCHES, is to promote healthy and supportive environments that protects the foetus and children form environmental and safety hazards. The Child has a right to health and to a safe environment, and to policy and legislation that reflects and respects the special vulnerability of the child. Please find more information about INCHES on the Internet at: http://www.inchesnetwork.org

Marie Louise Bistrup The International Network on Children's Health, Environment and Safety, INCHES Christian Ege The Danish Ecological Council Gunnar Bylin Karolinska Institute Huddinge University Hospital, Sweden

# The relationship between asthma and allergy and the external environment, especially air pollution (OH=1)

I will present two aspects of some recent studies in Sweden on the relationships between asthma and allergy and the external environment: (OH=2)

- factors in the environment that may increase the risk of inducing asthma, allergy, rhinitis and bronchitis, meaning that people acquire a disease; and
- the risk of triggering symptoms, difficulties in breathing and so on.

I will start with studies focusing on the first point, the influence of the external environment, on the risk of acquiring disease.

#### Asthma and allergy

The first study is from the Swedish National Institute of Public Health by Hans Fornfin, who has looked at studies on the frequency of asthma among children up to 16 years of age. The bottom line of both these papers is that, from the 1970 the prevalence was about 2% in both children and adults and it has increased to about 5% or 6%. I believe this is about the same in Denmark, as in many other countries. (OH=3)

#### Urban versus rural areas (OH=4)

The figure is from a study published in 1991 by two Swedish paediatricians, Bråbäck & Kälvesten, living in Sundsvall in northern Sweden. Bråbäck & Kälvesten were interested in whether living in the city is associated with an increased risk of acquiring allergy. The allergens are pollen and animal dander, and the subjects are schoolchildren. The percentage of positive prick-tests to pollen was 20% and about the same for animal dander among pupils living in urban areas. But interestingly, pupils living in the countryside, 50 km north of Sundsvall, had a significantly lower percentage of positive skin prick-tests. It was a clear, statistically significant effect.

The same authors, Bråbäck & Kälvesten, were interested in determining whether the data from the first study could be confirmed in another study. The frequency of at least one positive skin prick-test among schoolchildren, age 10-12 years, was measured in the city of Sundsvall and in the same rural area as in the previous study. There is a difference, which shows an increased risk for pupils in the city. It is interesting to note in this study that an investigation was conducted

with colleagues in Estonia, and the figures for positive skin prick-tests are much lower in Tallinn and also in Tartu. (OH=5)

Another author, Nilsson, also a paediatrician in Linköping in southern Sweden, was interested in determining whether the place of residence of children, urban versus rural areas affects the risk of acquiring asthma, allergy, atopic rhinitis and so on. Nilsson studied almost 2000 children when they were 13 or 14 years old. The new aspect in this study was that he examined the place of residence from birth and at 1 year, 2 years, 7 years and 13 or 14 years of age. (OH=6)

The place of residence the first year and the place of residence the second year is illustrated. Significant associations were found, but not for the place of residence at the 7 years of age or when the children were 13 or 14 years old. The percentage having bronchial asthma is listed. The place of residence was divided in three categories:

- rural places with less than 200 inhabitants',
- semi-urban, between 200 and 10,000 inhabitants', and
- Urban, more than 10,000 inhabitants. (OH=7)

The prevalence of bronchial asthma was only half for those children who lived the first year in rural areas compared to those who lived in an urban area. This pattern is the same also for atopic rhinitis, atopic dermatitis and any atopic disease. Here the difference is around 10% or 20%, so it is most pronounced for bronchial asthma. The same for the second year of life. Nilsson concluded that the place of living influenced the risk of getting bronchial asthma rather strongly but the effect was also observed for other atopic diseases.

A third study on this, urban versus rural areas, was published in 1998 by Eva Rönmark who works in northern Sweden. She is a nurse and defended her thesis in December 1999. She studied 3400 children 7 to 8 years old and examined several risk factors, in this case for showing a positive skin prick-test. Two variables were chosen: a family history of allergic disease, and urban versus rural living. The risk increased from 1.0 with no family history up to 1.6 with a family history of atopic disease. But interestingly, urban residence led also to a significant increased risk 1.4. (OH=8)

In these three studies the authors found an increased risk of urban residence compared to rural residence. In fact, some other studies show no effect of urban residence including some from Sweden. But I think that the data are quite convincing that urban residence can constitute a risk.

## Wheezy bronchitis

We then examine wheezy bronchitis, a disease seen in young children who are often hospitalised for difficulty in breathing. The place of the study is Stockholm. Rylander, a paediatrician, examined children from 4 months to 4 years who were hospitalised at St. Göran's hospital in Stockholm. This was Rylander's first paper, and her hypothesis was that traffic pollution influences the risk of getting wheezing bronchitis. As a marker of traffic emissions, she used, nitrogen dioxide, NO<sub>2</sub>. This paper shows the frequency of estimated NO<sub>2</sub> levels where the children lived and at the day care centres. At some of the children's residences the concentration is around 200 micro-

gram per cubic meter, as 1-hour means; but most of them had below 100 microgram per cubic meter. (OH=9)

The categories of  $NO_2$  exposure are: below 35, 35-45, 46-70 and above 70. It was a case-control study, and the risk for bronchitis for girls in the lowest category of  $NO_2$  exposure (L35) was set to 1. If you go further to the category 35-45, the risk was 1.5 for 46-70 the risk was 1.5 and the upper class 2.7. Girls had a significantly increased risk of acquiring wheezing bronchitis with increasing exposure. The same effect was not seen for boys, which is difficult to explain. (OH=10)

#### Asthma symptoms versus air pollution levels

Forsberg in Umeå was interested in the question of tracing and diagnosing symptoms and he followed 38 adult subjects with asthma, who kept a daily diary for 10 weeks. At the same time he got data for NO<sub>2</sub>, SO concentrations, temperature and humidity in the city of Landskrona. He looked for associations between exposure and asthma symptoms. He found a positive association with severe asthma symptoms and the 24-hour average NO<sub>2</sub> concentration in the city of Landskrona. This is quite a new finding to see such an association between asthma symptoms and low NO<sub>2</sub> levels. (OH=11)

The maximum 1-hour mean was 72 microgram per cubic meter in Landskrona. The subjects also monitored their own lung function, but there was no relation with the peak expiratory flow values, the lung function value. This contradiction is discussed, and Forsberg believes that the use of medicine (bronchodilators) blurs the association with exposure and the effect measured by changes in lung function. (OH=12)

## Severe breathlessness

Forsberg also studied the risk of severe breathlessness in 28 asthmatics during 61 winter days in Piteå, in northern Sweden. He found a clear association with exposure to soot, which means black particles. He divided the exposure levels in three categories: the lowest, middle and highest. He found that the risk of severe breathlessness in these asthmatics was 1.0 in the lowest category, 1.5 in the middle category, and 1.8 in the upper class. It is surprising that you can see this association at quite low levels of soot, because Piteå is considered a rather clean city in Sweden. (OH=13)

## **Hospital admissions**

Hospital admissions in Stockholm for airway diseases and cardiovascular diseases were related to air pollution for the year 1995. Preliminary data are just delivered by Bellander and co-workers in Stockholm, and they deduced data from studies in the United States on relationships between exposure to particulate  $PM_{10}$  and  $NO_2$  and hospital admissions. They calculated from these data the risk for increase in the number of admissions when the exposure level increases by  $10\mu/m^3$ . The table shows the Stockholm data on airway diseases and cardiovascular diseases and the numbers of actual admissions in Stockholm in 1995: 65,000 hospital admissions. Proceeding from the correlation taken over from the international studies, the estimated number of hospital admissions for airway diseases due to PM10 is 60, with a confidence interval from 40 to 80. There are uncertainties, of course, in these data. For airway diseases the total is 300 hospital admissions, for

cardiovascular diseases, 400. In total 700 hospital admissions were estimated to be caused by air pollution in Stockholm. Air pollution exposure has been calculated as the difference between the exposure in the City of Stockholm compared to the rural background, 50 km outside Stockholm, so this difference is used as an exposure level. It is not presumed that air pollution should be reduced to zero, but down to the rural background. (OH=14)

#### Particulate matter and mortality

Another study by Bellander is on mortality versus exposure of particulates in Stockholm in 1995. The data are analysed in the same way as in the previous slide. Exposure to PM2.5 is reduced to the rural background level. That is a difference of over  $3\mu/m^3$ . That would increase the average life expectancy of inhabitants of Stockholm by 2 months. And I at first thought, 2 months, that is not much, but it is an average for all inhabitants. A comparison may illustrate the importance: if all road accident deaths in Stockholm could be prevented, this would add 1 month to the average life expectancy in Stockholm.

## **Experiments**

I will then move to experimental data. We have performed exposure of human subjects in an environmental chamber. In such experiments you can expose subjects to low levels of a test gas and study the effects on the airways.

We have studied preferentially asthmatic subjects. In the first study we were interested in determining whether exposure to NO<sub>2</sub> at an ambient level could enhance the reactivity of the bronchi in asthmatics. The subjects were exposed for 30 minutes in a chamber to 500 microgram NO<sub>2</sub> per cubic metre. You can get these exposure conditions in large cities, in London, for instance, and in road tunnels in Stockholm, too. Then 30 minutes after exposure, 5 hours after exposure, 27 hours and 7 days after exposure a histamine inhalation test was performed, which is a method to measure the reactivity of the airways. (OH=15)

The figure shows the dose of inhaled histamine and the response, the contraction of the bronchi and the lung function measurement. Here you have a controlled experiment with filtered air and after  $NO_2$  the line was shifted somewhat to the left, more so after 5 hours. The morning after the situation was back to baseline and also after 7 days. To interpret the data, it means that these asthmatic subjects show increased bronchial reactivity 30 minutes after exposure and also 5 hours after exposure. This means that if they meet cold air or tobacco smoke, they will react more easily than in a normal state. (OH=16)

We were interested determining whether this change in reactivity could also change the reaction to inhaled allergens. This figure (OH 19) shows what happens when a person inhales allergen and gets allergic asthma. The person inhales allergen and the lung function goes down and spontaneously back to normal, and it can, as in this patient, go down again - a late asthmatic reaction after 3 or 4 hours. We were interested in determining whether NO<sub>2</sub> would affect this reaction. (OH=17)

In the study the subjects with allergic asthma were exposed to  $500 \ \mu \ NO_2 \ per \ m^3$  and also to air in a controlled experiment for 30 minutes. In the previous study we saw an effect after 5 hours. We therefore had them inhale allergen after 4.5 hours. The subjects then measured their lung function at home during the afternoon and evening (OH=18)

The results were that, in the morning, when they were exposed to  $NO_2$ , the lung function did not change. When the subject inhaled allergen, there was a decrease and then a decrease again during the afternoon and early evening. (OH=19)

After NO<sub>2</sub> this first decrease in lung function was enhanced and also the second one. For the whole group, the asthmatic reaction was enhanced after NO<sub>2</sub> compared to with air, it fell 13% in the controlled experiment and about 20% after NO<sub>2</sub>, which means that NO<sub>2</sub> can enhance an allergic asthmatic reaction, at least in this setting. (OH=19)

In the previous study I showed that a rather high dose of allergen was used. We were interested in determining whether low doses of pollen would have an effect. Such low doses of pollen are common in May in Stockholm due to birch pollen exposure. Repeated doses of allergen could also affect the allergic asthmatic reaction. In week one (a) is exposure to  $NO_2$  and allergen. In week two (b) there is exposure to  $NO_2$ , followed by allergen 4 hours later and then exposure to  $NO_2$ , 4 hours later allergen, next day,  $NO_2$ , allergen etc. for 4 days. There was also a control experiment with exposure to air and allergen. We were interested in determining whether  $NO_2$  and allergen affected lung function compared with air. (OH=21)

Air, NO<sub>2</sub> and allergen was studied for 4 days. After exposure to air and allergen: no change in lung function. But after NO<sub>2</sub> and air there was a decrease in lung function, not much, about 3%, but a consistently lower level of lung function after NO<sub>2</sub> and allergen than after air and allergen. (OH=22) The same happened 8 – 10 eight hours after allergen exposure. (OH=23)

The subjects in another study by Barck et al were also examined the morning after exposure to air,  $NO_2$  and allergen. They were examined by bronchoscopy with washing of the bronchi. You can suck up the fluid (lavage) and examine it. (OH=24)

The figure shows the number of neutrophils in the examined fluid after air and allergen and after NO<sub>2</sub> and allergen. The bottom line is that the number of neutrophils doubled after NO<sub>2</sub> and allergen compared to air and allergen. NO<sub>2</sub> enhances the allergic inflammation in the bronchi. (OH =25)

## Conclusions

I conclude from these studies in Sweden that living in an urban area, especially during the first years of life increases the risk of allergy and asthma. Which factor in urban living that is the main one is difficult to say. Air pollutants are probably a risk factor. (OH=26)

It is quite clear, on the other hand, that ambient levels of air pollutants can trigger symptoms in asthmatics, even in comparatively clean environments, as we have in Scandinavia. Ambient levels of air pollutants can also enhance an allergic asthmatic reaction. (OH=27)

Traffic is a major origin of these air pollutants and also, somewhat astonishing to me, wood burning, at least in Sweden, is a rather important cause of particle exposure, even in the area surrounding Stockholm. (OH=28)

#### Questions, comments and answers

Lis Keiding, National Institute of Public Health, Denmark: Thank you for a very interesting overview of Swedish studies. We had quite consistent results in Denmark from our national health survey. We have got increased prevalence of hay fever in the more urbanised areas and we've also got some results from ten years ago on asthmatic adults making diaries of symptoms and the use of medicine showing that there was some correlation with the levels of air pollution. But otherwise I think downloading the overview of the Danish studies, I think that we have more ongoing studies than we have got results of studies in Denmark about this source of ill health (?).

Gunnar Bylin: I saw the study from Odense and Aarhus - wasn't it published some years ago about the correlation with  $NO_2$  and also with PEF (Peak Expiratory Flow) values are highlighted in the Danish study.

Irene Tesseraux, Behörde für Arbeid, Gesundheit und Soziales, Germany: I come from Germany, from Hamburg, and you have certainly heard of the east-west comparisons for allergies and asthma and it is a fact that in East Germany before 1989 that we had less allergic diseases than in the west and now it's coming closer to the prevalence. But the air pollution in east Germany with the classical pollutants was much higher than in the western part and you focus very much experimentally on NO<sub>2</sub>, which certainly was higher in the east, too, in those times when the allergy rate was much lesser than in the west. I think what is more important are the particles. You mentioned some investigations, too, but I know it's difficult to do experimental work with particles, but do you think that particles are more important? Because we do not know if the particle pollution in West Germany, particularly with the fine particles is probably higher, even in the past, so this might be a reason.

Gunnar Bylin: I believe that particles may influence rather strongly on the situation but I do not think that you to day can conclude that that is the case and I think it is reasonable to believe, but in my mind it is not proven yet. It is very intriguing what has happened in East and West Germany and of course it is a multifactorial explanation of the increase of allergen asthma and it is not only caused by air pollution. I do not think that the NO<sub>2</sub> levels were higher in eastern Germany before, they are very related to traffic – and were traffic very high? I thought it was higher than oxidant air pollutants as NO<sub>2</sub> in the western part or more SO<sub>2</sub> and soot in the eastern part.

Irene Tesseraux: There were certainly more SO<sub>2</sub>, but I think the NO<sub>2</sub> levels were quite similar.

Friedrich Horak Jr. University Children's Hospital, Vienna, Austria

# Three-country study (Austria, France and Switzerland) of health effects of pollution from road traffic with a focus on obstructive lung disease

I will present the results of a project held between 1998 and 1999 in which we quantified the road traffic-related health costs due to air pollution in three different European countries using a common methodological framework. The project was planned as a case report for the Third Ministerial Conference on Environment and Health in London, June 1999 (www.who.dk/london99) The project was based on an interdisciplinary operation in the field of air pollution, epidemiology and economics. I worked in the epidemiological subgroup, together with Nino Künzli and Reinhard Kaiser from Basle in Switzerland, Sylvia Medina from Paris and Michael Studnicka and Gerd Oberfeld from Austria. (Künzli N et al. Public Health Impact of Outdoor and traffic-related air pollution: A tri-national European Assessment. Lancet 2000, 356; 795-801.)

We all know the positive impact of traffic on economic growth and mobility. But in recent years an increasing awareness of negative effects of transport, such as air pollution, noise and accidents, has been observed. The main part of these health effects and costs produced by traffic are not covered by the polluters themselves but rather imposed on everybody. To enable politicians to internalise such external costs, projects like this tri-national impact assessment are necessary.

## **Public health risks**

In December 1952 a fog episode in London caused about 4000 deaths. Ministry of Health. Mortality and morbidity during the London Fog of December 1952. London HMSO, 1954 (Reports on public health and medical subjects No. 95). An increase of particulate air pollution, mainly sulphur dioxide, was associated with a strong increase in death over the fog period. The main causes of deaths found were bronchitis and pneumonia, making the cause relationship of air pollution and mortality evident. Although the path of mechanisms is not clear right now, a partner in this project, Künzli, stated that public health risks may be assessed and actions successfully taken without detailed knowledge of the underlying patho-physiologic mechanisms. A very famous example for this principle can be found in history.

In 1855 one of the first epidemiologists, John Snow, suggested that drinking dirty water from the pond was the cause of cholera in London. Consequently, the closure of this pond on Broad Street successfully prevented new cases of cholera years before the causative bacteria could be detected. Similar to this example, there is strong epidemiological evidence today that air pollution causes

mortality and morbidity in our population, although the underlying patho-physiological mechanisms have not been fully explained yet.

## Effects of air pollution

A major part of our study was to assess the short-term and long-term effects of air pollution that are published in the scientific literature using time-series and cohort studies. In the last decade we can find about 100 to 200 studies that have assessed the short-term effects of air pollution. One of the studies in Austria was the Austrian Lung and Air Study (Frischer et al. Lung function growth and ambient ozone. A three years population study in school children. ASRCCM 1999; 160: 390-396), conducted in eight regions of southern Austria, that assessed the short- and long-term effects of particles less than 10 micrometer in diameter (PM10) on the lung functioning of school-children.

We found a significant negative correlation between PM10 concentration and different lung functioning parameters. We have illustrated the correlations by showing four quartiles. The first quartile shows people living in regions with relatively low PM10 concentrations. The three other areas have higher PM10 concentrations – highest in the fourth quartile. Different bars represent different lung function parameters, and there is a trend from the first towards the fourth quartile showing a decrease in lung function.

These are the multivariate analyses of our studies. We can see significant negative effects of PM10 but also for some of the other air pollutants on the different lung function parameters. This is a problem that we are always confronted in epidemiological studies: we always have a mixture of air pollutants, making it impossible to assess the effect of air pollution from just one air pollutant.

Another study being performed at the Children's Hospital in Vienna, the Austrian Project on Health Effects of Particulates, is assessing the effect of particulate air pollution on the respiratory health of pre-school children and schoolchildren in a time-series study.

In recent years, the focus of assessing air pollution impact has been changed to long-term effects. A very large study of the American Cancer Society, performed by Arnold Pope, observed more than 500,000 people in the United States. (CA Pope III et al. Particulate Air Pollution as a Predictor of Mortality in a prospective Study of U.S. Adults ASRCCM 1995; 151: 669-74) The study found a negative correlation between sulphur particles and mortality rate. Several regions were observed and the rise in sulphur particle concentration resulted in an increase in mortality rate.

When we talk about mortality, we have to discriminate between short-term effects of air pollution, accessible time series-studies and long-term effects observed on cohort studies, like that of Arnold Pope. Meta-analysis of these two kinds of studies shows us the different risk ratio. (0.7% vs. 4%). Therefore, different numbers of attributable deaths attributable to air pollution could be found. So which studies should be used? On figure (O\*H6) the whole lifetime is on the x-axis and the level of frailty on the ordinate that is finally leading to death. There are two possibilities to explain how air pollution affects our health. Either we are healthy our whole lifetime and are killed by one devastating air pollution episode, such as the cause of mortality in London in 1952. That would not hold true for the majority of us. The second possibility is that air pollution has an impact on our level of frailty for our whole lifetime, finally leading to death, which is the more valid assumption today. As time-series studies can only consider the very last part of the slope, we use long-term cohort studies for our impact assessment.

The results of the six-city studies (D.W. Dockey et al. An association between air pollution and mortality in six U.S. cities. NESM /1993; 329:1753-9.), a second long-term mortality study, are that smokers have a two-fold risk of dying and an eight-fold risk of dying from lung cancer compared with non-smokers. If you compare people living in the most polluted city versus people living in the least polluted city, there is an increase in the relative risk of 26%. So one could say that this risk of air pollution is much lower than of smoking is and consequently does not mean anything to society. But the air pollution impacts the whole society and not just the small population at a special risk.

### The methodology of the Three Country study

A major issue was the co-operation of three different study disciplines: air pollution, epidemiology and economics. The main task of the air pollution subgroup was to assess the exposure of the residential population in three countries: Austria, France and Switzerland. To do so, the population distribution map was combined with the air pollution map of each country. The results had to present a detailed register describing the number of people living in each category of air pollution concentration. The second task of the air pollution team was to estimate the road trafficrelated fraction from air pollution. This was achieved by using all available information on perceptive studies, dispersion modelling and emission inventories.

The epidemiological group used effect estimates of the international literature for relevant health outcomes and calculated the different numbers of additional mortality and morbidity cases due to air pollution.

At last, the economists had to estimate the health cost per case. To include production loss, treatment costs as well as pain and suffering, the willingness-to-pay approach was used. This approach includes all costs the individual can expect in case of disease. Summing up all health costs for all cases of morbidity and mortality, we could assess the external health costs due to air pollution.

As we could not take into account all air pollutants, we decided to concentrate on PM10, a marker for urban air pollution. Furthermore, concerning PM10, epidemiological evidence is strong and scientific literature is available to assess estimates.

An important issue for the epidemiological group was to select studies that were suitable for this project. Studies were included that published levels of particulate matter, that provide a clear exposure-response function and relative risk assessment and true and adequate study design. For

example, studies that only took two or three levels of exposure into account were omitted, as well as ecological studies that compared group effects, rather than individual based associations.

## "At-least" principle

In the selection of outcomes, the following principles was considered: first of all, for the whole study we used an "at-least" approach to finally estimate costs that are at least attributable to air pollution. To do so, we avoided health effects with clearly overlapping entities. For example, increasing hospital usage and emergency department visits that often lead to an increase in hospital usage. Or, if you can consider bronchial asthma into account, asthma attacks and the use of bronchial dilators will stand for the same outcome.

Secondly, we had to concentrate on health outcomes, which allowed financial evaluation. For example, reduction of lung function could not be taken into account, as financial assessment was impossible.

#### Selecting the health outcomes

In the figure (OH11) the left column shows the selected health outcomes. The right column represents the definition of the health outcomes used in literature that gave us the effect estimates. The following outcomes remained: long-term mortality from adults - the effect estimate for long-term mortality was taken from the two large long-term mortality studies, one of the American Cancer Society and the other of the six-city study. The second item was hospital admission. Respiratory and cardiovascular diseases defined by the International Classification of Diseases, 9<sup>th</sup> editions, the ICD-9 codes, are determined by the hospital admission diagnosis. For the hospital admission we used the effect estimate from five European studies. Chronic bronchitis incidents were defined as symptoms of cough and sputum production on most days for at least 3 months per year for 2 years or more. The estimate for new cases of chronic bronchitis was taken from one study of Abbey et al., who observed a non-smoking population of Seventh Day Adventists. We could only take into account one study to assess risk estimation for restricted activity days. Three studies could be used to estimate the risk ratio for bronchitis in children, including one Swiss study, the SCARPOL study. Concerning asthma attacks in children and adults, six studies conducted in the Netherlands, Germany and France could be included in our risk assessment.

## Cases attributable to air pollution

The next task was to assess the number of cases attributable to air pollution. The figure (OH15) shows a dose response relationship between PM10 levels and new numbers of cases for a special outcome. P zero represents the population that would experience a health outcome at an assumed baseline level of air pollution. This baseline level is set at 7.5 microgram per cubic meter, as a mean for a population group living in regions between 5 and 10 microgram per cubic metre PM10. The assumption of this level had two reasons. First, studies taken into account did not include populations living in regions with PM10 levels below 5 and 10 microgram per cubic metre. Second, this assumption was in line with our "at-least" approach, as we neglected possible health effects below 7.5 microgram per cubic metre. As we know the risk function slope from literature, we could assess the population exposed at a level of an increase of PM10 by a factor of 10. In the last step we took into account the mean population exposure, that we calculate by the air pollu-

tion group and calculated the respective numbers of people living of this level. The difference of P and Po gave us the attributable cases of the health outcomes that are due to air pollution.

The table (OH17) shows the baseline frequency of mortality in the three countries for 1 million inhabitants for 1 year. The first column presents the health outcome figures from local registers. In the second column shows the effect estimates derived from literature. This estimate accounts for an increase of mortality by 4.3%, associated with an increase of PM10 by 10 microgram per cubic metre.

The next step was a calculation of the attributable cases per 10 microgram per cubic meter PM10 using a central estimate, and the estimate from the upper and lower confidence interval. All the other health effects were calculated in the same way. For the last step we incorporated the population distribution of exposure from the air pollution group. Combining it with the epidemiological data, we could assess additional cases attributable to traffic-related air pollution for all health outcomes. The results of the impact assessment showed more than 25,000 new cases of hospital admissions and of chronic bronchitis in adults. Nearly 300,000 bronchitis episodes among children, more than half a million asthma attacks in children and adults and about 22,000 additional deaths, representing 3% of total mortality. Economic evaluation with the willingness-to-pay approach resulted in approximately EUR 306 per person per year in all three countries.

Looking into details, we can see that most of these costs are due to mortality. For example, in France, about EUR 16,000 had to be paid for additional cases of mortality but only 5749 for morbidity. The per capita costs were approximately the same.

I think we all have to concentrate on protecting our nature and demand that the politicians internalise external health costs caused by air pollution.

#### Questions, comments and answers

Christian Ege, The Ecological Council, Denmark: You said there was dose-response relationship between PM10 and health effects. Before I understood from Vyvyan Howard that there was no dose response relationship. I would like you two to discuss that.

Friedrich Horak: I think we have well enough evidence from the international literature that we have a kind of dose response relationship for PM10. We are not quite sure if it is PM10 per se that is responsible for these effects or if PM10 just stands for a mixture of air pollutants. So maybe PM10 stands for diesel particles, maybe it stands for, I do not know, iron.

Vyvyan Howard: The study is on exposure-response relationship but it is not an exact dose. Do you think the study underestimated the dose?

Friedrich Horak: We tried to underestimate impact, so in each step we had to decide which level we should go, we decided to take the at-least approach. So we definitely underestimated the effect.

Jens Steensberg, former Public Health Medical Officer: Could you say a few words about the willingness-to-pay method? It is a very interesting approach but it has been strongly criticised.

Friedrich Horak: I am not an economist and no specialist in this willingness-to-pay approach. However, the willingness-to-pay approach includes real material costs and intangible costs. I don't know if the intangible costs for pain and suffering you have for a special disease can be better taken into account with different approaches. So maybe "willingness-to-pay approach is not the best approach, but I think it was the most recent approach for this design.

Poul Larsen, The Danish Environment Protection Agency: You mentioned at the start of your speech that you estimated the impact of the traffic related particulate matter, but I think that the data we saw were for the total amount of air pollution.

Friedrich Horak: The data shown were just for traffic-related air pollution. Pollution was calculated for both situations, and then we took the amount of traffic-related air pollution and calculated it again, and the data shown rely only on data related to traffic-related mortality.

Poul Larsen: What share of the traffic compared to the total?

Friedrich Horak: It depends on the concentration where you are living, but it is about 40% to 60% of the whole air pollution; 40% is in lower levels of PM10 and 60% is in regions with high levels of PM10. So about 50/50.

Christian Ege: You showed figures for extra asthma attacks in relation to traffic exposure; did you consider whether these were attacks for people who already had asthma or if they could cause new asthmatics by road pollution?

Friedrich Horak: No, we have no epidemiological evidence yet that air pollution, and especially cause asthma per se. But we have evidence, that it can increase the number of asthma attacks.

Vyvyan Howard University of Liverpool, UK

# Environmental particulate aerosols and child health

This is not my specialised field, but I have been active in the Royal Microscopic Society and, as its president, I organised a meeting that tried to bring together all aspects of that Society, and that includes physicists, chemists, metallurgists, biologists and pathologists. The topic we lighted on was particles. It was an interesting meeting and it led to the book on the health effects of particles. I will give you some of the headlines from that book. I think it is going to be very relevant to today's discussions.

We know that if you smoke cigarettes, you might get cancer, but it is certain that you are going to damage your arterial system. This was some work done in approximately 1965 by Auerbach. He showed that, if you were a heavy smoker, more than 40 a day, the degree of arterial sclerosis increases in your coronary arteries. You are very much more likely to damage your arterial system. If you are a non-smoker, you may not get damage at all; the majority of people have a little bit of coronary artery disease – mild, but the message is very clear. You can see smokers as a group of voluntary exposees to particles because most of what smokers take in is in the form of particles. I will tell you what we know about the physical chemistry of particles and try to draw analogies with this and the diseases related to it.

Where does this aerosol that we are breathing come from? We have used more fossil fuel since 1940 than the whole rest of human history. So we rely very heavily on combustion processes. An example is of a diesel engine running, and all sorts of things affect the sort of particles that come out, but the first thing is to notice the scale. This is measured in microns ( $\mu$ m =1000 manometers), and they are in manometers. If you put the engine under load, so this is running at constant speed without a different sort of load, the level of these very small, less than 100 manometers, particles, defined as ultra-fine particles, goes up. Traffic is obviously a major source.

There are a number of standards laid down and some an EU directive with limit values 50 micrograms per cubic meter for PM10. In England they are saying they cannot achieve it. The main message here is that this is a gravimetric measure, meaning by weight, and I do not think it is going to be of very much use. We may have to go to something based on number or on surface area of particles.

This is how the United Kingdom looks for PM10 exposure. They say that a lot of it drifts across from Europe. We have mostly westerly winds, so maybe there is another explanation.

This is work by Harrison and his group. The main thing on this slide is this bottom line. In 1970 we were producing about 500,000 tonnes of PM10, estimated. That has now fallen to about 200,000. That is the trend.

We see the estimated emissions of PM10 in London. Out of the 9000 tonnes in total, by far the majority comes from road transport - 7500 tonnes.

Finally, these are the numbers of days in the year where the limit of 50 micrograms per cubic metre is exceeded. The trend is upwards – this is clearly related to traffic in London. So what are these particles and what do we know about them? This bar across here is 2 microns and these are a fraction of urban aerosol and you can see there are some very small particles, indeed.

From diesel exhaust you get different types of agglomerations, chain-like particles. The main thing is that we have very small particles here, in the 10-nanometre range.

Now we will look at the Millipore filter. I will say a little bit about the way you collect the particles, because they are very difficult to collect, because they are extremely small. Carbon black particles, about 120 manometers in diameter, are collected on a grid, which has got little holes that suck them down onto the grid.

Here are some other carbon black particles, and the way you treat them by sonication or deposition gives you a different picture than the microscope. You can actually produce different pictures just by the way you treat these things. People are really only starting to get a grip of the best way to prepare and collect materials to actually give them an idea of what is happening with these very fine particles.

This is a scanning electron microscope image by Kelly Berube, and it shows the difference in appearance between a sonicated and an impacted carbon black particle.

So there are methodological problems. If you collect particles on a cascade impactor, for instance, here these are soluble particles and you can see that the thing is predominated by solvents.

If you collect insoluble inorganic components, by a different method, you find that now the whole picture actually is predominated by iron oxide particles. It just depends on which way you collect the things - you can get a completely different picture.

This is actually urban aerosol, and it shows that there is 50% water soluble matter, about 12% insoluble carbon and about 30% water-insoluble matter, largely metal. And depending on what size range you collect, this is below 0.5 micron going up to 1.0 micron and to 3 microns, the proportions change. It is this fraction down here that we're going to think about.

So now what happens when we start to look at the different sources of the fractions? The watersoluble fraction is insoluble carbon compounds and inorganic, insoluble matter. If you just take these white ones. You see there is actually a lot of insoluble, inorganic matter here of the large size range, but, and I think this is important, there is a drop-off in this range, while here in this very small range, there is quite an appreciable amount. This is one of the major concerns - insoluble metal-type particles. If you take the same set of particles and you weight them in different ways and distributions, you find that here we are weighting the distribution by the mass and here we are weighting the distribution by the number of particles. Really, that is why at the beginning I said there's a gravimetric - 50 micrograms per cubic metre - standard, but actually the weight of this number of particles down at this end here is insignificant. There are millions of particles at this end, but the mass is almost zero. It is these that we have to worry about.

One of the most interesting talks at this particle conference was by David Jefferson and I think we need to start to learn more about what happens when you take relatively inert compounds and you atomise them down into things that just have a few hundred atoms. Because what happens is that the proportion of surface atoms to the total number of atoms there increases and as that happens, they actually take on a crystalline structure and you start to get imbalances between the number of electrons and the number of atoms. One of the ways we make heterogeneous catalysts is by making nanoparticles. Basically these things become highly chemically reactive, simply because you have made them smaller. These atoms can act as places where chemical reactions take place. There is a whole chapter on that – it is quite complicated but very illuminating. What happens is that the metal particles are the ones that give rise to free radical generation and become the most chemically reactive? The transition metals are iron, nickel, etc.

There were a number of studies on the toxicity - Roy Richards at Cardiff and Ken Donaldson at Napier College in Edinburgh. Basically, what they find is that, the smaller the particle becomes, the more toxic it becomes. The more insoluble it becomes, the more likely it is to be toxic – and the more free radical generation there is, which is associated with transitional metals. Basically, transitional metals in nanometre-size particles are bad news as far as the production of inflammation goes.

What are the effects of exposure to particles? Let us look at the work of Ken Donaldson. There are two systems for removing particles from the lung. We have evolved in a world where there are particles. There are wind-blown particles of pollen; things like that - we've got pretty good methods of removing them. Primarily, we have got a mechanism called the mucociliary escalator, which is a carpet of mucus that moves up from the lungs and then you swallow it and things land on it. Generally that will remove particles around 5-10 microns. Then if things get beyond that into the alveolar space, you've got macrophages wandering around on the alveolar surface and they can engulf things and then get up onto the mucociliary escalator, or they get internalised. If you present too many particles, then you get an overload situation where the macrophages are running around and they cannot collect everything and then things start to get engulfed or taken into the lymphatic system and can lead to fibrosis, eventually. This is the problem with these extremely small particles - they can be there in very high numerical densities but with virtually no mass - macrophages cannot actually pick them all up. The alveolar membrane separating the air from blood is only 0.2 micron so they can get taken up by pinocytosis and internalised. And those people who smoke - you can get a hit of nicotine very soon after inhaling. It is a very fast process. I think those are most of the conditions.

If you are exposed to PM10 for hours at a time over a period of years, it can lead to exacerbation of asthma and chronic constructive pulmonary disease. With fibres and insoluble particles, if you go on being exposed over a number of years, they can give rise to the chronic dust diseases that we know from the 19th century.

So now this is the work of Ken Donaldson's group. They looked at bronchial alveolar lavage and looked at the number of leukocytes in there, as you have seen before, and this has been exposed to carbon black with reasonably large particles and this is being exposed to ultra-fine carbon

black with particles below 100 manometers. You can see that, with exposure to ultra-fine particles there is an increase of production of bronchial white cells. They find changes in plasma Factor 7. Basically what they are seeing is a change - they challenge rats with these aerosols of ultra-fine particles and after about 8 hours the coagulability - the stickiness of the blood - increases. Then finally it stays elevated for about 14 days. We know that when there is poor air quality you can predict that 24 to 48 hours after that there will be an increased admission rate at the hospitals with coronary thrombosis and cerebral haemorrhage. And it is thought that this change in the coagulability of the blood that they're picking up experimentally may be involved in that – maybe bringing certain deaths forward. If you are getting ready to have one of these cardiovascular accidents, it can bring it forward. If you add to that the chronic damage that is probably going on to the arterial system from chronic exposure, those two things add together.

This was another study in the book. Here they are giving carbon black to rats. You can see as the particle size decreases (in manometers), the inflammation measured by looking at bronchial al-veolar lavage increases. So there is a clear relationship between smaller particle size and increased toxicity here.

This is again work done by Donaldson's group. Basically this is just about similar things looking at using particles, here, nickel. We see again the same inflammation. Metal particles are bad news when they are down in the nanometre size.

I would think that in a Westernised society we couldn't imagine living without combustion processes. We have to have some, for power production for some industries and for transport. We have to maybe ask ourselves, whether some processes are more toxic than others are. Do we need to be looking at the differential toxicity of certain processes and actually concentrate on those that cause the most damage? The understanding of the interaction between the chemistry and the physical properties of these very small nanoparticles is just in its infancy. It is an area, which we will see expand. I will take one example, waste incineration. The Minister for the Environment in the United Kingdom is very exercised by the fact that incineration of waste leads to the emission of various toxic pollutants and he emphasises that there is no safe dose to which we can be exposed. One of the problems toxicologists have with particle exposure is that it does not seem to be dose related. There does not seem to be any sort of linear dose relationship at all. It appears that if you are exposed to as little as 250 micrograms a day then the effects we have been talking about occur. That is why there has been quite a debate over the findings of Dockery and Pope from people in that large six-city study in the United States, where they are attributing up to 3% of the deaths, and 15% differential mortality rate between highly polluted cities and low-polluted cities.

Toxicologists say critically that no sort of dose response can be demonstrated at all. I think that it seems that the toxicity of these things is not like a chemical toxicity: It is much more based on the physical properties of the particles themselves. So the incineration plants emit toxic substances and the particulate aerosols will come from the plants – Now they have got very good gas cleaning devices that will get rid of 99% of the particle mass. But particles that are ultra-fine will tend to pass through unabated. Incineration plants are designed to produce an aerosol of high metal content, in the nano-particle range, because of what you put in. One would predict from what we have found so far in this area of research that this will be a rather toxic mixture.

Maybe one should think about what it might cost. People in the European Union have tried to put a cost on the health problems related to incineration, and it comes out about EUR 10 to 60 per tonne. If you take the chronic effects of primary and secondary particles, it increases to about twice that. If you take a large incinerator operating over a lifetime, you are talking about 3/4 of a billion Euros, if those figures are correct. That is for one incinerator burning about 400,000 tonnes of waste per year.

I do not know about this in your country, but we are seeing the cement companies rushing to say, 'we can burn waste, it is quite safe' and they are allowed 50 mg per cubic meter of gas emissions and this is something that has also happened in America.

Burning municipal waste produces  $5000 \text{ m}^3$  of gas per tonne and if you are allowed this emission of  $30 \text{ mg/m}^3$ , then you will produce 154 g per tonne of ultra-fine particles, because of the filtration system. We know how much is produced by cars. Diesel particle emission is about 0.8 g per km. So we can work out that actually operating a large incinerator is equivalent to 770 million vehicle-km a year. So it has a large impact.

Concerning child health, we have to think about the acute effects, which we have been hearing about. It can exacerbate existing conditions. These very small particles have been shown to be able to act as adjuvants. Nobody knows what that means for health, but we know that they are likely to be exposed to these during the time of imprinting of the immune system shortly after birth. I am not convinced that these particles cannot get from the mother into the foetus, and that is something we would like to investigate. If they can get across the lung, there is no reason why they cannot get across the placenta.

To conclude, I will say that we do not really know what these ultra-fine particles mean, except that they are probably bad news. I think society has to invest some money in identifying the combustion processes that are producing the most toxic aerosols and I have given you one example of a place where we might look. They are going to have to be fairly heavily controlled. Those are the first ones to go for.

#### Questions, comments and answers

Poul Bo Larsen, the Danish Environment Protection Agency: You focused very much on the soluble fraction of the particles but some of the epidemiological studies also find a very close relationship – and sometimes an even closer relationship – between the sulphate fraction and toxic effects. Do you consider that the reason is that the sulphate fraction is a surrogate for the ultrafine particles or can they have their own toxicological mechanism that can explain these findings?

Vyvyan Howard: I think that it is probably both because they will go hand in hand to some extent. Clearly sulphate particles are likely to be irritant in their own right, as well. I do not think that it is necessarily true that you will not find a positive correlation with these other particles. I think these small insoluble metal-type particles are likely to be involved more with the chronic long-term effects to things like the arterial system. Maybe sulphate particles are more concerned with more acute things, but we really do not know the answer. I think that this idea that one and the other may be positively correlated is quite possible. I do not think these other things have been looked into at any great depth in humans. They are starting to look at animal models closely. I do not know the answer to your question.

Poul Bo Larsen, The Danish Environment Protection Agency: Do you think that the emission of highly active aerosols in this occupied particles may explain the TH1 and TH2 shifts (?) and thereby the increased amount of allergies?

Vyvyan Howard: I am not an allergist, although I work with one. The answer is simply not known. I think that the general statement is that they certainly can act as adjuvants and therefore it is possible that there could be a mechanism whereby they could produce an effect. There are so many theories. One is that we are all getting too clean and we do not get exposed to enough micro bacteria and there are other theories. I think it is going to be a multi-factorial mechanism, whatever. It must be an area for further research.

Yuri L. Mizernitski Moscow Research Institute of Paediatrics and Child Surgery Russian Federation Ministry of Public Health

# Bronchial Asthma as a marker of air pollution

Bronchial asthma is one of the most frequent chronic diseases in children. As observed recently, the incidence rate of bronchial asthma increases world-wide.

The analysis of the incidence rate of bronchial asthma in Moscow children has revealed a close relation to the geography of industrial enterprises and main-line highways, the volume of traffic along streets adjacent to dwellings. In the neighbourhood of highways, the number of patients with bronchial asthma was generally 2- to 2.5-times higher than that in districts remote from the main-line highways. We have found that similar relations are common in other towns. Thus, after having children screened by pulmonologists in one of the towns near Moscow, the incidence rate of bronchial asthma has increased by the factor 3, averaged over this town, and by the factor 6 in certain of its districts. As a rule, the geography of such «unfavourable» districts is found to be in relation with that of industrial enterprises and highways (Fig.1). Thus, it appears that bronchial asthma in children is a sensitive marker of atmospheric pollution.

Why is it so? In the first place, the factors of indoor environment become more aggressive under the influence of outdoor pollution. This is due to the fact that even small amounts of xenobiotics, apparently, potentiate allergic reactions caused by the domestic, epidermal, and fungous allergens, injure the bronchopulmonary structures and respiratory tract epithelium, promote the outburst of allergy mediators from immunocompetent cells. On the other hand, the photochemical industrial smog can modify considerably the allergen effects on those with bronchial asthma, and, in the environment being far from healthy, air pollutants can aggravate the immunogenicity of pollen and other «natural allergens», which additionally contributes to the development of allergic diseases in persons featuring a pertinent genetic predisposition. At the same time, some chemicals directly exhibit the properties of allergens (or haptenes) and can sensitise children.

To estimate the contribution of chemical-allergen sensibilisation to the development of bronchial asthma in children, we, together with Prof. L.A. Dueva from the Research Institute of Occupational Medicine, Russian Academy of Medical Sciences, have examined 41 patients with bronchial asthma of various severities. Sensibilisation to environmental chromium, nickel, and formaldehyde was detected by a highly sensitive specific variant of the complement fixation test. In doing so, the change in total IgE was determined simultaneously after having tested sera with an appropriate haptene being preincubated under the conditions of dedicated specific method of invitro enzymo-immunoassay (EIA). The high rate of sensibilisation to industrial chemical allergens has been revealed in children with bronchial asthma. The antihaptene titres of high pathogenicity (1:160 and over) have been determined almost in 15 % of those examined, while the specific binding of IgE to various haptenes in-vitro has been revealed in 18 to 24 % of children (Fig.2). These reactions were higher in those children, who were atopics with high basal level of IgE (Fig.3). Such figures are typical for the clinical forms of occupational allergic diseases having a chemical aetiology and conform, in particular, with the inhalation-type provocative test results for patients exposed to an appropriate chemical allergen. This suggests that the chemical allergen sensibilisation of these children be of the pathogenic significance.

Of course, the environmental factors must be taken into account, however without overestimation. As a whole, the contribution of environmental factors to the bronchial asthma development is know to be at the level of 15 to 25 %. However, it is important to ensure the control over these factors. In modern conditions the problems of the increased incidence rate of bronchial asthma in children can be solved only with the help the large-scale environmental programs, including different aspects.

# Diesel particles – a health hazard

By: **Christian Ege**, Director, The Danish Ecological Council The pamphlet can be read and downloaded from our homepage: www.ecocouncil.dk

#### **Summary**

To all appearances small particles belong to the pollutants presenting the biggest health hazards. Particles come especially from diesel-powered vehicles. The Danish Environmental Agency has demonstrated that annually small particles apparently cause 3-400 premature deaths per million inhabitants in the cities. That is more than deaths in traffic accidents. Similar estimates came from WHO, and from the environmental authorities of several other European countries. Especially the elderly are affected. Also the small particles seem to aggravate the condition in asthma sufferers, including the many children with asthma. What makes the small particles so very dangerous is that they can enter the smallest vessels of the lungs.

There is a solution within sight to the grave health hazards created by small particles. The solution is called particle filters. But particle filters will not come automatically. They will require initiatives in the form of legislation, green taxes and subsidies. In the present leaflet The Danish Ecological Council offers a number of specific proposals on how to further the introduction of filters on diesel vehicles in Denmark, and we find that with a few modifications our proposal would also be applicable in other countries.

#### Introduction

The present leaflet was first prepared for use in a Danish context; however we decided to publish it in English since the issue of small particles from car exhausts is a general problem world-wide. We chose to retain descriptions of its political recommendations referring to a Danish context; we did however add a few circumstances that we knew to be different in other European countries. But the general idea is that interested parties in other countries can take our suggestions and proposals and adjust them to suit their own local conditions.

## Small particles – the number one traffic killer

There is every indication that small (ultra-fine) particles, mainly emitted by diesel vehicles, are among the pollutants that present the biggest health hazard. In 1997 the Danish Environmental Agency published a report indicating that annually such small particles apparently cause 3-400 premature deaths per million inhabitants in big cities. This rate is higher than e.g. deaths in traffic accidents. The figure quoted was based mainly on American studies; it has later been corroborated by WHO studies, studies in Norway, etc. Especially the elderly suffer. What is more, the small particles seem to aggravate asthma, also in the many children with this condition. What makes the small particles especially dangerous is that they can enter the smallest vessels of the lungs. Today it is widely agreed among researchers all over the world that the health implications of small particles are extremely serious. A large-scale WHO funded project has been conducted in France, Switzerland, and Austria. Some of its conclusions were that airborne pollution from traffic in the three countries altogether annually entailed some 295,000 days extra absence due to bronchitis and 560,000 additional asthma attacks, 165,000 of which in children. The principal cause appears to be small particles. A Dutch study concluded that *mean* life expectancy in city areas with small particle pollution is reduced by about 12 months due to those particles. This is quite a drastic effect compared to the calculated impacts of other environmental factors. By comparison estimates are that smokers can expect to live up to 7 years less than non-smokers do.

What is more, small particles from diesel vehicles are also a serious problem in the working environment of e.g. bus and taxi drivers, waste collectors, and those working for construction contractors.

Petrol-engined vehicles also emit small particles, though to a lesser extent. On an estimate twothirds of particles emitted by road traffic seem to come from diesel vehicles, and since they number far fewer than petrol-engined vehicles there is much to be said for prioritising particle filtering in diesel vehicles. Moreover most particles apparently come from older petrol-powered cars without a catalytic converter. Laboratory measurements have shown that the release of small particles from a petrol-engined vehicle with a catalytic converter is approx. a hundredth of that from a vehicle without one. And then the latter will emit fewer particles than a diesel vehicle. The implication is that particle emission from petrol-powered vehicles will automatically decrease in the coming years, as the proportion of vehicles with emission control systems gradually increases. Estimates are that today nearly 60 percent of Danish petrol cars have a catalytic converter, which has been mandatory for new cars registered since 1990. In most other EU countries the percentage is somewhat lower, since the statutory demand on catalytic converters only came into force in 1993; but presumably at least 50 percent of their cars have catalytic converters by now. In other words small particles from petrol cars will become a minor problem within few years. Therefore there is every reason to make a targeted effort in order to have particles removed from diesel vehicles.

#### Why filters?

Diesel vehicles are especially used for carriage of goods, while buses, taxies and private cars are of secondary importance. It is hard to imagine traffic restrictions that would in themselves be radical enough to solve the problem. Only a technical solution can bring about a drastic reduction in emissions. It is possible to make a little headway e.g. by better use of the capacity of lorries and vans. But even if a 20 or 30 percent decrease in diesel-powered transport could be achieved (which would be an extremely ambitious target) that reduction would be grossly inadequate in relation to the small particles issue. As for the diesel-powered passenger cars they could just be replaced by petrol-powered ones; however, in most EU countries, the passenger cars merely make up a small minority of the diesel-powered vehicles. In recent years there has been a dramatic surge in the market share of diesel-engined private cars. In Austria the majority of private cars sold today are diesels, due to the attached tax benefits. In environmental terms this has an inherent dilemma: A diesel engine will save fuel – 25-30 percent – which is a benefit where  $CO_2$  emissions are concerned due to the greenhouse effect. But then the particles will make it ex-

tremely hazardous to human health. This serves to further underline the fact that we need to solve the particle problem, before too many diesel cars have left the assembly line. Once the particle problem has been dealt with, the use of diesel will offer environmental benefits.

Thus, in order to achieve the required reduction of particle emissions, we have to resort to a technical solution. And such a solution actually *exists*, namely particle filters. These filters will withhold both large and small particles, after which the particles are burnt in the filter.

Yet another option is to change fuel – that is switch to gas or electric operation. However that is a lot more costly. Nor are particle filters inexpensive.

Prices are circa	
For a large van:	3,600 Euro
For a bus or medium-sized lorry:	5-6,000 Euro
For a heavy lorry:	9,000 Euro

As for private cars the change can be made at much less cost; but today filters are only offered for few models, and only for the more expensive cars. Particle filters will not come automatically. They need to be helped by legislation, green taxes and subsidies.

## Are filters effective?

It has been questioned whether particle filters actually work with the ultrafine particles– of 10-30 manometers (one billionth of a metre). In early 2000 the Danish Ministry of Transport and Odense municipality <sup>1</sup> launched a major trial of particle filters. The first report was published in May 2000. It showed that two out of the four filters tested satisfied the required 90 percent reduction – also in 10 nanometre particles – while there were problems with both the remaining filters. Other studies have also shown some filters to actually have the desired effect. The Odense trial will continue for a good while yet. In December 2000 - after 12 months' operation - the filters will undergo durability tests, to be repeated in December 2001. There is an obvious risk that the trial will be used for procrastinating all action in this area until the trial has been completed. That would be wholly unjustified. The political hearing on a taxation and subsidy mechanism might just as well take place in parallel. The scheme could then come into operation by mid-2001. Before then we will be clear about precisely which filter types can be recommended.

Both of the filters with particle cleaning problems in the Odense trial were the so-called CRT category. These filters also imply an increased release of nitric oxide. The reason is that these filters are combined with a catalytic converter. And when fitting a diesel vehicle with a catalytic converter, the only type available is one that cannot remove nitric oxides – as opposed to the so-called three-way catalytic converters mounted in petrol-powered cars. The catalytic converter of a diesel vehicle will transform nitric monoxide (NO) into nitric dioxide (NO<sub>2</sub>), which is even more toxic than NO. Still the Danish Environmental Protection Agency has declared that the health risks associated with small particles are considerably greater than those of nitric dioxide, so if a choice must be made - then go for particle removal.

<sup>&</sup>lt;sup>1</sup> Odense is Denmark's third largest city

But really such a choice is not necessary. The heartening outcome of the test is that the two filters that did well solve both problems. They fulfil the requirements for particle removal, while *not* increasing nitric dioxide. One of the filters depends on a diesel oil additive, however an iron or cerium compound that (for all we know) does not present any environmental problems or health hazards. The additive is to be added at the diesel pumps (which would require them to be modified), or the user will fill it into the petrol tank directly. The latter just amounts to what the Swedes are already doing if they need the particular additive (sodium or potassium) that has now replaced lead. They are to refuel "unleaded" petrol and themselves add an additive, since many pumps with petrol containing additives have been shut down. In the next few years the same thing will happen in Denmark.

Experiences from the Odense trial show that we need an independent testing scheme for filters, to decide which are eligible for subsidies (a point we will be coming back to).

#### Demands on vehicles and diesel oil

The EU has adopted more stringent demands on particle emissions from diesel vehicles; however they do not come into force until 2006 and apply to new vehicles only. This means that it will be quite some years before all diesel vehicles have filters fitted. Considering the serious health problem involved we cannot wait that long. The EU decides demands on vehicle emissions, and we cannot directly make demands more stringent than those of the EU. What we can do is to support the filters by means of green taxes and subsidies.

The pressure brought to bear since 1997, helped by the Danish Ecological Council, has placed the issue of small particles from traffic high up on the political agenda in Denmark. In 1998 the Danish government, supported by a couple of other parties, agreed on a compromise called the "Whitsun Package" that, among other things, included a new tax on diesel fuel with more than 50 ppm<sup>2</sup> of sulphur. This caused diesel to be replaced with low sulphur diesel (max 50 ppm) on the entire Danish market (except agriculture). This unambiguously illustrates how potent an instrument green taxes can be made to be. According to calculations low sulphur diesel should reduce particle emissions by 13 percent, while also easing the introduction of particle filters, some of which can tolerate very little sulphur. A further tightening of demands on sulphur contents (max 10 ppm) would be an advantage. Germany has decided to change their diesel tax so as to support the use of diesel with max 10 ppm sulphur, starting in 2003. It remains to be seen if Danish oil refineries can conform to such a demand without major conversions.

In August 2000, NERI (The National Environmental Research Institute)<sup>3</sup> published new streetlevel studies done in Odense and Copenhagen. The above-mentioned 13 percent reduction was calculated as a reduction in particle volume (weight), while the new studies measured *particle counts*. They showed a 50 percent reduction. The reason is that apparently the switch to low sulphur diesel has removed especially the small particles. They do not matter a lot when volume is measured, but they do when the count is. That was good news, since the smallest particles are also the most dangerous. Yet we would definitely hold that particle filters are still needed – a 50 percent reduction is not enough, given the serious nature of the health hazards.

<sup>&</sup>lt;sup>2</sup> ppm=parts per million=mg/kg

<sup>&</sup>lt;sup>3</sup> The research dept. of the Danish Ministry of Environment and Energy.

## Voluntary introduction of particle filters

Following the introduction of low sulphur diesel a smaller number of transport companies, especially among bus companies and refuse collection operators have introduced particle filters voluntarily. More specifically, the HT (Copenhagen Metropolitan Transport Company)<sup>4</sup> now impose the condition whenever new tenders are invited for bus transport contracts that all new diesel buses will have particle filters fitted, and the contractor is paid extra if filters are retro-fitted on the older buses. But in sum, given the total number of diesel vehicles in Denmark, exceedingly few have particle filters today.

All bus companies ought to make demands matching those of the HT; equally all diesel trains should be fitted with particle filters. Moreover other public institutions inviting tenders for transportation tasks (e.g. waste collection, school busses and conveyance of patients and disabled) should demand filters. Finally, this would also go for tasks carried out with diesel-powered construction machinery.

### The proposal of the Danish Ecological Council

We now propose that the Danish government and Folketing<sup>5</sup> set to work on organising a scheme with tax on diesel vehicles without particle filters, the revenue of which should be used for subsidising the fitting of particle filters. The Council has prepared a calculation example with a realistic tax that will enable a 50 percent subsidy for the installation of particle filters in 48,000 diesel vehicles annually, the equivalent of about 1/8 of all Danish diesel vehicles, or all diesel vehicles estimated to run in Copenhagen.

We therefore suggested that the government and the Folketing introduce an increase of the Danish green ownership tax<sup>6</sup> on diesel vehicles without particle filters, and to introduce subsidies for particle filters. Particle filters are encouraged further by a tax on all vehicles that do not satisfy the pending EU norms for particles (starting 2006), i.e. practically all diesel vehicles without filters. Gas-powered vehicles will normally be able to comply. More specifically we propose a rise of both the present ownership duty for diesel-powered private cars and of the present tax on motor vehicles according to weight<sup>7</sup> for lorries/vans and buses, at a total extra revenue of 155 million Euro/year. Increasing taxes with approx. 200 Euro a year for private cars, 270 for small vans and 470 for buses and lorries could do that. In the real world the levy scale would probably need more differentiation, so the indicated rates should be seen to rather reflect the order of magnitude. The duty revenue could then be used for a scheme to subsidise particle filters. The increase of duty would seem reasonable, since tax-wise the owners of diesel-powered vehicles have been given preferential treatment for quite some years. . It should also be noted that this is not a duty

<sup>&</sup>lt;sup>4</sup> HT covers Copenhagen and three neighbouring counties, with a total of about 2 million inhabitants.

<sup>&</sup>lt;sup>5</sup> The Danish parliament

<sup>&</sup>lt;sup>6</sup> An annual ownership tax on cars. It was formerly called 'weight duty', since based on vehicle weight; but in 1998 it became based on the car's fuel economy and was therefore renamed "green ownership tax", since meant to encourage the purchase of cars with a low fuel consumption.

<sup>&</sup>lt;sup>7</sup> Weight duty is still paid for heavy vehicles, as for private cars before 1998, comp. Note 6.

on diesel vehicles the revenue of which will end up in the public purse. What we suggest is solely a way of funding a solution of the particle problem.

For private cars that could equally well drive on petrol, a tax would have the positive effect of making fewer opt for diesel until diesel models with filters come on the market.

The indicated amount would enable a filter subsidy of some 50 percent, and for nearly 50,000 diesel vehicles a year – corresponding to circa every eighth diesel vehicle in Denmark, (refer to the box) We propose that both new and existing diesel vehicles are made eligible for subsidies. By way of comparison the Danish Technological Institute calculated in 1997 that 51,000 diesel-powered vehicles are running in Copenhagen. This serves to illustrate that in a matter of a few years the outlined scheme would accomplish to have filters mounted on all diesel vehicles running in big Danish cities.

It is necessary to keep a watchful eye on filter prices, so subsidies do not help maintain an overcharge.

We propose that subsidies be phased out with for instance 10 percentage points each year, thus creating an incentive for an early filter installation. Then the subsidy would be down at zero for new vehicles by 2006, when the EU demands come into force. For existing vehicles a more gradual phase-out could be adopted.

Estimated number of diesel vehicles in Denmark:		
Small vehicles, including taxis	90,000	
Vans less than 2 tons	12,000	
Lorries and vans above 2 tons	271,000	
Buses	8,000	

## **Duties and cross-frontier shopping**

Another solution would be to increase tax on diesel oil. However we need to keep in mind that in Denmark and several other countries it would be quite sensitive to cross-frontier shopping, so an increase risks producing a negative revenue. However, from a Danish viewpoint, the situation has changed for the better: Germany has adopted some gradual increases of her diesel tax, which will probably make room for a certain increase in the Danish diesel tax. Moreover, we will undoubtely see claims that an increase of the Danish weight tax will intensify the present trend that transport companies register in another country, e.g. Danish transport companies register in Luxembourg or a similar country where both duties and company taxes are low. Yet we do not believe that a tax of 470 Euro/year is of any consequence, compared with the book depreciation of heavy vehicles purchased for some 150,000 Euro in initial cost. Faced with the issue of registering abroad we should press for common minimum taxes, work environment codes etc. within the EU, so other EU countries cannot undercut and thus offer a "flag of convenience".

#### **Environmental zones**

A new Danish act that came into force on June 1, 2000 (similar laws have existed in Sweden for some years) has enabled the introduction of environmental zones in town centres. This means that municipal authorities can enforce more stringent demands on vehicles wishing to enter the most vulnerable areas, such as town centres. The big city municipalities could use this instrument for defining urban zones in which diesel vehicles are only allowed if fitted with filters. That would very specifically encourage owners of such vehicles that drive in the town centres to apply for subsidies and have filters installed.

Our proposals for a combined tax and subsidy scheme would be a good supplement to introducing environmental zones. Actually it is hardly realistic for any municipality to introduce environmental zones with mandatory particle filters, if not underpinned by a tax-cum-subsidy scheme. That would create a tremendous pressure from companies based within the zones, since their access to choosing between transporters and suppliers would be restricted.

If environmental zones are allowed to stand alone, they will merely amount to letting the state/ parliament wash their hands and pass the buck to the urban municipalities. In Denmark we have already experienced a similar situation, namely with our large shopping centres: No municipality in its right mind dared say no to such a shopping centre – if they did, they knew for certain that it would be built in the neighbouring municipality instead. The problem was solved in part with a government circular imposing tight restrictions on establishing new large shopping centres. But a tax-cum-subsidy scheme would make a potent incentive for road transport companies to have filters installed, while also making them financially feasible for them.

In other words: There is a solution within sight to the grave health hazards created by small particles. But it will require a political commitment to action.

#### Want to know more?

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