

Human health effects of aerosols and their gaseous copollutants

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1. Introduction

The Austrian Clean Air Commission (Kommission für Reinhaltung der Luft, KRL) promotes the study of ambient air pollutants and their effects on human health, formulates air quality criteria and gives recommendations for standard setting. Originally this work was limited to outdoor pollutants, however, as today most time is spent indoors (home and workplace) from which other pollutants originate, by example tobacco smoke (Moshhammer et al. 2004), asbestos (Neuberger and Vutuc 2003, Moshhammer and Neuberger 2009a), non-fibrous particulates (Moshhammer and Neuberger 2004a), spores from moulds (Moshhammer et al. 2000) and many organic pollutants including toxins with long half-lives (Neuberger et al. 1999), the task of the KRL was enlarged. Gases and small particles from outside reach indoor spaces easily, but even larger particles such as pollen (Moshhammer et al. 2005) enter rooms and may interact with indoor pollutants from cooking and heating (Moshhammer et al. 2006). For some air pollutants the inhalation route is of minor importance only, because most of them reach the human body via the food chain and are stored in human tissues for decades (Neuberger et al. 2000).

2. Indoor pollution

Besides gases and fine particulates entering rooms from outside the indoor pollution most frequently found in Austria is tobacco smoke. In tobacco control Austria is still a developing country, where a quarter of non-smokers is still insufficiently protected from tobacco smoke at the work place (Klimont et al. 2007). In this nationwide survey 10% of non-smokers aged 15+ years reported to be exposed to tobacco smoke in the home, however, in another survey (European Commission 2007) 32% of Austrian smokers admitted to smoke regularly inside their homes in the company of non-smokers and another 47% irregularly. Except for Spain and Hungary the Austrian rate of smokers never smoking inside their homes in the company of non-smokers (21%) was exceeded in all other EU member states and reached 70% in Sweden. Nevertheless passive smoking in Austria is even more prevalent in public rooms, e.g. for eating and drinking outside the home (Pletz and Neuberger 2011). About half of adult non-smokers in Spain and Austria reported passive smoking during leisure time (Table 1). Meanwhile Spain realized the failure of a partial smoking ban, amended its tobacco legislation and thereby reduced tobacco smoke exposure considerably (López et al. 2012), while the situation in Austria stayed unchanged (Neuberger and Moshhammer 2012).

Tab. 1: Percentage of passive smokers among 6545 non-smokers interviewed in 6 EU countries (12)

	E	A	SK	I	PL	IRL
home	28.7	18.1	11.0	13.3	22.1	11.5
work	35.2	19.2	25.0	34.2	40.2	1.3
leisure	56.1	47.8	37.0	7.8	21.5	8.5
transport	40.5	23.2	9.0	13.5	n.a.	6.2
TOTAL	71.9	66.2	41.0	36.8	35.9	23.4

Table 1: Percentage of passive smokers among 6545 non-smokers interviewed in 6 EU countries (Neuberger 2008a).

The result of the survey shown in table 1 corresponds to high exposures in the tourism industry measured in Austria and Spain compared to other countries (Nebot et al. 2005, Gorini et al. 2008). Compared to EU average, Austrians are not smoking more frequently, but make less quit attempts (European Commission 2007) and have a

high rate of relapse into smoking despite of using nicotine replacement (Neuberger 2008b) more frequently (European Commission 2007). This may be explained by a passive attitude expecting to be cured, disappointment from pharmacological treatment and lack of other help such as group therapy at the work place (Hutter et al. 2006, Moshhammer and Neuberger 2007). In addition, understanding for effects of passive smoking is poor in Austria (European Commission 2007), some politicians and media support tobacco industry and trade (Neuberger 2002) and disregard that both the World Bank and the World Health Organisation recommended high tobacco taxes (partly dedicated for tobacco prevention), smoke-free workplaces (without exemptions) and banning of all tobacco advertising as the most efficient methods to help the smoker to quit.

Among 29 European states Hungary, Spain and Austria reached the highest rate of smokers smoking at home in the presence of children (European Commission 2007). More than half of children in larger Austrian cities have to grow up in households with smokers (Neuberger et al. 2002, Moshhammer et al. 2006b). On average in the EU 9% of smokers did not care about children and 5% did not care about pregnant women when smoking in the car. In Austria this reckless behavior connected with extremely high air pollution was reported more frequently: 13% related to children and 8% related to pregnant women (European Commission 2007)¹. In a ranking of tobacco control in 30 European countries (based on taxation, legislation and funding) Austria got the poorest score (Joossens and Raw 2006). The KRL expressed its concern that the non-smokers protection in the Austrian tobacco law does not reflect the state of science (Maurer et al. 2008) and informed the public that indoor air pollution from tobacco smoke threatens the health of children (Moshhammer et al. 2007) and the health of adults, too: The high exposures measured in Austrian restaurants put patients with asthma or heart disease at risk for an attack already during the short time of a meal and are capable to induce a myocardial infarction in patients with coronary heart disease (Neuberger 2008c).

Groups at high risk without protection are the unborn and the children (Moshhammer et al. 2007). Austria participated in a multi-center study of children aged 6 to 12 years, which had been designed to investigate respiratory health effects from ambient air pollutants, but found most pronounced effects from parental smoking (Pattenden et al. 2006). Maternal smoking during pregnancy had long-lasting effects on wheeze, asthma, and nocturnal cough, independent of postnatal exposure. On the other hand postnatal exposure to maternal or paternal tobacco smoke was associated with bronchitis, wheeze and nocturnal cough (independently of smoking during pregnancy). In a sub-sample of 22 712 children respiratory functions were measured in eight regions in Europe and North America, including 2 825 elementary school children in Austria. Lung function growth was found impaired from passive smoking (Moshhammer et al. 2006a). Even if passive smoking was limited to the months before birth, respiratory functions of the children were impaired for many years. Children whose mothers smoked during pregnancy were 31% to 40% more likely to have poor lung function in elementary school. Postnatal exposure independently increased risk of poor lung function by 24% to 27% and independently from the effects of prenatal exposure.

In children from households with gas cooking vital capacity and forced expiratory volume in one second were reduced by 2% (Moshhammer et al. 2006). This reduction was attributed to the exposure to nitrogen dioxide (NO₂) and corresponded to even more pronounced impairments of respiratory functions associated with outdoor NO₂ from motor traffic and diesel engines in particular (Moshhammer et al. 2006b).

3. Outdoor pollution

Based on several severe air pollution episodes, a temporal correlation between high concentrations of particulate matter (PM), sulphur dioxide (SO₂) and acute increases in respiratory and cardiopulmonary mortality had been established in Vienna for the 1970's (Neuberger and Moshhammer 2004). Afterwards air pollution decreased in Austria, as documented by data on SO₂, and total suspended particles (TSP). During the 1980's Austria achieved the highest SO₂ reduction among the signatory states of the Helsinki Protocol, however, other sources of fine particulate matter like diesel traffic increased. Also other traffic related pollutants like NO₂ remained a problem, in particular in

¹ On average there are indications of improvements from the survey in Eurobarometer 2007 (European Commission 2007) to Eurobarometer 2010 (http://ec.europa.eu/health/tobacco/docs/ebs332_en.pdf), but not so in Austria (<http://www.aerzteinitiative.at/Eurobaro10.htm>).

cities with high density of population and cars. Therefore, short term effects of PM on lung function, morbidity and mortality were investigated in Vienna, Linz, Graz and a rural control area (Hauck et al. 2004, Neuberger et al. 2002, Neuberger et al. 2004). Long-term exposure and chronic disease – even more important for public health – were studied in repeated cross-sectional, a mixed longitudinal and a birth cohort study on school children in the city of Linz (Neuberger et al. 2002). After having established reference values of lung functions for Austrian children (Neuberger et al. 1999) their development was studied from entering elementary school to adulthood: Lung function growth was found impaired from long-term exposure to air pollutants and improved in districts where ambient air pollution had decreased. Where only TSP and SO₂ had decreased, no continuous improvement of small airway function was found and end-expiratory flow rates stayed impaired where NO₂-reduction from technical improvements of cars and industry was counterbalanced by increase of motorized (in particular diesel) traffic (Neuberger et al. 2002). In contrast to the study on effects of indoor NO₂ from gas stoves (Moshhammer et al. 2006), however, these effects of outdoor NO₂ may have been partly caused by ultrafine particles from diesel engines and other components of traffic pollution accompanying NO₂.

Active surface of particles inhaled several hours to days before spirometry was found related to short-term reductions in forced vital capacity, forced expiratory volume in one second and maximal expiratory flow rate at 50% of vital capacity. In pupils with asthma or previous airway obstruction 4-week-diaries proved that the following symptoms increased with daily exposure to urban particles characterized by their active surface area: wheezing, dyspnea, cough when going to sleep and cough at night (Neuberger et al. 2004, Moshhammer and Neuberger 2003). A rise in fine mode concentrations (PM_{2.5}) of 10 µg/m³ was linked to an increase in hospitalization of preschool girls by 8%, of school girls by 6%. A rise in carbonaceous fine particles (e.g. as caused by diesel-powered vehicles) in Vienna was also associated with adverse respiratory effects in kindergarten children (Horak et al. 2002). In adults effects were found on respiratory admission rates especially for the elderly. In persons older than 65 years bronchitis/asthma increased by 5.5% in men, and by 5.6% in women (Neuberger et al. 2004). First response of the respiratory system to a rise in fine particles seems to be a discrete, sub-clinical lung function impairment in children followed by symptoms of adverse effects in susceptible subpopulations and increased hospital admissions of elderly persons: Few hours after an increase of active particle surface in ambient air we recorded a slight increase in respiratory resistance and a slight decrease in peak flow (Moshhammer and Neuberger 2003). In the city of Vienna a significant increase in hospital admissions for bronchitis/asthma was found 2–4 days after a rise in PM_{2.5}: in elderly men and school girls after 2 days, in elderly women after 3 days, and in pre-schoolgirls after 4 days (Neuberger et al. 2004). A second significant peak in hospitalizations was observed in elderly persons 10 days after a rise in fine dust concentration. Besides these significant changes respiratory admissions stayed (insignificantly) elevated from the day of exposure to the end of the observation period of 14 days. Similar observations were made in Lower Austria (Neuberger et al. 2004).

The reaction of the cardiovascular system to fine particulates is even quicker and affects larger numbers of elderly persons (Neuberger 2007, 2008c). In the cities of Graz and Linz the increases of hospital admissions for cardiovascular disease per 10 µg/m³ were highest for PM₁ followed by PM_{2.5}, PM₁₀ and NO₂. No significant effects were found for O₃, not even during summer. Effect modification was seen by temperature, humidity, particle number, SO₂ and H₂S. In Linz where a steel plant contributes to PM exposure, significant effects were found in both sexes on the same day for PM and NO₂ and again after 2 days for NO₂ and after 6 days for PM. In Graz where local motor traffic dominates PM pollution, effects on admissions were found significant on the same day for NO₂ and after 6 days for PM. Admissions of females increased more per 10 µg/m³ of PM than admissions of males. Even levels of air pollution below the current EU standards were associated with cardiovascular admissions (Neuberger et al. 2008).

Recent mortality studies in the three largest Austrian cities considered cumulative and latent effects up to two weeks by use of time series with polynomially distributed lags (Neuberger et al. 2007a–c). Figure 1 shows that daily mortality in Vienna in 2000–2004 increased by 0.8% per 10 µg/m³ of NO₂ on the same and the following day, by 2.1% during the first week and by 2.9% over a period of two weeks (Neuberger et al. 2007c). This indicates that deaths accumulate during 14 days after an increase of air pollutants because of latent effects, while harvesting (reduced mortality after short increase due to premature deaths of most sensitive persons) is of minor importance for the net effect. A similar increase is seen in Figure 1 for fine particulates; however, PM₁₀ had only been monitored for 3

years and PM_{2.5} for 4 years, so that it had to be modeled for part of the observation period from TSP and meteorology.

In Graz a similar study was conducted from 1995 to 2005, evaluating daily pollution monitored at 5 stations, meteorology and incidences of influenza as registered by sentinels (Neuberger et al. 2007b). Like in Vienna effects of epidemic influenza exceeded air pollution effects on total and cardiopulmonary mortality, however, they were higher than the means reported from multicenter European studies (Ballester et al. 2008). This was attributed to the longer latency periods analysed, the high proportion of diesel vehicles in the car fleet and the aged population in these two cities. In Graz total mortality increased per 10 µg/m³ of NO₂ by 1.2, 2.0 and 2.6% if cumulating effects up to 1, 7 and 14 days. For PM₁₀ the corresponding increases were 1.3, 1.5 and 1.6%. The high mortality change with NO₂ indicates the importance of motor traffic, which also is the main source of fine and ultrafine particulates in Graz, followed by wood combustion. Somewhat lower increases of mortality per µg of pollutant (NO₂, PM_{2.5}, PM₁₀) per m³ of air were found in Linz. Nevertheless mortality increases were significant and accompanied by increases of cardiopulmonary emergency transports (Neuberger et al., submitted).

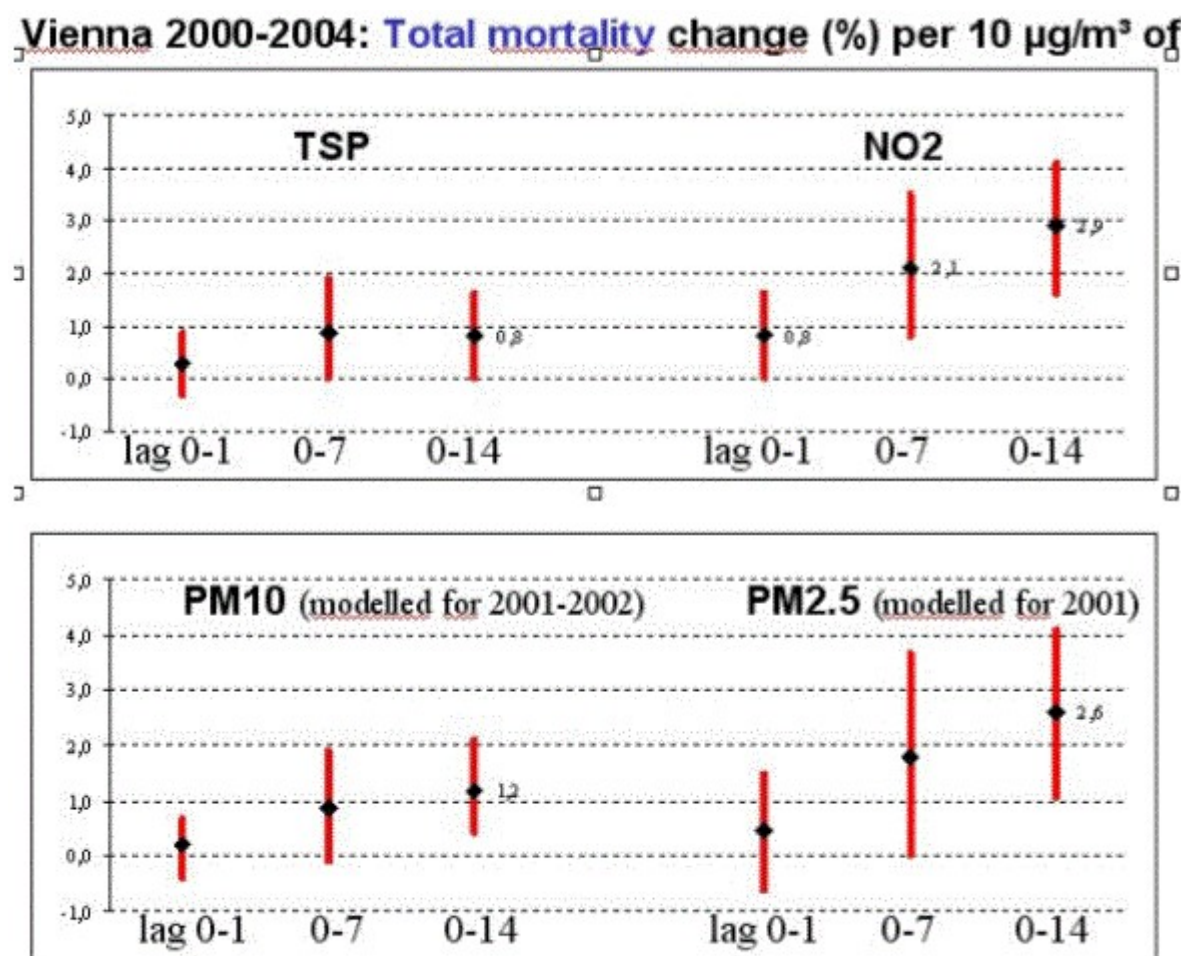


Figure 1: Total mortality increase in Vienna with daily air pollution

Figure 1: Total mortality increase in Vienna per 10 additional µg of the respective air pollutant per m³ (daily air pollution).

Besides NO₂ no other gaseous copollutants monitored influenced daily mortality. SO₂ was too low to be of importance and for ozone no effects on mortality could be proven if heat and humidity were taken into account. Neither for the total periods analyzed in Graz, Linz and in Vienna nor for the summer months a significant effect of daily ozone on mortality was detected. However, this could be related to the monitoring strategy for ozone, whis is measured mainly at elevated or downwind locations with lower population densities.

Per μg of fine particulates or NO_2 even higher increases of mortality were observed in cohort studies, which capture effects with latencies much longer than 2 weeks. Little is known on the mechanisms of irreversible long-term effects of PM such as myocardial infarction and cancer. In a prospective cohort study on 1630 dust-exposed and 1630 non dust-exposed male workers matched for age and smoking (Neuberger 1979) the exposure (nonfibrous, insoluble particulate matter in metal, ceramics, glass, stone industry) was related to cancer incidence (Neuberger et al. 1988). In dust exposed workers significant increases of deaths from lung cancer, stomach cancer, chronic obstructive and restrictive lung disease (underlying or main causes of death) were found. Lung cancer was significantly increased also in dust exposed workers without silicosis (Moshhammer and Neuberger 2004b).

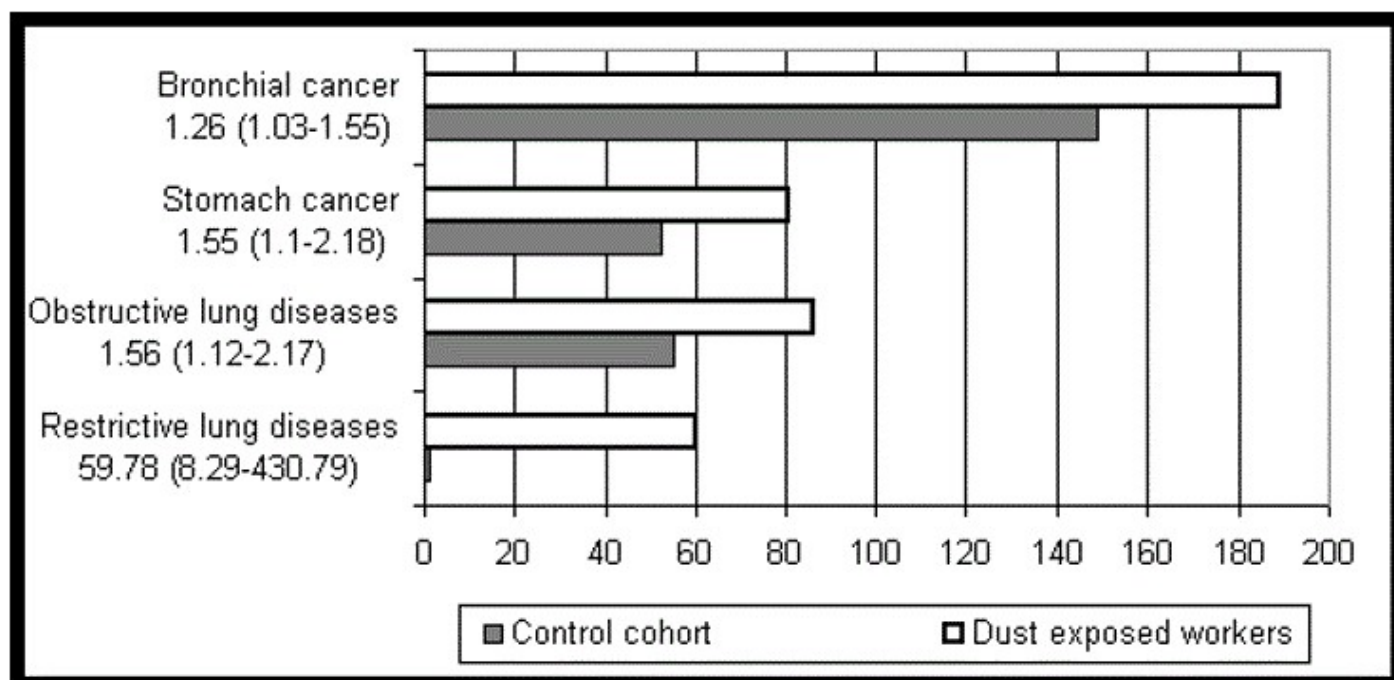


Figure 2: Deaths from bronchial cancer, gastric cancer and chronic lung disease in dust exposed and non dust exposed workers matched for smoking.

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Other studies were able to relate lung cancer to specific particles like those from diesel engines, and a large prospective study (Pope et al. 2002) linked lung cancer in the general population with long-term exposure to $\text{PM}_{2.5}$, mainly from combustion processes. All these epidemiological findings should have consequences for occupational and ambient air standards. Both NO_2 and fine particulates need to be reduced in ambient air. Health benefits of reducing NO_2 have been shown (Neuberger et al. 2002). From an earlier literature survey the Austrian Clean Air Commission decided already to recommend a lowering of the standard for the maximal daily mean of NO_2 to $80\mu\text{g}/\text{m}^3$ (Tvrdy et al. 1998).

The present standard for $\text{PM}_{2.5}$ in the European Union will not prevent acute and chronic health effects sufficiently (Neuberger et al. 2007c). Reducing annual mean levels of $\text{PM}_{2.5}$ to $15\mu\text{g}/\text{m}^3$ could lead to a reduction in the total burden of mortality among people aged 30 years and over that would be four times greater than the reduction in mortality that could be achieved by reducing $\text{PM}_{2.5}$ levels to $25\mu\text{g}/\text{m}^3$ (1.6% vs 0.4% reduction). The percentage reduction could grow by more than seven times if $\text{PM}_{2.5}$ levels were reduced to $10\mu\text{g}/\text{m}^3$ (3.0% vs 0.4%), equivalent to the standard recommended by the World Health Organization (Ballester et al. 2008).

4. Outlook

Recently new hazards were identified from nanoparticles (Neuberger and Moshhammer 2010) indoors and outdoors. At work places in Austria the most frequent source of hazardous particles in the submicron range (around

Advances of Atmospheric Aerosol Research in Austria (Interdisciplinary Perspectives 2), Austrian Academy of Science (ÖAW), Vienna 2012, p.103-115 (electronic version 2011: p.108-120). https://www.oeaw.ac.at/fileadmin/kommissionen/klimaundluft/2011_Advances_of_Atmospheric_Aerosol_Research_in_AustriaVorwort.pdf

100 nm) is environmental tobacco smoke (Neuberger 2009). Active and passive smoking is associated with 61% of male cancer mortality in Austria (Borsoi et al. 2010) and overall with about eleven thousand premature deaths annually (about 10 000 from active smoking and 1 000 from second hand smoke), mainly from cardiovascular causes (Maurer et al. 2011). Possibly effects of passive smoking have been underestimated (Pock et al. 2010), because of the non-linearity of the dose-response curve (Smith and Peel 2010) and the higher prevalence of second hand smoking in Austria compared to other countries (Wallner et al. 2010).

Other indoor sources of air pollution such as gas cooking have been found to be associated with only minor reductions in lung function of children (Moshhammer et al. 2010). In the future, however, furnaces could gain importance again as a source of indoor and outdoor pollution and related health risks (Haluza et al. 2012). From the replacement of all oil fired furnaces in Upper Austria without stricter particle emission standards for wood fired furnaces as recommended by the Clean Air Commission (Popp et al. 2007), 48 excess deaths are expected in the first year, increasing later to approximately 174 excess deaths per year (Moshhammer and Neuberger 2009b).

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