Health risks from long-range transboundary air pollution

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Introduction

Factors associated with the possible health effects of exposure to cadmium, lead and mercury have been investigated over many years. The types of adverse health effects are known to great extent, but because of the influence of confounding factors, it is very difficult to find thresholds for some outcomes such as impairment of cognitive functions in children exposed to lead or mercury.

In spite of the decreases in environmental exposure to these metals they are still present in the atmosphere and are carried to places remote from sources of emission by means of long-range atmospheric transport.
United Nations Economic Commission for Europe (UNECE) requested in 1999 the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution to assess the health effects of cadmium, lead and mercury from LRTAP and to document the supporting scientific information on the subject.
The preliminary assessment of the health risk of heavy metals from LRATP conducted in 2002 was revised using recent scientific evidence.

To finalize the assessment, the ninth meeting of the Joint WHO Convention Task Force on the Health Aspects of Air Pollution was convened in Berlin on 30 – 31 May 2006. The meeting reviewed the second draft.

On the basis of the conclusions formulated at the Berlin meeting the staff of the WHO European Centre for Environment and Health in Bonn prepared the final editing of the background material.
Health risks of heavy metals from long-range transboundary air pollution
Structure of the document

The report consists of one main chapter for each metal with the following sections:

► an introduction, summarizing sources, monitored and modelled levels for air and deposition, and the fate of the metal in the environment;
► pathways and levels of human exposure in relation to LRTAP;
► health hazard characterization, based on existing toxicological and epidemiological evidence;
► human health implication of the LRTAP.
Lead

Lead is released into the atmosphere from natural and anthropogenic sources.

The total emissions in Europe were about 35 kt in 1990 and 8.6 kt in 2003.

Lead is multimedia pollutant, *i.e.* several sources and media contribute to the exposure.

In general, ingestion of lead through food and water is the major pathway for lead in adults. For infants and young children, dust/soil and in some cases old lead-containing paints may constitute a major source of ingestion. Airborne lead is the minor exposure pathway.
Fig. 3.1. Reported emissions of lead in the EMEP region (24 countries).

Fig. 3.3. Anthropogenic lead emission sources in eight countries of the EMEP region in 1990 and 2003.
Atmospheric levels of lead

In Europe (2003) the concentrations of lead in air mainly ranged between 5 and 15 ng/m$^3$.

In cities and in the vicinities of industrial sources (i.e. at the distance of 1-10 km), higher concentrations prevail (up to an order of magnitude higher).

Atmospheric lead has the potential for long-range transportation.
Fig. 3.4. Modelled depositions of lead from British sources in 2003.
Lead in soils

Lead deposited from the air is generally retained in the upper 2 – 5 cm of undisturbed soil.

The natural lead content in soil typically ranges from below 10 mg/kg soil up to 30 mg/kg soil.

The lead concentrations in soil can be very high near the sources of emission (up to several thousand mg/kg) and decreases as the distance from the contaminating source increases.
Fig. 3.14. Concentrations of lead in topsoil; observations from field measurements and modelling.

Source: Salmiinen et al. (2005).
Pathways of human exposure

Lead is transferred from soil to food crops via root uptake. This is the main transfer rout for below ground plant parts, and seeds and fruits. For leafy vegetables, however, lead-containing dust deposits originating from long-range transport and from re-suspended particles play an important role.

The daily intake of lead with food amounted to 17 µg in Germany, 18 µg in Denmark, 27 µg in U.K., 15 µg in Croatia, 12 in Finland, 20.4 µg in the Czech Republic, and to 3 µg in adults in the USA.

WHO-FAO (1993) PTWI amounts to 1.5 mg Pb/week (25 µg/kg b.w.) which corresponds to 215 µg daily.
Lead in blood (Pb-B) levels can be used as a predictor of possible health effects. Human exposure can be calculated on the base of existing coefficients and models.

**Adults**

**Air**
- Rural areas: $0.1 \, \mu g/m^3 \times 16.2 = 1.6 \, \mu g/l$
- WHO guideline: $0.5 \, \mu g/m^3 \times 16.2 = 8.1 \, \mu g/l$

**Food**
- Present intake: $20 \, \mu g/day \times 0.4 = 8.0 \, \mu g/l$
- PTWI: $215 \, \mu g/day \times 0.4 = 86 \, \mu g/l$

**Water**
- WHO guideline: $10 \, \mu g/l \times 2 \, l = 20 \, \mu g \times 0.4 = 8.0 \, \mu g/l$
Children

Air
Rural areas 0.1 µg/m³ x 19.2 Pb-B 1.9 µg/l
WHO guideline 0.5 µg/m³ x 19.2 Pb-B 9.6 µg/l

Food
Germany (5-7 years) 17 µg/day x 1.6 Pb-B 27.2 µg/l

Water
WHO guideline 10 µg/l x 0.5 l = 5 µg x 1.6 Pb-B 8 µg/l

Soil and dust
50 mg Pb/kg x 100 mg of soil = 5 µg x 0.7 Pb-B 3.5 µg/l
Reports on the geometric mean values in different countries revealed that in women and children Pb-B levels are within the range of 10-30 µg/l considered as a „baseline” of minimal anthropogenic origin (Germany 16,3 µg/l, Kolossa-Gehring, 2007; Czech Republic 33 µg/l, Batariova et al. 2006)
Fig. 3.17. B-Pb levels (geometric means) in 3715 Swedish children for 1978–2005.

Source: Strömberg et al. (2003); Strömberg et al., unpublished data.
It is important to note that since the PbB levels are log normal distributed, then even at relatively low geometric mean values, a proportion of the population will have PbB above 100 µg/l.

At a geometric mean PbB level of 36 µg/l, approximately 1.5% of the population will have Pb-B above 100 µg/l, and at a geometric mean PbB level of 60 µg/l, approximately 6% of the population will have Pb-B above 100 µg/l.
Health hazard characterization

Lead is a well known neurotoxic metal. Impaired neurodevelopment in children is the most critical health effect.

In 1991 the Centers for Disease Control recommended that the Pb-B values in children should be below 100 µg/l.

The WHO Air Quality Guidelines for Europe (WHO, 2000) recommended that at least 98% of the population exposed in the general environment should have Pb-B below 100 µg/l, and the median blood lead level should not exceed 54 µg/l.

Recent publication suggest that the effects of environmental exposure of children can occur at Pb-B levels below 100µg/l.
Children

Blood lead concentrations, even those below 100 µg/l, are inversely associated with children’s IQ scores at ages 3 and 5 years, and the related declines in IQ are greater at these concentrations than at higher levels. For the sample of children whose maximal Pb-B remained below 100 µg/l, IQ declined by 7.4 points as the lifetime average Pb-B increased from 10 to 100 µg/l (Canfield et al., 2003).

Other recent findings also suggest a possible impairment of the neuropsychological functions (Canfield et al., 2004) and lead-related deficit in colour vision (Canfield et al, 2003a) as a result of low-level lead exposure in children.
During pregnancy, maternal calcium requirements increase and are maintained mostly through increased bone resorption. The latter facilitates active transfer of calcium to the fetus. Maternal lead transfer follows a pattern similar to that for calcium, without any barrier at the placental level. This is particularly true for the last part of pregnancy and the lactation period when maternal Pb-B increases by 25-100%. This increase derives from the further mobilization of lead from bones. Pb-B in infants is mainly the expression of maternal skeletal lead stores (Gulson et al., 1997; Tellez-Rojo et al., 2004).
Neurological toxicity is observed in children as a result of the ability of lead to cross the placental barrier and to cause neurological impairment in the fetus (Emory, et al. 2003; Gomaa et al., 2002)

If the main toxic event is prenatal exposure, attention should be paid to maternal lead stores and, if possible, mobilization of these stores during pregnancy should be avoided
All these findings indicate a need to reconsider the currently held notion that lead levels below 100 µg/l are acceptable from the public health perspective. In 2006 the Scientific Committee on Neurotoxicology and Psychophysiology and the Scientific Committee on the Toxicology of Metals of the ICOH (Landrigan et al., 2006) resolved that current exposure standards for lead urgently need to be reduced.

For children, the action level, which triggers community prevention efforts to reduce exposure sources, should be immediately reduced to a Pb-B concentration of 50 µg/l in nations worldwide.

Also for female industrial workers of reproductive age, the standard for Pb-B should be reduced to the lowest obtainable, preferably to 50 µg/l.
Human health implications in relation to LRATP

In many areas there has been a major decrease in Pb-B levels observed the last few decades, mainly because of the phasing out of leaded petrol, but also because of reductions in other sources of lead.

Elevated exposures are generally due to the local sources rather than being the result of LRTAP.

The annual lead inputs due to transboundary air pollution and the application of mineral and organic fertilizers to topsoils are of roughly the same magnitude.
Those inputs are relatively small in comparison to lead stores that have already accumulated and those that are from natural sources.

However LRTAP may contribute significantly to lead content of crops, through direct deposition.

Although uptake via plant roots is relatively small, rising levels in soils over the long term is a matter for concern, and should be avoided because of the possible health risk of low-level exposure to lead.
Cadmium

The anthropogenic sources of cadmium include non-ferrous metal production, stationary fossil fuel combustion, waste incineration, iron and steel production and cement production.

Food is the main source of cadmium exposure in the general population (above 90% of total intake in non-smokers). Besides, soil and dust account for a part of local population exposure in heavily contaminated areas.
Fig. 2.1. Reported emissions of cadmium in the EMEP region (24 countries).

Fig. 2.3. Changes in cadmium emissions from anthropogenic sources (various sectors) between 1990 and 2003 in eight countries.

Source: Vestreng et al. (2000).
Atmospheric levels of cadmium

In Europe the background concentrations of cadmium in air mainly ranged between 0.2 and 1 ng/m³.

In industrialized regions, cadmium concentrations were in the range 1-10 ng/m³ and in the proximity of industrial sources even higher (100 ng/m³).

Atmospheric cadmium has the potential for long-range transportation.
Fig. 2.4. Modelled depositions of cadmium from Belgian sources in 2003.

Source of the data: EMEP/INSC-E.
Cadmium in soils

The average cadmium contents in surface soils lies between 0.07 and 1.1 mg/kg; values above 0.5 mg/kg usually reflect anthropogenic input.

Cadmium concentrations in the topsoils of Europe vary from <0.03 to>0.8 mg/kg.

There are three main anthropogenic sources of terrestrial cadmium: atmospheric deposition, agricultural application of phosphate fertilizers, and use of municipal sewage sludges as a fertilizer on agricultural soils. About 90% of the cadmium in soil remains in the top 15 cm.
Fig. 2.13. Concentrations of cadmium in topsoils; observations from field measurements and modelling.

ICP-MS, detection limit 0.01 mg/kg. Number of samples 840. Median 0.140 mg/kg

Source: Salminen et al. (2005).
**Bioaccumulation**

The transfer of cadmium from soil to the food chain depends on a number of factors such as the type of plant, the type and pH of the soil, and zinc and organic matter content in the soil. Soil pH is the principal factor governing the concentration of cadmium in the soil solution. Plant uptake of cadmium decreases as the soil pH increases.

In animals, cadmium accumulates largely in the liver and kidney and not in the muscle tissue. There are large differences in the concentrations of cadmium in different kinds of food (milk - 1 µg/kg; meat, fish, fruit 1 - 50 µg/kg; wheat, rice, potatoes and leafy vegetables 10 - 300 µg/kg; kidney, liver, oysters 100-1000 µg/kg). Groups of population consuming excessive amounts of specific food items (mussels, kidney, liver, leafy vegetables) have higher risk of cadmium exposure.
Pathways of human exposure

Exposure via inhalation
Assuming a daily inhalation of 20 m$^3$, and based on the highest concentration of cadmium in rural, urban and industrialized areas, the amount of cadmium inhaled daily does not, on average, exceed 0.04, 0.2 and 0.4 µg, respectively.

Cigarette smoking may represent an additional source of cadmium which can exceed that from food. It can be estimated that a person smoking 20 cigarettes a day will absorb about 1 µg of cadmium.
Exposure via gastrointestinal tract

For non-smokers, food constitutes the principal environmental source of cadmium. In recent years, mean daily intake of cadmium from food amounted to 10-14 µg in Germany, 27 µg in France, 17.3 in Croatia, 11 - 29 µg in Spain, 11-19 µg in the Czech Republic, 11-16 in Sweden 1994, 23.3 µg in Poland (females).

The PTWI (WHO-FAO, 1993) for Cd is 500 µg (a weekly intake of 7 µg/kg b.w.), corresponding to the daily intake of 70 µg or 1 µg/kg body weight/day. The U.S.EPA Reference Dose (RfD) amounts to 1 µg/kg /day (IRIS, 1994).
Relevance of various routes of exposure

The uptake via food was estimated to amount to 97.2 % in Japan and 93.7% in China (Zhang et al., 1997).

In the Czech Republic, the daily intakes via respiratory route, water and food were estimated at 0.01; 0.17 and 18.2 µg/day (0.05; 0.92 and 99.3 %), respectively (Kliment, 1996).
Biomarkers of exposure

Cadmium concentration in blood (Cd-B) reflects the current exposure. Cadmium concentration in urine (Cd-U) is mainly influenced by the body burden and it is proportional to cadmium concentration in healthy kidney.

In Germany (Backer et al., 2003), the P50 of Cd-U (n=4740, adults 18-69 years) amounted to 0.22 µg/ g creat. (0.20 in non-smokers; 0.29 in smokers). The proposed reference value is 0.8 µg/ g creat.

In the Czech Republic (Batariova et al., 2006), the geometric mean Cd-U level amounted to 0.31 µg/ g creat. The proposed reference value (P95) is 1.2 µg/ g creat.
Critical effects

Kidney and bone are the critical target organs in chronic environmental exposure. The main critical effects include increased urinary excretion of low molecular weight proteins, as a result of proximal tubular damage, and also an increased risk of osteoporosis.
Kidneys

It has been suggested that for the general population the Cd-U levels should be below 2.0 – 2.5 µg/g creatinine. Recently the relation between cadmium exposure and tubular and glomerular function was investigated in women, aged 53-64 years, under a study conducted in Sweden in 1999-2000. Multiple linear regression showed cadmium in blood (median, 0.38 µg/l) and urine (0.67 µg/g creatinine) to be significantly associated with effects on renal tubules (indicated by increased levels of human complex-forming protein and NAG in urine). Tubular renal effects occurred at lower cadmium levels than previously demonstrated, and glomerular effects were also observed. Although the effects were relatively moderate, they may represent early signs of adverse effects, affecting large segments of the population. (Akesson et al., 2005).
**Bones**

In a study of men and women, aged 16--81 years, under environmental and occupational exposure to cadmium, Alfven *et al.* (2000) measured bone mineral density (BMD) in the forearm. The dose-response relationship was found between cadmium dose and osteoporosis. The OR for men was 2.2 (95% CI, 1.0-4.8) for the dose of 0.5 - 3 µg Cd/g creatinine and 5.3 (2.0-14) for the highest dose (> 3 µg Cd/g creatinine) compared with the lowest dose level (< 0.5 µg Cd/g creatinine). For women, the OR was 1.8 (0.65-5.3) for the dose of 0.5-3 µg Cd/g creatinine.
Trends of internal exposure

The recently published data do not show decrement of cadmium body burden in non-smokers over the last decade. The time trends for cadmium, mercury and lead were evaluated in Sweden. The concentrations in erythrocytes (Ery) were determined in a subsample of the population-based MONICA surveys in 1990, 1994, and 1999 in a total of 600 men and women aged 25-74 years. Annual decreases of 5 – 6 % were seen for Ery-Pb and EryHg levels. For Cd, the decline in Ery-Cd was seen only among smokers, indicating that Cd exposure from tobacco smoke has decreased while other environmental sources of Cd have not changed significantly (Wennberg et al., 2006).
Human health implications in relation to LRTAP

- The margin of safety between the present dietary daily intake of cadmium and the level of intake which can bring about health effects is very narrow. For highly exposed subpopulations, this margin may even be non-existing. Population groups at risk include the elderly, diabetics and smokers. Women may be at an increased risk because they absorb more cadmium than men due to the lower iron stores.

- In spite of the decreasing cadmium emissions, the recently published data do not show decreases of cadmium body burdens in non-smokers over the last decade.
The studies on cadmium balance in top layers of arable soil indicate that supply of this metal still exceeds its removal, thus increasing the risk of future exposure through food.

In view of the narrow margin of safety involved, sufficient effort should be made to achieve further reductions in cadmium emissions and in the direct input of cadmium to the soil.
Mercury

Mercury occurs normally in the environment. The largest anthropogenic source of mercury on a global scale is the combustion of coal and other fossil fuels.

Emissions of mercury into the air from both anthropogenic and natural sources are in inorganic forms:

- elemental mercury (Hg\(^0\)): more than 90%
- gaseous inorganic mercury (RGM): 1 – 5%
- particle bound mercury: 1 – 6%
Fig. 4.1. Reported emissions of mercury in the EMEP region (24 countries).

Fig. 4.3. Anthropogenic sources of mercury emission in 1990 and 2003 (for eight countries).

Source: Vestreng et al. (2005).
Elemental mercury is characterised by a long atmospheric lifetime and so it can be transported globally.

The spatial and temporal variability of atmospheric concentration is relatively low.

EMEP (1990-2003) : 1.3 – 2.0 ng/m$^3$

Arctic stations (1995-2002) : 1.52 – 1.62 ng/m$^3$

Only about one third of the mercury deposition in Europe originates from European emission.
Fig. 4.10. Annual deposition field of mercury from European anthropogenic sources. The red rectangle indicates the EMEP domain.
Fig. 4.15. Concentrations of mercury in topsoil; observations from field measurements and modelling.

Hg analyser, detection limit 0.0001 mg/kg. Number of samples 833. Median 0.040 mg/kg

Source: Salminen et al. (2005).
Mercury is deposited on the soil mainly in the form of inorganic compounds, which are readily bound to organic matter.

Processes involving mercury in the soil are:
- revolatilization
- methylation
- mobilization to the aquatic environment

Methylation to monomethylmercury occurs under anaerobic conditions via sulfate-reducing bacteria mainly in wetlands and other water-rich soils.
Estimated average daily intake and retention (in parentheses) of total mercury (ng/day) in the general population.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Elemental Hg vapour</th>
<th>Inorganic Hg compounds</th>
<th>Methylmercury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>30 (24)</td>
<td>2 (1)</td>
<td></td>
</tr>
<tr>
<td>Food</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fish</td>
<td>0</td>
<td>600 (42)</td>
<td>2400 (2300)</td>
</tr>
<tr>
<td>Non-fish</td>
<td>0</td>
<td>3600 (250)</td>
<td></td>
</tr>
<tr>
<td>Drinking water</td>
<td>0</td>
<td>50 (35)</td>
<td>0</td>
</tr>
<tr>
<td>Dental amalgams</td>
<td>1200-27000 (1000-22000)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total retained</td>
<td>1000-22000</td>
<td>330</td>
<td>2300</td>
</tr>
</tbody>
</table>
Methylmercury

As a result of increased transport of mercury to lakes, concentration in fish increased during the last century. In the southern parts of Finland, Norway, and Sweden, the mercury content in an 1-kg pike is about 0.5-1.0 mg/kg. The natural background value is estimated at about 0.2 mg/kg.

The recommended limit of 0.5 mg/kg is exceeded in about 50% of the lakes in Sweden and in 85% lakes in southern and central Finland.
Biological indicators of exposure

About 90% of mercury found in red blood cells was in the form of MeHg. THg in plasma was associated with both IHg and MeHg. **THg in hair reflects MeHg exposure at all exposure levels, and not IHg exposure.** The small fraction of IHg in hair is most probably emanating from demethylated MeHg. THg in urine reflected IHg exposure.

**THg in RBC and hair are suitable proxies for MeHg exposure.** THg in urine is a suitable proxy for IHg exposure. (Berglund et al., 2005)
Health hazard characterisation

The effects of methylmercury on the adult differ both quantitatively and qualitatively from the effects observed after prenatal or, possibly, postnatal exposure. The critical organ is the nervous system and the critical effects include developmental neurologic abnormalities in human infants, and paraesthesia in adults.

Prenatal exposure was reported to cause psychomotor retardation in infants.
Benchmark dose calculations have been performed for methylmercury-associated delays on evoked potential latencies in two cohorts of children from the Faroe Islands and from Madeira (Murata et al., 2002). The obtained BMDL 5 % of approximately 10 µg/g maternal hair was similar to that calculated for other neurological variables (Budtz-Jorgensen et al., 2002) in the Faroese children and in the New Zealand population (Crump et al., 1998).

The benchmark dose, based on a combination of all childhood neurological endpoints (onset of walking and talking, neurologic scores, mental symptoms, and seizures) was calculated by the U.S. EPA (IRIS, 1995). A benchmark level of 11 µg/g in maternal hair has been established, equivalent to maternal blood level of 44 µg/l or daily intake of 1.1 µg/kg/day.
The US National Academy of Sciences Committee on the Toxicological Effects of Methylmercury, applying an uncertainty factor of 10, arrived at the value of about 1 µg/g mercury in maternal hair. A daily intake of 0.1µg/kg/day, the USEPA current RfD, would result in such level (Bellinger, 2000, Rice et al., 2003)
The present background level of Hg-H associated with no or low fish consumption or low fish methyl mercury concentration amount to 0.25 µg/g in Germany (Drasch et al., 1997), 0.8 µg/g in Denmark (Grandjean et al., 1992), 0.28 µg/g in the north of Sweden (Oskarsson et al., 1996), 0.06 mg/kg in non-fish eating people in Sweden (Lindberg et al., 2004)
Much higher Hg-H levels result from the consumption of large amounts of fish or sea mammals. In the Faroe Islands, the mean Hg-H levels ranged from 1.6 µg/g (1 fish meal per week) to 5.2 µg/g (4 fish meals per week) (Grandjean et al., 1992).

In fishermen from Madeira (Portugal) and their families, Hg-H levels of 38.9 µg/g were found in men and 10.4 µg/g in women (Renzoni, 1998).

Bjornberg et al. (2005) investigated Swedish women with high fish consumption. The average total fish consumption was approximately 4 times/week. T-Hg in hair (median 0.70 mg/kg, range 0.08-6.6 µg/g) was associated with MeHg in blood).
These results show that not only on the islands such as Faroe Islands or Madeira but also in the continental Europe, high fish consumption can result in hair mercury levels several times higher than the 1 µg/g recommended by the US Academy of Sciences.
Human health implications in relation to LRTAP

- Airborne concentrations of mercury in Europe, and also globally, are generally well below the levels known to cause adverse health effects from inhalation exposure.

- Concentration of inorganic mercury species in surface water and groundwater are generally well below the levels known to cause adverse health effects from water consumption.

- Little information is available on the provenance of methylmercury in marine fish and on the contribution of long-range transport in the process. Evidence exists for increasing levels in marine fish and mammals in the Arctic, indicating the impact of long-range transport.
In general fish consumption has important beneficial effects on human health. However, in some populations consuming large amounts of fish, the intake of mercury can reach hazardous level. Thus, a high priority should be given to lowering the concentrations of methylmercury in fish.

Reducing the emissions into the atmosphere and long-range transport of pollution will be of great importance in the achievement of these goals.