

# MONITRAF

## Work Package 5

# Recommendations for a health impact assessment in the Alpine transit regions

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31.3.2006

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## Summary

Air pollution and noise have adverse effects on human health and traffic is one of the main contributors to air pollution and noise. One aspect of the project MONITRAF ("Monitoring of road traffic related effects in the Alpine space and common measures") aims at identifying and analysing the impact on human health due to air pollution and noise emitted by road traffic within the Alps along the four main transit corridors.

A method to quantify effects on human health is a so called "health impact assessment" (HIA). In a HIA, published exposure-response relationships from national or international large, high-quality epidemiological studies are pooled. The derived knowledge about the impact of air pollution (or noise) on health is transferred to the area of interest, in this case the Alpine transit area. Population exposure in the area must be known, and, subsequently, health effects are quantified.

In the past, several studies estimated the health impact from air pollution exposure for a larger area (e.g. on a country level), using PM<sub>10</sub> as an exposure surrogate for general air pollution. Within the project MONITRAF an assessment on a detailed scale is necessary. Such an impact assessment along the transit axis is methodologically more demanding and a scientifically established method does not exist yet. The goal of this study was to evaluate the feasibility of such a HIA and to propose a suitable method. By means of a literature review an appropriate surrogate was chosen to quantify the exposure-response associations for different outcomes.

We propose the following procedure for a future HIA from transit traffic:

1. **Study area:** The area of interest should be properly defined. Area of interest could be the Alpine transit corridors, only. If so, start, end and width of these corridors have to be defined. One might take into account that most likely health impact from transit traffic is much higher in the lowlands due to the larger exposed population.
2. **Exposure surrogate:** We recommend to select NO<sub>2</sub> as primary air pollution exposure surrogate. It is source specific and represents the spatial variation of transport-related air pollution. In addition, there are a number of epidemiological studies available that allow to quantify the health effects from NO<sub>2</sub>-exposure on a population level.
3. **Conversion factors:** In order to make value of epidemiological studies evaluating other suitable air pollution exposures (e.g. Black Smoke, ultrafine particles, elemental carbon, soot), conversion factors should be applied which allow to transfer the results

into NO<sub>2</sub>. These conversion factors have yet to be developed for the project MONITRAF, based on the air pollution situation in the Alpine transit areas.

4. **Exposure assessment:** The population exposure of NO<sub>2</sub> in the Alpine transit areas must be quantified. For this step, a GIS model with an appropriate spatial resolution may be developed. Other approaches such as passive sampler measurement campaigns are considered less efficient and reliable. It would be helpful to derive exposure distributions for the whole population as well as for different age groups separately (e.g. children < 14 years, adults, etc.)
5. **Outcome selection:** Air pollution related health effects to be considered include respiratory and cardiovascular morbidity, mortality, years of life lost and birth outcomes. The exact diagnoses that can be evaluated should be determined in the future HIA. Only diagnoses for which epidemiological studies are available can be considered. In order to avoid double counting the diagnoses must not overlap.
6. **Exposure-response association:** for each health outcome, a systematic literature review has to be performed to identify all eligible studies. With help of meta-analytical statistical techniques, a pooled exposure-response relationship from all selected publications has to be determined for each outcome separately. This step has yet to be carried out for the project MONITRAF.
7. **Prevalence and incidence data:** Because epidemiological studies quantify the relative excess risk from air pollution, knowledge of baseline prevalence or incidence of each selected outcome is required. These data should be derived preferably from national statistics or from representative surveys in the respective countries and should refer to the area of interest.
8. **Reference concentration:** In HIAs, a reference scenario has to be chosen. Usually it is not justified to quantify health effects down to the zero level of exposure. On the one hand, in general, nobody is exposed to such low levels. On the other hand there are no studies available which show health effects at very low pollutant levels. Only health effects above the reference concentration are quantified. One has to be aware that the selected level will substantially affect the result of the HIA. For NO<sub>2</sub>, the appropriate reference concentration has to be evaluated.
9. **Number of attributable cases:** In the last step, the number of attributable cases has to be calculated. The attributable cases refer to the number of cases that would not have occurred if the air pollution exposure would be at or below the reference level.

HIA of *noise* on health follows the same principle. Exposure assessment (step four) should be based on noise cadastral data. Relevant health outcomes (step five) include cardiovascular morbidity, sleep disorders and annoyance. In step eight, the "reference

concentration" of noise refers to the exposure level of zero effect. In the context of noise related HIA, it has to be considered that increasing traffic does not necessarily lead to an increased population exposure if noise protection measures are implemented. This is in particular relevant for cardiovascular morbidity where there is little evidence for harmful health effects below the standard limits. In contrast, relevant noise levels for annoyance can be well below the standard limits.

There is potential for cooperation of the Project MONITRAF with the projects MfM-U and ALPNAP. This includes e.g. expertise in assessment and modelling of the exposure(s) in the Alpine area and the development of region-specific exposure-response relationships for noise annoyance.

We conclude that a HIA in the Alpine area for air pollution and noise emission from transit traffic is a feasible and cost-effective way to quantify health impact from transit traffic. Whereas  $PM_{10}$  is an appropriate surrogate to assess health impact from the general air pollution for a larger area, we consider  $NO_2$  a more appropriate surrogate to represent the small area air pollution distribution in the alpine area from emissions of the transit traffic.

# 1. Health Impact-Assessment

## 1.1. Introduction

Air pollution and noise have adverse effects on human health and traffic is one of the main contributors to air pollution and noise. The project MONITRAF, ("Monitoring of road traffic related effects in the Alpine space and common measures") aims at identifying and analysing the impact of road traffic within and through the Alps along the four main transit corridors (see Figure 1).

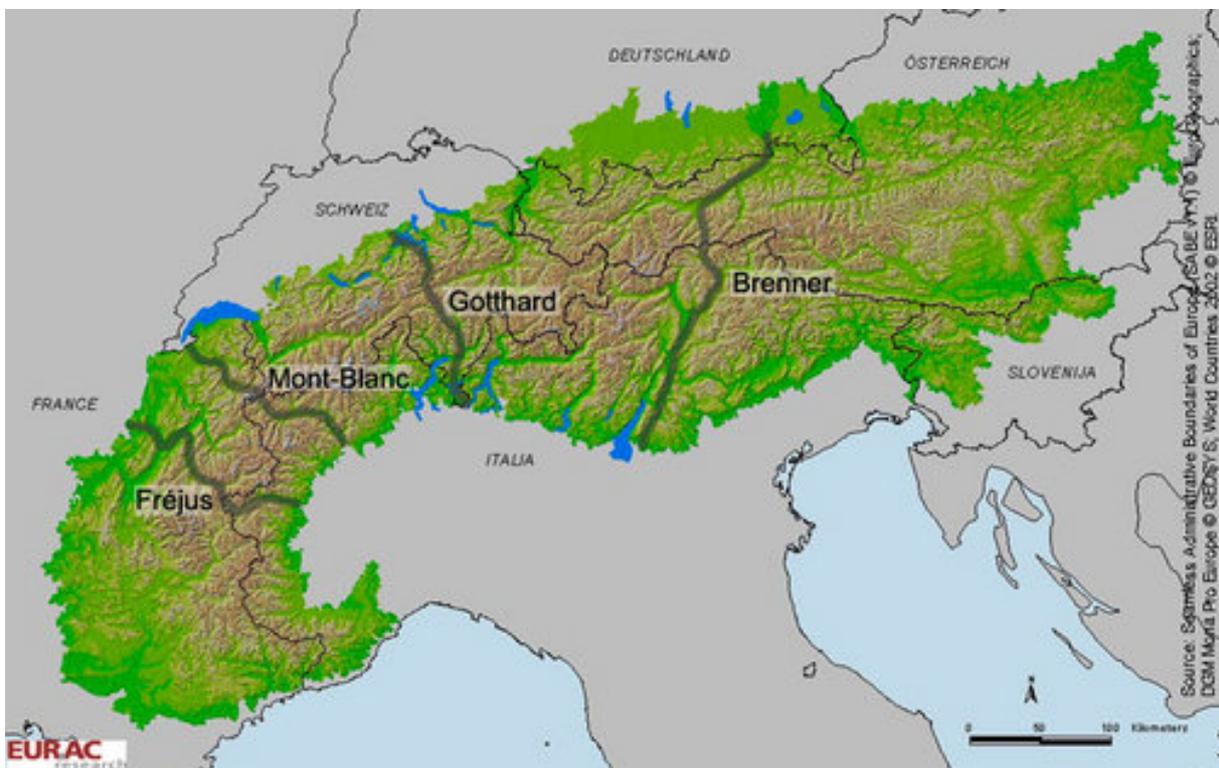


Figure 1: Project area MONITRAF, Source: [www.monitraf.org/44d228.html](http://www.monitraf.org/44d228.html)

The objective of the project is to develop comprehensive measures that aim at reducing the negative effects of road traffic, while simultaneously enhancing the quality of life within the Alpine region ([www.monitraf.org](http://www.monitraf.org)). One aspect of the project addresses the impact on human health due to air pollution and noise in the area. Within work package five, we will discuss methodological problems in dealing with a health impact assessment on a detailed spatial scale, give an overview over the literature and propose a methodology that considers the available data.

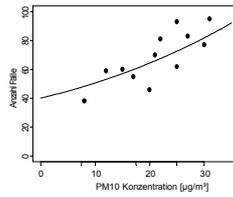
## **1.2. Advantage of a health impact assessment compared to a "public health study" in the Alpine transit areas**

There are high-quality epidemiological studies that measured the association between air pollution and health outcomes. In general, data on health outcomes and potentially confounding variables have to be assessed in these studies on an individual basis, i.e. measurements in every participating person are necessary. Examples are e.g. the cross-sectional study SCARPOL (Swiss Surveillance Program of Childhood Asthma and Allergies with respect to Air Pollution and Climate), where health outcomes of nearly 4500 children were investigated, (Braun-Fahrlander et al. 1997) or the large SAPALDIA-study (Swiss Study on Air Pollution and Lung Diseases in Adults), a cohort study in which data of more than 8000 persons was collected (Ackermann-Lieblich et al. 2005). An exception of studies where individual data are necessary are time-series studies, in which the temporal association between health outcomes such as mortality, emergency hospital admissions etc and air pollution data are analysed. An example for such an approach is the European study APHEA (Air Pollution and Health – A European Approach). In this study, nearly 3 million deaths occurring in 30 areas in Europe were set in temporal correlation to air pollution (Samoli et al. 2006). These large, high-quality studies give information on associations between air pollution and health. However, it would be difficult to implement any of these approaches in the Alpine transit areas. First of all, these studies require large population numbers, but the Alpine transit areas do not display high population numbers. Second, these studies are very money-intensive. In such situations, a health impact assessment can be carried out. In a HIA, published exposure-response relationships from national or international large, high-quality epidemiological studies are pooled. Subsequently, the derived knowledge about the impact of air pollution (or noise) on health is transferred to the area of interest, in this case the Alpine transit area. This procedure has the advantage of the simultaneous evaluation of a range of health outcomes and much lower costs.

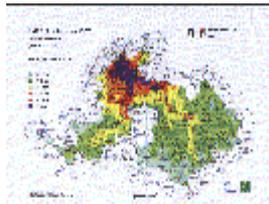
## **1.3. Health impact assessment**

What is a health impact assessment (HIA) and how is a HIA performed? In a HIA, known exposure-response relationships are transferred to an area of interest. (The exposure-response curve describes the relationship between the exposure ("dose") and its effect on health.) Population exposure in the area must be known, and, subsequently, health impact is quantified. This means that a HIA has three components (see Figure 2): First, an exposure-response association has to be extracted from the literature, preferably from several high-quality studies. Second, population exposure has to be assessed. Third, the exposure-response association and the population exposure are combined to calculate the number of attributable cases. The attributable cases refer to the cases that would not have occurred without exposures, which are sometimes referred to as the "preventable cases".

**1. Exposure-response association:**



**2. Exposure assessment:**



**3. Estimation of number of cases:**

**Number of cases = exposure x exposure-response association**

**Figure 2: General procedure of a health impact assessment**

HIA have been performed for assessing the impact of general air pollution ("PM<sub>10</sub>"). However, for evaluating the impact on human health in the Alpine transit corridors, there are two main problems: A suitable surrogate must be chosen that adequately represents transit-transport related air pollution and considers the spatial distribution on a detailed scale.

## **2. Health impact assessment in air pollution**

Air pollution can be defined as any substance in the atmosphere commonly recognised as harmful to humans or the earth. Air pollutants are classified as either primary or secondary; a primary air pollutant is one that is emitted directly to the air from a given source, such as carbon monoxide produced as a by-product of combustion, whereas a secondary air pollutant is formed in the atmosphere through chemical reactions involving primary air pollutants. The formation of ozone in photochemical smog is an example of a secondary air pollutant.

### **2.1. Exposure assessment**

#### **2.1.1. Concept of surrogate measurements**

Due to the complex mixture of air pollution, it is not possible to assess every single substance in the air. Epidemiological approaches take this into account by evaluating surrogate pollutants that are thought to represent a specific mixture. However, this means that health effects must be attributed to the whole mixture and not just to the chosen surrogate. In the scientific literature, these surrogates are also referred to as "proxy" or in German "Indikatorschadstoff".

The most important surrogates include  $PM_{10}$  ("particulate matter"),  $PM_{2.5}$ , nitrogen dioxide  $NO_2$  and nitrogen oxides  $NO_x$ , carbon monoxide CO, benzo[a]pyrene, benzene and "Black Smoke".

#### **Direct surrogates**

##### *$PM_{10}$*

PM is emitted by industry, in combustion processes and by agriculture. Transport related PM emissions come from exhaust fumes, abrasions of brakes, tyres and re-suspension of road dust. Larger particles are generally filtered by small hairs in the nose and throat. PM below 10 micrometers (" $PM_{10}$ ") aerodynamic diameter can penetrate into the thoracic part of the airways ("respirable fraction", see also Figure 3) and cause health problems.

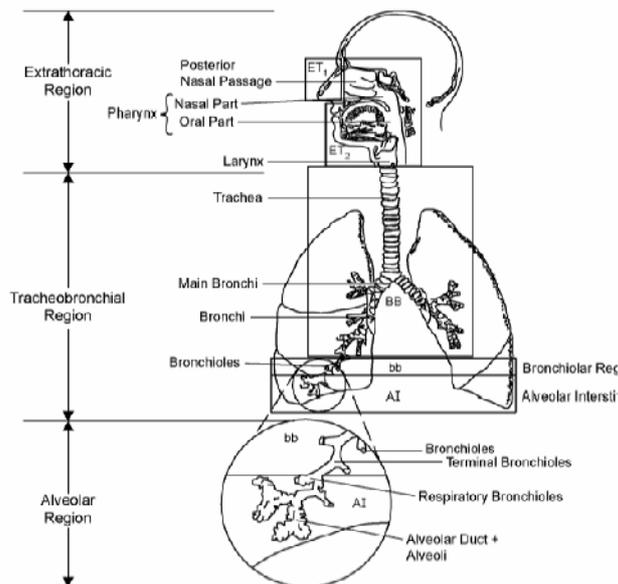


Figure 6-1. Diagrammatic representation of respiratory tract regions in humans.

**Figure 3: respiratory tract, source: (U.S. Environmental Protection Agency 2004)**

PM<sub>10</sub> is used in epidemiological studies to assess the effect of general air pollution on health (see Figure 4). However, for transport-related air pollution, PM<sub>10</sub> is not an ideal surrogate and more specific pollutants should be used that represent the specific mixture and the spatially less homogenous distribution.

*PM<sub>2.5</sub>, PM<sub>1</sub>, ultrafine particles*

Particles smaller than 1 micrometer (PM<sub>1</sub>) can penetrate into the alveolar region of the lung. PM<sub>2.5</sub> has a longer lifetime in the atmosphere and tends to have a even more homogenous distribution compared to PM<sub>10</sub>. (Gehrig et al. 2003)

Particles with diameters smaller than 0.1µm are referred to as ultrafine particles.

**Other direct, transport related, surrogates for air pollution**

*Black Smoke*

Black Smoke (BS) refers to a measurement method that uses the light reflectance of particles collected in filters to assess the "blackness" of the collected material. BS has been shown to be highly correlated with elemental carbon and to soot content. In modern urban settings, BS is closely related to diesel exhaust. (WHO Europe 2003)

*NO<sub>x</sub> and NO<sub>2</sub>*

Nitric oxide is a marker for combustion processes and an indicator of fresh exhaust-pipe emissions near roads. NO<sub>2</sub> is formed rapidly in the atmosphere from nitric oxide. In most

urban locations, NO<sub>2</sub> originates primarily from motor vehicles. NO<sub>2</sub> is a precursor for a number of other harmful secondary pollutants, such as the photo oxidant ozone. NO<sub>2</sub> levels are generally regarded to be a reasonable marker of exposure to traffic related emissions. (WHO Europe 2003)

## CO

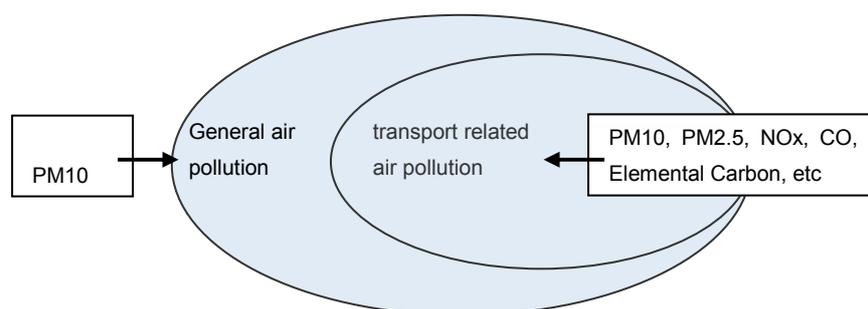
CO results from incomplete oxidation of carbon in combustion. WHO recently considered CO as having a reduced relevance as a traffic marker due to improved engines and fuels. (WHO Europe 2005)

## Benzene

Benzene is the "simplest" aromatic compound (six carbon atoms joined in a hexagonal ring). Gasoline motor vehicle exhaust is a source of benzene. Benzene is regarded as a marker of transport-related air pollution in general, and a more specific indicator of emissions from two-wheeled vehicles.

## PAHs (Polycyclic Aromatic Hydrocarbons) and benzo[a]pyrene

PAHs are organic chemicals that contain two or more aromatic benzene rings fused together. Vehicle emissions are the dominant source of PAHs in most urban areas. Some PAHs are carcinogenic, and benzo[a]pyrene is often used as a marker of the carcinogenic potency of ambient total PAHs. Roadside concentrations of benzo[a]pyrene in hot spots are in general three times higher than background levels when emissions from other sources (such as e.g. wood combustion) are minimal.



**Figure 4: Air pollution from different sources and examples for used surrogates for exposure**

## Indirect surrogates

Several indirect surrogates for exposure have been used in studies, such as traffic counts, distance to a major road, self reported traffic intensity, frequency of traffic jams at

intersections, etc. Some studies have used self-reported (perceived) exposure to road traffic. However, a drawback of this assessment is that it might overestimate the effects on (self-assessed) health.

### **Surrogate for transit traffic**

With respect to the goals of the project MONITRAF, a surrogate that represents the transit specific air pollution would be desirable. However, such a surrogate does not exist. Therefore, a surrogate representing transport-related air pollution was chosen (see Figure 4).

### **2.1.2. Different types of study examples based on different exposure measurements**

Which exposure concepts have been used so far in epidemiological studies to assess transport related air pollution effects on health? The following list gives examples of used methods to assess effect on health from transport related air pollution.

a) Use of a transport-related surrogate of air pollution and assessing its influence on mortality.

**Schwartz J, Laden F, Zanobetti A: The Concentration-Response Relation between PM<sub>2.5</sub> and Daily Deaths. *Env Health Persp* 2002, 110,10, 1025-1029**

"Harvard Six-Cities Study": Daily PM<sub>2.5</sub> measurements of one sampler per city were set in relation to daily deaths. They found a nearly linear relationship between air pollution and daily deaths with an increase of 1.5% in deaths in the case of an increase of mean 10µg/m<sup>3</sup> to 20µg/m<sup>3</sup> PM<sub>2.5</sub>. Restriction of the analysis to transport-related components of air pollution showed a 3% increase of deaths for the same air pollution increment.

b) long-term change (reduction) in air pollution levels

**Bayer-Oglesby L, Grize L, Gassner M, Takken-Sahli K, Sennhauser F H, Neu U, Schindler C, Braun-Fahrländer C. Decline of Ambient Air Pollution Levels and Improved Respiratory Health in Swiss Children. *Environ Health Perspect*. 2005 Nov;113(11):1632-7.**

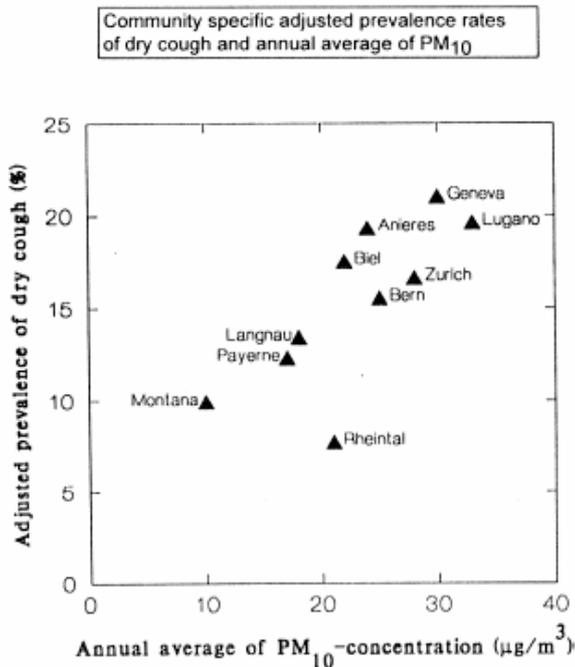
Bayer-Oglesby et al. investigated whether a rather moderate decline of air pollution levels in the 1990s in Switzerland was associated with a reduction in respiratory symptoms and diseases in school children. In nine Swiss communities, children participated in cross-sectional health assessments between 1992 and 2001. To each child, an estimate of regional particles since the first survey was assigned. Declining PM<sub>10</sub> was associated with declining prevalence of chronic cough, odds ratio per 10µg/m<sup>3</sup> decline was 0.65 (95% confidence interval 0.54–0.79), bronchitis, OR 0.66; (0.55–0.80), common cold, OR 0.78 (0.68–0.89), nocturnal dry cough OR 0.70 (0.60–0.83), and conjunctivitis symptoms OR 0.81 (0.70–0.95). The findings show that the reduction of air pollution exposures contributes to improved respiratory health in children.

c) High versus low exposure areas

**Braun-Fahrländer C, Vuille JC, Sennhauser FH, Neu U, Kunzle T, Grize L, Gassner M, Minder C, Schindler C, Varonier HS, Wüthrich B. Respiratory health and long-term exposure to air pollutants in Swiss schoolchildren. SCARPOL Team. *Swiss Study on Childhood Allergy and Respiratory Symptoms with Respect to Air Pollution, Climate and Pollen*.**

The impact of long-term exposure to air pollution on respiratory and allergic symptoms and illnesses was assessed in a cross-sectional study of schoolchildren (ages 6 to 15 yr, n = 4,470) living in 10 different communities in Switzerland. Air pollution measurements (PM<sub>10</sub>, NO<sub>2</sub>, SO<sub>2</sub>, and ozone) and meteorological data were collected in each community. Reported symptom rates of chronic cough, nocturnal dry cough, and bronchitis, adjusted for individual risk factors, were positively associated with PM<sub>10</sub>, NO<sub>2</sub>, and SO<sub>2</sub>. The strongest relationship was observed for PM<sub>10</sub> (adjusted odds ratios for chronic cough, nocturnal dry cough, and bronchitis between the most and the least polluted community for PM<sub>10</sub> were 3.07 (95% CI:

1.62 to 5.81), 2.88 (95% CI: 1.69 to 4.89), and 2.17 (95% CI: 1.21 to 4.89), respectively). The high correlation between the average concentrations of the pollutants makes the assessment of the relative importance of each pollutant difficult.



**Figure 5: Example of an exposure-response curve, in this case adjusted prevalence rates of dry cough and annual average of PM<sub>10</sub>. Source: (Braun-Fahrländer et al. 1997)**

d) multi-pollutant-models:

**Hirsch T, Weiland SK, von Mutius E, Safeca AF, Gräfe H, Csaplovics E, Duhme H, Keil U, Leupold W. Inner city air pollution and respiratory health and atopy in children. *Eur Respir J* 1999; 14: 669-677**

Hirsch et al. measured air pollution at 182 grid points within Dresden, Germany. Grid points had a distance of 1000m. Personal exposure of school children was estimated with data of the closest four measurement sites. Exposure at home and at school was calculated as a combined estimate. All measured traffic related pollutants, such as SO<sub>2</sub>, NO<sub>2</sub>, CO and Benzene increased the risk of bronchitis and morning cough. E.g., after adjusting for potential confounding factors an increase in the exposure to benzene of 1 µg/m<sup>3</sup> air was associated with an increased prevalence of morning cough, odds ratio 1.15 (95% C.I. 1.04-1.27) and bronchitis 1.11 (1.03-1.19).

Such multi-pollutant studies could give indication as to which pollutants are most relevant for human health. However, the use of single- versus multi-pollutant models may have a large influence on risk estimates. If the addition of a second pollutant changes the effect estimate for the pollutant of interest, this observation may be the result of collinearity among the pollutants, because the estimates can vary widely with the inclusion or exclusion of highly

correlated covariates. For example, in an Austrian panel study, the association between lung function parameters and air pollution, PM<sub>1</sub>, PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub> were assessed. The effect of PM<sub>2.5</sub> on PEF (peak expiratory flow, a measure of how fast a person can blow out) was estimated to result in a reduction of -0.41% per 10 µg/m<sup>3</sup> (p=0.027). However, when adjusted for NO<sub>2</sub>, this estimate was reduced to -0.18% for the same air pollution increment and was not statistically significant any more (p=0.4). (Moshhammer et al. 2006)

e) distinguishing between large-scale and small-scale effects

**Hoek G, Brunekreef B, Goldbohm S et al. Association between mortality and surrogates of traffic-related air pollution in the Netherlands: a cohort study. Lancet 2002; 360: 1203-9**

Long-term exposure to black smoke and nitrogen dioxide was estimated for the home address of a sample of 5000 people from the Netherlands Cohort study on Diet and Cancer. Exposure was characterised with the measured regional and urban background concentration and an indicator variable for living near major roads. Cardiopulmonary mortality was associated with living near a major road (relative risk 1.95, 95% CI 1.09-3.52). In addition, cardiopulmonary mortality increased by a factor of 1.34, (95%-CI: 0.68-2.64) per 10 µg/m<sup>3</sup> increase in the estimated ambient background concentration. In this assessment, both small- and large-scale air pollution contributions were relevant, but the effect on the more detailed scale was more important. Non-cardiopulmonary, non-lung cancer deaths were unrelated to air pollution.

By using this method an assumption is made that all individuals living within a certain distance of a major road are subjected to the same level of exposure, yet this is unlikely to be the case. Traffic on different roads varies, both in volume and in type, and meteorological conditions can alter dispersion of pollutants. However, advantage of this method is the rather easy measurement as compared to air pollutants.

f) traffic counts

**Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner C, von Mutius E. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. Eur Respir J 2003; 21: 956-963**

In this approach, Nicolai et al. used traffic counts in 50,150 and 300m perimeters around children's homes. Traffic counts in this perimeter were divided into low, medium and high traffic counts (e.g. 50m: 2600-15000 / 15001 – 30000, >30000 vehicles per day). They found that traffic counts were associated with current asthma, wheeze and cough. E.g. Odds ratio for current asthma was 0.232 (95% C.I. 0.032–1.700) for low, 1.130 (0.472–2.706) for medium and 2.047 (1.005–4.171) for high traffic counts.

The traffic count method has the advantage of being likely to be a more valid measure than distance to roads. Nevertheless, the actual exposure is influenced by many more factors.

Factors considered in the models so far included vehicle type and density, presence and type of buildings on a street, meteorological conditions, street width and distance from house to middle of the street amongst other factors. Some authors have analysed traffic counts from different vehicle types separately, suggesting truck pollution to be more detrimental to health than that from cars.

## **2.2. Exposure-response relationship for traffic related pollutants**

### **2.2.1. Evidence of health effects from transport related traffic**

What do we know about health effects of transport-related air pollution? In 2005, WHO published a report in which the evidence was summarised as following (WHO Europe 2005):

- *Birth outcomes*

There is "evidence" that implicates ambient air pollution in adverse effects on pregnancy, birth outcomes and male fertility. However, further studies are needed to estimate this more precisely, in terms of both pollution components and timing.

- *Non-allergic respiratory morbidity*

There is "rather substantial evidence" that points to transport-related air pollution as increasing the risk of non-allergic respiratory symptoms. Inflammatory processes are likely to be involved in the effect.

- *Allergic illness/ symptoms including asthma*

There is "rather substantial evidence" from laboratory experiments that transport-related air pollution can increase the risk of allergy development and exacerbation of allergic reactions. However, the results from population based studies are inconsistent.

- *Cardiovascular morbidity*

Only recent studies have focused on transport related air pollution and diverse indicators of cardiovascular diseases or functions of the cardiovascular system. Most studies show changes in various cardiovascular parameters without providing a consistent explanation of the possible mechanisms involved.

- *Cancer*

A few studies estimating the general population's exposure to transport-related air pollution suggest an increased incidence of lung cancer associated with increased exposure. Studies performed in susceptible subgroups or groups with higher levels of occupational exposure have indicated an increased risk in various types of cancer.

- *Mortality*

The few available studies that focus on transport-related air pollution indicate that it contributes substantially to the increased risk of death, particularly from cardiopulmonary causes.

### **2.2.2. Derivation of the exposure-response association**

Before an exposure-response curve can be transferred to the area of interest, this curve has to be estimated. Usually, for this purpose, two sorts of approaches are used: toxicological or epidemiological studies.

#### **Toxicology**

In toxicological studies, cells, animals or sometimes humans are exposed under controlled conditions and health related effects are measured. Toxicological studies can examine health effects of specific components, combined exposures to defined pollutants or exposures to ambient mixtures.

- Studies in humans

Disadvantages of studies in humans are that they are generally limited in duration, concentration range and the assessment of the range of endpoints.

- Animal/ cell studies

Both animal and cell studies have contributed to the knowledge on the hazardousness of air pollution. However, it is unclear whether these results can be extrapolated to humans. Cell studies are useful to explore the mechanisms of disease development. In this report, only epidemiological studies are considered.

#### **Epidemiology**

The advantage of epidemiological studies is their relevance to real life, in terms of both exposure patterns and coverage of target populations.

In epidemiological studies, study designs such as ecological, case-control-, time series or cohort designs are being used. All of these study types have inherent advantages and disadvantages. Epidemiological studies assess air pollution of populations at their living or work place and estimate the relationship to the frequency of e.g. illness, mortality, work absenteeism, medication, hospital admissions and so on. Thus, epidemiological studies are considered most suitable to derive quantitative exposure-response relationships, i.e. the excess risk per unit increase in the pollutant concentration. (Krzyzanowski et al. 2002)

### **2.2.3. Available publications**

One main criterion for an appropriate exposure surrogate in HIA is the availability of at least several studies assessing an exposure-response relationship for all health outcomes of interest. Table 1 gives an overview of epidemiological studies that display different concepts. This literature is based on papers selected for an evaluation by the World Health Organization, Regional Office for Europe, "Health effects of transport related air pollution", which represents the results of a systematic literature search. (WHO Europe 2005).

Health outcomes	birth outcomes	Respiratory morbidity children	Respiratory morbidity adolescents	Respiratory morbidity adults	Cardio-vascular morbidity	Cancer 1) childhood 2) adults	Mortality	Other: school / work absenteeism, fatigue
Surrogates								
PM2.5		(JANSSEN ET AL. 2003), (BRAUER ET AL. 2002) (GEHRING ET AL. 2002)		(BUCKERIDGE ET AL. 2002)	(PEKKANEN ET AL. 2002) (RIEDIKER ET AL. 2004)		(SCHWARTZ ET AL. 2002)	(CLENCH-AAS ET AL. 2000)
PM1 or ultrafine particles					(PEKKANEN ET AL. 2002)			
NO2	(HA ET AL. 2001) (LIU ET AL. 2003) (MAROZIENE ET AL. 2002)	(JANSSEN ET AL. 2003) (HIRSCH ET AL. 1999) (STUDNICKA ET AL. 1997) (BRAUER ET AL. 2002) (GEHRING ET AL. 2002) (NICOLAI ET AL. 2003) (PERSHAGEN ET AL. 1995) (OOSTERLEE ET AL. 1996) (BRAUN-FAHRLÄNDER ET AL. 1992) (GUO ET AL. 1999) (LEE ET AL. 2003) (KRÄMER ET AL. 2000) (FRIEDMAN ET AL. 2001)	(DE MARCO ET AL. 2002)	(FORSBERG ET AL. 1997) (OOSTERLEE ET AL. 1996)	(ATKINSON ET AL. 1999) (BALLESTER ET AL. 2001) (POLONIECKI ET AL. 1997) (D'IPPOLITI ET AL. 2003)	1) (NYBERG ET AL. 2000) 1) (NAFSTAD ET AL. 2003) 2) (FEYCHTING ET AL. 1998) 2) (RAASCHOU-NIELSEN ET AL. 2001)	(KATSOUYANNI ET AL. 2001) (LE TERTRE ET AL. 2002)	(CLENCH-AAS ET AL. 2000)
SO2	(HA ET AL. 2001) (LIU ET AL. 2003)	(BRAUN-FAHRLÄNDER ET AL. 1992) (HIRSCH ET AL. 1999) (GUO ET AL. 1999) (LEE ET AL. 2003)		(FORSBERG ET AL. 1997)	(ATKINSON ET AL. 1999) (BALLESTER ET AL. 2001) (POLONIECKI ET AL. 1997) (D'IPPOLITI ET AL. 2003)	1) (NYBERG ET AL. 2000) 1) (NAFSTAD ET AL. 2003)	(LE TERTRE ET AL. 2002) (HEDLEY ET AL. 2002)	

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CO	(HA ET AL. 2001) (LIU ET AL. 2003)	(HIRSCH ET AL. 1999) (GUO ET AL. 1999) (LEE ET AL. 2003)		(ATKINSON ET AL. 1999) (BALLESTER ET AL. 2001) (POLONIECKI ET AL. 1997) (D'IPPOLITI ET AL. 2003)	
Black Smoke		(STEERENBERG ET AL. 2001)	(FORSBERG ET AL. 1997)	(ATKINSON ET AL. 1999) (BALLESTER ET AL. 2001) (POLONIECKI ET AL. 1997)	(KATSOUYANNI ET AL. 2001) (LE TERTRE ET AL. 2002) (BALLESTER ET AL. 2002)
Soot		(JANSSEN ET AL. 2003) (BRAUER ET AL. 2002) (GEHRING ET AL. 2002) (NICOLAI ET AL. 2003),			
Benzene		(HIRSCH ET AL. 1999), (NICOLAI ET AL. 2003),	(HAGEN ET AL. 2000) (OFTEDAL ET AL. 2003)		2) (RAASCHOU-NIELSEN ET AL. 2001).
Benzo[a]pyren, toluene, formaldehyde, 1-hydroxypyrene (1-HP)			(NORTHRIDGE ET AL. 1999)	(HAGEN ET AL. 2000), (OFTEDAL ET AL. 2003)	
Distance to a major road/ distance to a petrol station		(VENN ET AL. 2001), (EDWARDS ET AL. 1994) (LIN ET AL. 2002) (WALDRON ET AL. 1995) (WILKINSON ET AL. 1999)	(NITTA ET AL. 1993)		2) (HARRISON ET AL. 1999) (ROEMER ET AL. 2001) (HOEK ET AL. 2002)
Traffic counts		(BRUNEKREEF ET AL. 1997) (VAN VLIET ET AL. 1997)	(Wylers et al. 2000)		2) (Reynolds et al. 2004)

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		(WJST ET AL. 1993)		
		(VENN ET AL. 2000)		
		(NICOLAI ET AL. 2003)		
		(ZMIROU ET AL. 2004)		
		(CICCONE ET AL. 1998)		
		(WYLER ET AL. 2000)		
		(JANSSEN ET AL. 2003)		
Self reported traffic intensity		(CICCONE ET AL. 1998)	(DUHME ET AL. 1996)	(LERCHER ET AL. 1995)
		(LERCHER ET AL. 1995)		(FORSBERG ET AL. 1997)
		(FORSBERG ET AL. 1997)		
Other: frequency of traffic jams at residences/ traffic flow/ "exposure to traffic", traffic density	(RITZ ET AL. 1999; RITZ ET AL. 2000; WILHELM ET AL. 2003)	(EDWARDS ET AL. 1994)		(Peters et al. 2004)
Change in fuel composition		(WONG ET AL. 1998)		

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**Table 1: Epidemiological studies only, no laboratory studies with controlled exposure, no workplace exposure or cell-/animal studies.**

## **2.3. Proposed method for a health impact assessment**

### **2.3.1. Surrogate selection**

#### *Primary surrogate*

Many air pollutants are highly correlated in the ambient air due to their common source. Summarising health effects from different surrogates would lead to an overestimation of the overall health impact. As a consequence, a primary air pollution surrogate should be used in the HIA. In epidemiologic studies, a range of surrogates are being evaluated. As long as the chosen primary surrogate displays spatial correlation with the respective other surrogates, conversion factors can be used to transfer the exposure-response relationships between the surrogates and the respective health effects.

#### *Not appropriate surrogates*

Both PM<sub>2.5</sub> and PM<sub>10</sub> display a relatively homogenous spatial variability and therefore do not represent the detailed-scale contrasts in exposure that are attributable to road traffic in the Alpine space. In addition, they are not source specific with regard to transport-related air pollution. They are therefore not appropriate surrogates to assess health impact from traffic specific air pollution on a detailed scale.

SO<sub>2</sub> is not a traffic specific surrogate.

CO is not considered to be a good surrogate of traffic related exposure anymore as it is emitted from other sources as well and CO emissions from traffic were reduced due to improved engines and fuel. (WHO Europe 2005) WHO also concluded that "nitric oxide is a better marker for traffic air pollution near roads, though nitrogen dioxide is a more relevant marker for adverse health effects".

The air pollutants benzene and PAH's have been linked with cancer. The effect of these surrogates is not specific for the mixture that it is thought to represent. For this reason, benzene and PAH's were excluded from the list of appropriate surrogates for this HIA.

#### *NO<sub>2</sub> as primary surrogate*

For a HIA in the Alpine transit areas, we propose to select NO<sub>2</sub> as a primary surrogate for several reasons: NO<sub>2</sub> is

- a traffic specific indicator and displays spatial heterogeneity on a detailed scale ("useful for estimation of the spatial variation in traffic-related pollution" (Lewné et al. 2004))
- has been evaluated in a range of epidemiological studies on the effect of air pollution on health in Europe,
- is being monitored in several spots in the Alpine transit area,

- there is expertise in modelling the spatial distribution in a Geographic Information System e.g. in Switzerland. Although this models needs improvement, this expertise might be useful in modelling exposure in the Alpine transit areas.

*Conversion factors: spatial correlation of NO<sub>2</sub> with other surrogates*

Several studies have compared temporal correlation of air pollution variables. (Ito et al. 2001; Qian et al. 2001; Ito et al. 2005) However, for health impact assessments, known spatial correlation between specific pollutants are of specific interest. For example, CO, BC and particle number concentrations were highly correlated in a study estimating the spatial correlation between these pollutants near a highway. The authors suggested that "these three pollutants can be used interchangeably to estimate the concentration of the other two pollutants near freeways". (Zhu et al. 2002) However, there is limited knowledge on the spatial correlation between transport-related air pollutants in general and potential differences across geographical regions.

Black Smoke, EC and soot as well as benzene and ultrafine particles display spatial heterogeneity. A next step for a HIA would include assessment of conversion factors, so that health effects of these surrogates can be converted into each other.

### **2.3.2. Selection of health outcomes**

Because health is not an entity that can be assessed with one parameter, a range of health outcomes are evaluated. In the context of air pollution, respiratory and cardiovascular morbidity and mortality are of main interest. The specific health outcomes (diagnoses) should be determined in a future HIA. Only diagnoses for which epidemiological studies are available can be considered. In order to avoid double counting of health outcomes, they must not overlap. Overlapping outcomes are for example cardiovascular hospital admissions and cases with a myocardial infarction (see chapter 4). If one aims at quantifying the costs related to the health effects from air pollution, this will affect the outcome selection. In this case all selected outcomes should be monetary quantifiable.

The following gives exemplary references chosen from Table 1 for the selected health outcomes. For a HIA, a literature search should be conducted to complement this list, and all relevant studies should be included.

In more detail, the health outcomes can be grouped into:

#### **a) respiratory outcomes**

##### **mortality**

- subgroup of total mortality: respiratory mortality (HOEK ET AL. 2002; LE TERTRE ET AL. 2002)

##### **morbidity**

## children/ adolescent respiratory morbidity

### self-reported outcomes

- self-reported respiratory symptoms including e.g. cough, wheeze or use of medication (BRAUN-FAHRLÄNDER ET AL. 1992; LERCHER ET AL. 1995; OOSTERLEE ET AL. 1996; STUDNICKA ET AL. 1997; VAN VLIET ET AL. 1997; GUO ET AL. 1999; HIRSCH ET AL. 1999; KRÄMER ET AL. 2000; BRAUER ET AL. 2002; DE MARCO ET AL. 2002; GEHRING ET AL. 2002; JANSSEN ET AL. 2003; LEE ET AL. 2003; NICOLAI ET AL. 2003)
- doctor-diagnosed asthma (STUDNICKA ET AL. 1997; VAN VLIET ET AL. 1997; GUO ET AL. 1999; HIRSCH ET AL. 1999; KRÄMER ET AL. 2000; BRAUER ET AL. 2002; DE MARCO ET AL. 2002; LEE ET AL. 2003; NICOLAI ET AL. 2003)

### measured outcomes

- sensitization to common allergens (HIRSCH ET AL. 1999; KRÄMER ET AL. 2000; JANSSEN ET AL. 2003; NICOLAI ET AL. 2003)
- acute care visits and hospitalizations for asthma (asthma events) and non-asthma events (PERSHAGEN ET AL. 1995; FRIEDMAN ET AL. 2001)
- Bronchial hyperresponsiveness (HIRSCH ET AL. 1999; JANSSEN ET AL. 2003)
- Lung function (BRUNEKREEF ET AL. 1997; HIRSCH ET AL. 1999; NORTHRIDGE ET AL. 1999; KRÄMER ET AL. 2000; STEERENBERG ET AL. 2001; JANSSEN ET AL. 2003; NICOLAI ET AL. 2003)

## adult respiratory morbidity

- lung cancer (adults) (NYBERG ET AL. 2000; NAFSTAD ET AL. 2003)
- self reported respiratory symptoms (FORSBERG ET AL. 1997); (OOSTERLEE ET AL. 1996)

## **b) cardiovascular outcomes**

### **mortality**

- subgroup of total mortality: cardiovascular mortality (HOEK ET AL. 2002; LE TERTRE ET AL. 2002)

### **morbidity**

- cardio-vascular morbidity: emergency hospital admissions for cardiovascular diseases (POLONIECKI ET AL. 1997; BALLESTER ET AL. 2001; D'IPPOLITI ET AL. 2003; PETERS ET AL. 2004), ST-segment depression (PEKKANEN ET AL. 2002)

## **c) other**

### **mortality**

- total mortality (KATSOUYANNI ET AL. 2001; ROEMER ET AL. 2001; BALLESTER ET AL. 2002; HOEK ET AL. 2002; LE TERTRE ET AL. 2002)

### **morbidity**

- childhood cancer: childhood leukaemia, central nervous system tumours (FEYCHTING ET AL. 1998; RAASCHOU-NIELSEN ET AL. 2001) or malignant lymphomas (RAASCHOU-NIELSEN ET AL. 2001)

- **general emergency hospital emergency visits** (HAGEN ET AL. 2000; OFTEDAL ET AL. 2003)
- **low birth weight** (HA ET AL. 2001; MAROZIENE ET AL. 2002; LIU ET AL. 2003); (RITZ ET AL. 1999; WILHELM ET AL. 2003) **and preterm birth** (RITZ ET AL. 2000; MAROZIENE ET AL. 2002; WILHELM ET AL. 2003), **intrauterine growth retardation** (LIU ET AL. 2003)
- **self-reported symptoms in adults** (CLENCH-AAS ET AL. 2000)

## **3. Transport related noise and health**

### **3.1. Exposure assessment**

#### **3.1.1. Source of transport related noise**

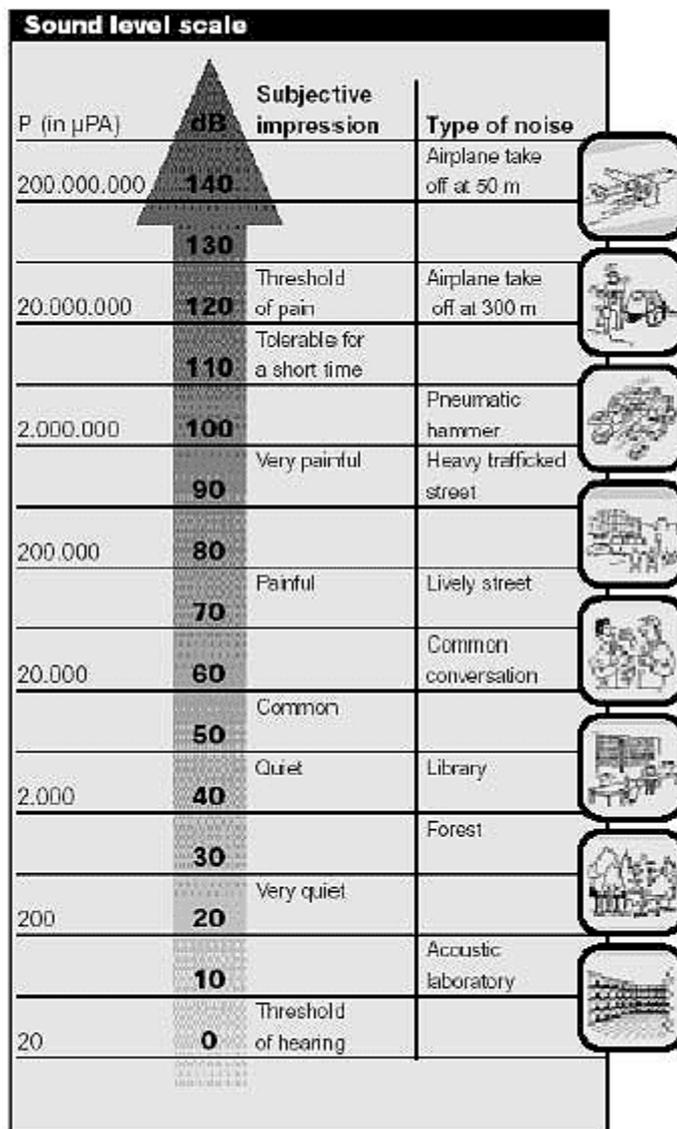
Noise is defined as unwanted sound. Transportation noise, including noise from road, rail and air traffic, is the main source of environmental noise pollution. Usually, larger and heavier vehicles emit more noise than smaller and lighter vehicles. Noise from roads is mainly generated from the engine and from frictional contact between the vehicle and the ground and air. Road contact noise will exceed engine noise at speeds higher than 60km/h. (Berglund et al. 1999) Sound pressure levels from traffic can be predicted from the traffic flow rate, the speed of the vehicles, the proportion of heavy vehicles and the nature of the road surface. Special problems, however, can arise in areas where the traffic movements involve a change in engine speed and power (e.g. hills), or where topography, meteorological conditions and low background levels are unfavourable (e.g. in mountain areas).

#### **3.1.2. Measurement of noise**

Because the range of sound pressure levels that human listeners can detect is very wide, these levels are measured on a logarithmic scale with units of decibels (dB). Each three-decibel increase doubles the acoustic pressure.

In the measurement of loudness, an A-weighting filter is commonly used to emphasise frequencies around 3–6 kHz where the human ear is most sensitive, while attenuating very high and very low frequencies to which the ear is insensitive. The aim is to measure loudness in a way so that it corresponds well with perceived loudness. These weighted measurements are also referred to as dB(A). Next to pitch or loudness of a sound, there are also other factors influencing the perception of noise, such as a repetitive nature of a sound, the distraction it causes and the lack of control over it. A voice in normal conversation is normally around 60 dB. Normal city or freeway traffic registers 70 dB (see Figure 6).

The sum of the total sound energy over some time period gives a level equivalent to the average sound energy over that time. Thus,  $L_{Aeq,T}$  is the energy average equivalent level of A-weighted sound over a period T.  $L_{Aeq,T}$  should be used to measure continuing sounds, such as road traffic noise. (Berglund et al. 1999) Standards for noise measurements have been defined by the International Organization for Standardization ([www.iso.org](http://www.iso.org)) in the ISO 1996-1:2003.



**Figure 6: Sound level scale in  $\mu\text{PA}$ , dB, subjective impression and type of noise. Source: World Health Organization, Regional Office Europe, [www.euro.who.int/eprise/main/WHO/Progs/NOH/Activities/20030724\\_1](http://www.euro.who.int/eprise/main/WHO/Progs/NOH/Activities/20030724_1)**

### Intermittent noise

Intermittent noise is more disturbing than continuous noise, regarding sleep parameters. Intermittent noise is often accompanied by continuous noise, as with particularly loud trucks passing in a background of general traffic noise. Under these conditions, it is better to measure both the long-term average ( $L_{\text{Aeq}}$ ) and the loudest sounds ( $L_{\text{Amax}}$ ). Number of noise events (e.g. number of events exceeding 50-55  $L_{\text{Amax}}$ ) are an additional factor that is used to explain effects on sleep quality. A range of studies have used  $L_{\text{Aeq, day}}$  or  $L_{\text{Aeq, night}}$  (usually in the hours between 6am and 10pm and vice versa) to assess effects of noise on health.

## 3.2. Exposure-response relationship for traffic related noise and health

### 3.2.1. Evidence of health effects of noise

It is common to classify noise related effects in a hierarchical order. Based on the classification proposed by Babisch (Babisch 2002), mortality represents the highest level and annoyance the lowest (see Figure 7). Ouis (Ouis 2001) argues that annoyance due to noise is the most common outward symptom of stress – which should therefore be considered as an indicator of more serious health problems. However, so far it is controversial in how far annoyance translates into health effects.

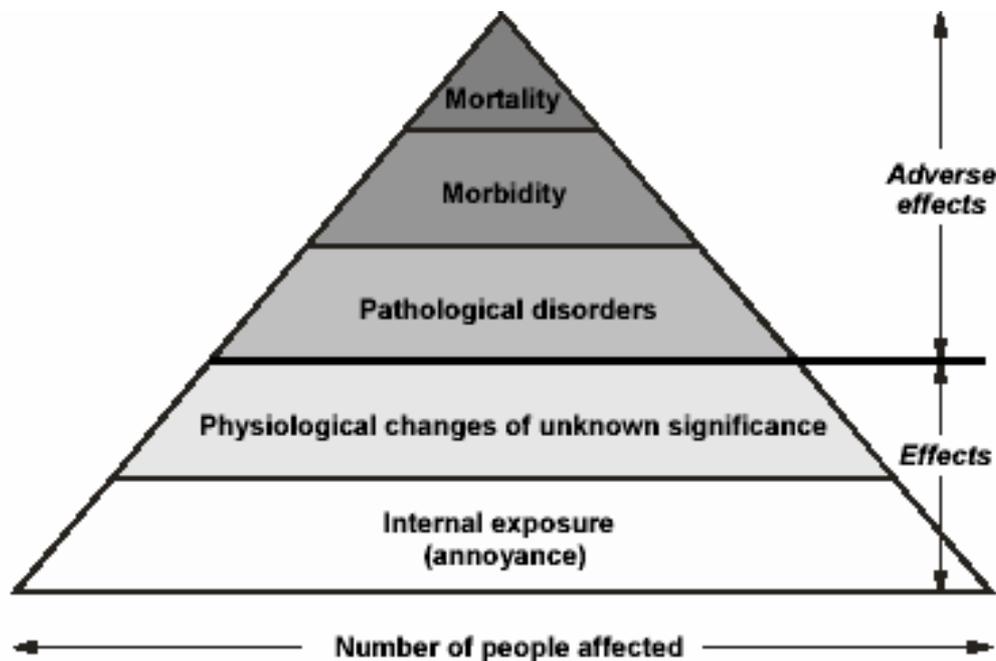


Figure 7: Grading of noise effects on health. Quelle: (Babisch 2002)

In 1999, WHO published a report that summarises adverse health effects of noise as following (Berglund et al. 1999):

- *Hearing impairment*

Hearing impairment is defined as an increase in the threshold of hearing, and may be accompanied by tinnitus. Hearing impairment is not expected to occur at  $L_{Aeq,8h}$  levels of 75dB(A) or below, or at levels of 70dB(A) at  $L_{Aeq,24h}$  even after a lifetime exposure. A consequence of hearing impairment, the inability to understand speech in daily living conditions, is considered to be a severe social handicap.

- *Speech intelligibility*

Speech interference is a process in which interfering noise renders speech incapable of being understood. The difference between speech level and interfering noise should be at least 15dB(A). Since the sound pressure of normal speech is at approximately 50dB(A),

noise levels of above 35dB(A) or more interferes with speech intelligibility in smaller rooms. The inability to understand speech results in a number of personal handicaps and behavioural changes. Especially vulnerable groups are e.g. the hearing impaired, the elderly and children in the process of language acquisition.

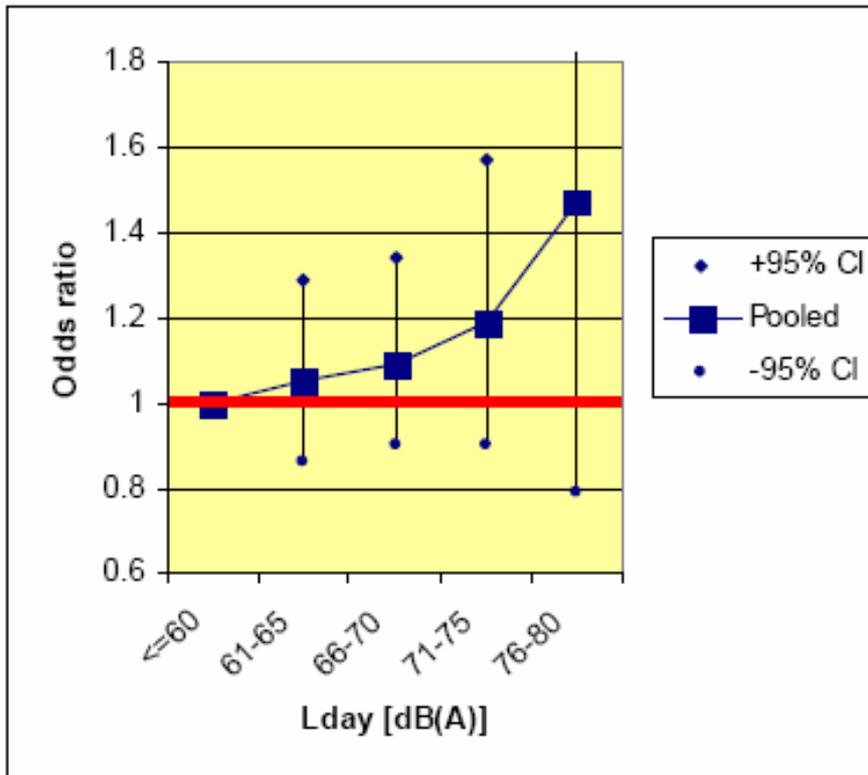
- *Sleep disturbance*

The difference between sound levels of a sound event and background noise, rather than the absolute noise level may determine probability of reaction. However, for a good night's sleep, WHO recommends levels not exceeding 30dB(A) for continuous background noise and 45dB(A) for noise events. Sleep disturbances are divided into primary effects (e.g. difficulties falling asleep, awakenings, increased blood pressure or heart rate) and secondary effects on the following day(s), such as increased fatigue, depressed mood or well-being or decreased performance.

In a technical meeting on exposure-response relationships of noise on health, strongly deviating exposure-response relationships were developed for sleep disorders. (WHO Europe 2003) This is due to the fact that assessing sleep disturbance by noise exposure is difficult to measure. There are large differences between laboratory and field studies ("habituation phenomenon"), several ways of assessing the exposure towards noise have been applied and a whole range of sleep disorder-associated outcomes have been assessed. (Carter 1996; Franssen et al. 2003)

- *Cardiovascular effects*

After prolonged exposure, susceptible individuals may develop permanent effects such as hypertension and ischaemic heart disease. Magnitude and duration of effects are determined by individual characteristics, lifestyle behaviour and environmental conditions. Cardiovascular effects have been shown to occur after long-term exposure to air- and road-traffic with  $L_{Aeq,24h}$  values of 65-70dB(A). Babisch (Babisch 2006) developed an exposure-response relationship for road traffic noise and the incidence of myocardial infarction (see Figure 8).



**Figure 8: pooled effect estimates for the association between road traffic noise level and the incidence of myocardial infarction (odds ratio and 95% CI). Source: (Babisch 2006)**

- *Mental illness*

Environmental noise is believed to accelerate and intensify the development of latent mental disorders. However, the findings on environmental noise and mental-health effects so far are inconclusive. (Stansfeld et al. 2000)

- *Performance*

Noise can adversely affect performance, especially in the case of more complex cognitive tasks. Reading, attention and memorisation are strongly affected by noise. Noise may also increase errors at work and some accidents may be an indicator of performance deficits.

- *Annoyance*

Annoyance effects of noise are often complex, subtle and indirect. Equal levels of noise can cause different magnitudes of annoyance, because annoyance also depends on other factors, such as social, psychological and economic factors.

Annoyance is a highly subjective concept that can only be explained to a small extent by the measured acoustic pressure, e.g. Ising reports prediction of a maximum of 33% of the individual annoyance that can be explained by acoustic parameters. (Ising et al. 2004). This percentage is even lower for aircraft noise. (Brink et al. 2006) The source of noise is therefore a relevant factor for assessment of perceived annoyance (see Figure 9).

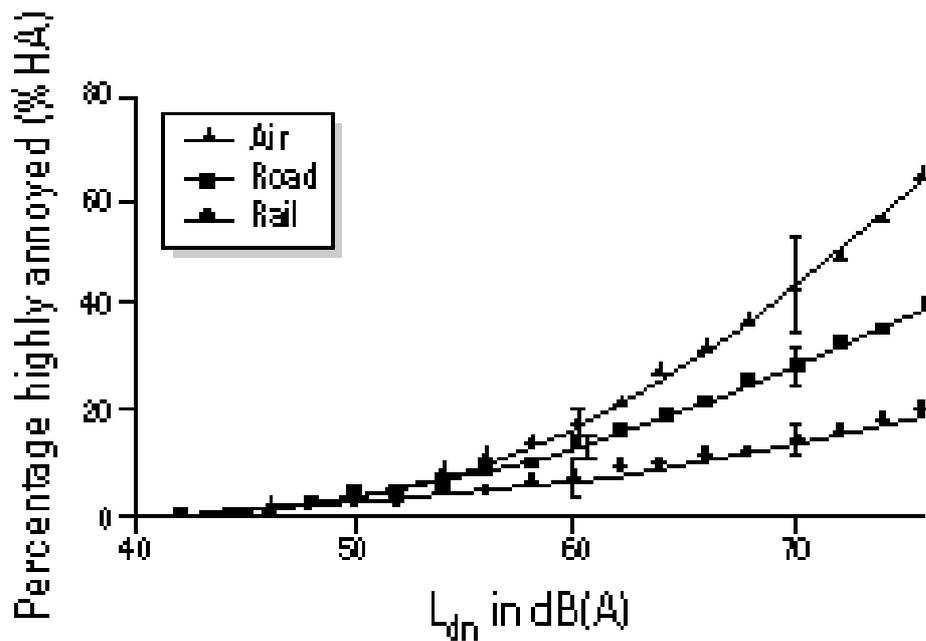


Figure 9: Relationships between the percentage of highly annoyed persons and  $L_{dn}$  for air, road, and railway traffic noise. The vertical bars at 60 and 70 dB(A) represent 95% confidence intervals. Source: (Miedema et al. 1998; Passchier-Vermeer et al. 2000)

### 3.2.2. Available studies

The following table includes peer-reviewed, epidemiological studies only. A range of publications is available as reports only, some are quoted in the text.

Health outcomes	Hearing impairment	Speech interference	Sleep disturbance	Physiological functions, etc.	Cardio-vascular morbidity	Mental health and well-being in general	Performance, memory tests, etc.	Annoyance
Noise exposure								
Modelled or measured		(HALL ET AL. 1985)	(SASAZAWA ET AL. 2004) (ÖHRSTRÖM 2004) (BLUHM ET AL. 2004) (WEHRLI ET AL. 1978) (EBERHARDT 1988) (LANGDON ET AL. 1977) (WILKINSON ET AL. 1984) (GRIEFAHN ET AL. 1986) (HALL ET AL. 1985) (MEIJER ET AL. 1985) (ÖHRSTROM 1989) (ÖHRSTROM 1991) (YOSHIDA ET AL. 1997) (YOSHIDA ET AL. 1997) (XIAOTU 1990) (GARCIA ET AL. 1990) (NIVISON ET AL. 1993) (LERCHER ET AL. 1996) (BELOJEVIC ET AL. 1997)	(VALLET ET AL. 1983) (WILKINSON ET AL. 1984) (GRIEFAHN ET AL. 1986) (HOFMAN ET AL. 1995) (CARTER ET AL. 1994) (YOSHIDA ET AL. 1997) (BELOJEVIC ET AL. 2002) (EVANS ET AL. 2001) (HERBOLD ET AL. 1989) (PULLES ET AL. 1990)	(BABISCH ET AL. 2003) (BABISCH ET AL. 2005) (MASCHKE 2003) (BABISCH ET AL. 2005) (HOEK ET AL. 2002) (REGECOVA ET AL. 1995) (TOBIAS ET AL. 2001)	(LERCHER ET AL. 2002) (STANSFELD ET AL. 1996) (MASCHKE 2003) (ÖHRSTROM 1991) (YOSHIDA ET AL. 1997) (LERCHER ET AL. 1996) (GOMEZ-JACINTO ET AL. 1999)	(STANSFELD ET AL. 2005)	(BABISCH ET AL. 2003) (SATO ET AL. 2002) (ALI 2004) (BLUHM ET AL. 2004) (RYLANDER ET AL. 2002) (WALLENIUS 2004) (MEIJER ET AL. 1985) (LERCHER ET AL. 1996)
Self assessment					(BELOJEVIC ET AL. 2002)			(HOFFMANN ET AL. 2003) (NIEMANN ET AL. 2005)

Traffic counts

(EBERHARDT 1988)

(KAGEYAMA ET AL.  
1997)

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**Table 2: peer-reviewed epidemiological studies only, no laboratory studies with controlled exposure, no workplace exposure or cell-/animal studies.  
No studies on aircraft-noise.**

### **3.3. Proposed method for a health impact assessment from noise**

#### **3.3.1. Surrogate selection**

##### *Primary surrogate*

In contrast to the air pollution surrogate  $\text{NO}_2$ , we propose to select more than one primary surrogate for noise. A-weighted sound levels over a period  $T$ , the  $L_{\text{Aeq},T}$ , have been recommended for measuring continuing sounds, such as road traffic noise. (Berglund et al. 1999) For the outcome sleep disorders, as a more appropriate measurement, the loudest sounds ( $L_{\text{Amax}}$ ) has been recommended. (WHO Europe 2003) Number of noise events (e.g. number of events exceeding 50-55  $L_{\text{Amax}}$ ) are also a factor that is used to explain effects on sleep quality. However, many of the studies assessing sleep disorders rely on  $L_{\text{Aeq, night}}$ . (Franssen et al. 2003) In addition to  $L_{\text{Aeq}}$ , the noise source must be known for evaluating the impact of noise on annoyance.

We propose to select  $L_{\text{Aeq,day}}$  and  $L_{\text{Aeq,night}}$  as primary surrogate for the HIA. In order to be able to estimate population annoyance from noise,  $L_{\text{Aeq},T}$  –values must be available for traffic separately.

##### *Conversion factors*

If the source of the sound (in this case transit traffic in the Alpine region) is known and continually displays similar patterns, it might be possible to convert health effects from  $L_{\text{Amax}}$  and noise events into  $L_{\text{Aeq}, T}$ . Potential conversion factors into  $L_{\text{Aeq},T}$  should be evaluated for the project MONITRAF.

##### *Noise cadastral data*

Available noise cadastral data usually display yearly average levels in the day or in the night (see e.g. Linz: [www.linz.at/Umwelt/umwelt\\_10125.asp](http://www.linz.at/Umwelt/umwelt_10125.asp), or Basel: [www.geo-bs.ch/stadtplan\\_laermkataster\\_karte.cfm](http://www.geo-bs.ch/stadtplan_laermkataster_karte.cfm) or Zürich: [www.geo-bs.ch/stadtplan\\_laermkataster\\_karte.cfm](http://www.geo-bs.ch/stadtplan_laermkataster_karte.cfm)). Such data are necessary to evaluate population exposure for a HIA in the Alpine region.

#### **3.3.2. Selection of health outcomes**

Noise could have an effect on a range of health outcomes. There is a large body of publications on noise and health originating from the 1980' and since then, for several health outcomes, publications on exposure-response relationships for sleep disorders, myocardial infarction and annoyance have emerged. These outcomes were considered in our report.

### Sleep disorders

The largest body of publications addresses perceived sleep quality and this measure seems to be the most promising to include into a quantitative meta-analysis. (Franssen et al. 2003)

### Cardiovascular diseases

The association between the risk of myocardial infarction (MI) and noise was described in a review by Babisch (see Figure 8). (Babisch 2006) No exposure-response association could be derived for hypertension and noise.

### Annoyance

Exposure-response relationships for noise annoyance have been derived for exposure to the three main types of transport noise: road, railway, and aircraft. The relationships shown in Figure 9 refer to populations chronically exposed to noise at specified levels for periods of more than a year.

The source of noise is therefore a relevant factor for the assessment of perceived annoyance. Because annoyance is such a subjective concept, for a HIA within the project MONITRAF, it would be preferable if an exposure-response relationship can be extracted from the literature that relies on data from residents of Alpine regions.

## **3.4. Available noise cadastral data in the Alpine transit areas**

Noise measurement (points) of the MONITRAF Region were described in (ökoscience 2005) and will not be repeated here.

In Austria, noise cadastral maps are being developed ([www.umweltbundesamt.at/umweltschutz/laerm/laermenschutz/b\\_laerm\\_g/laermkarten/](http://www.umweltbundesamt.at/umweltschutz/laerm/laermenschutz/b_laerm_g/laermkarten/)). The project will be finished in 2012.

In Switzerland, within the project MfM-U a noise cadastral map will be developed.

## **4. Future steps**

The following summarises (a) where we stand and (b) which next steps are necessary (or which data must be available) for a HIA.

### **4.1. Steps for a HIA of air pollution on health**

#### **4.1.1. Step1: Definition of the area of interest**

- a) First, the region of interest should be properly defined. Does MONITRAF consider only the Alpine transit corridors? If so, start, and width of these corridors should be defined. What about e.g. cities like Lugano, Lucerne or Verona, which also get a fair amount of traffic due to the Alpine transit transport? One might take into account that most likely the health impact from transit traffic is much higher in the lowlands due to the larger exposed population.
- b) To define the area of interest in accordance with the specific project goals of MONITRAF.

#### **4.1.2. Step 2: select a primary surrogate**

- a) Many air pollutants are highly correlated in the ambient air. Summarising health effects from different surrogates would lead to an overestimation of the overall health impact and as a consequence, a primary air pollution surrogate should be used in the HIA. For the reasons delineated in chapter 2.3.1, we propose to select NO<sub>2</sub> as primary surrogate.
- b) (done)

#### **4.1.3. Step 3: evaluate conversion factors of other pollutants into NO<sub>2</sub>**

- a) A range of surrogates for air pollution have been evaluated in epidemiologic studies. As long as the chosen primary surrogate is correlated with the respective other surrogates, conversion factors can be used to transfer the exposure-response relationships between the other surrogates into the primary surrogate, NO<sub>2</sub>. Black Smoke, EC and soot as well as ultrafine particles display spatial heterogeneity. So far there are few studies that assess spatial correlation between NO<sub>2</sub> and these surrogates.
- b) Step three for a HIA in the Alpine transit areas covers the evaluation of conversion factors. This step could be implemented e.g. in the next work package of the project MONITRAF; data analysing measurements of these pollutants of the Alpine space are required.

#### **4.1.4. Step 4: assess population exposure of NO<sub>2</sub>**

- a) The HIA requires knowledge about the distribution of NO<sub>2</sub>-exposure of the population in the respective area. This means that annual mean exposure levels, preferably in 5µg/m<sup>3</sup> increments, must be known. In Switzerland, there exists a NO<sub>2</sub>-exposure model in a Geographical Information System (GIS). In this model, NO<sub>2</sub> concentrations are simulated on the basis of known NO<sub>x</sub> emissions. This model displays a 200m-grid. However, the model does not predict exposure reliably enough for a HIA. The model is currently being revised and a meeting between the agency in charge of the modelling and the MfM-U project will take place in May 2006.
- b) In order to avoid replication of the model revision, it would be sensible to await the outcome of the meeting in May 2006, before planning a potential NO<sub>2</sub> GIS-model for the project MONITRAF. Annual mean exposure distribution in the population must then be modelled in an adequate resolution for the defined area. This might pose some difficulties, as data concerning emission sources (see (Bafu 2004)) must be available for each of the participating four countries. It might be useful to integrate the existing modelling expertise into an NO<sub>2</sub> GIS model of the Alpine transit areas. Another approach would be to use passive sampler measurements of NO<sub>2</sub>. However, this approach is considered less efficient and reliable.

#### **4.1.5. Step 5: select health outcomes**

- a) Health can not be measured with one parameter. The selected health outcomes must consider the publications of epidemiologic studies that evaluated the respective transport-related air pollution surrogates, reported the air pollution levels, exposure-response relationships and the statistical precision of this estimate. As can be seen from the list in chapter 2.3.2, health outcomes from the available literature include respiratory and cardiovascular morbidity, mortality (with the subgroups: total, cardiovascular or respiratory mortality), years of life lost and birth outcomes.
- b) Needs to be done in accordance with the exact goals of MONITRAF.

#### **4.1.6. Step 6: Derive exposure-response association**

- a) For each selected outcome an exposure-response relationships has to be derived from the literature. This may either be obtained from the most suitable epidemiological study that represents the condition in the Alpine area most adequately. If there is no such study, the exposure-response association will be calculated as a pooled effect estimate from all available and appropriate studies by means of meta-analytical statistical techniques.
- b) Next work step is to perform a systematic literature review to identify suitable studies to derive the exposure-response associations for NO<sub>2</sub>. For each outcome the most

suitable study has to be selected or a pooled exposure-response association has to be calculated.

#### **4.1.7. Step 7: Assess base prevalence/ incidence of health effect**

- a) In order to be able to quantify cases of illness or death attributable to transport related air pollution, the base prevalence or incidence of the respective health outcome (see step five) must be known. Necessary are standardised statistics (e.g. mortality, hospital statistics) or representative surveys (e.g. respiratory symptoms).
- b) For step seven, population frequencies of prevalence and/or incidence of all selected outcomes must be collected. It might be cumbersome to obtain adequately standardised data from regions out of four different countries.

#### **4.1.8. Step 8: Evaluate reference concentration**

- a) In a HIA, a reference scenario has to be chosen. It is usually not justified to quantify health effects down to the zero level: On the one hand nobody is exposed to such low levels. On the other hand there are no studies available which show health effects at very low pollutant levels. For example, studies assessing the health effects of PM<sub>10</sub> have not been performed in areas with annual levels below 5-10µg/m<sup>3</sup>. Therefore, in a HIA of general air pollution, a reference level of 7.5µg/m<sup>3</sup> (mean of lowest assessed exposure level) was chosen. (ARE et al. 2004) Only health effects above this level were quantified. One has to be aware that the selected level will substantially affect the result of the HIA.
- b) Evaluate the reference concentration for NO<sub>2</sub>.

#### **4.1.9. Step 9: Quantify health impact**

- a) From the population exposure and the exposure-response association attributable cases can be quantified. "Attributable" refers to the proportion of illness or deaths that can be ascribed to NO<sub>2</sub>-air pollution exposure of the population.
- b) Estimate attributable cases.

### **4.2. Steps for a HIA of noise on health**

According to the HIA of transport-related air pollution on health, the same eight steps are discussed. Again, the points summarise (a) where we stand and (b) which next steps are necessary (or which data must be available) for a HIA. In general, most of the points that are important for the HIA of air pollution on health, also apply for a HIA of noise on health.

#### **4.2.1. Step 1: Definition of the area of interest**

- parallel to point 4.1.1-

#### **4.2.2. Step 2: select a primary surrogate**

- a) The energy average equivalent level of A-weighted sound over a period is used. The surrogate to measure noise is the  $L_{Aeq, day}$  and  $L_{Aeq, night}$  for traffic.
- b) (done)

#### **4.2.3. Step 3: evaluate conversion factors**

- a) The loudest sounds ( $L_{Amax}$ ) and the number of noise events (e.g. number of events exceeding 50-55  $L_{Amax}$ ) are important surrogates for sleep outcomes. If the source of the sound (in this case transit traffic in the Alpine area) is known and continually displays similar patterns, it might be possible to convert health effects from  $L_{Amax}$  and noise events into  $L_{Aeq, night}$ .
- b) Step three covers the evaluation whether there are conversion factors of noise, i.e.  $L_{Amax}$  and number of noise events into  $L_{Aeq, day}$  and  $L_{Amax, night}$ . This step could be implemented in the next work package of the project MONITRAF.

#### **4.2.4. Step 4: assess population exposure of noise**

- a) As with the  $NO_2$ -exposure-model, a HIA of noise on health requires the population exposure of noise.
- b) Noise cadastral data are necessary for the HIA, these must be specific for traffic noise and cover  $L_{Aeq, day}$  and  $L_{Aeq, night}$ .

#### **4.2.5. Step 5: select health outcomes**

- a) Health outcomes selected from the literature cover cardiovascular morbidity, sleep disorders and annoyance.
- b) Needs to be done in accordance with the exact goals of MONITRAF.

#### **4.2.6. Step 6: Perform meta-analysis to assess effect estimates**

- parallel to point 4.1.6 –

#### **4.2.7. Step 7: Assess base prevalence/incidence of health effect**

- parallel to point 4.1.7 –

#### **4.2.8. Step 8: evaluate reference value**

- a) In step seven, the "reference concentration" of noise refers to the exposure level of zero effect. E.g. below 60dB(A) for  $L_{day}$ , no notifiable increase in MI was observed, and this level was defined as NOAEL ("no observed adverse effect level") by Babisch (2006).
- b) Zero effect levels of noise on health have to be extracted from the literature for the respective health outcomes.

#### **4.2.9. Step 9: quantify health impact**

- parallel to point 4.1.9 -

### **5. Conclusion**

We conclude that a HIA in the alpine area for air pollution and noise emission from transit traffic is a feasible and cost-effective way to quantify health impact from transit traffic. Whereas PM<sub>10</sub> is an appropriate surrogate to assess health impact from the general air pollution for a larger area, we consider NO<sub>2</sub> a more appropriate surrogate to represent the small area air pollution distribution in the alpine area from emissions of the transit traffic.

## 6. APPENDIX

### 6.1. Work steps M. Rössli/ A. Huss

- Participation at Workshop and Conference: Dr. M. Rössli and Dr. A. Huss; oral presentation by Dr. Rössli in Innsbruck, 4<sup>th</sup>-5<sup>th</sup> Oct 2005 on "Assessment of Health Impact from Transit Traffic – State of the Art and Outlook". Oral presentations by Dr. Huss in Lucerne, 2<sup>nd</sup> Dec 2005 on "Assessment of public health impact from alpine transit traffic: methods and limitations" and 9<sup>th</sup> of March 2006 in Bozen/ Bolzano.
- Literature search air pollution and noise and health.
- Overview over methods and methodological limitations.
- Selection of an air pollution surrogate from the literature that represents the spatial heterogeneity and source specificity of transport-related exposures.
- Selection of a surrogate for noise.
- Selection of health outcomes from the literature that can be used to assess effects of exposures on health in the Alpine transit areas.

### 6.2. overview over ongoing studies in the Alpine space

- Contact to Dr. M. Hazenkamp, Institute of Social and Preventive Medicine, Basel, coordinator in the project MfM-U. Covered subjects in the framework of this project include:

#### Exposure:

- Air pollution: Black Carbon or Elemental Carbon were listed as indicators for diesel exhaust and NO<sub>2</sub> as an indicator for transport-related air pollution in general. For NO<sub>2</sub> there exists already a GIS (geographic information system) which is currently being improved with respect to the challenges of modelling the exposure in Alpine valleys. Results will be discussed in May 2006, a report can be expected in 2007.
- Traffic counts are now available. These will be used for validation of a GIS-traffic model for Switzerland. However, so far this model still has methodological limitations, e.g. a linkage to person-exposure is difficult. In addition, traffic count modelling including freight traffic is not yet available, but expected by 2007.
- A noise cadastral map will be developed.

#### Health effects:

- Evaluations of health effects of Alpine transit traffic so far refer to the studies SCARPOL and SAPALDIA (see also [pages.unibas.ch/ispmb/](http://pages.unibas.ch/ispmb/)). However, these

studies do not include enough participants living close to highways for a reliable estimate. A questionnaire study that aims at filling up these data gaps has been conducted in areas along the A2 and A13 highway by the department "Landschaft und Gesellschaft" der Eidg. Forschungsanstalt für Wald, Schnee und Landschaft (WSL)". In this questionnaire, an annoyance-score was inquired and supplemental questions were integrated that assessed respiratory symptoms as well as some risk factors (such as smoking). 1700 persons were surveyed. Data analysis of this survey will start in April 2006, a publication or report can be expected in 2007.

- For the project MONITRAF, it would be desirable if an exposure-response relationship for noise and annoyance could be used that is derived from reports of residents of the Alpine region. Such a – regionally assessed – exposure-response relationship is more likely to take account of the subjective nature of annoyance. However, so far it is unclear when results of the WSL-survey might be expected.

There is potential for synergy of the Project MONITRAF with the projects MfM-U (see [www.umwelt-schweiz.ch/buwal/de/fachgebiete/fg\\_ubeobacht/rubrik3/mfm-u/index.html](http://www.umwelt-schweiz.ch/buwal/de/fachgebiete/fg_ubeobacht/rubrik3/mfm-u/index.html)) and ALPNAP (see [www.alpnep.org](http://www.alpnep.org)).

- The first three-year project phase of MfM-U is now (March 2006) finished. If MONITRAF wants to cooperate with MfM-U, a contact should be established preferably in April 2006, so that a potential cooperation can be planned and included in the next project phase. Contact person is Klaus Kammer, Bundesamt für Umwelt (BAFU); 3003 Bern, Tel. +41 (0)31 323 03 10, email [klaus.kammer@bafu.admin.ch](mailto:klaus.kammer@bafu.admin.ch).
- The contact between the projects ALPNAP and MONITRAF has already been established. A cooperation within ALPNAP-work packages 5 (Traffic Monitoring and Emission Data) and 7-10 (Air Pollution - Monitoring and Prediction, Noise - Monitoring and Prediction Impact Assessment, Planning and Integration) is indicated. Contact person is Dr. Dietrich Heimann, DLR Institut für Physik der Atmosphäre, 82234 Oberpfaffenhofen, Tel +49 (0)8153 28 2508.

### 6.3. Glossary

Attributable cases	Refers to the number of cases of illness or death that would not have occurred if the air pollution were at or below the reference level.
BS	Black smoke, a measurement that uses the light reflectance of particles collected on filters to assess the "blackness" of the collected material.
Cardiovascular disease	Disorders of the heart and circulatory system.
CI	Confidence interval (and 95% CI). The computed interval with a given probability, e.g. the 95%, that the true value of a variable such as a mean, proportion, or rate is contained within the interval.
CO	Carbon monoxide
CO <sub>2</sub>	Carbon dioxide
dB	Decibel, a unit for expressing the relative intensity of sounds on a logarithmic scale from zero for the average least perceptible sound to about 130 for the average level at which sound causes pain to humans. For traffic and industrial noise measurements, the A-weighted decibel dB(A) is widely used.
dB(A)	Unit of weighted sound pressure levels. The A-weighted decibel scale corresponds approximately to the frequency response of the human ear and thus correlates well with perceived loudness.
Dose-response-relationship	A dose-response relationship is said to exist when a change in the exposure is associated with a change of the outcome.
EC	Elemental carbon
Epidemiology	The study of disease patterns in populations and the factors that influence these diseases.
Exposure-response-relationship	see dose-response relationship
GIS	Geographical Information System
HIA	Health impact assessment
L <sub>Aeq</sub>	The "equivalent" average sound level measured using the A-weighting.
L <sub>Amax</sub>	Maximum A-weighted noise level (noise metric for a single event)
Longitudinal studies	Epidemiological techniques that involve following populations or individuals over a period of time
Map scale	A map scale refers to the extent to which reality is reduced to display it on a map. Large scale maps (e.g. 1:1000) show a small are of the Earth's surface in a lot of detail. Small scale maps (e.g. 1:1000 000) show large areas in very little detail. Due to potential confusing of the German word "kleinräumig" and English "small-scale", the wording "detailed scale" was used in the text.
MfMU	The Swiss project "Monitoring Flankierende Massnahmen (MFM)" has two subprojects: The MFM-Umwelt (environment) and MFM-Verkehr (traffic).
MI	Myocardial infarction

µPa	Micro-Pascal
NO	Nitrous oxide
NO <sub>2</sub>	Nitrogen dioxide
NO <sub>x</sub>	Oxides of nitrogen
NOAEL	no observed adverse effect level
O <sub>3</sub>	Ozone
OR	The ratio of two odds, an odds being a ratio of probabilities (in this instance, the ratio of the probability of occurrence of an event to the probability of non-occurrence). For example, if the ordinary risk of getting a disease is 2 in a 1000 but for smokers it's 5 per 1000, then a smoker's odds ratio is 2.5. They are two and half times more likely to get the disease than a non-smoker.
PM	Particulate matter
PM <sub>x</sub>	Particles that have an aerodynamic diameter of less than x µm
ppb	Parts per billion
ppm	Parts per million
Proxy	See surrogate
Reference level	Reference level or reference concentration refers to the chosen scenario of lowest air pollution concentration. Only health effects above this reference level are calculated in a Health Impact Assessment.
SO <sub>2</sub>	Sulfur dioxide
surrogate	air pollutant that is thought to represent a complex mixture of air pollutants
TSP	Total suspended particulate

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