Years of life lost and morbidity cases attributable to transportation noise and air pollution: a comparative health risk assessment for Switzerland in 2010

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ABSTRACT

Background: There is growing evidence that chronic exposure to transportation related noise and air pollution affects human health. However, health burden to a country of these two pollutants have been rarely compared.

Aims: As an input for external cost quantification, we estimated the cardiorespiratory health burden from transportation related noise and air pollution in Switzerland, incorporating the most recent findings related to the health effects of noise.

Methods: Spatially resolved noise and air pollution models for the year 2010 were derived for road, rail and aircraft sources. Average day-evening-night sound level (Lden) and particulate matter (PM_{10}) were selected as indicators, and population-weighted exposures derived by transportation source. Cause-specific exposure-response functions were derived from a meta-analysis for noise and literature review for PM_{10}. Years of life lost (YLL) were calculated using life table methods; population attributable fraction was used for deriving attributable cases for hospitalisations, respiratory illnesses, visits to general practitioners and restricted activity days.

Results: The mean population weighted exposure above a threshold of 48 dB(A) was 8.74 dB(A), 1.89 dB(A) and 0.37 dB(A) for road, rail and aircraft noise. Corresponding mean exposure contributions were 4.4, 0.54, 0.12 $\mu g/m^3$ for PM_{10}. We estimated that in 2010 in Switzerland transportation caused 6,000 and 14,000 YLL from noise and air pollution exposure, respectively. While there were a total of 8,700 cardiorespiratory hospital days attributed to air pollution exposure, estimated burden due to noise alone amounted to 22,500 hospital days.

Conclusions: YLL due to transportation related pollution in Switzerland is dominated by air pollution from road traffic, whereas consequences for morbidity and indicators of quality of life are dominated by noise. In terms of total external costs the burden of noise equals that of air pollution.

KEYWORDS: Transportation; noise; air pollution; burden of disease; health impact assessment; external costs

HIGHLIGHTS:

- Link between transportation noise and cardiovascular outcomes, independent of air pollution.
- The impact of transport noise was only partially accounted in past burden studies.
- Mortality is dominated by air pollution from road traffic.
- Noise has a larger impact on quality of life indicators.
- In Switzerland, transportation related air pollution and noise amount to similar external costs.

INTRODUCTION

There is a large body of evidence on the health effects of air pollution, specifically fine particle matter (PM) generated by traffic sources in urban areas. There is robust evidence for a link of PM fractions with long-term mortality (Hoek et al., 2013) and infant mortality (Woodruff et al., 1997), and various morbidity outcomes such as cardiorespiratory hospital admissions (Atkinson et al., 2014), bronchitis (Abbey et al., 1995; Schindler et al., 2009), asthma (Hoek et al., 2012; Weinmayr et al., 2010) and restricted activity days (Ostro, 1987). This evidence has been used for estimating the burden of air pollution in different settings (Lim et al., 2012; WHO, 2013a).

Less is known about the health effects of transportation related noise, although there has been substantial growth in the body of evidence in the last years. While the negative health impact from noise were principally linked to annoyance, auditory and other non-auditory health effects (Basner et al., 2013), new studies are finding an association between chronic exposure to transportation related noise and cardiovascular outcomes, such as ischemic heart disease (IHD), hypertensive diseases and stroke, independent of the effects of air pollution (Sørensen et al., 2011; van Kempen and Babisch, 2012; WHO, 2011).

In Switzerland, the political consensus is that heavy vehicles (above 3.5 tonnes) must cover the entirety of the costs they generate, including the external costs from damage to environment and health. Thus the LSVA (performance related heavy vehicle charge) has been traditionally derived in part on calculation of external costs of noise and air pollution, revised every 5 years (ARE, 2004a, b, 2008, 2014a). So far, external cost of noise were principally driven by the effects of quality of life indicators (annoyance and sleep disturbance) and were reflected by calculating the loss of rents in noise exposed apartments (ARE, 2008). Health effects represented by mortality due to hypertension and ischemic heart disease have also been included in past evaluations but cost contributions were minor compared to loss of rents (ARE, 2008, 2014a). The recent epidemiological literature shows that the mortality effects of noise are much higher than earlier studies suggested. The impact of noise from transportation was thus most likely only partially accounted in past burden and cost evaluations studies in Switzerland and elsewhere.

As an input for the latest external traffic cost estimates in Switzerland, this study estimates the years of life lost (YLL) and attributable burden for different cardiorespiratory outcomes due to the noise and air pollution generated from road, rail and aircraft transport in 2010 in Switzerland, incorporating the most recent findings related to the health effects of noise and air pollution.

MATERIALS AND METHODS

We combined population exposure to noise and air pollution with exposure-response functions and baseline cardiorespiratory morbidity and mortality data to estimate the years of life lost (YLL) and the number of morbidity cases attributable to noise and air pollution from transportation on the roads, railways and in the air.

Population exposure

Exposures to noise were obtained from existing models for year 2010. For road and rail noise population exposures were derived from SonBase, the Swiss GIS-based noise model (Karipidis et al., 2014). SonBase models the noise propagation from source to reception points, taking account of building height, first order reflections and noise barriers. Noise levels at source points are first calculated with CADNA-A and STL-86+ models using data from a detailed Swiss national traffic model for 2010 from the Federal Office for Spatial Development (ARE, 2014b). SonBase calculates equivalent continuous noise level (Leq) at the most exposed façade of each building per floor in Switzerland, with noise in steps of 1 dB(A). Estimates of aircraft noise for the national airports of Zurich and Geneva come directly from the airport operators, which annually evaluate the airport-specific noise. The data for Basel and 10 regional airports were derived from the SonBase model developed by the Federal Office of Civil Aviation (ARE, 2004a; Huss et al., 2010).

The noise metric used in our study was Lden [dB(A)], the average sound level over all 24 hour periods of a year with a respective 5 and 10 dB(A) penalty for evening (18:00 to 22:00) and night (22:00-06:00) hours. Noise levels modelled at residential addresses were combined with population counts to determine total exposure in 1 dB(A) steps from 40 – ≥80 dB(A) (in burden calculations, population in areas with modelled road and rail noise <40 dB(A) were assigned a level of 40 dB(A)). For subsequent burden calculations, a threshold of no effect of 48 dB(A) was assumed (see next Section "Derivation of exposure-response relationship"). We thus calculated the population-weighted mean exposure over this threshold for each noise source.

For air pollution, PM_{10} was used as the pollutant indicator to allow for comparability with past studies. Exposure levels for 2010 were obtained from a 200 x 200m dispersion model for PM_{10} which accounted for primary particulates, secondary particle formation from precursor emissions (NO_x, SO_2 NH_3 and NMVOC) and transboundary large-scale PM_{10} (BAFU, 2013). The dispersion model was run for total air pollution and separately for each transport source (road, rail and air). Population counts in each grid cell were combined with PM_{10} levels to obtain population-weighted concentrations by source type.

Derivation of exposure-response relationship

We conducted a literature review to derive or obtain exposure-response relationships reflecting the most current scientific evidence in the association between noise, particulate matter and cardiorespiratory mortality or morbidity.

We had previously developed meta-analytic estimates of the effects of noise on several cardiovascular outcomes (ARE, 2014a; Vienneau et al., 2015). This included a meta-analysis to derive an exposure-response function for ischemic heart disease (IHD) and stroke, and the pooling of two existing meta-analysis estimates to derive a summary estimate for hypertension (Table 1). The methods in brief were as follows. For IHD, we combined the results of 10 studies conducted since the mid-1990s, providing 13 relative risk estimates for morbidity or mortality. Most were conducted in Europe for road noise; 4 investigated exposure to aircraft noise, two of which were in North America; none were found for railway noise. Six studies were combined for stroke, contributing a total of 8 relative risk estimates for meta-analysis: 3 road, 4 aircraft and 1 rail noise. For hypertension, we combined the two recent meta-analyses, van Kempen and Babisch (2012) on road and Babisch and van Kamp (2009) for aircraft, to derive the exposure-response function. To specify the starting point for the noise exposure-response associations, we globally pooled the study specific reference values (i.e. for three outcomes) using the derived meta-analysis weights of each study. This resulted in a threshold of 48 dB(A) below which no effects were considered. We did not include annoyance, sleep disturbance and cognitive impairment as outcomes to allow for comparability with past cost evaluations in Switzerland, and to avoid potential double counting of effects.

For air pollution related health effects we applied the recommendations of the HRAPIE (Health risks of air pollution in Europe) project (WHO, 2013a, b) (Table 2). For some outcomes such as mortality, HRAPIE proposes an exposure-response function for $PM_{2.5}$. In this case the exposure-response function was converted to PM_{10} by applying the ratio of the population-weighted means for $PM_{2.5}$/ PM_{10} of 0.73 (calculated in the Swiss dispersion model).

Table 1. Exposure-response relationships and baseline data used for the estimation of mortality and morbidity due to noise (per 10 dB(A) increase in Lden)

Outcome	Approach	Relative Risk (95% confidence interval) per 10 dB(A) increase in Lden	Baseline health data
Ischemic heart disease	≥30 years mortality; all ages morbidity. Meta-analysis including 13 estimates from 10 studies on effects of road and aircraft transportation noise and IHD (Babisch et al., 2005; Babisch et al., 1999; Babisch et al., 1994; Beelen et al., 2009; Correia et al., 2013; Gan et al., 2012; Hansell et al., 2013; Huss et al., 2010; Selander et al., 2009; Sørensen et al., 2012)	1.046 (1.015, 1.079)[a]	ICD10 I20-I25. 2011 mortality rates; 283,443 hospital days (BfS)
Stroke	≥30 years mortality; all ages morbidity. Meta-analysis of 8 estimates from 6 studies on road, aircraft and rail transportation noise and stroke (Beelen et al., 2009; Correia et al., 2013; Gan et al., 2012; Hansell et al., 2013; Huss et al., 2010; Sørensen et al., 2011)	1.014 (0.964, 1.066)[b]	ICD10: I60-I64 exc. I63.6. 2011 mortality rates; 300,472 hospital days (BfS)
Hypertensive diseases	≥30 years mortality; all ages morbidity. Pooling of the effect estimate from 2 existing meta-analysis (Babisch and van Kamp, 2009; van Kempen and Babisch, 2012)	1.076 (1.032, 1.121)[b]	ICD10: I10-I15. 2011 mortality rates; 51,871 hospital days (BfS); 990,440 general practitioner visits extrapolated from Swiss Health Survey (BfS, 2010)

a. Exposure-response functions were developed in a previous version of Vienneau et al. (2015).
b. Exposure-response functions were developed in ARE (2014b).
BfS: Bureau of Federal Statistics, Switzerland

Table 2. Exposure-response relationships and baseline data used for the estimation of mortality and morbidity due to air pollution (per 10 µg/m^3 increase in PM_{10})

Outcome	Relative Risk (95% confidence interval) per 10 µg/m^3 increase in PM_{10}	Source[a]	Baseline health data[a]
All-cause (natural) mortality	1.045 (1.029, 1.060)	Hoek et al. (2013)	2011 mortality rates, ICD10 A00-R99 (BfS)
Post-neonatal infant mortality, all cause	1.04 (1.02, 1.07)	Woodruff et al. (1997)	2011 mortality rates (BfS)
Hospital days for cardiovascular diseases (includes stroke), all ages	1.007 (1.001, 1.012)	Atkinson et al. (2014)	1,393,409 hospital days, all ages, ICD-10 I00-I99, (BfS)
Hospital days for respiratory diseases, all ages	1.014 (0.999, 1.029)	Atkinson et al. (2014)	579,939 hospital days, ICD-10 J00-J99, (BfS)
Incidence of chronic bronchitis in adults (≥18 years)	1.117 (1.040, 1.189)	Abbey et al. (1995) Schindler et al. (2009)	24,869 cases. Annual incidence is 3.9 per 1000 adults to be applied to ages above 18, SAPALDIA study
Prevalence of bronchitis in children (6-18 years)	1.08 (0.98, 1.19)	Hoek et al. (2012)	198,109 cases. Prevalence average PATY study, 18.6% to be applied to ages 6-18
Asthma attacks in adults with asthma (≥18 years)	1.029 (1.013, 1.045)	ARE (2004a)	1,339,058 attacks. Estimated as 0.21 asthma attacks per adult per year (this includes average 3-4 attacks per year per asthmatic)
Days with asthma symptoms in asthmatic children (5-17 years)	1.028 (1.006, 1.051)	Weinmayr et al. (2010)	3,333,635 symptom-days. Estimated in children based on "Symptoms of severe asthma" over population 5-19 and average "severe asthma" for Western Europe (4.9%) in ISAAC study (Lai et al., 2009). The daily incidence of symptoms among this group is assumed 17%, multiplied by 365 gives the number of days of symptoms among asthmatics for one year
Restricted activity days (≥18years)	1.034 (1.030, 1.038)	Ostro (1987)	121,152,911 days with restricted activity. As in original paper, 19 restricted activity per person per year (for population over 18 years of age)

a. Following the recent World Health Organization guidelines (WHO, 2013a, b).
BfS: Bureau of Federal Statistics, Switzerland

Calculation of morbidity and mortality burden

We used mortality rates observed in Switzerland to calculate changes in YLL for a reference and the counterfactual scenario using the life table approach (Miller and Hurley, 2003; Röösli et al., 2005). In the reference scenario, 1-year age interval life tables for the Swiss population were calculated extrapolating observed survival probabilities in the year 2011, obtained from Federal Statistics Office (BfS), to 2010 population (differentiated for male and female). For the counterfactual scenario, life tables were rerun with modified survival probabilities that assumed no one in the population was exposed to source-related transportation noise (above 48 dB(A)) or PM_{10} concentrations. Thus cause-specific mortality rates were changed according to the relevant relative risk (RR) and source-specific exposure contribution, keeping unchanged rates for the remaining outcomes not affected by the exposure. The counterfactual scenario assumed a return to previous exposure levels after 2010, thus mortality rates are only modified in 2010. For both scenarios, life years were calculated for the next 105 years and summed. The difference between the reference and counterfactual scenario is interpreted as the YLLs attributed to noise or air pollution in year 2010 in Switzerland. No discounting for time or age was applied.

For morbidity outcomes, we used population attributable fraction (PAF) applied to baseline heath data to obtain the number of cases per year attributable to noise or air pollution transportation in Switzerland in 2010. Baseline health data were obtained from Federal Statistics Office (BfS) or, following recommendations by WHO, extrapolated from past studies if not available for Switzerland (Tables 1 and 2).

We evaluate uncertainty by calculating health impacts based on the 95% confidence intervals of the relevant exposure-response function.

RESULTS

Population exposure

Approximately 6.6 of the 7.8 million residents (84%) in Switzerland were found to be exposed to road noise in excess of our 48 dB(A) (Lden) threshold. The majority of these persons (61%) lived in areas with noise levels between 48 and 60 dB(A). On this basis, we computed the mean population-weighted excess (>48 dB(A)) exposure as 8.74 dB(A) for road noise (Table 3). Substantially fewer persons were exposed to rail (1.5 mil) and aircraft (0.58 mil) noise, respectively reflecting a mean excess exposure of 1.89 and 0.37 dB(A) (Lden) (Figure S1, online supplement).

The population-weighted exposure to total PM_{10} in 2010 was 19.4 µg/m^3 (see Table S1). The transportation sources accounted for 26% of the total PM_{10} load. The remaining load is largely caused by household, industry, agriculture and forestry sources (71%) and a small amount from natural origin (3%). The contribution of transportation related sources applied to the burden calculation were 4.4, 0.54, 0.12 μg/m^3 for road, rail, and aircraft transport, respectively (Table 3).

Table 3. Population weighted excess concentrations for noise and air pollution to transportation sources in Switzerland, 2010

Pollutant	Road traffic	Rail traffic	Aircraft traffic	Total transport
Noise (Lden, dB(A))[a]	8.74	1.89	0.37	11.00
Air pollution (PM_{10}, μg/m^3)	4.40	0.54	0.12	5.06

a. Calculated as population weighted mean for levels above 48 dB(A). This threshold level was determined by pooling the study specific reference values using the derived meta-analysis weights of each study.

Exposure-response relationship

Relative Risk (RR) estimates derived from a meta-analysis (ARE, 2014a; Vienneau et al., 2015) used in the evaluation of the noise burden in Switzerland (per 10 dB(A) increase, Lden) are presented in Table 1. The relative risk for increased morbidity and mortality per 10 dB(A) increase in Lden is 1.046 (95% CI 1.015, 1.079) for IHD, 1.014 (0.964, 1.066) for stroke and 1.076 (1.032, 1.121) for hypertensive diseases. Given the small number of available studies for each outcome, we assumed that the same risk estimate would apply to both mortality and morbidity. The available risk estimates for hypertension, in particular, related only to morbidity. A stratified analysis in the meta-analysis for IHD indicated that the difference between risk estimates by disease state were not statistically significant (Vienneau et al., 2015). The baseline health data related to each outcome are presented in Table 1.

We followed the recent review of the literature by the WHO to select the exposure-response functions for long-term exposure to PM_{10} and health outcomes (WHO, 2013b). The relative risk (RR) for all-cause (natural) mortality in adult populations (age 30+) is 1.062 (1.040, 1.083) per 10 μg/m^3 increase in the long term $PM_{2.5}$ exposure based on a meta-analysis of 13 cohort studies (Hoek et al., 2013). The corresponding risk estimate for PM_{10} using the population-weighted ratio of 0.73 results in an RR of 1.045 (1.029, 1.060). For the first year of life we applied an increase in mortality by 1.04 (1.02-1.07) according to Woodruff et al (1997). The full set of relative risk estimates and baseline health data in relation to PM_{10} is presented in Table 2.

Estimated burden

The results of the YLL in Switzerland due to transport noise and air pollution exposure are shown in Table 4. We estimated that exposure to transportation related noise caused 6,000 YLL in 2010, most of which were associated with death by IHD (4,100), followed by death from hypertensive diseases (1,400). We estimated that exposure to transportation related air pollution caused 14,000 YLL in 2010. For both, noise and air pollution, the largest contributor to the YLL originates from road traffic (78% and 86%, respectively).

The burden related to transportation on the road, railway and by aircraft in Switzerland in 2010 amounted to 20,000 YLL with 70% of total contributed by air pollution and 30% by noise. By source, the largest contribution to YLL from road traffic remains air pollution (72%). The burden from rail and aircraft traffic is more equally distributed between these sources (60% and 62%, respectively, from air pollution).

Table 5 shows the estimated impact of noise and air pollution on morbidity in 2010. We obtained 13,800, 4,600 and 4,100 hospital days for IHD, stroke and hypertensive diseases due to transportation noise in Switzerland in 2010. The number of general practitioner visits due to hypertensive diseases was estimated as 77,700.

We estimated a total of 4,700 and 4,000 cardiovascular and respiratory hospital days, respectively, due to exposure to air pollution from transportation in Switzerland in 2010. In addition, for adults (children) we estimated there were 1,400 (7,600) bronchitis cases and 19,300 (45,900) asthma related symptoms per year, as well as 2,047,000 restricted activity days due to this exposure.

Table 4. *Estimated* Years of life lost (undiscounted) due to noise and air pollution by transportation source with 95% confidence interval for Switzerland, year 2010

Outcome		Total transport	Road traffic	Rail traffic	Aircraft traffic
Noise (Lden): Cardiovascular disease	IHD	4100 (1400, 6800)	3300 (1100, 5400)	710 (240, 1200)	140 (50, 230)
	Stroke	470 (0, 2100)[a]	370 (0, 1700)[a]	80 (0, 360)[a]	20 (0, 70)[a]
	Hypertensive diseases	1400 (610, 2100)	1100 (480, 1700)	240 (100, 360)	50 (20, 70)
	Total noise	6000 (2000, 11100)	4700 (1600, 8800)	1000 (340, 1900)	200 (70, 370)
Air pollution (PM_{10}): All-cause (natural) mortality	age≥30	13000 (8600, 17000)	11000 (7500, 15000)	1400 (920, 1800)	310 (200, 400)
	age 0-1	460 (240, 790)	400 (210, 690)	50 (30, 80)	10 (6, 20)
	Total air pollution	14000 (8800, 18000)	12000 (7700, 16000)	1400 (940, 1900)	320 (210, 420)
Total YLL (noise + air pollution)[b]		20000	17000	2500	520
%contribution to total[b]	Noise	30%	28%	40%	38%
	Air pollution	70%	72%	60%	62%

a. If confidence intervals of the exposure-response function included 1.0, the burden estimates were censored at zero to prevent calculating beneficial effects.
b. Based on central estimate.

YLL are rounded thus totals do not necessarily sum: 9-999 to the nearest 10; 1000-99999 to nearest 100; >100000 to nearest 1000.

IHD: Ischemic Heart Disease; YLL Years of Life Lost

Table 5. *Estimated* Morbidity due to noise and air pollution by transportation source with 95% confidence interval on the exposure-response function

Outcome	Total transport	Road traffic	Rail traffic	Aircraft traffic
Noise (Lden)				
Hospital days for IHD (≥30 years)	13800 (4600, 23100)	10900 (3700, 18200)	2400 (800, 4000)	470 (150, 790)
Hospital days for stroke (≥30 years)	4600 (0, 20600)[a]	3600 (0, 16300)[a]	790 (0, 3600)[a]	150 (0, 700)[a]
Hospital days for hypertensive diseases (≥30 years)	4100 (1800, 6300)	3200 (1400, 4900)	710 (310, 1100)	140 (60, 220)
General practitioner visits for hypertensive diseases (>15 years)	77700 (33900, 119000)	61400 (26900, 94100)	13600 (5900, 21100)	2700 (1100, 4100)
Air pollution (PM_{10})				
Hospital days for cardiovascular diseases (includes stroke), all ages	4700 (870, 8500)	4000 (760, 7400)	500 (90, 900)	110 (20, 200)
Hospital days for respiratory diseases, all ages	4000 (0, 8400)[a]	3500 (0, 7300)[a]	430 (0, 900)[a]	100 (0, 200)[a]
Incidence of chronic bronchitis in adults (≥18 years)	1400 (490, 2100)	1200 (430, 1900)	150 (50, 230)	30 (10, 50)
Prevalence of bronchitis in children (5-17 years)	7600 (0, 17000)[a]	6600 (0, 14700)[a]	810 (0, 1800)[a]	180 (0, 410)[a]
Asthma attacks in adults with asthma (≥18 years)	19300 (8800, 29700)	16800 (7600, 25800)	2100 (940, 3200)	460 (210, 700)
Days with asthma symptoms in asthmatic children (5-17 years)	45900 (10000, 82600)	40000 (8700, 71800)	4900 (1100, 8800)	1100 (240, 2000)
Restricted activity days (≥18years)	2047000 (1834000, 2301000)	1779000 (1595000, 2000000)	219000 (196000, 246000)	48500 (43400, 54500)

a. If confidence intervals of the exposure-response function included 1.0, the burden estimates were censored at zero to prevent calculating beneficial effects.

Numbers are rounded thus totals do not necessarily sum: 9-999 to the nearest 10; 1000-99999 to nearest 100; >100000 to nearest 1000.

IHD: Ischemic Heart Disease

DISCUSSION

This study comparatively estimated the attributable burden due to road, rail and aircraft traffic noise and air pollution in 2010 in Switzerland, incorporating the most recent findings related to effects of air pollution and noise on health. Stratified by source, we found that road traffic remains the largest single contributor to cardiorespiratory mortality.

Our noise estimates do not include the well-established effects on sleep disturbances and annoyance (Frei et al., 2014; Héritier et al., 2014). These effects are usually estimated and expressed by means of disability adjusted life years (DALYs), as done in burden of disease from environmental noise (WHO, 2011). Given our objective to determine external costs for Switzerland, however, we opted against first calculating DALYs then translating these into monetary costs. The additional step likely would have introduced greater uncertainty in the external cost. Direct quantification of reduced housing and renting prices likely better reflects what citizens are willing to pay for the absence of noise induced annoyance and sleep disturbances, and furthermore is more widely accepted by policy makers. We calculated annual external costs of 1,050 Mil Swiss Francs (CHF) due to reduced housing and renting prices in Switzerland for the year 2010 (for full data see ARE 2014a). The impact of transportation related noise exposure on cardiovascular diseases was 560 Mil CHF due to YLL and 190 Mil CHF due to noise induced morbidity (ARE, 2014a). This yields total external health costs from noise exposure of 1,800 Mil CHF (1,250 Mil CHF due to YLL and 510 Mil CHF due to morbidity), which is similar to the total air pollution related external costs of 1,760 Mil CHF. In 2005, for road and rail transport the external cost of noise was estimated at only 60% of the health costs due to air pollution.

Our estimate includes the noise impact on cardiovascular diseases in Switzerland. Impacts related to hypertension contribute second to YLL after IHD (largely myocardial infarction). While the pathophysiological pathways by which noise is related to hypertension still need to be understood, our finding is relevant for public health because the overall prevalence of hypertension – a primary cause of cardiovascular mortality in Switzerland – remains high and targeted policy actions for prevention are needed (Danon-Hersch et al., 2009; Dratva et al., 2012).

While recent noise studies have accounted for the effect of air pollution, the older studies included in our meta-analysis have not. Confounding, however, may be minimal. In a recent, review Tétreault et al. (2013) reported minimal confounding of the association between noise exposure and cardiovascular disease by air pollution. In a population-based study on transportation noise and blood pressure in adults in Switzerland, the effect estimate for road or railway noise also did not change after adjusting for home outdoor air pollution levels (Dratva et al., 2012). Similarly the air pollution studies we used for exposure-response functions did not adjust for confounding by noise. Recent studies from the European Study of Cohorts for Air Pollution Effects (ESCAPE) project on the effects of air pollution on several cardiovascular and respiratory outcomes found no major change in effects when adjusting for noise (Stafoggia et al., 2014) . Moreover, as pointed out by Foraster (2013), true personal exposure to traffic related noise may be substantially lower and misclassified in particular among subjects living close to noisy streets as they may adopt coping strategies. Given that night-time noise in bedrooms was not modelled in any of the studies used in our noise meta-analyses, associations between noise and health outcomes are likely to be underestimated, thus, resulting in conservative estimates of the burden as well.

Aircraft traffic is a rather moderate contributor to mortality at a Swiss-wide level as it represents less than 3% of total noise and air pollution exposure due to transportation. The contribution of aircraft noise to the total source-specific burden, however, is rather large at 38%. In more general terms this result raises the issue of exposure to concomitant sources. Our analysis is based on an average for the full population. It does not account for aspects of susceptibility in the population that may have important consequences in burden and cost estimates if the susceptibility profile of the Swiss population differed from the populations where the exposure-response functions had been derived. It has now been shown that noise and air pollution could lead to acceleration of the progression of some often clustered metabolic disorders and chronic respiratory diseases in individuals (Adam et al., 2015; Eze et al., 2014; Jerrett et al., 2014). As a consequence, there may be a disproportionate earlier age of onset in diseases and premature deaths in certain population groups. There is a need to collect data to help establish susceptibility risk profiles in Switzerland.

We did not consider potential interactions between the various exposure sources. Some areas and individuals may be exposed to several sources at the same time. Pershagen et al. (2014), for example, saw a clear upward trend in the odds ratios for the relationship between noise and abdominal obesity with increasing number of transportation sources from one to all three (e.g. road, rail and aircraft). Given, however, that the synergetic or sub-additive effects between pollutants or within different levels of pollutants are still unknown, we abstained from additional disaggregation to evaluate the distribution and impact of concomitant exposures.

The above point also directly relates to the main limitation of our study - the attribution of health effects to a specific traffic source. Given that only a few studies on the effects of noise on cardiovascular disease for specific transportation modes exist, we abstained from applying different exposure-response functions in our evaluation. We further assumed a log linear relationship with noise exposure for all health outcomes. The same exposure-response function was applied for all traffic noise sources despite known differences in the acoustic characteristics for the different noise sources. As indicated by existing exposure-response functions for noise annoyance and hypertension, the type of noise source (road, rail or aircraft) may be strongly related to health outcome or characteristics of individuals (Basner et al., 2013). Using a single exposure-response for all noise sources may thus be problematic. We explored the implications of our decision to pool noise sources in our meta-analysis for IHD and noise exposure through stratified analyses. We did not see indications for heterogeneity between studies on road versus aircraft noise, although the threshold for the association may be higher for aircraft compared to road traffic noise (Vienneau et al., 2015).

Similarly, we used the same exposure-response function for PM_{10} for all modes of transportation. A large contribution of air pollution exposure is from road traffic. Thus, exposure-response functions deriving from cohort studies based on exposure to $PM_{2.5}$ (e.g. land use regression models representing both regional and local pollution) should reflect the effects of road traffic exposure to some extent. We cannot, however, rule out that we may be over or underestimating burden from railway and aircraft with these exposure-response functions. In particular, little research has been done on differences in toxicity according to the source of particles. Particles from road traffic, for example, may have a different effect on long term morbidity and mortality than particles from railway traffic which is primarily mechanically generated from wear on the rail. PM_{10} is also a limited indicator for air pollution from aircraft which mainly emit NOx and ultrafine particles. Nevertheless, assessment of total transportation related air pollution effects is expected to be less critical since a large contribution of total air pollution exposure is from road traffic. Estimated health effects of $PM_{2.5}$ and PM_{10} from cohort studies thus largely reflect the effect of road traffic exposure.

Comparative health impact assessment like ours relies on several assumptions that can rarely be validated. In our study we followed a conservative approach. We tried to make the most accurate choice but in case of doubt we applied the assumption that would likely yield a conservative estimate, which may also be generalizable beyond Switzerland. For instance, we only evaluated noise effects at people's homes; we do not consider potential effects from noise in work places, in recreation areas and below the threshold of 48 dB(A). We also only selected outcomes that were economically quantifiable and minimized double counts of the same cases. Burden and associated costs, however, would be accrued if air pollution effects including low birth weight, respiratory symptoms and days with cough, drug prescriptions for respiratory and cardiac/circulatory diseases, self-medication, avoidance behaviour as well as acute and chronic physiological changes (e.g., lung function) and metabolic changes including diabetes (Eze et al., 2014) had been considered. Evidence for additional non-auditory health effects from transportation noise which we could not quantify, for example, include cognitive impairment in children (Clark and Stansfeld, 2007; Stansfeld et al., 2005) and metabolic outcomes in adults (Eriksson et al., 2014; Sørensen et al., 2013). Air pollution effects on restricted activity is an important driver of the morbidity (Table 5) and the subsequent cost, but our morbidity estimate relies on a single very old exposure-response function from the United States which adds some uncertainty to the cost estimates (Ostro, 1987). Finally, our counterfactual scenarios intrinsically assume immediate changes after one year of intervention. In reality, more time would be needed to see the health benefits (and costs) of transportation interventions, thus it could be argued that the external costs incurred per year may be inflated. Past sensitivity analyses, however, demonstrated that the effect is minor (Röösli et al., 2005). In terms of quantifying cost we used a discount rate of 1% in all calculations.

Our estimates of the cardiorespiratory health burden from transportation related noise and air pollution in Switzerland are influenced by a number of factors including disease incidence, the risk profile of the population, selection of the exposure metrics (e.g. Lden, PM_{10}) and the simulation of population exposure. Only studies from Europe and North America were available for the derivation of the exposure-response functions and selected threshold levels. In order to generalize these associations, more studies in different cultural contexts are needed, specifically with regards to noise, on the health effects of exposure to individual and combined transportation sources. This will serve to reduce uncertainty in the exposure-response functions and make more them generalizable to populations beyond Europe. Strikingly, the global burden of disease study lists ambient air pollution as one of the leading causes for DALYs on a global scale, but does not even mention community noise (Lim et al., 2012). We have confirmed that in Switzerland noise exposure is an important a risk factor in its own right. This demonstrates that noise exposure should not be ignored at the global scale.

CONCLUSIONS

Transportation related air pollution and noise in Switzerland is widespread and contributes largely to the health burden from these exposures. While exposure assessments are becoming more precise with availability of source-specific exposure models, uncertainties about the exposure-response functions for different transport sources remain, especially regarding noise. In Switzerland transportation related noise and air pollution cause similar external costs in the range of 1,700-1,800 million CHF each year. For air pollution the effects on mortality is most relevant in terms of costs whereas for noise the effects representing impaired quality of life from annoyance and sleep disturbances is the strongest cost contributor.

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Online Supplement

Years of life lost and morbidity cases attributable to transportation noise and air pollution: a comparative health risk assessment for Switzerland in 2010

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Table S1. Population weighted PM_{10} exposures in 2010 by sector activity

Sector activity	All residents $\mu g/m^3$	Ages 0-14 $\mu g/m^3$	Ages ≥30 $\mu g/m^3$
Road traffic			
Car	2.985	2.951	2.991
Light goods vehicle	0.378	0.375	0.379
Heavy goods vehicle	0.774	0.767	0.775
Private bus	0.066	0.066	0.066
Public transport Bus	0.176	0.172	0.176
Motorcycles	0.026	0.026	0.026
Rail traffic			
Passenger transport	0.339	0.330	0.340
Good transport	0.202	0.199	0.203
Ship traffic			
Passenger transport	0.050	0.050	0.050
Good transport	0.037	0.036	0.037
Air traffic	0.120	0.120	0.119
Residential	3.353	3.334	3.357
Industrial	6.294	6.287	6.297
Agricultural	4.144	4.163	4.140
Natural	0.500	0.500	0.500
Sum	**19.442**	**19.375**	**19.456**

Figure S1. Number of noise-exposed persons to road, rail and air transport in 5 dB(A) categories (Lden)