Estimated health impacts of changes in air pollution exposure associated with the planned by-pass Förbifart Stockholm

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SAMMANFATTNING

För att minska problem med trängsel på vägnätet i Stockholm och möta framtida transportbehov planeras en 21 km lång förbifart i nord-sydlig sträckning, varav cirka 18 km skulle gå i tunnel. Denna nya led, Förbifart Stockholm, förväntas förbättra luftkvaliteten i områdets central delar som avlastas trafik på ytvägar, samtidigt kommer de som färdas I tunnel att kunna utsättas för höga koncentrationer av trafikföroreningar.

Trafikföroreningarna utgörs dels av motoravgaser med avgaspartiklar (bl a dieselsot), kolväten och kväveoxider (NO_X) som viktiga komponenter. Dessa föroreningar brukar studeras som indikatorer på hur farlig luften är, I synnerhet NO_2 och NO_X. I tunnelmiljö är NO_X (NO + NO_2) en bättre indikator än NO_2 på hur hög avgashalten är i jämförelse med i utomhusluft och hälsostudier, eftersom NO inte oxideras till NO_2 i samma utsträckning som i utomhusluft.

Förutom avgaser består trafikföroreningarna av slitagepartiklar från vägbanor och fordon (främst däck och bromsar). Dessa partiklar ingår i det man brukar kalla vägdamm, vilket består av större partiklar än avgaserna. För vägdamm kan masskoncentrationen (vikt/volym luft) av PM_{10} (partiklar mindre än 10 mikrometer) användas som ett relevant mått på koncentrationen. För de mycket mindre avgaspartiklarna används inte så ofta masskoncentrationen i luft. Hur höga halter det skulle bli i tunnelmiljön beror förutom på utsläppens omfattning på ventilationslösningarna, varför beräknade förhållanden blir mer osäkra. För tunnelhalterna är det dessutom svårare att uppskatta antal exponerade personer och avskiljningen som fordonskupen ger. Beräkningarna för hur utomhusluften påverkas är förenade med mindre osäkerhet.

Skillnaderna i befolkningsexponering via ändrade årsmedelhalter i omgivningsluften har beräknats för NO_X och PM_{10} med en spridningsmodell och upplösningen 100x100 m, vilket sedan kopplats till befolkningen vid årsskiftet 2011/12 (1 628 528 invånare) med samma geografiska upplösning.

Exponeringstillskottet från nyttjandet av tunnel har beräknats utifrån förväntat antal fordon, antal personer i fordonen, halten längs olika sträckor samt restiden längs olika sträckor. Som indikator på avgaser används NO_X eftersom det finns kända dos-responssamband för viktiga effekter som inverkan på dödlighet. För vägdamm används PM_{10} som mått på halterna. Innan förändringen i hälsokonsekvenser beräknas för antalet personer som förväntas nyttja tunneln, dras ifrån den lägre exponering som skulle erhållas vid motsvarande resor på ytvägnätet.

Hälsokonsekvenserna av förändrad exponering har beräknats med etablerade metoder och beräkningsprogrammet AirQ utvecklat av WHO. För dessa beräkningar har relevanta exponerings-responsantaganden eftersträvats. För avgasernas effekt på dödlighet har vi hämtat sambandet från en studie genomförd i Oslo, med en relative ökning på 8% per 10 μgm^{-3} i NO_X, och för vägdamm från en studie genomförd i Stockholm där dagligt antal dödsfall ökade 1.68% per 10 μgm^{-3} högre halt av PM_{10}. För beräkningarna av tunnelexponeringens betydelse

för förtida dödsfall har antagandena om de exponerades åldersfördelning stor betydelse, eftersom en relativ riskökning får större konsekvenser ju högre risken är från början. Ur ett biologiskt perspektiv kan man se det som att äldre är mer känsliga för exponeringen exempelvis för att deras lungor och hjärtan inte är lika friska som hos yngre.

Beräkningarna visar att för befolkningen skulle förbifarten minska antalet förtida dödsfall med 23,7 (95% CI 17.7–32.3) per år, huvudsakligen genom lägre exponering för avgaser. Minskad exponering för vägdamm står bara för 0,5 fall per år färre. Andra former av ohälsa beräknas också minska något till följd av lägre halter. Samtidigt skulle exponeringstillskottet som tunnelluften ger jämfört med resa på ytvägnätet bland annat leda till förtida dödsfall. Under rusningstid skulle avgashalterna i tunneln mätt som NO_X nå närmare 2000 μgm^{-3}. Att passera hela tunneln skulle två gånger om dagen, 5 dagar i veckan, under rusningstid, skulle ge ett tillskott till årsmedelexponeringen motsvarande 9.6 μgm^{-3} NO_X. Om man antar ett genomsnitt på drygt 55 000 fordon per dygn vardera riktning och 1.3 person per fordon, fördelade som åldersgruppen 30–74 år, förväntas en årlig ökning av antalet förtida dödsfall om 20,6 (95% CI 14.1–25.6). Skulle det bli fler personer per fordon eller en högre andel känsliga t ex på grund av äldre resenärer, blir resultatet av exponeringen fler förtida dödsfall. De verkliga effekterna skulle alltså kunna bli annorlunda därför att scenarierna vi antagit inte stämmer.

ABSTRACT

To reduce problems with traffic congestion and meet increased needs of transports, a 21 km long by-pass (18 km in a tunnel) is planned. The by-pass is expected to reduce traffic emissions in central Stockholm but at the same time tunnel users could be exposed to high concentrations of air pollutants from traffic.

For the reduction in urban air pollution concentrations, the change in annual ambient NO_X and PM_{10} levels were modelled using 100x100 m grids and the population (1 628 528 inhabitants) average exposure was calculated for Greater Stockholm area. The tunnel exposure was estimated based on annual average NO_X and PM_{10} levels, time spent in tunnel and number of persons using the tunnel. Health risks were calculated based on health impact assessment principles using equations and the WHO AirQ software. In these calculations the E-R coefficient for non-external mortality was 8% per 10 μgm^{-3} increase of NO_X (vehicle exhaust indicator) and for daily number of deaths 1.68% per 10 μgm^{-3} increase of non-exhaust (road dust) PM_{10}.
It appeared that for the general population there would be annually 23.7 (95% CI 17.7–32.3) premature deaths less; mainly from lower exposure to vehicle exhaust (indicated by NO_X) and somewhat from a reduction in coarse particles (indicated by PM_{10}), contributing 23.2 and 0.5 fewer deaths, respectively. Other adverse health effects of exposure are also expected to be reduced. At the same time, tunnel users will be exposed to vehicle exhaust components in terms of NO_X up to near 2000 μgm^{-3} during rush-hours. Passing the whole tunnel twice on working days would correspond to an additional annual NO_X exposure of 9.6 μgm^{-3}. Assuming there would be on average approximately 55 000 vehicles per day each way and 1.3 persons in each vehicle from the range 30–74 years of age, this exposure would result in 20.6 (95% CI 14.1–25.6) more premature deaths. If there would be more persons per vehicle or older and more vulnerable people travelling, the adverse effect of exposure in traffic could become larger. Hence, the effects in reality may be different as these results are based on now presented scenarios.

CONTENTS

1. INTRODUCTION

The planned Stockholm bypass – Förbifart Stockholm – will be a new motorway linking southern and northern Stockholm, which is divided by water. This bypass should meet the growing transport needs due to the increased population in the region. By 2030, the population of the Stockholm region is expected to have increased from 2 million today to roughly 2.4 million. More than 18 km of the total of 21 km of the bypass are going to be road tunnels. When the link opens for traffic it will be one of the longest road tunnels in the world. By 2035, the Swedish Transport Administration (Trafikverket) estimates that Förbifart Stockholm will be used by approximately 140,000 vehicles per day. The by-pass is expected to reduce traffic emissions in central Stockholm but at the same time tunnel drivers could be exposed to high concentrations of vehicle exhaust.

Traffic air pollution has been associated both with respiratory and cardiovascular effects (HEI, 2010). The cardiovascular effects of air pollution include myocardial ischemia, atherosclerosis, infarctions, heart failure, arrhythmias, strokes etc. The respiratory outcomes range from acute symptoms like coughing and wheezing to more chronic conditions such as asthma, chronic bronchitis, chronic obstructive pulmonary disease etc. There is also increasing evidence suggesting vehicle emissions to be also associated with the development of cancer, particularly lung cancer, hormonal, and reproductive effects and allergy. Many of these conditions are also associated with the increase in mortality seen in exposed populations.

1.1. Exposures in traffic environments and health effects

Traffic induced air pollutants have a substantial impact on ambient air exposures, indoor air exposures, and personal exposures. The populations who either spend a considerable amount of time in traffic (such as professional drivers and commuters) or who live or work near busy roads are potentially at greatest risk. Often the in-vehicle concentrations are higher than ambient concentrations for most airborne pollutants (Kaur et al., 2007). Also the roadway concentrations are higher compared with ambient concentrations measured at air-monitoring stations; however, highly variable (HEI panel on the Health Effects of Traffic-Related Air Pollution, 2010). Several studies concentrating on professionals, like taxi and truck drivers, have investigated the air pollution induced health effects associated with driving a vehicle. A study in Denmark of 28,744 men with lung cancer found an increased risk among taxi drivers and truck drivers when compared with other employees, probably due to exposure to benzene (Hansen et al., 1998), and increased levels of respiratory conditions have also been associated with professional driving in Shanghai (Zhou et al., 2001). However, the long-term effects of traffic pollutants on the general population are mainly investigated using the area of residence as basis for the exposure estimation.

1.2. Health impacts assessments and their epidemiological base

The general principle for a health impact assessment (HIA) is to use information on how a change in a specific risk factor (for example an air pollutant) is expected to modify the risk of disease or death in the population. Previously found relative changes in health risks are combined with known base-line frequencies in the population in order to estimate the quantitative impact. The most important indicator in air pollution HIAs has been long-term exposure impact on mortality, resulting in loss of life expectancy. Despite the few cohort studies of long-term exposure and mortality, these studies are considered most relevant for HIA, since the time-series studies of short-term effects on mortality do not fully quantify the number of attributable deaths (Krzyzanowski et al., 2005). Other chronic effects have less often been included in HIAs. Even most HIAs of ambient air pollution have dealt with large populations and areas, often bigger than one country (Anenberg et al., 2010; Boldo et al., 2006; Kunzli et al., 2000; Pascal et al., 2013), some studies have also dealt with parts of a city (Orru et al., 2009) or with specific traffic projects (Johansson et al., 2009).

The most often used exposure indicator in HIAs has been particulate matter (PM) mass concentration for the effects of long-term exposure on mortality, based on exposure-response functions from the American Cancer Society (ACS) cohort (Pope et al., 2002). Even expert reports from WHO Regional Office for Europe have concluded that although studies indicate that some components of PM, especially combustion-derived particles, are more toxic than others, it is currently not possible to quantify the contribution to health effects from different components due to limited epidemiological evidence (WHO, 2013). Though an analysis of ACS participants from Los Angeles County, where traffic-induced particles explain a bigger proportion of gradients in the $PM_{2.5}$ concentrations and where exposure-response function (ERF) are nearly threefold higher coefficient for the same indicator (Jerrett et al., 2005). The use of more specific indicators, such as elemental carbon, results in quite different coefficients per mass concentration (Smith et al., 2009). The coarse fraction and mineral particles do not seem to be associated with the survival of cohort members (Brunekreef and Forsberg, 2005). However, recently road dust particles (coarse fraction of PM_{10}) have been associated with short-term effects on daily mortality in Stockholm (Meister et al., 2012).

While waiting for motor traffic specific ERFs for PM to become available, other indicators may be used to indirectly assess the effect of traffic related particles. Road traffic contributes to atmospheric particle pollution in several ways. There are emissions of particles and combustion gases which results in an increased concentration of ultrafine particles (< 100 nm). These particles usually only cause a small increase in the local mass concentration expressed as PM_{10} or $PM_{2.5}$, but a large increase in the particle number concentration (PNC) (Johansson et al., 2007). Due to the common major source (traffic exhaust) there is a good correlation between PNC and NO_X in Stockholm (Johansson et al., 2007). Exhaust gases also form secondary particles such as nitrates and sulphates, but this process occurs on a regional scale (Wexler et al., 1994). A third type of traffic particle is road dust, mainly road wear material but also brake and tire wear. In Stockholm the local contribution to the PM_{10} levels of

road dust is approximately 10 times higher than the mass concentration of exhaust particles (Johansson et al., 2007).

Since motor vehicle emissions of primary exhaust particles have a very local influence, their effect on long-term effects exposure must be studied with a fine spatial resolution. However the ACS results that are frequently used for HIAs do not examine associations at the intra-community level. Epidemiological studies from Europe that use a fine spatial resolution which can capture the gradients in exposure to local traffic pollutants indicate an important effect of local traffic emissions, resulting in high relative risks. Of particular interest is a Norwegian study of 16,000 men from Oslo, of whom 25 % died during the follow up, which used modelled nitrogen oxides (NO_X) in the residential area as the exposure indicator (Nafstad et al., 2004). This cohort, with people of between 40–49 years of age at the start of the study, was followed from 1972/73 through 1998. NO_X was estimated in a model with 1000 m grids, and a street contribution added for the largest streets. When the median concentration of NO_X for 1974–78 was used (10.7 μgm^{-3}), the relative risk for total non-violent mortality was 8 % per 10 μgm^{-3} (95% CI 6–11%).

In a city like Oslo, NO_X is a good indicator of the gradients in levels of motor vehicle exhaust. Due to its long atmospheric lifetime (days) it may be considered as inert and modelled without considering photochemical processes, as in the Norwegian cohort study. Moreover, on a yearly basis there is in general a good spatial correlation between NO_X and NO_2. Other studies from the Netherlands (Hoek et al., 2002), Germany ((Gehring et al., 2006); later follow up by (Heinrich et al., 2012)), France (Filleul et al., 2005), US (Hart et al., 2011), Toronto (Jerrett et al., 2009) and Auckland (Scoggins et al., 2004) have found deaths from non-external causes to increase by 12–14% per 10 μgm^{-3} of NO_2 (however using slightly different exposure metrics), which are in line with the Norwegian result.

A large number of time-series studies have found effects of air pollution on the daily number of hospital admissions for respiratory and cardiovascular disease (Ruckerl et al., 2011), while the effects on induction of such diseases are far less investigated. However, there are increasing support for an effect of traffic pollution on asthma incidence in children and adults (Perez et al., 2013), which is important to consider since it is not reflected in mortality studies.

The short-term associations between NO_2 and hospital admissions and daily mortality remain in many studies after adjustment for PM_{10} or $PM_{2.5}$. The WHO REVIHAAP report (WHO, 2013) concludes ”As there is consistent short-term epidemiological evidence and some mechanistic support for causality, particularly for respiratory outcomes, it is reasonable to infer that NO_2 has some direct effects.” In order to avoid double counting when calculating effects on mortality, we choose to use one pollution indicator only.

The main objective of this study is to estimate the balance between expected health benefits for the general population associated with improved ambient air quality and the expected adverse effects on commuters from high exposure levels while driving in the new road tunnel.

2. METHODS

2.1. Emission factors and scenarios

Road traffic prognoses for two alternative scenarios by the year 2030 have been analysed using emission factors for 2020:

1. No bypass constructed but congestion tax (road traffic fee) on Essingeleden (E4 highway). Presently there is no tax on Essingeleden;
2. The bypass is built and there is congestion tax on Essingeleden (as in 1).

In modelling of ambient concentrations, emissions are described for all important sectors but the difference in emissions in the two scenarios compared here are only due to differences in road traffic emissions. Traffic prognoses for the future scenarios are obtained from a national traffic prognosis model system called SAMPERS (Beser-Hugosson, 2005); a travel demand forecasting tool. In short, it is mainly based on travel enquiries and describes the transports using cars, public transport, cycling and walking depending on the distances, destination, availability of different transportation systems etc. It also includes a model that considers peoples willingness to pay in order to account for taxes e.g. the congestion tax in central Stockholm (Johansson et al., 2009). The scenarios have the same land-use (e g with respect to locations of residential areas).

A detailed local emission database is administered and updated annually by the Stockholm and Uppsala air quality management association. It covers the two counties of Stockholm and Uppsala, including some 30 municipalities with approx. 2 million inhabitants (Johansson et al., 1999). The estimates of total traffic volumes are primarily based on measurements *in situ*. Such measurements are of different kinds: regular automatic traffic counting by the local traffic and street authorities within municipalities, automatic traffic counting on main roads by the Swedish Transport Administration and manual surveys of traffic volumes. Variations of vehicle compositions and temporal variation of the traffic volumes are described for different road types. Future vehicle fleet composition and vehicle exhaust emission factors for 2020 are based on the Swedish application of the Artemis model (Sjödin et al., 2006). Additionally to the vehicle exhaust emissions there are large non-tailpipe emissions of PM due to wear of road surfaces, brakes and tires. In Stockholm the non-tailpipe emissions dominate and emission factors are estimated based on local measurements (Ketzel et al., 2007; Omstedt et al., 2005).

Since most of the transit road will be constructed as an underground highway tunnel, the location of the emissions will be very different compared to current highway on the ground. Most emissions from the tunnel will be ventilated in 10 to 20 meter high towers. Some emissions will occur at tunnel exits at ground surface level. The largest difference between the two alternatives is that there will be much less traffic emissions close to the city centre with the transit road. Special actions are planned to keep the emissions of PM_{10} inside the tunnel as low as possible (in order to minimize exposures of drivers inside the tunnel). Other

emissions than road traffic include combustion of fuels in small and large scale power plants, sea traffic and residential heating. These emissions are assumed to be the same with or without the by-pass Förbifart Stockholm.

2.2. Modelling of air pollution concentrations

The emission and dispersion module of the Aiviro air quality management system (SMHI, Norrköping, Sweden; http://airviro.smhi.se) of the Stockholm Uppsala Air Quality Management Association (www.slb.nu/elvf) has been used for calculating the emissions and exposure concentrations for the Metropolitan area of Stockholm (greater Stockholm).

The annual mean concentrations were calculated using a wind model and a Gaussian air quality dispersion model part of the Airviro system. Meteorological conditions were based on a climatology that was created from 15 years of meteorological measurements (15 minute averages) in a 50 m high mast located in the southern part of Stockholm. The climatology consists of a list of hourly events, each of them with a certain frequency of occurrence, which together will yield a distribution of different weather conditions that is similar to the distribution of the full scenario period. We have used a scenario that consist of 60 wind direction classes with 6 stability classes within each wind sector, making a total of 360 hourly events. The wind field for the whole model domain was calculated based on the concept first described by Danard (1976). This concept assumes that small scale winds can be seen as a local adaptation of large scale winds (free winds) due to local fluxes of heat and momentum from the sea or earth surface. Any non-linear interaction between the scales is neglected. It is also assumed that the adaptation process is very fast and that horizontal processes can be described by non-linear equations while the vertical processes can be parameterized as linear functions. The large scale winds as well as vertical fluxes of momentum and temperature are estimated from profile measurements in one or several meteorological masts (called principal masts). For the model domain analysed in this study (35x35 km^2) only one principal mast is used. This is located in the southern part of the city. Topography and land use data for the Danard model are given by 500 m resolution. Since the topography of Stockholm is relatively smooth, without dominating ridges or valleys, the free wind can be assumed to be horizontally uniform in the whole domain.

The dispersion calculations were performed on a 25 m resolution. Individual buildings and street canyons are not resolved but treated using a roughness parameter. In an open area the calculation height is 2 m above ground level. Over a city the simulation will reflect the concentrations at 2 m above roof height. A special treatment of the Gauss model plume length is introduced to avoid unrealistically long plumes. This length depends on the stability and persistency of weather conditions. A detailed description of the model is given in the Airviro User Documentation (SMHI, 1997). Chemical and physical transformation processes of particles as well as dry and wet deposition were neglected in the model calculations of annual mean PM_{10} and number concentrations.

2.3. Assessment of additional exposure from short-term high concentrations of air pollutants in traffic

The calculated air pollution concentrations along the by-pass including the road links in the tunnel have been modelled by WSP for the Förbifart Stockholm project and sent to us to be used in this study. The concentration was modelled for every 100 m (used to calculate road link mean values) and as daily average (00-24) and morning (08) and afternoon (17) rush-hour mean concentration. The concentrations are modelled assuming specific conditions: vehicle emission factors for 2020 (Artemis), 90 km/n speed during free flow traffic, 18 h ventilation in the tunnel, concrete road (not asphalt) and only 50% of the cars having studded tyres.

For the alternative scenario, E4/Essingeleden, the concentrations were modelled as a length weighted mean value for each road link. Rush hour mean concentrations were estimated by adjustment using distributions from two road-side monitoring stations.

In calculation the contributions of short-term very high concentrations of air pollutants to regular exposure, the time-weighted average microenvironmental (tunnel) exposure (Kornartit et al., 2010) concept was used

$$E_i = \sum_{j}^{J} C_j \, t_{ij}$$

Where,

Ei is the time-weighted average air pollutant exposure for person i over the specified time period;

Cj is the air pollutant concentration in microenvironment j (e.g. tunnel link);

tij is the aggregate time that person i spends in microenvironment (e.g. tunnel link);

J is the total number of microenvironments (e.g. tunnel links) that the person i moves through during the specified time period in transit.

Moreover, the microenvironmental exposures per average traveller were adjusted to contribution to annual total exposure, weighted by number of cars, and time spent in different links in the tunnel.

When assessing the additional risk for users of the by-pass road tunnel, the corresponding exposure using the current E4 (as main alternative passing Stockholm) was subtracted.

2.4. Population exposure, baseline mortality and morbidity and calculations of health impacts

Population exposure

Population weighted exposure for the two scenarios was obtained by multiplying the calculated concentrations with the number of people in each 100x100 m grid square, summing all products and dividing by the total population. This procedure has been used in several earlier studies (Johansson et al., 2009; Johansson et al., 2007; Orru et al., 2012; Orru et al., 2009). In order to use data matching the epidemiological studies, the ambient concentration are used as "exposure level".

The population data for Greater Stockholm area was obtained from the Statistics Sweden on 31.12.2012 and was gridded by 100x100 m according to address and registration in the following age groups: 0–9, 10–19, 20–29, 30–39, 40–49, 50–59, 60–64, 65–69, 70–79, 80+ years. The population is kept constant in our scenarios since the future population and its geographical distribution is not so well known.

Impact calculation

For the quantification of the health impacts the following equation was used:

$$\Delta Y = (Y_0 \times pop) \times (e^{\beta \times X} - 1)$$

where Y_0 is the baseline rate; *pop* the number of exposed persons; β the exposure-response relationship (relative risk) and X the estimated excess exposure.

The number of Years of Life Lost (YLL) and decrease of life expectancy were assessed using the WHO software AirQ 2.2.3.

Exposure-response relationships and baseline frequencies

For non-external mortality analysis the following exposure-response (E-R) relationships from previous studies were used: RR=1.08 (95% CI 1.06–1.11%) for 10 μgm^{-3} increase of annual mean NO_X concentration (Nafstad et al., 2004) and RR=1.0168 (95% CI 1.0020–1.0325%) for 10 μgm^{-3} increase of annual mean traffic related (road dust) PM_{10} concentration (Meister et al., 2012). The PM coefficient is for daily mortality and not possible to use to estimate years of life lost.

For calculation of respiratory hospitalisations, RR=1.0114 (95% CI 1.0062–1.0167) per 10 μgm^{-3} increase of PM_{10} was used (Atkinson et al., 2005) and for cardiovascular hospitalisations the RR= 1.009 (95% CI 1.005–1.013%) from the COMEAP meta-analysis (2006) was implemented.

The baseline non-external mortality (A00–R99) as well as hospitalization data for cardiovascular (I00–I99) and respiratory (J00–J99) causes in Stockholm County was retrieved from the databases of The National Board of Health and Welfare of Sweden for year the 2011. The number of cases in Greater Stockholm was calculated based on the distribution of the population.

Regarding the asthma, in a study on air pollution effects in children initiated by Naturvårdsverket (Nerhagen et al., 2013) the literature search 2010 identified combinations of design (follow-up, cross-sectional), spatial exposure assessment (hi-resolution, low-resolution), age group (0–2, 2–5, 5–18 years) and outcome (doctor's diagnosis, wheeze) for which there were at least are three reports with a meta-OR statistically different from unity. Only two such combinations, both for the outcome wheeze in the 5–18 age group and with hi-resolution NO_2 as the exposure indicator, were found. There were four studies with cohort design and five cross-sectional studies. From the prevalence studies a meta-OR of 1.38 (95% CI 1.16–1.64) was estimated per 10 µg/m³.

Of the children with wheeze it can be expected that 50–75% will develop asthma in the future. Since the OR express an estimated change in the prevalence odds associated with a change in exposure level, we have to know the underlying prevalence of the diseases in the population. We make an assumption based on results from a Swedish study (Bjerg et al., 2010), and expect 13/100 children to have wheeze symptoms; hence the current individual risk is 13%. Under the assumption of a linear relation between exposure level and prevalence of wheeze, the estimated meta-OR above corresponds approximately (the alteration between OR and RR made according to Zhang and Yu (Zhang and Yu, 1998)) to a decrease in the prevalence of wheeze by (1.378–1)/10*13 = 0.29 % units (0.29 less per 100) for each 1 µg/m³ decrease in NO_2. The reduction in childhood asthma prevalence is calculated as half of this.

Our exposure scenarios present differences in NO_X, but the concentrations of NO_X to NO_2 are closely correlated. In Stockholm the following conversion formula has been utilized:

$NO_2 = NO_X \text{^} (0.66+37/(NO_X+100))$.

For changes in morbidity associated with ambient air concentrations of vehicle exhaust we have converted modelled NO_X into NO_2.

Adult onset asthma over more than 8 years was studied among 3,609 cohort members from three Swedish cities included in the Respiratory Health in Northern Europe (RHINE) cohort (Modig et al., 2009). Exposure at each participant's home was calculated using dispersion models. The incidence rate was 0.19/100 persons and year, and the OR per 10 μgm^{-3} was 1.54 (95% CI 1.00–2.36). Assuming a similar incidence would approximately correspond to a decrease in the yearly incidence by 1.02 cases per 10,000 persons for each 1 µg/m³ decrease in NO_2. We assume this effect to occur within the range 20–65 years of age.

For effects the bronchitis, the computed RR of 1.22 (95% CI 1.02–1.38) per PM_{10} 10 μgm^{-3} annual, based on Schindler et al. study (2009) was utilized. In their study they expected 43 incidence cases per 1,000 people per 10 μgm^{-3} of PM_{10} during 11 years among adults 20+.

The health effects in the tunnel were assessed for three different age group: (1) expecting that tunnel users would be between age 30–69 with the same probability to travel, (2) between age 30–74 and (3) 30–84 adjusted for the probability to travel, where it would be 50% for persons between 70–79 and 25% for persons aged 80–84 years.

3. RESULTS

3.1. Reduced risk for the Greater Stockholm population

There are all together 1,628 528 persons in Greater Stockholm who's air pollution exposures with and without bypass Förbifart Stockholm was estimated. The population density is larger in central Stockholm (number of persons in 100 m grids), where the current E4 crosses (Fig 1).

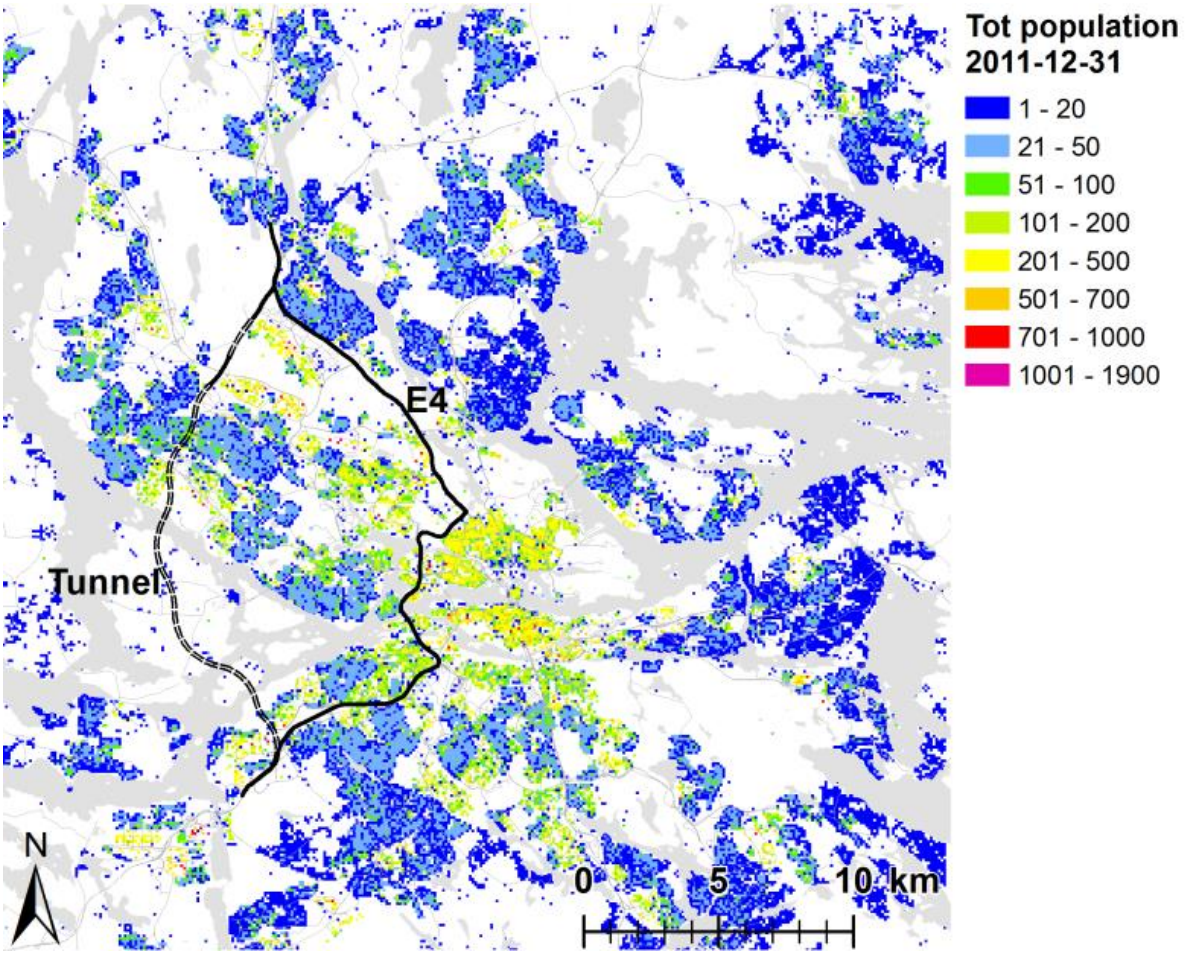

Fig 1. Total population in 31.12.2011 in 100x100 m grids in Greater Stockholm area.

The persons in the study population are distributed by age groups with different base-line rates of non-external mortality and hospitalization due to cardiovascular or respiratory diseases as well as asthma and chronic bronchitis cases. The baseline rate mortality for different age groups is described in Fig 2.

With the by-pass the annual ambient concentrations of the traffic-related pollutants will change (mostly decrease due to traffic intensity decrease, however in some places close to ventilation stacks and entrances, also increase) and all people in Greater Stockholm will be exposed to somewhat different concentrations. The change in air pollutants was calculated with congestion tax on Essingeleden. The larger reduction was notable in central Stockholm and largest increase close to tunnel entrances (Fig 3).

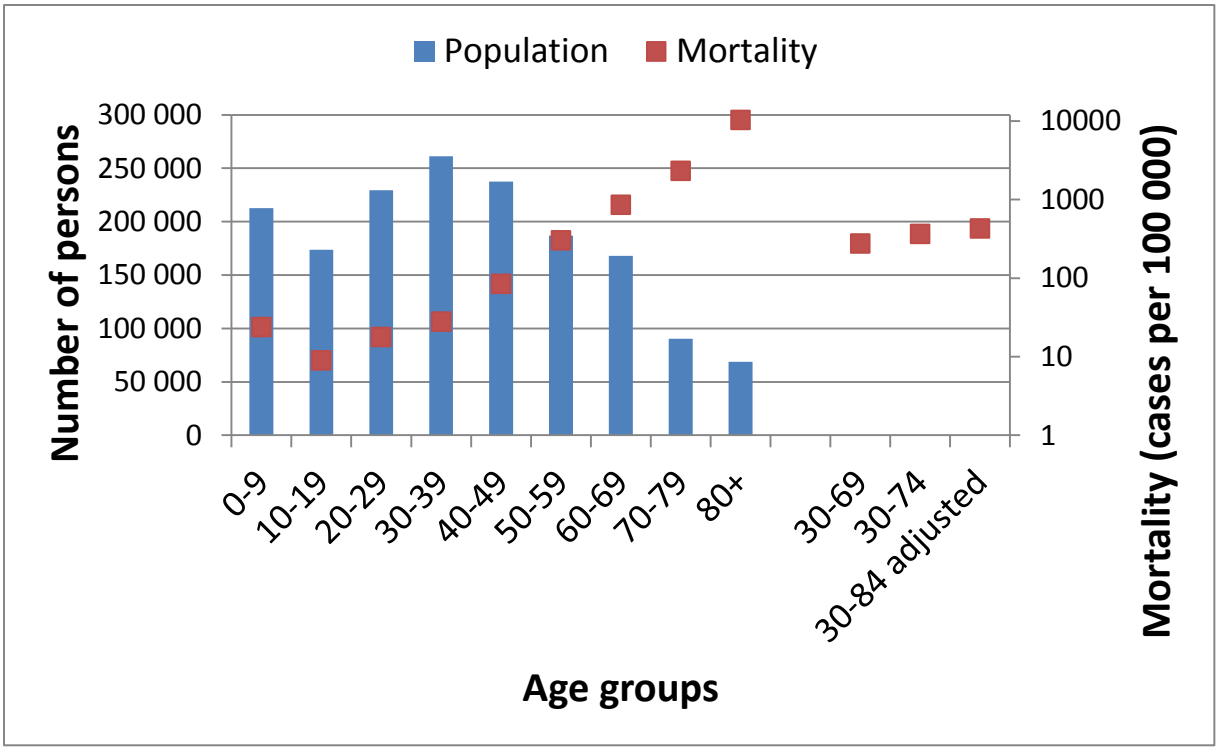

Fig 2. Number of persons in different age groups and base-line mortality rate (red) for the Greater Stockholm area.

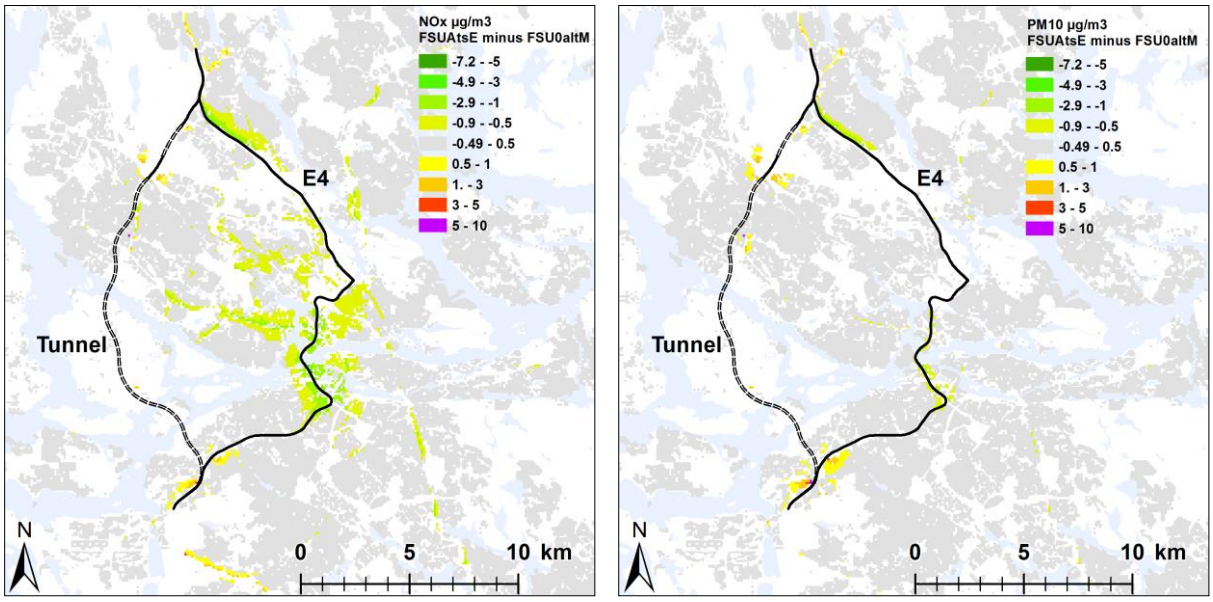

Fig 3. Change in NO_X and PM_{10} annual levels with the by-pass Förbifart Stockholm.

The population average decrease in annual NO_X and PM_{10} levels among adults in Greater Stockholm area are 0.222 μgm^{-3} and 0.020 μgm^{-3}, respectively. This reduction will result in 23.2 (17.6–31.5) fewer premature deaths from traffic exhaust using NO_X as indicator, and 0.5 (0.1–0.8) less death from PM_{10}. The long-term effects due to NO_X exposure will results in

282.9 (196.5–369.3) less Years of Life Lost annually.

Nevertheless, the air pollution reduction will also result in morbidity effects. Among the total population, the PM_{10} exposure decrease would give 1.2 (0.1–2.3) and 0.6 (0.1–1.1) fewer acute cardiovascular and respiratory hospitalizations, respectively (Table 1).

The annual NO_2 exposure would decrease by 0.149 μgm^{-3} among children and 0.207 μgm^{-3} among adults at age 20–65. The asthma prevalence would decrease by 564.4 (237.6–950.6) cases among children and asthma incidence 21.2 (0.1–53.3) cases among adults. In addition PM_{10} exposure decrease among adults by 0.027 μgm^{-3} is expected to decrease chronic bronchitis cases by 2.9 (0.3–5.0) (Table 1).

Table 1. The reduction in health effect in the general population due to decreased exposure in the Greater Stockholm area as a result of Förbifart Stockholm

	Premature mortality (30+)	**Hospitalizations (all ages)**		**Asthma prevalence (<19)**	**Asthma incidence (20–65)**	**Chronic bronchitis incidence (20+)**
		Cardio-vascular	**Respiratory**			
	Number of cases (annually, except asthma prevalence) (95% CI)					
NO_X	23.2 (17.6–31.5)			564.4 (237.6–950.6)	21.2 (0.1–53.3)	
PM_{10}	0.5 (0.1–0.8)	1.2 (0.1–2.3)	0.6 (0.1–1.1)			2.9 (0.3–5.0)

3.2. Additional risk due to increased exposure of persons using Förbifart Stockholm versus exposure on the E4

Due to limited ventilation, the air pollution concentrations in the tunnel can be very high. For instance, during rush hours, the NO_X concentration in some parts of tunnel could increase up to 1957 μgm^{-3}. The rest of the time levels are lower, being for the same tunnel link 382.2 μgm^{-3} as annual average (Table 2). These concentrations are very high compared to maximum concentration along open road, where during rush hours the NO_X concentration is 47.8 μgm^{-3}. Nevertheless, the time spent to pass the tunnel is relatively short being 21.28 minutes during rush hours and 17.61 minutes as annual average. The time-weighted average Förbifart Stockholm micro-environmental exposures is then estimated to be 4.3 μgm^{-3} and 8.4 μgm^{-3} as annual average exposure without and with adjustment for the rush hour situation (Table 2). The comparable exposures in alternative open road link E4 (despite longer travel/exposure time) would be 0.5 and 1.2 μgm^{-3}, respectively. Besides vehicle exhaust also concentrations of wear particles (road dust) will be high in the tunnel, but a recent study has shown that only a small fraction of the coarse particles will penetrated into vehicles, causing marginal increase in exposure (Johansson et al., 2013).

Table 2. Concentrations of pollutants, travel time, daily mean number of vehicles in tunnel and calculated annual average applicable exposures in different by-pass links and in current main road E4

Road type on by-pass	Link no	Annually			During rush hours (6–9 AM and 3–6 PM)		
		NO_x yearly mean (μgm^{-3})	Travel time in tunnel (min)	Daily number of vehicles	NO_x yearly mean (μgm^{-3})	Travel time in tunnel (min)	Daily number of vehicles
Open road	29750	27.6	0.18	41530	42.5	0.25	21837
Open road	29723	36.6	0.16	41530	47.8	0.70	21837
Tunnel	29721	606.5	1.14	41530	168.4	1.76	21837
Tunnel	29734	449.1	4.22	63422	332.0	3.23	26895
Tunnel	30983	601.3	0.51	50901	458.6	0.84	24811
Tunnel	28164	341.9	4.30	61080	418.0	2.83	26451
Tunnel	28163	446.9	0.59	50011	914.4	0.84	24319
Tunnel	28192	469.9	0.52	60897	748.4	0.63	26676
Tunnel	28193	353.3	0.85	60897	348.7	0.83	24258
Tunnel	28365	282.8	0.47	60897	547.7	3.16	26965
Tunnel	28366	382.2	0.91	60897	1096.1	0.82	24173
Open road	28531	25.8	0.50	59297	419.8	2.72	26890
Tunnel	28530	126.5	1.90	58641	989.3	1.73	22056
Open road	28547	16.5	1.12	58641	47.8	0.70	22056
Open road	33105	19.6	0.27	58641	42.5	0.26	22056
Total travel time			17.61			21.28	
Average number of vehicles (one way)				55254			24208
Tunnel exposure (one way)		4.3			8.4		
E4 exposure (one way)		0.5			1.2		

As people while travelling on the by-pass with long tunnels are exposed to high concentration of exhaust pollutants, this will be reflected in health effects as well. However, the majority of users will be of working age and younger seniors, and likely not so many from the oldest and most sensitive group. Currently in congestion charging areas there have been counted 1.3 persons per car, but due to buses and the expected higher costs to travel this number per vehicle could be assumed to be bigger in the future (1.5).

It appeared that the annual mean exposure in Förbifart Stockholm compared to with the current route (E4) would cause 15.1 (CI 95% 11.0–19.8) more premature deaths assuming travellers to come from the age group 30–74 (Table 3). If we expect tunnel users to be younger (30–69) or older (30–84 adjusted), the effects would be smaller or bigger: 11.4 (95% CI 8.2–14.9) and 17.8 (95% CI 12.9–23.2) premature deaths, respectively. If we expect 1.5 passengers in the car, the effects would be 15% larger.

Nevertheless, during rush hours the pollutant concentration are much higher, travel (exposure) time longer and more people use the tunnel, thus we expect that using the annual concentration gives an underestimation of the real effects. We developed the scenario where during peak rush hours (7–8 AM and 4–5 PM) as one hour before and after, users would be exposed to the "rush hours concentrations" with "rush hours travel time", and the rest of the day we assume the "free flow travel time" with "annual concentrations". With this kind of exposure the Förbifart Stockholm scenario would cause 20.6 (95% CI 14.1–25.6) more premature deaths using age group 30–74 (Table 3). Using the age groups 30–69 and 30–84 adjusted, the effects would be 15.5 (95% CI 10.6–19.3) and 24.2 (95% CI 16.6–30.0) more premature deaths per year, respectively. In these age groups this would mean on average from 382.3 to 496.3 Years of Life Lost. In the worst scenario adjusted for rush-hour exposure, the age group 30–84 adjusted 1.5 persons per vehicle, the effect would be an annual increase of 27.9 (95% CI 19.1–34.7) premature deaths with 572.6 (95% CI 400.6–741.9) Years of Life Lost (Table 3).

Table 3. Additional risk from using the tunnel in different age groups (number on annual premature mortality cases, 95% CI)

	30–69	**30–74**	**30–85 adjusted***	**30–69**	**30–74**	**30–85 adjusted***
	1.3 persons per car			**1.5 persons per car**		
Based on annual average concentrations						
Increase of NO_X annual average by 3.8 μgm^{-3}, weighted by number of tunnel users in every hour	11.4 (8.2–14.9)	15.1 (11.0–19.8)	17.8 (12.9–23.2)	13.1 (9.5–17.2)	17.5 (12.6–22.8)	20.5 (14.8–26.8)
Years of Life Lost	284.1 (198.9–368.0)	328.6 (229.9–425.8)	366.6 (255.9–476.3)	327.8 (229.5–424.6)	379.2 (265.3–491.3)	423.0 (295.3–549.6)
Based on morning (6–9 AM) and evening (3–6 PM) rush hours concentrations and free flow as annual average						
Increase of NO_X annual average by 7.2 μgm^{-3} during rush hours, rest as annual average with free flow travel time	15.5 (10.6–19.3)	20.6 (14.1–25.6)	24.2 (16.6–30.0)	17.9 (12.3–22.2)	23.8 (16.3–29.5)	27.9 (19.1–34.7)
Years of Life Lost	382.3 (268.8–493.1)	443.3 (311.3–572.2)	496.3 (347.–643.0)	441.2 (310.2–569.0)	511.5 (359.2–660.2)	572.6 (400.6–741.9)

*From age 30–69 all, from age 70–79 half and from age 80–84 quarter using tunnel.

Subsequently also the additional risk of increased personal exposure using Förbifart Stockholm *vs* the current E4 route was calculated. It appeared that while a daily commuter using E4 as alternative for by-pass would increase mortality risk by 1.4% (95% CI 1.0–1.9) , and by using Förbifart Stockholm with average concentrations the mortality increase would be more than four times higher, 6.1% (95% CI 4.6–8.4) (Table 4). If passing the whole tunnel every working during morning and evening rush hour, the increase in mortality would be as high as 9.6% (7.2–13.2); meaning life expectancy decrease by 0.36 (95% CI 0.25–0.47) years for people 30–74 years of age. However, not all people travel the whole distance, passing the half, would decrease the risk by 50% (Table 4).

Table 4. Increase in mortality and decrease of life expectancy among adults (intermediate age group, 30–74) in different commuting scenarios (95% CI)

	NO_X yearly mean (μgm^{-3})	**Increase in mortality (%, 95 CI)**	**Decrease of life expectancy (years, 95% CI)**
Average commuter	7.6	6.1 (4.6–8.4)	0.23 (0.16–0.30)
Using whole tunnel twice a day 5 times a week during rush hours	12.0	9.6 (7.2–13.2)	0.36 (0.25–0.47)
Using half of tunnel twice a day 5 times a week during rush hours	6.0	4.8 (3.6–6.6)	0.18 (0.13–0.24)
Using whole E4/E20 twice a day 5 times a week during rush hours	1.7	1.4 (1.0–1.9)	0.05 (0.04–0.07)

4. DISCUSSION – CRITICAL ASSUMPTIONS

4.1. Number and age-distribution of tunnel users

The main challenge has in this study been to assess the impact of air pollution exposure in special traffic situations. Especially those who will use the bypass tunnel will during short periods be exposed for very high concentration of pollutants that are not common in ambient air. Most epidemiological air pollution studies, in particular studies of long-term exposure, deal with concentrations in ambient air. The fact we used current population and baseline rates instead of expected future values is not so critical. However, the impact calculations are very sensitive to the predicted number and age distribution of tunnel users (older people are much more susceptible with a higher risk of dying). These factors can affect the results to the extent that conclusions may become very different regarding the total impact on health.

4.2. Outdoor concentrations and indoor/in-cabin exposure

This impact assessment deals both with traffic pollution impacts on the general population (using the registered population at residential address), and with effects on commuters due to exposure in vehicles. The epidemiological studies that provided the applied exposure-response functions, were all using outdoor air pollution concentrations, despite the fact that people spend most of their time indoors. It is known that indoor levels of air pollutants from traffic are much lower than outdoor concentrations; however with an indoor/outdoor ratio that vary between study sites, buildings, seasons and pollutants. In different studies the infiltration factors have ranged from 0.1 to 0.95 (Hoek et al., 2013; Hoek et al., 2008), being especially high for PNC (Fuller et al., 2013). According to several studies in cars, the reduction of actual exposure will be similar as for the buildings where subjects in the epidemiological spent most of their time (e.g. (Xu and Zhu, 2009)). Most studies addressing the issue of penetration efficiencies in vehicles deal with ultrafine particles and results largely depend on ventilation, vehicle age and driving speed (Hudda et al., 2012; Wu et al., 2013). Wu et al. (2013) compared $PM_{2.5}$ concentrations while using different commuting modes: in-cabin of bus, taxi and metro, versus on-roadway walking or cycling and showed higher concentrations in road ways compared to in-cabins. They also showed that the use of air-conditioning can effectively reduce exposure levels.

It can be concluded that the variability in penetration of particles into buildings and into vehicle cabins is very large, but not significantly different. Thus we assume that we can apply the exposure-response function found in epidemiological studies for the effect of vehicle exhaust on mortality (NO_X being the indicator). We have not included any estimates for morbidity in relation to in-traffic exposure, but do not exclude the possibility that these effects may exist in a dose-dependent manner. We assume from recent measurements that the wear particles (mostly coarse fraction) have a negligible infiltration rate, and do not calculate any additional impacts from PM_{10} in traffic (Johansson et al., 2013).

4.3. Effect of short-term very high exposures using long-term coefficients

The risk estimates per concentration unit from long-term exposure studies are usually several times bigger than those of short-term exposure studies, demonstrating that long-term effects are not merely a sum of short-term effects. However, parts of the higher mortality and morbidity among persons with a higher long-term exposure is probably related to higher or more frequent short-term peaks in exposure. Most persons susceptible to the effects of short-term exposures suffer from some (known or unknown) conditions or disease. In this group, traffic pollution exposure may trigger acute exacerbations of disease and premature deaths. Induction of disease due to traffic pollution exposure may take years and be associated e.g with acceleration of inflammatory processes in the case of cardiovascular and respiratory diseases. It is not known how the long-term effects of a given dose depends on the exposure pattern, if repeated short episodes of very high exposure results in different total burden.

The recent WHO REVIHAAP project (2013) discussed the lack of data on effects of very short exposures. There are few epidemiological studies of shorter than daily air pollution exposures, especially since daily 1-hour maximum values and daily means usually have a high correlation. A few panel studies have associated very-short term changes in ambient or personal particle exposure to adverse physiological effects. Such studies have reported physiological changes that occur within hours of changes in PM exposure (Burgan et al., 2010; Delfino et al., 2010; Schneider et al., 2010). In exposure chamber studies of diesel exhaust exposure and airway inflammatory responses, there are few studies and inconsistent results on 100 μgm^{-3} PM exposure over 1–2 hours (Ghio et al, 2012). Experimental studies have also investigated health effects of traffic-generated air pollution using volunteers exposed to real-life mixtures of air pollutants from traffic. Studies using concentrated ambient particles from city air (Gong et al., 2008; Graff et al., 2009), studies of exposure during car and bus trips (Adar et al., 2007a; Adar et al., 2007b; Langrish et al., 2012; Laumbach et al., 2010), of cyclist, pedestrians and being close to traffic (Langrish et al., 2012; Weichenthal et al., 2011) (Dales et al., 2007; McCreanor et al., 2007; Rundell et al., 2007) have indicated that concentrations of traffic-related $PM_{2.5}$ in the range of 20–100 μgm^{-3} over under 0.5–2 hours is enough to show mild effects in blood, lungs and on the circulation also in small study populations. Measured as elemental carbon or black carbon it may in some of these with the lowest exposure have been concentrations in the range of only 3–6 μgm^{-3} which resulted in the observed effects. In the most susceptible persons, these changes might further lead to more serious exacerbations of chronic disease, but these toxicological experimental studies are not possible to use for statements on the role of repeated, short high exposures for the cumulative effects on mortality or induction of new cases.

A few small human exposure studies have been done in a Stockholm road tunnel. In healthy subjects 2 hours of exposure to 64 µgm-3 PM2.5 (median concentration) resulted in airway inflammatory response (Larsson et al, 2007). In asthmatics a 30 minutes exposure session with

95 µgm-3 PM2.5 (median concentration) resulted in increased hyperresponsiveness to inhaled allergens (Svartengren et al, 2000), and in a later tunnel study asthmatics showed increased symptoms and decreased peak expiratory flow after 2 hours in 80 µgm-3 (median level) PM2.5 (Larsson et al, 2010). A large proportion of PM2.5 in these studies should have been wear particles and not exhaust particles.

In our health impact assessment we have chosen to use geographically the most relevant study from Oslo (Nafstad et al., 2004) of long-term exposure (with good resolution) to traffic pollution and mortality to estimate both the effects of small reductions in annual mean ambient levels and the effects of the increased in exposure among those using the tunnel. Nevertheless, the latter application could be questioned in several ways, e.g. if the high peak exposures trigger more fatal events than a linear association would suggest.

5. CONCLUSIONS

The analysis showed that depending on the in-vehicle exposure of tunnel users, age (and sensitivity) of tunnel users and number of persons in the vehicle, the total impact on health of the changes in air pollution exposure driven by the bypass project can be beneficial or adverse. The calculations were highly sensitive to assumptions that have to be included.

First of all, the higher the concentrations of harmful pollutants in the tunnel are, the bigger the increase in risk will be. Moreover, the longer the time spent in the tunnel (especially during more congest situations during rush hours), the higher dose will become. Second, if we expect older more vulnerable people using the tunnel users, this would increase the mortality risk. As older people probably will work and commute in the future, this exposure of elderly will likely increase. Third, the more persons in the car, the larger number of people will be exposed and the larger the impact will become. Forth, if the tunnel ventilation or air pollution dispersion in reality will be poorer, increasing air pollution exposure, this would increase the also the negative effects on health.

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APPENDIX

Underlag för befolkningsexponering och resenärsexponering

I denna rapport redovisas resultat av exponeringsberäkningar som utförts som en del av forskningsprojektet ”Total hälsokonsekvensbedömning av luftföroreningsexponering för olika transportlösningar”. Syftet med hela projektet är att beräkna hälsokonsekvenserna av tunnelexponering i ett helhetsperspektiv som omfattar dels olika normer för tunnelluften (halter), dels tunnelns konsekvenser genom Stockholmarnas lägre exponering via omgivningsluften samt även inkluderar trafikanternas exponering i gatumiljön.

I denna rapport beskrivs hur modelleringen av befolkningens totala exponering för kväveoxid och partiklar har utförts för ett scenario med Förbifart Stockholm utbyggd samt två nollalternativ. Vidare beskrivs metod och resultat av trafikantexponering för olika vägval, med och utan Förbifart Stockholm.

Projektet har genomförts för området Storstockholm, i ett område omfattande 35 gånger 35 km. Beräkningar av PM_{10} och NO_2 halter har utförts med SMHI-Airviro gaussmodell. Haltberäkningar för partiklar (PM_{10} och PM_{avgas}) och kväveoxider (NO_X) har utförts för två scenarier år 2030:

- FS_0altM: nollalternativ med avgift på Essingeleden
- FS_FSUAtsE: utbyggnadsalternativ där Förbifart Stockholm är byggd och avgift är införd på Essingeleden.

Förutsättningar för spridningsberäkningarna:

- 90 km/h för Förbifarten vid fritt flöde.
- Emissionsfaktorer för år 2020 (Artemis) med hänsyn till vägens lutning.
- 50 % dubbade vinterdäck på ytvägnätet.
- Betongbeläggning som antas reducera PM_{10} emissionerna jämfört med asfalt
- 18 h ventilation i Förbifartens tunnlar, mellan kl 5:30 och kl 23:30.
- Max 800 μgm^{-3} PM_{10} i tunnelluften.

Befolkningsexponering

Befolkningen är indelad i åldersklasser. Åldersindelning är i 10 årsgrupper, 0-9 år, 10-19 år t o m 100 samt två extraklasser med 60-64 år och 64-69 år. Totala befolkningen i beräkningsområdet Storstockholm är 1 628 528 personer. Prognos för 2030 har erhållits från WSP och visar prognostiserad befolkning år 2030 i större statistikområden. Befolkning år 2030 för Storstockholmsområdet är beräknad till ca 1 816 000, ca 187 500 fler personer än SCB statistiken per 2011-12-31.

Halterna har beräknats i 25 m x 25 m och 100 m x 100 m rutor. I vår analys redovisas resultatet från båda beräkningsupplösningarna. Halterna som har tagits fram är det lokala haltbidraget från trafiken. Ingen urban eller regional bakgrundshalt har adderats till resultatet.

Befolkningsviktade medelvärden för fallet med Förbifarten, Nollalternativet med avgifter på Essingeleden och Nollalternativet utan avgifter framgår av nedanstående tabell.

Scenario	**NO_X µgm^{-3}**	**PM_{10} µgm^{-3}**	**Skillnad µgm^{-3}**	
Med Förbifart	4,17	1,67		
Noll m avg.	4,38	1,69	0,21	0,02

Skillnaderna i exponering är små. För NO_X minskar exponeringen med 0,21 µgm^{-3} med Förbifarten jämfört med nollalternativet med avgifter på Essingeleden.

För PM_{10} minskar exponeringen med 0,02 µgm-3 med Förbifarten jämfört med nollalternativet med avgifter på Essingeleden.

Resvägsexponering

Resvägsexponering har beräknats för två alternativa resvägar. Lindvreten tpl till Häggvik tpl via E4:an (resrutt 1) alternativt via Förbifart Stockholm (resrutt 2). Sträckan är 27,9 km respektive 21,5 km. För resrutt 1, via E4:ans ytvägnät, beräknades ett längdviktat medelvärde för trafikantexponeringen som summan av alla produkter mellan halt och längden av väglänkar dividerat med summan av längden av alla smålänkar. För att få haltbidraget i rusningstid för resrutt 1 används variationen i uppmätta värden över dygnet vid stationerna Lilla Essingen intill Essingeleden (E4/E20) och Häggvik intill E4:an i Sollentuna.

För resrutt 2, via Förbifartens tunnlar, har WSP levererat data för halten NO_X och PM_{10} inne i tunneln. Halten inne i Förbifartens tunnlar har beräknats och halten anges var 100:de meter dels som vardagsmedeldygn och dels för rusningstimme kl 08:00 och 17:00. För PM_{10} har även ett vinterdygn i rusning eftermiddag beräknats. Halten för varje väglänk har beräknats genom medelvärdesbildning av i länken ingående 100 meters halter.

Exponeringsdosen beräknades som halten gånger restiden för båda resvägarna. Halterna inne i fordonskupéerna är väsentligt lägre än halterna i omgivningsluften längs vägarna. Detta studeras i ett separat projekt, som innefattar mätningar av i och utanför fordonskupéer i olika fordon; tre olika personbilar, en liten lastbil och en buss.

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