Background

Health impact assessment (HIA) is a combination of procedures, methods and tools by which a policy, programme or project may be evaluated based on its potential effects on the health of a population, and the distribution of those effects [1]. Knowledge of the exposure, baseline mortality or morbidity in the population and exposure-response functions from epidemiological studies helps us to estimate trends in negative health effects associated with alternative scenarios.

One of the first important air pollution HIAs was conducted by Künzli et al. [2]. This study estimated the impacts of traffic air pollution in Austria, France and Switzerland. It showed that in these countries, particulate pollutants cause 40 000 premature deaths, 25 000 new cases of chronic bronchitis, more than 290 000 chronic bronchitis episodes in children, more than 1.5 million asthma attacks and 16 million person-days of restricted activities annually. Other early HIA reports found that men in Holland and the population in the US lose 1-3 years of life due to air pollution [3,4].

Recommendations for HIAs of environmental factors have been published by the World Health Organisation (WHO) and European Centre for Environment and Health in 2000 [5,6]. Even though several authors [7,8] later discussed the details of HIAs in this field, the principles have remained unchanged.

A recent large detailed outdoor air pollution HIA was carried out in the Apheis project that covered 23 European cities [9]. The influence of fine particles ($PM_{2.5}$) on health was assessed as a number of premature deaths and Years of Life Lost (YLL). The study showed that a reduction of $PM_{2.5}$ (particulate matter with diameter less than 2.5 μm) concentration to 15 μg/m^3 in these cities would avert almost 17 000 premature deaths. The average life expectancy at birth would increase more than 2 years in heavily polluted cities like Bucharest, Rome, Tel Aviv [9]. If the WHO air quality guidelines ($PM_{2.5}$ annually <10 μg/m^3) were followed in these cities, the premature death rate would be reduced by 41/100 000 [10].

The average life expectancy at birth among all European Union (EU) citizens in 2000 was estimated to be shortened by 8.6 months due to $PM_{2.5}$ levels in a WHO report [11]. European Commission (EC) has declared that $PM_{2.5}$ has been causing the premature death of 348 000 people in Europe annually [12]. Globally, the annual number of premature deaths due to outdoor fine particles is considered to be between 800 000 [13] and 3 million [14].

If the EU Directive 99/30/EC requirements for PM_{10} (particulate matter with diameter less than 10 μm) were fulfilled (there is presently no limit value for $PM_{2.5}$), the life expectancy would increase by 2.3 months per person [11]. This is equal to 80 000 avoided premature deaths and more than one million YLL annually.

Golub & Strukova [15] analysed numerous HIAs in Russia and found that air pollution causes 87 000 deaths annually in the Russian Federation, which comprises ~4% of the total mortality. Yorifuji et al. observed that if annual $PM_{2.5}$ level in Tokyo, Japan were lowered to below 12 μg/m^3, the mortality would decrease 8% and 6 700 premature deaths would be avoided [16]. A Swedish study on the impact of particulate matter established that it could cause annually more than 4700 premature deaths in cities and almost 600 premature deaths in the countryside of Sweden [17]. All of these findings confirm that air pollution has an important role in causing premature mortality.

The economic costs of health loss due to outdoor pollution can be estimated as well. In the EU, for example, the external costs of air pollution are 50-161 billion EUR annually from premature mortality and 29 billion € from morbidity, which represents more than 1% of the Union's GDP [11]. It is also important to note that majority of the morbidity-related external costs from air pollution are generated in the public health sector not in the health care sector [18].

Even though several indicators are being used for HIAs, their main goal is to quantify negative effects of risk factors and provide guidelines for policymakers, developers, planners, etc. to decrease exposure to air pollution in order to mitigate the negative health effects.

Tallinn

The sources of air pollution in Estonian capital Tallinn (~390 000 inhabitants) are quite complex with an important role played by local heating. Thus, the health impacts of the air pollution are best characterized using $PM_{2.5}$. The negative effects of chronic exposure to fine particles is shown in various epidemiological studies, even at low concentrations [19,20].

HIAs in such towns as Tallinn give valuable knowledge about the health effects of air pollution in less polluted average-size cities and in less studied regions where the economic transition has been very rapid. The current study enhances the explanatory power of HIA methods by incorporating modelling and sectioning approaches for cities, where the air quality measuring network is rare or absent.

Methods

In the current HIA study, data on population, baseline mortality and morbidity, air pollution exposure, exposure-response functions, socio-economical condition and health-care expenses was gathered and applied.

Baseline population, mortality and morbidity data

Population data for Tallinn is based on enquiry from Population Register (02.02.2007) according to address registration in the following age groups: 0–6, 7–17, 18–27, 28–37, 38–47, 48–57, 58–67, 68+ years. The citizens' residences were divided into sections according to neighbourhoods (regions with similar geographical, socio-economic, etc. patterns), forming the smallest administrative units (smaller than city districts) used in city planning and management. Altogether 84 sections (Fig. 1, 2, 3) were formed in order to identify site-specific exposure to air pollution and identify the areas with greatest risk. Neighbourhood is a small and homogeneous section, where air pollution as a risk factor is assumed to be similar. The age-structure of the population in each section was identified and used for calculation of YLL with life-tables methodology. Each section also belongs to one of the 8 city districts.

The total regional baseline mortality was retrieved from statistics on Estonia (International Classification of Diseases – ICD-10, A00-Y98). The morbidity calculations were carried out using hospitalisation data from Estonian Health Insurance Fund (EHIF), which covers the whole population and is the sole purchaser of health care services in the country. Hospitalisations due to two main disease groups were included in the calculations: cardiovascular (I00-I99) and respiratory causes (J00-J99). Additionally cardiac admissions (I20-I25) and cerebrovascular admissions (I60-I69) were used to match the exposure-response functions for cardiovascular hospitalisations. The short-term effects of high pollution levels on mortality were not calculated separately as according to several authors

[2,9,17] these are already included in exposure-response function of long-term mortality.

Exposure assessment

The annual levels of locally emitted PM2.5 as well as PM_{10} for model validation were estimated with model AirViro [21] based on emission data for traffic, industry, local and central heating along with meteorological parameters with grid resolution 200x200 meters. A database of local heating emissions was developed during the current study, using previous measurements [22] and European Environmental Agency's emission factors for small combustion devices. The traffic flows were measured in Tallinn in 2005 and 2006. The emission factors were taken from CORINAIR [23] for traffic and from a database of pollution licenses for industry, central heating etc.

For model validation the $PM_{2.5}$ and PM_{10} modelled levels were compared with air quality monitoring data from Rahu, Õismäe and Liivalaia receptor points (Fig. 1) for three meteorological years, 2004-2006. The Liivalaia monitoring station is located in city centre and represents a typical city hotspot. The Õismäe monitoring station is an urban background station located in a typical city region with apartment houses where the majority of people of Tallinn reside. The Rahu monitoring station is located in a region of residential houses with a railway with a number of the diesel trains passing by on a daily basis located nearby. In each monitoring station, the concentration of PM_{10} is measured by beta-attenuation analyzers (Thermo Andersen FH-62). In Õismäe station, the PM_{10} levels are measured by reference method (Digitel DHA-80) as well. $PM_{2.5}$ is monitored only at the Õismäe station by beta-attenuation analyzer (Thermo Andersen FH-62).

The difference of the measured and modelled $PM_{2.5}$ concentration at Õismäe station was 21% in 2006. The approximate 2 $\mu g/m^3$ lower $PM_{2.5}$ values from the model indicate somewhat lower background than expected. The average difference for all three monitoring stations above modelled PM_{10} levels for three meteorological years was 18.8%. The biggest difference was in Rahu monitoring station close to a railway with diesel locomotives, where measured and modelled PM_{10} values differed by 37%, while at both Liivalaia and Õismäe monitoring stations the measured and modelled concentrations of PM_{10} differed by only 11%. As concurrence of the measured and modelled PM_{10} levels for the monitoring stations was fairly good, we assume that the model also represents real particle levels fairly good at other receptor points in the city.

The annual levels of $PM_{2.5}$ were calculated for all 84 territorial sections as the average concentration of modelled grid cells in a section. The average concentration of each section was assigned to all citizens of that neighbourhood. Only individuals of age 28+ were included in analyses, as the US cohort study did not include younger persons.

Short-term effects of air pollution were calculated using mean daily average concentrations of PM_{10} from 3 monitoring stations and morbidity data for all age-groups; the small neighbourhoods were combined in this analysis.

Exposure-response functions, calculation of mortality and morbidity

To describe the effects of air pollution on mortality, the broadly employed US ACS study relative risk (RR) 1.06 per 10 $\mu g/m^3$ increase of $PM_{2.5}$ was used as exposure-response relationship [24]. For calculation of respiratory hospitalisations due to short-term air pollution episodes, RR 1.0114 per 10 $\mu g/m^3$ increase of PM_{10} was used [25]. RR of cardiovascular hospitalisations was found as a weighted average based on occurrence of cardiac and cerebrovascular admission with RRs from COMEAP meta-analysis [26].

The cases (mortality and morbidity) were calculated in absolute and relative numbers for all sections in Tallinn. The following equation was used:

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

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

The number of YLL was calculated using "life-tables" methodology, where the hypothetical life expectancy is compared with the life expectancy affected by air pollution. The calculation of YLL and changes in life expectancy were facilitated by a WHO Centre for Environment and Health developed program AirQ 2.2.3 (Air Quality Health Impact Assessment Tool) [27]. For calculation of hospitalisation, the short-term effects module of AirQ was used. The number of hospitalisation cases was determined based on occurrence of different exposure intervals (10–19,9; 20–29,9; …); no effect was assumed below 10 μg/m^3.

Assessment of socio-economic external costs

Air pollution affects economic and social well-being through mortality and morbidity. Morbidity, in turn, affects the health and productivity of the labour force. In this study, the direct costs related to morbidity were calculated using costs of hospitalisation, salary compensation during sick leave and loss of labour input (based on GDP per capita). The data for hospitalisation cost calculations were provided by EHIF, where the average costs of hospitalisation case due to respiratory disease and general internal disease in 2005 was €1239 and €778 respectively [28]. The same source was used to identify the time spent in hospital (6.9 days) and the value of the average compensation of a workday (~€10).

For the country as a whole and its development prospects, the long-term outcomes and costs of air pollution effects are even more important than the direct costs. This means that in a case of premature death, people can lose decades of life-years but direct costs appear only in actual year of death. The concept of *Value of Statistical Life* (VSL) and *Value Of Life Year* (VOLY) are used to express the cost of lost lives and life-years. These concepts stem from people's contribution to GDP, typical work time, salary and sometimes health care (compensation and decreased productivity) costs [29,30]. As there are no comprehensive statistical life valuation studies in Estonia, the conversion coefficient between GDP and the statistical value of life was derived from international meta-analyses (statistical value of life being on average equal to 120 times GDP *per capita* in a country) [31,29]. Value of a life year was calculated from the value of a statistical life following the formula:

$$VOLY = \frac{VSL_A}{T - A}$$

where *VOLY* is statistical value of life year; *VSL* value of statistical life; *A* age when the case happened; *T* life expectancy; *T* – *A* loss of life.

A sensitivity analysis was performed using minimal and maximal economic values of statistical life and life-year to describe the range of potential errors.

Results

Baseline population, mortality and morbidity data

Altogether, 388 964 registered residents of Tallinn were identified in 84 sections of the city. Population-wise the biggest sections had more than 15 000 residents while some of the smallest had less than 100. The population density varied a great deal as well. In most sections, the number of residents ranged from 3 000 to 16 000. Differing number of inhabitants in neighbourhoods gave different proportions to sections in calculation of health impacts.

Based on mortality data, the mortality rates in different age groups were found (average 1 136 cases per 100 000 citizens per year) and calculated in all 84 sections for the reference year. The baseline hospitalisation rates were determined separately for cardiovascular and respiratory admissions per 100 000 people using the same principles. The analysis showed 3 945 and 1 266 annual admission cases respectively per 100 000 people.

Exposure levels

The city centre and nearby regions with local heating can be clearly differentiated as areas with higher exposure to fine particles (Fig. 1). The concentrations are also higher in other regions, especially adjacent to the city centre. High concentrations also appear in smaller residential areas, particularly near busy streets (Fig. 1). The lowest concentrations were in the Tallinn fringe area with small but densely populated residential neighbourhoods.

The exposure to fine particles was calculated by sections, using the modelled annual $PM_{2.5}$ levels in 200x200m grids in Tallinn. To minimize the influence of other sources the minimal modelled value for areas outside the borders of Tallinn (0.42 $\mu g/m^3$) was subtracted.

The content of particulate matter (PM_{10}) in all 3 monitoring stations was quite different. Generally, the concentration was the highest in the town centre and the lowest in residential areas. This is expected as the former has busy traffic and the latter is a blockhouse area. It is noteworthy that in spring for some time the concentration at Õismäe station was even higher than in centre town. As $PM_{2.5}$ levels at that time were not so high, the high pollution episodes were presumably driven by coarse particles ($PM_{2.5-10}$).

Health impacts

As some neighbourhoods had very few inhabitant cases, the number of premature deaths caused by air pollution is presented at the level of city district (Table 1), whereas YLL is given at the level of neighbourhood (Fig. 2).

Our analysis shows that locally emitted air pollutants could cause 296 (76–528) premature deaths in Tallinn. It causes 3 859 (1 023-6 636) YLL in a year, which is 988 YLL per 100 000 citizens. As a total number, the greatest loss (235-650 YLL) was in neighbourhoods with a large number of citizens (25 000-50 000) e.g. Mustamäe, Lilleküla, Väike-Õismäe and Laagna (Fig. 2). The relatively largest loss appeared in the neighbourhoods of city centre Kompassi, Südalinn, Tõnismäe.

Air pollution in Tallinn decreases the life-expectancy of the residents by 0.64 (0.17-1.10) years. The decrease is much greater in city centre, e.g. in Kompassi neighbourhood where it reaches up to 1.17 years; whereas in the least polluted neighbourhoods the decrease of life-expectancy remains between 0.1-0.3 years (Fig. 3). Many of the negative health influence emerge in risk groups (people with respiratory and cardiovascular disease, elderly etc.) [19]. Nevertheless, healthy people may be affected as well. Synergistic interactions with air pollution can appear especially in concurrence with other diseases. However, the average number of YLL per premature mortality case is around 13 years.

Regarding morbidity, short-term exposure to air pollution will cause 71 (43-104) respiratory hospitalisation cases per year in Tallinn. While baseline cardiovascular hospitalisation was higher than respiratory, the influence in absolute numbers is even greater – 204 (131-260) air pollution related hospitalisation cases per year.

In the current study, the assumption was made that negative effects also appear in people with chronic respiratory or cardiovascular symptoms at concentrations below the PM limit values in outdoor air. The threshold value at which negative effects begin to appear is scientifically debated. According to the source of pollutants, their toxicity, and climatic conditions, the number of cases as well as threshold values can differ.

Economic costs

There were 275 short-term air pollution related hospitalisations in Tallinn. Drawing from the average treatment cost data the total direct cost to treat these air pollution related hospitalisations would be ~€245 000. For the days spent in the hospital ~€2 600 was paid to compensate temporary loss of income. The national economy lost input from hospitalised individuals' worth €44 000.

The value of a statistical life in Estonia in 2005 was estimated around 1 million € and the statistical value of a life-year ~€77 000. Based on these values the total indirect loss from air pollution caused premature deaths (296) adds up to €22.9 (5.3-105.4) million.

In summary, the biggest external economic costs related to exposure to outdoor air pollution add up to €23.2 (5.5-105.8) million. The majority stems from loss of life-years from premature deaths. This represents ~0.5% of Tallinn's GDP (€5.2 billion in 2005).

Discussion

Exposure assessment and benefits from methodological advances

While the methodology for HIA follows generally accepted principles [32], major differences appear in exposure assessment. In the case of Tallinn, air pollution is measured in only 3 monitoring sites. Thus, it was necessary to use dispersion modelling to gain an adequate level of detail for exposure assessment. The model validation showed fairly good agreement with monitored levels although the model generally underestimated the particle concentrations. The reason may be incomplete emission database and very high levels of PM at springtime because of road dust.

In our study, the sectioning was determined by a variety of natural and social factors, but the final results largely followed the municipal distribution in Tallinn. Detailed population data was easily available even for small neighbourhoods. In this case, it was feasible to employ it in order to increase the accuracy of analysis. Nevertheless, such detailed data is not always available. The population exposure is usually calculated as the average of monitored particulate matter in a large area, and later adjusted to population density. Our study also used GIS technologies for calculation of average concentration by neighbourhood and explicit demonstration of the variation.

The place of residence was used as the exposure position presuming that the greatest portion of the day is spent there. This is similar to other epidemiological studies from which exposure-response coefficients were taken. Furthermore, site of dwelling was the only data available from the population register. The amount of time a person spends in the residence area and outside of it (work, studies, etc.) affects individual exposure levels, however current methodology does not permit consideration of these variations. Neither could they be considered in the studies providing our exposure-response functions. When doing analysis with such accuracy (which is possible with modelling), individual factors such as home's exact distance from street, other pollution sources, individual sensibility to pollutants, etc. play an important role.

The exposure was highest in the city centre and close to busy streets. People who live or work there, rather than the people who drive through, are more exposed to pollution. The number of people living in Tallinn is possibly bigger than indicated by the data used because of optional registration. In the sectioning process we also lost ~3% of persons, due to miss matching in Population and Land Register datasets.

Critical issues

Firstly, the baseline population and health data as well as exposure information are vital determinants affecting HIA results. Similar statements were made by Tainio et al. [33] in their statistical modelling study. When looking at the absolute numbers, the highest number of casualties occurred in Lasnamäe as the largest number of people lives in this neighbourhood. If we look at relative values (e.g. YLL per 100 000), the greatest numbers are in neighbourhoods of city centre and Kristine. Thus, the absolute number of casualties in Mustamäe and Lasnamäe is in the same range as air pollution exposure is much higher in Mustamäe. The age structure of the population plays a role as well, but it is quite minor.

Questions may arise about the possibility of over(under)estimation of the health impacts. The main basis for overestimation is the high baseline mortality rate (driven by external causes) in Estonia that magnifies the relative exposure impact. On the one hand, it is an indication that people's health is generally weak and thus, they could be more sensitive to air pollution. In some cases, as in the Reshetin & Kazazyan study, where air pollution was said to cause 15-17% of mortality in Russia [34], it is probably overestimated because of very high base-line mortality (on large extent related to alcohol consumption). Of course, we should not be too conservative in our estimations. In Helsinki, where the air pollution influence of busses was assessed, the results could be underestimated because of taking into account only exhaust particles, what are more toxic [35]. Mostly the outcomes are given with 95% CI, what gives an indication of the statistical precision.

Secondly, the choice of dose-response relationship represents an even more critical determinant of HIA results than baseline data. For long-term mortality assessment, we applied Pope et al. [24] relative risk from the ACS study that is broadly used in HIA studies. Since most of the stations in that survey were in urban surroundings, the combustion particles (prevailing in the city centre) may cause relative risk up to 1.17 per 10 $\mu g/m^3$ increase of $PM_{2.5}$ as Jerret et al. found in the California cohort in ACS study [36]. Thus, we may underestimate the effect on mortality. There are many more exposure-response coefficients available, but there is a great deal of variation and additional studies are needed.

A third problematic aspect lies in the choice of pollutant as the pollution indicator. We assumed that most of the heath effects of air pollution could be quantified with $PM_{2.5}$ especially due to the impact of local heating. In HIA by Tonne et al., where effects of $PM_{2.5}$ and NO_2 from traffic were compared – slightly greater influence on health was recognised when NO_2 was the indicator [37]. This means that we may have underestimated the exposure rate in the city centre. One possibility in such cases would be to conduct HIA with several pollutants that would give evidence for designing alternative exposure rate scenarios. However, adding up a cumulative sum of the effects of different pollutants would lead to overestimation and would be methodologically wrong.

The fourth critical issue is the threshold level of particulate matter health effects. Studies have shown that fine particulate matter can cause negative effects on concentrations below limit values [38]. The existence of a threshold value is still unclear, as most studies do not suggest it. In principle, we assume the local contribution to have an impact. The background concentration is often used as the threshold value, as we did in this study of

Tallinn. However, as it is here presumed to be low, we might have slight over-estimation in our results.

Broader relevance of the results

Even though air pollution exposure in Tallinn is relatively low, the number of premature deaths and hospital admissions is alarmingly high. As baseline cardiovascular hospitalisation is much higher in Estonia compared to respiratory hospitalisation, the big difference between them was predictable. The negative effects on morbidity also appear in family physician and other doctors' outpatient records. Due to lack of relevant data, these morbidity cases were not taken into account.

The average loss of life expectancy (at birth) is slightly less (7.7 months) than among all EU citizens (8.6 months) [11]. The rate of premature deaths (76/100 000) is almost the same as EC study's 75/100 000 among EU-25 residents [12].

The total external costs estimated at €23.1 million EEK make up 0.5% of the Tallinn GDP (in 2005). This is quite a conservative estimate compared to findings from Russia 2.6-6.5% [15] and Beijing 6.55% [39], which are of course much more polluted. But even compared to WHO European assessment's 1.5%, it is modest [11].

The use of HIA as an assessment tool is based on assumptions that local situation and social factors are similar to the reference conditions used in the epidemiological studies from which the exposure-response coefficients are derived. The design of epidemiological studies for long-term effects of air pollution is complicated, especially in small or average size towns where air quality and social patterns vary. Thus, the current study design is the most applicable in the case of limited resources.

Conclusions

The methodology we used helped to assess health impacts of air pollution a town with sparse monitoring network but where dispersion modelling was available. The variety of factors in the city itself was large. Dividing the town into sections helped improve the comprehensiveness of the assessment. To some extent, all the citizens of Tallinn are affected by poor air quality. From the heath-impact point of view, exposure to $PM_{2.5}$ in the long-term and exposure to PM_{10} in the short-term are important.

Altogether, 296 premature deaths and 3859 YLL in a year, average loss of 7.7 months life expectancy and 275 hospital admissions due to air pollution make particle pollution a significant environmental health issue in Tallinn. People suffering from chronic diseases should be informed about the air quality in different regions. Sectioning the city for analysis and using GIS implementations helped to improve the accuracy of impact estimations.

The main reasons for the high levels of air pollution in the city centre are traffic and local heating in close-by areas. To reduce exposure, traffic emissions should be minimised and diverted from the dwelling areas; use of light traffic (walking, cycling) and public transport as well as access to environmental health information should be promoted. Even the levels on particulates are not big; still the negative health effects appear. It means that these kind of studies are needed in areas with average pollution levels as well

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

HO and BF developed the overall concept of current HIA methodology; ET conducted dispersion modelling; TL made economic evaluation and determined baseline health data; TT made GIS designs; MK and VK improved pollution emission database; EM contributed to general health impact background analysis; KK contributed to the interpretation of the analysis results and their applicability in urban risk regulation, and HO performed most of the analyses and drafted the manuscript. All authors have read and approved the final manuscript.

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Figures

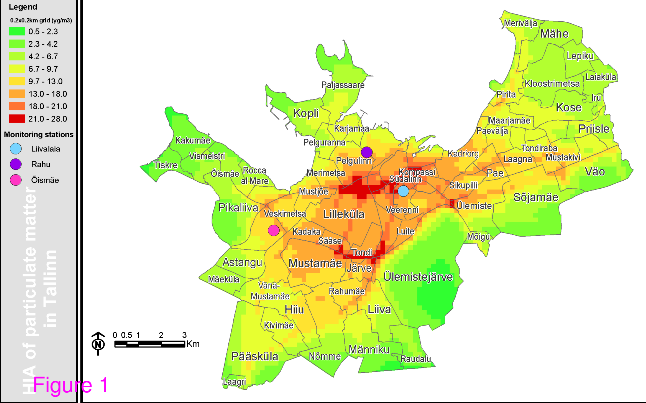
Figure 1. Modelled (200x200 m grid) annual average concentration of $PM_{2.5}$ in Tallinn, $\mu g/m^3$.

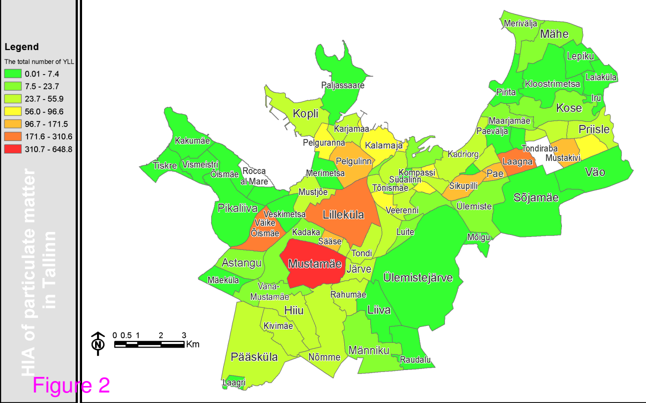
Figure 2. The total number of YLL due to $PM_{2.5}$ pollution in Tallinn.

Figure 3. Decrease of life-expectancy due to $PM_{2.5}$ pollution in Tallinn.

Tables

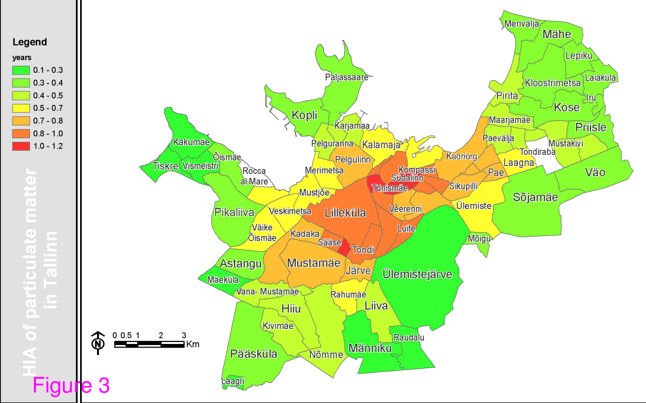
Table 1. The number of premature death due to $PM_{2.5}$ pollution in Tallinn


HIA of particulate matter in Tallinn
Legend
0.2x0.2km grid (yg/m3)
0.5 - 2.3
2.3 - 4.2
4.2 - 6.7
6.7 - 9.7
9.7 - 13.0
13.0 - 18.0
18.0 - 21.0
21.0 - 28.0
Monitoring stations
Liivalaia
Rahu
Õismäe
0 0.5 1 2 3 Km
N
Merivälja
Mähe
Lepiku
Laiaküla
Kloostrimetsa
Iru
Pirita
Kose
Maarjamäe
Paevälja
Priisle
Tondiraba
Laagna
Mustakivi
Väo
Kadriorg
Pae
Sõjamäe
Paljassaare
Kopli
Karjamaa
Pelguranna
Pelgulinn
Kakumäe
Vismeistri
Tiskre
Õismäe
Rocca al Mare
Merimetsa
Kompassi
Südalinn
Sikupilli
Mustjõe
Ülemiste
Pikaliiva
Veskimetsa
Lilleküla
Veerenni
Kadaka
Sääse
Luite
Mõigu
Tondi
Astangu
Mustamäe
Järve
Ülemistejärve
Mäeküla
Vana-Mustamäe
Rahumäe
Hiiu
Liiva
Kivimäe
Pääsküla
Nõmme
Männiku
Raudalu
Laagri
Figure 1



HIA of particulate matter in Tallinn
Legend
The total number of YLL
0.01 - 7.4
7.5 - 23.7
23.7 - 55.9
56.0 - 96.6
96.7 - 171.5
171.6 - 310.6
310.7 - 648.8
0 0.5 1 2 3 Km
N
Merivälja
Mähe
Lepiku
Laiaküla
Kloostrimetsa
Pirita
Iru
Kose
Paljassaare
Kopli
Karjamaa
Maarjamäe
Paevälja
Priisle
Pelguranna
Kalamaja
Tondiraba
Kakumäe
Mustakivi
Laagna
Pelgulinn
Kadriorg
Tiskre
Vismeistri
Õismäe
Rocca al Mare
Kompassi
Südalinn
Pae
Väo
Merimetsa
Tõnismäe
Sikupilli
Mustjõe
Sõjamäe
Pikaliiva
Veskimetsa
Lilleküla
Veerenni
Ülemiste
Väike Õismäe
Kadaka
Luite
Mõigu
Sääse
Tondi
Astangu
Mustamäe
Järve
Ülemistejärve
Mäeküla
Vana-Mustamäe
Rahumäe
Hiiu
Liiva
Kivimäe
Männiku
Pääsküla
Nõmme
Raudalu
Laagri

Figure 2



Legend
years
0.1 - 0.3
0.3 - 0.4
0.4 - 0.5
0.5 - 0.7
0.7 - 0.8
0.8 - 1.0
1.0 - 1.2
HIA of particulate matter in Tallinn
Merivälja
Mähe
Lepiku
Laiaküla
Kloostrimetsa
Paljassaare
Pirita
Iru
Kose
Kopli
Karjamaa
Maarjamäe
Priisle
Paevälja
Mustakivi
Kalamaja
Pelguranna
Tondiraba
Kakumäe
Õismäe
Kadriorg
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Laagna
Tiskre
Vismeistri
Rocca al Mare
Kompassi
Väo
Merimetsa
Südalinn
Pae
Tõnismäe
Sikupilli
Mustjõe
Sõjamäe
Lilleküla
Ülemiste
Veskimetsa
Pikaliiva
Veerenni
Väike Õismäe
Luite
Kadaka
Mõigu
Sääse
Tondi
Astangu
Mustamäe
Järve
Ülemistejärve
Mäeküla
Vana- Mustamäe
Rahumäe
Hiiu
Liiva
Kivimäe
Raudalu
0 0.5 1 2 3 Km
N
Männiku
Pääsküla
Nõmme
Laagri

Figure 3

Table 1. The number of premature death due to $PM_{2.5}$ pollution in Tallinn

City district	**Number of population**	**Annual exposure to local $PM_{2.5}$ (µg/m^3)**	**Number of premature deaths (95% CI)**
Haabersti	38 031	9.5	23 (6–42)
Mustamäe	62 589	14.0	63 (16–112)
Nõmme	38 268	7.2	18 (5–31)
Kesklinn	47 105	17.1	51 (13–91)
Kristiine	28 878	16.2	30 (8–54,0)
Lasnamäe	107 280	10.2	73 (19–131)
Pirita	13 192	6.4	5 (1–8)
Põhja-Tallinn	53 621	9.3	33 (9–59)
Total	**388 964**	**11.6**	**296 (76–528)**

Figure 4