Economic cost of the health burden caused by selected pollutants in the Haifa Bay Area

Final report submitted to the Ministry of Environmental Protection

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Abstract:

In Israel, only a few studies conducted detailed analyses of air pollution damage costs, and most of these studies based their cost analysis on environmental emission. During the last two decades, hundreds of epidemiological studies worldwide evaluated the associations between air pollution and adverse health outcomes. Worldwide valuations of the additional burden of disease/mortality from air pollution were usually done on a country scale rather than on a spatial, geo-statistical scale.

The goal of this study was to evaluate the burden of disease and mortality and their economic costs, caused by local emissions of air pollution. The study year was 2015 and all economic data are given in 2017 terms. Pollution effects were estimated for NO2, PM2.5, O3 and Benzene. No VOC was included due to lack of data in the study year, 2015.

The current valuation was detailed, in terms of the symptoms, the pollutants and the different geographical areas and neighborhoods in the Haifa Bay area (HBA). The assessment of the total burden was conducted in three phases:

A. Assessment of the additional pollution levels to the ambient, environmental levels based on local emissions data for the following three pollutants: NO2, PM2.5 and Ozone. Pollutant levels reflect only the local anthropogenic contribution, above or below the background levels (addition or reduction). For Benzene we assessed additional pollution omitted from transportation only.

B. Based on phase A, we performed a risk assessment in order to estimate the attributable number of cases attributable to the exposure difference (i.e., the number of cases that could be avoided if we reduce the pollution from the value in phase A to the reference level). For Benzene we calculated the attribution based on function of transformation from emissions.

C. Estimation of the economic burden of the extra morbidity and mortality assessed in phase B.

Phase A was conducted by using dispersion models of pollutant emissions from energy sources, other industrial sources, and transportation. The data from phase A was processed by GIS into different areas in Haifa.

Calculation was done using the AERMOD model. To run the model, the following input data and parameters were used:
A. Data on anthropogenic pollutant emissions.
B. Meteorological data
C. Topographic data
D. Receptor network
E. Durations for mean concentrations
F. Contribution of anthropogenic sources of pollution to pollutant concentrations were calculated once as a contribution of all sources and once separately, for the different industrial/energy and transport sources.

For Ozone, which is a secondary pollutant, the distribution of concentrations was received from CHIMERE model. The spatial resolution was set to a receptor grid of approx. 3 x 3 km. The computed concentrations for Ozone include background values as well as the photochemical effect of the anthropogenic sources. This effect is reflected in lower than natural background values (represented by the values for the Mediterranean Sea area).

Mean air pollution level was calculated for each statistical area, using a GIS software. The area was divided to 179 statistical areas of which 156 have population-at-risk for morbidity and mortality.

For each statistical area we calculated an exposure difference, representing the difference between the mean value of air pollution (from Phase A) and the reference level for all the pollutants except Ozone. For Ozone we calculated the difference between the Ozone levels calculated in phase A and the value over the Mediterranean Sea.

**In Phase B,** to select the concentration response functions (CRF or dose-response function) we first conducted a wide literature review, which includes the WHO’s review performed for the “Health risks of air pollution in Europe—HRAPIE” project, ~50 meta-analyses and 20 Israeli studies. The criteria for inclusion and selection of the dose-response function were: studies with exposure levels and population similar to Israel, statistically significant CRF, CRF from cohort studies on long term exposures that allows to integrate all the long-term effects, and studies that were the most recent published. Where no relevant study was found for long term exposure, a CRF for short-term exposure was selected (based on a daily or 8 hours mean values), this is an underestimation of the exposure-outcomes effects.

A total of eight disease groups were found ( Type 2 Diabetes mellitus, Term Low birth weight, Lung cancer, Stroke, Cardiovascular Morbidity, Chronic obstructive pulmonary
disease (COPD), Asthma, Acute lower respiratory infection(ALRI) and non-accidental mortality. 21 CRfs were defined, one for each pollutant-health outcome-pair.

In order to calculate the attributable morbidity and mortality from local emissions, in each statistical area the total number of patients was multiplied by the specific PAF (population Attributable Function- percentage of avoidable morbidity/mortality cases in each statistical area, if pollution level was equal to the reference level). The total number of patients was calculated based on the national morbidity rates in Israel and the population-at-risk. The specific PAF was calculated based on CRF taken from the literature, and the delta between the mean pollution values calculated in Phase A and the reference level.

Data on national background morbidity rates for Israel were taken from hospitalization and mortality data from the Ministry of health and were calculated from epidemiological studies from Israel. The population-at-risk size in each statistical area relevant for each symptom, according to literature, was based on the 2008 Census population.

The results of phase B where categorized according to additional symptoms by disease and early death. Maximal and minimal values were calculated assuming zero correlation or full correlation between the different pollutants. Attributable morbidity and mortality were calculated according to the specific emission sector – industrial or transportation.

Number of incidences for morbidity and mortality are given in tables 1 and 2 respectively.

Table 1. Total occurrence of cases of symptoms “contributed” by local emissions

<table>
<thead>
<tr>
<th>Disease</th>
<th>Studied morbidity</th>
<th>NO₂</th>
<th>O₃</th>
<th>PM2.5</th>
<th>Benzene</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Heart Disease</td>
<td>Hospitalizations or Death</td>
<td>85</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>Incidence</td>
<td></td>
<td></td>
<td></td>
<td>5</td>
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<td>Stroke</td>
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<td>Diabetes</td>
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<td>1,070</td>
<td>-353</td>
<td>262</td>
<td></td>
</tr>
<tr>
<td>Lung cancer</td>
<td>Incidence</td>
<td>2</td>
<td>0</td>
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</tr>
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<td>ALRI</td>
<td>Incidence</td>
<td></td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>Disease</td>
<td>Event</td>
<td>Count</td>
<td>Rate</td>
<td></td>
<td></td>
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<td>-------------</td>
<td>-----------</td>
<td>-------</td>
<td>------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td>Hospitalizations</td>
<td>138</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Term LBW</td>
<td>Births</td>
<td>37</td>
<td>13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukemia</td>
<td>Incidence</td>
<td></td>
<td>1.8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Total number of occurrences of death cases from disease, “contributed” by local emissions.

<table>
<thead>
<tr>
<th>Pollution source</th>
<th>NO₂</th>
<th>O₃</th>
<th>PM₂.₅</th>
<th>Benzene</th>
<th>All pollutants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Industrial sources</td>
<td>7.21</td>
<td></td>
<td>7.16</td>
<td>14.36</td>
<td></td>
</tr>
<tr>
<td>Transportation sources</td>
<td>72.75</td>
<td></td>
<td>14.59</td>
<td>87.33</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>79.96</td>
<td>-5.35</td>
<td>21.75</td>
<td>0.69</td>
<td>101.69</td>
</tr>
</tbody>
</table>

In Phase C, the economic value was quantified using the Cost of Illness (COI) method and Value of Life estimates. Data on both morbidity and mortality was derived from a meta-analysis of 47 studies we conducted that estimated the COI for the different morbidity symptoms. For mortality cost, we used widely used international value adapted by the OECD and adjusted them to Israel. We used both the value of statistical life (VSL) estimated at 12.95 million ILS per life saved as well as the value of a year life lost (VOLY) which was estimated at 0.350 million ILS per year saved.

Summing all the above resulted in a total monetary value for the HBA pollution and provided the basis to conduct further segmentation according to the different pollutants, cells and sectors.

Depending on the assumption of VOLY or VSL, the monetary value therefore ranges between 561 and 1,352 million ILS for the year of 2015. This is an annual burden of 1,835 ILS per resident on average.
Table 3. The total cost of morbidity and mortality from local emissions in the Haifa Bay Area (₪/year)

<table>
<thead>
<tr>
<th></th>
<th>Mortality cost by Voly</th>
<th>Mortality cost by VSL</th>
<th>Morbidity cost</th>
<th>Total cost (using Voly)</th>
<th>Total cost (using VSL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>360,473,493</td>
<td>1,033,138,220</td>
<td>79,889,728</td>
<td>440,363,221</td>
<td>1,113,027,948</td>
</tr>
<tr>
<td>PM2.5</td>
<td>98,082,538</td>
<td>281,110,319</td>
<td>42,549,606</td>
<td>168,762,988</td>
<td>323,659,926</td>
</tr>
<tr>
<td>Benzene</td>
<td>3,100,485</td>
<td>8,886,172</td>
<td>1,170,020</td>
<td>4,270,505</td>
<td>10,056,192</td>
</tr>
<tr>
<td>Total(min)*</td>
<td>360,473,493</td>
<td>1,033,138,220</td>
<td>79,889,728</td>
<td>440,363,221</td>
<td>1,113,027,948</td>
</tr>
<tr>
<td>Total(max)**</td>
<td>437,478,479</td>
<td>1,253,839,036</td>
<td>106,834,981</td>
<td>544,313,460</td>
<td>1,360,674,018</td>
</tr>
</tbody>
</table>

This study is based on data which was available for 2015. There are changes that occurred in the last 5 years; hence, a more accurate report should take those changes into account.

We should note also that we did not include emissions from Haifa port or airport and no economic valuation was done for Volatile Organic Compounds (VOC’s) that were not included in the 2015 emissions inventory. In addition, we did not quantify the effects of SO2 since the attribute values were below threshold values for health effects. Last, Benzene was estimated only for transportation sources due to lack of data for 2015.
הערכה כלכלית של העלויות הבריאותיות качוצאת מתורמת מזיהמי אוויר ב חיפה

תקציר:
עד כה, בוצעה בישראל הערכה של עלויות חיצוניות של זיהום אוויר בסך מעטים מחקרים וברובם התבססו על עלויות המהתבוננות ומיעוט פלטת לטבע. הערכה זו התבססה על המוחלט והנתונים של מזהמים אזורים ו_CHUNK1307 (Burden of disease/Mortality) ששרטולרה על מדיניות אל בדקל ;-) במטרה לשפר את התוספת לחובות של אנרגיה סטטי.- 3." targeting="_blank"

הערכה שNguồnה מחויבות חירום יישימה בברית המועצות שלהנהגמה בשנית מזיהמי אוויר. הערכה זו מצא notifyingполнение ה productList במטרה לשפר את התוספת לחובות של אנרגיה סטטי.- 3." targeting="_blank"

לישראל, מדדי הקשר מובהקים סטטיסטית, מדדי קשר ממחקרי עוקבה של חשיפות כרוניות ייחוס. בפערת של חשיפה ארוכת ימים קצרים, נבחר מדד קשור לחשיפה קצרים מועד (ממוצע לשישה ימים ו ikea השפעה הביא ל الأو(153,584),(844,904)

במידה ולא היה מחקר רלוונטי לחשיפה כרונית, נבחר מדד קשר לחשיפה קצרים מועד (ממוצע ימומי או ממוצע 8 שעות) שהנו הערכה חסר של ההשפעה הבריאותית לאותו המזהם.

ובאילות הנמצאו 8 קבוצות של מחלות (סוכרת מסוג 2, לידת תינוקות במשקל נמוך, סרטן ריאה, מחלות לב וכלי דם הכוללים מחלות לב ושבץ מוחי, מחלות נשימה כולל מחלת ריאות חסימתית כרונית, אסתמה ודלקת ריאות חריפה) ותמותה כללית במ關鍵ה, למעט מתוניות

והוגדרו 21 מדדי קשר לתחום כל תוצאה בריאותי וمخものの אוויר, במידה או שינוי המדידות לקורטירונים מקוונים.

בכדי לחשב את תוספת התחלואה/תמותה הנמצאות המחלות הממוצעיות בכל אזור סטטיסטי (cases attributable) והכלולים sanit בلعب (Attributable Fraction PAF- Population) של חולים בשתי המקרים של חולים בכל מבט סטטיסטי (אן: מקורי התחלואה)

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הנמצא sanit בקצת פלטוגרפים פלטוגרפים פלטוגרפים פלטוגרפים פלטוגרפים

וג_hello (טבלת 1) המחלות (טבלת 1)

טבלת 1 - סDRAM כדי מפריך מקורי הירחוט של סימפטומים "התרוממות" ממלית מקוונים

<table>
<thead>
<tr>
<th>Disease</th>
<th>Studied morbidity</th>
<th>NO_2</th>
<th>O_3</th>
<th>PM2.5</th>
<th>Benzene</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Heart Disease</td>
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<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>O3 cost reduction</td>
<td>-24,178,037</td>
<td>-69,295,675</td>
<td>-16,774,372</td>
<td>-40,952,409</td>
<td>-86,070,048</td>
</tr>
<tr>
<td>Total (min)*</td>
<td>360,473,493</td>
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<td>79,889,728</td>
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<td>106,834,981</td>
<td>544,313,460</td>
<td>1,360,674,018</td>
</tr>
</tbody>
</table>

יש להדגיש שהנתונים באומדן אומנו על שיקוף את התמונות בתחום דברי 2015. בכל הפרמטרים בוכרי לקביל מתון במגזר עדכני להרוךساعدسطحفقد נמוך. דוגמה ששבשמורת בכל פליטת ממקלח החלים מסולבים התועפות וכנל נושאת הערכה לכלכלת לכלכלת. וארדנס נדיפים-voc שבלכל פלישה פליאט 2015. וערדנס, אנו נושאת ערכה של התמונות ונפרדת מערכיה העביב התועפותooled העביב כלכלך הבוראות. בנם שמד רד פליאטוח התבורהית עקב מתחור בנותינו בהתייחס לסנט 2015.
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1. Introduction

1.1 General introduction

**Haifa Bay Area demographics**

The Haifa Bay Area (HBA) is composed of two sub-districts: the Haifa sub-district and the Acre Sub-District. The Haifa sub-district consists of a number of towns and rural settlements. In addition to Haifa (the third largest city in Israel, with approximately 100,273 residents in 2013), urban localities in the Haifa district include Kiryat Ata (app. 50,000 residents), Kiryat Bialik, Kiryat Yam, Kiryat Motzkin (app. 40,000 people) and Nesher (app. 25,000 residents). Other localities in the Haifa metropolitan area near the Haifa Bay industrial zone are Yagur, Kfar Hassidim and Rekhasim. In addition, some of Acre’s population is located in Haifa Bay, mainly Acre’s residents (app. 50,000) and a number of small rural settlements (Grotto et al. 2015). The total population in the HBA in 2015 was 521,230 individuals.

**Air pollution in the Haifa Bay Area**

HBA has been Israel's primary industrial region since the 1930s. It currently hosts petroleum refineries, a power plant, and several large petrochemicals, chemical and agrochemical industries. It is also the home of many medium and small size factories, Israel's largest seaport, and a dense metropolitan area with transportation pollution (Ministry of Environmental Protection Air quality and climate change department 2016).

In 2007, the Ministry of Environmental Protection (MoEP) conducted environmental samplings in HBA, which indicated high aerial concentrations of non-methane volatile organic compounds (NMVOC). In 2008, in the wake of these findings, the MoEP determined that an action plan was necessary in order to reduce these pollution emissions. The action plan pinpointed and focused on mainly 15 facilities and installations with the highest potential for the release of NMVOCs. Following the MoEP’s activities, air pollution emissions from factories in the HBA dropped significantly. The Volatile Organic Compounds (VOCs) emissions from the largest enterprises in HBA (chemicals, petrochemicals, fuel tank farm, and hazardous waste management) dropped by approximately 60% from 2009 to 2015 for Nitrogen oxides (NOx, mostly NO and NO2) by 45%, for Sulfur oxide (SOx, mainly Sulfur dioxide (SO2)) by 90% and by 60% for fine particulate matter (PM2.5). The Ministry’s activities included increased supervision, enforcement, and regulation actions of polluting
businesses, such as individual orders, setting new business license terms including stricter emission standards, as well as requirements to use Best Available Technologies (BAT)\(^1\) for reducing emissions. In addition, during 2011, a shift from low sulfur oil to natural gas began in the petrochemical factories and the power plant (Matzner and Netanyahu 2015; Yinon and Thurston 2017).

However, according to the Pollutant Release and Transfer Register (MIPHLAS, an environmental inventory of pollutants released to air, water and soil), in 2013, aerial emissions in the HBA of VOC/sqkm, were still higher compared to any other areas in Israel. Furthermore, emissions per square kilometer of NOx and SOx in the HBA are ranked third in the country. For Particulate Matter with particles aerodynamic diameter of <10 μm (PM10), the emission per square kilometer is ranked second in the country (Matzner and Netanyahu 2015). The MoEP continues its efforts to reduce pollution, and in September 2015, a national plan to reduce pollution and risks in the HBA for the years 2015-2020 was implemented (Government decision No. 529). (Ministry of Environmental Protection Air Quality and Climate Change Department 2016)

**Morbidity and Mortality in the Haifa Bay Area**

A recent report published by the Ministry of Health summarized in detail the epidemiological data on excess morbidity and mortality in HBA and the epidemiological studies that evaluated the association between these diseases and air pollution or residence in the HBA (Grotto et al. 2015). Briefly, in the Haifa sub-district, there is excess morbidity compared to the national average for heart and respiratory diseases, including worsening of asthma in children, and higher cancer incidence among adult Jews. In Israel there is no national registry for cardiovascular and respiratory diseases (like the national cancer registry in Israel) and therefore the data used refers to crude, unadjusted prevalence rates only and could not identify differences in geographical distribution of incidence rates.

In 2009, the Central Bureau of Statistics (CBS) conducted a health survey that included questions about the diagnosis of a myocardial infarction by a physician among 8,728 households, including 28,968 participants\(^2\). Compared to the national average crude rates

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\(^1\) BAT is not based on cost efficiency at most cases.

(without adjustment) of 22 cases per 1,000 residents, excess morbidity was reported for Haifa and the Haifa district: 32 cases per 1000 residents in the Haifa District (Haifa and Hadera sub-districts) and 35 cases per 1000 residents in the city of Haifa. In addition, the Haifa District and the City of Haifa were found to be higher than the national average in self-reports unadjusted rates for other heart diseases: 32 cases per 1,000 residents, and 39 cases per 1,000 residents, respectively, compared to the national average of 27 cases per 1,000 Residents. In addition, data from the Health and Social Profiles file of the Israeli CBS of 2005-2009, in the Haifa sub-district, the crude rates (per 1000 residents) of chronic diseases of myocardial infarction in 2009, was higher than the national average (74 vs. 68, for ages 50-74).

The report "Health status in Israel 2010" analyzed hospital admissions data for asthma in 2008 by sub-district. According to the report, in the Haifa sub-district, the rate of asthma admissions in 2008 for children aged 0-4 and 5-15 years was the highest in the country. These findings are consistent with the data on hospitalizations due to asthma, in children aged 0-4 years and between 2004-2006, which indicates that the highest rates are in the north of Israel, especially in the Haifa District.

Excess asthma morbidity (measured in crude unadjusted rates) in the Haifa sub-district and the city of Haifa was also reported in the aforementioned CBS health survey: for the question about the diagnosis of asthma by a physician, 40 cases per 1000 residents aged 0-24 in Haifa district and 62 cases per 1000 residents in the city of Haifa were reported, compared to the national average of 30 cases per 1,000 residents.

With regard to cancer incidences, in October 2018, a report by the National Cancer Registry in collaboration with the Israel Cancer Association on cancer incidence for Jews and Arabs by sub-district was published. The measure used was the Standardized Incidence Ratio (SIR), which reflects the observed incidence of cancer (age-adjusted) in a defined area, compared with the expected incidence in that area according to standard population data. It was found that in the Haifa sub-district for both males and females, there is a significant excess of SIRs.

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http://www.cbs.gov.il/reader/?Mlval=cw_usr_view_SHTML&ID=391

(a 9% to 14% increase above expected incidence) in all cancer types, compared to the national average, from 2001 to 2015.

In addition, there was a higher cancer incidence for Jews and Arabs in city of Haifa compared to other Israeli cities\(^5\). Although among Jews aged 0-14 there was no excess incidence of cancer, for ages 15-34 (mainly women), middle-aged adults (ages 35-64, both sexes), and in the elderly (aged 65 and over) excess incidence of cancer was reported.

Furthermore, epidemiological studies in the Haifa sub-district have found associations between industrial air pollution and respiratory disease and asthma in children. In addition, associations between industrial air pollution and cardiovascular diseases were reported. Additionally, epidemiological studies in the Haifa sub-district reported an association between industrial air pollution, residential proximity to roads, fuel tank farms and ambient air pollution and cancer. Although these studies had some methodological limitations, the consistency of the findings in the studies, may be an indication of the strength of these associations in the HBA.

In order to take into account the above data and results, and incorporate it into policy making, one needs to translate these added symptoms into monetary terms. This is the goal we are seeking by the end of the report.

**1.2 Aims of the study**

The objective of this study is to evaluate the attributable burden and economic costs of air pollution emissions (of NO\(_2\), PM\(_{2.5}\), benzene, and O\(_3\)) from industry, energy generation, and transportation in the HBA.

**1.3 The innovation of the study**

In the current study, we used dose-response estimates that were based on a comprehensive scientific literature review of reports published by the WHO and other peer-review journals.
Most of the previous studies evaluated the burden of disease, based on national or other large-scale regions. This is one of the first studies that evaluated the burden of disease from a small-scale analysis.

In addition, the economic burden of disease was evaluated for different sectors. The analysis used air dispersion models to apply the chain of valuation into each one of the statistical regions in the HBA. This was further segmented into stationary and mobile sources of air pollution to distinguish between industry (including energy production) and transportation.

Most of the previous international analyses evaluated the economic burden of air pollution emissions on mortality. This is one of the first projects that aimed to integrate a detailed ambient concentration together with dose-response and translate that into the monetary burden of disease for high resolution of the HBA. This enables us to zoom into what seems to be more problematic areas and simulate changes over time to better understand the impact of policy on emissions of major pollution sources.

1.4 Literature Review

The burden of disease from air pollution

Ambient air pollution is estimated to cause about 4.5 million premature deaths each year worldwide and ranks among the leading risk factors in the Global Burden of Disease (GBD) Study (Forouzanfar et al. 2015). Until now, estimates of the GBD have mainly been produced on national and sub-national levels. The GBD estimates have provided an understanding of the relative importance of different contributors to disease burden and changes in these contributions over time. In the 2015 GBD estimates, ambient PM2.5 was the fifth-ranking mortality risk factor (Cohen et al. 2017). Exposure to PM2.5 caused 4.2 million (95% uncertainty interval [UI] 3.7 million to 4.8 million) deaths and 103.1 million (90.8 million to 115.1 million) disability-adjusted life-years (DALYs) in 2015, representing 7.6% of total global deaths and 4.2% of global DALYs, 59% of these in east and south Asia. Deaths attributable to ambient PM2.5 increased from 3.5 million (95% UI 3.0 million to 4.0 million) in 1990 to 4.2 million (3.7 million to 4.8 million) in 2015. Exposure to ozone caused an additional 254 000 (95% UI 97 000–422 000) deaths and a loss of 4.1 million (1.6 million to 6.8 million) DALYs from chronic obstructive pulmonary disease in 2015.
The GBD estimates, however, are less useful for a small area that has to take decisions on the local scale with direct impacts on residents' daily lives. A recent study in France demonstrated that using exposure levels on a national scale compared to finely spatially-resolved models led to an underestimation of the Population Attributable Fraction (PAF) for PM2.5 for lung cancer (population-weighted median or median of pollution concentrations by 11% and 72% respectively) (Wang et al. 2016; Kulhánová et al. 2018).

**The burden of air-pollution in Israel - previous studies**

In Israel, the attributable burden of premature deaths and disease from air pollution was mainly estimated either on a national level or for a local national project.

Within the GBD 2013 study, for Israel, the estimate of attributable deaths from exposure to ozone and particulate matter for was 2250 or approximately 5% of total deaths (Forouzanfar et al. 2015). According to a report by the Organization for Economic Cooperation and Development (OECD), there was an increase in premature mortality rates due to particulate air pollution during 2015 (265 per million) compared to 2010 (238 per million) and the total estimated cost from premature death from particulate and ozone air pollution was estimated to be $7514 million (2.5% of the GDP), during 2015 (and from PM - 7,043 premature death and from the GDP 2.3%) (Roy and Braathen 2018).

Researchers from the Ministry of Health (Ginsberg et al.; Ginsberg, Kaliner, and Grotto 2016)) calculated a total of 1,908 premature deaths from the exposure to PM2.5, that constitutes 4.7% Population Attributable Fraction (PAF) of total deaths in Israel during 2015. Age group (in five-year increments) specific Concentration Response Functions (CRFs), based on the WHO burden, were used for ischemic heart disease and cerebrovascular disease mortality from PM2.5 in adults, aged 25 years or older. Non-age specific CRFs were obtained for chronic obstructive pulmonary disease (COPD) and lung cancer, as well as for acute lower respiratory infection (ALRI), in children under 5 years of age. The national population weighted average of PM2.5 exposure level was obtained based on mid-2015 data, multiplied by the relevant local monitoring stations annual average PM2.5 level, and divided by the national exposed population figure of 8,608,500. The national average of the PM2.5 levels calculated was 21.57 μg/m³. The Ginsberg (et al. 2016) estimate is comparable to the GBD

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6 PAF is the proportional reduction in population disease or mortality that would occur if exposure to a risk factor were reduced to an alternative ideal exposure scenario.
estimate. Furthermore, Ginsberg et al. (2016) also estimated, for the first time in Israel, the attributable burden of disease from exposure to PM2.5. The researchers estimated that PM2.5 pollution accounted for between 183,000 to 591,000 hospitalization days due to specific diseases (Acute Lower Respiratory Infections, Asthma Incidences or lifetime prevalence, Circulatory Diseases, Coronary Heart Failure, Chronic Obstructive Pulmonary Disease, Type 2 Diabetes mellitus, Ischemic Heart Disease, Lung Cancer, Low Birth Weight, Respiratory Diseases and Stroke). They estimated that the total cost of the burden of disease from PM2.5 pollution (including premature burial costs) amounted to $544 million, $1030 million and $1749 million respectively (or 0.18 %, 0.35 % and 0.59 % of GNP) (Ginsberg et al. 2016).

Samet (2016) in his commentary on Ginsbereg et al. (2016) discussed the need to improve the methods used. Specifically, he argued? the need to improve methods of assessing exposure levels to air pollution, and to provide a more accurate calculation of the burden of diseases and the costs associated with the actual exposure range of all population groups. In Ginsbereg et al. (2016), the authors used a single exposure level for all the population. Samet (2016) discussed the choice of the counterfactual value that is critical in interpreting the findings. Furthermore, the selection of risk values for the adverse effects of air pollution would ideally come from locally based studies and not from studies of populations outside of Israel. The generalization of the risk estimates from other countries is further complicated by the substantial diversity that exists in the the Israeli population.

The report by Ginsberg et al. (2016) sets a much-needed foundation in the field of economic costs of air pollution in Israel and should initiate a further discussion on the role of burden estimation and risk assessment more broadly in regulations intended to advance environmental health in Israel.

2. **Scientific Background**

Valuation of the cost of air pollution is complex when we try to calculate adverse health effects such as illness and mortality risk (Jaramillo et al. 2016). An accepted approach to mortality risk valuation is the Value of a Statistical Life (VSL) method (Viscusi and Aldy, 2003) which attributes a monetary value to small changes in mortality risk. In particular, the VSL of
approximately $9.6 million (in 2000 dollars, which the USEPA has used in their benefit-cost assessments of the Clean Air Act is a common value to use (WHO 2015). The concept used by the OECD is not similar to the one used in the USA. It is based on stated preferences rather than on wage-risk tradeoffs. The current estimate is roughly $4 million in 2015 dollars (Martuzzi and George, 2020).

A second approach is to use the value of a year of life lost (VOLY) (Leksell & Rabl 2001). Here, the age of the individual is of particular importance since a person who dies at the age of 65 will be considered less than an individual who dies at the age of 45. Usually, the value of a year of life is derived by dividing the VSL by 40. This is based on an average life expectancy of 80 years (Hammitt, 2007).

To estimate the cost of morbidity, the leading approach is to calculate the Cost of Illness (COI). These costs are comprised of two major elements. Direct cost refers to the medical costs involved in the identification, treatment, prevention, rehabilitation, and long-term treatment of the disease. The main medical expenditures are specialists, medication, self-monitoring equipment, caregivers, periodic examinations, emergency room visits, and hospitalization. The main non-medical costs are travel, transportation, and patient time. The second element is the indirect cost. These costs are defined as those impairing productivity due to morbidity, such as absence from work, reduction in employment due to a disability, and the time the family and relatives have taken to visit and treat the patient.

There is also a third element which includes the intangible value of the morbidity. Here we may include pain, anxiety, suffering, and loss of quality of life as a result of the disease. Given the difficulty in pricing these costs, most of the research papers and reviews we have explored do not discuss the assessment of these costs. The primary reason is the fact that the only way to receive information about these values is by asking people direct questions which most likely result in an uninformed and probably biased answer. The COI approach is a lower bound and studies have shown an approximate of a 60% markup if these intangible values would have been included (Alberini & Krupnick 2000; Berger et al. 1987; Zhu et al. 2019).

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7 The ExternE project series does take welfare costs into account though (Rabl and Spadaro, 2002).

8 This is correct for studies related to developed countries. Developing countries may have different mark-up for pain and suffering of course.
The OECD report on the cost from road air pollution emission, reported an estimate of the morbidity cost as 10% of the total cost of mortality (OECD 2014). They used the same estimate for evaluating the morbidity cost for ambient air pollution (WHO 2015). The figure of 10% applies when the VSL is used for the mortality assessment, whereas a higher markup is necessary when valuing mortality using the VOLY.

A critical component of the calculation methodology of health costs of air pollution is to use concentration–response functions (or exposure-response function) for key pollutants and to include all the known adverse health outcomes. The European Commission published, as part of the WHO framework, a comprehensive literature review on recommended concentration–response functions and associated background information for several mortality and morbidity effects associated with short and long-term exposure to particulate matter, ozone, and nitrogen dioxide (WHO 2013). Van der Kamp (2015) reviewed the methodological developments from the 1990s to 2014, with a focus on classical air pollution-induced human health damage costs. It was demonstrated that there is a trend of an increase in the fraction of morbidity cost compared to the mortality cost during the last decades in cost-benefit analysis. In Israel, only a single comprehensive economic analysis was conducted to calculate the externalities from air pollution emitted by the transportation and industrial sectors (Becker et al. 2012). Becker (2012) used a detailed analysis of ambient air pollution maps calculated from dispersion models, expected changes in health effect outcomes based on concentration-response function (dose-response), the incidence and prevalence rates for the health endpoints and the population demographic data.

One of the foundations of a comprehensive cost-benefit analysis for air pollution is the inclusion of all known adverse health outcomes, and this requires a systematic literature review of meta-analyses that were published during recent years. Since Becker’s report was published, hundreds of scientific epidemiological studies were published on the associations between air pollution and adverse health outcomes for the general population and sensitive sub-groups (children, elderly, pregnant women and fetuses). Moreover, various meta-analysis studies were conducted and published to evaluate the pooled odds ratio (dose-response) for these adverse health outcomes. For example, few meta-analyses that evaluated the pooled dose-response between exposure to air pollutants and type 2 diabetes were published (Balti et al., 2014; Janghorbani et al., 2014; Wang et al., 2014; Eze et al.,
2015; He et al. 2017). It is important to note that different meta-analyses report different dose-response values. Different inclusion criteria such as the research type, long or short term exposure, the study population, all may affect the pooled dose-response that was evaluated in each study (for example, for a 10 µg/m³ increase in PM2.5, Janghorbani (2014) reported a 2% increased risk and He et al. 2017 reported a 25% increased risk for type 2 Diabetes). This issue had required a careful selection strategy for the specific dose-response selected in our analysis.

Other health outcomes related to air pollution are adverse pregnancy outcomes, like low birth weight (Pedersen et al. 2013; Dadvand et al., 2013), congenital malformation (Chen et al., 2014) and respiratory outcomes such as COPD hospitalization (Li et al., 2015).

In addition, the International Agency for Research on Cancer (IARC) published a monograph on the carcinogenic effect (group A) of ambient particulate matter and lung cancer risk. However, the IARC evaluation did not include a quantitative summary of the evidence and meta-analysis, and this was published later (Hamra et al., 2014).

Currently, decision-makers in Israel face major knowledge gaps regarding adverse health outcomes related to air pollution, and there is a real need for a comprehensive literature review on available meta-analyses. This is the first step towards the estimation of the actual cost of air pollution on adverse health outcomes.

2.1 Selection of reference level

During the last decade, it became clear that the health effects are observed under the air pollution standards (Brunekreef et al., 2015). In many studies, the calculation of the attributable cases is done by comparison of the exposure levels (that are calculated from dispersion model or ambient levels) to a reference level. For example, in recent estimates of the attributable burden and economic costs of Particulate Matter exposure and preterm birth in the U.S., a reference level of 8.8 µg/m³ was selected, and sensitivity analyses of a 5.8 µg/m³ threshold in (PM2.5) was used (Trasande et al., 2016). These were also the reference levels that applied to other health effects of PM2.5 in the 2010 Global Burden of Disease estimates of PM2.5-attributable disease burden (Lim et al., 2012). In a risk analysis that was published recently, the researchers evaluated the risk of adverse health outcomes from air pollution levels based on emission models. The reference level corresponded to a yearly PM2.5 average of 10 µg/m³ as per the current WHO’s air quality guidelines (Morelli et, al. 2016). This
variation in reference levels demonstrates that there is no conventional level agreed upon as a reference level to be used.

In the current study, we used a different approach. Our approach was to evaluate only the contribution of the local anthropogenic emissions to the burden of disease. Therefore, the “reference level” in the current study was set to zero.

### 2.2 Population Attributable Fractions

The Population Attributable Fraction or the population attributable risk fraction (PAF) indicates the percentage reduction, if the exposures were eliminated altogether, in each population in the incidence ratio (IR) of a disease.

Three categories of cases occur among the exposed population over a certain period of time: A0, cases which would have happened regardless, even in the absence of exposure—these would typically be estimated from the number of cases occurring in an unexposed control population; A1, cases that would have occurred anyway but were accelerated by exposure; and A2, cases which would not have occurred, ever, without exposure. A1 is also called the ‘accelerated,’ but can also be called ‘premature.’ What we usually call the attributable fraction among the exposed is equivalent to the attributable risk (RR-1)/RR which in Greenland’s paper is denoted as the etiologic fraction, (A1 + A2)/(A0 + A1 +A2). Moreover, etiologic cases are A1 +A2, and excess cases are A2 (Greenland and Robins 1988). It is almost impossible to isolate excess cases from the etiological cases. The distinction between excess cases and accelerated cases only makes sense for morbidity endpoints or cause-specific mortality and not for mortality in general. The limitations of calculating and using the numbers of premature deaths make the calculation of years of life lost a preferable approach (Leksell and Rabl, 2001).

### 3. Methodology

**Framework of the study**

To evaluate the attributable economic burden of disease (BD) from local anthropogenic activity in HBA, we followed a quantitative health risk assessment methodology (Figure 3.1)
(WHO, 2016a). The methodology aimed to calculate PAF at each level of exposure by combining air pollution level modeling (based on emissions and dispersion), the share of the population at risk, specific IR of the health outcome, and exposure-risk estimates at each level of exposure. We did not include latency periods (Wang et al. 2016). In more detail, the study framework includes 10 steps, as follows:

1) The reference level was set to zero.

2) The addition of the air pollutants based on numerical estimations of concentrations obtained from numerical dispersion model. Specifically, AERMOD was used for all pollutants, except ozone, and the spatial resolution of the results is 200m*200m. CHIMERE was used for ozone, at a resolution of 3km*3km.

3) Mean levels of the air pollutant for every census tract were calculated in a specific time unit (i.e. daily or annual).

4) The “exposure difference” was estimated as the difference between values derived in stages 3 and 1.

5) The exposure response functions (ERFs) or Concentration response function (CRFs) - quantifying the association between exposure and mortality or specific morbidity) were based on the literature review.

6) The relative risks (RR) for exposure difference were calculated for every census tract.

7) The population attributable fraction (PAF) for each exposure difference from the RRs was calculated.

8) Calculation of incidence cases within every census tract was based on the specific mortality rate in the population of the national health or mortality outcome and the size of the population at risk.

9) Calculation of the estimated number of excess cases (contributed cases) within each census tract was based on the specific PAF multiplied by the specific expected morbidity/mortality rates within every census tract.

10) Quantification of the economic value was done separately for mortality and morbidity, by applying the COI method for the different morbidity effects and estimation of the VSL and VOLY for mortality.
During the last decade, it has become clear that health effects are observed under the air standards (Cohen et al. 2017). The calculations of the PAFs were done by comparing CURRENT exposure levels (that are calculated from dispersion model) to a DESIRED reference level. The process of selecting the reference levels is therefore one of the most important issues in risk assessment and burden analysis. The reference level that was chosen for the study was chosen according to the contribution of local emissions. The reference level was set as zero to evaluate the contribution of local emissions to the burden of disease, without taking into consideration the natural sources and anthropogenic air pollutants transported over long distances.

3.1 Stage 1: Counterfactual Level

Figure 3.1: Flow chart of research stages

1. Counterfactual Exposure- set to zero
2. Emission model -> outcome: pollutant levels, scale for AERMOD -200m*200m and for CHIMERE - 3km*3km.
3. Current Exposure- from 1, the mean in every small census tract was calculated.
4. Exposure Difference (3 - 1 = 4)
5. Dose-response/Concentration Response Functions (CRF)/concentration–response coefficient
6. RR for exposure difference for every census tract
7. Calculation of PAF for every census tract
8. Calculation of expected morbidity = national incidence ratio*population at risk within census tract
9. Calculation of estimated access number of cases within the census tract = PAF * expected morbidity
10. Quantify the economic value
3.2 Stage 2: Emission and Dispersal Models

Exposure to air pollution was estimated for the following pollutants: NO2, PM2.5, benzene and O3. Exposure was estimated based on the emission from various polluting sources in the Haifa bay area. These sources include industry, energy generation, and transportation.

The Ministry of Environmental Protection carries out daily air quality predictions throughout the country by running an elaborate photochemical dispersion model called CHIMERE. The CHIMERE model has been in operation for several years. The spatial resolution of the model is approximately 3X3-squared km.

Since we needed a different resolution for our study, we used a dispersion model that can reach finer spatial resolution - the AERMOD model, which was commissioned and approved by the USEPA - was chosen. This model is also approved by the Israeli Ministry of Environmental Protection (IMEP) and serves as a leading evaluation tool for estimating pollution dispersion from industry and transportation. AERMOD is used in various studies concerning exposure of population to air pollution. Dionisio et al. (2013), EPA (2014), and Michanowicz et al (2016) are three recent examples of such studies.

It should be noted that AERMOD is not a photochemical model, and therefore cannot be employed to compute ozone concentrations and was thus used only for the primary pollutants NO2, PM2.5 and Benzene. Ozone (a secondary pollutant) concentrations were obtained from the CHIMERE model results, albeit their coarse spatial resolution.

Below is a description of the data input for the AERMOD model runs. The data input is comprised of:

a. Emissions data

The primary pollutants are generated by the industrial and energy sectors, as well as by transportation. These are mainly generated by the combustion of fossil fuels. The data on input emission sources for these models was the same as those used by CHIMERE for the Haifa bay area.

i. **Emissions inventory for the industrial and energy sectors** in the Haifa Bay region for 2015 (see Appendix A). The inventory contains measured emission rates and geographic layout of the major pollution sources in the Bay area (approximately 290).
ii. Traffic data for the region was provided by the IMEP. This data includes the spatial layout of the roads system, and the emissions inventory for traffic on this system. The transportation sector causes high rates of NO$_2$, PM2.5 and Benzene emissions. It should be noted that the roads system represents data from 2008. Consequently, it does not include two major traffic projects that were constructed after 2008, that had an impact on traffic in Haifa. These projects are the Carmel Tunnel and Highway 22 ("Krayot Bypass"). The new projects significantly changed traffic patterns and emissions. However, traffic amounts and characteristics were updated to those of 2015. The method is described in the MOE report "A methodology Document – The National Plan to Reduce and Abate Air Pollution", 2012 (in Hebrew).

b. Topographic data of the region, taken from NASA’s STRDM3 topographical database. The spatial resolution of this data is approximately 30 meters. (http://www.src.com/datasets/SRTM_Info_Page.html)

c. A spatial grid of 200X200 meters, covering the geographical region of the Haifa Townships Association for Environmental Quality.

d. Meteorological database, which represents the region and is based on hourly measurements at the IGUD monitoring/meteorological station, in Haifa Bay next to BAZAN Refineries, at an altitude of 27 meters above sea level. The database contains hourly data on wind speed, wind direction, temperature, cloud coverage and radiation (radiation data was taken from the nearby Technion meteorological station). Figs 3.2 and 3.3 present the annual wind rose and stability rose, respectively, based on this meteorological database. The roses indicate that wind direction frequencies in the West-north-West and South-East sectors are the highest. At the same time, wind direction frequencies perpendicular to the dominant directions are very low. This is consistent with the topographic channeling effect of the Jezreel/Kishon valley, which is aligned with the west-north and east-south directions.
3.3 Stage 3: pollutants levels attributable to emission in each census tract

The study area is around 24 km long and 22 km wide and covers an area of about 262 km² (see Figure 3.4, which includes the city of Haifa, the Haifa Bay area (mostly industrial and commercial area) and the Krayot region which is a cluster of small satellite towns north of Haifa).
Two main sources were used for pollution level analyses in the Haifa Bay area. The first source was a GIS layer National Layer of Statistical Areas – Census 2008 which divides the study area to separate statistical areas (Statistics 2017). A statistical area is usually one big neighborhood, or a cluster of several neighborhoods, given that they are small in terms of size and/or population. The studied area includes 179 statistical areas (see figure 3.4 above).

The second source was the pollutant spatially dispersed concentrations, as described in the previous stage of research.

The spatial analysis of the pollutant’s dispersion was performed using ArcGIS software (version 10.5.1). The pollutants concentrations? were arranged into separate excel tables. Each table contained data on the average pollutant measured in different areas of the study
area. The table was uploaded into the software and a point-shaped layer was created using the X and Y points of each measuring point (see Figure 3.5). In order to also discover the spatial data on pollutant levels between measured points (i.e., in areas that were not directly measured), we first converted the point shapefile into a raster file. Then, we used the IDW interpolation tool, which interpolates a surface from points using an inverse distance weighted technique (Watson and Philip, 1985) (see Figure 3.5). Once we had the interpolation layer and the statistical area layer together, we could combine data from both layers, based on geographic location. By doing so, each statistical area got the average pollution calculation.

![Figure 3.5: An example of pollution distribution in the bay area](image)

3.4 Stage 4: Exposure difference

The exposure difference is the concentration difference between the level calculated in stage 3 and the reference level reported in stage 1 (the x–x₀ or ΔX).
3.5: Stage 5: Literature review of CRF

An important component of calculating health outcomes attributable to air pollution is the use of concentration–response functions (or dose response function) for key pollutants and inclusion of all known adverse health outcomes. The European Commission published a comprehensive literature review on recommended concentration–response functions and background information for several mortality and morbidity effects associated with short- and long-term exposure to particulate matter (PM), ozone (O3) and nitrogen dioxide (NO2) (WHO 2013). To calculate the number of “cases” per year that can be attributed to air pollution, we used risk estimates based on cohort studies, when possible, as recommended previously for mortality and morbidity (WHO 2013; Héroux et al. 2015). The CRF from the literature was reported as effect estimates [(Relative risks (RRs), Odds Ratios (ORs) and Hazard Ratios (HR)). OR could be estimated as a proxy for RR due to the low incidence rates of the outcomes in the population (rare outcomes). It is important to note that when the only available CRF for exposure-outcome pair was for acute effects (of short-term exposure), it does result in underestimation of the overall effect of the health outcome (World Health Organization 2013).

3.6 Stage 6: RRs for exposure difference

The estimated RRs of exposure differences were based on the exposure difference that was calculated in stage 4 (Trasande et al. 2016) (see example of calculation below, in table 3.2). The RRs for exposure difference were calculated per census tract.

3.7 Stage 7: Population Attributable Fraction-PAFs for Each Census Tract

We calculated the population attributable fraction (PAF) for each ‘exposure difference’ using the following expression:

$$\text{PAF} = \frac{\sum_{i=1}^{n} P_i \times RR_i - \sum_{i=1}^{n} P'_i \times RR'_i}{\sum_{i=1}^{n} P_i \times RR_i}$$

Where:
\[ P_i = \text{proportion of population at exposure level } i, \text{ current exposure} \]
\[ P'_i = \text{proportion of population at exposure level } i, \text{ recommended level of exposure} \]
\[ R_{Ri} = \text{the relative risk at exposure level } i, \text{ current exposure} \]
\[ R_{R'i} = \text{the relative risk at the target concentration} \]
\[ n = \text{the number of exposure levels} \]

For each census tract, we calculated:
\[ P_i = 1 \] (the analysis is done for the exposed population (i.e. census tracts) only, if the entire population in the census tract is exposed to the same mean level).
\[ R_{R'i} = \text{the relative risk for the exposure difference, calculated for each census tract.} \]
\[ R_{R'i} = \text{the relative risk at the target concentration=1} \]

Therefore:
\[ P_{AFi} = \frac{(R_{Ri} - 1)}{R_{R'i}} \]

An example of the calculation for a representative tract is given in table 3.1 below. This sums up stages 3 – 7.

**Table 3.1: Example - Calculation of the exposure difference and the RR for the exposure difference for census tract 1120001 in Haifa. (stages 3-7)**

<table>
<thead>
<tr>
<th># Census tract</th>
<th>Annual mean attributable to local emissions of PM$_{2.5}$ $\mu g/m^3$</th>
<th>Relative Risk (R$_{R'i}$) in the census tract</th>
<th>Population attributable fraction (P$_{AFi}$) in the census tract</th>
</tr>
</thead>
<tbody>
<tr>
<td>1120001</td>
<td>1.27 $\mu g/m^3$</td>
<td>1.00267</td>
<td>0.00266</td>
</tr>
</tbody>
</table>

Air pollution and Ischemic Heart Disease hospitalization

Census tract: 1120001

Exposure: PM2.5

Exposure response function (ERF): RR= 1.0212 per 10 $\mu g/m3$ PM2.5 (Shah et al. 2013)

Concentration in the census tract $=1.27 \mu g/m3$
RR exposure difference = $RR_i = \exp(\beta \Delta C)$, where $\beta = \ln(1.0212)/10$

$RR_i = \exp (((\ln(1.0212))/10)*1.27) = 1.00267$

$PAFi= (1.00267-1)/1.00267=0.00266$

3.8 Stage 8: Calculation of Morbidity Cases Within Each Census Tract

The number of morbidity cases or deaths within each census tract was calculated by multiplying the national incidence rates by the population at risk in each census tract. We obtained incidence rates specific for sex and age group from the literature, where available.

In addition, for non-accidental deaths and hospitalizations for selected morbidities, data obtained from the databases of the Israeli Ministry of Health (Ginsberg, Kaliner, and Grotto 2016). Data on other health outcomes was taken from different sources as described in the results section, in Table 4.4 (Table of incidence of disease).

Example: Calculation of the Estimated Total Number of Ischemic Heart Disease Cases Within Census Tract 1120001 in Haifa (stage 8)

A. calculation of the annual national incidence ratio (IR)

For ischemic heart disease (IHD), we first calculated the total numbers of national incidence cases (hospitalization) within five years (2009-2013) in the relevant sex and age group (both sexes age >20) ($N= 492,953$).

We calculated the mean annual incidence ratio (IR) for IHD (both sexes age >20). We divided the annual mean IHD cases ($N=98,590.6$) by the population size (the total size of the population, both sexes age >20, $N= 4,979,300$).

The mean annual incidence ratio (IR) for IHD (both sexes age >20): 1980 per 100,000 persons

B. calculation of the annual number of cases within census tract

The calculation is described in table 3.2 below.

<table>
<thead>
<tr>
<th>#</th>
<th>Population</th>
<th>Expected annual Incidence ratio (IR) in</th>
<th>Total new IHD annual cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3.2: Example: Calculation of the Estimated Total Number of Cases Within Census Tract 1120001 in Haifa (stage 8)
To calculate the specific census tract IHD cases, we multiplied the national IR of IHD hospitalization in people aged ≥20 years in Israel (1980 per 100000) by the total size of the population at risk in the census tract. Thus, for census tract # 1120001 there was a total of 11.68 new annual cases of IHD (590 * (1980/100000)).

**Stage 9: Calculation of the Estimated Access Number of Cases Within the Census Tract.**

This was calculated based on multiplying the PAF for each census tract by the incidence cases within that census tract. An example is given in table 3.3 below.

**Table 3.3: Example: Calculation of the Estimated Number of Cases Attributable to Excess PM$_{2.5}$ Exposure for Census Tract 1120001 in Haifa (stage 9)**

<table>
<thead>
<tr>
<th># Census tract</th>
<th>Estimated number of cases attributable to PM$_{2.5}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1120001</td>
<td>0.00266* 11.68 = 0.031</td>
</tr>
</tbody>
</table>

Thus, the estimated number of cases attributable to excess PM2.5 exposure for census tract 1120001 in Haifa is found by multiplying PAFi * expected morbidity incidence rate in census tract # 1120001(11.68). There were 31 cases per 1000 population ≥20 years of IHD estimated to be attributable to the excess PM$_{2.5}$ annual mean level of 1.27 μg/m$^3$.

**3.10 Stage 10: Measuring the Economic Value of Morbidity and Mortality.**

Since a symptom can occur only once but may be the result of more than one pollutant, a problem of double counting may exist. To challenge this problem, we report a range of two estimates. The first one is the conservative one in which only the highest pollution cost out
of the four pollutants is considered. The second estimate is more liberal and assumes that the costs are fully addable.

Table 3.4 below presents the costs associated with the different morbidity symptoms used in this study, based on the literature. (see research study titles in the table).

<table>
<thead>
<tr>
<th>Category/Disease</th>
<th>Research Study title</th>
<th>Type of Direct Cost</th>
<th>Age Groups</th>
<th>Differentiation by gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular Diseases</td>
<td>The Shifting Burden of Cardiovascular Disease in Australia</td>
<td>Hospitalization, nursing homes, professional services, medication, research</td>
<td>Costs by age group (0-4, 5-14, 15-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+) for all cardiovascular diseases (CVD), and by stroke, ischemic heart disease (IHD), and peripheral vascular disease (PVD)</td>
<td>Yes</td>
</tr>
<tr>
<td>Stroke</td>
<td>Cost of Stroke In Ireland</td>
<td>Hospitalization, ambulatory care, professional services, medication and assistive devices, etc.</td>
<td>Costs by age group (0-14, 15-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+) only for some types of costs</td>
<td>Yes</td>
</tr>
<tr>
<td>Type 2 Diabetes mellitus</td>
<td>Economic Costs of Diabetes in The U.S. In 2012</td>
<td>Hospitalizations, professional services, medication and assistive devices</td>
<td>Cost by age group (&lt;45, 45-64, 64+)</td>
<td>No</td>
</tr>
<tr>
<td>Lung Cancer</td>
<td>Cost of Cancer In NSW</td>
<td>Health system costs.</td>
<td>Cost by age groups (&lt; 15, 15-64, 64+)</td>
<td>Yes</td>
</tr>
<tr>
<td>COPD</td>
<td>Economic Case Statement: Chronic Obstructive Pulmonary Disease</td>
<td>Hospital care, outpatient care, medication and miscellaneous</td>
<td>Graphic representation of costs by 5-year cohorts and 90+, and only for some of the costs</td>
<td>Yes, but only graphical representation for some costs</td>
</tr>
<tr>
<td>Asthma</td>
<td>The Hidden Cost of Asthma</td>
<td>Hospitalization, specialists, diagnosis, screening, medications</td>
<td>Graphic representation of costs by 5-year cohorts and 90+, for costs of the care system and all costs including Biochemical Oxygen Demand (BOD)</td>
<td>Yes</td>
</tr>
</tbody>
</table>

As presented, these costs are affected by gender, age and type of treatment given. Costs are usually given in present value terms for the entire duration of the symptom. Therefore, the
estimated cost was based on *incremental cases per year*. This is true to all symptoms besides asthma, where no information was found about new cases, but only on cases in general. Thus, the costs were amortized to annual value instead of present value.

**Value of Mortality:** To estimate the VSL we used a study by Roy and Braathen (2018), which estimated these values for the OECD countries based on benefit transfer. Its results are given in table 3.5 below.

<table>
<thead>
<tr>
<th>Country</th>
<th>VSL</th>
<th>Country</th>
<th>VSL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brazil</td>
<td>1.885</td>
<td>Italy</td>
<td>3.428</td>
</tr>
<tr>
<td>China</td>
<td>1.36</td>
<td>Japan</td>
<td>3.428</td>
</tr>
<tr>
<td>India</td>
<td>0.737</td>
<td>Korea</td>
<td>3.53</td>
</tr>
<tr>
<td>Indonesia</td>
<td>1.226</td>
<td>Latvia</td>
<td>2.406</td>
</tr>
<tr>
<td>Russia</td>
<td>3.269</td>
<td>Luxembourg</td>
<td>7.5</td>
</tr>
<tr>
<td>South Africa</td>
<td>1.501</td>
<td>Mexico</td>
<td>2.022</td>
</tr>
<tr>
<td>Australia</td>
<td>4.452</td>
<td>Netherlands</td>
<td>4.429</td>
</tr>
<tr>
<td>Austria</td>
<td>4.236</td>
<td>New Zealand</td>
<td>3.434</td>
</tr>
<tr>
<td>Belgium</td>
<td>3.96</td>
<td>Norway</td>
<td>5.496</td>
</tr>
<tr>
<td>Canada</td>
<td>4.138</td>
<td>Poland</td>
<td>2.622</td>
</tr>
<tr>
<td>Chile</td>
<td>2.516</td>
<td>Portugal</td>
<td>2.798</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>3.032</td>
<td>Slovakia</td>
<td>2.932</td>
</tr>
<tr>
<td>Denmark</td>
<td>4.161</td>
<td>Slovenia</td>
<td>2.919</td>
</tr>
<tr>
<td>Estonia</td>
<td>2.783</td>
<td>Spain</td>
<td>3.282</td>
</tr>
<tr>
<td>Finland</td>
<td>3.783</td>
<td>Sweden</td>
<td>4.158</td>
</tr>
<tr>
<td>France</td>
<td>3.725</td>
<td>Switzerland</td>
<td>4.528</td>
</tr>
<tr>
<td>Germany</td>
<td>4.141</td>
<td>Turkey</td>
<td>2.634</td>
</tr>
<tr>
<td>Greece</td>
<td>2.447</td>
<td>UK</td>
<td>3.923</td>
</tr>
<tr>
<td>Hungary</td>
<td>2.695</td>
<td>U.S</td>
<td>4.88</td>
</tr>
<tr>
<td>Ireland</td>
<td>5.137</td>
<td>Iceland</td>
<td>4.417</td>
</tr>
</tbody>
</table>
As shown, the VSL in Israel was estimated at 3.301 million dollars.

The value of the loss of one year was obtained from the equating between the annual value discounted over 40 years and the present value presented as the value of statistical life. That is: \[ \sum_{i=1}^{T} = \frac{\text{VOLY}}{(1+r)^T} = \text{VSL}. \]

Since the effective exchange rate (in PPP form) was 3.924 at 2015, the VSL was estimated at 12.95 and VOLY at 0.353 Million ILS.

**Value of Morbidity:** The following tables describe the costs per symptom based on our literature review. The benefit transfer mode will follow the tables. Results per case will be presented in the next section.

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liou et al.</td>
<td>Taiwan</td>
<td>2017</td>
<td>$1,423</td>
<td></td>
<td>$1,423</td>
</tr>
<tr>
<td>EPA</td>
<td>USA</td>
<td>2020</td>
<td>$1,150</td>
<td></td>
<td>$1,150</td>
</tr>
</tbody>
</table>

**Table 3.6: Cost of Acute lower respiratory infection (ALRI)**

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accordini et al.</td>
<td>EU</td>
<td>2010</td>
<td>€588</td>
<td>€995</td>
<td>€1,583</td>
</tr>
<tr>
<td>Smith et al.</td>
<td>USA</td>
<td>1997</td>
<td>$2,272</td>
<td>$312</td>
<td>$2,584</td>
</tr>
<tr>
<td>Gibson et</td>
<td>EU</td>
<td>2013</td>
<td>€1,950</td>
<td>€1,450</td>
<td>€3,400</td>
</tr>
</tbody>
</table>

\(^9\) We used the suggested discount factor for Israeli programs which is 3% (Barak, 2016).
<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mahmoud et al.</td>
<td>UK</td>
<td>2011</td>
<td>n.a.</td>
<td>n.a.</td>
<td>£52,110</td>
</tr>
<tr>
<td>Mahmoud et al.</td>
<td>USA</td>
<td>2011</td>
<td>n.a.</td>
<td>n.a.</td>
<td>$251,979</td>
</tr>
<tr>
<td>Blankart et al.</td>
<td>Germany</td>
<td>2008</td>
<td>€9,753</td>
<td>€13,939</td>
<td>€23,692</td>
</tr>
<tr>
<td>Chen et al.</td>
<td>USA</td>
<td>2015</td>
<td>n.a.</td>
<td>n.a.</td>
<td>$147,000</td>
</tr>
</tbody>
</table>

Table 3.8: Cost of Blood cancer\(^{10}\)

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foster et al.</td>
<td>USA</td>
<td>2005</td>
<td>$3,568</td>
<td>$2,682</td>
<td>$6,350</td>
</tr>
<tr>
<td>Menzin et al.</td>
<td>USA</td>
<td>2008</td>
<td>n.a.</td>
<td>n.a.</td>
<td>$6,300</td>
</tr>
<tr>
<td>Ramsey and Sullivan</td>
<td>USA</td>
<td>2003</td>
<td>n.a.</td>
<td>n.a.</td>
<td>€3,668</td>
</tr>
<tr>
<td>Lodenkemper et al.</td>
<td>UK</td>
<td>2003</td>
<td>€2,754</td>
<td>€820</td>
<td>€3,574</td>
</tr>
<tr>
<td>Mannino et al.</td>
<td>USA</td>
<td>2013</td>
<td>n.a.</td>
<td>n.a.</td>
<td>$21,753 (present value)</td>
</tr>
<tr>
<td>Gibson et al.</td>
<td>EU</td>
<td>2013</td>
<td>€1,013</td>
<td>€1,091</td>
<td>€2,104</td>
</tr>
<tr>
<td>EPA</td>
<td>USA</td>
<td>2020</td>
<td></td>
<td></td>
<td>$15,903</td>
</tr>
</tbody>
</table>

Table 3.9: Chronic obstructive pulmonary disease (COPD)

\(^{10}\) Survival rate is 62.7% ([https://seer.cancer.gov/statfacts/html/leuks.html](https://seer.cancer.gov/statfacts/html/leuks.html)) so we attribute 37.3% of the VSL to the COI (225K ILS).
Table 3.10: Cost of Diabetes

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADS</td>
<td>USA</td>
<td>2017</td>
<td>$7,577</td>
<td>$2,023</td>
<td>$9,600</td>
</tr>
<tr>
<td>Fernandes and Fernandes</td>
<td>India</td>
<td>2017</td>
<td>INR 8,250</td>
<td>INR 4,185</td>
<td>INC 12,435</td>
</tr>
<tr>
<td>Dall et al.</td>
<td>USA</td>
<td>2007</td>
<td>7,001</td>
<td>2,974</td>
<td>$9,975</td>
</tr>
</tbody>
</table>

Table 3.11: Cost of Ischemic heart disease (IHD)

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADS</td>
<td>USA</td>
<td>2017</td>
<td>$7,577</td>
<td>2,023</td>
<td>$9,600</td>
</tr>
<tr>
<td>EPA</td>
<td>USA</td>
<td>2020</td>
<td>n.a.</td>
<td>n.a.</td>
<td>$33,357</td>
</tr>
</tbody>
</table>

Table 3.12: Lung cancer

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Im et al.</td>
<td>EU</td>
<td>2013</td>
<td>n.a.</td>
<td>n.a.</td>
<td>€16,022</td>
</tr>
<tr>
<td>Kutikova et al.</td>
<td>USA</td>
<td>2005</td>
<td>n.a.</td>
<td>n.a.</td>
<td>$42,990</td>
</tr>
<tr>
<td>Gibson et al.</td>
<td>EU</td>
<td>2013</td>
<td>€11,473</td>
<td>n.a.</td>
<td>€11,473</td>
</tr>
</tbody>
</table>

Table 3.13: Cost of Stroke

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Year</th>
<th>Direct cost</th>
<th>Indirect cost</th>
<th>Total cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cha</td>
<td>South Korea</td>
<td>2015</td>
<td>$3,169</td>
<td>$4,078</td>
<td>$7,247</td>
</tr>
<tr>
<td>Rajsic et al.</td>
<td>Meta-analysis</td>
<td>2015</td>
<td>$2,745</td>
<td>n.a.</td>
<td>$2,745</td>
</tr>
<tr>
<td>Xu et al.</td>
<td>UK</td>
<td>2016</td>
<td>£13,452</td>
<td>£8,977</td>
<td>£22,429</td>
</tr>
<tr>
<td>Patel et al.</td>
<td>UK</td>
<td>2016</td>
<td>£12,869</td>
<td>£11,134</td>
<td>£24,003</td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Year</td>
<td>Direct cost</td>
<td>Indirect cost</td>
<td>Total cost</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------</td>
<td>------</td>
<td>-------------</td>
<td>---------------</td>
<td>------------</td>
</tr>
<tr>
<td>Almosn et al</td>
<td>USA</td>
<td>2004</td>
<td></td>
<td></td>
<td>$90,000</td>
</tr>
<tr>
<td>Cavallo et al.</td>
<td>Italy</td>
<td>2015</td>
<td>€20,502</td>
<td>€3,707</td>
<td>€24,209</td>
</tr>
<tr>
<td>Tongo et al.</td>
<td>USA</td>
<td>2008</td>
<td>$37,939</td>
<td>$13,350</td>
<td>$51,289</td>
</tr>
</tbody>
</table>

Table 3.14: Term Low birth weight (LBW)

Adjusting different monetary values to Israel:

To make an adjustment in a value, two elements should be considered:

*Adjustment to changes in prices and currency rates:* All values were adjusted to inflation and exchange rate between years.

Israel's GDP per capita in terms of purchasing power in 2015 divided by the purchasing power parity of the country examined in 2015.

The adjustment coefficient (AC) looks like so:

\[
AC = \frac{\text{index}}{\text{index}_{\text{year}}} \times \text{EX}_{\text{currency}} \times \left(\frac{\text{GDP PC PPP Israel}}{\text{GDP PC PPP}_c}\right)^\beta
\]

Where:

\(\text{Index}_{\text{year}}\) = Price index of country C in year \(\text{year}\).

\(\text{EX}_{\text{currency}}\) = exchange rate between currency and ILS.

\(\text{GDP PC PPP Israel}\) = GDP per capita in terms of purchasing power in Israel.

\(\text{GDP PC PPP}_c\) = GDP per capita in terms of purchasing power of a country \(c\).

\(\beta\) = Elasticity of income\(^{11}\).

\(^{11}\) Since COI is not a welfare measure, a value of 1 was used.
Table 3.15 below presents a sample of adjustment coefficient for different studies from 4.9 different countries in different years.

### Table 3.15: Sample of Adjustment Coefficients for Israel for Symptom Costs in Various Countries in Previous Years

<table>
<thead>
<tr>
<th>Disease</th>
<th>Country</th>
<th>Currency</th>
<th>Year</th>
<th>Index - end of year index examined</th>
<th>Exchange rate at the end of 2017</th>
<th>GDP capita measured in terms of purchasing power, 2017</th>
<th>Adjustment Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td>IHA</td>
<td>Australia</td>
<td>AUD</td>
<td>2004</td>
<td>79.6</td>
<td>112</td>
<td>3.47</td>
<td>50,334</td>
</tr>
<tr>
<td>Stroke</td>
<td>Ireland</td>
<td>euro</td>
<td>2007</td>
<td>94.9</td>
<td>99.6</td>
<td>4.15</td>
<td>75,538</td>
</tr>
<tr>
<td>Stroke</td>
<td>Australia</td>
<td>AUD</td>
<td>2007</td>
<td>86.7</td>
<td>112</td>
<td>4.15</td>
<td>50,334</td>
</tr>
<tr>
<td>Stroke</td>
<td>France</td>
<td>euro</td>
<td>2007</td>
<td>88.62</td>
<td>101.95</td>
<td>4.15</td>
<td>43,761</td>
</tr>
<tr>
<td>Stroke</td>
<td>Germany</td>
<td>euro</td>
<td>2007</td>
<td>88.1</td>
<td>103</td>
<td>4.15</td>
<td>50,425</td>
</tr>
<tr>
<td>Diabetes</td>
<td>US</td>
<td>USD</td>
<td>2012</td>
<td>227.7</td>
<td>248.19</td>
<td>3.47</td>
<td>59,501</td>
</tr>
<tr>
<td>Lung Cancer</td>
<td>Australia</td>
<td>AUD</td>
<td>2005</td>
<td>81.7</td>
<td>112</td>
<td>3.47</td>
<td>50,334</td>
</tr>
<tr>
<td>COPD</td>
<td>Australia</td>
<td>AUD</td>
<td>2008</td>
<td>89.3</td>
<td>112</td>
<td>3.47</td>
<td>50,334</td>
</tr>
<tr>
<td>Asthma</td>
<td>Australia</td>
<td>AUD</td>
<td>2014</td>
<td>106.5</td>
<td>112</td>
<td>3.47</td>
<td>50,334</td>
</tr>
</tbody>
</table>

### 4. Results

#### 4.1 Stage 1: Counterfactual Level

We calculated the economic valuation of the PAF for morbidity and mortality in the area caused only by the total local emissions of air pollutants. Therefore, the counterfactual levels were set to zero.

#### 4.2 Stage 2: Emission and Dispersal Models

---

The dispersion models produced hourly concentration values for all hours of 2015 and all grid receptors for the pollutants considered. These results are used as input for the next stages of the research. Since the present study focuses on economical and epidemiological outcomes of long-range exposure to air pollution, the results presented here are annual averages of the computed concentrations. The following is a short description of the results for each of the pollutants considered.

**NO2**

**General**

This pollutant is generated in all sectors - industry, energy and transportation, mainly by fuel combustion. In the past, NO and NO2 were treated as one pollutant – NOX. In recent years, following updated epidemiological findings, the focus has been on NO2 instead of NOX. Fig. 4.1 presents annual NO2 concentrations for 2015: all sources, and by sectors.

*Figure 4.1(a)- Annual attributable NO2 concentrations (µg/m³) for 2015 - Haifa region.*
Comparison with measured data and a similar study

Although it was not within the scope of the present research, the computed annual NO$_2$ concentration for the receptor representing the IGUD monitoring/meteorological station in Haifa Bay area was compared to:

- The annual NO$_2$ value measured at this station
- The average value for the statistical region that includes this station (produced by CHIMERE with measurements).

The following table presents the comparison:

<table>
<thead>
<tr>
<th>Value Source</th>
<th>NO$_2$ Concentration ($\mu$g/m$^3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1  Present study</td>
<td>14.5</td>
</tr>
<tr>
<td>2  Measured value</td>
<td>14.7</td>
</tr>
<tr>
<td>3  Average value from CHIMERE with measurements</td>
<td>16.8</td>
</tr>
</tbody>
</table>
The comparison reveals a reassuring picture of similar estimates across sources. However, it does not indicate that such differences are evident in other places in the Haifa region.

**Benzene**

This pollutant is generated in all sectors - industry, energy and transportation. However, as noted earlier, the 2015 emissions inventory for the industrial and energy sectors did not include Benzene data. Consequently, the contribution of these sectors to Benzene concentrations was not computed. It should also be noted that the updated 2018 emissions inventory does include Benzene data as well. However, data on benzene concentrations from transportation emissions was available for 2015, and the contribution of this sector was computed. Fig. 4.2 presents annual benzene concentrations from transportation for 2015.

**Fig 4.2- Annual Attributable Benzene concentrations (µg/m³) in 2015 – HB originated by the transportation**

The pattern of the concentration isopleths for the transportation sector is typical to those generated by ground level fugitive sources).
PM2.5

This pollutant is different from the others presented in this study because Israel is part of the global desert dust belt that spans through North Africa and west Asia. A considerable fraction of the dust in this region is PM2.5. Fig 4.3 presents global Satellite Derived Annual PM2.5 concentrations (NASA, 2017). Israel is located in the northern region of this belt, and it is estimated that annual natural (non-anthropogenic) PM2.5 concentrations in our region are above 20 µg/m³.

![Figure 4.3: Global annual PM2.5 concentration Source: NASA (2017)](image)

Therefore, PM2.5 concentrations in Israel are a superposition of natural background dust and the anthropogenic contributions. In this study, only attributable anthropogenic PM2.5 is calculated.

Anthropogenic PM2.5 is generated by all sectors - industrial, energy, buildings and transportation. Fig. 4.4 presents annual anthropogenic PM2.5 concentrations for 2015: all sources, and by sectors.
Ozone

Unlike the other primary pollutants in the present research, ozone is a secondary pollutant, i.e. – a chemical product of a photochemical reaction between primary pollutants and the
atmosphere. The results presented here are based on CHIMERE hourly concentration for the Haifa region during 2015. This data was provided by IMEP. It should be noted that:

a. The spatial resolution CHIMERE grid is 3x3-squared km. This is due to the geographical resolution limitations of CHIMERE.

b. The computed values are the atmospheric concentration of ozone. It is not possible to isolate the anthropogenic contribution.

Figure 4.5 presents annual ozone concentrations.

![Figure 4.5: Annual attributable O₃ concentrations (PPB) for 2015 - Haifa region](image)

The fact that ozone concentrations in the Haifa Bay area are lower than its concentrations in surrounding areas is probably due to the characteristics of air pollution emissions in this region. In a study by Dayan, Mahrer and Levi (2002), the phenomenon was attributed to the emission regime in Israel, characterized by near 1:1 VOC/NOx ratios, where additional NOx causes an increased titration effect, and reduction in ozone concentrations. Hence, Ozone concentration decreases with emission.

### 4.3 Stage 3 & 4: Levels of pollutant in every census tract and exposure difference
The average level of pollution is summed up and given in figure 4.5.1 below for the four pollutants.

Figure 4.5.1: Mean yearly incremental concentration map for the HBA

(Only from anthropogenic sources)
The area with the highest pollution level of MP 2.5 is in the statistical area in the center of the industrial area of Haifa Bay area. The areas with the highest pollution level of NO2 were found in five statistical areas scattered across the research area:

In the city of Haifa – Vadi salib, Ramot HaCarmel

In the Bay area – Checkpoint, the Bay area, and Kiryat Haim East

The areas with the highest O3 pollution level were all the statistical areas found along the coastlines of Haifa, western Kyriat Haim and Kyriat Yam.

The areas with the highest pollution level of Benzen were Haifa - Wadi Nisnas and Hadar, and in the Bay area – the Checkpoint and the Bay area.

Exposure to annual means of O3, NO2, PM2.5, and Benzene and summertime O3 (April–September mean of daily maximum 8-hour (µg/m$^3$)) was assessed for 521,230 inhabitants in all ages living in all the relevant census tracts. The levels represent the addition to exposure to pollutants contributed by emissions from local sources. Table 4.1 (below) summaries the spatial range, mean and percentiles of the pollutants’ distribution in the area (by census tracts). The median (and IQR) of O3, NO2, PM2.5, and Benzene and summertime O3 levels in 2015 were (82.3(3.3); 12.5(8.3); 1.05(0.7); 0.19(0.1); 100.9(3.1), respectively).

Table 4.1: Descriptive statistics of the difference in exposure for annual mean during 2015 of O3, NO2, PM2.5, and Benzene (and summertime mean for O3), (µg/m$^3$).

<table>
<thead>
<tr>
<th></th>
<th>O3 Annual</th>
<th>O3 Summertime mean of daily maximum 8-hour</th>
<th>NO2 Annual</th>
<th>PM2.5 Annual</th>
<th>Benzene Annual</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>82.2</td>
<td>100.4</td>
<td>13.7</td>
<td>1.23</td>
<td>0.22</td>
</tr>
<tr>
<td>SE</td>
<td>0.2</td>
<td>0.6</td>
<td>0.5</td>
<td>0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>Min.</td>
<td>76.7</td>
<td>0.0</td>
<td>4.1</td>
<td>0.30</td>
<td>0.03</td>
</tr>
<tr>
<td>Max.</td>
<td>88.9</td>
<td>105.5</td>
<td>34.0</td>
<td>4.02</td>
<td>0.67</td>
</tr>
<tr>
<td>Percentiles</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>78.9</td>
<td>98.5</td>
<td>6.6</td>
<td>0.60</td>
<td>0.08</td>
</tr>
<tr>
<td>20</td>
<td>80.2</td>
<td>99.1</td>
<td>7.9</td>
<td>0.76</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>Q1</td>
<td>Median</td>
<td>Q3</td>
<td>80</td>
<td>90</td>
</tr>
<tr>
<td>---</td>
<td>------</td>
<td>--------</td>
<td>------</td>
<td>-----</td>
<td>------</td>
</tr>
<tr>
<td>30</td>
<td>80.8</td>
<td>82.3</td>
<td>83.6</td>
<td>82.5</td>
<td>85.5</td>
</tr>
<tr>
<td>40</td>
<td>81.3</td>
<td>100.4</td>
<td>102.4</td>
<td>101.4</td>
<td>103.7</td>
</tr>
<tr>
<td>Median</td>
<td>82.3</td>
<td>100.9</td>
<td>12.5</td>
<td>1.05</td>
<td>23.6</td>
</tr>
<tr>
<td>60</td>
<td>82.5</td>
<td>101.4</td>
<td>14.2</td>
<td>1.20</td>
<td>2.21</td>
</tr>
<tr>
<td>70</td>
<td>83.2</td>
<td>102.0</td>
<td>15.5</td>
<td>1.34</td>
<td>0.43</td>
</tr>
<tr>
<td>Q3</td>
<td>83.6</td>
<td>102.4</td>
<td>17.2</td>
<td>1.49</td>
<td>0.21</td>
</tr>
<tr>
<td>80</td>
<td>84.2</td>
<td>102.6</td>
<td>18.9</td>
<td>1.60</td>
<td>0.28</td>
</tr>
<tr>
<td>90</td>
<td>85.5</td>
<td>103.7</td>
<td>23.6</td>
<td>2.21</td>
<td>0.43</td>
</tr>
</tbody>
</table>

Q1-25th percentile, Q3-75th percentile, Min.-minimum, Max.-maximum, SE-standard error

The area with the highest level of pollution of MP 2.5 is in statistical area number 214, which is the center of the industrial area of Haifa Bay. With regards to the level of NO2 pollution, the highest level was found in four statistical areas scattered across the research area. The statistical areas were statistical areas 222 (Checkpoint), 314 (Vadi Salib, Haifa), 932 (Ramot Begin, Haifa), and 211 (Old Industrial Area, Kyriat Haim). Concerning the pollution level of O3, the highest pollution was found in all the statistical areas located along the coast. Lastly, the statistical areas with the highest levels of Benzene were 314 (Wadi Nisnas), 231 (Bay Area) and 222 (Checkpoint).

4.5 Stage 5: Literature review of CRF

Morbidity:

To calculate the number of “cases” that can be attributed to air pollution every year, we used risk estimates based on cohort studies, when possible, as recommended previously for mortality and morbidity (World Health Organization 2013; Héroux et al. 2015). When the only available risk estimates for health-exposure pairs are for acute effects of short-term exposure, it is important to note that it can possibly result in underestimation of the overall effect (World Health Organization 2013). For some of the health-exposure pairs presented in table 4.2 there was more than one CRF, therefore a selection between CRF’s was made based on the following criteria: 1) if possible - long-term exposure; 2) the associations must be statistically significant; 3) The most recent studies; 4) the estimates are based on cohort studies; 5) The study population or the exposure levels in the studies were the most similar.
to Israel’s pollution levels (background and incertment). The selected pairs are indicated in bold in table 4.2. All other health-exposure pairs are summarized in appendix B.

Benzene’s main effect on the general population is blood cancer (Collins, 2014). The concentrations of airborne benzene associated with an excess lifetime risk of leukemia is 1 Leukemia incidence per 100,000 for every 1.7 μg/m3. No safe level is reported hence the reference level is zero. Survival levels from leukemia were reported by the US national cancer institute13 as 62.7% within five years of detection, we calculated mortality rates as 37.3% of the annual incidence.

Previous studies on air pollution and health outcomes were conducted in Israel. These were evaluated to use local CRFs; however, we could not identify in any of those studies’ risk estimates for pairs of long-term exposure and morbidity outcomes from the Israeli population with significant associations. These non-significant effect estimates may be used for future sensitivity analysis.

Table 4.2 summary of the CRF that were used and the population at risk based on recent literature (selected pairs are indicated with bold text).

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ICD-9/ICD-10 codes</th>
<th>Exposure, Per unit</th>
<th>Short/long term exposure</th>
<th>Study design</th>
<th>Age group</th>
<th>Sex</th>
<th>Reference for risk estimate</th>
<th>Concentration-response function (CRFs) Risk Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 2 Diabetes mellitus</td>
<td>ICD-10 code: E11 ICD-9: 250</td>
<td>PM2.5, annual Mean, RR (95% CI), per 10 μg/m3</td>
<td>Long term</td>
<td>Meta-analysis</td>
<td>Adults&gt;21</td>
<td>Both</td>
<td>(He et al. 2017)</td>
<td>*after excluding (mortality): RR=1.170 (95% CI: 1.103-1.241)</td>
</tr>
<tr>
<td>Type 2 Diabetes mellitus</td>
<td>ICD-10 code: E11 ICD-9: 250</td>
<td>NO2, (After conversion to the estimate of NOx to its NO2 corresponding value), annual mean, Hazard Ratio (HR, 95% CI), per 10 μg/m3</td>
<td>Long term</td>
<td>Meta-analysis</td>
<td>Adults&gt;21</td>
<td>Both</td>
<td>(Balti et al. 2014)</td>
<td>Hazard Ratio (HR) across cohort studies: HR=1.13 (95% CI: 1.04-1.22)</td>
</tr>
<tr>
<td>Type 2 Diabetes mellitus</td>
<td>ICD-10 code: E11 ICD-9: 250</td>
<td>O3, summer average ozone, daily mean, per 10-μg/m3 increase</td>
<td>Short and long-term, combined</td>
<td>Meta-analysis</td>
<td>Adults&gt;21</td>
<td>Both</td>
<td>(Janghorbani, Momeni, and Mansourian 2014)</td>
<td>OR=1.07 (95% CI:1.05–1.09)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disease/Condition</th>
<th>Exposure</th>
<th>Dose</th>
<th>Duration</th>
<th>Pooling Level</th>
<th>Age Group</th>
<th>Gender</th>
<th>Study References</th>
<th>Effect Size (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Term Low birth weight</td>
<td>PM2.5, during pregnancy</td>
<td>5 μg/m3</td>
<td>Long term</td>
<td>Pooled effect estimate, Europe</td>
<td>20-49</td>
<td>Female</td>
<td>(Pedersen et al. 2013)</td>
<td>OR=1.18 (95% CI:1.06-1.33)</td>
</tr>
<tr>
<td>Term Low birth weight</td>
<td>NO2, during pregnancy</td>
<td>10 μg/m3</td>
<td>Long term</td>
<td>Pooled effect estimate, Europe</td>
<td>20-49</td>
<td>Female</td>
<td>(Pedersen et al. 2013)</td>
<td>OR=1.09 (95% CI:1.00-1.09)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>PM2.5, annual Mean</td>
<td>10 μg/m3</td>
<td>Long term</td>
<td>Meta-analysis</td>
<td>Adults, Age 30+</td>
<td>Both</td>
<td>(Huang et al. 2017)</td>
<td>RR=1.08 (95% CI: 1.03, 1.12)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>NO2, annual Mean</td>
<td>10 μg/m3</td>
<td>Long term</td>
<td>Meta-analysis</td>
<td>Adults, Age 30+</td>
<td>Both</td>
<td>(Kulhánová et al. 2018)</td>
<td>RR=1.04 (95% CI: 1.01, 1.08)</td>
</tr>
<tr>
<td>Stroke</td>
<td>PM2.5, annual Mean</td>
<td>5 μg/m3</td>
<td>Long term</td>
<td>Pooled effect estimate, Europe</td>
<td>Adults, 60+ years</td>
<td>Both</td>
<td>(Stafoggia et al. 2014)</td>
<td>HR=1.40 ( 95% CI: 1.05, 1.87)</td>
</tr>
<tr>
<td>Stroke hospitalization</td>
<td>NO2(per 10ppb)</td>
<td>10 μg/m3</td>
<td>Short term</td>
<td>Meta-analysis</td>
<td>Adults, Age 30+</td>
<td>Both</td>
<td>(Shah et al. 2015)</td>
<td>RR=1.014 (95% CI:1.009-1.019)</td>
</tr>
<tr>
<td>Heart Morbidity</td>
<td>NO2(per 10 ppb)</td>
<td>10 μg/m3</td>
<td>Short term</td>
<td>Meta-analysis</td>
<td>Adults ≥ 20 years</td>
<td>Both</td>
<td>(Shah et al. 2013)</td>
<td>RR=1.017 (1.0125–1.0216)</td>
</tr>
<tr>
<td>COPD Emergency department visit and hospitalization (Data on Mortality also available)</td>
<td>PM2.5 (per 10 ug/m3)</td>
<td>10 μg/m3</td>
<td>Short term</td>
<td>Meta-analysis</td>
<td>Adults, (age not stated, when possible RR were derived from 65+)</td>
<td>Both</td>
<td>(DeVries, Kriebel, and Sama 2017)</td>
<td>RR= 1.025 (95% CI: 1.016–1.034)</td>
</tr>
<tr>
<td>Asthma incidence or lifetime</td>
<td>NO2 (per 10 PPB)</td>
<td>10 μg/m3</td>
<td>Long-term Annual, cumulative</td>
<td>Meta-analysis</td>
<td>Children, age &lt;18</td>
<td>Both</td>
<td>(Khreis et al. 2017)</td>
<td>RR= 1.26 (1.10–1.37)</td>
</tr>
<tr>
<td></td>
<td>PM2.5 (per 10 μg/m3)</td>
<td>10 μg/m3</td>
<td>Long-term Annual, cumulative</td>
<td>Meta-analysis</td>
<td>Children, age &lt;18</td>
<td>Both</td>
<td>(Anenberg et al. 2016)</td>
<td>RR= 1.34 (1.11–1.63)</td>
</tr>
</tbody>
</table>
Mortality:

We used PM2.5 CRF for long term exposure and the all-cause non accidental mortality as a single mortality outcome, as recommended previously (World Health Organization 2013; Héroux et al. 2015). For NO2, a recent meta-analysis summarized that per 10 µg/m3 increment in NO2 there was an increase in all-cause mortality, however the authors concluded that given the many uncertainties and the sensitivity of health impact calculations to small changes in the magnitude of the HRs, calculating the impact of policies to reduce long-term exposure to NO2 on health should use prediction intervals and report ranges of impact rather than focusing on point estimates (Atkinson et al. 2018). In addition, in the WHO HRAPIE report, the short term CRF for NO2, adjusted for PM, was recommended (rather than the long term CRF) (World Health Organization 2013). For O3, a recent meta-analysis on the long-term effects on mortality was published. The authors concluded that the associations with all-cause mortality were not significant (Atkinson et al. 2016), and significant associations were reported only for specific causes of mortality (Cardiovascular: per 10 ppb: 1.01 (95% CI 1.00 to 1.02); Respiratory: 1.03 (95% CI 1.01 to 1.05)) (Atkinson et al. 2016). Therefore, similar to NO2, for O3, we used the short term CRF, previously recommended, and also adjusted for PM, and therefore could be additive to the PM results (World Health Organization 2013; Héroux et al. 2015). A summary of the CRF for mortality effects is given below in table 4.3.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>ICD-9/ICD-10 codes</th>
<th>Exposure</th>
<th>Per unit</th>
<th>Study design</th>
<th>Age group</th>
<th>Sex</th>
<th>Reference for risk estimate</th>
<th>Risk Estimate</th>
<th>Background health data source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma emergency room visits and hospital admissions</td>
<td>J45.901</td>
<td>O3, Per 10 ppb, all lags</td>
<td>RR, Short term, daily mean</td>
<td>Meta-analysis</td>
<td>Children, age &lt;18</td>
<td>Both</td>
<td>(Zheng et al. 2015)</td>
<td>RR=1.03 (1.02–1.04)</td>
<td></td>
</tr>
<tr>
<td>ALRI-Acute lower respiratory infection</td>
<td>ICD 10: J10-J22</td>
<td>PM2.5 (per 10 µg/m3)</td>
<td>Long term, (subchronic and chronic) effects studies</td>
<td>Meta-analysis</td>
<td>age &lt;5</td>
<td>Both</td>
<td>(Mehta et al. 2013)</td>
<td>RR=1.12(1.03-1.30)</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.3 Summary of the CRF from recent literature and the population at risk.
<table>
<thead>
<tr>
<th>Mortality data by code from the ministry of health</th>
<th>PM2.5 (per 10 μg/m3)</th>
<th>Meta-analysis</th>
<th>Adult, 30+</th>
<th>M,F</th>
<th>(World Health Organization 2013; Héroux et al. 2015)</th>
<th>RR=1.062 (95 CI: 1.04-1.083)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause, non-accidental mortality*</td>
<td>A00-U99</td>
<td>Annual mean Long term</td>
<td>NO2 (per 10 μg/m3)</td>
<td>Meta-analysis</td>
<td>Adult, 30+</td>
<td>M,F</td>
</tr>
<tr>
<td>All-cause, non-accidental mortality*</td>
<td>A00-U99</td>
<td>Daily, maximum 1-hour mean Short term</td>
<td>NO2 (per 10 μg/m3)</td>
<td>Meta-analysis of European cities</td>
<td>All ages</td>
<td>M,F</td>
</tr>
<tr>
<td>All-cause, non-accidental mortality*</td>
<td>A00-U99</td>
<td>Daily maximum 8 hour mean Short term</td>
<td>O3 (per 10 μg/m3) (above 20 μg/m3)</td>
<td>Meta-analysis of European cities</td>
<td>All ages</td>
<td>M,F</td>
</tr>
</tbody>
</table>

**4.6 Stage 6 & 7: RRs for Exposure Difference and Population Attributable Fraction-PAFs**

RRs for exposure difference are affected by the population at risk and the spatial distribution of the pollutants. RRs and PAFs for each statistical area were calculated for each health-pollutant pair (based on exposure differences and the assumption that the entire population is subject to the same level of exposure, explained in the methods section.

Below are the tables (table 4.4-table 4.12) describing the distribution of the RRI and the PAFi by disease, for each pollutant, in the 163 statistical areas (the populated areas from the 179).

**Table 4.4: Type 2 Diabetes mellitus, descriptive statistics, (N=163 statistical areas)**

<table>
<thead>
<tr>
<th></th>
<th>RRI (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRI (O3)</th>
<th>PAFi (O3)</th>
<th>RRI (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.19</td>
<td>0.15</td>
<td>0.95</td>
<td>-0.05</td>
<td>1.04</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>SE</td>
<td>0.01</td>
<td>0.00</td>
<td>0.001</td>
<td>0.001</td>
<td>0.00</td>
</tr>
<tr>
<td>-----</td>
<td>--------</td>
<td>-------</td>
<td>-------</td>
<td>-------</td>
<td>-------</td>
<td>-------</td>
</tr>
<tr>
<td>Min</td>
<td>1.05</td>
<td>0.05</td>
<td>0.93</td>
<td>-0.08</td>
<td>1.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Max</td>
<td>1.51</td>
<td>0.34</td>
<td>1.00</td>
<td>0.00</td>
<td>1.22</td>
<td>0.18</td>
</tr>
</tbody>
</table>

Percentiles

<table>
<thead>
<tr>
<th>Percentile</th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1.09</td>
<td>0.08</td>
<td>0.94</td>
<td>-0.07</td>
</tr>
<tr>
<td>20</td>
<td>1.10</td>
<td>0.09</td>
<td>0.94</td>
<td>-0.06</td>
</tr>
<tr>
<td>Q1</td>
<td>1.12</td>
<td>0.11</td>
<td>0.94</td>
<td>-0.06</td>
</tr>
<tr>
<td>30</td>
<td>1.13</td>
<td>0.11</td>
<td>0.95</td>
<td>-0.06</td>
</tr>
<tr>
<td>40</td>
<td>1.15</td>
<td>0.13</td>
<td>0.95</td>
<td>-0.05</td>
</tr>
<tr>
<td>Median</td>
<td>1.17</td>
<td>0.14</td>
<td>0.95</td>
<td>-0.05</td>
</tr>
<tr>
<td>60</td>
<td>1.19</td>
<td>0.16</td>
<td>0.96</td>
<td>-0.05</td>
</tr>
<tr>
<td>70</td>
<td>1.21</td>
<td>0.17</td>
<td>0.96</td>
<td>-0.04</td>
</tr>
<tr>
<td>Q3</td>
<td>1.23</td>
<td>0.19</td>
<td>0.96</td>
<td>-0.04</td>
</tr>
<tr>
<td>80</td>
<td>1.26</td>
<td>0.21</td>
<td>0.96</td>
<td>-0.04</td>
</tr>
<tr>
<td>90</td>
<td>1.32</td>
<td>0.24</td>
<td>0.97</td>
<td>-0.03</td>
</tr>
</tbody>
</table>

**Table 4.5: Term LBW, descriptive statistics, (N=163 statistical areas)**

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.127</td>
<td>0.110</td>
<td>1.046</td>
<td>0.043</td>
</tr>
<tr>
<td>SE</td>
<td>0.005</td>
<td>0.003</td>
<td>0.002</td>
<td>0.002</td>
</tr>
<tr>
<td>Min</td>
<td>1.036</td>
<td>0.034</td>
<td>1.010</td>
<td>0.010</td>
</tr>
<tr>
<td>Max</td>
<td>1.340</td>
<td>0.254</td>
<td>1.238</td>
<td>0.192</td>
</tr>
</tbody>
</table>

Percentiles

<table>
<thead>
<tr>
<th>Percentile</th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1.059</td>
<td>0.056</td>
<td>1.020</td>
<td>0.020</td>
</tr>
<tr>
<td>20</td>
<td>1.072</td>
<td>0.067</td>
<td>1.026</td>
<td>0.025</td>
</tr>
<tr>
<td>Q1</td>
<td>1.082</td>
<td>0.076</td>
<td>1.027</td>
<td>0.027</td>
</tr>
<tr>
<td>30</td>
<td>1.087</td>
<td>0.080</td>
<td>1.028</td>
<td>0.028</td>
</tr>
</tbody>
</table>
Table 4.6: Lung cancer, descriptive statistics, (N=163 statistical areas)

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.0556</td>
<td>0.0521</td>
<td>1.0104</td>
<td>0.0102</td>
</tr>
<tr>
<td>SE</td>
<td>0.0019</td>
<td>0.0017</td>
<td>0.0005</td>
<td>0.0005</td>
</tr>
<tr>
<td>Minimum</td>
<td>1.0160</td>
<td>0.0158</td>
<td>1.0023</td>
<td>0.0023</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.1424</td>
<td>0.1247</td>
<td>1.0509</td>
<td>0.0484</td>
</tr>
</tbody>
</table>

Percentiles

| 10  | 1.0266     | 0.0259     | 1.0047      | 0.0047       |
| 20  | 1.0323     | 0.0313     | 1.0060      | 0.0059       |
| Q1  | 1.0364     | 0.0351     | 1.0063      | 0.0063       |
| 30  | 1.0388     | 0.0373     | 1.0065      | 0.0065       |
| 40  | 1.0453     | 0.0433     | 1.0073      | 0.0072       |
| Median| 1.0509    | 0.0485     | 1.0084      | 0.0083       |
| 60  | 1.0575     | 0.0544     | 1.0097      | 0.0096       |
| 70  | 1.0633     | 0.0595     | 1.0109      | 0.0107       |
| Q3  | 1.0698     | 0.0652     | 1.0123      | 0.0121       |
| 80  | 1.0769     | 0.0714     | 1.0146      | 0.0144       |
| 90  | 1.0928     | 0.0849     | 1.0192      | 0.0188       |
### Table 4.7: Stroke, descriptive statistics, (N=163 statistical areas)

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (O3)</th>
<th>PAFi (O3)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.0096</td>
<td>0.0095</td>
<td>0.997</td>
<td>-0.0003</td>
<td>1.10</td>
<td>0.08</td>
</tr>
<tr>
<td>SE</td>
<td>0.0003</td>
<td>0.0003</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Minimum</td>
<td>1.0028</td>
<td>0.0028</td>
<td>0.9995</td>
<td>-0.0005</td>
<td>1.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.0239</td>
<td>0.0233</td>
<td>1.0000</td>
<td>0.0000</td>
<td>1.54</td>
<td>0.35</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Percentiles</th>
<th>RRi</th>
<th>PAFi</th>
<th>RRi</th>
<th>PAFi</th>
<th>RRi</th>
<th>PAFi</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>1.0047</td>
<td>0.0046</td>
<td>0.996</td>
<td>-0.0004</td>
<td>1.0417</td>
<td>0.0401</td>
</tr>
<tr>
<td>20%</td>
<td>1.0056</td>
<td>0.0056</td>
<td>0.996</td>
<td>-0.0004</td>
<td>1.0535</td>
<td>0.0508</td>
</tr>
<tr>
<td>Q1</td>
<td>1.0064</td>
<td>0.0063</td>
<td>0.996</td>
<td>-0.0004</td>
<td>1.0564</td>
<td>0.0534</td>
</tr>
<tr>
<td>30%</td>
<td>1.0068</td>
<td>0.0067</td>
<td>0.996</td>
<td>-0.0004</td>
<td>1.0584</td>
<td>0.0552</td>
</tr>
<tr>
<td>40%</td>
<td>1.0079</td>
<td>0.0078</td>
<td>0.997</td>
<td>-0.0003</td>
<td>1.0656</td>
<td>0.0616</td>
</tr>
<tr>
<td>Median</td>
<td>1.0088</td>
<td>0.0088</td>
<td>0.997</td>
<td>-0.0003</td>
<td>1.0756</td>
<td>0.0703</td>
</tr>
<tr>
<td>60%</td>
<td>1.01</td>
<td>0.0099</td>
<td>0.997</td>
<td>-0.0003</td>
<td>1.0878</td>
<td>0.0807</td>
</tr>
<tr>
<td>70%</td>
<td>1.0109</td>
<td>0.0108</td>
<td>0.997</td>
<td>-0.0003</td>
<td>1.0990</td>
<td>0.0901</td>
</tr>
<tr>
<td>Q3</td>
<td>1.012</td>
<td>0.0119</td>
<td>0.998</td>
<td>-0.0002</td>
<td>1.1124</td>
<td>0.1010</td>
</tr>
<tr>
<td>80%</td>
<td>1.0132</td>
<td>0.013</td>
<td>0.998</td>
<td>-0.0002</td>
<td>1.1355</td>
<td>0.1193</td>
</tr>
<tr>
<td>90%</td>
<td>1.0159</td>
<td>0.0156</td>
<td>0.998</td>
<td>-0.0002</td>
<td>1.1806</td>
<td>0.1530</td>
</tr>
</tbody>
</table>

### Table 4.8: Ischemic Heart Disease, descriptive statistics, (N=163 statistical areas)

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.0117</td>
<td>0.0115</td>
<td>1.0028</td>
<td>0.0028</td>
</tr>
<tr>
<td>SE</td>
<td>0.0004</td>
<td>0.0004</td>
<td>0.0001</td>
<td>0.0001</td>
</tr>
<tr>
<td>Minimum</td>
<td>1.0034</td>
<td>0.0034</td>
<td>1.0006</td>
<td>0.0006</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.0290</td>
<td>0.0282</td>
<td>1.0136</td>
<td>0.0134</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Percentiles</th>
<th>RRi</th>
<th>PAFi</th>
<th>RRi</th>
<th>PAFi</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>1.0057</td>
<td>0.0056</td>
<td>1.0013</td>
<td>0.0013</td>
</tr>
</tbody>
</table>
Table 4.9: COPD, descriptive statistics, (N=163 statistical areas)

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.0584</td>
<td>0.0546</td>
<td>1.0033</td>
<td>0.0033</td>
</tr>
<tr>
<td>SE</td>
<td>0.0020</td>
<td>0.0018</td>
<td>0.0002</td>
<td>0.0002</td>
</tr>
<tr>
<td>Minimum</td>
<td>1.0168</td>
<td>0.0165</td>
<td>1.0007</td>
<td>0.0007</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.1499</td>
<td>0.1304</td>
<td>1.0161</td>
<td>0.0158</td>
</tr>
</tbody>
</table>

Percentiles

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1.0280</td>
<td>0.0272</td>
<td>1.0015</td>
<td>0.0015</td>
</tr>
<tr>
<td>20</td>
<td>1.0339</td>
<td>0.0328</td>
<td>1.0019</td>
<td>0.0019</td>
</tr>
<tr>
<td>Q1</td>
<td>1.0382</td>
<td>0.0368</td>
<td>1.0020</td>
<td>0.0020</td>
</tr>
<tr>
<td>30</td>
<td>1.0407</td>
<td>0.0391</td>
<td>1.0021</td>
<td>0.0021</td>
</tr>
<tr>
<td>40</td>
<td>1.0476</td>
<td>0.0454</td>
<td>1.0023</td>
<td>0.0023</td>
</tr>
<tr>
<td>Median</td>
<td>1.0535</td>
<td>0.0508</td>
<td>1.0027</td>
<td>0.0027</td>
</tr>
<tr>
<td>60</td>
<td>1.0604</td>
<td>0.0570</td>
<td>1.0031</td>
<td>0.0031</td>
</tr>
<tr>
<td>70</td>
<td>1.0665</td>
<td>0.0623</td>
<td>1.0035</td>
<td>0.0035</td>
</tr>
<tr>
<td>Q3</td>
<td>1.0733</td>
<td>0.0683</td>
<td>1.0039</td>
<td>0.0039</td>
</tr>
</tbody>
</table>
Table 4.10: Asthma Incidences or lifetime prevalence (based on prevalence cases), descriptive statistics, (N=163 statistical areas)

<table>
<thead>
<tr>
<th></th>
<th>RRi (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRi (O3)</th>
<th>PAFi (O3)</th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.186</td>
<td>0.152</td>
<td>0.991</td>
<td>-0.009</td>
<td>1.041</td>
<td>0.039</td>
</tr>
<tr>
<td>SE</td>
<td>0.007</td>
<td>0.005</td>
<td>0.000</td>
<td>0.000</td>
<td>0.002</td>
<td>0.002</td>
</tr>
<tr>
<td>Minimum</td>
<td>1.051</td>
<td>0.048</td>
<td>0.985</td>
<td>-0.015</td>
<td>1.009</td>
<td>0.009</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.513</td>
<td>0.339</td>
<td>1.000</td>
<td>0.000</td>
<td>1.210</td>
<td>0.174</td>
</tr>
</tbody>
</table>

Table 4.11: ALRI, descriptive statistics, (N=163 statistical area)

<table>
<thead>
<tr>
<th></th>
<th>RRi (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.0153</td>
<td>0.0150</td>
</tr>
<tr>
<td>SE</td>
<td>0.0007</td>
<td>0.0007</td>
</tr>
</tbody>
</table>
### Table 4.12: All cause Non-accidental mortality, descriptive statistics, (N=163 statistical areas)

<table>
<thead>
<tr>
<th></th>
<th>RRI (NO2)</th>
<th>PAFi (NO2)</th>
<th>RRI (O3)</th>
<th>PAFi (O3)</th>
<th>RRI (PM2.5)</th>
<th>PAFi (PM2.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.0276</td>
<td>0.0267</td>
<td>0.998</td>
<td>-0.002</td>
<td>1.0081</td>
<td>0.0080</td>
</tr>
<tr>
<td>SE</td>
<td>0.0009</td>
<td>0.0009</td>
<td>0.000</td>
<td>0.000</td>
<td>0.0004</td>
<td>0.0004</td>
</tr>
<tr>
<td>Minimum</td>
<td>1.0081</td>
<td>0.0080</td>
<td>0.997</td>
<td>-0.003</td>
<td>1.0018</td>
<td>0.0018</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.0695</td>
<td>0.0650</td>
<td>1.000</td>
<td>0.000</td>
<td>1.0396</td>
<td>0.0381</td>
</tr>
<tr>
<td>Percentiles</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1.0134</td>
<td>0.0132</td>
<td>0.997</td>
<td>-0.003</td>
<td>1.0037</td>
<td>0.0036</td>
</tr>
<tr>
<td>20</td>
<td>1.0162</td>
<td>0.0159</td>
<td>0.998</td>
<td>-0.002</td>
<td>1.0047</td>
<td>0.0046</td>
</tr>
<tr>
<td>Q1</td>
<td>1.0182</td>
<td>0.0179</td>
<td>0.998</td>
<td>-0.002</td>
<td>1.0049</td>
<td>0.0049</td>
</tr>
<tr>
<td>30</td>
<td>1.0194</td>
<td>0.0190</td>
<td>0.998</td>
<td>-0.002</td>
<td>1.0051</td>
<td>0.0051</td>
</tr>
</tbody>
</table>
4.8 Stage 8: Calculating Incidence Cases Within Each Census Tract.

**National IR**

The national IR is summarized in the two following tables. The main sources were peer review scientific papers and official reports. When the IR was not reported, a proximation was calculated based on different data as explained in table 4.13 below.

**Table 4.13(a) National IR for main symptoms**

<table>
<thead>
<tr>
<th>Health outcome [refers to outcome in table 4.13-4.14]</th>
<th>Population at Risk Gender and age</th>
<th>Annual average of the total cases in Israel*</th>
<th>Population at risk in Israel (thousands)</th>
<th>Calculated incidence rates (per 100,000)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality from non-accidental causes</td>
<td>Both, 30+ All ages</td>
<td>37,479.4</td>
<td>3820.1</td>
<td>981</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>38,529.6</td>
<td>7766.1</td>
<td>496.14</td>
<td></td>
</tr>
<tr>
<td>IHD hospitalizations discharge**</td>
<td>Both, 20+</td>
<td>98,590.6</td>
<td>4979.3</td>
<td>1980.01</td>
<td>Hospitalizations used as a proxy for incidence</td>
</tr>
<tr>
<td>[Heart failure hospitalization or death]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALRI hospitalizations discharge</td>
<td>Both, 0-5</td>
<td>12,639.8</td>
<td>795.5</td>
<td>1591.31</td>
<td>Hospitalizations discharge for 0-4 used as a proxy for incidence 0-5</td>
</tr>
<tr>
<td>[ALRI-Acute lower respiratory infection]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke hospitalizations discharge</td>
<td>Both, 30+ 60+ All</td>
<td>21,158.4</td>
<td>3820.1</td>
<td>553.87</td>
<td>Hospitalization discharge used as a proxy for incidence (underestimate)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>17,438.4</td>
<td>1143.2</td>
<td>1525.40</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>21,400.4</td>
<td>7766.1</td>
<td>275.56</td>
<td></td>
</tr>
</tbody>
</table>
[Stroke hospitalization])

| COPD hospitalizations discharge ([COPD Emergency department visit and hospitalization]) | Both, Adults (calculated as 30+) | 31,713.0 | 3820.1 | 830.16 | Hospitalization discharges used as a proxy for incidence (underestimate) |

* The annual average of the total cases in Israel was calculated from the total cases in five years divided by 5.

** Hospitalization discharge was used to estimate the number of hospitalizations.

For the other health outcomes, IR was obtained from other sources as described below:

### Table 4.13 (b): National IR for the other symptoms

<table>
<thead>
<tr>
<th>Health outcome</th>
<th>Population at Risk Gender and age</th>
<th>Population at risk size</th>
<th>Data source</th>
<th>Original rates reported in source</th>
<th>Year</th>
<th>How the IR was calculated</th>
<th>Calculated IR (per 100,000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke incidences</td>
<td>Both 60+</td>
<td>125,845</td>
<td>ICDC</td>
<td>3.3 (95% CI:3.3-3.4) per 1,000 for the whole population</td>
<td>2015</td>
<td>Direct use of ICDC data (X100) since no data is available on IR of 60+ (making this IR an underestimate for 60+)</td>
<td>330</td>
</tr>
<tr>
<td>Asthma Incidences or lifetime prevalence ([Asthma incidence or lifetime prevalence from birth until 18 years old]*)</td>
<td>Both 0-18</td>
<td>131,612</td>
<td>Shohat et al. 2000</td>
<td>Asthma in Israel, affecting at least 7% of children.</td>
<td>2000</td>
<td>Since no data was available of new occurrences per year, prevalence was used to estimate IR based on population distribution</td>
<td>7000</td>
</tr>
<tr>
<td>Diabetes [Type 2 Diabetes mellitus]</td>
<td>Both 21+</td>
<td>344,826</td>
<td>Clalit Health Services (Jaffe et al. 2017)</td>
<td>Annual IR for Arabs 2.9% (95%CI: 2.7–3.1) and Jews 1.7% (95%CI: 1.6–1.8)</td>
<td>2015</td>
<td>The ratio of Jews/Arabs in Haifa district (=4.46 CSB 2010) was used.</td>
<td>1919.6</td>
</tr>
<tr>
<td>Lung cancer IRs [Lung cancer incidence]</td>
<td>Both 30+</td>
<td>313,620</td>
<td>Cancer in Israel (2019)</td>
<td></td>
<td>2018</td>
<td>The ratio of F/M in Israel (=1.0202 CBS 2019) was used</td>
<td>11.32</td>
</tr>
</tbody>
</table>
Population at risk, by census tract

In the study area, there were 163 census tracts with a population larger than zero. Mean RR’s and concentrations were not calculated for census tracts in non-populated areas. Distribution according to population size and age was not normal, and for all ages, the population size ranged between 10 to 11360 individuals. The total population size in the study area was 521,230, of which 313,620 individuals were 30 years old or older and 106,900 were children younger than 15. Table 4.14 summarizes the distribution of population size at risk by groups, in the 179 census tracts in the study area (163 populated areas). The median of the population density in the area by census tract was 7,779 individuals per square km (Interquartile range-(IQR): 7245 and 10th and 90th percentiles were 1052 and 15910, respectively). Figure 4.6 below displays the total population numbers residing in the different areas in the HBA. The differences in the excess cases could be explained by the differences in the population at risk and the differences in the air pollution levels.

Table 4.14: Descriptive statistics of the population size by the population at risk groups in the 163 census tracts in the study area.

<table>
<thead>
<tr>
<th></th>
<th>Total population</th>
<th>Male Total</th>
<th>Female Total</th>
<th>Age 0-14</th>
<th>Age 0-5 (extr.)</th>
<th>Age 15-19</th>
<th>Age 0-18 (extr.)</th>
<th>Age 20-29</th>
<th>Age 30-64</th>
<th>Age &gt; 65</th>
<th>Adult (&gt;20)</th>
<th>Adults (&gt;21, extr.)</th>
<th>Adults (&gt;30)</th>
<th>Adults (30-60, extr.)</th>
<th>Females (20-49, extr.)</th>
<th>Adult (&gt;60, extr.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>3198</td>
<td>1542</td>
<td>1656</td>
<td>656</td>
<td>226</td>
<td>190</td>
<td>807</td>
<td>427</td>
<td>132</td>
<td>602</td>
<td>2351</td>
<td>2308</td>
<td>1924</td>
<td>1190</td>
<td>611</td>
<td>772</td>
</tr>
<tr>
<td>SD</td>
<td>1683</td>
<td>822</td>
<td>867</td>
<td>502</td>
<td>173</td>
<td>133</td>
<td>601</td>
<td>290</td>
<td>688</td>
<td>335</td>
<td>1170</td>
<td>1148</td>
<td>976</td>
<td>619</td>
<td>316</td>
<td>409</td>
</tr>
<tr>
<td>----</td>
<td>------</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
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<td>------</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>Min.</td>
<td>10</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Percentiles</th>
<th>10</th>
<th>804</th>
<th>400</th>
<th>404</th>
<th>184</th>
<th>64</th>
<th>70</th>
<th>228</th>
<th>122</th>
<th>300</th>
<th>100</th>
<th>562</th>
<th>534</th>
<th>422</th>
<th>270</th>
<th>160</th>
<th>135</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q1</td>
<td>2040</td>
<td>980</td>
<td>1040</td>
<td>370</td>
<td>128</td>
<td>110</td>
<td>460</td>
<td>270</td>
<td>830</td>
<td>330</td>
<td>1490</td>
<td>1461</td>
<td>1200</td>
<td>747</td>
<td>389</td>
<td>451</td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>2262</td>
<td>1112</td>
<td>1172</td>
<td>392</td>
<td>135</td>
<td>120</td>
<td>496</td>
<td>292</td>
<td>914</td>
<td>380</td>
<td>1636</td>
<td>1608</td>
<td>1328</td>
<td>823</td>
<td>415</td>
<td>555</td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>3370</td>
<td>1580</td>
<td>1730</td>
<td>630</td>
<td>217</td>
<td>170</td>
<td>768</td>
<td>390</td>
<td>1370</td>
<td>620</td>
<td>2500</td>
<td>2462</td>
<td>2050</td>
<td>1233</td>
<td>636</td>
<td>814</td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>3640</td>
<td>1714</td>
<td>1908</td>
<td>714</td>
<td>246</td>
<td>200</td>
<td>882</td>
<td>434</td>
<td>1504</td>
<td>720</td>
<td>2748</td>
<td>2666</td>
<td>2278</td>
<td>1354</td>
<td>687</td>
<td>938</td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>3954</td>
<td>1870</td>
<td>2098</td>
<td>778</td>
<td>268</td>
<td>220</td>
<td>958</td>
<td>518</td>
<td>1714</td>
<td>848</td>
<td>3058</td>
<td>3011</td>
<td>2506</td>
<td>1543</td>
<td>761</td>
<td>1074</td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>4140</td>
<td>1990</td>
<td>2160</td>
<td>840</td>
<td>290</td>
<td>240</td>
<td>1028</td>
<td>550</td>
<td>1800</td>
<td>900</td>
<td>3180</td>
<td>3122</td>
<td>2660</td>
<td>1620</td>
<td>816</td>
<td>1115</td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>4470</td>
<td>2222</td>
<td>2322</td>
<td>892</td>
<td>308</td>
<td>252</td>
<td>1091</td>
<td>590</td>
<td>1890</td>
<td>930</td>
<td>3264</td>
<td>3205</td>
<td>2722</td>
<td>1701</td>
<td>878</td>
<td>1166</td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>5006</td>
<td>2416</td>
<td>2602</td>
<td>1150</td>
<td>396</td>
<td>322</td>
<td>1350</td>
<td>666</td>
<td>2120</td>
<td>1010</td>
<td>3764</td>
<td>3699</td>
<td>3136</td>
<td>1908</td>
<td>997</td>
<td>1272</td>
<td></td>
</tr>
<tr>
<td>Max.</td>
<td>11360</td>
<td>5810</td>
<td>5550</td>
<td>4880</td>
<td>1683</td>
<td>1270</td>
<td>5896</td>
<td>2470</td>
<td>3300</td>
<td>1300</td>
<td>5310</td>
<td>5193</td>
<td>4140</td>
<td>2970</td>
<td>1726</td>
<td>1571</td>
<td></td>
</tr>
</tbody>
</table>

Q1: 25th percentile, Q3: 75th percentile, Min.: minimum, Max.: maximum, SD: standard deviation, Extr.: extrapolation based on age distribution from the Central Bureau of Statistics.
Figure 4.6: Population distribution in the HBA

Incidence cases, by census tract

Descriptive statistics of new cases per year

The total number of new cases in 2015 for ALRI, asthma, COPD, diabetes, ischemic heart disease, lung cancer, stroke and term LBW in the area were 587, 1184, 2604, 7221, 7586, 36, 2152, 288 respectively. Table 4.15 summarizes the distribution of new cases that were calculated in the 179 census tracts in the study area.
Table 4.15: Descriptive Statistics of the Pollution Attributed Cases of morbidity in 2015, Categorized by Health Outcomes

<table>
<thead>
<tr>
<th></th>
<th>ALRI</th>
<th>Asthma Incidence s or lifetime prevalence</th>
<th>COPD</th>
<th>Type 2 Diabetes</th>
<th>Ischemic Heart Disease</th>
<th>Lung cancer</th>
<th>Stroke</th>
<th>Term LBW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>3.60</td>
<td>1.22</td>
<td>16.00</td>
<td>5.99</td>
<td>46.50</td>
<td>0.20</td>
<td>0.11</td>
<td>1.80</td>
</tr>
<tr>
<td>SE</td>
<td>0.20</td>
<td>0.07</td>
<td>0.60</td>
<td>0.33</td>
<td>1.80</td>
<td>0.00</td>
<td>0.01</td>
<td>0.10</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.03</td>
<td>0.20</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Maximum</td>
<td>26.80</td>
<td>5.11</td>
<td>34.40</td>
<td>22.50</td>
<td>105.10</td>
<td>0.50</td>
<td>0.38</td>
<td>5.00</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Percentiles</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1.00</td>
<td>0.23</td>
<td>3.50</td>
<td>0.73</td>
<td>11.10</td>
<td>0.10</td>
<td>0.02</td>
<td>0.50</td>
</tr>
<tr>
<td>20</td>
<td>1.60</td>
<td>0.52</td>
<td>8.60</td>
<td>2.22</td>
<td>26.90</td>
<td>0.10</td>
<td>0.05</td>
<td>1.00</td>
</tr>
<tr>
<td>Q1</td>
<td>2.00</td>
<td>0.62</td>
<td>10.00</td>
<td>2.89</td>
<td>29.50</td>
<td>0.10</td>
<td>0.06</td>
<td>1.10</td>
</tr>
<tr>
<td>30</td>
<td>2.20</td>
<td>0.75</td>
<td>11.00</td>
<td>3.34</td>
<td>32.40</td>
<td>0.20</td>
<td>0.07</td>
<td>1.20</td>
</tr>
<tr>
<td>40</td>
<td>2.80</td>
<td>0.92</td>
<td>14.70</td>
<td>4.41</td>
<td>43.70</td>
<td>0.20</td>
<td>0.09</td>
<td>1.60</td>
</tr>
<tr>
<td>Median</td>
<td>3.50</td>
<td>1.09</td>
<td>17.00</td>
<td>5.37</td>
<td>49.50</td>
<td>0.20</td>
<td>0.10</td>
<td>1.80</td>
</tr>
<tr>
<td>60</td>
<td>3.90</td>
<td>1.23</td>
<td>18.90</td>
<td>6.09</td>
<td>54.40</td>
<td>0.30</td>
<td>0.12</td>
<td>2.00</td>
</tr>
<tr>
<td>70</td>
<td>4.30</td>
<td>1.51</td>
<td>21.30</td>
<td>7.86</td>
<td>60.60</td>
<td>0.30</td>
<td>0.14</td>
<td>2.20</td>
</tr>
<tr>
<td>Q3</td>
<td>4.60</td>
<td>1.61</td>
<td>22.10</td>
<td>8.63</td>
<td>63.00</td>
<td>0.30</td>
<td>0.15</td>
<td>2.40</td>
</tr>
<tr>
<td>80</td>
<td>4.90</td>
<td>1.83</td>
<td>22.60</td>
<td>9.09</td>
<td>64.60</td>
<td>0.30</td>
<td>0.16</td>
<td>2.50</td>
</tr>
<tr>
<td>90</td>
<td>6.30</td>
<td>2.23</td>
<td>26.00</td>
<td>11.89</td>
<td>74.50</td>
<td>0.40</td>
<td>0.20</td>
<td>2.90</td>
</tr>
</tbody>
</table>

4.9 Stage 9: Calculation of the Estimated Excess Number of Cases Within the Census

Descriptive statistics of new cases per year attributable to exposure to pollutants

4.9 Stage 9: Calculation of the Estimated Excess Number of Cases Within the Census

Table 4.16 below summarizes the distribution of the new cases in the study area calculated in the 179 census tracts.
### Table 4.16: Descriptive Statistics of Pollution Attributed Cases Categorized by Health Outcomes

<table>
<thead>
<tr>
<th>ALRI</th>
<th>COPD</th>
<th>Type 2 Diabetes mellitus</th>
<th>Ischemic Heart Disease</th>
<th>Lung cancer</th>
<th>Stroke</th>
<th>Term LBW</th>
<th>Asthma Incidences or lifetime prevalence</th>
<th>Asthma Incidences or lifetime prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>PM2.5</td>
<td>NO2</td>
</tr>
<tr>
<td>Mean</td>
<td>0.05</td>
<td>0.77</td>
<td>0.04</td>
<td>5.97</td>
<td>-2.17</td>
<td>1.46</td>
<td>0.47</td>
<td>0.11</td>
</tr>
<tr>
<td>SE</td>
<td>0.00</td>
<td>0.04</td>
<td>0.00</td>
<td>0.31</td>
<td>-0.10</td>
<td>0.08</td>
<td>0.03</td>
<td>0.01</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>-4.18</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.32</td>
<td>2.86</td>
<td>0.16</td>
<td>21.92</td>
<td>0.00</td>
<td>5.49</td>
<td>1.87</td>
<td>0.40</td>
</tr>
<tr>
<td>Q1</td>
<td>0.02</td>
<td>0.34</td>
<td>0.02</td>
<td>0.04</td>
<td>-2.95</td>
<td>0.07</td>
<td>0.24</td>
<td>0.05</td>
</tr>
<tr>
<td>Q2</td>
<td>0.04</td>
<td>0.72</td>
<td>0.04</td>
<td>5.69</td>
<td>-2.05</td>
<td>1.28</td>
<td>0.44</td>
<td>0.09</td>
</tr>
<tr>
<td>Q3</td>
<td>0.07</td>
<td>1.13</td>
<td>0.06</td>
<td>8.81</td>
<td>-1.17</td>
<td>2.06</td>
<td>0.68</td>
<td>0.15</td>
</tr>
</tbody>
</table>

Figure 4.7 below presents the distribution of mortality incidences in the HBA. It is split between maximum and minimum values according to our hypothesis regarding the double counting effects from different pollutants (see section 3.10). Figure 4.8 describes the mortality distribution according to pollution source. Industrial pollution on the right and pollution caused by transportation on the left.
Figure 4.7: Map of mortality distribution

(Map 4.7: Map of mortality distribution (Minimum on the left side and Maximum on the right side))

Map 4.8: Mortality distribution according to pollution sources
4.10 Stage 10: Economic analysis

Table 4.17 gives an estimated cost per one symptom case for Israel, for 2015. This is a mean value of the relevant studies used in section 3. These values were adjusted based on the GDP ratio and price index described in section 3.

Table 4.17: summary of morbidity and mortality costs, adjusted to Israel 2015

<table>
<thead>
<tr>
<th>Disease</th>
<th>Mean Cost per incidence case (ILS)</th>
<th>C.I. (95%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Heart Disease</td>
<td>112,154</td>
<td>99,817 - 124,491</td>
</tr>
<tr>
<td>Stroke</td>
<td>178,000</td>
<td>96,120 - 259,880</td>
</tr>
<tr>
<td>Asthma Incidences or lifetime prevalence</td>
<td>10,126</td>
<td>5,671 - 14,581</td>
</tr>
<tr>
<td>Type 2 Diabetes mellitus</td>
<td>45,000</td>
<td>28,800 – 61,200</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>120,000</td>
<td>69,600 – 170,400</td>
</tr>
<tr>
<td>ALRI</td>
<td>1,233</td>
<td>1,097 – 1,352</td>
</tr>
<tr>
<td>COPD</td>
<td>24,499</td>
<td>16,414 – 32,584</td>
</tr>
<tr>
<td>Term LBW</td>
<td>147,449</td>
<td>95,842 – 199,056</td>
</tr>
<tr>
<td>Blood cancer</td>
<td>318,000</td>
<td>178,080 – 457,920</td>
</tr>
<tr>
<td>VSL</td>
<td>12.95 mill. ILS</td>
<td></td>
</tr>
<tr>
<td>VOLY</td>
<td>0.35 mill ILS</td>
<td></td>
</tr>
</tbody>
</table>

The cost per symptom (table 4.17) multiplied by the number of symptom cases attributable to pollution, as explained in section 4.9 (table 4.16), allowed us to calculate the cost of each morbidity case (table 4.18). By using the different valuation methods of VSL and VOLY and multiplying them by the number of mortalities attributed to pollution, we calculated the cost of morbidity (table 4.18). Table 4.18 presents calculations of morbidity from each combination of symptom and pollutant. Table 4.19 shows the cost of morbidity by both VSL and VOLY as well as the sum of morbidity and mortality.
Table 4.18: Summary of morbidity costs from symptoms attributed to the different pollutants.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Gender and Age</th>
<th>Total population size</th>
<th>Background IR (per 100,000)</th>
<th>NO₂</th>
<th>Cost (Mill. ILS)</th>
<th>O₃</th>
<th>Cost (Mill. ILS)</th>
<th>PM2.5</th>
<th>Cost (Mill. ILS)</th>
<th>Benzene</th>
<th>Cost (Mill. ILS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Heart Disease – incidence²</td>
<td>All 20+</td>
<td>383,140</td>
<td>1,980.01</td>
<td>85</td>
<td>9.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>18.81</td>
<td>2.10</td>
</tr>
<tr>
<td>Stroke – incidence²</td>
<td>All 60+</td>
<td>125,845</td>
<td>330.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>131.65</td>
<td>23.43</td>
<td></td>
</tr>
<tr>
<td>Stroke – Hospitalizations³</td>
<td>All 30+</td>
<td>313,620</td>
<td>553.87</td>
<td>16</td>
<td>2.84</td>
<td>-0.53</td>
<td>-0.09</td>
<td>2.24</td>
<td>0.39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma Incidences or lifetime prevalence – ⁴</td>
<td>All 0-18</td>
<td>131,612</td>
<td>7,000.00</td>
<td>1,326</td>
<td>13.43</td>
<td>-86.42</td>
<td>-0.87</td>
<td>308.67</td>
<td>3.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma Incidences or lifetime prevalence – ⁵</td>
<td>All 0-18</td>
<td>131,612</td>
<td>900.00</td>
<td>171</td>
<td>120.88</td>
<td>-11.11</td>
<td>-7.80</td>
<td>39.69</td>
<td>28.13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 2 Diabetes mellitus – Incidence</td>
<td>All 21+</td>
<td>344,826</td>
<td>1919.6</td>
<td>1,068</td>
<td>48.07</td>
<td>-353.32</td>
<td>-15.9</td>
<td>261.45</td>
<td>11.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lung cancer – Incidence</td>
<td>All 30+</td>
<td>313,620</td>
<td>11.32</td>
<td>2</td>
<td>0.22</td>
<td></td>
<td></td>
<td>0.32</td>
<td>0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALRI – Incidence</td>
<td>All 0-5</td>
<td>36,862</td>
<td>1591.31</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9.15</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COPD – Hospitalizations</td>
<td>All adults</td>
<td>313,620</td>
<td>830.16</td>
<td>138</td>
<td>3.4</td>
<td></td>
<td></td>
<td>7.62</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Term LBW-births</td>
<td>Females 20-49</td>
<td>99,650</td>
<td>288.557</td>
<td>36</td>
<td>5.27</td>
<td></td>
<td></td>
<td>12.70</td>
<td>1.87</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukemia - Incidence²</td>
<td>All ages</td>
<td>521,230</td>
<td>1.70</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.84</td>
<td>1.17</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Short term exposure.
2. IR given for the general population and not for age 60+, therefore costs are an underestimation.
3. Short term exposure
4. Based on prevalence of 7% used as the main figure for summary calculations.
5. Based on incidence rates from the U.S.A in 2009 of 900 cases per 100,000. Used as a secondary figure.
6. Instead of IR, this calculation uses a direct estimate in the number of attributed cases per increase of 1 µm/m³ of Benzene.
As can be seen from table 4.18, if we start from the left column, for each symptom we describe the relevant age range and the total population size at that age range in the HBA. The fourth column is the background incidence rate (see explanation in sections 3.8, and 4.8 and full list in table 4.13). Using the relevant risk ratio functions, we were able to show the number of new cases attributable to each pollutant (as listed in table 4.16). Later, by multiplying that number by the cost of the symptom (see explanation in section 3.10 and a full list of costs in table 4.17), we calculated the total monetary cost for that symptom for each pollutant.

Summing up the total morbidity effects yields the estimates presented in table 4.19 which also shows the mortality effects (also divided by pollution sources). Please note that the total cost calculations are not summaries of the lines in the table but of all census tracts.

<table>
<thead>
<tr>
<th></th>
<th>Mortality from industrial pollution</th>
<th>Mortality from transport pollution</th>
<th>Mortality from total pollution</th>
<th>Mortality cost by VOLY</th>
<th>Mortality cost by VSL</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO₂</td>
<td>7.21</td>
<td>72.75</td>
<td>79.96</td>
<td>360,473,493</td>
<td>1,033,138,220</td>
</tr>
<tr>
<td>PM2.5</td>
<td>7.16</td>
<td>14.59</td>
<td>21.75</td>
<td>98,082,538</td>
<td>281,110,319</td>
</tr>
<tr>
<td>Benzene</td>
<td>-</td>
<td>-</td>
<td>0.69</td>
<td>3,100,485</td>
<td>8,886,172</td>
</tr>
<tr>
<td>O₃ mortality reduction</td>
<td>-</td>
<td>-</td>
<td>-5.35</td>
<td>-24,178,037</td>
<td>-69,295,675</td>
</tr>
<tr>
<td>Total(_{\text{min}})*</td>
<td>7.66</td>
<td>72.75</td>
<td>80.45</td>
<td>360,473,493</td>
<td>1,033,138,220</td>
</tr>
<tr>
<td>Total(_{\text{max}})**</td>
<td>14.36</td>
<td>87.33</td>
<td>101.69</td>
<td>437,478,479</td>
<td>1,253,839,036</td>
</tr>
</tbody>
</table>

* Total\(_{\text{min}}\) = Conservative sum of only one (highest) pollutant cost in each census tract to avoid overlap.

** Total\(_{\text{max}}\) = Sum of all 4 pollutant costs assuming a neglectable overlap.
Table 4.19 shows the number of deaths caused by industrial pollution, transportation pollution, the sum of the two sources, and the years of lost lives (estimates were not adjusted to quality of life). Our estimates are based on life tables described in the Global Burden of Disease study (2017)\(^\text{14}\). Total number of deaths is estimated to be 2365, while the total number of years lost is estimated to be 30,390 (for life expectancy at birth of 82.5 years in Israel). That is a mean value of 14.44 years per life lost.

It should be noted, that since four pollutants have been examined, we may face a problem of double counting as previously explained, therefore minimum and maximum effects are presented. See table legend for an explanation on the difference between these values. In addition, as shown in table 4.19, mortality is in the range of 94 – 183 deaths annually. We use two values for the mortality cost. The first column is the cost derived from the number of years lost times the cost of one year of life (VOLY). The second column shows the results based on an equal value for statistical life (VSL).

Table 4.20: Summaries of mortality and morbidity costs (ILS/Year)

<table>
<thead>
<tr>
<th></th>
<th>Mortality cost by VOLY</th>
<th>Mortality cost by VSL</th>
<th>Morbidity cost</th>
<th>Total cost (using VOLY)</th>
<th>Total cost (using VSL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO(_2)</td>
<td>360,473,493</td>
<td>1,033,138,220</td>
<td>79,889,728</td>
<td>440,363,221</td>
<td>1,113,027,948</td>
</tr>
<tr>
<td>PM2.5</td>
<td>98,082,538</td>
<td>281,110,319</td>
<td>70,680,450</td>
<td>168,762,988</td>
<td>323,659,926</td>
</tr>
<tr>
<td>Benzene</td>
<td>3,100,485</td>
<td>8,886,172</td>
<td>1,170,020</td>
<td>4,270,505</td>
<td>10,056,192</td>
</tr>
<tr>
<td>Total(_{\text{min}})*</td>
<td>360,473,493</td>
<td>1,033,138,220</td>
<td>200,772,882</td>
<td>561,246,375</td>
<td>1,113,027,948</td>
</tr>
<tr>
<td>Total(_{\text{max}})**</td>
<td>437,478,479</td>
<td>1,253,839,036</td>
<td>247,973,059</td>
<td>685,451,538</td>
<td>1,360,674,018</td>
</tr>
</tbody>
</table>

* Total\(_{\text{min}}\) = Conservative sum of only one (highest) pollutant cost in each census tract to avoid overlap.

** Total\(_{\text{max}}\) = Sum of all 4 pollutant costs assuming neglectable overlap.

\(^{14}\) [http://ghdx.healthdata.org/gbd-results-tool](http://ghdx.healthdata.org/gbd-results-tool)
Table 4.20 shows the results in terms of monetary costs. The first two columns on the left were taken from table 4.19 and show the total cost of mortality summed up, given for the years of life lost and the value of statistical life. To consider the possible double counting, we show again the minimum and maximum endpoints. Added to this is the summed value of morbidity costs as calculated in table 4.18. Summary columns on the left are also given for both VOLY and VSL methods of calculating mortality costs.

The total cost that results from air pollution in the HBA, in 2015, ranges from 685 to 1,352 million ILS depending on the assumption. Figures 4.9 and 4.10 below present the total cost of mortality and morbidity in the 179 geographical census tracts. The left figure presents max values while the right figure presents min values.
5. Discussion

5.1 Population characteristics and pollution level

Total damage from air pollution is a combination of several components. The first component is the pollutants concentrations in each statistical area. The other component is the number of individuals affected by pollution and especially the population at risk. While the population at risk is relatively easy to calculate, the number of individuals affected is subject to uncertainty. This uncertainty is reflected in any stage of the estimation and especially the population attributable fraction e stage as well as estimation of the economic burden of mortality and morbidity. Therefore, a confidence interval is suggested for the selected functions in this study. This is not presented in the report since the major uncertainty is related to the assumption regarding VSL vs. VOLY and the double counting assumption. Given these reservations we can deduce several conclusions regarding the spatial outcome in the HBA.

Figure 5.1 below presents the percentages of the most vulnerable population at risk in the HBA (under 15 and more than 60 years of age).
The densely populated areas are outside the municipality of Haifa. They are presented in red and include Nesher, Kiryat Ata, Neve Ganim, Shefa-ram and Rekhasim.

The areas with higher rates of population at risk are located both in the city of Haifa and outside of Haifa. In Haifa, the areas are Neve Shaanan, Neve Yosef, Savionei HaCarmel and Ramat Vizhnitz. In the Bay and the Krayot the high-rate area is Kiryat Yam. However, in order to estimate the burden of pollution both population at risk AND pollution level play an equal role. It is the combination of the two that makes a given region relatively vulnerable.

The areas most affected by pollution from industry are most of the areas in the vicinity of the Bay Area:

In the Haifa city - Neve Shaanan, Kiryat Yam and Kiryat Elyiau.

In the Bay and Krayot areas - Rekhasim, Kiryat Ata, Kiryat Haim East, and Tivon.

With regards to the mortality from pollution, it is quite similar to the above result: the areas with a higher risk of mortality are the populations near the Bay area industrial area. This may be a result of the wind flow pattern in the bay area.

In the Haifa city – Neve Shaanan, Nesher, Hadar and Kyriat Elyiau.

In the Bay and Krayot areas – Kiryat Bialik and Kiryat Ata.

The areas that appear in almost all figures (of either high pollution or high mortality) in terms of both the number of cases and the economic cost are:

In the Haifa city – Neve Shaanan, and to a lesser extent Hadar.

In the Bay and Krayot areas – Kiryat Ata, Kiryat Haim East, and to a lesser extent Rekhasim and Nesher.

The information is summarized in Table 5.1
### Table 5.1: High damage cost neighborhoods in the HBA

<table>
<thead>
<tr>
<th></th>
<th>Population</th>
<th>Population at risk</th>
<th>Damage cost (maximum values)</th>
<th>Damage cost (minimum values)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haifa city</strong></td>
<td></td>
<td>Neve Shaanan Neve Yosef Savionei HaCarmel Ramat Vizhnitz.</td>
<td>Neve Shaanan Hadar</td>
<td>Neve Shaanan Hadar Kiryat Shprintzak</td>
</tr>
<tr>
<td>The bay area</td>
<td>Nesher</td>
<td>Kiryat Yam</td>
<td>Nesher Rekhasim Kiryat Ata Kiryat Haim East</td>
<td>Nesher Rekhasim Kiryat Ata Kiryat Haim East Neve Ganim Tsur-Shalom Shfaram.</td>
</tr>
</tbody>
</table>

Note: Maximum and minimum values relate to the double counting assumption explained earlier in section....

The damage costs are presented in the two columns on the right-hand side of the table. However, if we look at areas like Nesher, Kiryat Ata, and Rekhasim, part of the problem is not necessarily the high pollution level but the presence of many residents in these areas. The neighborhood of Neve Shaanan is not classified as a high-density neighborhood but it has a higher rate of a population at risk. This suggests the need to calculate statistical area-specific mortality/morbidity rates.

As seen in figure 4.5, the highest polluted areas are the Checkpoint, Wadi-Salib and Wadi Nisnas, Ramot HaCarmel as well as the coastal areas in Haifa, Kiryat Haim west and Kiryat Yam. These areas are defined as polluted areas but are not defined as areas with high damage cost.

#### 5.2 Comparison with other studies
The total population in Israel in 2015 was 8.462 million. The population in the HBA was 0.521 million in that year - 6.16% of the total population in Israel for that year.

Lavee and Menachem, (2018) found a total cost of pollution resulting from PM and O3 in Israel to be about 7,812 million ILS in 2015. Using the relative share of residents out from the total population in the HBA, we should receive a total damage of 481 million ILS. Correcting for non-inclusion of NO2 in our study yields an estimated damage cost of 1,045 million ILS. This is more than double, compared to the relative share of the residents of the HBA.

The Ministry of the Environment carried out a study (unpublished) regarding air pollution (among other types of pollution types) in Israel. This study was based on damage cost per ton of pollutant and not pollution concentration. The total damage cost for the HBA was estimated at 1,387 million ILS. The study was based on VSL estimates only so compared to the mean value between the min. and max. values in the current study, this is slightly higher than the current result in this study, which is 1,361 million ILS.

6. Conclusions

Air pollution is a significant problem in major industrial cities, and the HBA is no exception. In this study, we estimated the monetary costs of added symptoms of morbidity and mortality in the area.

Two elements influence the total value of pollution damage: (1) Is VSL the same regardless of age? (2) Since the dose-response function does not consider the fact that other pollutants operate in the background and one can have a disease caused by probably one symptom only (and die only once), there is a possibility of double counting.

We addressed element (1) by considering both the VSL (not age adjusted) and VOLY. To address element (2) we considered two extreme cases. One is where there is no double counting effect, while the other case is where every symptom is counted exactly once. The former case indicates a simple summation of all the effects while the latter indicates a full double counting. To deal with that, we considered the highest incidence rate in each statistical cell.

Since air pollution damage is a combination of pollution level and the number of individuals living in the given area, it does not mirror the map in Figure 4.6. However, a few conclusions can be made from looking at the region as a whole.
The number of individuals who died from air pollution is estimated to between 94 and 183. This is based on the assumptions about double counting discussed above. The number of years lost ranges from 2,657 and 4,908, again according to the double counting assumptions. Two new cases of Leukemia and two new cases of lung cancer are detected. The total cost of air pollution damage is shown in Table 6.1

Table 6.1: Total monetary cost for the HBA (in ILS)

<table>
<thead>
<tr>
<th></th>
<th>VOLY</th>
<th>VSL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum estimation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>due to double</td>
<td>561,246,375</td>
<td>1,113,027,948</td>
</tr>
<tr>
<td>counting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No double counting</td>
<td>685,451,538</td>
<td>1,360,674,018</td>
</tr>
<tr>
<td>Assumption</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Most of the pollution is due to transportation. If we look at mortality, the results are given in Table 6.2 below.

Table 6.2: Mortality early cases due to air pollution divided by source for minimum and maximum effects (projected cases for 2015)

<table>
<thead>
<tr>
<th></th>
<th>Industrial</th>
<th>Transportation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum estimation</td>
<td>7.66</td>
<td>72.75</td>
</tr>
<tr>
<td>due to double</td>
<td></td>
<td></td>
</tr>
<tr>
<td>counting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No double counting</td>
<td>14.36</td>
<td>87.33</td>
</tr>
<tr>
<td>assumption</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Taking the mean value, we observe 11 and 80 early death cases for industry and transportation, respectively.

Table 6.3 below shows the problematic neighborhoods and towns in the HBA damage costs and population at risk rates.
Table 6.3: High damage cost neighborhoods in the HBA

<table>
<thead>
<tr>
<th></th>
<th>Population</th>
<th>Population at risk</th>
<th>Damage cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haifa city</td>
<td></td>
<td>Neve Shaanan</td>
<td>Neve Shaanan, Hadar</td>
</tr>
<tr>
<td>The Bay area</td>
<td>Neve Shaanan, Nesher, Kiryat Ata, Rekhasim</td>
<td></td>
<td>Nesher, Rekhasim, Kiryat Ata, Kiryat Haim East</td>
</tr>
</tbody>
</table>

Neighborhoods such as Neve Shaanan suffer from high damage from pollution, but part of the reason is the density of population at risk. The towns of Nesher, Rekhasim and Kiryat Ata suffer from high damage from air pollution partially because they are highly dense areas. However, Hadar neighborhood as well, as Kiryat Haim East, suffer from high pollution damage without being classified as highly dense or having a high rate of population at risk.

7. Potential Contribution for Israel

This study is one of the first studies that has taken an interdisciplinary approach, using several air pollution models, epidemiological responses, and the monetary valuation of the consequences. This was applied to one of the most polluted areas in Israel - the Haifa Bay Area. The analysis was based on dividing the Bay area into 179 cells, 163 of which were populated. A previous study (Kulhánová et al. 2018) demonstrated that the attributable burden of disease depends on the spatial resolution of the exposure, the assumptions made regarding the dose-response function and the reference level of the pollutants. Kulhánová (2018) demonstrated that decreasing the spatial distribution of exposure and the spatial resolution of the population weighted density data, underestimated the number of attributable cases. Applying the fine scale method developed and demonstrated in the current study on a national scale, could improve the precision of the evaluation of the real burden of air pollution.
In addition, the results can be thought of as a ‘crystal ball’, that may serve policy makers in analyzing different strategies to reduce the negative consequences of damage? cost more effectively. This can be done easily by changing the relevant parameters (year, pollution levels, etc.) and analyzing the implications of that change. For example, the current study uses data from 2015. More recent data may reveal changes because the transport network has changed (e.g., the Carmel tunnels and road 22). In addition, several polluting companies invested in pollution reduction equipment and this parameter change will most likely have implications on the damage costs.

Changes like this may serve to trace improvements (or worsening) in air pollution levels over time. It can also set the foundation for cost-benefit analysis. More importantly, such models may serve as a tool for ex-ante simulations of suggested projects to compare their benefits vs. costs.

Air pollution damage is a function of the amount of pollution in a given statistical area but also a function of the number of people living in the area. The number of people may eventually change over time, which gives rise to changes in the damage from air pollution. This may include natural immigration or migration, as well as the addition of new neighborhoods.

8. Recommendations for Future Research

The current study has several limitations, and future research may resolve a major part of these issues.

i. The current study estimates local emission. It is an underestimation of current pollution levels from several reasons and a future study may relate to several more issues such as: swell into some of the issues.

   1. The dataset provided by the MOE on emissions should be expanded in order to include missing data such as emission from Haifa port and the trains, home heating using wood, or burning of agricultural waste.

   2. The transportation data sources (year/s) were not compatible with the other data in our study, which used data from 201?. We have current data (year) on emissions but no current data on roads distribution. Future
research may use the 2018 data set, which is now readily available at the Ministry of the Environment’s database (link/ref).

3. The data available on the IR of specific morbidities is limited, at least for the same parameter combination that we were working on (age and sex categories). Therefore, in future research we intend to rely on figures from the literature.

ii. We had to deal with double counting of symptoms when aggregating different pollutants. Therefore, we provided two endpoint cases, as discussed previously. Future research may narrow the range by providing estimates for more intermediate scenarios.

iii. We did not have information on the severity of the diseases. Adding this information could possibly shed more light on this issue.

iv. Our results are biased towards transportation at the expense of industrial pollution. Future research may use some agreed-upon assumptions to "reallocate" residents to the industrial areas where currently there is zero population (even though many people are working there and the fact that there is significant economic activity during daytime.

9. Summary

This study aimed to evaluate the economic valuation of local anthropogenic emissions of air pollution in the Haifa Bay Area. We used three main methodologies to reach our goal. The first one was estimating the air pollution ambient concentration for every census tract in the studied area. The second is to determine how it will affect mortality and morbidity, while the third is monetarily valuing these symptoms. Dose–response functions were estimated based on a meta-analysis from the literature. We also mapped the pollutants that were relevant to estimate the health endpoints and their economic value. These were O₃, PM₂.₅, NO₂, and Benzene.

We have also estimated the value of several symptoms and the value of statistical life as well as a year life lost.

The results are presented in tables and maps. The maps were produced using GIS, which was also the base for extracting the number of people living in each of the 179 statistical cells, as
well as their age distribution. These were further used to derive the relative weights between populations at risk and those not at risk (under 14 years old and above 60 years old are defined as at-risk populations).

The three ingredients together, with the information received from the GIS section, was the basis to achieve the monetary value of air pollution. This was further reviewed by different areas, different pollutants, and different pollution sectors (mobile vs. stationary). The evaluation methods developed in this current study can be used in future studies. More specifically, they can be used to produce updated valuations for updated pollution or population data that will be available.
References:


Cha, Y. J. (2018). The economic burden of stroke based on South Korea’s national health insurance claims database. *International journal of health policy and management, 7*(10), 904.


## APPENDIX A: INDUSTRIAL SOURCES INVENTORY - HAIFA REGION - 2015

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### APPENDIX B: Summary of the CRFs that were summarized and the population at risk based on recent literature (selected pairs are indicated in bold and presented also in table 4.2).

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<th>Outcome</th>
<th>ICD-9/ICD-10 codes</th>
<th>Exposure, Per unit</th>
<th>Short/long term</th>
<th>Study design</th>
<th>Age group</th>
<th>Sex</th>
<th>Reference for risk estimate</th>
<th>Concentration-response function (CRFs)</th>
<th>Risk Estimate</th>
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<td>Type 2 Diabetes mellitus</td>
<td>ICD-10 code: E11 ICD-9: 250</td>
<td>PM2.5, annual Mean, RR (95% CI), per 10 μg/m³</td>
<td>Long term</td>
<td>Meta-analysis</td>
<td>Adults&gt;21</td>
<td>Both</td>
<td>(He et al. 2017)</td>
<td>RR=1.25 (95% CI: 1.10–1.43)</td>
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<td>Type 2 Diabetes mellitus</td>
<td>ICD-10 code: E11 ICD-9: 250</td>
<td>NO2, (After conversion to the estimate of NOx to its NO2 corresponding value), annual mean, Hazard Ratio (HR, 95% CI), per 10 μg/m³</td>
<td>Long term</td>
<td>Meta-analysis</td>
<td>Adults&gt;21</td>
<td>Both</td>
<td>(Balti et al. 2014)</td>
<td>Hazard Ratio [HR]: HR=1.13 (95% CI: 1.04–1.22)</td>
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<td>Type 2 Diabetes mellitus (mortality and morbidity together)</td>
<td>ICD-10 code: E11 ICD-9: 250</td>
<td>O3, summer average ozone, daily mean, per 10-μg/m³ increase</td>
<td>Short and long-term, combined</td>
<td>Meta-analysis</td>
<td>Adults&gt;21</td>
<td>Both</td>
<td>(Janghorbani, Momeni, and Mansourian 2014)</td>
<td>OR=1.07 (95% CI:1.05–1.09)</td>
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<td>Term Low birth weight</td>
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<td>PM2.5, during pregnancy, OR (95% CI), per 5 μg/m³</td>
<td>Long term, during pregnancy</td>
<td>Pooled effect estimate, Europe</td>
<td>20-49</td>
<td>Female</td>
<td>(Pedersen et al. 2013)</td>
<td>OR=1.18 (95% CI:1.06–1.33)</td>
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<td>Pooled effect estimate, Europe</td>
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<td>Female</td>
<td>(Pedersen et al. 2013)</td>
<td>OR=1.09 (95% CI:1.00–1.09)</td>
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<td>Meta-analysis</td>
<td>20-49</td>
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<td>WHO, Meta-analysis</td>
<td>Adults, Age 30+ (Kulhánová et al. 2018)</td>
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<td>(Hamra et al. 2014)</td>
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<td>Meta-analysis</td>
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<td>(Huang et al. 2017)</td>
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<td>Pooled effect estimate, Europe</td>
<td>Adults, Age 30+ (Kulhánová et al. 2018)</td>
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<td>[Raaschou-Nielsen et al. 2013]</td>
<td>HR= 1.18 (95% CI: 0.96–1.46)</td>
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<td>Adults, Age 30+ (Kulhánová et al. 2018)</td>
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<td>Meta-analysis</td>
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<td>Adults, &lt;60 years</td>
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<td>(Stafoggia et al. 2014)</td>
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<td>Both</td>
<td>(Cesaroni)</td>
<td>HR=1.13 (95% CI: 0.98–1.00)</td>
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<td></td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Condition</th>
<th>CRF</th>
<th>Air Pollutants</th>
<th>Time Period</th>
<th>Analysis Type</th>
<th>Age of Subjects</th>
<th>Study Or Range of RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic Heart Disease</td>
<td>IHD: I20-I25</td>
<td>CO (per 1ppm) SO2 (per 10 ppb) NO2 (per 10 ppb) PM2.5 (per 10 μg/m3)</td>
<td>Short term</td>
<td>Meta-analysis</td>
<td>Adults ≥ 20 years</td>
<td>Both (Shah et al. 2013) RR=1.0352 (1.0252-1.0454) RR=1.0236 (1.0135-1.0338) RR=1.017 (1.0125-1.0216) RR= 1.0212 (1.0142-1.0282)</td>
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<tr>
<td>Heart failure hospitalization or death</td>
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<tr>
<td>Lung Morbidity</td>
<td>COPD: J44</td>
<td>PM2.5 (per 10 μg/m3) NO2 (per 10 μg/m3) SO2 (per 10 μg/m3)</td>
<td>Short term, RR, daily mean (cumulative lags) No long term RR from meta-analysis</td>
<td>Meta-analysis</td>
<td>Adults, (age not stated, when possible RR were derived from 65+)</td>
<td>Both (DeVries, Kriebel, and Sama 2017) RR= 1.025 (95% CI: 1.016–1.034) RR=1.042 (1.025–1.060) RR=1.021 (1.017–1.035)</td>
</tr>
<tr>
<td>COPD Emergency department visit and hospitalization (Data on Mortality also available)</td>
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<tr>
<td>Asthma incidence or lifetime prevalence from birth until 18 years old</td>
<td>J45.901</td>
<td>NO2 (per 4 μg/m3) NOx (per 30 μg/m3) PM2.5 (per 1 μg/m3) Or other units (Anenberg et al. 2018): NO2 (per 10 PPB) PM2.5 (per 10 μg/m3)</td>
<td>Long-term, Annual, cumulative exposure</td>
<td>Meta-analysis</td>
<td>Children, age &lt;18</td>
<td>Both (Kreis et al. 2017) RR=1.05 (1.02, 1.07) RR= 1.48 (0.89, 2.45) RR= 1.03 (1.01, 1.05)</td>
</tr>
<tr>
<td>Asthma emergency room visits and hospital admissions</td>
<td>J45.901</td>
<td>O3, Per 10 ppb, all lags</td>
<td>RR, Short term, daily mean</td>
<td>Meta-analysis</td>
<td>Children, age &lt;18</td>
<td>Both (Zheng et al. 2015) RR=1.03 (1.02–1.04)</td>
</tr>
<tr>
<td>ALRI-Acute lower respiratory infection</td>
<td>ICD 10: J10-J22</td>
<td>PM2.5 (per 10 μg/m3)</td>
<td>Long term, (subchronic and chronic) effects studies</td>
<td>Meta-analysis</td>
<td>Age &lt;5</td>
<td>Both (Mehta et al. 2013) RR=1.12(1.03-1.30)</td>
</tr>
</tbody>
</table>

* Bold text are the CRFs that were used based on the selection criteria mentioned above