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JSM Environmental Science & Ecology

Research Article

Characterizing the Health Risks Attributable to Tropospheric Ozone in Finland

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Submitted: 10 February, 2016

Accepted: 18 March, 2016

Published: 21 March, 2016

ISSN: 2333-7141

Keywords
- Health impact assessment
- Premature mortality
- Morbidity
- Air quality
- Exposures

Abstract

Ozone has been the second most important air pollutant for which environmental health risk estimates have been presented. In earlier estimates only mortality and subsequent 'harvesting' deaths due to acute exposure have been accounted for. Now for the first time World Health Organization working group recommended concentration response functions for mortality caused by long-term exposure. This work aimed at comparing the magnitude of environmental burden of disease for acute and premature mortality for ozone. We estimated the exposures to ozone as population weighted ambient concentrations using simple statistical approach and data from the air quality monitoring network in Finland. The environmental burden of disease was estimated using population attributable fraction methods and WHO Global Health Estimates background burden of disease. The results showed that chronic mortality substantially increases the environmental burden of disease attributable to ozone exposures from previous estimated burden of 240 disability adjusted life-years (DALY) to 450-1100 DALY in 2013. Even in a Nordic country with traditionally low exposure levels such as Finland the health losses due to ozone are significant.

ABBREVIATIONS

AI: Attributable Incidence; BOD: Burden Of Disease; CVD: Cardiovascular Disease; C-R: Concentration Response; DALY: Disability-Adjusted Life Year; EBD: Environmental Burden Of Disease; LRS: Lower Respiratory Symptoms; MRAD: Minor Restricted Activity Days; PAF: Population Attributable Fraction; $PM_{2.5}$: Particulate matter smaller than 2.5 μm in aerodynamic diameter; RR: Relative Risk; SOMO35: Annual sum of 8-hour running average ozone levels over 35 ppb (70 μg m^{-3}); UR: Unit Risk; YLL: Years of Life Lost (due to premature mortality); YLD: Years Lived with Disability

INTRODUCTION

Most health impact assessments for ambient air quality tend to focus on particulate matter and ozone. In Europe the first widely used assessment was conducted as part of the Clean Air for Europe (CAFE) –programme for year 2005. Six health endpoints were estimated for ozone and eleven for fine particulate matter ($PM_{2.5}$) [1]. The ozone health impacts were dominated by acute mortality, estimated to be 21 000 deaths in EU and 58 deaths in Finland in year 2000. In comparison, for fine particles the corresponding figures were 348 000 and 1 270, respectively. At that time epidemiological studies were able to associate ozone with mortality only using time-series approaches, thus estimating the ozone impacts only for acute mortality and estimated as one year of life lost per case. The $O_3/PM_{2.5}$ mortality ratio for Finland was 58/1 270=4.5%, but the same ratio for years of life lost only 58/13 840 = 0.42%. In contradiction to the ozone estimates, for $PM_{2.5}$ premature deaths clearly dominate the number of total deaths with about 10 years of life lost due to every $PM_{2.5}$ attributable death [2,3].

These CAFE-estimates were calculated using SOMO35-indicator as the health relevant annual ozone exposure metric [4].SOMO35 is the annual sum of the maximum daily 8-hour concentrations that are exceeding 35 ppb (70 μg m^{-3}).

The previous mortality figures demonstrate well that the numbers of deaths are not always completely comparable. The same difficulty applies to any estimates consisting of a number of cases. To allow for comparability across different types of endpoints ranging from asthma or cough days to mortality, World Health Organization developed together with World Bank and Harvard University a new approach. This burden of disease (BoD) methodology measures health gaps as opposed to health expectancies. It measures the difference between a

current situation and an ideal or alternative situation [5]. BoD is quantified using disability adjusted life years (DALY), which apply disability weights to make morbidity, quantified as years lived with disability (YLD), comparable with years of life lost (YLL) due to premature death (eq 1).

$$BoD = YLL + YLD \quad (1)$$

Burden of disease (BoD) attributable to environmental risk factors (EBD) such as air pollution can be calculated from background burden of disease using the estimated population attributable fraction (PAF):

$$EBD = PAF \times BoD \quad (2)$$

World Health Organization maintains a global database of national background burden of disease. The most recent Global Health Estimates dataset covers year 2012 [6].

In this paper we (i) summarize the previous health impact assessments conducted for ozone including Finland, (ii) discuss the reasons for the differences and the merits and weaknesses of various approaches, as well as (iii) present the original research results from new environmental burden of disease estimates for ozone in Finland using the latest exposure and background health data. Specifically we (iv) show the sensitivity of the burden of disease estimates to methodological choices representing model uncertainty in the assessment.

MATERIALS AND METHODS

A literature review was conducted to identify the most important previous health impact assessments for ozone covering Finland. Based on the European Environmental Agency (EEA) reports and the projects where the authors had previously participated and studies cited therein, four previous studies were identified that presented ozone health impact estimates for Finland (Table 1). Three identified studies presented estimates for year 2005 (TSAP, SETURI, EBoDE), and one for 2012 (EEA).

As part of the current work we calculated an updated estimate for year 2013, accounting for changes in exposures and in population size and health status (ISTE 1). In addition, for comparison purposes we calculated here two additional estimates. ISTE 2 calculation was conducted using WHO working group recommendation on using chronic non-violent mortality as the main endpoint and ISTE3 using the acute mortality that was used in all of the year 2005 assessments.

Table 1: Inclusion of health end-points in various assessments.

Project	Target year	C-R id (Table2)	Reference
TSAP	2005	n/a	[17]
SETURI	2005	1, 2, 3, 4	[18]
EBoDE	2005	1, 2, 3, 4	[2]
ISTE 1	2013	6, 7, 8	[20]
ISTE 2	2013	5, 6	[14]
ISTE 3[a]	2013	1, 2, 3, 9	–
EEA	2012	n/a	[19]

[a]Acute mortality estimates calculated here for comparison only

For the new ISTE1-3 calculations we collected air quality monitoring data from 23 monitoring stations available for ozone in 2013. Population weighted outdoor ozone levels were calculated by combining population data from 313 municipalities and using two alternative complementary simple modelling approaches. First, an allocation method was used by choosing representative monitoring stations for each municipality. Second, a simple regression model was developed accounting for the community size and inverse distance from Central Europe as an indicator of long-range transportation of ozone. The results were very similar, the annual mean ranging from 55.2 to 55.7 μg m^{-3}.

For the health impact calculations the hourly ozone times eries data were used to calculate annual SOMO35 levels. Earlier estimates for SOMO35 in Finland in 2005 were 2 580 μg $m^{-3}$for the population weighted ozone exposures by the European Topic Centre on Air and Climate Change (ETC/ACC) using Air Base data and air quality maps [7]. The more recent updates vary from year to year and were 2 050 and 1 650 μg m^{-3} for 2011 and 2012, respectively [8,9]. Our population weighted estimate from the Finnish monitoring network with 23 stations for year 2013 was 1 940 μg m^{-3} [10]. For the health impact calculations we averaged WHO estimate 1 320 μg m^{-3}, EEA estimate 1 650 μg m^{-3} and the national estimate to reduce model specific uncertainties, yielding SOMO35 value of 1 640 μg m^{-3}.

The general methodology for the environmental burden of disease (EBD) calculations follows the Comparative Risk Assessment Approach [11,12]. To estimate the morbidity component from years lived with disabilities (YLD), the number of disability cases (n) is multiplied by the average duration of the disease (L, in years) and a disease specific disability weight (DW) (eq 2):

$$YLD = n \times DW \times L \quad (3)$$

Years of life lost (YLL) due to premature mortality is calculated as the difference between the standard life expectancy at the age of death and the age of death (eq 3).

$$YLL = n \times L \quad (4)$$

Where n = number of deaths in a given age category and L = remaining years to standard life expectancy at age of death (in years).

The fraction of disease caused by the ozone exposure is estimated by calculating the population attributable fraction (PAF) as (eq 4):

$$PAF = \frac{f \times (RR - 1)}{f \times (RR - 1) + 1} \quad (5)$$

where f is the fraction of population exposed to a given factor and RR is the relative risk of the exposed population. The relative risk at the current exposure level (E) can be estimated from epidemiological relative risk (RR°) expressed per a standard exposure increment (e.g. 10 μg m^{-3}) [2]:

$$RR = \exp(\beta \ln RR^{\circ}) = RR^{\circ \frac{E}{\Delta}} \quad (6)$$

For ozone, exposure data and a relative risk derived from

epidemiological data are used to calculate the population attributable fractions (PAF) for total non-violent and cardiovascular and respiratory cause specific mortality. For health end-points with unit risk exposure functions, PAF is derived indirectly. The unit risk and exposure information are used to estimate the attributable incidence (AI). The PAF is indirectly estimated from dividing the total incidence by this AI. Subsequently, the PAF is applied to the WHO burden of disease data for both YLL and YLD. In cases where no appropriate burden of disease data were available from the WHO database, the EBD was calculated by multiplying the estimated number of attributable cases with WHO disability weights (DW) and corresponding estimates of disease duration or years lost due to premature death (L) [2].

The earlier estimates compared here were based on burden of disease figures were extracted from the WHO global burden of disease database for year 2004 [13]. These data included number of deaths and BoD expressed as DALYs for a comprehensive list of different diseases. WHO updated the methodologies and estimates recently for year 2012 [6]. These are used to calculate the updated EBD estimates presented here. In the updated estimates two major methodological changes were applied compared to earlier ones: WHO discontinued discounting and age weighting and instead of previous incidence based estimation prevalence was used.

In the literature review we identified a relatively large number of health endpoints proposed for ozone in health impact assessments. As the main update of the earlier estimations for Finland, we performed the health impact assessment using the WHO working group recommendations published recently [14] (Table 2).

The health endpoints corresponding concentration-response relationships presented in Table 2 were used in various combination in different assessments (Table 1; for the EEA assessment we were not able to identified the used functions due to omissions in reporting).

RESULTS AND DISCUSSION

Annual ozone levels in Finland are low in comparison to countries in other climatological regions (mean 55 μg m^{-3} in 2013). Due to the summer season and daily variability, the daily maximum 8-hour averages used to calculate SOMO35 exceeded the threshold level of 70 μg m^{-3} on the average by 4-5 μg m^{-3}, yielding population weighted SOMO35 exposure levels of 1 600–1 900 μg m^{-3}. Why our monitoring network based estimates are higher than those from WHO and EEA remained unclear. There was no indication of differences for long-range transport contribution to the ozone levels in Finland, with levels being equal in rural areas in southern Finland and Lapland. Urbanozone levels were slightly lower than the rural ones. We do not see a clear reason why our estimate might be an overestimation.

Besides exposure levels, there have been several independent methodological changes in the assessments that affect the magnitude of the estimates substantially. The number of attributable mortality was 90-100 deaths for 2005. The corresponding figures for most of the assessments for 2012-2013 were 60-62 deaths (Table 3), representing merely the direct effect of the decrease in the exposures. However, when looking at the other indicators we see that the years of life lost due to ozone mortality increased from values around 90 for 2005 to 700-1 085 in 2012-2013. This increase is attributable to the inclusion of chronic mortality in the estimates due to more comprehensive scientific evidence accumulated during this period. Many of the European assessments did not pay attention to the morbidity component and the data is sparser. Also the trend in the morbidity estimates is not so clear (Table 3).

The oldest estimates for overall burden in 2005 were around 250 DALY, but the estimates for 2012-2013 range from a mere 150 to 1 100 DALY, representing a substantially larger

Table 2: Concentration-response functions proposed for ozone health impact assessments.

				C-R function (1 μg m^{-3})$^{-1}$			
C-R id	**Endpoint**	**Agegroup**	**Type of function**	**Estimate**	**Lower 95% CI**	**Upper 95% CI**	**Ref.**
1	MRAD	18 - 64	UR	0.0115	0.0044	0.0200	[15,2]
2	Coughdays	5 - 14	UR	0.0930	0.0190	0.2200	[15,2]
3	LRS days (excl. cough)	5 - 14	UR	0.0160	-0.043	0.080	[15,2]
4	Acutemortality (non-violent)	>30	RR	1.0003	1.0001	1.000	[16,2]
5	Mortality (natural)	All	RR	1.0003	1.0001	1.0004	[14]
6	MRAD	All	RR	1.0015	1.0006	1.0025	[14]
7	CVD	All	RR	1.0003	1.0001	1.0004	[14][a]
8	Respiratorydiseases and infections	All	RR	1.0003	1.0001	1.0004	[14][a]
9	Acutemortality (natural)	All	RR	1.0003	1.0001	1.0004	[14][b]

Abbreviations: C-R Function: Concentration-Response Function: Est.: Central Estimate: CI: Confidence Interval: MRAD: Minor Restricted Activity Days: LRS: Lower Respiratory Symptoms: UR: Unit Risk: RR: Relative Risk

[a]Natural mortality function (5) was used for calculating cause-specific mortality

[b]Natural mortality function (5) was used for calculating acute mortality by adding disability weight 1.0 and duration of disease 1.0

Table 3: Ozone burden of disease estimates from various studies in comparison with the current estimates.

Project	Discountrate	Type of mortality[a]	Targertyear	Deaths	YLL	YLD	DALY	Ref.
TSAP report	n/a	AA	2005	99				[17]
Seturi	3 %	AA	2005	90	90	141	232	[18]
EBoDE	3 %	AA	2005	94	94	151	245	[2]
ISTE 1	0 %	CC	2013	29[c]	442	129	571	[20]
ISTE 2	0 %	CA	2013	62	1 085	32	1 117	[14]
ISTE 3[b]	0 %	AA	2013	62	62	90	152	–
EEA report	n/a	CA	2012	60	700	-	700	[19]

[a]AA acute all cause mortality; CA chronic all cause mortality; CC chronic cause specific mortality
[b]Calculated here for comparison only; includes acute mortality effects similar to the earlier estimates for year 2005.
[c]Includes cardiovascular and respiratory mortality instead of total non-violent mortality.

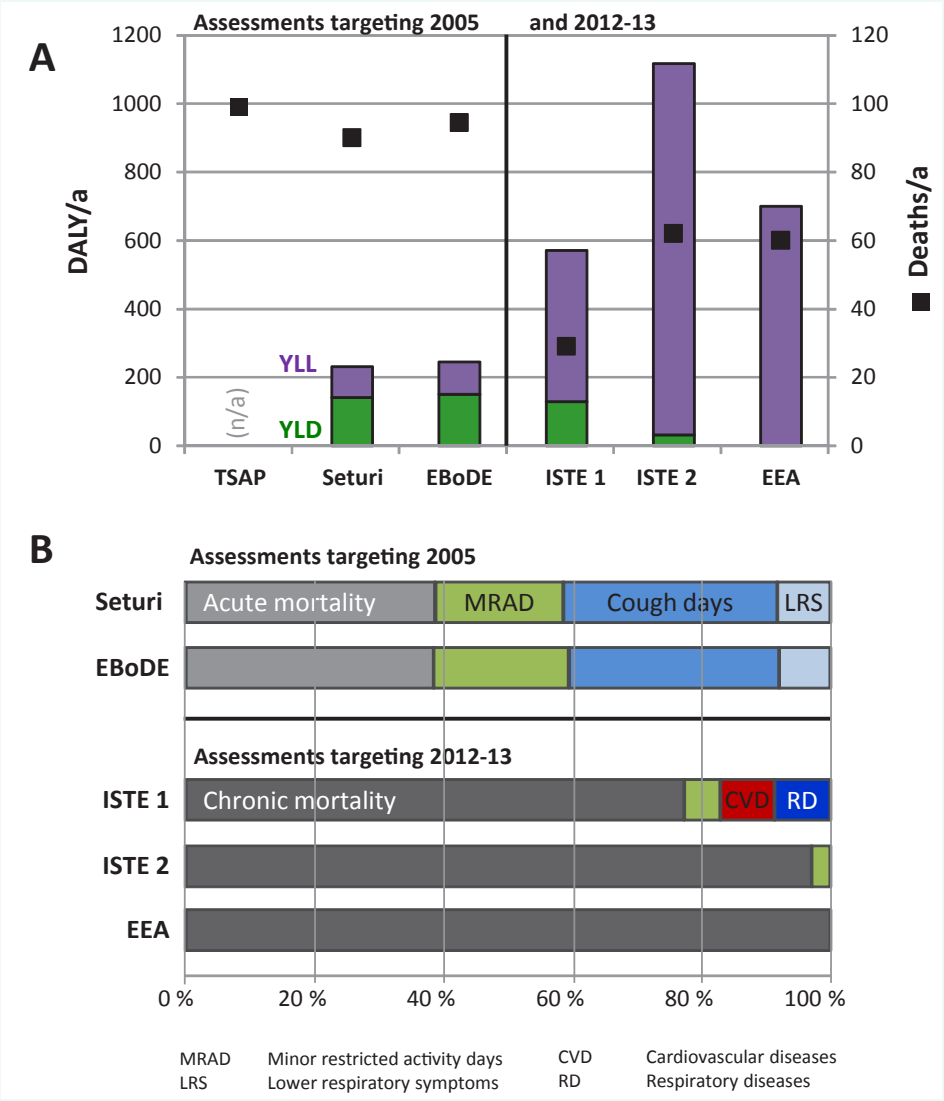

Figure 1 A. Comparison of ozone disease burden estimates from different projects representing parallel evolution of methods and input data and B. relative contribution of various health endpoints to them. In the earlier evaluations mortality accounted for less than half of the DALYs (SETURI, EBoDE), whereas after inclusion of chronic mortality as endpoint mortality makes up for clearly more than half of the total burden (ISTE1-2, EEA).

methodological uncertainty. Part of these differences are due to the choice of using cause-specific mortality in the official ISTE estimates [20], while following the WHO working group recommendation leads to substantially lower estimates (while producing at the same time larger estimates for YLL) [14] (Table 3, Figure 1)

Originally the WHO methodologies included age weighting and discounting as improving economical interpretation of the burden figures. However, recently the international scientific community chose to drop these and to present non-discounted results without any age weighting. All the assessments conducted since 2012 have adopted this approach. Discounting did not affect much ozone estimates due to the fact that earlier only acute mortality, not affected by discounting, was assessed for ozone. For the new chronic mortality estimates it would play some role, but we did not specifically address that as the international assessments by WHO and IHME have also stopped using discounting. However, when comparing ozone figures to particulate matter and other risk factors, the decrease in these estimates due to discounting (up to 50%) lead to a relative increase in ozone impacts.

While the exposure levels to ozone are relatively low in Finland, the burden can be considered significant: There are around 60 deaths, leading to the loss of 400-1 000 life years per year, even though the model choices and uncertainty is relatively large here. In contrast with other air pollutants, rural ozone levels are practically constant over Finland from Southern coast to Lapland (data not shown).

CONCLUSION

Public health impacts caused by ozone have been characterized using a number of various metrics including number of caused deaths, years of life lost (YLL), years lived with disability (YLD), symptom days associated with various symptoms such as cough, lower respiratory symptoms, and minor restricted activity days. Such metrics are not readily useful for prioritization. I integrating impacts into a harmonized measure such as disability adjusted life year (DALY), allows for comparing the overall burden across different types of end points and exposures.

When ozone and particulate matter ($PM_{2.5}$) impacts are quantified as number of attributable deaths, ozone accounts for roughly 3% compared to $PM_{2.5}$ deaths. In the earlier assessments where only acute mortality was accounted for, the corresponding ratio of years of life lost was only 0.5%.

Recent systematic review by a WHO working group found sufficient evidence on ozone effects on chronic mortality, which according to our calculations now roughly doubles the risk estimates. In comparison with PM2.5 impacts without discounting the ratio between the risks remains approximately at the previous level.

Harmonization of health metrics used in health impact assessments would improve comparability of data across studies. Bias to use mortality as health loss descriptor leads to underestimation of morbidity impacts and may be poorly justifiable from mechanistic toxicology point of view.

ACKNOWLEDGEMENTS

This work was funded by the Ministry of Environment (YM80/481/2015), the Ministry of Social Affairs and Health (005/HTO/EER/2015 and STM/1069/2015), NordForsk under the Nordic Programme on Health and Welfare project #75007 (NordicWelfAir), Academy of Finland project BATMAN (285672) and intramural funding by the National Institute for Health and Welfare,

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Cite this article

Hänninen O, Lehtomäki H, Korhonen A, Rumrich I, Asikainen A (2016) Characterizing the Health Risks Attributable to Tropospheric Ozone in Finland. JSM Environ Sci Ecol 4(1): 1023.