

## 22.20 Morbidity, Mortality and Years of Life Lost due to poor air quality

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**Author/s and affiliations:** Laura Wendling<sup>1</sup>, Ville Rinta-Hiiri<sup>1</sup>, Maria Dubovik<sup>1</sup>, Arto Laikari<sup>1</sup>, Johannes Jermakka<sup>1</sup>, Zarrin Fatima<sup>1</sup>, Malin zu-Castell Rüdenhausen<sup>1</sup>, Ana Ascenso<sup>2</sup>, Ana Isabel Miranda<sup>2</sup>, Peter Roebeling<sup>2</sup>, Ricardo Martins<sup>2</sup>, Rita Mendonça<sup>2</sup>

<sup>1</sup> VTT Technical Research Centre Ltd, P.O. Box 1000 FI-02044 VTT, Finland

<sup>2</sup> CESAM – Department of Environment and Planning, University of Aveiro, Campus Universitário de Santiago, 3810-193 Aveiro, Portugal

Morbidity, Mortality and Years of Life Lost due to poor air quality	Air Quality Health and Wellbeing
<b>Description and justification</b>	Air pollution has been related to numerous adverse health effects, typically expressed in several morbidity and mortality endpoints (see Costa et al., 2014). In particular, an increasing amount of epidemiological and clinical studies observes that exposure to air pollution is associated with increased risk of heart disease, myocardial infarction and stroke as well as lung cancer (e.g., Costa et al., 2014). While the impact of these health effects may appear low at the individual level, the overall public-health burden is sizable as the entire population is exposed (Pascal et al., 2011).
<b>Definition</b>	Reduction in years of life (y) due to premature mortality in comparison with standard life expectancy (Morbidity): Long-term (annual) incidence of chronic bronchitis due to poor air quality calculated using atmospheric NO <sub>2</sub> and PM <sub>10</sub> data (Mortality): Long-term (annual) incidence of mortality due to poor air quality calculated using atmospheric PM <sub>2.5</sub> , PM <sub>10</sub> , O <sub>3</sub> and NO <sub>2</sub> data
<b>Strengths and weaknesses</b>	+ The indicator is easy to define - The method needs a lot of input data
<b>Measurement procedure and tool</b>	The general approach in health impact assessment is to use exposure-response functions, linking the concentration of pollutants to which the population is exposed to the number of health events occurring in that population (Costa et al., 2014; Silveira et al., 2016). Therefore, the following aspects are usually considered: i) involved pollutants and their air concentration levels, ii) health indicators analysed in terms of morbidity and mortality, iii) affected age groups, and iv) exposure time. The health response is usually calculated by:

$$\Delta R = IR \times CRF \times \Delta C \times Pop$$

Where,

- $\Delta R$  is the response as a result of the number of the unfavourable implications (cases, days or episodes) over all health indicators;
- IR is the baseline morbidity/mortality annual rate (%); this information is available in the national statistical institute of each country;
- CRF is the correlation coefficient between the pollutant concentration variation and the probability of experiencing a specific health indicator (%; i.e., Relative Risk (RR) associated with a concentration change of  $1 \mu\text{g m}^{-3}$ );
- $\Delta C$  indicates the change in the pollutant concentration ( $\mu\text{g m}^{-3}$ ) after adoption of the adaptation/mitigation measure;
- Pop is the population units per age group exposed to pollution.

Morbidity (chronic bronchitis) due to poor air quality is calculated using  $\text{NO}_2$  and  $\text{PM}_{10}$  to determine CRF and  $\Delta C$  in the preceding equation.

Mortality, assessed as total mortality, is calculated using  $\text{PM}_{10}$ ,  $\text{PM}_{2.5}$ ,  $\text{O}_3$  and  $\text{NO}_2$  to determine CRF and  $\Delta C$  in the preceding equation.

Both morbidity and mortality are based on long-term (annual) effects (Table). Where air quality data are derived from WRF-Chem results can be calculated on a daily/weekly/monthly/annual basis at the grid, neighbourhood or city scale.

*Table. Air pollutant health indicators (WHO, 2013)*

Pollutant	Health outcome	Age group
$\text{PM}_{10}$	Chronic bronchitis (incidence)	>18 y
	Chronic bronchitis (prevalence)	6-18 y
	Total mortality	<1 y
		>30 y

	PM <sub>2.5</sub>	Total mortality	>30 y
	NO <sub>2</sub>	Total mortality	>30 y
		Prevalence of bronchitic symptoms in asthmatic children	5–14 y
	O <sub>3</sub> (April-September)	Total mortality (respiratory diseases)	>30 y
	<p>Years of life lost (YLL) is an often-used health indicator, and refers to the total number of years of reduced life due to premature mortality. Using the mortality indicator, the YLL can be calculated as the number of deaths multiplied by a standard life expectancy at the age at which death occurs (see Gardner &amp; Sanborn, 1990).</p>		
<b>Scale of measurement</b>	Street to metropolitan scale		
<b>Data source</b>			
<b>Required data</b>	i) involved pollutants and their air concentration levels, ii) health indicators analysed in terms of morbidity and mortality, iii) affected age groups, and iv) exposure time		
<b>Data input type</b>	Quantitative		
<b>Data collection frequency</b>	Daily, weekly, monthly or annually		
<b>Level of expertise required</b>	Moderate		
<b>Synergies with other indicators</b>	Other indicators in the <i>Air quality</i> indicator group		
<b>Connection with SDGs</b>	SDG 3 Good health and well-being, SDG 15 Life on land		
<b>Opportunities for participatory data collection</b>	No opportunities identified		
<b>Additional information</b>			
<b>References</b>	Costa, S., Ferreira, J., Silveira, C., Costa, C., Lopes, D., Relvas, H., ... Teixeira, J.P. (2014). Integrating Health on Air Quality Assessment-Review Report on Health Risks of Two Major European Outdoor Air Pollutants: PM and NO <sub>2</sub> . Journal of		

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## 22.21 Prevalence and incidence of autoimmune diseases

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**Author/s and affiliations:** Adina Dumitru<sup>1</sup>, Catalina Young<sup>2</sup>, Irina Macsinga<sup>2</sup>

<sup>1</sup> *University of A Coruña, Spain*

<sup>2</sup> *West University of Timisoara, Romania*

Prevalence and incidence of autoimmune diseases	Health and Wellbeing
<p><b>Description and justification</b></p>	<p>Numerous authors stress the relevance of immune-regulatory mechanisms in the manifestation of the generally expected beneficial effects of exposure to nature (<a href="#">Hanski et al, 2012</a>; <a href="#">Kuo, 2015</a>; <a href="#">Rook, 2013</a>; <a href="#">von Hertzen et al., 2015</a>). <a href="#">Rook (2013)</a> argue that multiple physiological consequences of exposure to the natural environment (e.g., sunlight, physical exercise) supplement the immune-regulatory effects of microbial biodiversity (i.e., low CRP levels, low inflammation, low cytokine response to stress) and the psychological rewards of interaction with nature (e.g., relaxation, restoration, exercise, social capital). These notions have been brought forth by <i>the hygiene hypothesis (i.e., Old Friends mechanism, biodiversity hypothesis)</i> that explains the increasing prevalence of chronic inflammatory diseases (autoimmunity, allergy and inflammatory bowel diseases) in urban communities in high-income countries by a predisposition to poor regulation of inflammation gradually developed through</p>