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Quantifying the effects of different environmental stressors on the health of the population in Germany, taking into account the population-based exposure approach

(Distribution-based analysis of health effects from environmental stressors, VegAS)

Summary

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Summary

In October 2009, as part of the Framework of the Environmental Research Plan 2009, the Federal Environment Agency promoted the research project "Quantifying the effects of different environmental burdens on the health of people in Germany, taking into account the population-based exposure assessment" (short title: Distribution-based analysis of health outcomes of environmental stressors; acronym: VegAS). The project ran for a total of 30 months.

In the VegAS project, the procedure for determining the environmental burden of disease (EBD) was shown in detail for the **environmental stressors** benzene, cadmium, particulate matter, noise, ozone, second-hand smoke and perfluorinated compounds (PFC). The environmental stressor second-hand smoke was carried out as a personal contribution of the research consortium.

The VegAS project had the following **objectives**:

- contribute to improving the methodological and empirical basis for determining the environmental burden of disease in Germany;
- create a basis for future comparative and methodologically consistent quantification of the environmental burden of disease in Germany;
- determine whether the environmental burden of disease concept of the World Health Organization (WHO) and its methods are suitable for prioritizing the environmental stressors listed above on the basis of their estimated health effects in Germany;
- if possible, develop a ranking based on the VegAS project estimate of the environmental burden of disease which can help decide on interventions for each environmental stressor;
- identify opportunities and limitations of the method which result from the method itself, from the availability of data and from scientific knowledge about health problems;
- describe uncertainties qualitatively and, where possible, quantify them;
- identify data gaps, research needs and recommendations for action regarding further procedures for determining the environmental burden of disease in Germany.

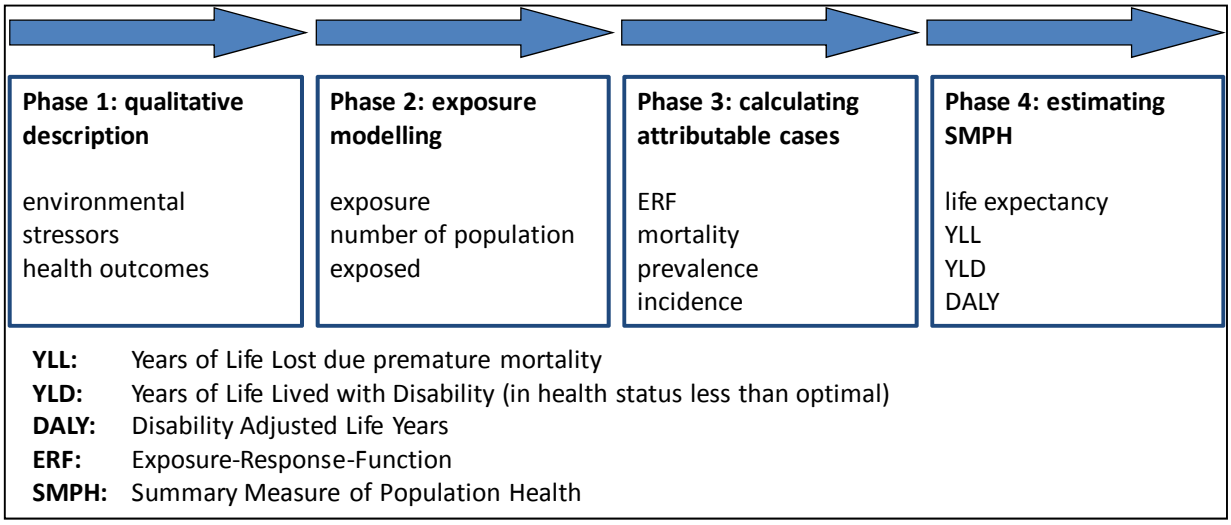
The purpose of the project for the various environmental stressors was to:

- screen the scientific data (including exposure-response functions), document and evaluate their usefulness for EBD estimates in Germany;
- model the exposure distribution in the population of Germany. Aspects such as age, gender, social status, groups at risk or geographic specifications were to be taken into account wherever possible;
- as far as technically feasible, develop and adjust models of disease and mortality burden on the available data;

- estimate risks using EBD methodology, including appropriate reduction or avoidance scenarios;
- describe and analyze uncertainties and limitations;
- evaluate the results with regard to the quality of data, models and literature;
- where possible, standardize the methodology so that the relative abundance of comparatively increasing adverse health effects (i.e., in terms of rankings) can be classified;
- develop policy recommendations and show possible areas for action and measures to reduce exposure to the environmental stressors examined.

The approach shown in Figure 1 is divided depending on the quality of information and availability of data at different stages. The aim of the project was not always to achieve phase 4 for all environmental stressors, but rather give a detailed description of the necessary steps, data requirements, limitations and uncertainties, allowing the VegAS consortium to relate feasibility and validity of the results according each stressor.

Figure 1: Model of the phases and target parameters for progressive description of the environmental burden of disease as a function of information quality and availability of data



Source: Own illustration

For particulate matter and ozone, special emphasis was placed on detailed population-weighted exposure modeling. These were analyzed using spatial-temporal interpolation based on the method described by Horálek et al. (2005).

Certain assumptions and uncertainties had to be made for stressor-specific analyses. Due to insufficient data and limitations on subgroups or exposure categories (e.g. residents in metropolitan areas, non-smokers, background-stressor concentration, etc.), it was often not possible to consider the total population in Germany (differentiated by age, gender, risk groups, etc.) for each individual stressor. The original data contained partial inconsistencies or lacked stratification. With modeling or extra- and intrapolations, certain analytical parameters were derived and used. Co-morbidities and links between health outcomes could not be considered. The disease

duration of most health outcomes is conceptually and empirically difficult to determine, so that some simplifications had to be made whose assumptions cannot be safely considered satisfactory (incidence * duration = prevalence). Due to the current state of research and data availability, instead of determining the total burden of disease across the environmental stressors, it was only possible to estimate the burden of disease associated with individual health outcomes. Included were only those health outcomes whose association with the stressor – classified according to a framework of criteria developed in the VegAS project – was considered at least moderate.

Results differentiated by environmental stressors

Benzene

The analysis of the VegAS project to benzene-related burden of disease in Germany pointed to leukemias (total leukemia according to ICD-10: C91-C95), confirming the results of a previous study by Hänninen & Knol (2011). The burden of disease attributable to leukemia for benzene could be determined, although evidence on the association between benzene and leukemia is limited to individual subtypes of leukemia. Further health outcomes seem to be associated with benzene, but could not be included in the analysis due to insufficient knowledge, incomplete data and inconsistent exposure-response functions, so an additional, suspected benzene-related burden of disease could not be quantified. Uncertainty and sensitivity analyses were conducted to observe the variation of results of included parameters.

With 181 DALYs (0.22 DALYs per 100,000), the estimated burden of disease for benzene-caused leukemia in Germany is relatively low. This is a result of regulatory measures which target a continuous reduction of benzene concentrations in Germany. In the VegAS project, only the average benzene exposure from background exposure was evaluated. Extraordinary burdens could not be included. The VegAS evaluation documents the health benefits due to benzene reduction measures (such as the EU Directives 2008/50/EC of benzene in ambient air).

Cadmium

For cadmium, the burden of disease attributable to lung cancer by inhalation of cadmium in background air can be determined with 7 DALYs for all of Germany. The data for cadmium is characterized by the fact that a variety of data on all dimensions of the EBD model is available, although it is mostly incompatible with the model specifications and so could not be combined. The quantity of data reveals fundamental differences in the exposure database. Difficulties in linking data are becoming apparent, particularly in the categories of exposure-response functions and for similar stratified health data. It was thus possible to attribute only one health outcome – lung cancer – to cadmium.

It was found that evidence for the association between exposure to cadmium and kidney damage, bone disease and certain cancers (lung, kidney, prostate) can be classified as high or at

least moderate. Only a small part of the burden of disease could be quantified. Regarding the state of research, however, it is likely that the burden of disease attributable to cadmium in Germany is significantly greater. Hence, the burden of disease due to cadmium can only be incompletely shown. Further research is needed for a evidence-based environmental health policy (including regulations).

Particulate matter

Premature deaths during the study period (2005 to 2009) due to short-term exposure to PM_{10} is estimated at 7,500-10,600 per year only for anthropogenic particulate matter. If including PM from natural sources, the number rises to between 12,000 and 15,000 cases. There are total of 1-1,5 DALYs per 100,000 for PM from anthropogenic sources, and 1,6-2,1 DALYs per 100,000 for total PM. These are estimates for the burden of disease from short-term exposure to PM. Due to non-differentiation from exposure-related long-term changes in mortality, the calculated DALYs should either not be added, or at least added with strong reservations when considering other health outcomes.

For mortality from acute respiratory infections in children under 5 years, in average 36 deaths per year are reported in Germany from 2007 to 2009, of which one death can be attributed to PM.

Furthermore, restricted activity per adult per year for three-quarters of a day or for an entire day may be caused by short-term exposure (1.2-1.5 days when considering all PM sources), and 2-3 additional days (3-4 if considering all PM sources) with lower respiratory tract problems in children <15 years and young adults >15 years with preexisting chronic lung disease.

The burden of disease for long-term exposure to PM is significant, with 12.3-15.3% of cardiopulmonary deaths being estimated to be attributable to exposure to PM, and 18-22% for lung cancer. Including PM from natural sources increases the estimated mortality from cardiopulmonary causes to 34-36%, and to 46-49% for lung cancer.

Further health outcomes were estimated from 34-63 additional cases of chronic bronchitis per year per 100,000 adults >27 years. For adolescents and adults, the risk is 1.2 times higher for evolving respiratory allergies. Adding PM from natural sources, chronic bronchitis increases to 72-91 estimated additional cases per year per 100,000. The risk of respiratory allergies increases by a factor of 1.7.

Tables 1 and 2 show the sum of the absolute and relative DALYs for each year for those health outcomes where calculation was possible.

Table 1: Sum of absolute and relative DALYs for particulate matter from 2005 to 2009. Base concentrations were 7 µg/m^3 for PM_{10} and 5.11 µg/m^3 for $PM_{2.5}$.

	2005	**2006**	**2007**	**2008**	**2009**
DALYs	576,051	551,410	494,445	465,954	503,735
DALYs per 1,000 inhabitants	7.01	6.71	6.03	5.67	6.13

Table 2: Sum of absolute and relative DALYs for particulate matter from 2005 to 2009. No base concentrations were used.

	2005	**2006**	**2007**	**2008**	**2009**
DALYs	1,337,585	1,319,552	1,277,870	1,257,026	1,284,666
DALYs per 1,000 inhabitants	16.29	16.08	15.57	15.31	15.64

Noise

Environmental noise is a risk factor for health restrictions such as annoyance, sleep disturbances, cognitive impairment as well as cardiovascular and cerebrovascular diseases and for hearing disorders. Exposure to environmental noise cannot be treated as a singular entity, however, but must be divided into different, independent types of noise.

The available data allows quantification estimates of DALYs from noise for some combinations of noise types and health outcomes, but not for others. It was possible to quantify the following combinations:

- DALYs attributable to road, rail and air traffic noise causing annoyance,
- DALYs attributable to road, rail and air traffic noise causing sleeping disorders,
- DALYs attributable to road traffic noise causing myocardial infarction,
- DALYs attributable to road and air traffic noise causing hypertension, and
- DALYs attributable to road traffic noise causing stroke.

Wherever possible, these estimates were stratified by age and gender. The regional distribution of the individual metropolitan areas based on currently available data could not be mapped into the models.

It was not possible to quantify the environmental noise-related burden of disease in terms of

- hearing loss and one of these types of noise,
- tinnitus and one of these types of noise,
- cognitive impairments and one of these types of noise,
- neighborhood noise and one of the relevant health outcomes,
- leisure-time noise and any of the relevant health outcomes
- heart attack and noise from air traffic or rail traffic,
- hypertension and noise from rail traffic,
- stroke and noise from air traffic or rail traffic,
- ischemic heart disease and one of these types of noise.

Calculations were based on estimates of exposure to noise by the Noise Observation and Information Service for Europe (NOISE). Currently, only statements regarding urbanized areas in Germany with at least 250,000 inhabitants can be made which cannot be reliably generalized to the total population of Germany. For the next stage of noise exposure estimation, it is planned to include a reference to metropolitan areas with at least 100,000 inhabitants. No comprehensive estimate of exposure is currently underway.

A **total estimate of the burden of disease attributable to environmental noise in Germany** cannot be made at present. No exposure estimate is available for the general population, and the DALYs are modeled only for a few relevant combinations of noise type and health outcome. The results are based on German urban areas with at least 250,000 inhabitants. The calculations cannot be summed up to even a minimum empirical total quantifiable estimate because it is not possible to include correlations and interactions of the various types of noise and health outcomes in the models.

Taking into account these limitations and uncertainties, YLLs, YLDs and DALYs can be determined for noise (Table 3):

Table 3: Summarized results of model calculations for health outcomes due to noise[a]

Model calculation	YLLs	Attributable years of disease	Disability weights	YLDs	DALYs per 100,000[b]
Road traffic noise Annoyance	0	597,423	0.02[c]	11,948.46	69.41
Rail traffic noise Annoyance	0	275,295	0.02[c]	5,505.90	31.97
Air traffic noise Annoyance	0	69,442	0.02[c]	1,388.84	9.53
Road traffic noise Insomnia	0	128,784	0.07[c]	17,684.94	102.74
Rail traffic noise Insomnia	0	77,679	0.07[c]	5,437.53	31.57
Air traffic noise Insomnia	0	9,993	0.07[c]	699.51	4.80
Road traffic noise Hypertension	2,816	459,475	0.091	41,812.25	264.73
Air traffic noise Hypertension	133	16,484	0.091	1,500.05	11.22
Road traffic noise Myocardial infarction	3,105.79	30.72	0.405	12.44	18.11
Road traffic noise Stroke	31,040	14,105	0.92 0.259	4,206	204.75

[a] Based on stated assumptions and without considering the uncertainties mentioned.

[b] A summation of the health outcome-related DALYs for a total DALY burden of disease caused by noise ***cannot*** be made.

[c] Average *disability weights* for the WHO recommendations.

As expected, the burden of disease for frequent, long-term health problems with a relatively low impact (e.g., annoyance and sleep disturbance) is given more emphasis than less frequent health outcomes of short duration but resulting in severe impairment or death (e.g., myocardial infarction).

Ozone

The burden attributable to ozone for respiratory allergies and hay fever is significant. The estimated attributable fraction of the total burden of disease, depending on the survey year, is 39-44%.

The estimated attributable fraction of the total mortality rate for ozone is 0.4-0.55%, again depending on the survey year. In absolute numbers, this means 2,400-3,300 deaths, which is 26,000-37,000 premature years of life lost (DALYs). The ozone concentrations from 2005 to 2007 also were responsible for about one estimated additional “coughing day” per child per year.

Second-hand smoke

A number of health problems in the non-smoking population could be attributed to second-hand smoke. Analysis of this single environmental stressor alone shows the complexity of the EBD estimate.

The effects of exposure to second-hand smoke *in utero* and the associated full premature loss of life expectancy for the two (relatively rare) health outcomes sudden infant death syndrome (SIDS) (2,186 YLLs) and death due to low birth weight (2,043 YLLs) are nearly identical. Theses are nearly the same for premenopausal breast cancer by non-smoking women (3,792 DALYs), whereas breast cancer is the leading cause of overall cancer death in women.

As expected, the highest attributable burdens were estimated for acute myocardial infarction (over 14,000 DALYs) and stroke (over 25,000 DALYs). Even for non-smokers there is clearly an enormous prevention potential. This is also noticeable notwithstanding the limitations by the methodological and data-related EBD estimates for this stressor.

Perfluorinated organic compounds/chemicals

The perfluorinated compounds PFOA (perfluorooctanoic acid) and PFOS (perfluorooctane sulfonate) were examined whether fulfilling the requirements for the use of the EBD concept. These requirements refer to the data available for public exposure, to the description of exposure-related health impacts and appropriate exposure-response relationships, as well as the assignment of disability weights to the relevant health outcomes.

The exposure situation of the German population can – based on the present data – only be inadequately measured. The ratio for human biomonitoring data for particular regions and data for external exposure (food, drinking water, dust, contact with products and packaging surfaces, etc.) on the current state of science for risk assessment is still insufficient, especially for an all age group analysis. Currently new information is generated from the study programs begun in recent years. Reviews, meta-analyses and summary risk assessment (description of exposure-response relationships) are largely missing. Related to the possible associated internal exposure (described as PFOA and PFOS in plasma) biological effects, such as changes in lipid metabolism and uric acid substitution, reducing the persistent immunization, alteration of the onset of puberty/menarche and menopause, there are no reference distributions and no disability weights available today.

Since EBD assessments only consider those effects, that fulfill the above-mentioned requirements with regard to the available data and knowledge, there are significant limitations for this environmental stressor. The exposure description of the population through human biomonitoring data is regularly not provided in the usual EBD concept, methodological approaches still have to be developed. The analysis and evaluation of possible exposure-related health effects of PFOA and PFOS is at present sufficiently incomplete. The exposure-response relationships de-

scribed in the literature are based on individual studies and required an overall assessment to justify them. A risk assessment for the general population is therefore currently not feasible. The scientific discussion of an assignment of disability weights to effect sizes, which are described by clinical laboratory parameters and not by a classification of diseases, is currently under consideration. A professionally justifiable EBD modeling for the perfluorinated compounds PFOA and PFOS cannot be recommended, due to the above-stated reasons.

Overall results

The VegAS project yielded the following **products**:

1. a description of the current state of research on (a) the association(s) between a particular environmental stressor and the health outcome(s) and the current evidence regarding (b) health, (c) exposure data, (d) exposure-response functions and (e) disability weights (DWs);
2. a description of the gaps in research;
3. modeling stressor-specific exposures in Germany;
4. as far as possible, calculations of YLLs, YLDs and DALYs;
5. a discussion of the EBD methodology as well as designing and identifying uncertainties and sensitivities;
6. recommendations for dealing with EBD estimates as part of health-related environmental protection.

The main findings were part of a two-day technical hearing and discussion with external experts at the Federal Environment Agency in Berlin. The suggestions of these experts were incorporated into the final report.

The VegAS project has shown how complex the estimation of the total burden of disease for even one single environmental stressor is. The stages shown in Figure 1 for each environmental stressor were implemented wherever possible. DALYs were determined for 6 of 7 environmental stressors and for 11 different health outcomes, although some (e.g., lung cancer) could be attributed to more than one stressor. YLLs could be estimated for 5 health outcomes. Compilation of data, information and EBD estimates in Germany have yielded a so far unique source of information for EBD studies, which also were evaluated for usefulness. There is strong variation in the quantification of the environmental stressors. Several problem areas were identified which may affect the validity of the results:

- differences in the availability, quality and transferability of information according to spatial and temporal distribution of exposure, risk groups, health outcomes and exposure-response functions;
- requirement of greatly simplifying EBD models to yield useful results (partly without considering combined effects, comorbidity and disease sequences);

- suitability of the EBD method is given, especially for recognized environmental stressors and health outcomes associated with them but suitability is restricted due to incomplete toxicological and epidemiological data, especially for "new" environmental stressors;
- estimating the environmental burden of disease for various environmental stressors on the basis of different models;
- using disability weights for quantifying the burden of disease, which should be evaluated critically from an ethical point of view;
- lack of disability weights for a whole range of health outcomes known to be adverse effects of environmental stressors;
- potentially using EBD results as a basis for prioritizing and subsequently economizing, when their limitations reduce their significance.

These problem areas should always be discussed in detail when addressing specific target groups. The estimates should not be used without adequate knowledge of individual partial results and values to prevent misinterpretation. However, this should not lead to the conclusion that EBD results cannot be used for decision-making in health-related environmental protection. First and foremost, the EBD concept provides a **framework for the systematic collection of data on environmental health risks**, which also includes the most objective assessment of these risks. The latter has so far worked to a limited extent and definitely warrants critical discussion. The results and critical discussion of the objectiveness of individual parameters and comparability of the EBD analysis should not be ignored, as they are being used worldwide for international cooperation and decision-making. This becomes clear with intervention scenarios where achievable health gains can be quantified by regulating well-understood environmental stressors, such as reducing second-hand smoke or particulate matter in the air, or arsenic in drinking water.

Conclusions

The **EBD method provides estimates of the environmental burden of disease** from available data, in so far supports the systematic allocation of health risks to environmental stressors. This was shown by examples in the VegAS project which, although it has both strengths and weaknesses, made use of the **strengths** while the **limitations and uncertainties** were carefully considered.

A variety of **data gaps** were found for Germany. Furthermore, processing available data is time-consuming. For each environmental stressor, the data available was presented transparently. In order to make the estimates, it was necessary to make determinations, which were also described. In order to limit the number of assumptions and findings based on the data available, tools for estimating missing epidemiological data need to be developed further.

For further research within the framework of environmental and health monitoring, it is necessary to expand the exposure database and close existing gaps in surveys.

In particular, **research needs were identified while dealing with disability weights**. On the one hand, these are essential for unifying mortality and morbidity for DALYs, but on the other hand, the underlying method of derivation, the unexplained transfer and the assumption of universality of the evaluation of diseases, impairments and disabilities need to be reviewed and developed further. Although calculation of DALYs is only one aspect of the EBD concept, the wide international distribution of DALYs makes open discussion of risk assessment necessary. Otherwise, this assessment could, as part of a primarily economically motivated prioritization, be carried out less objectively and without transparently developed criteria.

Comparing the burden of disease across all considered environmental stressors within a comparative risk assessment is currently not possible at an evidence-based, meaningful and robust level. Although, the EBD method can be suitable for routine application in the context of health-related environmental monitoring in Germany of single stressors if a good data base is available, the application is currently not suitable regarding comparisons of several stressors. However, this trend might change fundamentally,

1. if epidemiological knowledge and the available data improved, and
2. if the open question of a fundamental, normative weighting of health problems across a wide professional and public socio-political debate and legitimacy were brought to a (preliminary) conclusion.

In this case, the EBD method could be used, with transparent reference to opportunities and limitations, as an important but not unique component for preparing political decisions and prevention measures for health-related environmental protection.

The ultimate step in the EBD concept, an entirely and expressly consented, across-the-board comparison of the health relevance of environmental stressors with each other, however, is of theoretical nature and will most probably never be attained.

Even so, however, this overall objective is of vital importance to show clearly how further to improve this method and critically discuss the various options for estimating the environmental burden of disease. The findings will be indispensable for further improving health-related environmental protection, as the debate at the VegAS conference in 2011 and at other international conferences meanwhile has shown. A number of new essential components were thus developed by VegAS, and should be used in this sense.

References

are listed in the final report