ChemObs

Economic Cost of Inaction Calculator

Documentation

Step-by-step Guidance

Calculator for the estimation of adverse health outcomes and associated productivity losses resulting from chemicals exposure.

Integrated Health and Environment Observatories for the Sound Management of Chemicals and Waste in Africa (GEF ID 9080)

Summary

The purpose of this guidance is to assist users in completing the ChemObs Economic Calculator. The calculator uses existing environmental exposure data to approximate attributable disease and productivity losses. The guidance is not intended to be overly descriptive. For more detailed information on the underlying assumptions and calculations refer to the associated methods document.

Steps

Step 1. Assemble data sources

The first step in the process is to gather necessary data on exposures. This could include both the grey literature and peer-reviewed publications. What is needed are the results of environmental analyses of a chemical in a human exposure pathway (i.e. water, soil, dust or dietary; the calculator does not assess air exposure). Utilise only trusted sources and do not use data that are more than 5 years old.

Step 2. Extract and organize data

The second step is to extract the study results and organize them in a useful way. Specifically, users should identify or calculate the central tendency (e.g. median, mean) of analysis results for chemicals in a human exposure pathway.

Step 3. Enter data in the calculator

Begin on the 'Summary' sheet. Choose the country for which you are using the calculator. Then move to any of the other sheets and enter the data you have assembled. Each exposed group should be entered on a separate row. Try to be as specific as possible. At a minimum, children and adults should be entered on separate rows. Groups with higher or lower exposures – where known – could also be entered on separate rows.

Step 4. Assess the results

After all the available data have been entered, refer back to the 'Summary' sheet. Are the results tallying correctly? Do the amounts shown seem to make sense with the information you have entered? Go through each step in the process and check for any possible errors.

Step 5. Communicate the results

Communicate the results of your research to policy makers and colleagues. Discuss the results with other researchers. The 'Primer on Costs of Inaction' includes useful information on understanding the results of your work and how best to communicate that information to different audiences.

Inputs

The calculator relies on the inputting of environmental analysis data. This section describes some select parameters in the calculator's different worksheets.

If environmental analysis data are available, the user enters them in the appropriate worksheet following the guidance given below and in the step-by-step for each exposure pathway (Dietary, Soil, Dust and Water). The tables below list select parameters and describe the required inputs.

Dietary Worksheet

Food	**Weight ingested (kilos)**	**Frequency**	**Population**	**Adult or Child**	**Chemical**	**Concentration (mg/kg)**
Broccoli	0.125	Daily	medium	Child (0-7 years)	Chlorpyrifos	70
Enter the food for which chemical analysis is available	Enter the amount of the relevant food ingested in a single meal by the specified population (adults or children).	Pull down menu. Choose the frequency with which the mean is eaten: daily, 2-3 times per week, or 1-2 times per week.	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Pull down menu. Select the appropriate age group.	Pull down menu. Select the chemical for which analysis information is available.	Enter the central tendency available for analysis. Mean preferred.

Soil Worksheet

Sirte name	Latitude	Longitude	**Population**	**Concentration (mg/kg)**	**Adult or Child**	**Chemical**	**Estimated Dust Concentration (mg/kg)**
East side dumpsite	-1.43	33.9432	medium	400	Child (0-7 years)	Chlorpyrifos	290.7
Enter the common name for the site	Enter latitude in decimal degrees (from -90 to 90)	Enter longitude in decimal degrees (from -180 to 180)	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Enter the central tendency available for analysis. Mean preferred.	Pull down menu. Select the appropriate age group.	Pull down menu. Select the chemical for which analysis information is available.	Populates automatically following USEPA Integrated Environmental Uptake Biokinetic Model guidance.

Dust Worksheet

Sirte name	Population	Concentration (mg/kg)	Concentration (mg/kg)	Adult or Child	Chemical	Estimated Soil Concentration (mg/kg)
Smith town	medium	400	400	Child (0-7 years)	Chlorpyrifos	560.7285714
Enter the common name for the site	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Enter the central tendency available for analysis. Mean preferred.	Enter the central tendency available for analysis. Mean preferred.	Pull down menu. Select the chemical for which analysis information is available.	Pull down menu. Select the chemical for which analysis information is available.	Populates automatically following USEPA Integrated Environmental Uptake Biokinetic model guidance.

Water Worksheet

Water	Volume ingested daily (liters)	Population	Adult or Child	Concentration (ppb)
Smith town	0.125	large	Child (0-7 years)	70
Enter the common name for the site	Enter the amount of the relevant food ingested daily by the population (adults or children).	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Pull down menu. Select the chemical for which analysis information is available.	Enter the central tendency available for analysis. Mean preferred.

Determining the size of the exposed population and frequency of exposure

The model utilizes environmental sampling data to approximate the level of exposure (i.e. dose) of a given chemical to an individual. In all cases – with the exception of dietary sources – the frequency of exposure is set to daily. In the case of dietary exposures, the user must determine how frequently receptors (i.e. humans) ingest the contaminated food. Three options are provided: daily, 2-3 times per week, and 1-2 times per week. Once the exposure is quantifier, the appropriate dose response relationship is then applied to estimate the attributable disease burden for an individual. Population wide impacts are simply the product of the individual disease burden multiplied by the population. Thus a key challenge for the individual user of the model is to identify *the number of people potentially exposed at the specified dose.*

For example if the user enters a certain organophosphate pesticide sampling result for a given food available at a market – say dimethoate on spinach in a market in Dar es Salaam – he or she must determine the number of people eating that particular spinach and the quantity they are consuming at the level of frequency specified. Likewise, if the user enters a certain soil lead concentration for a residential area, the user must enter the number of people likely to be exposed to that soil on a *daily* basis.

This parameter introduces a large amount of uncertainty to the model. Early testing found that individual users' estimates varied widely from those of other uses for similar exposure scenarios. In some cases, entire

national populations were entered as the exposed population. This resulted in a both over- (and in some cases under-) estimations of the disease burden that undermined the integrity and usefulness of the model. Recognizing these challenges, the available inputs for this parameter have been restricted. Users are now asked to select from a simple list of exposed populations defined as small, medium, large. The text below provides guidance on these options:

- **Small.** This selection uses a default value of 100 people exposed. Small groups could be those living near contaminated playgrounds, confined worker exposures in smaller settings, or remote lakes where fish are consumed only by local populations.
- **Medium.** This selection uses a default value of 1,000 people exposed. Medium groups might be entire neighbourhoods or sections of an informal settlement where hazardous chemicals are being handled, larger occupational settings, or markets in medium-sized towns selling vegetables with high levels of pesticides.
- **Large.** This selection uses a default value of 10,000 people exposed. Large groups might be individuals residing in an industrial neighbourhood or around a large open dumpsite. People consuming spinach purchased at a large city market where multiple samples have shown elevated levels of a given pesticide might also be in this category.
- **Very Large.** This selection uses a default value of 100,000 people exposed and should rarely be selected. Very large groups might represent a regional population consuming mercury-contaminated fish or cities built around mining-smelting complexes

Additional information

Refer to the associated methods document to better understand the underlying assumptions and calculations. Contact Africa Institute, WHO, or UNEP for more information or further assistance. Contact details in the table below.

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Methods for Underlying Calculations

Calculator for the estimation of adverse health outcomes and associated productivity losses resulting from chemicals exposure.

Integrated Health and Environment Observatories for the Sound Management of Chemicals and Waste in Africa (GEF ID 9080)

TABLE OF CONTENTS

INTRODUCTION

Summary

This document and the accompanying 'step-by-step' and economic calculator are intended for use by environmental and health professionals to rapidly characterise economic costs associated with chemicals exposures in the project countries. Results could be used in concert with other studies and observations to inform decisions relating remedial options and regulatory policy, among other applications.

The purpose of the model is to estimate attributable Disability Adjusted Life Years (DALYs), Full Scale Intellectual Quotient (IQ) decrement, and associated economic costs resulting from chemicals exposure. The model is intended to accept different types of data inputs form multiple environmental media. To allow for this level of flexibility in the model, a number of assumptions are employed at different stages. The assumptions are based on existing studies, surveys, reports, or expert opinion.

The model is integrated into an accompanying Microsoft Excel workbook which automates the calculations.[1] The model can also be used manually following the guidance presented here. The model was initially developed by Pure Earth in 2019 under contract with the United Nations Environment Programme and later modified by PAN-UK under contract with Africa Institute in 2021. Resources for the Future supported the development of the underlying economic model.

The model is not intended to produce a definitive calculation of health and economic outcomes, but rather an indicative estimate based on the best available information. Estimates are static and represent a snapshot in time. Future efforts might consider refining and improving the methods presented here. Future efforts might consider refining and improving the methods presented here. Given the paucity of efforts in this area, the model is intended as a 'proof of concept' of a tool that – following further improvement, review and testing – could be used in addressing a significant and credible health risk.

The purpose of this document is to outline the steps in the model and underlying assumptions. This document is accompanied by an Excel calculator and 'step-by-step' guide for employing the model in testing.

Sources of Data

The equations embedded in the calculator rely on known concentrations of chemicals in various media. Users enter concentrations of multiple chemicals in four different media (soil, dust, water, food) in either ppm (all media but water) or ppb (water). Sources of data could include government monitoring reports or peer-reviewed literature. In some cases reputable NGO data could also be considered. The Toxic Sites Identification Programme of the NGO Pure Earth offers one possible source.[2] In the absence of site specific information, other inputs can be approximated based on third party information. Dietary information from the Food and Agriculture Organisation of the United Nations could provide useful guidance as could population density data from CIESEN, for instance.[3]

[1] Microsoft Corporation, 'Microsoft Excel for Mac 16.9'.
[2] Pure Earth, 'No Title' (2018) <https://www.pureearth.org/projects/toxic-sites-identification-program-tsip/> accessed 19 February 2018.
[3] FAO, 'Home | Food-Based Dietary Guidelines | Food and Agriculture Organization of the United Nations' <https://www.fao.org/nutrition/education/food-dietary-guidelines/home/en/> accessed 3 May 2022; Center for International Earth Science Information Network-CIESIN-Columbia University and Centro Internacional de Agricultura Tropical- CIAT, 'Gridded Population of the World, Version 3 (GPWv3): Population Density Grid' <http://dx.doi.org/10.7927/H4XK8CG2>.

Metrics

The disability adjusted life year (DALY) is a metric developed by the World Bank in the 1990s and widely employed in burden of disease studies.[4] A DALY represents a productive year of life lost either due to morbidity or mortality. It is the sum of two separate metrics, years lived with disease (YLD) and years of life lost (YLL). YLD is calculated by multiplying the duration (in years) of an adverse health outcome by its sequalae-specific disability weight ranging from 0–1, with 0 representing perfect health and 1 representing death. The Institute for Health Metrics and Evaluation (IHME) is the global leader in calculating burden of disease estimates, releasing annual reports in the medical journal *The Lancet* and sharing detailed results on their website (http://ghdx.healthdata.org/gbd-results-tool). Since 2018, they have done so jointly with the World Health Organization. IHME's list of disability weights is the most comprehensive that exists and is based on surveys of individuals' perceptions of the severity of a given health outcome.[5] The economic cost of a DALY in this model could be understood as an annual cost, or rate.

Full scale intelligence quotient (IQ) is a measure of intelligence and cognitive ability most commonly assessed in children with the Wechsler Intelligence Scale for Children. In any given population, IQ is normally distributed with a mean of 100 and a standard deviation of 15. Economic costs for IQ decrement in this model are quantified as a percentage of lifetime earnings. They represent an assessment of the cost at any one point in time. They therefore cannot be summed with costs associated with DALYs, which represent an annual rate.

Interpreting Results

The results of the calculator are intended to be indicative first pass estimates that could be refined through further effort. Each step in the model contains assumptions and uncertainties. Results should not be interpreted as definitive. Annex 1 (Primer on Costs of Inaction) includes a detailed discussion on the interpretation and communication of the results of this sort of analysis. Annex 2 (Summary of underlying assumptions) outlines key steps in the model and an assessment of the strength of evidence.

Inputs

If environmental analysis data are available, the user enters them in the appropriate worksheet following the guidance given below and in the step-by-step for each exposure pathway (Dietary, Soil, Dust and Water). The tables below list select parameters and describe the required inputs.

Dietary Worksheet

Food	Weight ingested (kilos)	Frequency	Population	Adult or Child	Chemical	Concentration (mg/kg)
Broccoli	0.125	Daily	medium	Child (0-7 years)	Chlorpyrifos	70
Enter the food for	Enter the amount of the relevant	Pull down menu. Choose the	Pull down menu. Select the population	Pull down menu.	Pull down menu. Select	Enter the central tendency available

[4] Christopher JL Murray and Alan D Lopez, 'Measuring the Global Burden of Disease' (2013) 369 New England Journal of Medicine 448 <http://www.nejm.org/doi/abs/10.1056/NEJMra1201534> accessed 26 September 2016.
[5] IHME, 'Global Burden of Disease Study 2017 (GBD 2017) Disability Weights | GHDx' <http://ghdx.healthdata.org/record/ihme-data/gbd-2017-disability-weights> accessed 30 October 2021.

which chemical analysis is available	food ingested in a single meal by the specified population (adults or children).	frequency with which the mean is eaten: daily, 2-3 times per week, or 1-2 times per week.	of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Select the appropriate age group.	the chemical for which analysis information is available.	for analysis. Mean preferred.

Soil Worksheet

Site name	Latitude	Longitude	Population	Concentration (mg/kg)	Adult or Child	Chemical	Estimated Dust Concentration (mg/kg)
East side dumpsite	-1.43	33.9432	medium	400	Child (0-7 years)	Chlorpyrifos	290.7
Enter the common name for the site	Enter latitude in decimal degrees (from -90 to 90)	Enter longitude in decimal degrees (from -180 to 180)	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Enter the central tendency available for analysis. Mean preferred.	Pull down menu. Select the appropriate age group.	Pull down menu. Select the chemical for which analysis information is available.	Populates automatically following USEPA Integrated Environmental Uptake Biokinetic Model guidance.

Dust Worksheet

Sirte name	Population	Concentration (mg/kg)	Concentration (mg/kg)	Adult or Child	Chemical	Estimated Soil Concentration (mg/kg)
Smith town	medium	400	400	Child (0-7 years)	Chlorpyrifos	560.7285714
Enter the common name for the site	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very large). Refer to the population guidance for assistance.	Enter the central tendency available for analysis. Mean preferred.	Enter the central tendency available for analysis. Mean preferred.	Pull down menu. Select the chemical for which analysis information is available.	Pull down menu. Select the chemical for which analysis information is available.	Populates automatically following USEPA Integrated Environmental Uptake Biokinetic model guidance.

Water Worksheet

Water	Volume ingested daily (liters)	Population	Adult or Child	Concentration (ppb)
Smith town	0.125	large	Child (0-7 years)	70
Enter the common name for the site	Enter the amount of the relevant food ingested daily by the	Pull down menu. Select the population of children or adults exposed to soil with this concentration daily (small, medium, large, very	Pull down menu. Select the chemical for which analysis information is available.	Enter the central tendency available for analysis. Mean preferred.

	population (adults or children).	large). Refer to the population guidance for assistance.		

ESTIMATION OF HEALTH OUTCOMES

Lead

Introduction and underlying assumptions

The biokinetics of lead are relatively well understood. In the present model, USEPA tools (i.e. IEUBK and ALM) are used to approximate biological concentrations of lead (blood lead level; BLL) based on environmental sampling data.[6] These results are in turn used to estimate adverse health outcomes associated with elevated BLLs. Specifically, the calculator approximates attributable IQ decrement in children and cardiovascular disease in adults. IQ decrement calculations follow Budtz-Jørgensen, et. al (2013).[7] Cardiovascular disease calculations follow Fewtrell (2003). The specific steps in the method are given below.

Method

Determining Blood Lead Levels from Soil and Dust Concentrations

For children, the USEPA Integrated Environmental Uptake Biokinetic (IEUBK) model is used to estimate blood lead levels (BLLs) associated with soil and dust lead concentrations ranging from 0–10,000 mg/kg.[8] Users input data on soil or dust lead concentrations (in the Soil or Dust Worksheet) from available sources as well as an estimated size of the exposed population.

The accompanying Excel calculator allows users to enter either values for soil or dust lead concentrations. Where dust concentrations are entered into the Soil and Dust Worksheet, they are converted to soil lead concentrations using IEUBK guidance.[9] The soil lead value is then used in subsequent equations based on the IEUBK model.

BLLs are calculated for each one-year interval of age for children age 0–7 years. The average of these values is then taken and used for subsequent calculations. The following assumptions are made while using the IEUBK:

- Soil values are rounded to the closest 100 mg/kg increment;
- All other exposure sources are zeroed out in the IEUBK;
- The upper bound intake of 135 mg/ day is used for all age groups.

[6] OSRTI US EPA, OSWER, 'Lead at Superfund Sites: Software and Users' Manuals' <https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals> accessed 13 August 2016.
[7] Esben Budtz-Jørgensen and others, 'An International Pooled Analysis for Obtaining a Benchmark Dose for Environmental Lead Exposure in Children' (2013) 33 Risk Analysis 450.
[8] US EPA, OSWER (n 6).
[9] OSWER, 'Guidance Manual for the IEUBK Model for Lead in Children' (1994) <https://semspub.epa.gov/work/HQ/176284.pdf> accessed 1 January 2018.

For adults and foetuses, the USEPA Adult Lead Methodology (ALM) is used to calculate blood lead levels associated with soil concentrations from 0–10,000 mg/kg.[10] As above for children users input data on soil or dust lead concentrations (in the Soil or Dust Worksheet) from available sources as well as an estimated size of the exposed population.

As above, the accompanying Excel calculator allows users to enter either values for soil or dust lead concentrations which are then converted to soil lead concentrations using IEUBK guidance.[11] Also as above, the soil lead value is then used in subsequent equations based on the ALM model.

Dietary Ingestion

Blood lead levels attributable to diet are calculated for adults and foetuses following the Adult Lead Methodology in increments of 5 µg/ day. Children's dietary ingestion were calculated using the IEUBK in 5 µg/ day increments and averaged for age 0–7 years as above for soil.

Converting Blood Lead Level to Burden of Disease

Figure 1. Simple flow chart of steps in the model to calculate the burden of disease in children attributable to soil, dust or dietary lead exposure.

For children, IQ decrement is calculated first, then attributable DALYs are calculated for intellectual disability only. IQ decrement is calculated for each BLL increase using values from Budtz-Jorgensen et al.[12] which assumes a log-linear distribution of IQ decrement (proportionally more IQ points are lost at lower BLLs). The resulting IQ points lost are multiplied by the exposed population (entered in the Soil or Dust Worksheet) and summed to arrive at the total IQ points lost.

To determine the DALYs associated with IQ decrement, the attributable increase in intellectually disabled children is calculated and multiplied by a given disability weight. Specifically, the increased proportion of children with borderline, mild, moderate, severe, and profound intellectual disability is determined. These groups are associated with the following IQ intervals: 70–89, 50–69, 35–49, 20–34, and < 20, respectively. These IQ intervals are in turn associated with the following IHME disability weights: 0.0034, 0.127, 0.293. 0.383, and 0.444, respectively.

Thus to determine the increased proportion of children in a given group, a normal distribution of IQ with a mean of 100 and an SD of 15 is assumed. The influence of population-wide shifts in IQ are then modelled for IQ losses ranging from ~1 to ~9 points. The increased proportion of children in each interval is

[10] US EPA, OSWER (n 6).
[11] OSWER (n 9).
[12] (2013)

determined as a percent and multiplied by the exposed population and appropriate disability weight. Additional adjustments are made following WHO guidance on regional adjustment ratios.[13]

By way of example, in a given population approximately 2.8 % of children have an IQ below 70. If population-wide IQ is lowered by 5 points due to lead-exposure, this value increases to 4.8 %. In the model this 2 % increase is captured at the lead-attributable disease burden.

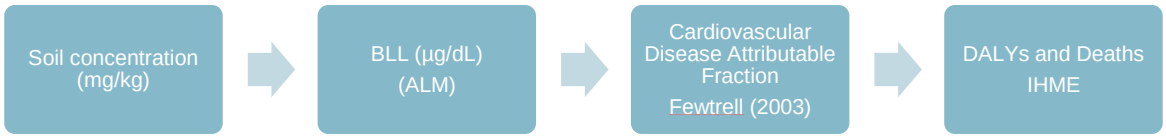

Figure 2. Simple flow chart of steps in the model to calculate the burden of disease in adults attributable to soil and dust lead exposure.

For adults, a log-linear distribution of BLLs is assumed. The mean BLL calculated from the ALM and the standard deviation of 1.6 are used.[14] The distribution of BLLs within the following intervals is calculated: 5–10, 10–15, 15–20, >20 µg/dL. The percentage increase in each interval is then multiplied by its respective relative risk (Following Fewtrell, 2003) for four types of cardiovascular disease: ischaemic, hypertensive, cerebrovascular and other.[15] No sex disaggregation is conducted. Relative risk is averaged across sexes, resulting in an underestimate of cardiovascular disease. This value is then used in the following Equation 1 from Fewtrell et al. (2003) to calculate attributable fraction:

$$AF = \frac{\sum P_i RR_i - 1}{\sum P_i RR_i}$$

(1)

Where:

AF=Attributable fraction

P_i=proportion of the population at exposure interval i

RR_i=relative risk at exposure interval i, compared to the reference level.

The resulting attributable fraction for each type of cardiovascular disease is then multiplied by the total DALYs and deaths calculated by IHME for that type of cardiovascular disease in 2017. Where IHME does not calculate DALYs for a given cardiovascular disease, the attributable fractions are summed and multiplied by the DALYS for the category 'other cardiovascular diseases.

[13] Lorna Fewtrell, Rachel Kaufmann and Annette Prüss-Üstün, *Lead: Assessing the Environmental Burden of Disease at National and Local Levels* (World Health Organization 2003) <http://www.who.int/quantifying_ehimpacts/publications/en/leadebd2.pdf?ua=1> accessed 10 October 2016.
[14] SD of 1.6 taken from IEUBK
[15] Fewtrell, Kaufmann and Prüss-Üstün (n 13).

The attributable fraction of DALYs and deaths for each type of cardiovascular disease is then multiplied by the population entered in the Soil and Dust Worksheet to calculate total attributable DALYs and deaths for a given cardiovascular disease. All DALYs from all three types of cardiovascular disease (ischaemic, hypertensive, and other) are then summed to calculate total attributable DALYs.'

Organophosphate Pesticides

Introduction and underlying assumptions

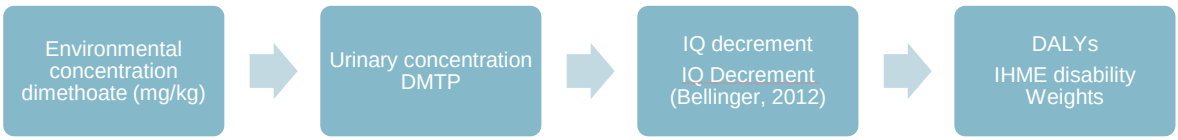

Figure 3. Simple flow chart of steps in the model to calculate the burden of disease attributable pesticides exposure

The model calculates IQ decrement in children and attributable Disability Adjusted Life Years (DALYs). The literature on organophosphate (OP) pesticides exposure and IQ decrement is less robust than that for lead and mercury. Bouchard, et. al (2011) and Engel, et al (2011) evaluated urinary metabolites of dialkyl phosphate (DAP) in pregnant women living in agricultural areas.[16] The researchers found decrements of 1.39 (Bouchard) and 5.6 (Engel) IQ points in children of prenatally exposed women associated with a 10-fold increase in DAP. Bellinger 2012 pooled and employed a sample-weighted 4.25 IQ point decrement associated with a 10-fold increase in DAP to determine the number of IQ points lost in children due to pesticides exposure in the US.[17] He accomplished this by calculating a slope of -0.01 IQ points/nmol/L DAP in urine for concentrations > 50 nmol/L DAP. Put differently, Bellinger (2012) assumed every one nmol/L increase (above 50 nmol/L) in DAP metabolites in urine would result in a 0.01 decrease in IQ. He then applied that slope to data from the US National Health and Nutrition Examination Survey (NHANES) (1994–2004) to calculate the total IQ points lost for the population.

Urinary concentrations of pesticide metabolites can be used to calculate dose by correcting for creatinine and determining the concentration of the metabolite's parent chemical in the pesticide.[18] Fenske (2000) measured the presence of the DAP metabolites dimethyl thiophosphate and dimethyl dithiophosphate and estimated daily OP pesticides exposure levels for children of farmworkers as well as a control group.[19] The study found daily doses of 0–72 µg/kg/day and seasonal doses of 0–36 µg/kg/day. These values are

[16] Maryse F Bouchard and others, 'Prenatal Exposure to Organophosphate Pesticides and IQ in 7-Year-Old Children' [2011] Environmental Health Perspectives; Stephanie M Engel and others, 'Prenatal Exposure to Organophosphates, Paraoxonase 1, and Cognitive Development in Childhood' (2011) 119 Environmental Health Perspectives 1182.
[17] David C Bellinger, 'A Strategy for Comparing the Contributions of Environmental Chemicals and Other Risk Factors to Neurodevelopment of Children' (2012) 120 Environmental Health Perspectives 501.
[18] Richard A Fenske and others, 'Biologically Based Pesticide Dose Estimates for Children in an Agricultural Community' [2000] Environmental Health Perspectives; David T Mage and others, 'Estimating Pesticide Dose from Urinary Pesticide Concentration Data by Creatinine Correction in the Third National Health and Nutrition Examination Survey (NHANES-III)' 457.
[19] Fenske and others (n 18).

significantly higher than doses in the general population. Mage, et al (2004) for instance calculated a median exposure to chlorpyrifos of 0.134 µg/kg/day for the US based on NHANES data and a maximum exposure of 4.38 µg/kg/day.[20]

Different pesticides have different metabolites, and few have been evaluated for their relationship with IQ. Bouchard, et al. (2001) note that the most commonly used pesticides in their study area were chlorpyrifos, diazinon, malathion and oxydemeton-methyl, though did not endeavour to determine the individual effect of each on IQ.

Method

The present model calculates IQ decrement attributable to exposure to a range of OP pesticides. However the impact of most pesticides on IQ remains poorly characterized. In the absence of pesticide-specific dose response information, the calculator assumes a uniform impact of all OP pesticides on brain development. The values from the commonly used pesticide dimethoate are used in all cases.

Dimethoate has a molecular mass of 229.26 g/mol. Estimates of absorption rates for ingested dimethoate range from 60–90 %.[21] The calculator assumes that a) 75% of dimethoate is absorbed, that b) all absorbed dimethoate is excreted from the body as the metabolite DAP, and that c) all ingested dimethoate is excreted from the body within 24 hours of ingestion. Thus to determine the daily urinary excretion rate of DAP, the following equation (2) derived from Rigas et al., 2001[22] is used:

$$UER_{mg} = \left(1 * \left(0.75 * I_{mg}\right)\right) \quad (2)$$

Where:
UER_{mg} = urinary excretion rate (mg/day)
I_{mg} = ingestion (mg/day)

The resulting urinary excretion rate (mg/day) is converted to nmols/day using the molecular mass of dimethoate in Equations 3 and 4, thus:

$$UER_{nmol} = \frac{UER_{mg} * 0.001}{229.26} * 1e9 \quad (3)$$

Where:
UER_{nmol} = urinary excretion rate (nmol/day)

[20] Mage and others (n 18).
[21] National Center for Biotechnology Information, 'Dimethoate, CID=3082' (*PubChem Database*, 2019).
[22] Marc L Rigas, Miles S Okino and James J Quackenboss, 'Use of a Pharmacokinetic Model to Assess Chlorpyrifos Exposure and Dose in Children, Based on Urinary Biomarker Measurements' (2001).

$$C = \frac{UER_{nmol}}{2}$$

(4)

Where:
C = concentration DAP in urine (nmol/L)

The concentration of DAP in urine above 50 nmol/L is then multiplied by -0.01 following Bellinger (2012) to estimate IQ points lost for an individual. Bouchard, et al. (2011) found a relationship between urine concentrations of pregnant women and the IQ decrement in their children (age 7 years). Thus the algorithm utilizes modelled adult urinary concentrations and applies the resulting IQ decrement to all children age 0–7 in the population. The inclusion of all children 0–7 years effectively presupposes that the same level of exposure has occurred over the previous 7 years. Once IQ decrement is determined, DALYs are calculated as above for lead.

Mercury

Introduction underlying assumptions and method

Methyl mercury exposure is associated with IQ decrement in children.[23] In the calculator IQ decrement is calculated based on dietary concentrations of methyl mercury following Spadaro and Rabl (2008).[24] The study includes a comprehensive model that calculates IQ decrement from dietary exposure to methyl mercury. This is distinct from methods presented above where different models are linked together to develop a functional dose (mg/kg)–response (IQ decrement) model. The underlying equation is as follows:

$$IQ\ decrement = 0.036\ x\ I$$

(5)

Where:
I = ingestion (µg/day)

Dietary calculations are calculated by the user utilizing environmental sampling data. Soil and dust concentrations are calculated as above for lead. After IQ decrement is calculated, DALYs are calculated as above for lead and OP.

(5)

[23] Daniel A Axelrad and others, 'Dose-Response Relationship of Prenatal Mercury Exposure and IQ: An Integrative Analysis of Epidemiologic Data' (2007) 115 Environmental Health Perspectives 609.
[24] Joseph V. Spadaro and Ari Rabl, 'Global Health Impacts and Costs Due to Mercury Emissions' (2008) 28 Risk Analysis 603 <http://doi.wiley.com/10.1111/j.1539-6924.2008.01041.x> accessed 16 August 2019.

Carcinogens (including organochlorine pesticides)

Introduction and underlying assumptions

The USEPA maintains a database of slope factors for the increased risk associated with exposure to carcinogens in the form of the Integrated Risk Information System (IRIS).[25] The linear slope factors contained in IRIS are based largely on animal studies and calculate increased cancer risk for different exposure routes. Oral (mg/kg/day), water (µg/L), and air (µg/m^3) concentrations are considered for most chemicals. In the present effort, only oral (i.e. ingestion) concentrations are used. Increased risk is taken to be analogous with increased incidence. While the calculator focuses on organochlorine pesticides, multiple non-pesticide carcinogens can also be assessed.

The Institute for Health Metrics and Evaluation (IHME) calculates different disability weights for multiple adverse health outcomes to capture the perceived severity of each. In the case of cancers, each cancer site is given a different disability weight, as are different phases of the cancer (i.e., diagnosis, metastasis). For each cancer, disability weights for the 'diagnosis' phase range from 0.1 to 0.5; for 'metastasis' they are all set at the same value of 0.451. In the present effort all cancers are given the uniform disability weights of 0.2 for diagnosis and 0.451 for metastasis. Cancers are assumed to endure 5 years, with 3 years being in the diagnosis phase and 2 years being in the metastasis phase. The 5-year survival rate for all cancers is assumed to be 50 %. In practice survival rates for different cancers vary widely but are generally below 20 % in Africa.[26] Thus the use of 50 % is intended to be conservative, meaning the overall number of deaths is likely underestimated.

Method

First the environmental concentration of a given carcinogen is entered in mg/kg in the Dietary and Soil or Dust Worksheet. The concentration is taken from environmental sampling data. The ingestion rate is calculated by the user for the dietary spread sheet. For soil and dust, this calculation is automated as above for lead.

$$Ingestion\ Exposure\ Dose = \frac{C * IR * EF}{BW} \quad (6)$$

Where:
C = concentration (mg/kg)
IR = ingestion rate (mg/kg/day)
EF = exposure factor (set to 1 assuming complete absorption)
BW = body weight (kg)

Once the ingestion exposure dose is calculated, increase cancer risk is calculated as follows:

[25] US EPA, 'Integrated Risk Information System' (2016) <https://www.epa.gov/iris> accessed 26 September 2016.
[26] R Sankaranarayanan and others, 'An Overview of Cancer Survival in Africa, Asia, the Caribbean and Central America: The Case for Investment in Cancer Health Services.' [2011] IARC scientific publications 257.

$$Cancer\ Risk = \frac{Exposure\ dose * \ slope\ factor * \ years\ of\ exposure}{70\ years\ (lifetime)} \quad (6)$$

Lifetime cancer risk is then divided by 11 to determine the likelihood of a case of cancer occurring within the 5-year duration evaluated by the calculation, assuming a life expectancy of 70 years and an equal chance of cancer occurring from age 15 to 70 years. The disability weights for diagnosis, metastasis and mortality are applied to the number of cancer cases. As noted above, the diagnosis phase is assumed to be 3 years with a disability weight of 0.2 and the metastasis phase is assumed to be 2 years with a disability weight of 0.451. Also as noted above, death is expected to occur in 50 % of all cases within 5 years and have a disability of 1.

Determining the size of the exposed population and frequency of exposure

The model utilizes environmental sampling data to approximate the level of exposure (i.e. dose) of a given chemical to an individual. In all cases – with the exception of dietary sources – the frequency of exposure is set to daily. In the case of dietary exposures, the user must determine how frequently receptors (i.e. humans) ingest the contaminated food. Three options are provided: daily, 2-3 times per week, and 1-2 time per week. Once the exposure is quantifier, the appropriate dose response relationship is then applied to estimate the attributable disease burden for an individual. Population wide impacts are simply the product of the individual disease burden multiplied by the population. Thus a key challenge for the individual user of the model is to identify *the number of people potentially exposed at the specified dose.*

For example if the user enters a certain organophosphate pesticide sampling result for a given food available at a market – say dimethoate on spinach in a market in Dar es Salaam – he or she must determine the number of people eating that particular spinach and the quantity they are consuming at the level of frequency specified. Likewise, if the user enters a certain soil lead concentration for a residential area, the user must enter the number of people likely to be exposed to that soil on a *daily* basis.

This parameter introduces a large amount of uncertainty to the model. Early testing found that individual users' estimates varied widely from those of other uses for similar exposure scenarios. In some cases, entire national populations were entered as the exposed population. This resulted in a both over- (and in some cases under-) estimations of the disease burden that undermined the integrity and usefulness of the model. Recognizing these challenges, the available inputs for this parameter have been restricted. Users are now asked to select from a simple list of exposed populations defined as small, medium, large. The text below provides guidance on these options:

- **Small.** This selection uses a default value of 100 people exposed. Small groups could be those living near contaminated playgrounds, confined worker exposures in smaller settings, or remote lakes where fish are consumed only by local populations.

- **Medium.** This selection uses a default value of 1,000 people exposed. Medium groups might be entire neighbourhoods or sections of an informal settlement where hazardous chemicals are being handled, larger occupational settings, or markets in medium-sized towns selling vegetables with high levels of pesticides.
- **Large.** This selection uses a default value of 10,000 people exposed. Large groups might be individuals residing in an industrial neighbourhood or around a large open dumpsite. People consuming spinach purchased at a large city market where multiple samples have shown elevated levels of a given pesticide might also be in this category.
- **Very Large.** This selection uses a default value of 100,000 people exposed and should rarely be selected. Very large groups might represent a regional population consuming mercury-contaminated fish or cities built around mining-smelting complexes.

ESTIMATION OF COSTS OF INACTION

Introduction and underlying assumptions – valuation of YLDs and deaths

The economic costs of adverse health outcomes calculated below reflect the loss in output during the year in which people are ill, measured by YLDs, and the loss in the present value of output when people die prematurely.

The value of a YLD is calculated as the average value of output per worker in the country, multiplied by the probability that a person is working. We refer to this as the expected value of market output. If the probability of working varies by age, the expected value of market output will vary by age. Average output per worker—which is assumed the same for workers of all ages—is calculated by multiplying a country's GDP by labour's share of GDP and dividing by the number of workers employed.

This value could be modified to allow for the value of non-market output by adding the average value of non-market output (multiplied by the probability that a person is not working) to market output. The average value of non-market output has been estimated for several countries (see Appendix), and is usually expressed as a fraction (e.g. 0.3) of average output per worker, based on GDP. The expected value of market plus non-market output is multiplied by the number of YLDs to calculate the cost of morbidity, measured in terms of lost output.

If a person dies prematurely due to pollution, their market and non-market output is lost for the remainder of their life. To illustrate, if a person dies at age 20 the output lost by his or her death is the present discounted value of what he or she would have produced over the remainder of his or her working life. Output at each age is weighted by the probability that a 20-year-old survives to each future age, times the probability that he or she is working at that age. This flow of output is discounted to the present at an appropriate rate of interest (r). In the calculations presented below, average output per worker in the current year is calculated as above (for YLD), but is assumed to grow at a rate (g) that reflects the projected

growth in future labour productivity (For details, see the Appendix). Average worker output can, as above, be modified to include expected non-market output.

To provide an illustration of the magnitude of output losses associated with a YLD, as well as the present value of output lost due to premature mortality, Tables 1-3 show the expected loss in output (both market and non-market), by age, for 2019, as well as the present value of the loss in output, beginning at the same ages, for the project countries. All values are expressed in 2019 USD. They can be converted to international (PPP) dollars by multiplying by an appropriate factor.

The values for a YLD reflect GDP and employment in 2019, as well as estimates of labour's share of GDP for 2019.[27] Labour force participation rates, by age, are from the International Labour Organisation.[28] In all cases, the non-market output produced by an adult who is not working is assumed to equal 0.3 times the average output per worker.

The present value of lost output is calculated using survival rates estimated using current life tables for each country and the same labour force participation rates, by age, used to calculate the expected loss in output associated with a YLD.[29] The effective annual rate at which future output is discounted (see Appendix) equals (1+g), where g is the rate of growth average output per worker, divided by (1+r), where r is the social discount rate. Following the Lancet Commission Report on Pollution and Health, we present the present value of future output using two assumptions about (1+g)/(1+r). In Table 2 we assume that (1+g)/(1+r) = 1/(1.015). In Table 3 we assume that (1+g)/(1+r) = 1/(1.03).[30]

Introduction and underlying assumptions – IQ approach

Estimates of the present value of output lost due to premature death can also be used to estimate the value of a loss in IQ points once the link between IQ and lifetime earnings (output) has been established. If x represents the fraction of lifetime earnings (output) lost due to the loss of one IQ point, then the monetary value of this loss equals the present value of lifetime earnings (output) multiplied by x. To illustrate, if exposure to pollution at age 5 reduces IQ and, subsequently, lifetime earnings, by 2%, the value of this could be estimated by multiplying the present value of lifetime earnings in Table 2 discounted to age 5 by 0.02. To illustrate, in Kenya this would be 0.02 x USD 63,255 or, in PPP terms, 0.02 x USD 63,255 x 2.08 International Dollars.

[27] Robert C Feenstra and Marcel P Inklaar, Robert C., Robert Timmer, 'The Next Generation of the Penn World Table' (2015) 105 American Economic Review 4 <https://www.rug.nl/ggdc/productivity/pwt/?lang=en> accessed 14 November 2021; ILO, 'ILO Data Explorer' <https://ilostat.ilo.org/data/> accessed 13 November 2021.
[28] ILO (n 27).
[29] WHO, 'GHO | By Category | Life Tables' WHO.
[30] Philip J Landrigan and others, 'The Lancet Commission on Pollution and Health' [2017] The Lancet <http://linkinghub.elsevier.com/retrieve/pii/S0140673617323450> accessed 22 October 2017.

Tables for estimation of costs of inaction

Table 1. Values per YLD in 2019 USD

	Ethiopia	Gabon	Kenya	Madagascar	Mali	Senegal	Tanzania	Zambia	Zimbabwe
< 1	0	0	0	0	0	0	0	0	0
1-4	0	0	0	0	0	0	0	0	0
5-10	0	0	0	0	0	0	0	0	0
10-15	0	0	0	0	0	0	0	0	0
15-19	691	4,911	2,099	393	920	2,037	1,004	941	981
20-24	795	5,648	2,414	451	1,058	2,342	1,155	1,082	1,128
25-29	824	5,851	2,500	468	1,096	2,427	1,196	1,121	1,169
30-34	835	5,931	2,535	474	1,111	2,460	1,213	1,137	1,185
35-39	859	6,102	2,608	488	1,143	2,531	1,247	1,169	1,219
40-44	847	6,017	2,572	481	1,127	2,496	1,230	1,153	1,202
45-49	850	6,034	2,579	482	1,130	2,503	1,234	1,156	1,206
50-54	849	6,032	2,578	482	1,130	2,502	1,233	1,156	1,205
55-59	837	5,945	2,541	475	1,114	2,466	1,215	1,139	1,188
60-64	817	5,802	2,479	464	1,087	2,406	1,186	1,112	1,159
65+	692	4,912	2,099	393	920	2,037	1,004	941	981

Note: These calculations are based on the Appendix. All figures reflect labour force participation rates from the International Labour Organization and assume that non-market output equals 35% of each country's GDP. All figures are in 2019 USD.

Table 2. Present Discounted Value of Lifetime Output per person in 2019 USD, Low Discount Rate

	Ethiopia	Gabon	Kenya	Madagascar	Mali	Senegal	Tanzania	Zambia	Zimbabwe
< 1	22,040	137,897	58,682	10,712	25,083	63,507	30,175	22,675	20,967
1-4	23,220	144,421	61,583	11,278	27,106	66,661	31,780	24,064	22,211
5-10	23,917	148,331	63,255	11,614	28,536	68,532	32,746	24,924	22,931
10-15	24,416	151,357	64,547	11,918	29,371	69,961	33,504	25,475	23,396
15-19	24,907	154,680	65,816	12,189	30,068	71,338	34,125	25,966	23,906
20-24	23,431	145,611	62,020	11,478	28,337	67,112	32,123	24,503	22,585
25-29	21,613	134,546	57,347	10,613	26,211	61,956	29,672	22,722	20,984
30-34	19,649	122,587	52,324	9,678	23,921	56,370	27,039	20,804	19,249
35-39	17,587	110,141	47,134	8,696	21,516	50,501	24,290	18,829	17,469
40-44	15,391	96,986	41,691	7,649	18,941	44,226	21,359	16,749	15,620
45-49	13,173	83,761	36,219	6,596	16,317	37,885	18,386	14,666	13,809
50-54	10,859	69,949	30,434	5,492	13,550	31,251	15,253	12,440	11,876
55-59	8,427	55,242	24,113	4,330	10,620	24,307	11,925	10,006	9,739
60-64	5,830	39,070	17,036	3,069	7,442	16,889	8,315	7,215	7,169
65+	2,970	20,429	8,850	1,617	3,862	8,673	4,275	3,848	3,909

Note: These calculations are based on the Appendix, assuming that the discount rate (r) is 1.5 percentage points higher than the rate of growth in output per worker (g). All figures are in 2019 USD.

Table 3. Present Discounted Value of Lifetime Output per person in 2019 USD, High Discount Rate

	Ethiopia	Gabon	Kenya	Madagascar	Mali	Senegal	Tanzania	Zambia	Zimbabwe
< 1	18,909	117,607	50,149	9,184	22,073	54,284	25,880	19,597	18,088
1-4	19,764	122,577	52,272	9,598	23,581	56,633	27,061	20,597	18,950
5-10	20,475	126,925	54,128	9,994	24,630	58,668	28,096	21,363	19,620
10-15	21,196	131,629	56,008	10,372	25,587	60,707	29,039	22,096	20,344
15-19	20,234	125,743	53,557	9,912	24,470	57,955	27,740	21,159	19,503
20-24	18,940	117,904	50,254	9,300	22,969	54,293	26,002	19,911	18,388
25-29	17,473	109,012	46,530	8,606	21,272	50,127	24,045	18,501	17,117
30-34	15,871	99,391	42,534	7,847	19,416	45,572	21,920	16,992	15,764
35-39	14,094	88,814	38,178	7,005	17,346	40,500	19,559	15,338	14,304
40-44	12,241	77,837	33,658	6,129	15,163	35,206	17,085	13,629	12,832
45-49	10,240	65,963	28,699	5,179	12,778	29,470	14,383	11,731	11,199
50-54	8,064	52,863	23,075	4,143	10,163	23,260	11,412	9,576	9,319
55-59	5,661	37,941	16,543	2,980	7,227	16,400	8,075	7,006	6,962

60-64	2,927	20,132	8,721	1,594	3,806	8,547	4,213	3,792	3,852
65+	18,909	117,607	50,149	9,184	22,073	54,284	25,880	19,597	18,088

Note: These calculations are based on the Appendix, assuming that the discount rate (r) is 3 percentage points higher than the rate of growth in output per worker (g). All figures are in 2019 USD.

Method

The underlying method for the economic valuations is attached as an Annex. Certain assumptions are worth outlining here. YLD values are calculated for the population as whole using labour participation rates for all 5-year age groups age 1–64 and are not discounted. Results of YLD calculations represent lost productivity for a single year.

Productivity losses attributable to IQ decrement are calculated as 2 % of present value discounted lifetime earnings for a 5 year old. The higher of the two discount rates given above is used.

ANNEXES

Annex 1: Primer on Costs of Inaction
Annex 2: Summary of underlying assumptions
Annex 3: Manganese supplement

ChemObs

Economic Cost of Inaction Calculator

Documentation

Anney 1

Primer on the Costs of Inaction

About the Authors

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About RFF

Resources for the Future (RFF) is an independent, nonprofit research institution in Washington, DC. Its mission is to improve environmental, energy, and natural resource decisions through impartial economic research and policy engagement. RFF is committed to being the most widely trusted source of research insights and policy solutions leading to a healthy environment and a thriving economy.

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Sharing Our Work

Contents

1. Introduction

To support the African ChemObs project (the Integrated Health and Environment Observatories and Legal and Institutional Strengthening for the Sound Management of Chemicals in Africa), we provide a critical review of methodologies for valuing the health damages from policy inaction associated with chemical exposures. In particular, we discuss how disability-adjusted life years (DALYs) and IQ loss should be valued. We conclude by providing advice on communicating the costs of inaction and the benefits and costs of action to policymakers.

By the *social costs of inaction*, we mean the private or market costs, as well as the external costs, from pollution exposures compared with no exposure. Knowledge of these damages can then lead to policies designed to force investment and operating decisions in the market to account for (internalize) such costs/damages. The costs of inaction can be distinguished from the benefits and costs of action. The benefits of action are the value of, for example, the health improvements from regulations or other forms of action. These actions usually come with a cost of resources to bring about such actions. The *net* benefits to society of an action are the benefits minus the costs of action. In general, as regulations of chemicals rarely eliminate all exposures, the costs of inaction generally exceed (in absolute terms) the benefits of action.

2. Approach

The material in this report is based on a review of the available conceptual and empirical literature on social cost estimation—in the context of health—to identify the strengths and weaknesses of the various methods used to estimate these costs. Advice on how to communicate results draws from a different literature on communicating costs and benefits to policymakers—in the United States, in other developed countries, and in developing countries—as well as our own experience and the experiences of ChemObs team members. We considered use of this report by project developers, planners, research analysts, and government policymakers.

3. Health Valuation

3.1. Overview of Approaches

The standard approach to estimating the health effects of environmental pollution is the damage function approach. Epidemiologists estimate the association between pollution and premature mortality, morbidity, or other health effects, such as the impact of pollution on cognitive development or functioning. These impacts may, in turn, be valued using either the cost-of-illness or willingness-to-pay approach.

The cost-of-illness (COI) approach to health valuation measures the direct medical expenditures associated with disability or illness, including hospital, physician, and medication costs, as well as long-term rehabilitation costs. The indirect costs of illness include time lost from work due to illness and the value of caregivers' time (Landrigan et al. 2018). It also includes losses in productivity over an individual's lifetime due to chronic medical conditions or a loss in cognitive function. When properly measured, these costs include out-of-pocket costs borne by affected individuals, as well as costs reimbursed by insurance or paid for by the government. When a person dies prematurely due to pollution, the COI approach measures this loss in human capital by the individual's lost output. For workers, this output is their earnings over the remainder of their working life. For people not in the workforce, there are approaches to measuring their "earnings," such as using market wages for providing services in the home (e.g., child-rearing services).

The COI approach does not capture the discomfort caused by illness, including the physical burden borne by people who do not receive treatment for their condition. The COI approach also fails to capture the anxiety and loss in enjoyment that a person facing death risks suffers, as well as losses by family members related to these risks are after his death.

The willingness-to-pay (WTP) approach is the theoretically correct approach to measure preferences of people to avoid being ill or dying prematurely. There are *revealed preference* approaches to capture these preferences through statistical analyses of individual behavior (such as by estimating the wage premium paid to workers in more risky jobs) and *stated preference* approaches, which use survey techniques to elicit and monetize preferences for improved health by posing hypothetical questions (Cropper et al. 2011). These approaches can, in principle, capture the pain, suffering, and loss of enjoyment that the COI approach cannot capture. An individual's WTP is, however, necessarily limited by income. Taking the distribution of income as a given is consistent with measuring the benefits to society of improving health (under modern welfare economics), but this caveat is important for understanding the context of such metrics. The WTP approach also may fail to

capture costs that a person does not pay for, such as the cost of treatment in a government clinic.

3.2. Valuing Mortality

Epidemiological studies often link pollution exposures to mortality rates; for example, they may estimate how risk of death, by age and cause, is increased by exposure to ambient air pollution ($PM_{2.5}$), relative to some minimum level of exposure (GBD 2017 Risk Factor Collaborators 2018). The damages attributable to the current level of air pollution in a city (i.e., the costs of inaction) can be expressed as the number of deaths, by age and cause, attributable to current air pollution levels. These deaths can be expressed as death rates, such as 10 in 10,000 people in a city. The WTP approach asks how much people would pay to reduce their risk of death from this baseline.

The WTP approach estimates what a person would pay for a reduction in risk of death—for example, for a reduction in risk of death from 10 in 10,000 to 9 in 10,000. It sums these WTPs across individuals to determine what 10,000 people would pay for risk reductions that sum to one "statistical life." This sum, termed the value of a statistical life (VSL), is multiplied by the number of statistical lives associated with air pollution to determine the cost of premature mortality associated with, for example, current $PM_{2.5}$ levels using the WTP approach. To illustrate, if each person in the city were willing to pay $25 to reduce the risk of death from 10 in 10,000 to 9 in 10,000, 10,000 people together would pay $250,000 for risk reductions that sum to one statistical life being saved. This implies a VSL of $250,000.

The COI approach, in contrast, would value the statistical lives lost due to pollution by the loss in output associated with each death. This would, in general, vary with age at death and would be measured by the present value of future output lost when a person dies prematurely.

3.2.1. The WTP Approach

In the mortality context, stated preference studies confront respondents with hypothetical situations asking them, for example, what they would pay for a medicine that would reduce the risk of death from 10 in 10,000 to 9 in 10,000 over the coming year. The revealed preference literature infers WTP from studies of wage differentials in the labor market that indicate how much workers must be paid to work in jobs carrying higher risks of death (Cropper et al. 2011).

Although dozens of WTP studies to value mortality risks have been conducted in Organisation for Economic Co-operation and Development (OECD) countries, fewer studies have been conducted in low- and middle-income countries (LMICs). While in-country studies are preferred, the lack of such studies implies that it is often necessary to transfer VSL results from high- to low-income countries. The most prevalent approach is to adjust the VSL according to per capita income (Robinson, Hammitt, and O'Keeffe 2019; Narain and Sall 2016). The VSL used by the OECD, $3.83 million (2015 US$), is approximately 100 times per capita income. The official VSL of the US Environmental Protection Agency (EPA), $9.4 million (2015 US$), is approximately 160 times per capita income. The ratio of the VSL to income, divided by 100, represents the fraction of income that would be given up for a 1 in 10,000 reduction in risk of death. EPA's VSL implies that a 1 in 10,000 reduction in risk of death is worth 1.6 percent of income; the OECD value implies that it is worth 1 percent of income (see Appendix B). Well-executed studies of the VSL in LMICs imply that the ratio of the VSL to per capita income falls as per capita income falls (Hammitt and Robinson 2011).

A recent Gates Commission study, after reviewing the international VSL literature, suggests transferring EPA's VSL to LMICs using an income elasticity of 1.5 (Robinson et al. 2019; Robinson, Hammitt, and O'Keeffe 2019). Table 1 shows the ratio of the VSL to per capita income implied by this approach. It presents gross national income (GNI) per capita in international (purchasing power parity, or PPP) dollars, as well as the VSL in international (PPP) dollars. The ratio of the VSL to per capita income (Y) is 21 times per capita income for a country with a per capita GNI of $1,000 international dollars, 48 times per capita income for a country with per capita income of $5,000 international dollars, and 67 times per capita GNI for a country with per capita GNI of $10,000 international dollars. Note that once the VSL/Y ratio is determined, it can easily be used to solve for the VSL at market exchange rates, since the VSL/Y ratio is identical in PPP and market exchange rate (MER) terms. Applying this transfer implies a VSL/Y ratio of 43 for Ghana, 42 for Zambia, 39 for the Côte d'Ivoire, and 37 for Kenya.

3.2.2. The COI Approach (also called the human capital approach)

The cost-of-illness approach uses forgone earnings, rather than the VSL, to measure the value of premature mortality. This is often referred to as the human capital approach, since an individual's output (or income) is often used as a measure of the person's human capital, or accumulated skills and knowledge. To illustrate, if a person dies at age 25, the output lost by his death is the present discounted value of what he would have earned (produced) over the remainder of his working life.

Earnings (output) at each age is weighted by the probability that a 25-year-old survives to each future age, times the probability that he is working at that age. This flow of output is discounted to the present at an appropriate rate of interest. (For details, see Appendix A.)

One way to measure earnings at each age is to use results from a national survey that records labor earnings; however, such data may not be available for all countries. In the Lancet Commission report (Landrigan et al. 2018), earnings per worker were approximated by output per worker, calculated by multiplying labor's share of gross domestic product (GDP) by GDP and then dividing by the number of workers employed. This number can also be adjusted to reflect the value of nonmarket output produced by labor, as described in Appendix A.

The value of the human capital lost when a person dies clearly depends on age at death. Other things equal, the output lost when a 60-year-old dies is less than the output lost when a 25-year-old dies: the human capital measure of mortality varies with age at death. Does this also hold for the VSL? Whether WTP to reduce risk of death varies with age is an empirical question—it depends on the utility people receive from living longer, as well as their wealth. How the VSL varies empirically with age is not well established (Krupnick 2007). For this and other reasons, policymakers in the United States (and other countries) apply the same VSL to deaths of all ages.

3.2.3. International Examples

The Global Burden of Disease program at the Institute for Health Metrics and Evaluation regularly publishes, by country, estimates of deaths associated with environmental risk factors, including outdoor air pollution, household air pollution, unsafe water and sanitation, and exposure to lead (GBD 2017 Risk Factor Collaborators 2018). The Lancet Commission report (Landrigan et al. 2018) valued the mortality burden associated with these pollution sources, by country, using both the human capital and VSL approaches to value damages. Table 2 shows the human capital losses as a percentage of GDP, aggregated by World Bank Income Group. Table 3 shows mortality losses, valued using the VSL, as a percentage of GNI, aggregated by World Bank Income Group.

A comparison of Tables 2 and 3 highlights the difference between the two approaches. For low-income countries, aggregating across pollutants, human capital losses range from 1.33 to 1.9 percent of GDP, depending on the rate used to discount future earnings to the present. The corresponding figure for mortality losses valued using the VSL is 8.33 percent of GNI. Two factors explain the differences: human

capital losses last only over a person's working life, whereas the same VSL is applied to premature deaths at all ages. Table 2 uses the International Labour Organization's definition of working life as 15 to 64 years. A high percentage of deaths from environmental exposures occurs among persons over 65. The second factor is that because the VSL captures losses beyond productivity (i.e., earnings) losses, the VSL is a multiple of forgone earnings at all ages.

3.3. Valuing Morbidity

The difficulty in valuing morbidity lies in finding epidemiological studies linking pollution to specific illnesses and then valuing the consequences of these illnesses using COI or WTP studies. This is made difficult by the large number of illnesses associated with pollution and, for a given illness, variation in the severity and duration of the illness relative to the small number of valuation studies available.

One solution is to translate illness into years lived with disability (YLDs). Disability weights measure the level of disability associated with a particular disease (or condition), where a disability weight of 0 indicates no disability and a disability weight of 1 equals death (Salomon 2010). Equivalently, (1 – disability weight) indicates the fraction of a year in good health lost due to the disease. For example, a case of mild chronic obstructive pulmonary disease (COPD) might have a disability weight of 0.46; a severe case of COPD, a disability weight of 0.77. The Global Burden of Disease (GBD) estimates the YLDs, by age and gender, associated with 354 diseases for 195 countries. The GBD also estimates the fraction of YLDs, by disease and country, associated with various environmental risk factors (GBD 2017 Risk Factor Collaborators 2018).

One approach that has been taken to valuing YLDs is the human capital approach (see Appendix A). This assumes that the value of a YLD equals the average value of income (or output) per worker in the country, multiplied by the probability that a person is working. For example, the value of a YLD experienced by a 60-year-old would equal average labor income (output) multiplied by the probability that a 60-year-old is working. This value could be modified to allow for the value of nonmarket output (see Appendix A). This approach assumes that disability weights reflect an inability to work or productivity lost while working.

The human capital approach to valuing YLDs simplifies the valuation of illness but has limitations. It does not capture the medical expenditures (direct costs) associated with illness, nor does it capture pain and suffering. To estimate medical costs requires attributing medical costs to specific illnesses, and then attributing

these illnesses to pollution. The GBD study provides a link between YLDs and pollution but does not provide an estimate of medical costs by country and disease. Such estimates may be available for some countries based on survey or administrative data. For example, estimates are available for the United States (Dunn et al. 2015) and other OECD countries (OECD 2013). Estimates of hospitalization costs, by disease, are available for India (Kastor and Mohanty 2018) and may be available for other countries.

3.3.1. Valuing DALYs

In describing the burden of ill health associated with pollution and other risk factors, YLDs are often added to years of life lost (YLLs) to calculate disability-adjusted life years (DALYs) lost. YLLs are measured by remaining life expectancy, based on life tables for the country in question. For example, a person who dies in India at age 25 has a remaining life expectancy of 48 years; hence his death is associated with 48 YLLs. To illustrate the calculation of DALYs, in India in 2017 the GBD estimates that 5.4 million YLDs and 1.24 million deaths were associated with particulate air pollution (both ambient and household air pollution). The 1.24 million deaths resulted in 31 million YLLs, implying that air pollution was associated with 36.4 million DALYs in India in 2017, about 8 percent of all DALYs (India State-Level Disease Burden Initiative Air Pollution Contributors 2018).

How should DALYs be monetized? If a human capital approach is used to value premature mortality and also YLDs, then by adding the monetized value of YLDs to the value of premature deaths, measured by the present value of lost output, one has valued DALYs. Using the approach described in Appendix A, in India, the present value of output (human capital) lost due to premature mortality associated with particulate air pollution was US$20.27 billion. The value of output lost due to YLDs was US$8.17 billion, implying a total loss of US$28.44 billion, or about 1.2 percent of India's GDP.

How can DALYs be valued using a WTP approach? It has been suggested that DALYs should be valued by apportioning the VSL into a value per statistical life year (VSLY) and using the VSLY to value each DALY. In practice, the VSLY is computed based on the mean age of respondents in a stated preference survey or the mean age of workers in a compensating wage study. The VSL is divided by the (discounted) remaining life expectancy of a person of the mean age to produce the VSLY. For example, if the VSL is $1,200,000 and the discounted remaining life expectancy of the average worker in the compensating wage study is 30 years, the VSLY is $40,000. A premature death at any age is then valued by multiplying the YLLs lost by the VSLY. A 25-year-old with 52 life years remaining would be assigned a value of 52 times the VSLY ($2,080,000), whereas a 75 year-old with 11 life years

remaining would be assigned a value of $440,000. YLDs are valued by multiplying each YLD by the VSLY.

There are several issues with this approach. Valuing premature mortality using the VSLY implies that the value of a premature death declines with age, since the VSLY is constant and remaining life expectancy (YLLs) declines monotonically with age. The main criticism of this method of valuing premature mortality is lack of evidence that WTP to reduce risk of death declines monotonically with age (Krupnick 2007). To value YLDs using a metric that measures WTP to avoid risk of death is problematic, given that YLDs measure morbidity rather than mortality.

3.4. Valuing Cognitive Impairment

Much of the ChemObs project focuses on exposures to heavy metals. Some of these, such as lead, but also air pollutants such as fine particles ($PM_{2.5}$), can affect cognitive development, lower IQ, and reduce a child's potential for learning (Brockmeyer and D'Angiulli 2016). For lead, there is a literature linking blood lead levels to test scores and performance on IQ tests (Grosse et al. 2002; Lanphear et al. 2005). IQ, in turn, has been shown to affect lifetime earnings. Other costs associated with lead exposure include the costs of treatment to reduce high blood lead levels (chelation therapy) and the costs of additional schooling for children with high blood lead levels.

In the literature linking lead exposure to future earnings through its effect on IQ, there are at least two channels of effects: the effect of IQ on earnings and on the amount of education attained. Studies in the United States suggest that the total impact of a 1-point reduction in IQ is to reduce annual earnings between 0.75 and 0.9 percentage points for people in their early 30s and by about 1.4 percentage points for people in their early 50s (Grosse 2007). This, of course, reflects outcomes in US labor markets. A recent expert elicitation of US and Canadian labor economists to estimate the effect of IQ on earnings in India (Lutter et al. 2017) found that a 2-point decrease in IQ was associated with a 2 percentage point reduction in earnings each year from age 25 to age 60.

The above results suggest that a rough estimate of the impact of an IQ point on future earnings is about 1 percentage point. This can be applied to estimates of future earnings computed using the methods for estimating the present value of future earnings described in Appendix A.

4. Communicating to Policymakers

It is one thing to generate technical documents on the costs of inaction or the costs and benefits of possible government actions to reduce pollution. It is quite another to successfully communicate those results to appropriately and persuasively influence policymakers, who may lack economics training and are subject to tight timelines and influences from many sides. This section of the primer offers our thoughts on communication challenges and strategies, developed from interviews, our experiences, and what literature is available.

4.1. Prerequisites

It should go without saying that the analyses need to be on point, methodologically sound, transparent, and clearly written, and they should have executive summaries with clear headlines that can appeal to policymakers who may be unable to read the entire analysis. In addition, important assumptions need to be highlighted for transparency.

Also, the baseline must be clear and reasonably accurate. This is true both for a study that is describing the costs of inaction—the damages that will occur if pollution is not remediated—and for cost-benefit analyses (CBAs) that describe the costs and benefits of policies to reduce pollution. The baseline is what changes as a result of a rule or other government action. Costs and benefits are measured from the policy or activity-induced change to the baseline. The current baseline is factual and therefore can be checked (although a future baseline is obviously not observable). Errors in characterizing the current situation can seriously damage a study's credibility with decisionmakers.

4.2. What to Communicate

In this section, we consider the substantive issues of effectively communicating a study of the costs of inaction, a CBA, or other forms that a policy analysis might take.

In communicating pollution damages in a cost-of-inaction study, or benefits (i.e., reduced damages) in a CBA, damages (or benefits) should first be described in physical terms. This might include morbidity, mortality, impacts on IQ, or other impacts. It is useful to present physical impacts (when appropriate) by age, gender, and the geographic region in which they occur. When valuing these impacts, it may be prudent to present both conservative estimates of damages—e.g., estimates of the costs of illness associated with morbidity and earnings losses associated with premature mortality—as well as what economists call welfare benefits, which include

what people would pay to reduce both the monetary and nonmonetary costs of illness, such as pain and suffering.

When estimating welfare benefits, such as what people would pay to reduce their risk of dying, it is often necessary to transfer estimates from other countries to the country where the CBA is being conducted—that is, to use benefits transfer. Benefits transfer (Johnston and Rosenberger 2009; Czajkowski et al. 2017) refers to using analyses, data, or results from one setting to apply to another setting or context. A widely practiced benefits transfer is to apply a value of statistical life (VSL) estimated from one country, such as the United States, adjusted for income differences, to an analysis in a country that does not have studies estimating the VSL for its own population. Multicountry comparisons of the burden of disease and health impacts of pollution (Narain and Sall 2016; Landrigan et al. 2018) use such income elasticities to make these transfers.

One way to avoid monetizing the benefits of a policy, especially when they can be expressed using a single metric, such as lives saved or DALYs avoided, is to use cost-effectiveness analysis (CEA). CEA divides the costs of the policy by a measure of effectiveness, such as lives saved, to obtain a cost per life saved. When computed for different options of an action or across several actions, one looks for the option with the lowest cost per effectiveness metric. This is consistent with a Eurocentric point of view that starts with setting targets for environmental improvements and then seeks to meet those targets at the lowest possible cost.

But this focus comes with several disadvantages. The main advantage of CBA is that it provides the net benefits of an action. Actions with positive net benefits improve social welfare—in other words, the efficiency of the allocation of resources. CEA lacks this normative element, although cost-effectiveness targets or benchmarks appear in the literature, along with the recommendation to reject options that fail to meet the target. But such targets are generally arbitrary. The second disadvantage is the construction of the effectiveness measure. Government actions usually deliver benefits over a number of physical endpoints, such as premature deaths and a variety of morbidity effects. Standard practice is to pick the most important endpoint as the effectiveness measure, but this leaves out the other endpoints. Seen in this light, the advantage of a CBA is that all the endpoints, in principle, are included, weighted by their monetary value. When a policy, action, or rule has one major metric, such as CO_2 emissions, and targets are set for that metric, then CEA can be used to identify the lowest-cost way to meet the target.

The rate of return (ROR) on investment is another popular metric. The rate of return is calculated to be consistent with the interest earned (i.e., the benefits realized)

from costs "invested" in the policy. This type of metric may appeal to finance ministries more than a CEA or net benefit metric, but it is just another way of expressing net benefits. A further advantage is that the ROR on a government action can be readily compared with that of private projects or returns on financial investments, such as bonds, to help benchmark the efficacy of government actions.

Another issue is whether, and how, to communicate uncertainties in an analysis. It is a perennial complaint in reports from the U.S. National Academy of Sciences that government CBAs should do more to address uncertainties in benefits and costs (Abt et al. 2010). EPA's Council for Regulatory Environmental Modeling (CREM) has initiated several projects with implications for the treatment of uncertainty in regulatory impact analysis (RIA), such as a draft guidance document on environmental models (Gaber et al. 2009), an online Models Knowledge Base, and a series of regional seminars.

The first question an analyst must address is whether to present uncertainties in damages (benefits) and costs at all because of the complexities this adds to the narrative. The alternative is to present only best estimates, or perhaps to present uncertainty analyses in appendixes, where they can be ignored if desired. The latter approach emphasizes the analyst's best judgment but forecloses the judgment of the decisionmaker over uncertain outcomes. For instance, it is sometimes observed that decisionmakers are risk averse, meaning, by one interpretation, that they want to make the decision on the basis of a worst-case set of assumptions or forecasts. Such an approach is foreclosed unless uncertainty distributions are provided.

If uncertainties are to be presented, the next question is which type of uncertainties. There are both quantifiable uncertainties, which include statistical and model uncertainties, and unquantified uncertainties, which can be described qualitatively. A useful set of qualitative descriptors is provided by the Intergovernmental Panel on Climate Change (IPCC; Mastrandrea et al. 2010).

Statistical uncertainties refer to the error bounds around estimated relationships; these can also be error bounds around collections of study results, as in a meta-analysis. Model uncertainties refer to differences in results from different approaches taken to a problem. For instance, a concentration-response relationship could be estimated using a variety of assumptions for the shape of that relationship. While the results from each assumed shape (e.g., linear, log-linear, quadratic) can be described with their statistical errors, the differences in results across these assumed shapes can be described as model uncertainty.

Given that uncertainties of various types are to be presented, another question is in what form they should be presented. This issue is taken up below in the section on "How to Communicate to Decisionmakers."

4.3. With Whom to Communicate

Our interviews reveal that when groups outside of government are performing CBAs and other analyses, a local champion—a person with credibility and access to the senior decisionmakers relevant to the analyses in question—is needed.

Even so, based on our own experience, we have found that it is far easier to gain access to the top bureaucrats (i.e., civil servants) than to the political appointees at the top of an agency. The top bureaucrats typically have long tenures and are therefore more likely to be in networks both inside and outside the government. They also tend to be more technically oriented than their political bosses and thus more skilled at understanding analyses. In addition, such civil servants will rise to positions of importance in an agency precisely because they are good at communicating to political appointees above them. Depending on the government's norms, such top bureaucrats may also be able to consult on outside projects.

CBAs are often performed within agencies, as well. For instance, the US government requires that all major proposed and final rules be accompanied by a RIA, which includes a CBA. Trained economists often develop the RIAs and have direct access to the top civil servants and political appointees in their agencies. Access to ultimate decisionmakers may be limited, however, particularly for major controversial actions that are decided at the highest levels of the government.

Government decisionmakers are, of course, not the only group with which to communicate. Legislators who may be writing bills on the topic are an equally important audience. And public opinion, particularly in democratic systems, can provide leverage for implementing a policy. Thus a media strategy may be needed. Our interviewees recommended op-eds and news stories using various digital media platforms and covering all the major languages in a country. Meeting local editors will aid in getting pieces published, as will coauthoring such pieces with local champions of the work or local writers.

4.4. When to Communicate

For in-house CBAs, governments with extensive administrative procedures will have schedules for making the rules (whether in proposed or final form public, schedules

for developing CBAs for rules or other actions, and possibly opportunities for public comment, setting their scheduling vis-à-vis the overall regulatory process.

Institutions or individuals offering analyses from outside the government should want those analyses made available as early in the regulatory process as possible to help shape decisions. But not all analyses are of single rules or actions. For instance, the Copenhagen Consensus Center (2017) offers estimates of the return on investments for a large list of possible actions a government could take. Such analyses are designed to help a government set priorities. Thus ideal timing for these priority-setting CBAs is at the start of new government leadership that is seeking to set its priorities or when new planning cycles begin, such as China's Five-Year Plans. Our interviewees advise that one avoid releasing and pushing such analyses in the lead-up to a leadership change, as the work and findings could become politicized or simply get lost in the politics leading up to an election or other political transition.

4.5. How to Communicate to Decisionmakers

We have already discussed some of the technical issues in communicating analytical information to policymakers evaluating prospective government policies, actions, and rules. This section provides some conclusions about communicating complex analyses to decisionmakers.

The form of the information, the relevance of the material to the expressed interests of the decisionmaker, and the uncertainty tied to the findings are all aspects to consider when addressing decisionmakers. While relevance can seem self-evident from the point of view of the scientist, the manner in which the information's importance is conveyed can influence the decisionmaker's acceptance of the information. Research in the medical field on translating scientific discoveries into public policy has highlighted the importance of getting the attention of and evoking interest in policymakers in order to convey significance (Brownson et al. 2018). One study emphasizes the effectiveness of story-focused briefings based in evidence and personalized in context, as compared with data-focused briefings, in getting policymakers to grapple with the relevance of a discovery in certain scenarios (Brownson et al. 2011).

4.6. Uncertainty

Much of the literature covers communicating about uncertainties. This section is based on Krupnick et al. (2006). Although a great deal of research has been

conducted on the communication of uncertainty and risk, little attention has been focused on the means of communicating the results of such analyses to policymakers. Instead, the orientation has been toward understanding how to present uncertainty to lay audiences and help them put low-probability risks in appropriate context. The issue of communicating uncertainties associated with climate change to policymakers has been garnering increasing attention, with a focus on high-consequence outcomes (Webster 2003). But the issue of communicating uncertainty in a typical regulatory decisionmaking process remains less unexplored (van der Bles et al. 2019).

Experiments on the interpretation of standardized uncertainty language in IPCC reporting by nonprofessional participants suggest that jargon and discrepancies in understanding of probabilities are significant obstacles in disseminating uncertain information to those outside of the academic arena (Patt and Schrag 2003; Budesen et al. 2009). One study by van der Bles et al. (2019) proposes that while potential sources of uncertainty can be broadly broken into four categories—sample variability, measurement inadequacy, knowledge limitation, and expert disagreement—the independent effects of these different sources of uncertainty on audience understanding are not yet understood. Further, the issue of communicating uncertainty specifically in the regulatory decisionmaking process is underexplored.

Psychological research on decisionmaking under uncertainty has uncovered numerous instances in which decisions are influenced simply by the manner in which a problem is presented. Because decisionmakers (and even experts) are just as susceptible to these cognitive biases as the general population, the data analyst's choice of presentation format could influence a policymaker's decision. Furthermore, some evidence suggests that as the emphasis on uncertainties increases, so does the probability that decisionmakers will lose confidence in the overall analysis.

Ways to convey the importance of uncertain variables is one emerging area of interest. Some research has emphasized that the most effective means of communicating information and the associated uncertainties is largely dependent on the type of decisions facing the decisionmaker regarding the topic (Fischhoff and Davis 2014). Research on the effectiveness of different graphic techniques in communicating uncertainties has demonstrated that box-and-whisker plots, probability density functions, and cumulative density functions perform relatively well in allowing a well-educated audience to accurately extract quantitative information. Beyond standard tornado graphs, novel approaches such as radar graphs, cobweb plots, and pairwise scatterplots offer ways to present large amounts of information in an economical manner, although these approaches might be too

complex for a nontechnical audience. Area and volume presentations can be misleading and cause viewers to underestimate large magnitudes and thus should be avoided.

We conducted in-depth interviews with seven former EPA assistant or deputy administrators in which we presented the basic results of the case study we conducted on tightening the US cap on power plant NO_x emissions. Using alternative metrics and graphics, we then solicited their opinions about these presentations. From their responses, a number of observations can be made. First, the interviewees were rather heterogeneous in backgrounds and in their interest in and familiarity with uncertainty assessments. This heterogeneity no doubt led to differences in the ease with which they interpreted alternative metrics and graphics portraying the results of our case study. Therefore, we found it difficult to generalize about the techniques used and challenges encountered in communicating these types of results. Nevertheless, even with the limited sample, interviewees were most comfortable with the use of probability density functions (PDFs) and simple tabular formats, rather than the complex graphics more commonly used by analysts (and favored by us), such as box-and-whisker plots, cumulative density functions, and circle charts. We conjecture that as the number of variables considered increases, the box-and-whisker plot would be increasingly useful and the PDFs less so.

Beyond PDFs and simple tabular formats, the former decisionmakers also favored other graphics. For example, they were particularly interested in the graphic displaying the relative importance of the various factors considered in the uncertainty analysis (Figure 1). On several occasions, they specifically asked about relative importance even before the graphic was presented. The interviewees also were interested in identifying any factors for which uncertainty might be an important issue but that had been excluded from formal uncertainty analysis.

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6. Tables and Figures

Figure 1. Relative Importance of the Factors Considered in the Uncertainty Analysis

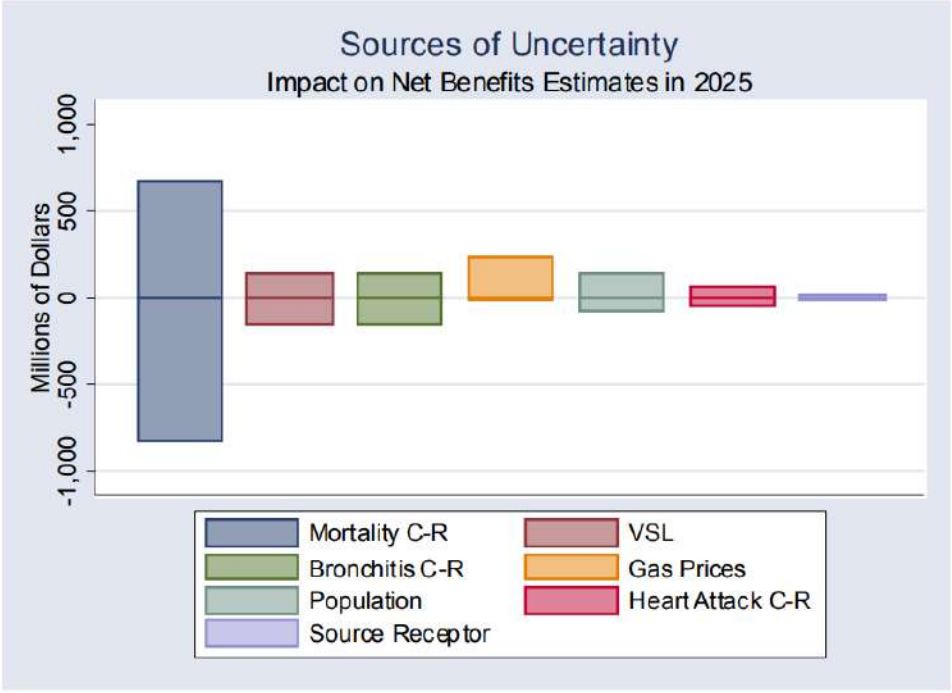

Table 1. Examples of Extrapolated VSL Estimates Using an Income Elasticity of 1.5 to Transfer the US VSL

	GNI per capita (2015 international dollars)					
Approach	$1,000	$5,000	$10,000	$15,000	$20,000	$25,000
Reference VSL = $9.4 million Elasticity = 1.5	$0.021 million (21*GNI per capita)	$0.24 million (48*GNI per capita)	$0.67 million (67*GNI per capita)	$1.2 million (83*GNI per capita)	$1.9 million (95*GNI per capita)	$2.7 million (110*GNI per capita)

Source: Robinson et al. (2019a).

Table 2. Productivity Losses as a Percentage of GDP by Pollutant and Income Group

World Bank Income region	World Bank Income region	UW and US combined[a]	Lead exposure	Total
High income	0.044% (0.048%)	0.0028% (0.0033%)	0.0027% (0.0029%)	**0.050%** **(0.054%)**
Upper-middle income	0.13% (0.15%)	0.019% (0.027%)	0.0054% (0.0059%)	**0.15%** **(0.18%)**
Lower-middle income	0.32% (0.40%)	0.28% (0.40%)	0.012% (0.013%)	**0.61%** **(0.82%)**
Low income	0.62% (0.86%)	0.70% (1.03%)	0.012% (0.013%)	**1.33%** **(1.90%)**
World	**0.092%** **(0.11%)**	**0.033%** **(0.047%)**	**0.0042%** **(0.0046%)**	**0.13%** **(0.16%)**

Source: Landrigan et al. (2018).

Note: Results in parentheses discount future output at the rate of growth in per capita GDP plus 1.5%. Base case results (those without parentheses) discount future output at the rate of growth in per capita GDP plus 3%.

[a] Includes no handwashing with soap.

Table 3. Welfare Damages in Billions of Dollars and as a Percentage of GNI by Pollutant and Income Group (2015 US$)

World Bank Income region	World Bank Income region	UW and US combined[a]	Lead exposure	Total
High income	1,691 (3.52%)	159 (0.33%)	303 (0.63%)	**2,153** **(4.48%)**
Upper-middle income	1,691 (8.37%)	89 (0.44%)	118 (0.59%)	**1,898** **(9.40%)**
Lower-middle income	367 (6.38%)	143 (2.49%)	28 (0.49%)	**538** **(9.36%)**
Low income	18 (4.83%)	12 (3.30%)	0.740 (0.20%)	**31** **(8.33%)**
Total	**3,767** **(5.06%)**	**404** **(0.54%)**	**451** **(0.61%)**	**4,622** **(6.21%)**

Source: Landrigan et al. (2018).

Note: Results in parentheses discount future output at the rate of growth in per capita GDP plus 1.5%. Base case results (those without parentheses) discount future output at the rate of growth in per capita GDP plus 3%.

[a] Includes no handwashing with soap.

Appendix A. Measurement of Output Losses due to Pollution

This appendix describes methods for measuring output losses associated with pollution morbidity (years lived with disability, or YLDs) and mortality (deaths associated with pollution) in a given year (e.g., 2017), following the human capital/cost-of-illness (COI) approach. It is assumed that the user has YLDs and premature deaths associated with pollution by age. Premature mortality is often measured using years of life lost (YLLs); however, the human capital approach calls for valuing the present value of output loss associated with each death, which will vary with age at death, rather than valuing YLLs. Valuing disability-adjusted life years (DALYs) using the human capital/COI approach thus calls for valuing YLDs and premature deaths separately.

The present value of lifetime earnings for a person of a given age represents the output lost if the person dies prematurely. It can also be used to value the impact of a decrement in IQ, if the loss in IQ points is expressed as a percentage reduction in earnings. This is discussed in section A.4.

A.1. Output Losses Associated with Pollution Mortality

We begin by estimating the present discounted value of the loss in gross domestic product (GDP) attributable to mortality associated with pollution in 2017. The loss in GDP in country i in 2017 if a worker dies is equal to labor's share of GDP (α) multiplied by GDP (Y_i), divided by the number of persons who are employed (L_i). We assume that workers of all ages in a country produce the same output per worker. Because not all persons of age j are working, the expected value of GDP per worker for a person of age j (W_{ij2017}) is equal to ($\alpha Y_i/L_i$) times the ratio of the number of workers of age j, L_{ij}, to the population of age j, N_{ij},

$$W_{ij2017} = (\alpha Y_i/L_i)*(L_{ij}/N_{ij}) \quad (1)$$

In our calculations below, we assume that labor's share of GDP (α) is constant over time. We also assume that the ratio of L_{ij}/N_{ij} remains constant over time.

To calculate the loss in market and nonmarket output in 2017, we modify equation (1) to allow for household production. The US Bureau of Economic Analysis

estimates that household production equals 25 percent of GDP (BEA 2019). The comparable estimate for Ghana is 35 percent (Ofosu-Baadu 2015) and for India is 30 percent (Pandey 2001). We therefore calculate W'_{ij2017} as

$$W'_{ij2017} = (\alpha Y_i/L_i)*(L_{ij}/N_{ij}) + \lambda_j(\alpha Y_i/L_i)*[1-(L_{ij}/N_{ij})], \quad (1')$$

where λ_j represents the fraction of output attributable to nonmarket production for a person of age j. For children and the elderly, $(L_{ij}/N_{ij}) = 0$, so the first term in (1′) is zero. We also assume that nonmarket output is zero for children and the elderly. This implies, for example, that $\lambda_j = 0$ for $j < 15$ and $j > 84$, and $\lambda_j > 0$ for $10 < j < 85$.

If a person of age j dies in the current year, her contribution to GDP will be lost for all future years of her working life. To compute the value of GDP lost in future years, we assume that GDP per worker in country i grows at rate g_i. If labor's share of GDP and the fraction of population of working age (L_{ij}/N_{ij}) remain constant for all i and j, this implies that lost GDP at age t of a person currently of age j will equal $(\alpha Y_i/L_i)*(L_{it}/N_{it})*(1+g_i)^{t-j}$. This must be weighted by the probability that an individual would have survived to age t, where $\pi_{ij,t}$ is the probability that a person of age j in country i survives to age t. We therefore weight the loss in GDP in future years by the probability that an individual who dies this year would have survived to each future year of his working life. We discount the value of GDP lost in the future at the annual rate r_i.

Given the previous assumptions, the present discounted value of lost market and nonmarket output for a person of age j in country i who dies in 2017, PV_{ij}, is

$$PV_{ij} = \sum_{t=j}^{84} \pi_{ij,t} \left[\left(\frac{L_{it}}{N_{it}}\right)\left(\frac{\alpha Y_i}{L_i}\right) + \lambda_t \left(1 - \frac{L_{it}}{N_{it}}\right)\left(\frac{\alpha Y_i}{L_i}\right)\right]\left(\frac{1+g_i}{1+r_i}\right)^{t-j}. \quad (2)$$

In practice, equation (2) would be calculated for $j = 0, \ldots, 84$. The value of λ_t would presumably equal 0 for small children (e.g., $t = 0, \ldots, 14$) and would be set equal to a positive value (e.g., 0.3) for larger values of t.

The total output lost due to pollution is the product of PV_{ij} and D_{ij}, the number of deaths due to pollution in 2017 of persons of age j in country i, summed over all j.

A.2. Output Losses Associated with Pollution Morbidity

We compute the lost output due to morbidity associated with pollution in 2017 by multiplying the number of YLDs associated with pollution in 2017 by the expected loss in output per person, which is given by equation (1'). YLDs associated with pollution are assumed to be available by country *i* and age *j*, YLD_{ij}. The output loss associated with morbidity in 2017 for persons of age *j* in country *i*, M_{ij} is given by

$$M_{ij} = W'_{ij2017} * YLD_{ij} . \quad (3)$$

A.3. Data

To compute GDP per worker, gross domestic product (Y_i) (World Bank 2019) is divided by the size of the labor force in country i (L_i) (World Bank 2019) to compute (Y_i/L_i). Labor's share of GDP (α) can be obtained from Penn World Table (Feenstra et al. 2015) or the International Labour Organization (ILOSTAT 2019).

Other parameters that vary by country include the ratio of worker to total population and survival rates. The ratio of worker to total population (L_{ij}/N_{ij}) for each country and age group can be obtained from ILOSTAT (2019). Because only aggregate data are reported for ages 65 and older, (L_{ij}/N_{ij}) can be estimated for each age over 65 by assuming that the worker-population ratio declines linearly from age 65 to age 85, becoming zero at age 85. The annual survival rate from age j to age t in each state, $\pi_{ij,t}$, can be computed from life tables provided by the Global Burden of Disease Study (GHDx 2019).

The present value of lost output depends on the rate of growth in output per worker (g_i) and the discount rate (r_i). As equation (2) indicates, it is the ratio of $(1+g_i)/(1+r_i)$ that determines the present discounted value of future earnings. Determining appropriate values of r_i and g_i for each country is difficult. Should this not be possible, a default is to use the assumptions underlying the Lancet Commission report (Landrigan et al. 2018)—in other words, that the discount rate exceeds the rate of growth in output per worker by (a) 1.5, (b) 3.0 percentage points. This implies that the term $[(1+g_i)/(1+r_i)]^{t-j}$ in equation (2) is replaced by $[1/(1+d)]^{t-j}$, where d = .015 or .03.

A.4. Valuing Output Losses due to Reductions in IQ

An extensive literature links the impact of lead exposure in children to IQ loss (see main text) and values the loss in IQ by its impact on lifetime earnings. If the total impact of the loss of one IQ point is to reduce lifetime earnings by, for example, 1 percent, then the present value of lifetime earnings (equation (2)) can be multiplied by .01 times the number of IQ points lost. An important question is the timing of the earnings loss. One approach is to calculate the loss discounted to the beginning of an individual's working life (i.e., to age 15). Exposure to lead may, however, occur earlier (e.g., between ages 0 and 7), which raises the question of whether the earnings loss should be discounted to the time of exposure. Attina and Trasande (2013) discount lifetime earnings to age 5.

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Appendix B. Willingness to Pay to Avoid Premature Mortality

B.1. Methodology

The willingness-to-pay (WTP) approach captures individuals' preferences for avoiding increases in risk of death by analyzing their behavior in risky situations (the revealed preference approach) or in hypothetical choice situations involving changes in their risk of death (the stated preference approach) (Cropper et al. 2011). An example of the former approach is a labor market study of jobs with different mortality risks, in which the analyst uses knowledge of those different risks and the wages that different jobs command to derive a wage premium (the willingness to be paid) for bearing extra risk (Viscusi and Aldy 2003). A good example of the latter is a survey that asks participants to choose among several hypothetical situations where mortality risks can be reduced at a cost (Lindhjem et al. 2011).

Either of these approaches yields values consistent with the centrality of individual preferences in modern welfare economics, in contrast to the human capital approach discussed in Appendix A, which is regarded as embedded in WTP values. The amount that a person would pay (or accept) in exchange for a small change in risk of death should reflect losses in output when the individual dies—losses that may exceed the person's contribution to gross domestic product (GDP). WTP should also reflect the utility received from living and should therefore exceed the value of output losses.

The value of mortality risk reductions is typically expressed in terms of the value per statistical life (VSL)—the sum of what people would pay for small risk reductions that sum to one statistical life saved. To illustrate, if each of 10,000 people were willing to pay $25 over the coming year to reduce their risk of dying by 1 in 10,000 during this period, on average, one statistical life would be saved and the VSL would equal $25 × 10,000, or $250,000. To evaluate WTP to reduce risk of death by 1 in 10,000, one would multiply the VSL by .0001.

A large body of literature has used revealed and stated preference approaches to estimating the VSL, primarily in Organisation for Economic Co-operation and Development (OECD) countries but also in middle-income countries (Lindhjem et al. 2011; Hammitt and Robinson 2011). Because many countries have no studies representing preferences of their population toward reducing risk of death, analysts typically transfer estimates from one country (a base country) to other countries, adjusting for differences in per capita income (Hammitt and Robinson 2011). This

adjustment is made using the following equation, where Y denotes per capita income and ε denotes the elasticity of the VSL with respect to income:

$$VSLTransfer = VSLBase*(YTransfer/YBase)^{\varepsilon} \quad (B.1)$$

The base value is typically selected based on VSL values in OECD countries. In the Lancet Commission report (Landrigan et al. 2018), a base VSL was selected based on a meta-analysis of stated-preference studies reported by the OECD (2012). This meta-analysis forms the basis of the VSL used by the OECD for policy analysis and is also the basis of VSL transfers by the International Monetary Fund in its computation of health-based fuel taxes (Parry et al. 2014). It is the same baseline value used in the Institute for Health Metrics and Evaluation (IHME)–World Bank study The Cost of Air Pollution (World Bank 2016). The base VSL is $3.83 million 2015 international dollars. In a recent Gates Commission study (Robinson et al. 2019), benefits transfers were based on the US Environmental Protection Agency's (EPA's) VSL of $9.4 million 2015 US$ (equivalent to $9.4 million 2015 international dollars). In transferring the VSL to other countries, per capita income is usually measured in international (i.e., in purchasing power parity, or PPP) dollars. This implies that the VSL is also measured in PPP terms.

What income elasticity (ε) should be used in transferring the base VSL? The elasticity of the VSL with respect to income (ε) represents the percentage change in the VSL for a 1 percent change in income (Y). If the VSL were proportional to income (i.e., if $\varepsilon = 1$), then the ratio of the VSL to income (Y) would be the same in all countries. Using the OECD VSL as a base value implies a ratio of VSL/Y of ~ 96:1. Studies in low- and middle-income countries (LMICs), however, suggest that the ratio of the VSL/Y falls as per capita income falls (Hammitt and Robinson 2011), implying a value of $\varepsilon > 1$.

The exact value of ε to be used should be guided by information on the VSL/Y ratio at different income levels. The ratio of the VSL to income, divided by 100, represents the fraction of income that would be given up for a 1 in 10,000 reduction in risk of death. EPA's VSL implies that a 1 in 10,000 reduction in risk of death is worth 1.6 percent of income; the OECD value implies that it is worth 1 percent of income. Well-executed studies of the VSL in LMICs imply that the ratio of the VSL to per capita income falls as per capita income falls. To achieve a target value of the VSL/Y at a particular income level, the value of ε must increase with the size of the base VSL. The Lancet Commission report used a value of $\varepsilon = 1.2$ in transferring the OECD VSL to low- and low-middle-income countries. The Gates Commission report (Robinson et al. 2019), using the EPA VSL as a base, recommends a value of $\varepsilon = 1.5$.

B.1.1. Treatment of Age

Both the Lancet Commission (Landrigan et al. 2018) and Gates Commission (Robinson et al. 2019) reports use the same VSL irrespective of age at death and use the same VSL for children as for adults. The age distribution of deaths associated with pollution varies widely, raising the question of whether the same VSL should be used to evaluate the deaths of children and the elderly, who lose very different numbers of life years. There is limited and contradictory evidence that VSLs are lower for elderly people than for younger adults (Krupnick 2007). In the case of children, the VSL should be based on parents' WTP to reduce their children's risk of death. There is a growing literature on parents' WTP; however, it consists primarily of studies in high-income countries (Alberini et al. 2010). Because of the lack of studies in low- and middle-income countries and differences in child mortality between high- and low-income countries, we do not recommend transferring studies of parents' WTP to reduce child mortality to low- and middle-income countries.

B.2 References

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Annex 2: Summary of underlying assumptions

Assumption	Strength of evidence	Sources	Reference
Contaminant soil concentration	moderate	Laboratory analysis	--
Contaminant dust concentration	moderate	Laboratory analysis	--
Contaminant water concentration	moderate	Laboratory analysis	--
Contaminant food concentration	moderate	Laboratory analysis	--
Exposed population	low	Assessor estimate	--
Dietary intake	low	Assessor estimate	--
Geometric mean blood lead level children	high	USEPA IEUBK used to calculate BLLs for a range of different exposures	OSRTI US EPA, OSWER, 'Lead at Superfund Sites: Software and Users' Manuals' <https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals> accessed 13 August 2016.
Geometric mean blood lead level adults	high	USEPA ALM used to calculate BLLs for a range of different exposures	OSRTI US EPA, OSWER, 'Lead at Superfund Sites: Software and Users' Manuals' <https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals> accessed 13 August 2016.
Geometric mean blood lead level foetus	high	USEPA ALM used to calculate BLLs for a range of different exposures	OSRTI US EPA, OSWER, 'Lead at Superfund Sites: Software and Users' Manuals' <https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals> accessed 13 August 2016.
IQ decrement (Pb)	high	Peer-reviewed study	Esben Budtz-Jørgensen and others, 'An International Pooled Analysis for Obtaining a Benchmark Dose for Environmental Lead Exposure in Children' (2013) 33 Risk Analysis 45
Incidence of intellectual disability	moderate	WHO report provides typical incidence of ID in a population	Lorna Fewtrell, Rachel Kaufmann and Annette Prüss-Üstün, Lead: Assessing the Environmental Burden of Disease at National and Local Levels (World Health Organization 2003) <http://www.who.int/quantifying_ehimpacts/publications/en/leadebd2.pdf?ua=1> accessed 10 October 2016.
Incidence of cardiovascular disease	moderate	WHO report provides associations of BLLs with CV disease rates	Lorna Fewtrell, Rachel Kaufmann and Annette Prüss-Üstün, Lead: Assessing the Environmental Burden of Disease at National and Local Levels (World Health Organization 2003) <http://www.who.int/quantifying_ehimpacts/publications/en/leadebd2.pdf?ua=1> accessed 10 October 2016.
Disability weights for intellectual disability and cardiovascular disease	high	IHME Disability Weights (from 2017 GBD)	IHME, 'Global Burden of Disease Study 2017 (GBD 2017) Disability Weights | GHDx' <http://ghdx.healthdata.org/record/ihme-data/gbd-2017-disability-weights> accessed 30 October 2021.
IQ decrement (Hg)	moderate	Peer-reviewed study provides IQ decrement associated with Me-Hg ingestion	Joseph V. Spadaro and Ari Rabl, 'Global Health Impacts and Costs Due to Mercury Emissions' (2008) 28 Risk Analysis 603 <http://doi.wiley.com/10.1111/j.1539-6924.2008.01041.x> accessed 16 August 2019.
Urinary concentration of dimethoate	moderate	Peer-reviewed study provides DAP urinary concentrations associated with different levels of exposure	Marc L Rigas, Miles S Okino and James J Quackenboss, 'Use of a Pharmacokinetic Model to Assess Chlorpyrifos Exposure and Dose in Children, Based on Urinary Biomarker Measurements' (2001).
IQ decrement pesticide exposure	moderate	Peer-reviewed study provides IQ decrement associated with different urinary DAP concentrations	David C Bellinger, 'A Strategy for Comparing the Contributions of Environmental Chemicals and Other Risk Factors to Neurodevelopment of Children' (2012) 120 Environmental Health Perspectives 501.
Uniform impact of all OP pesticides on brain development	low	Assumption made in the absence of pesticide specific urinary DAP concentrations	--
Cancer incidence from oral exposure	moderate	IRIS slope factors provide a slope to approximate risk with increased carcinogen exposure	US EPA, 'Integrated Risk Information System' (2016) <https://www.epa.gov/iris> accessed 26 September 2016.

Cancer mortality rates in Africa	moderate	Peer-reviewed study provides typical survival rates from multiple cancers in different African countries	R Sankaranarayanan and others, 'An Overview of Cancer Survival in Africa, Asia, the Caribbean and Central America: The Case for Investment in Cancer Health Services.' [2011] IARC scientific publications 257.
Disability weights for cancers	moderate	IHME Disability Weights (from 2017 GBD)	IHME, 'Global Burden of Disease Study 2017 (GBD 2017) Disability Weights | GHDx' <http://ghdx.healthdata.org/record/ihme-data/gbd-2017-disability-weights> accessed 30 October 2021.
Gross Domestic Product	high	World Bank	https://data.worldbank.org/indicator/NY.GDP.MKTP.CD
Labour's contribution to GDP	high	Penn World Tables	Robert C Feenstra and Marcel P Inklaar, Robert C., Robert Timmer, 'The Next Generation of the Penn World Table' (2015) 105 American Economic Review 4 <https://www.rug.nl/ggdc/productivity/pwt/?lang=en> accessed 14 November 2021.
Labour force participation	high	ILO	ILO, 'ILO Data Explorer' <https://ilostat.ilo.org/data/> accessed 13 November 2021.
Age specific death rates	high	WHO GHO	WHO, 'GHO | By Category | Life Tables' WHO.
Discount rates	high	Annex 1; Peer-reviewed study provides the underlying assumptions and discount rate	Philip J Landrigan and others, 'The Lancet Commission on Pollution and Health' [2017] The Lancet <http://linkinghub.elsevier.com/retrieve/pii/S0140673617323450> accessed 22 October 2017.
Overall method for the valuation of DALYs	high	Annex 1; Peer-reviewed study provides the underlying assumptions and required equations	Philip J Landrigan and others, 'The Lancet Commission on Pollution and Health' [2017] The Lancet <http://linkinghub.elsevier.com/retrieve/pii/S0140673617323450> accessed 22 October 2017.

Annex 3: Manganese supplement

Context

At the request of the Ministry of Water and Forests, the Sea and the Environment of Gabon, an effort was made to include manganese soil exposure into the economic calculator. A review of the epidemiological literature confirmed that the available data are not adequately robust for this chemical and exposure route to be fully integrated. As an alternative the crude model below is presented as an interim measure. Results should be taken as broadly indicative and are not immediately comparable with other results from the calculator.

Exposure Model

Manganese is a naturally occurring element typically found at < 900 mg/kg in surface soils. It is an essential nutrient, though elevated levels of exposure are associated with a number of adverse health outcomes, including Parkinsonism.[1] There are multiple significant exposure routes to manganese including incidental soil and dust ingestion, inhalation of airborne particles, and deliberate ingestion of food and water.[2] Of these, the inhalation dose-response has been most extensively evaluated. A study in Hamilton, Canada for instance found that the odds ratio for Parkinson's disease increased by 3 % for each 10 ng/m^3 increase in manganese particulate levels in ambient air.[3] Oral exposure by contrast is much less well characterized. The USEPA maintains a no observed adverse effect level (NOAEL) for chronic oral exposure to inorganic manganese of 0.14 mg/kg-day, or 10 mg/ day for a 70 kg adult.[4] Though given the limited available data there is some uncertainty around this value, with adverse outcomes having been associated with chronic exposures as low as 0.103 mg/kg-day.[5]

Manganese is ubiquitous in the environment. Elevated exposures typically result as the aggregate dose from multiple routes. This is somewhat distinct from other chemicals assessed by calculator, where a dominant exposure route can often be associated with an adverse response. In the case of lead, for example, exposure to relatively low soil concentrations (~400 mg/kg) alone can result in adverse health impacts. Manganese soil levels however would need to be exceptionally high for the associated dose to be considered elevated. To illustrate, soil and dust incidental ingestion rates are commonly approximated at 30–100 mg/day.[6] Thus for exposure from this source alone to exceed the adult manganese NOAEL of 10 mg/ day, soil manganese levels would need to exceed 10 % (i.e. > 100,000 mg/kg). Additional routes of exposure are therefore highly relevant. To further illustrate, typical dietary manganese exposures in England, the Netherlands and US have been estimated at 2.3–8.8 mg/ day.[7] In

[1] Atsdr, 'TOXICOLOGICAL PROFILE FOR MANGANESE'; Roberto G Lucchini, Christopher J Martin and Brent C Doney, 'From Manganism to Manganese-Induced Parkinsonism: A Conceptual Model Based on the Evolution of Exposure' (2009) 11 NeuroMolecular Medicine 311 <https://link.springer.com/article/10.1007/s12017-009-8108-8> accessed 14 February 2022.

[2] Roberto Lucchini and others, 'Manganese and Developmental Neurotoxicity' (2017) 18 Advances in neurobiology 13 </pmc/articles/PMC6057616/> accessed 14 February 2022.

[3] Murray M Finkelstein and Michael Jerrett, 'A Study of the Relationships between Parkinson's Disease and Markers of Traffic-Derived and Environmental Manganese Air Pollution in Two Canadian Cities' (2007) 104 Environmental Research 420.

[4] Us Epa and Integrated Risk Information System Division, 'Manganese (CASRN 7439-96-5) | IRIS | US EPA'.

[5] Atsdr (n 1); Vanita Sahni and others, 'Case Report: A Metabolic Disorder Presenting as Pediatric Manganism' (2007) 115 Environmental health perspectives 1776 <https://pubmed.ncbi.nlm.nih.gov/18087599/> accessed 14 February 2022.

[6] 'About the Exposure Factors Handbook | US EPA' <https://www.epa.gov/expobox/about-exposure-factors-handbook> accessed 14 February 2022.

[7] USEPA, 'Manganese CASRN 7439-96-5 |IRIS|US EPA, ORD' <https://iris.epa.gov/ChemicalLanding/&substance_nmbr=373> accessed 14 February 2022.

this context, the incremental increase of exposure associated with living on moderately contaminated land becomes more important. Soil manganese concentrations of ~10,000 mg/kg for instance might result in a dose in exceedance of the NOAEL.

Lucchini et al (2007) evaluated the prevalence of Parkinson's in populations in Brescia, Italy where historical ferroalloy production resulted in elevated environmental manganese levels. The authors fiound the prevalence of Parkinson's in the province as a whole to be 407/ 100,000 and within populations residing in the vicinity of ferroalloy plants to be 492/100,000. For comparison, the comparable rate for all Italians was 157.7/ 100,000.[8] A related study assessed soil manganese concentrations in Brescia. The authors evaluated the distribution of manganese in surface soils (< 30 cm) in the municipality of Bagnolo Mella which contains an active ferroalloy plant.[9] Surface soil manganese concentrations ranged from a mean of 693 mg/kg in their control area to a mean of 23,627 mg/kg in residential areas adjacent to the plant. The maximum concentration identified was 79,000 mg/kg.

The proposed model crudely combines the data from the two Brescia studies to estimate an indicative number of excess cases of Parkinson's disease associated with environmental soil manganese levels. As noted above, it is not assumed that soil manganese levels alone are adequate to explain the increased burden of disease. Rather the soil manganese level is employed here as an indicator of wider environmental contamination of various media (e.g. water, food, air). Three exposure scenarios are presented: low, moderate and high.

As a base (low), the Italian prevalence of 157.7 cases per 100,000 population is taken as zero cases of *excess* Parkinson's in the exposed population attributable to manganese. This value of zero excess cases is applied to any area with a mean soil manganese concentration of < 10,000 mg/kg. A second scenario (moderate) considers any area with a mean soil manganese concentration of 10,000–20,000 mg/kg and approximates prevalence of Parkinson's at 407/ 100,000 or 0.002493 excess cases per capita (i.e. 0.00407 minus 0.001577). Finally a third scenario (high) approximates prevalence of Parkinson's at 492/ 100,000 or 0.003343 excess cases per capita. **Table 1** summarizes the theoretical scenarios.

Exposure scenario	Soil concentration	Approximate prevalence of Parkinsonism	Excess Parkinsonism per capita
Low	< 10,000 mg/kg	157.7/100,000	0
Moderate	10,000 – 20,000 mg/kg	407/100,000	0.002493
High	> 20,000 mg/kg	492/100,000	0.003343

Table 1. Theoretical exposure scenarios and estimated excess cases of Parkinson's disease

Economic Valuation

Following the approach used elsewhere in the calculator the IHME disability weight is employed. Because morbidity only is assessed the value of 0.267 for moderate Parkinson's is taken as analogous to a disability adjusted life year (DALY). The economic calculations outlined elsewhere calculate the cost of a DALY in Gabon (2019 international USD) at USD 4,306. Thus the annual economic cost a single of

[8] Roberto G Lucchini and others, 'High Prevalence of Parkinsonian Disorders Associated to Manganese Exposure in the Vicinities of Ferroalloy Industries.' (2007) 50 American journal of industrial medicine 788.

[9] Marco Peli and others, 'Profiles and Species of Mn, Fe and Trace Metals in Soils near a Ferromanganese Plant in Bagnolo Mella (Brescia, IT)' (2021) 755 Science of The Total Environment 143123.

manganese induced Parkinson's would be valued at USD 1,150 in Gabon. The resulting calculation therefore is the total exposed population multiplied by the appropriate value for excess Parkinsonism from Table 1 and multiplied again by USD 1,150. By way of example the annual manganese attributable cost of Parkinson's disease in a highly contaminated city of 100,000 people in Gabon would be USD 384,445 (i.e. 100,000 x 0.002493 x 1,150). For a moderately contaminated city, the value would be USD 286,695. For a low contamination city the associated cost would be USD 0. **Table 2** presents the estimated per capita losses associated with these scenarios.

Exposure scenario	Soil concentration	Per capita losses (USD)
Low	< 10,000 mg/kg	0
Moderate	10,000 – 20,000 mg/kg	2.50
High	> 20,000 mg/kg	3.43

Table 2. Economic per capita losses associated with theoretical exposure scenarios