Controlling Lead Concentrations in Human Blood by Regulating the Use of Lead in Gasoline

After having been emitted at maximum rates in the 1960s and 1970s, lead has become less ubiquitous in industrialized countries as a result of increasingly stringent policies to limit the use of this heavy metal as an anti-knock additive in gasoline. Using a detailed reconstruction of lead emissions in Europe (PbE), of the air concentration of lead in Europe (PbC) and repeated measurements of lead concentrations in human blood (PbB) in Germany since about 1980, we have constructed an empirical model that estimates PbB given PbE. This model is used for 2 purposes: *i)* To estimate PbB levels for the 1960s and 1970s in Germany, when emissions were maximum and monitoring blood levels had not yet begun. It turns out that PbB peak emissions were reaching a mean level, which health officials considered potentially harmful for fetuses and small children. *ii)* To estimate how PbB levels may have developed if regulations of the use of lead in gasoline had been implemented differently. In case of no or delayed regulations, the model estimates that PbB levels well beyond the critical level would have emerged. Thus, the regulation instituted in Germany since the 1970s has reduced significant health hazards.

INTRODUCTION

Since the first half of the 20th century, lead has been emitted into the environment on a large scale through its use as an efficient anti-knock additive in gasoline. Lead in gasoline was the most significant emission source for lead in the atmosphere. According to an analysis by Olendrzynski et al. (1), about 70% of the total emissions in Europe were related to road traffic, 15% to industrial production, 5–10% to power generation and 2% to waste incineration. In the early 1970s, peak lead concentrations in highly affected urban areas in Germany amounted to 2500 ng m^{-3} and more, while in rural areas the concentrations were of the order of 100 ng m^{-3} (not shown).

The health implications of lead in the environment have been subject to many studies. Lovei prepared a review according to which lead, even at low concentrations, was found to have adverse effects such as "retarded the mental and physical development of children, causing reading and learning disabilities; changes in behavior, such as hyperactivity; reduced attention span; and hearing loss, even at low levels of exposure" (2). Effects on blood pressure, hypertension and cardiovascular diseases were shown to be associated with the presence of lead in humans (3, 4). Other implications of enhanced levels of lead in humans are "reduced birth weight, disturbed mental development, spontaneous abortion, or premature birth at relatively low blood lead levels". A distinct effect on the IQ of school-age children was identified (5); an increase in the lead concentration in human blood by 100 µg L^{-1} was associated with a decrease of 2.5 IQ points. In the US, the United States Center for Disease Control (CDC) has set limits for lead concentrations in blood over which medical intervention is recommended. Before 1975, this limit was set at 600 µg L^{-1}. It was lowered to 300 µg L^{-1} in 1975, to 250 µg L^{-1} in 1985 and to 100 µg L^{-1} in 1991 (2).

Also in Germany limits were lowered in the course of time. The Humanbiomonitoring–Kommission defined 3 categories of lead concentrations in blood labelled HBM 1, 2 and 3. HBM 1 represents unobtrusive conditions, HBM 2 is not associated with health hazards but monitoring is recommended, whereas the category with the highest values, HBM 3, comprises significantly elevated lead levels, including the possibility of health effects and the need for clarification and mitigation. The categories are set differently for children, for women in a child-bearing age and for other adults. In 1987, the categories were defined for HBM 1: ≤ 150 µg L^{-1} (all groups), and for HBM 3: > 250 µg L^{-1} (children and women of child-bearing age) and > 350 µg L^{-1} (other adults). HBM 2 covers the range of concentrations between 150 µg L^{-1} and 250 µg L^{-1} (6). In 1996, the limits for the 3 categories were lowered (7). The "unobtrusive" category HBM 1 was decreased to levels of up to 100 µg L^{-1} for children and women of child-bearing age, while 150 µg L^{-1} was kept for other adults. The potentially health threatening category HBM 3 was set to begin at 150 µg L^{-1} for children and women of child-bearing age, and to 250 µg L^{-1} for other adults.

When dealing with such critical levels, one has to keep in mind that such limits are questionable, as Schwartz (8) has demonstrated that adverse effects emerge in adults and children even at slightly elevated lead concentrations.

Thus, the ubiquitous presence of lead in the environment posed, and in many parts of the world still poses, a health hazard (9). The present paper attempts to quantify this hazard for the case of West Germany.

The consumption of gasoline in Germany rose steadily from the 1950s until the present. Without legislation the emission of lead into the environment would have risen in parallel. However, legislation was introduced to curb emissions of this neurotoxin. In 1972 and 1976, the amount of lead added to gasoline was abruptly lowered, from originally 0.6 g L^{-1} to 0.4 g L^{-1} in 1972, and to 0.15 g L^{-1} in 1976. Later, in 1985, "unleaded" gasoline, with no more than 0.013 g L^{-1}, was offered to the consumers. After a few years, leaded gasoline was phased out entirely in West Germany. The volume of gasoline sold in Germany, and lead emissions gasoline use are shown in Figure 1, assuming that about $^3/_4$ of lead added to gasoline was emitted into the atmosphere (Alfke, Mineralölwirtschaftsverband, pers. comm.).

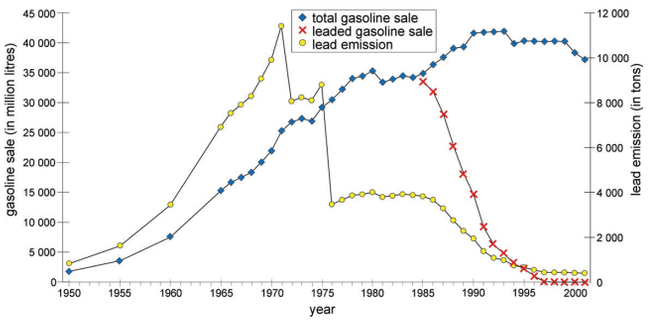

Figure 1. Annual gasoline sales and lead emissions in Germany. Volume of gasoline sold (millions of liters per year; solid) and of leaded gas (after 1985; red crosses); amount of lead emitted by gasoline (in tonnes; yellow).

Table 1.
List of a series of analyses of lead concentrations in humans ($\mu g\ L^{-1}$), samples across Germany in former West (AB = Alte Bundesländer) and East (NB = Neue Bundesländer) Germany. The analyses are different in terms of the analytical procedure, in selecting the samples, in the age and gender composition of the samples. The abbreviation "95%-q" stands for 95%-quantile, i.e., 95% of all concentrations in that sample are below the given concentration and 5% above. In the second column, usually the sample mean is given, but numbers marked by a * are medians (6, 16).

Year	mean LHB	range	std.dev	sample	Name of the study
1979	136	35-566	53	1423 adults	Brockhaus et al. (29)
1979	120		60	2953 adults	Wagner et al. (30)
1979	118		54	2301 children	Wagner et al. (30)
1981	103		49	2292 adults	Wagner et al. (30)
1981	111		63	2097 children	Wagner et al. (30)
1982	126		49	122 men	Eickmann et al. (31)
1982	105		35	149 women	Eickmann et al. (31)
1982	110		24	145 boys	Eickmann et al. (31)
1982	105		25	94 girls	Eickmann et al. (31)
1983	114	47-308	43	213	Ewers et al. (32)
1983	67	95%-q:115		2346 adults	Krause et al. (33)
1984	60		17	45 men	Myslak/Bolt (34)
1984	57		16	56 women	Myslak/Bolt (34)
1984	50		20	25 children	Myslak/Bolt (34)
1986	66	30-135	13	361 children	Brockhaus et al. (29)
1987	70		30	351 children	Michels et al. (35)
1988	72	13.5-175		105 women	Timm (36)
1988	65	15-220	17	72 adults	Jermann et al. (37)
1989	77		30	77 adults	Krause et al. (33)
1989	79		29	79 adults	Krause et al. (33)
1990	90		36	1703 men	Hense et al. (38)
1990	65		26	1661 women	Hense et al. (38)
1990-91	60	95%-q.:113		1219 men (AB)	Krause et al. (7)
1990-91	44	95%-q.:92		1261 women (AB)	Krause et al. (7)
1990-91	36	95%-q.:62		512 children (AB)	Krause et al. (7)
1991	50	13-150	15	213 Turkish children	Begerow et al. (39)
1991	53	15-174	15	1283 children (NB)	Begerow et al. (39)
1991-92	68			715 men (NB)	Krause et al. (7)
1991-92	42			772 women (NB)	Krause et al. (7)
1991-92	34	95%-q.:66		201 children (NB)	Krause et al. (7)
1991-92	60	1-137	35	100 children (Hamburg)	Dulon et al. (40)
1991-92	79	1-260	41	205 adults (Hamburg)	Dulon et al. (40)
1993	48	8-142	22	100) women (NB	Heinrich et al. (41)
1993	42	14-180	19	681 children	Bergerow et al. (39)
1995-96	25	6.3-43.8	7	226 children	LGA BW (42)
1995-97*	26	0.05-164.4		259 men (Berlin)	Hoffmann/Schäcke (43)
1995-97*	19	0.05-163		145 women (Berlin)	Hoffmann/Schäcke (43)
1998	41	79 (95%)		2342 men	Becker et al. (16)
1998	31	62 (95%)		2303 women	Becker et al. (16)

Table 2. Blood lead levels of students in Münster (Germany) (in $\mu g\ L^{-1}$). (Data source: Human-Probenbank Münster; pers. comm).

year	sample size	quantiles			mean
		50%	90%	95%	
1981	95	84.6	135.5	161.2	88.9
1984	145	78.1	127	147	82.3
1985	139	79.8	123.5	165.9	85.2
1986	98	62.4	105	116	69.1
1986	138	70	103	118	71.5
1987	121	60.3	113	133	69.4
1987	116	54.8	80.8	91.7	59.3
1988	125	63.5	83.5	102	65.8
1988	130	55.5	72.4	82.9	56.7
1989	119	50	67.2	92.4	54.5
1989	143	55.9	74.7	81.3	57.3
1990	89	56.5	75.4	80.6	58.2
1990	113	53.2	71.9	79.9	56.5
1991	99	48.7	74.1	92.9	53.5
1992	125	48.5	66	76.3	49.1
1993	97	44.6	61.9	70.4	45.6
1995	115	30.5	46.6	54.4	33.1
1995	122	29	48.2	50	31.2
1996	120	25.9	43.7	48.2	28.3
1996	112	26.8	40.9	49	28.7
1997	113	21.5	40.2	50.8	25.1
1998	119	10	36.9	47.6	21.4
1999	119	19.5	30.8	34.9	21.7
2000	113	18.1	25.9	33.4	20.1
2001	129	21.8	33.6	40.3	24.7

Few studies on emissions, deposition, and assessment of lead in the environment in the "gasoline decades" 1950-1990 are available. Two such studies are Thomas et al. (10) and von Storch et al. (11). The Thomas et al. (10) study reviews 19 reports from 6 countries, dealing with the relationship of lead use in gasoline and the presence of lead in blood. They conclude that lead in gasoline is a dominant source of lead in blood, and that a linear link between the two exists. They suggested lead concentrations in blood of about 30 $\mu g\ L^{-1}$ and in air of about 20 ng m^{-3}, as likely values in cases with no anthropogenic emissions of lead into the atmosphere.

Lead emissions and depositions in Europe were analyzed in some detail in the "GKSS lead study" (11, 12). Specific questions were: How did the lead emissions, atmospheric concentrations and depositions develop in Europe since the 1950s? Was the decline in air concentrations of lead matched by corresponding declines in the environment? The strategy included the preparation of a spatially disaggregated atlas of emissions (13), together with an analysis of the sequence of political decisions taken at German and European levels, and of their economic impacts (14). These emissions were fed into an atmospheric transport model (15), which calculated air concentrations and depositions in Europe in spatial detail.

DATA ON LEAD IN HUMAN BLOOD IN GERMANY

We have 2 data sets with time series of lead concentrations in human blood (PbB), beginning in the late 1970s. To our knowledge, no earlier data about lead in human blood in Germany are available. Data set "G" ("Germany") (Table 1) (6) is unsystematically collected, with samples at different locations, different methods, different age and gender groups. Data from a survey in 1998 have been added to this data set (16). The data most likely exhibit variations larger than those in the second data set, which is considerably more homogeneous. This second data set "M" ("Münster"; Human-Probenbank Münster 2002; pers. comm.) (Table 2) is derived from a controlled sampling strategy—for groups of students (age: 23–24 years, 50% women and 50% men) living in the town of Münster (ca. 200 000 inhabitants) in Nordrhine-Westfalia, close to the Ruhr area with heavy industry and intensive road traffic. Both data sets show the presence of lead in human blood in a rather similar manner, even if data set G exhibits larger variability, presumably because of the heterogeneous character of that data set. Children have usually somewhat lower lead concentrations than adults (7). These data sets describe a steady reduction of the presence of lead in human blood over time, with values levelling off at about 30 $\mu g\ L^{-1}$ in

the mid-1990s. These results are consistent with the comparative analysis of Thomas et al. (10) of a series of blood concentration studies, which did not include the 2 data sets G and M.

To determine the relationship between lead concentration in gasoline and in blood, an "efficient" lead concentration in gasoline in West Germany is determined for the years following the gradual introduction of unleaded (0.013 g L^{-1}) gasoline in the year 1985. First the volume of leaded and of unleaded gas is calculated, and then the ratio of the total mass of lead is divided by the total volume of gasoline. In 1990, the effective concentration was 0.05 g L^{-1}, in 1993 0.03 g L^{-1} and in 1995 it was down to 0,013 g L^{-1}. When relating these lead concentrations in gasoline to those in human blood, we find in the data set M (G) an intercept of 29 μg L^{-1} (31 μg L^{-1}) and a slope of 36.3 μg L^{-1}/(g L^{-1}) (48.0 μg L^{-1}/(g L^{-1})). These numbers are within the range of numbers found by Thomas et al. (10) in her analysis of several studies in a number of countries (not including Germany).

Both data sets contain information about the variability within the examined samples. In case of G, sometimes the standard deviation is given, at times the 95%-quantiles. There is a linear relationship between the mean concentration and the dispersion of the sample distributions. In fact, the best fit for the standard deviation is

standard deviation = 0.43 x mean – 3.9 Eq. 1

and for the 95% quantiles:

95% quantile = 1.63 x mean + 10.9 Eq. 2

If the mean concentration in a sample is 150 μg L^{-1}, then the standard deviation is 60 μg L^{-1}. If the mean blood concentration is 150 μg L^{-1}, then on average one sixth of the population will have more than 200 μg L^{-1} or less than 100 μg L^{-1}. This rough estimate is based on the assumption of a normal distribution, which is not really valid as the distribution of lead levels is skewed, with a long tail towards large values (not shown). Thus, it is more appropriate to use percentiles as in Equation 2.

In case of M, the 90% and 95% quantiles are given. Also in this case, a clear linear relationship between the mean and the quantiles is emerging, with

90% quantile = 1.46 x mean – 2.2 Eq. 3

95% quantile = 1.75 x mean – 4.6 Eq. 4

According to Equations 3 and 4, a mean concentration of 150 μg L^{-1} is associated with 5% (10%) of the population having more than 258 μg L^{-1} (217 μg L^{-1}) lead in their blood.

There are 2 major conclusions to be drawn from the above. *i)* The 2 data sets, G and M, describe the presence of lead in human blood rather similarly, even though 1 data set is drawn from a variety of different studies, and the other is a controlled sample from one location with similar individuals. The data in G are less homogenous and exhibit to some extent variability unrelated to our topic. Therefore, we will use in the following the set M. *ii)* In modelling the lead concentrations in human blood, it is reasonable to first deduce the mean concentration from the ambient conditions; the dispersion is then estimated by applying the Equations 2–4.

DATA ABOUT EMISSIONS AND AIR CONCENTRATIONS OF LEAD

Pacyna and Pacyna (13) provided expert estimates of European atmospheric lead emissions, in t yr^{-1}, for the years 1955, 1965, 1975, 1985, 1990 and 1995, and projection estimates for the year 2010. Road transport, nonferrous metal manufacturing, stationary fuel combustion, iron and steel production, waste disposal, cement production and other small sources were considered in all European countries. The sources were assigned to grid cells in a grid with 50 by 50 km^2. Estimates for intermediate years were obtained by linear interpolation between the emission values in the 2 nearest years of the estimate. Our reconstruction of the population's past contamination with lead is based on this data set.

For constructing scenario's of plausible alternative past contamination, we have used sales numbers of the Mineralwirtschaftsölverband (17, 18). From these volumes, we estimate the amount of emitted lead into the atmosphere by assuming that only 75% of the added lead is reaching the atmosphere, while a quarter is deposited in the engine and in the oil (Alfke, pers. comm).

From the already mentioned detailed simulation of aerial transport and deposition of lead, time series of air concentrations, are available on a 50 x 50 km^2 grid covering all Europe, which are consistent with the limited amount of data on atmospheric concentration (11).

MODELS

We adopt a simple dynamical relationship between 3 variables, namely the emission of lead PbE_t in an area A_E in the year t, the atmospheric concentration PbC_t in an area A_C in the year t and the mean concentration of lead in human blood, PbB_t, in the year t in the area A_C, according to Tahvonen et al. (19).

$$PbC_{t+1} = \alpha\ PbC_t + \beta\ PbE_{t+1} \quad \text{Eq. 5}$$

$$PbB_{t+1} = \gamma\ PbB_t + \delta\ PbC_{t+1} + \epsilon \quad \text{Eq. 6}$$

The dispersion of the blood levels is then determined through the 90% or 95% quantiles, using equation 3 or 4.
Equation 6 is equivalent to:

$$(PbB_{t+1} - \eta) = \gamma\ (PbB_t - \eta) + \delta\ PbC_{t+1} \quad \text{Eq. 7}$$

with $\eta = \epsilon/(1-\gamma)$. Equation 7 describes the dynamics of "anomalies" PbB_t-η relative to a "normal" state η towards which the system converges as soon as the forcing PbC_t ceases if $|\gamma| < 1$. For $0 < \gamma < 1$ the air concentration approaches η asymptotically with a time scale of $1/(1-\gamma)$ if $PbC_t=0$.
Both equations 5 and 6/7 are linear difference equations of first order, or discretized linear ordinary differential equations of first order, with memory ($\alpha\ PbC_t$ and $\gamma(PbB_t-\eta)$) and forcing terms (βPbE_t and $\delta\ PbC_t$). Large constants α and γ indicate that concentrations in the air and in human blood in a year are important for the concentrations in the next year, while small values indicate that the concentrations are mainly given by the forcing.

The linear formulation of Equation 5 is supported by the simulated 1955-1995 air concentrations and lead emissions (15). The linearity of Equations 6/7 is motivated by the analysis of Thomas et al. (10), who examined several data sets of emission data, air concentrations and blood concentrations in different countries. The linearity is not implicitly assuming that the airborne lead would be taken in mainly through breathing. The most important source of human intake for adults is *via* food which is contaminated by the deposition of airborne lead on food crops (20, 21). Lead is also taken in by humans in others ways, for instance with drinking water flowing through lead pipes or food kept in lead-soldered cans. The relative importance of these other factors has been discussed (22, 23). However, it seems that gasoline contributions are, in most cases, the largest (23). In special cases, in particular in the vicinity of certain industries and large-scale usage of leaded plumbing systems, other factors may be dominant (22). Not only the absorption of lead by food, but also the dynamic interchange of the body lead pool cause a delay in the

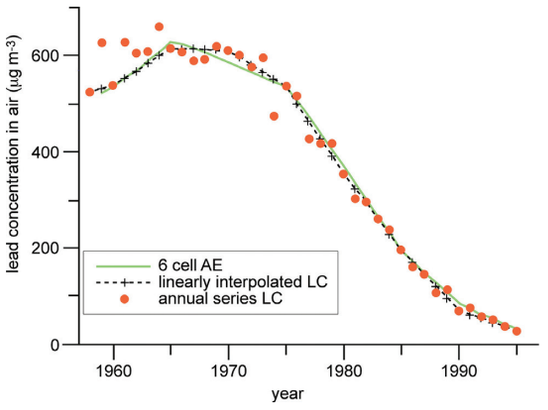

Figure 2. Lead concentration in the air (PbC), in ng m^{-3} in the 50 x 50 km^2 grid cell containing the town of Münster as simulated in the von Storch et al. (15) reconstruction (raw: red dots; interpolated: black dashed), and as estimated using Münster emissions (according to Pacyna and Pacyna (13)) and the linear model (Eq. 5) (green continous line)

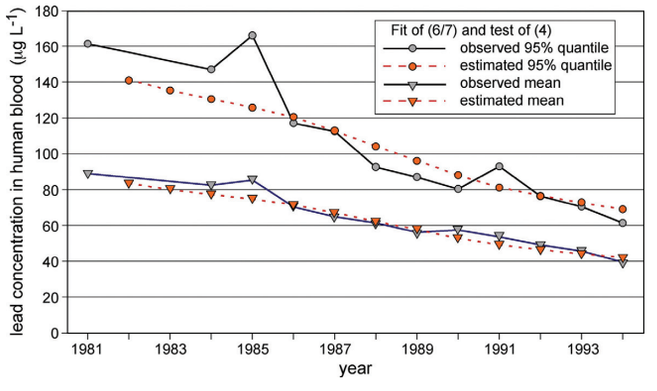

Figure 3. Fit of equation (6/7) and test of equation (4) for data set M. The upper 2 curves refer to the 95%-iles, and the lower 2 to the means. The solid lines are the data from the data set M; the dashed lines are estimated. The estimated mean curve is derived by using simulated air concentrations in Münster and the 1981 observed blood level as initial value; the curve for the estimated 95%-iles is obtained by using the estimated means and applying equation (4).

effect of decreasing emissions, which is integrated in the model by the variable γ (Eqs 6/7) (21).

The model (Eqs 5-7) has been fitted to 2 different sets of variables. First, PbB is the lead level in blood in Münster, i.e. given by the data set M. In that case we speak of the Münster model M. A_E is an area surrounding Münster and A_C the grid box containing Münster. Because of the considerably better quality of the data set M, this case is discussed and presented in more detail, while results for the second case are reported only in brief. The second case refers to German blood levels, as described by the heterogeneous data set G. The model is called Germany model G. The emission area covers West Germany, The Netherlands, and Belgium and northern France. The predictor is German lead levels in blood (as given by the data set G).

The Münster model

For fitting Equation 5 for the Münster model we have chosen as A_E a grid of 6 50 x 50 km^2 cells, with 2 columns (north-south oriented) and 3 rows (east-west oriented). Münster is contained in the cell in the right column and middle row, i.e. there is one row north and south of Münster, and one west of the town, but none to the east. Having tried several choices of A_E regions we found this setup most efficient. The yearly interpolated Pacyna emission data for this region A_E from 1958 until 1995 as well as the simulated air concentrations in the grid cell A_C containing Münster were used to fit Equation 5, which gave as best estimates for the 2 constants: $\alpha = 0.25$ and $\beta = 1.02$ ng yr^{-1} m^{-3} t^{-1}. The fit was made with concentrations linearly interpolated, using as nodes the years with Pacyna-estimates of the emissions (i.e. 1955, 1965, 1975, 1985, 1990 and 1995). The value of α may be compromised by the linear segments in the concentration and emission data Equation 5 is fitted to.

The similarity between the simulated concentrations in A_C and the estimate using (Eq. 5) with PbC_{1958} as initial value is reasonably good with a root mean square error of 7.7 ng m^{-3} (linearly interpolated concentrations) and 31.1 ng m^{-3} (full data; Fig. 2). Applying statistical tests to determine the quality of the fit is not possible because of the very strong temporal memory in the sample. The value of $\alpha = 0.25$ indicates that little lead emitted in one year will be found in the atmosphere in the next year, but that annual air concentrations are mainly given by the emissions at the same time.

For fitting the Equations 6/7 we have used the simulated air concentrations in the grid cell A_C of Münster and the samples means of lead concentrations in human blood from data set M, 1981 to 1995. This set-up gave as optimal constants $\gamma = 0.31$, $\delta = 0.15$ and $\epsilon = 22.2$ µg L^{-1}, or, equivalently, $\eta = 32.2$ µg L^{-1}. Also in this case, the memory of lead concentration in blood is relatively low, even if not as low as in the case of the atmosphere. If lead concentrations on average are larger than η by X µg L^{-1} then in the next year 31% of this excess X will still be there, even if the ambient atmosphere is free of lead. When lead is banned from the atmosphere for good, then the blood level will settle at about 30 µg L^{-1}, which is consistent with the intercept found in the emissions and air concentrations section.

The fit of the Equations 6/7 with the original data is demonstrated in Figure 3. The forward integration of Equation 6 was begun in 1984, when observations became continuously available for blood levels. Already in 1981, a study on blood levels was conducted, and that value was used to initiate Equation 6. The fit is very good, with a root mean square (rms) of 4.2 µg L^{-1}. This suggests the adequacy of a linear approximation for the scope of the present study. It can be assumed that the levelling off of the blood lead with increasing exposure, as it is described in the literature, happens on a level which is beyond the pollution level of our samples (24).

As a further independent check, the 95%-iles were estimated, using Equation 4 with the *estimated* means. The resulting 2 curves are also rather similar, as shown in Figure 3 and as quantified by an rms of 12.1 µg L^{-1}.

The Germany Model

The Germany model predicts lead levels of blood in West Germany, using emissions in The Netherlands, Belgium, West Germany, and northern France as predictor (A_E). The data used to fit Equations 5–7 are the Pacyna emissions, the simulated air concentrations and the heterogeneous data set G. The air concentration is averaged over A_C = West Germany. The constants found are $\alpha = 0.30$, $\beta = 0.0054$ ng yr^{-1} m^{-3} t^{-1}, $\gamma = 0.46$, $\delta = 0.24$, $\epsilon = 16.2$ µg L^{-1}, corresponding to $\eta = 30$ µg L^{-1}. The rms of the Eqs 6/7 fit is 12.3 µg L^{-1}.

The constants in the German model and in the Münster model are not identical, but rather similar, in particular with respect to the memory time for the air concentration ($\alpha = 0.30$ and 0.25) and the expected blood level in case of no lead in the atmosphere ($\eta = 32$ and 30 µg L^{-1}). β is very different in the 2 cases, as the absolute mass of emissions, which depends on the size of the area A_E, is related to the air concentrations, which are dependent on the size of the considered area A_C. That the rms of Equations 6/7 for the German model is considerably larger than the rms for the Münster model was to be expected because of the heterogeneity of the G-data set.

RECONSTRUCTION OF PAST DEVELOPMENT

The reconstruction of the emissions by Pacyna and Pacyna (13) is very detailed in its spatial resolution but, unfortunately, is available only at certain times, namely 1955, 1965, 1975, 1985, 1990 and 1995. Thus, this reconstruction fails to describe the maximum use of lead in gasoline in Germany around 1970 (Fig. 1). Because of the availability of the sales numbers of gasoline in Germany (16, 17), we are able to improve the estimation of the emissions for Germany, by using

$$PbE_t^+ = PbE_t + \theta_t (V_t^+ - V_t) \qquad \text{Eq. 8}$$

where PbE_t is the linearly interpolated estimate of emissions according to Pacyna and Pacyna (13) available on the 50 x 50 km^2 grid. θ_t is the concentration of lead in gasoline in the year t. V_t^+ and V_t are estimated volumes of gasoline consumed in the year t in the area A_E. In case of the Münster data set M, it is assumed that 3.3% of the gasoline consumed in West Germany is consumed in the 6-grid box area A_E. V_t is the linearly interpolated time series of volume, using as nodes the years 1955, 1965, 1975, 1985, and 1990 and 1995, whereas V_t^+ is the time series of annual sales volumes according to MWV, after subtraction of the estimated former east German (GDR) consumption.

The modifications are in some years very large, in particular in 1976, when the reduction amounts to 150 t, so that the emissions are only 220 t. This dramatic reduction is entirely meaningful, as the Pacyna estimates between 1975 and 1985 are linearly interpolated, and a significant reduction by 5/8 of the lead concentration in the gasoline from 0.4 g L^{-1} to 0.15 g L^{-1} took place in 1976. On the other hand, Eq. 8 increased the emissions by 58 t in 1970, shortly before the first regulation was initiated. On average, the emissions in the region are reduced by 17 t yr^{-1} compared to the interpolated Pacyna-values; the standard deviation of the modification is 45 t yr^{-1}.

For consistency, the fit of the model (Eq. 5) is made with the linearly interpolated PbE_t and the simulated air concentrations based on the linearly interpolated emissions in all of Europe (14). Only when applying the model (Eq. 5) the improved estimates for Germany PbE_t^+ are used—for reconstructing past blood levels and for constructing scenarios of blood levels in case of different lead regulations.

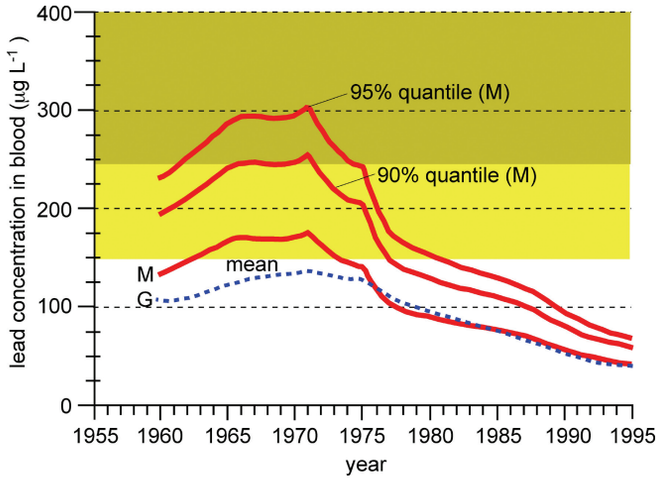

Figure 4. Estimated mean, 90%-ile and 95%-ile PbB (μg L^{-1}) in Münster, according to (6/7) and (3/4) (red solid lines, M). Additionally the mean level estimated with the Germany model is given as dotted blue curve (G). A level of more than 150 μg L^{-1} is considered in Germany as potentially harmful for children and women in child-bearing age (HBM 3). For other adults the limit for serious concern is set to 250 μg L^{-1}.

The Münster Model

The applications of the Münster model, Eqs 5–7 and Eq. 4, resulted in the curves shown in Figure 4. The estimated mean Münster blood levels pass the critical 150 µg L^{-1} between 1963 and 1973. Obviously, the first regulation in 1971 prevented a worse development. But, in spite of the limited threat to most people, a minority of them was actually more severely endangered. According to our estimate, 10% of the population was beyond the 150 µg L^{-1} limit, stipulated by German authorities (Humanbiomonitoring-Kommission), during all of the 1960s until the second regulation in 1976 took effect. For 5% of the population even the serious threshold of 250 µg L^{-1} was likely passed between the early 1960s und the early 1970s. After the second regulation in 1976 the health situation for more than 95% of the population returned to levels not associated with significant health threats (according to published knowledge).

The Germany Model

The Germany model uses emissions in northern France, West Germany, The Netherlands, and Belgium as input. In this case, only the German emissions are corrected according to Equation 8, while the other contributions are specified according to Pacyna. The estimated mean values are also shown in Figure 4. From 1975 until 1995 the estimate coincides closely with the reconstruction with the Münster model. Before that, however, significant differences emerge. The peak value is also obtained in 1971, but with a lower level of about 140 µg L^{-1} as compared to about 180 µg L^{-1} in case of the Münster model.

The difference between the M- and G-model reflects the inherent uncertainty in the fitted models. Obviously, an extrapolation using the limited evidence available, only since 1980, has to suffer from significant uncertainties. The overall similarity of the curves, however, indicates that the guesses deserve some credit. The fact that the Münster model is based on a considerably better blood-level data set, makes considering the results with that model more reliable than the lower values suggested by the G-model.

SCENARIOS

Emissions scenarios PbE_t^* are constructed by first assuming hypothetical lead concentrations in gasoline θ^*_t and inserting these into

$$PbE_t^* = PbE_t^+ + (\theta^*_t - \theta_t)\, V_t^+ = PbE_t - \theta_t V_t + \theta^*_t V_t^+ \qquad \text{Eq. 9}$$

The quantities without a * refer to the development, which actually took place: PbE_t^+ is the refined estimates (Eq. 8) of West German emissions of lead in the year t. The quantities with a * represent the scenarios, i.e. the emission scenario PbE_t^* in the area A_E, and the assumed lead concentrations in gasoline θ^*_t. If the assumed concentration θ^*_t is unchanged, then the emission is unchanged, i.e., $PbE_t^* = PbE_t^+$. If a higher concentration is assumed, i.e., $\theta^*_t > \theta_t$, then the emissions are increased $PbE_t^* > PbE_t^+$. The change is made to the estimate based on the MWV data, i.e. V_t^+.

Three scenarios for the concentration θ^*_t of lead in gasoline in West Germany were designed. In scenario 1 no reduction of lead is imposed so that the concentration is constant throughout 1955-1995 at 0.6 g L^{-1}. In scenario 2, the 2 abrupt reductions of lead concentration, first from 0.6 to 0.4 g L^{-1} in 1972 and then from 0.4 to 0.15 g L^{-1} in 1976 are taking place, while there is no introduction of "unleaded" gas in 1985. In scenario 3, the reduction from 0.6 to 0.4 g L^{-1} is already made in 1961, and the second step from 0.4 to 0.15 in 1965. In 1985, the introduction

of "unleaded" gasoline is assumed to have taken place in this scenario of an earlier limitation of the emission of lead into the atmosphere.

The Münster model

The most health threatening scenario is scenario 1, in which no regulations are introduced at all so that the gasoline lead concentrations remain at a constant level of 0.6 g L^{-1} through time. Then, according to our fitted series of models, the emissions of lead in the 6 boxes surrounding Münster rose to 1995 values of about 560 t yr^{-1} in the mid-1990s, and the air concentrations in the Münster grid box to 760 ng m^{-3} (not shown). The resulting estimated blood levels, in terms of populations means, are displayed in Figure 5. They reach levels well beyond what has been recorded in Germany with mean values of 200 µg L^{-1}. In this scenario, 10% of the population would have a lead concentration in their blood of 300 µg L^{-1} and more, while 5% would even have levels beyond 320 µg L^{-1} (not shown).

The mean levels for the scenarios 2 (no introduction of unleaded gas) and 3 (early regulation) are also displayed in Figure 5. The difference between scenario 2 and the real (estimated) development is that in the 1990s about 50% of the population

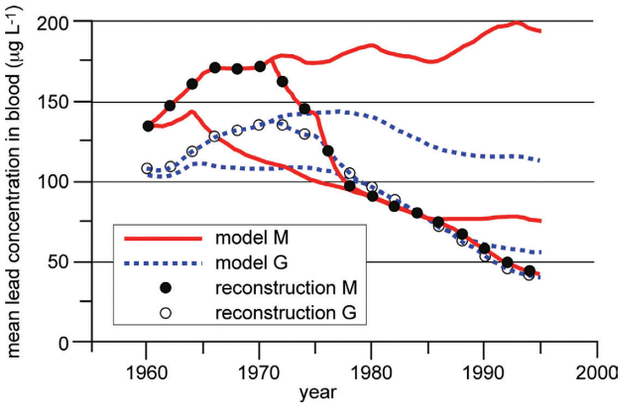

Figure 5. Scenarios for mean PbB (µg L^{-1}), as derived by the Münster model (M, red, continuous) and for the Germany model (G, blue, dashed). Scenario 1 describes an evolution without regulation (i.e., ongoing use of 0.6 g L^{-1} lead in gasoline in Germany, upper curves). In scenario 2 no unleaded gasoline has been introduced in Germany in 1985 (middle curves), and in scenario 3, regulation was instituted in Germany already in 1961 (lower curves). The reconstructed lead levels in blood are also given as open (Germany model) and full circles (Münster model).

would have stayed at levels of about 75 µg L^{-1}, whereas the real blood-lead level was about 40 µg L^{-1}. The early regulation would have avoided the "critical phase" of elevated blood levels between 1960 and 1970. In the 1990s, 10% of the adults in the Münster area would have had levels of 110 µg L^{-1} in scenario 2, while the introduction of unleaded gas in the mid-1980s lowered this level to about 80 µg L^{-1} (not shown).

The Germany model

The Germany model suggests considerably smaller values than the M-model. In fact, even in the no-regulation scenario the concentrations level off since about 1980, due to the regulations in the neighboring countries France, The Netherlands, and Belgium and end up at values of about 115 µg L^{-1} in the 1990s, i.e. well below the critical levels of HBM 3. In fact, an emittor-receptor analysis of depositions in Germany (Costa-Cabral, pers. comm.) finds that while 70% of all depositions in Germany are "homemade", 10% originate from France, and 10% from Belgium and The Netherlands. Also in the scenario without an introduction of unleaded gas in Germany a significant difference in 1995 between the M-estimate and the G-estimate emerges, with lower values in the G-estimate due to the effect of the introduction of unleaded gas in the neighboring countries. In the scenario with early regulation, the 2 models are rather similar since the early 1970s.

DISCUSSION

The present study has a double purpose, namely; *i)* to demonstrate that retrospective analysis of past environmental regulation is possible and meaningful; and *ii)* to assess the success of the lead regulation in terms of its impact.

The general approach of this study, of combining detailed transport calculations with the construction of robust empirical models, may be used for other substances as well. If the substance is not inert, then more complex transport and transformation models are needed, which will add to the technical complexity of the task, but which will not change the general approach. The advantage of the approach is that is condenses the complexity of the numerical transport simulation into a simple conceptual model, which relates emissions to air concentrations. Then, in the spirit of empirical downscaling in climate change studies (e.g. 25), the atmospheric concentration is related to the impact relevant variable, in this case lead concentration in human blood. This approach explains the complexity of the system in a simplified, structured manner which, in principle, allows for the construction of scenarios of future development, conditional upon societal decisions. As an alternative, we have derived an empirical model, which directly relates emissions to blood levels (25). This even simpler model resulted in rather similar estimates for the past and for the scenarios.

The same 2-step procedure was originally developed for the global climate change problem (19) to assess the relative importance of mitigation and adaptation measures, by optimizing expected future costs. This could be done in principle also in the present case of reducing the use of lead in gasoline. However, this could not really be done, as the health costs associated with the emission of gasoline lead can hardly be determined—likely the costs were high. Although there had been changes of market shares in the gasoline trade and automobile sectors in Germany, the macroeconomic costs of the regulation seem to be insignificant in spite of contemporarily voiced concerns claiming significant costs (14). In fact, the case of gasoline lead may turn out to be an excellent example, demonstrating the limited utility of purportedly objective cost-benefit analyses, as the costs claimed at the time of the regulations turned out to be significantly biased due to vested interests.

The stepwise out-phasing of lead as an anti-knock additive in gasoline in Germany was a clear success. Our analysis indicates that unacceptable levels of lead have prevailed in parts of the population, having caused significant but undocumented health damage in many people. In the 1960s, ever-increasing emissions of lead were causing lead levels in human blood to increase to levels beyond liberally defined limits; likely, the majority of people was not affected, but it seems probable that a significant number of people were adversely affected. The regulation just came in time to avoid expected widespread health hazards.

However, even if the present analysis is concluding with a rather positive assessment of the lead regulation policy, one must not forget that lead is not only a factor for human health, but also for the environment as a whole. And the lead once emitted into the air and later on deposited is not just disappearing from the system. In plants and some mammals, including humans, the lead content is slowly decreasing (12), while concentrations in

animals living in rivers and coastal seas do not show such a clear reduction. Much of the lead is simply deposited in the soil and sediments (12), where it may remain for a few hundred years. Johansson et al. (26) presented a detailed analysis of soil pollution in Sweden, showing that in peat bogs, the increased depositions of lead related to the use in gasoline are well preserved (27).

Thus, the conclusion of a successful regulation in terms of limiting risks for human health should not downplay the consequences of the introduction of tetraethyllead as an anti-knock additives in gasoline, in particular since alternatives were known and available already in the 1920s and 1930s (28). Heavy metals pose a large-scale and long-term environmental problem (26), and as such, reduced emissions "have only a significant effect on the pools" (in the soil) "in the perspectives of decades or centuries. The strategy of environmental protection has to be based on continuous assessment and precautionary principles" (26).

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