

NILU: OR 43/2000
REFERENCE: O-98129
DATE: SEPTEMBER 2000
ISBN: 82-425-1207-8

Blood lead concentrations in the population of Tbilisi, Georgia

Results of model estimates

Jocelyne Clench-Aas¹
Manana Juruli²
Kari Arnesen¹



¹ Norwegian Institute for Air Research

² Georgian Toxicological Center, Lab of Toxicological Analyses

Preface

The Geo-2110 Country Programme for Phasing Out Lead in Gasoline in Georgia is funded by United Nations Office for Project Services (UNOPS). The project is a cooperative project within The Norwegian Consortium for Energy and Environment (NORCE), including Center for Economic Analysis AS (ECON), Institute for Energy Technology (IFE), and Norwegian Institute for Air Research (NILU).

The study has three phases.

- *Phase I: Mobilization and Preparation of the Project – already reported*
- *Phase II: Assessment of the Existing Situation and Development of Projections*
- *Phase III: Georgia Lead Phase-Out Country Program*

Phase II includes an activity “Assessment of Lead – Concentrations-Air and Health Implications”.

This report is a contribution to an evaluation of the impact of lead from gasoline on health using estimated lead concentrations in the blood of the general population in Georgia as indicators.

Table of Contents

	Side
Preface	1
Summary	5
1 Introduction	9
2 Health effects of lead pollution	10
3 Measured concentrations of lead in blood in the population of Tbilisi	11
4 Description of the Compartment Model.....	13
4.1 Exposure to lead through food ingestion.....	15
4.2 Exposure to lead through inhalation.....	16
4.3 Time-use factors used.....	19
4.4 Choice of values for physiological parameters used	20
5 Dose-response relationships.....	22
6 Estimated blood lead concentrations	23
7 Estimated benefits for the total population of Tbilisi.....	25
8 Discussion	26
9 References.....	27
Appendix A Relationship between estimated and measured blood lead using compartment model – results from earlier report.....	29
Appendix B Assumptions used to run the lead compartment model in Tbilisi	33
Appendix C Benefit estimates for different levels of lead in gasoline.....	39

Summary

This study aims at predicting the health benefits of emission reductions of lead from vehicular traffic in Tbilisi, Georgia. The method chosen uses a compartment model that estimates blood lead concentrations in population subgroups as a function of inhalation and ingestion of lead.

The compartment model is a useful tool to estimate anticipated reductions in blood lead concentrations after pollution reduction measures by comparing estimates before and after implementation of measures. The model accounts for changes in concentrations in food, drinking water and ambient air and the resulting effects on blood lead concentration via changes in intake from nutrition and inhalation. The model uses as input, time-use, food intake, physiological parameters and pollution concentrations. The model calculates blood lead concentrations for 76 different population subgroups. The criteria for defining a subgroup are sex, age, work site, home site, extra features as jogging, etc.

The model was run for three situations:

1. The target situation, when the lead in gasoline does not exceed the legal limit of 13 mg lead per liter gasoline.
2. The estimated current situation, with lead levels in gasoline of 50 mg/liter.
3. The estimated worst case in 2005, with lead levels of 80 mg/liter gasoline.

Concentrations of lead in blood are in reality composed of:

- a base level that reflects exposure to food imported from the local region, exposure to paint and other sources of lead in coloring, possibly drinking water etc.
- a portion from exposure from lead in gasoline that is consumed either directly through inhalation, or indirectly through ingestion of deposited lead.

The sum of the two should not exceed the WHO guideline value of 10 $\mu\text{g}/\text{dl}$. Estimates of concentrations of lead in blood originating from lead in gasoline can be derived that include both the portions reaching the blood through direct inhalation, and the portion reaching the blood indirectly via ingestion. Concentrations of blood lead were estimated to be as in Tables A and B (lead originating from special sources such as contaminated drinking water from lead pipes, food stored in painted ceramic pots etc not included).

Table A: Reductions in blood lead concentrations reflecting the reduction between the estimated current situation (lead in gasoline 50 mg/liter) and the target situation (13 mg/liter).

Population subgroup	Blood lead levels (µg/dl) reflecting lead in gasoline 50 mg/l		Blood lead levels (µg/dl) reflecting lead in gasoline 13 mg/l		Resulting reduction in estimated blood lead levels with ambient lead removal measures (µg/dl)	
	Living suburbs	Living city center	Living suburbs	Living city center	Living suburbs	Living city center
Babies	47.4	71.9	14.9	21.0	32.5	50.9
Children	29.5	43.9	10.6	14.2	18.9	29.7
Adult women	25.0	30.2	8.3	9.7	16.7	20.5
Adult men	22.3	27.2	7.6	8.9	14.7	18.3
Retired persons	30.8	41.4	10.0	12.7	20.8	28.7

Table B: Reductions in blood lead concentrations reflecting the reduction between the worst case in 2005 (lead in gasoline 80 mg/liter) and the target situation (13 mg/liter).

Population subgroup	Blood lead levels (µg/dl) reflecting lead in gasoline 80 mg/l		Blood lead levels (µg/dl) reflecting lead in gasoline 13 mg/l		Resulting reduction in estimated blood lead levels with ambient lead removal measures (µg/dl)	
	Living suburbs	Living city center	Living suburbs	Living city center	Living suburbs	Living city center
Babies	53.8	82.2	14.9	21.0	38.9	61.2
Children	42.1	63.7	10.6	14.2	31.5	49.5
Adult women	36.0	43.9	8.3	9.7	27.7	34.2
Adult men	32.0	39.3	7.6	8.9	24.4	30.4
Retired persons	44.6	60.6	10.0	12.7	34.6	47.9

The benefits associated with removal of lead from gasoline are calculated for reductions from the estimated current situation and the worst case in 2005 separately (Tables C and D). They reflect the reduction of lead in gasoline first to the target level, and subsequently, to the total elimination of lead from gasoline. The calculations were based on the following assumptions:

- the portion of lead in blood arising from exposure to lead in gasoline is reduced to 0 with future complete removal of lead from gasoline,
- the background blood lead level of 2.0 µg/dl (assuming zero lead in ambient air),
- population of 1.5 million,
- 50% of the population living in the city center.

Table C: Estimated benefits from measures to reduce ambient lead from the current estimated situation (lead in gasoline 50 mg/liter) to the target level (13 mg/liter) and to total removal of lead from gasoline (0 mg/liter). The numbers are rounded.

Benefit	Estimated benefit for removal of lead in gasoline from 50 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 13 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 50 mg/l to 13 mg/l
Nr. of adults with reduced hypertension	365 000	165 000	200 000
Nr. of adults with reduced risk for non fatal heart attacks, pr. year	1400	800	600
Nr. of adults with reduced risk for cardiovascular death, pr. year	1500	800	600
IQ points improved per child	9.23	2.75	6.48

Table D: Estimated benefits from measures to reduce ambient lead from the worst case in 2005 situation (lead in gasoline 80 mg/liter) to the target level (13 mg/liter) and to total removal of lead from gasoline (0 mg/liter). The numbers are rounded.

Benefit	Estimated benefit for removal of lead in gasoline from 80 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 13 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 80 mg/l to 13 mg/l
Nr. of adults with reduced hypertension	440 000	165 000	275 000
Nr. of adults with reduced risk for non fatal heart attacks pr. year	1600	800	800
Nr. of adults with reduced risk for cardiovascular death pr. year	1700	800	900
IQ points improved per child	13.56	2.75	10.81

These numbers can only be considered as rough estimates. All the benefits would not be achieved immediately as

- Lead is stored in the bone reservoir and would gradually be released,
- When lead is removed from the gasoline, it will remain in the soil for many years, contributing to intake of lead through the ingestion, and via resuspension of particles, to the air concentrations to be inhaled.

Blood lead measurements should be made to confirm the estimates described in this report.

Blood lead concentrations in the population of Georgia

Results of model estimates

1 Introduction

Lead is a highly toxic compound that has known effects on the hematopoietic and nervous system. The primary routes of human exposure to lead are through inhalation and ingestion of water, food, air, soil and dust. The relative importance of any single source of exposure is difficult to predict and will vary with geographic location, climate and local geochemistry. The level of exposure will vary as a function of age, sex, occupation, socio-demographic status, diet and cultural practices. In addition the amount of lead absorbed into the body varies depending on the concentration and composition of the inhaled or ingested lead.

The health effects of air borne lead are dependent upon particle size. A high proportion of inhaled small particles ($< 2.5 \mu\text{m}$) is deposited in the deepest alveolar portion of the respiratory system, from which lead is absorbed with almost 100% efficiency in the blood. Particles ranging in size from 2.5 to $10 \mu\text{m}$ are deposited in the tracheobronchial and naso-pharyngeal region, from which they are brought up into the throat where they are then swallowed. The absorption of lead from the gastrointestinal tract is far less efficient in adults (10 to 15%) but can be up to 50% in small children. Thus, a large proportion of lead in these sizes does not enter the blood. Especially children (hand-to-mouth activity) can ingest larger particles polluted with lead originating from traffic sources as surface dust.

A majority of the lead particles originating from vehicular exhaust is in the size range under $2.5 \mu\text{m}$, making vehicular traffic exhaust an important source of blood lead concentrations.

Food ingestion is a major component of most individuals total lead uptake, although the relative contribution is a function of body size, weight, age and type of diet. The occurrence of lead in the diet may be a result of natural sources of lead, deposition of air borne lead particles onto crops, forage, feed, soils and water; fertilizer use; harvesting, processing, transport, packaging, preparation and storage of food. Other sources of lead in foods include leaching from ceramic containers in which lead glazes have been used, uptake of lead from lead-rich urban garden soils into homegrown vegetable crops, and uptake of lead into crops from sewage sludge applied to farmland.

Lead concentrations in drinking water result primarily from lead leaching from water delivery systems and from previously contaminated drinking water sources. However, natural lead from the ground can also occasionally enter the drinking water.

This study is intended to predict the effects of lead emission reduction measures aimed at curtailing the ambient concentrations of lead from vehicular traffic. The method chosen to predict the impact of pollution reduction measures is a compartment model that estimates blood lead concentrations in population

subgroups as a function of the inhalation and ingestion of lead (food locally produced) originating from lead in gasoline. It would be considered beneficial to confirm estimated blood lead concentrations with selected blood lead measurements.

2 Health effects of lead pollution

The most common observed health effects of lead pollution are on the hematopoietic system. Children up to 6 years of age are considered the most at risk population. The reasons for children being the subpopulation most at risk are as follows (reviewed in WHO, 1994):

- *Behavioral – increased exposure due both to being more outdoors and to greater hand-mouth activity,*
- *Nutritional – lead intake is increased with a relatively higher intake of food and drink per unit of body weight,*
- *Physiological – lead absorption from the gastrointestinal tract is higher in children; nutritional deficiencies which enhance uptake are more prevalent; and the blood-brain barrier is not fully developed enhancing the transfer of lead into the brain; and*
- *Toxicological - health effects occur at a lower threshold of exposure.*

The lowest observed adverse-effect levels in children are as follows:

Table 1: Summary of lowest observed adverse effect levels for lead induced health effects in children.

Lowest observed effect blood lead level (µg/dl)	Hematological effects	Nervous system effects
800 – 1000		Encephalopathic signs and symptoms
700	Frank anemia	
400	Increased urinary ALA and elevated coproporphyrin	
250 - 300	Reduced hemoglobin production	
150-200	Erythrocyte protoporphyrin elevation in males	
100 - 150	Vitamin D3 reduction	Cognitive impairment
100	ALAD – inhibition	Hearing impairment

Blood lead has also been associated with neurodevelopmental effects in children. These effects include (EPA, 1987):

- Decreased intelligence
- Short-term memory loss
- Reading and spelling underachievement
- Impairment of visual motor functioning
- Poor perception integration
- Disruptive classroom behavior

- Impaired reaction time

Again no threshold has been identified.

The lowest observed adverse-effect levels in adults are as follows:

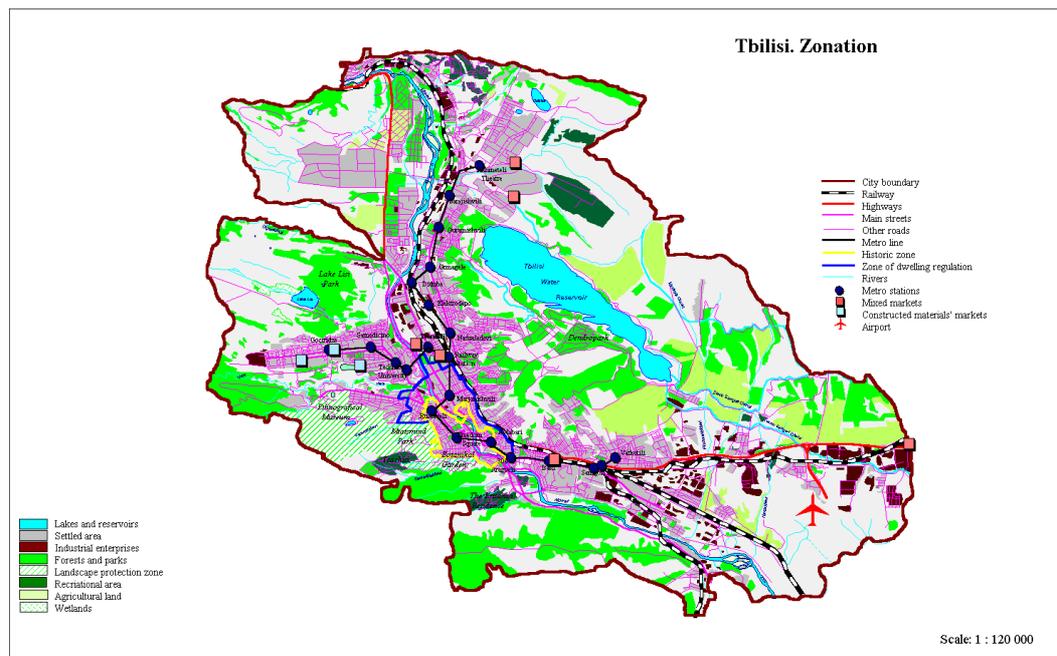
Table 2: Summary of lowest observed adverse effect levels for lead induced health effects in adults.

Lowest observed effect blood lead level ($\mu\text{g}/\text{dl}$)	Hematological effects	Nervous system effects
1000 – 1200		Encephalopathic signs and symptoms
800	Frank anemia	
500	Reduced hemoglobin production	Overt subencephalopathic neurological symptoms, cognition impairment
400	Increased urinary ALA and elevated coproporphyrin	
300		Peripheral nerve dysfunction (slowed nerve conduction velocities)
200-300	Erythrocyte protoporphyrin elevation in males	
150 – 200	Erythrocyte protoporphyrin elevation in females	

Blood lead concentrations have been associated with increased blood pressure across a broad range of doses. This has been reviewed by Ostro, 1992 and 1997. No threshold has been observed in a range of 7 to 34 $\mu\text{g}/\text{dl}$. Increases in blood pressure have in themselves been associated with increased risk of cardiovascular events and disease.

3 Measured concentrations of lead in blood in the population of Tbilisi

It should be stated that no data is available on the levels of non-occupational exposure to lead before 1990s in Georgia. In recent years (1995-1996), the Research Laboratory of the State Tbilisi Medical University has paid some attention to the impact of air pollution on the health of the population living in the area of the main highway of Tbilisi. A report of the Working Group N2 on Public Health, Demographics, and Social Issues demonstrates that high concentrations of lead were found in air and soil samples of Tsereteli and Agmashenebeli Avenues. It is also pointed out that the 25% of the population had blood concentrations (PbB) of 38.2 $\mu\text{g}/\text{dl}$ or more. These values are higher than PbB limits recommended by the WHO.



Performed investigations have demonstrated:

- 7.1 per cent of investigated children (564) from various districts of Tbilisi were found to have low hemoglobin levels.
- 32.5 per cent of children had eosinophilia,
- 25.4 per cent had monocytosis,
- 6.4 per cent of children had iron-deficiency anemia,
- 7.4 per cent were observed to have latent iron-deficiency anemia,
- 25 per cent of children were observed to have changes in the heart tones were: tonelessness, Cystol murmur.

During the first year of observations many of the examined children showed morphofunctional disorders. In the third year, a growth of chronic diseases was marked among children with subcompensation and decompensation, which may indicate to the reduction of compensatory ability of the body.

The observed negative trends are especially acute among children brought up in districts with heavy traffic. They exceed by 3-15 times the data for the children living in the control district (Nutsubidze Plateau).

Investigation of the health of the population of the Tsereteli, Agmashenebeli Avenues and Nutsubidze Plateau as well as of the control region (673 persons) has shown that 20.7 per cent of the studied population of the area showed cardiovascular system pathology, in particular, hypertension and insufficiency of the heart, stenocardia, etc. This pathology was mainly seen among people over 40. In addition, 40 per cent of the people of the same age had high thrombogen levels.

It should be particularly noted that carboxihemoglobin in the blood was found in 60 per cent of the examined people (108 persons) and 25 per cent of people were observed to have high levels of lead in the hair and urine.

In the control area, 7 per cent of the examined people (108 persons) exhibited pathology of the cardiovascular system, and 2 per cent pathology of the respiratory system. There were few cases of carboxihemoglobin in blood and lead in the hair and urine.

In the absence of detailed description of measurement techniques and analytical methods there would seem to be some questions as to whether this PbB is valid or not.

The absence of any valid information concerning the lead concentration in non-occupationally exposed Georgian population increases the difficulty of determining the necessary procedures to be taken to stop exposure to lead and more importantly when these must be done¹.

4 Description of the Compartment Model

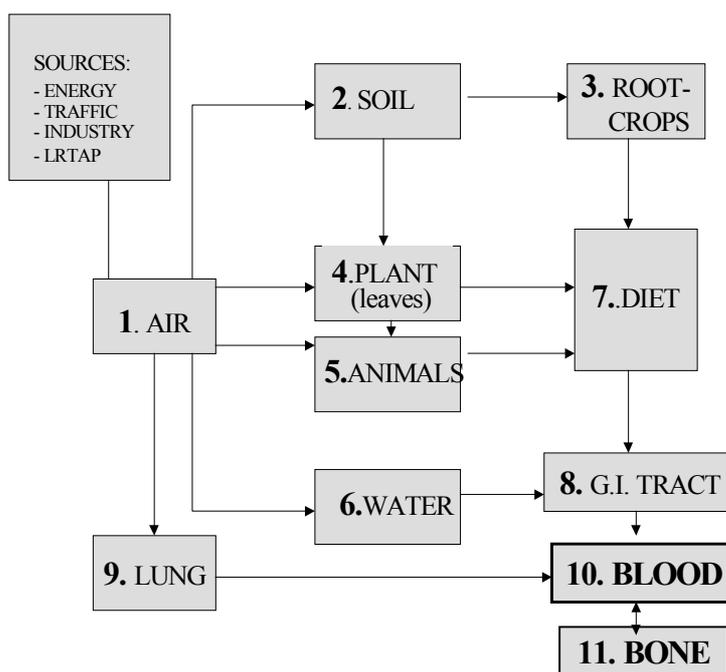
The compartment model estimates blood lead concentrations in different population subgroups. It also estimates what portion of the blood lead concentration that originates from inhalation and what portion that originates from food ingestion. The method is based on a path model where each box is in steady state with its neighboring boxes having a defined flux coefficient (Figure 1). In the steady state or equilibrium situation, the rates of pollutants entering and leaving a compartment are equal, and the concentration of the pollutant in the reference compartment does not change with time. The primary path components are air, soil, water, then vegetation and animals that then enter the lung or digestive system to end up in the blood. The mathematical details are described in Sivertsen, 1985.

Some of the primary ingredients of the model are given in the following list:

Steady state concentration in soil
Leaf surface
Soil surface
Steady state flux air-soil
Residence time (of lead) in soil
Mixing depth in soil
Density of soil
Deposition velocity to soil
Steady state concentration in Plant A, from soil
Steady state concentration in Diet A, from soil
Steady state concentration in GI-tract
Steady state concentration in blood from Diet A
Steady State flux soil – plants (roots), diet A
Steady state flux roots diet (diet A)
Steady state flux diet A – GI tract
Steady state flux GI-tract to blood of Diet A
Residence time of lead in blood

¹ As a first step, a pilot project was undertaken by a group of scientists (1998-1999). This study included among others determining the blood lead content of the people living in the heavy traffic area of Tbilisi. Blood of 50 subjects were collected and prepared for analytical measurements by Atomic Absorption Spectrophotometer. Due to the lack of funds the testing has been postponed.

Blood volume
Deposition velocity to leaf surface
Steady state flux air-plant surface
Steady state flux plant-diet
Residence time of lead on grain
Production of grain per m²
Deposition velocity to fruit, berries
Residence time of lead on fruit, berries
Production of fruit, berries per m²
Residence time of lead on vegetables
Retention of lead inhaled into pulmonary regions of the lung
Breathing rate factor
Absorption of ingested lead into blood
Intake of food not included in component model
Fraction lead into lung absorbed in blood
Time spent in each microenvironment
(Lung ventilation) breathing rate
Consumption potato
Consumption grain
Consumption fruit/berries
Consumption meat
Consumption milk
Consumption surface vegetables
Number of persons in each population subgroups



LRTAP = Long Range Transport

Figure 1: Simple, quasistationary compartment model for calculation of steady-state concentration of lead in blood for selected groups of people, based upon estimated air concentrations from the different source categories.

4.1 Exposure to lead through food ingestion

As can be seen in the above list and in Figure 1, foods are divided into food types and the transfer of lead from one box to the next is then calculated. The primary food categories are grain, fruit, meat, dairy products, potatoes and other root vegetables, surface vegetables and fish. Levels of lead in each of these food types is multiplied by the consumption per day and then summed up over all food types. The details of the calculations and the flux coefficients are all given in Sivertsen, 1985.

The consumption of the main foodstuffs is assumed as indicated in Table 3 for a selected group of individuals.

Table 3: Consumption (g/day) of foodstuffs by populations subgroup, input to compartment model.

Population	Potato	Cereals	Fruit/Berr	Meat	Milk	Surface
Adult men						
Non-smokers	190	240	50	120	420	80
Smokers	190	240	50	120	420	80
Adult women						
Nonsmokers	130	150	50	90	360	60
Smokers	130	150	50	90	360	60
Retired persons						
Nonsmokers	70	150	30	50	100	40
Smokers	70	150	30	50	100	40
Children						
Unexposed to passive smoking	100	230	30	80	550	40
Exposed to passive smoking	100	230	30	80	550	40
Babies						
Unexposed to passive smoking	50	100	20	20	400	20
Exposed to passive smoking	50	100	20	20	400	20

4.2 Exposure to lead through inhalation

The amount of lead in blood that results from inhalation of polluted air is calculated on the same principles as those used in calculating the burden coming from food ingestion. Lifestyles of each population subgroup together with estimated lead concentrations in different microenvironments outdoors and indoors is used in estimating the portion of blood lead concentrations coming from inhalation

Population subgroups were defined as described in Table 4.

Table 4: Abbreviations used in defining population subgroups

1	Personal
A	Adult
C	Child
B	Baby
R	Retired
2	Sex
X	Male
Y	Female
3	Smoking habits
M	Smoker (passive smoking in children)
N	Non-smoker
4	Occupation
U	Unemployed
E	City center location
F	Suburban location
O	Residential location
S	School
D	Lead exposed industry
5	Living
I	City center location
J	Suburban location
K	Residential location
6	Specialties
L	Jogging along roads
H	Only indoors
P	Commuting more than 20 minutes

These are combined so that for example XNDJ is an adult man, living in a suburban area, working at the factory and is a non-smoker.

The amount of time each population subgroup spends in each of 30 microenvironments is estimated. The microenvironments and their concentration of air lead used in this study are those given in Table 5 (Tønnessen, 2000).

Table 5: Definition and concentration of lead ($\mu\text{g}/\text{m}^3$) in air in the thirty microenvironments used in this study. Values are based on estimates done in other parts of this study and represent estimated concentrations assuming three concentrations of lead in gasoline, 13 mg/l, 50 mg/l and 80 mg/l.

	Microenvironment	Air lead concentration ($\mu\text{g}/\text{m}^3$)	Air lead concentration ($\mu\text{g}/\text{m}^3$)	Air lead concentration ($\mu\text{g}/\text{m}^3$)
		Gasoline lead	Gasoline lead	Gasoline lead
		13 mg/l	50 mg/l	80 mg/l
CSA(1)	City sidewalk – low pollution	0.4	1.6	2.4
CSA(2)	City sidewalk – medium pollution	1	4	6
CSA(3)	City sidewalk – high pollution	2	8	12
CSA(4)	Suburban sidewalk	0.1	0.4	0.6
CSA(5)	Suburban medium pollution area	0.1	0.4	0.6
CSA(6)	Suburban open area, low pollution	0.035	0.035	0.035
CSA(7)	Residential “polluted”	0.1	0.4	0.6
CSA(8)	Residential clean	0.035	0.035	0.035
CSA(9)	Along Hiway < 10 m	0.7	2.8	4.2
CSA(10)	Along Hiway 10-50 m	0.35	1.4	2.1
CSA(11)	Along small road < 10m	0.1	0.4	0.6
CSA(12)	Industrial area low pollution	0.1	0.4	0.6
CSA(13)	Industrial area medium pollution	0.1	0.4	0.6
CSA(14)	Industrial area high pollution	0.1	0.4	0.6
CSA(15)	Background area	0.035	0.035	0.035
CSA(16)	Nonsmokers home in city	0.8	3.2	4.8
CSA(17)	Nonsmokers home in suburban	0.4	1.6	2.4
CSA(18)	Nonsmokers home in residential	0.1	0.4	0.6
CSA(19)	Smokers home in city	0.82	3.28	4.92
CSA(20)	Smokers home in suburban	0.42	1.68	2.52
CSA(21)	Smokers home in residential	0.12	0.48	0.72
CSA(22)	Store/restaurant	0.2	0.8	1.2
CSA(23)	Parking Garage	2	8	12
CSA(24)	Work place residential	0.8	3.2	4.8
CSA(25)	Work place suburban	0.8	3.2	4.8
CSA(26)	Work place city	0.8	3.2	4.8
CSA(27)	Work place in lead exposed industry	0.8	3.2	4.8
CSA(28)	Private car	1	4	6
CSA(29)	Bus/train	1	4	6
CSA(30)	Bicycle	1	4	6

In addition it is necessary to provide a breathing rate for each environment. These are given in Table 6.

Table 6: The breathing rate factor used for each of the thirty microenvironments.

	Microenvironment	Breathing rate
CSA(1)	City sidewalk – low pollution	1.5
CSA(2)	City sidewalk – medium pollution	1.5
CSA(3)	City sidewalk – high pollution	1.5
CSA(4)	Suburban sidewalk	1.5
CSA(5)	Suburban medium pollution area	1.5
CSA(6)	Suburban open area, low pollution	1.5
CSA(7)	Residential “polluted”	1.5
CSA(8)	Residential clean	1.5
CSA(9)	Along Highway < 10 m	1.5
CSA(10)	Along Highway 10-50 m	1.5
CSA(11)	Along small road < 10m	1.5
CSA(12)	Industrial area low pollution	1.5
CSA(13)	Industrial area medium pollution	1.5
CSA(14)	Industrial area high pollution	1.5
CSA(15)	Background area	1.0
CSA(16)	Nonsmokers home in city	0.8
CSA(17)	Nonsmokers home in suburban	0.8
CSA(18)	Nonsmokers home in residential	0.8
CSA(19)	Smokers home in city	0.8
CSA(20)	Smokers home in suburban	0.8
CSA(21)	Smokers home in residential	0.8
CSA(22)	Store/restaurant	1.0
CSA(23)	Parking Garage	1.0
CSA(24)	Work place residential	1.0
CSA(25)	Work place suburban	1.0
CSA(26)	Work place city	1.0
CSA(27)	Work place in lead exposed industry	1.0
CSA(28)	Private car	1.0
CSA(29)	Bus/train	1.0
CSA(30)	Bicycle	3.0

4.3 Time-use factors used

In order to estimate the portion of lead coming from inhalation, it is necessary to calculate the realistic exposure based on time spent in each of the 30 microenvironments. This was done as indicated in Table 7. The full table for all population subgroups can be found in Appendix B. Time-use factors were based on data collected in Norwegian studies using diaries (Clench-Aas, et al., 1984, 1986 and 1989).

Table 7: The amount of time (in hours) spent in each microenvironment by each of the population subgroups, input to compartment model. Examples only, where work place is city center, and living site is suburban.

CSA	Babies		Children		Adult Men		Adult Women		Retired persons	
	NS	S	NS	S	NS	S	NS	S	NS	S
1	0.0	0.0	0.0	0.0	0.3	0.3	0.3	0.3	0.0	0.0
2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.1	0.1
3	0.0	0.0	0.0	0.0	0.2	0.2	0.2	0.2	0.0	0.0
4	1.0	1.0	1.0	1.0	0.1	0.1	0.2	0.2	0.0	0.0
5	1.0	1.0	1.0	1.0	0.4	0.4	0.5	0.5	1.0	1.0
6	0.0	0.0	2.0	2.0	0.3	0.3	0.4	0.4	1.0	1.0
7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
9	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
10	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
11	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
12	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.0	0.0
13	0.0	0.0	0.0	0.0	0.1	0.1	0.4	0.4	0.0	0.0
14	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.0	0.0
15	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
16	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
17	20.0	0.0	15.7	0.0	15.3	0.0	15.6	0.0	20.5	0.0
18	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
20	0.0	20.0	0.0	15.7	0.0	15.3	0.0	15.6	0.0	20.5
21	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
22	1.0	1.0	0.5	0.5	0.5	0.5	1.0	1.0	1.0	1.0
23	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.0	0.0
24	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
25	0.0	0.0	3.0	3.0	0.0	0.0	0.0	0.0	0.0	0.0
26	0.0	0.0	0.0	0.0	5.5	5.5	4.1	4.1	0.0	0.0
27	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
28	0.4	0.4	0.3	0.3	0.4	0.4	0.4	0.4	0.2	0.2
29	0.4	0.4	0.3	0.3	0.4	0.4	0.4	0.4	0.2	0.2
30	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

4.4 Choice of values for physiological parameters used

It was necessary to assume certain physiological constants in order to run the model. The values used for these constants (summarized in Table 8) were found in the literature as follows.

Gastrointestinal absorption

Gastrointestinal absorption has been found to vary from 10 to 15% in adults. We used values of 15% in this study since the value has appeared more often in more recent literature. Values as high as 50% have been reported in children (EPA, 1987). However, these are for younger children than we are assuming in this study. Therefore we retain values of 15% for children as well. We used values of 20% for babies.

Retention of lead in the lungs

Lead retention values for adults vary from 30 to 50% dependent on particle size (EPA, 1987). Values for smokers are substantially higher (55%) than for non-smokers (35%) (Camner et al., 1973). In this study values of 35% were used for non-smoking adults and 55% for smoking adults. The same values were used for children exposed and unexposed to passive smoking.

Absorption of lead from the lungs

The lead that is retained by the lungs is totally absorbed (Chamberlain 1983; EPA, 1987). Absorption was thus set at 100%.

Ventilation rates

Standard ventilation rates of 16 to 25 m³/day were used.

Blood volume

Blood volume varies by age and sex. Values for adult men were assigned as 55 dl, whereas adult women were assigned 40 dl. Since the majority of nonsmoking retired persons were women, that group was also assigned a value of 40 dl, whereas the majority of smoking retired person were men and therefore given the value of 55 dl. Values for children can be estimated using 60 ml per kg body weight (Åstrand, Rodahl, 1977). Examination of the population age group revealed that value of 25 dl was a good estimate.

Table 8: Assumed coefficients for blood volume, ventilation rate, lung retention of lead and gastrointestinal absorption of lead in different population subgroups.

Population subgroup	Blood volume (dl)	Ventilation rate (m ³ /day)	Lung retention (%)	Gastro-intestinal absorption (%)
Adult men				
Non-smokers	55	25	35	15
Smokers	55	25	55	15
Adult women				
Nonsmokers	40	20	35	15
Smokers	40	20	55	15
Retired persons				
Nonsmokers	40	16	35	15
Smokers	55	16	55	15
Children				
Unexposed to passive smoking	25	25	35	15
Exposed to passive smoking	25	25	55	15
Babies				
Unexposed to passive smoking	15	20	60	15
Exposed to passive smoking	15	20	85	20

Correction for blood-bone transfer

After comparing estimated values (using the compartment model) to measured values (Clench-Aas, Sivertsen, 1989), a correction for the transfer from blood to bone storage that differs by age was done. This is documented in the literature to occur (Hernández-Avila et al., 1998; O’Flaherty, 1993, 1995 a, b, 1997), but the actual transfer amounts is under discussion. We used the algorithms calculated in our report, as seen in Appendix A. This correction led to a decrease of the final blood concentration due to transfer of lead from blood to bone for children, and an increase for retired persons for an outward flux of lead from bone reserves to blood in the elderly.

The algorithms, based on our own comparisons in Appendix A are (where X is estimated PbB values and Y is adjusted PbB):

For children: $Y = 0.78X$

For retired non-smokers: $Y = 1.77X$

For retired smokers: $Y = 2.76 X$

5 Dose-response relationships

The effects of lead upon the cardiovascular and nervous system originate from exposure to concentrations of lead in blood. Therefore, the blood lead concentrations are representative of the dose.

The relationship between blood lead and mortality from cardiovascular disease in adults and decrease in IQ in children is reviewed elsewhere and not repeated here (Ostro, 1997; Dubourg, 1996). A series of assumptions is needed however, as is elucidated in the European Commission’s working group document for lead.

1. Current measured “baseline” blood lead levels of minimal anthropogenic origin are probably between 1 and 3 $\mu\text{g}/\text{dl}$.
2. Various international expert groups have determined that the earliest adverse effects of lead in populations of young children begin at 10 to 15 $\mu\text{g}/\text{dl}$. It is assumed to be prudent to derive a guideline value based on the lowest value of this range (10 $\mu\text{g}/\text{dl}$).
3. Inhalation of airborne lead is a significant route of exposure for adults, but for young children other pathways of exposure (ingested lead) are generally more important than inhaled air lead.
4. It is recommended that efforts should be undertaken to ensure that at least 98% of an exposed population should have blood lead-levels that do not exceed 10 $\mu\text{g}/\text{dl}$. In this case, the median blood lead would not exceed 5.4 $\mu\text{g}/\text{dl}$. On this basis, the annual average air lead should not exceed 0.5 $\mu\text{g}/\text{m}^3$ (with the assumption that the upper limit of non-anthropogenic blood is 3 $\mu\text{g}/\text{dl}$).

Exposure-response relationships are provided (Table 9) for mortality from cardiovascular disease originating from increased blood pressure (DBP) in adults, and decreased IQ in children, since these are the best documented. However, high levels of lead can lead to other forms of mortality such as renal disease, and death due to infections.

Table 9: Algorithms of estimating change of risk of a health end point associated with a decreased blood lead level (PbB in µg/dl).

Health End-point	Algorithms for exposure-response relationships*
Cardiovascular morbidity in adults as change in blood pressure	$\Delta\text{DBP}=2.74(\ln\text{PbB}_2-\ln\text{PbB}_1)^{**}$
Increase in hypertension***	$\Delta\text{HYPER}=(1+\exp(2.744-0.793\ln\text{PbB}_2))^{-1} - (1+\exp(2.744-0.793\ln\text{PbB}_1))^{-1}$
Cardiovascular morbidity in adult males as change in probability of a CHD event (non-fatal myocardial infarction) in the following 10 years.****	$\Delta\text{MORB}=(1+\exp(4.996-0.030365\text{DBP}_2))^{-1} - (1+\exp(4.996-0.030365\text{DBP}_1))^{-1}$
Cardiovascular mortality in adult males as change in the 12 year probability of death from all causes as originated in DBP changes.****	$\Delta\text{MORT}=(1+\exp(-5.3158-0.03516\text{DBP}_2))^{-1} - (1+\exp(-5.3158-0.03516\text{DBP}_1))^{-1}$
Total decrements in IQ in all children	0.25 pts/(PbB ₂ -PbB ₁)
<p>* Reference (Ostro, 1997; Dubourg, 1996) ** PbB₁ or DBP₁ refers to a reference situation, and PbB₂ or DBP₂ refers to the current or maximum situation in Tbilisi as estimated by the model. *** This factor multiplied by the population provides the number at any point in time with hypertension (>90 mm Hg), not the number of new cases per year. **** This factor is multiplied by the population to provide the 10 year or 12 year rates of morbidity or mortality. The yearly rate is provided by dividing the final number by 10 or 12. The risk, according to Ostro, 1997, is considered to be half for women.</p>	

6 Estimated blood lead concentrations

The results of the model estimates can be summarized as in Table 10 and Table 11 for the population living in the suburbs and working in the city, or living in the city and working in the city.

The model was run for three situations:

4. The target situation, when the lead in gasoline does not exceed the legal limit of 13 mg lead per liter gasoline.
5. The estimated current situation, with lead levels in gasoline of 50 mg/liter.
6. The estimated worst case in 2005, with lead levels of 80 mg/liter gasoline.

The differences resulting from successful ambient lead reduction measures are also provided.

These values represent only model estimates, and especially for children and babies are highly uncertain. Ostro, 1997, reviews the literature concerning the relationship between ambient lead exposure and blood lead concentrations. The uncertain element is the amount of lead coming from food sources. Therefore the relationship 1.8 for adults and 4.2 for children is for the air lead to blood lead ratio. The model values calculated using this method were compared to blood lead values in lower exposure (Appendix A). In Tbilisi currently known measures of

blood lead in adults seems to agree with the current model estimates. Therefore we have made no adjustments. However, it is necessary to measure blood lead in the different population groups described further to compare estimates to measurements.

It should be pointed out the values for children as estimated by this method, do not seem too high relative to the measured adult levels. Furthermore the mean values estimated for 13 mg/l lead in gasoline exceed the WHO guidelines values that specify that 98% of the population should have values under 10 µg/dl.

Table 10: Reductions in blood lead concentrations reflecting the reduction between the estimated current situation (lead in gasoline 50 mg/liter) and the target situation (13 mg/liter).

Population subgroup	Blood lead levels (µg/dl) reflecting lead in gasoline 50 mg/l		Blood lead levels (µg/dl) reflecting lead in gasoline 13 mg/l		Resulting reduction in estimated blood lead levels with ambient lead removal measures (µg/dl)	
	Living suburbs	Living city center	Living suburbs	Living city center	Living suburbs	Living city center
Babies	47.4	71.9	14.9	21.0	32.5	50.9
Children	29.5	43.9	10.6	14.2	18.9	29.7
Adult women	25.0	30.2	8.3	9.7	16.7	20.5
Adult men	22.3	27.2	7.6	8.9	14.7	18.3
Retired persons	30.8	41.4	10.0	12.7	20.8	28.7

Table 11: Reductions in blood lead concentrations reflecting the reduction between the worst case in 2005 (lead in gasoline 80 mg/liter) and the target situation (13 mg/liter).

Population subgroup	Blood lead levels (µg/dl) reflecting lead in gasoline 80 mg/l		Blood lead levels (µg/dl) reflecting lead in gasoline 13 mg/l		Resulting reduction in estimated blood lead levels with ambient lead removal measures (µg/dl)	
	Living suburbs	Living city center	Living suburbs	Living city center	Living suburbs	Living city center
Babies	53.8	82.2	14.9	21.0	38.9	61.2
Children	42.1	63.7	10.6	14.2	31.5	49.5
Adult women	36.0	43.9	8.3	9.7	27.7	34.2
Adult men	32.0	39.3	7.6	8.9	24.4	30.4
Retired persons	44.6	60.6	10.0	12.7	34.6	47.9

7 Estimated benefits for the total population of Tbilisi

The benefits assumed to be derived from total elimination of lead from gasoline could be estimated and are shown in Table 12 and Table 13. The details of the calculations are shown in Appendix C for all three situations lead content in gasoline 13, 50 and 80 mg/l. The calculations were based on the following assumptions:

- the portion of lead in blood arising from exposure to lead in gasoline is reduced to 0 with future complete removal of lead from gasoline,
- the background blood lead level of 2.0 µg/dl, assuming zero lead in ambient air,
- population of 1.5 million,
- 50% of the population living in the city center

The numbers calculated for women are too high. The values for women have thus been adjusted down by 50% of the values calculated using the male coefficients (Ostro, 2000). The numbers also were calculated using the same coefficients for both those under 65 and those over 65 years, probably leading to an underestimate for the retired group. This table provides a range of benefits calculated for removing lead from gasoline, assuming the current maximum official concentrations.

Table 12: Estimated benefits from measures to reduce ambient lead from the current estimated situation (lead in gasoline 50 mg/liter) to the target level (13 mg/liter) and to total removal of lead from gasoline (0 mg/liter). The numbers are rounded.

Benefit	Estimated benefit for removal of lead in gasoline from 50 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 13 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 50 mg/l to 13 mg/l
Nr. of adults with reduced hypertension	365 000	165 000	200 000
Nr. of adults with reduced risk for non fatal heart attacks per year	1400	800	600
Nr. of adults with reduced risk for cardiovascular death per year	1500	800	600
IQ points improved per child	9.23	2.75	6.48

Table 13: Estimated benefits from measures to reduce ambient lead from the worst case in 2005 situation (lead in gasoline 80 mg/liter) to the target level (13 mg/liter) and to total removal of lead from gasoline (0 mg/liter). The numbers are rounded.

Benefit	Estimated benefit for removal of lead in gasoline from 80 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 13 mg/l to 0 mg/l	Estimated benefit for removal of lead in gasoline from 50 mg/l to 13 mg/l
Nr. of adults with reduced hypertension	440 000	165 000	275 000
Nr. of adults with reduced risk for non fatal heart attacks per year	1600	800	800
Nr. of adults with reduced risk for cardiovascular death per year	1700	800	900
IQ points improved per child	13.56	2.75	10.81

8 Discussion

The compartment model can be used to assess the effects of lead pollution reduction measures in Georgia on blood lead concentrations. It is, however, necessary to make many assumptions. The primary assumptions used in this report have to do with time-use, food consumption patterns and of course the estimated ambient lead concentrations. The errors inherent in these assumptions must be kept in mind when interpreting the final results.

The correctness of the estimates can always be discussed. The absolute levels are however, important in discussing whether the measures will successfully keep blood lead concentrations under the guidelines. The World Health Organization guideline is set at 10 µg/dl.

98% of the population should have values under 10 µg/dl. WHO therefore concludes that the median levels should be around 5.4 µg/dl.

The estimates revealed in this report indicate that blood lead concentrations in the population of Tbilisi is unacceptably high, and will become higher if nothing is done to improve the situation. The current maximum allowable limits of 13 mg/l lead in gasoline leads to estimated blood lead concentrations that exceed the WHO guideline limits.

It is vitally necessary to sample blood in the different population subgroups and check the estimates.

9 References

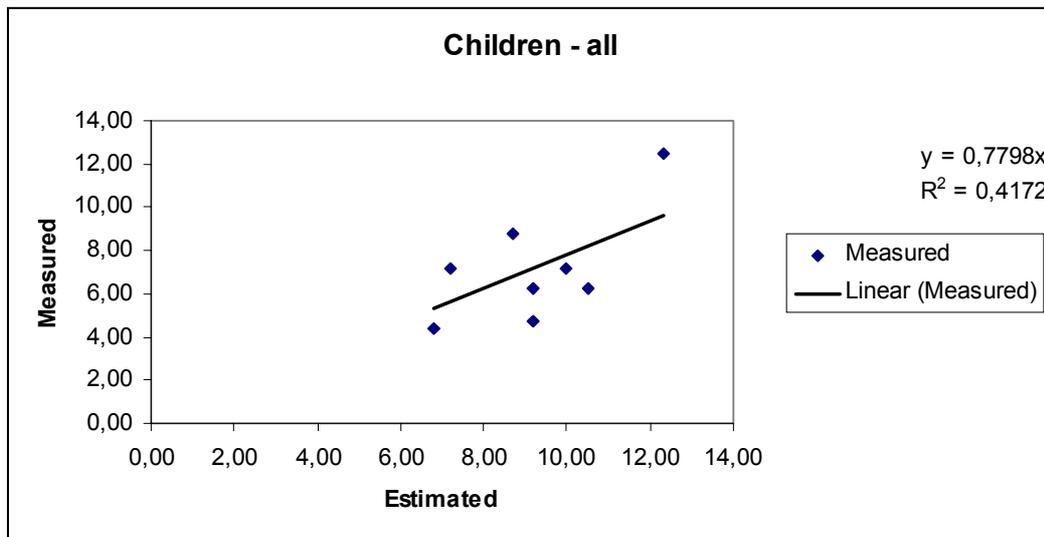
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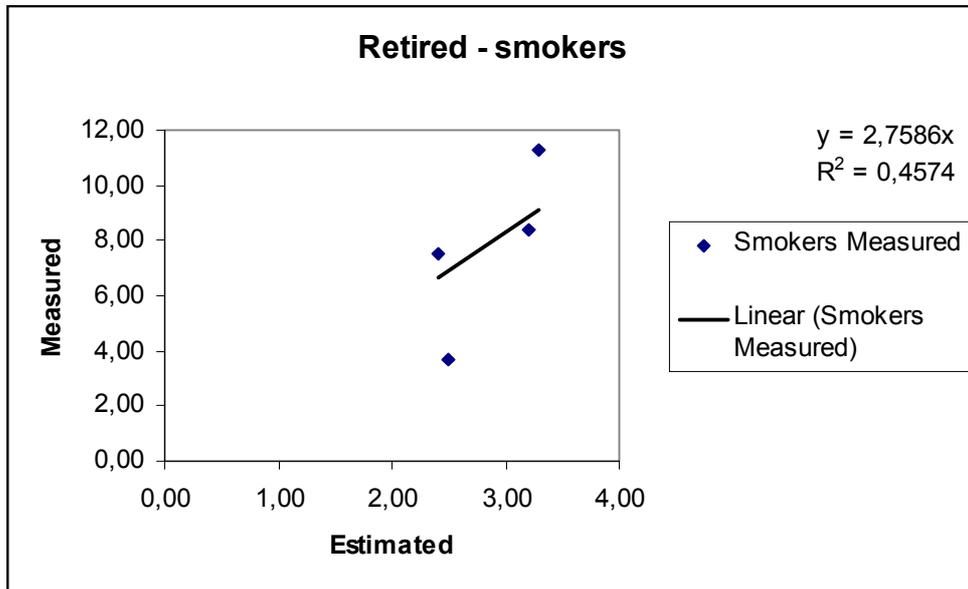
Appendix A

Relationship between estimated and measured blood lead using compartment model – results from earlier report

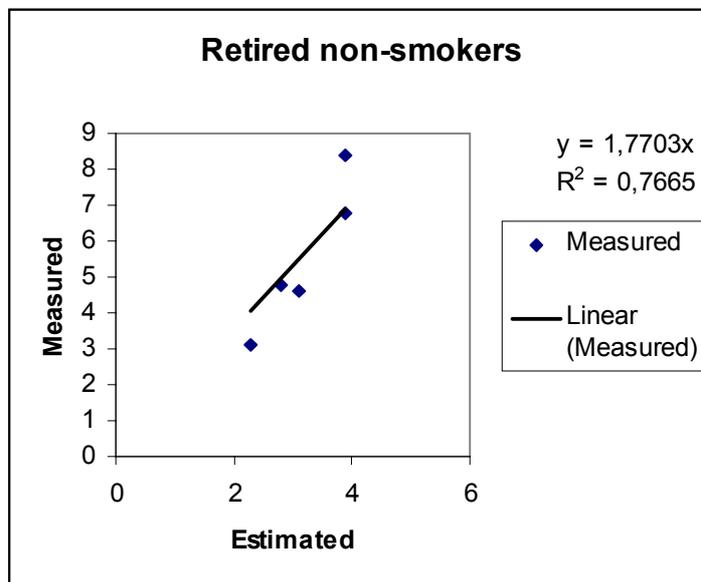
Results for children



Results for Retired Persons Smokers



Non-smokers



Appendix B

Assumptions used to run the lead compartment model in Tbilisi

Abbreviations used in compartment model.

Abbreviation	Definition	Unit
CS2	STEADY STATE CONC.IN SOIL	(UG/UG)
A4	LEAF SURFACE	(M2)
SA2	SOIL SURFACE	(M2)
FS12	STEADY STATE FLUX AIR SOIL	(UG*5**1)
T2	RESIDENCE TIME (OF LEAD) IN SOIL	(S)
MD2	MIXING DEPTH IN SOIL	(M)
RO2	DENSITY OF SOIL	(UG/M ³)
VD12	DEPOSITION VELOCITY TO SOIL	(M/S)
CS3A	STEADY STATE CON.IN PLANT A, FROM SOIL	(UG/UG)
CS7A	STEADY STATE CON.IN DIET A, FROM SOIL	(UG/UG)
CS8A	STEADY STATE CON.IN GI TRACT	(UG/UG)
CS10A	STEADY STATE CON.IN BLOOD FROM DIET A	(UG/S)
FS23A	PLANTS (ROTS) , DIET A	(UG/S)
FS37A	DIET (DIET A)	(UG/S)
FS78A	ST.STATE FLUX DIET A GI TRACT	(UG/S)
FS810A	ST.STATE FLUX GI TRACT TO BLOOD OF DIET A	(UG/S)
T10	RESIDENCE TIME OF LEAD IN BLOOD	(S)
M10	BLOOD VOLUME	(DL)
VD14B	DEP. VELOCITY TO LEAF SURF.B	(M/S)
FS14B	STEADY STATE FLUX AIR PLANT SURFACE	
FS47B	STEADY STATE FLUX PLANT DIET	
TB4	RESIDENCE TIME OF PB ON CEREALS	(S)
PRB	PRODUCTION OF CEREALS PR.M2	(UG/M ²)
VD14C	DEP. VELOCITY TO FRUIT, BERRIES	(M/S)
TC4	RESIDENCE TIME OF PB ON VEGETABLE	(S)
PRC	PRODUCTION FRUIT/BERRIES PR.M2	(UG/M ²)
TF4	RESIDENCE TIME OF PB ON VEGETABLE	(S)
FR	RETENTION OF LEAD INHALED INTO PULMONARY REGIONS OF LUNG (0.35)	
KBR	BREATHING RATE FACTOR	
RF(I)	ABSORPTION OF INGESTED PB INTO BLOOD (0.1 ADULTS, 0.5 CHILDREN)	
BACK	INTAKE OF FOOD NOT INCLUDED IN COMPONENT MODEL	(UG/D)
FABS	FRACTIONS LEAD INTO LUNG ABSORBED IN BLOOD (0.5)	
T(I)	TIME SPENT IN EACH ENVIRONMENT (H)	
BRJ	(LUNG VENTILATION) BREATHING RATE	(M ³ /DAY)
MA	CONSUM POTATO	(G/D)
MB	CONSUM CERCALS	(G/D)
MC	CONSUM FRUIT/BERRIES	(G/D)
MD	CONSUM MEAT	(G/D)
ME	CONSUM MILK	(G/D)
MF	CONSUM SURF.VEG.	(G/D)
MN(J)	NUMBER OF PERSONS IN GROUPS J	

The amount of time spent in each micronvironment by each of the population subgroups, input to compartment model

XNFI	0.2	0.5	0.1	0.1	0.2	0.2	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	15.8	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XNFJ	0.0	0.2	0.0	0.2	0.4	0.3	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.015.9	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XNFK	0.2	0.0	0.2	0.4	0.3	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.016.4	0.0	0.0	0.0	0.0	0.5	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XNOIL	0.2	0.8	0.1	0.0	0.0	0.0	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15.7	0.0	0.0	0.0	0.0	0.0	0.5	0.0	5.5	0.0	0.0	0.0	0.4	0.4	0.0
XNOJ	0.0	0.2	0.0	0.2	0.4	0.3	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.015.7	0.0	0.0	0.0	0.0	0.0	0.5	0.0	5.5	0.0	0.0	0.0	0.4	0.4	0.0
XNOK	0.0	0.2	0.0	0.0	0.0	0.0	0.5	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.016.0	0.0	0.0	0.0	0.0	0.5	0.0	5.5	0.0	0.0	0.0	0.4	0.4	0.0
XNEI	0.4	0.7	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	15.4	0.0	0.0	0.0	0.0	0.0	0.5	0.1	0.0	0.0	5.5	0.0	0.4	0.4	0.0
XNEJ	0.3	0.2	0.2	0.1	0.4	0.3	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	0.015.3	0.0	0.0	0.0	0.0	0.0	0.5	0.1	0.0	0.0	5.5	0.0	0.4	0.4	0.0
XNFKP	0.0	0.2	0.0	0.1	0.2	0.2	0.3	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.015.6	0.0	0.0	0.0	0.0	0.5	0.0	0.0	5.5	0.0	0.0	1.0	0.0	0.0
XNOKP	0.0	0.2	0.0	0.0	0.0	0.0	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.016.2	0.0	0.0	0.0	0.0	0.5	0.0	5.5	0.0	0.0	0.0	1.0	0.0	0.0
XNEJP	0.1	0.2	0.2	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	0.016.0	0.0	0.0	0.0	0.0	0.0	0.5	0.1	0.0	0.0	5.5	0.0	1.0	0.0	0.0
XNUI	0.2	2.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	18.8	0.0	0.0	0.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
XNUJ	0.0	1.0	0.0	0.2	0.4	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.019.3	0.0	0.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
XNUK	0.0	1.0	0.0	0.0	0.0	0.0	0.5	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.019.2	0.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
XMFI	0.2	0.5	0.1	0.1	0.2	0.2	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.015.8	0.0	0.0	0.5	0.0	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XMFI	0.0	0.2	0.0	0.2	0.4	0.3	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.015.9	0.0	0.5	0.0	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XMFK	0.1	0.2	0.0	0.1	0.2	0.2	0.3	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.016.4	0.5	0.0	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XMOI	0.2	0.5	0.1	0.0	0.0	0.0	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.016.0	0.0	0.0	0.5	0.0	5.5	0.0	0.0	0.0	0.0	0.4	0.4	0.0
XMOJ	0.0	0.2	0.0	0.2	0.4	0.3	0.2	0.2	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.015.7	0.0	0.5	0.0	5.5	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
XMOK	0.0	0.2	0.0	0.0	0.0	0.5	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.016.0	0.5	0.0	5.5	0.0	0.0	0.0	0.0	0.4	0.4	0.0
XMEI	0.4	0.7	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	0.0	0.0	0.015.4	0.0	0.0	0.5	0.1	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XMEJ	0.3	0.2	0.2	0.1	0.4	0.3	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.1	0.0
	0.0	0.0	0.0	0.015.3	0.0	0.5	0.1	0.0	0.0	5.5	0.0	0.0	0.4	0.4	0.0
XMFKP	0.0	0.2	0.0	0.1	0.2	0.2	0.3	0.3	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.015.6	0.5	0.0	0.0	5.5	0.0	0.0	0.0	1.0	0.0	0.0
XMOKP	0.0	0.2	0.0	0.0	0.0	0.0	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.016.2	0.5	0.0	5.5	1.0	0.0	0.0	0.0	0.0	0.0	0.0
XMEJP	0.3	0.2	0.2	0.2	0.4	0.3	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0	0.0
	0.0	0.0	0.0	0.016.0	0.0	0.5	0.1	0.0	0.0	5.5	0.0	1.0	0.0	0.0	0.0
XMUI	0.2	2.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.018.8	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.8	0.0
XMUJ	0.0	0.1	0.0	0.2	0.4	0.3	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.019.3	0.2	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.8	0.0
XMUK	0.1	0.0	0.0	0.0	0.0	0.0	0.5	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.019.2	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.8	0.0
YNFI	0.2	0.5	0.1	0.1	0.3	0.3	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	16.6	0.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	4.0	0.0	0.0	0.4	0.4	0.0
YNFJ	0.0	0.2	0.1	0.2	0.5	0.4	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.016.7	0.0	0.0	0.0	0.0	1.0	0.0	0.0	4.0	0.0	0.0	0.0	0.4	0.4	0.0
YNFK	0.0	0.2	0.1	0.1	0.3	0.3	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.017.1	0.0	0.0	0.0	1.0	0.0	0.0	4.0	0.0	0.0	0.0	0.4	0.4	0.0
YNOI	0.2	0.5	0.1	0.0	0.0	0.0	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	16.8	0.0	0.0	0.0	0.0	0.0	1.0	0.0	4.0	0.0	0.0	0.0	0.4	0.4	0.0
YNOJ	0.0	0.2	0.0	0.2	0.5	0.4	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.016.3	0.0	0.0	0.0	0.0	1.0	0.0	4.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YNOK	0.0	0.2	0.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.016.8	0.0	0.0	0.0	1.0	0.0	4.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YNEIL	0.5	0.9	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	16.0	0.0	0.0	0.0	0.0	0.0	1.0	0.1	0.0	0.0	4.0	0.0	0.4	0.4	0.0
YNEJ	0.3	0.2	0.2	0.2	0.5	0.4	0.0	0.0	0.0	0.0	0.0	0.1	0.4	0.1	0.0
	0.015.6	0.0	0.0	0.0	0.0	1.0	0.1	0.0	0.0	4.0	0.0	0.0	0.4	0.4	0.0
YNFKP	0.0	0.2	0.0	0.1	0.2	0.2	0.3	0.3	0.3	0.1	0.0	0.0	0.0	0.0	0.0
	0.0	0.016.3	0.0	0.0	0.0	1.0	0.0	0.0	4.0	0.0	0.0	0.0	1.0	0.0	0.0
YNOKP	0.0	0.2	0.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0

	0.0	0.016.6	0.0	0.0	0.0	1.0	0.0	4.0	0.0	0.0	0.0	1.0	0.0	0.0	
YNEJP	0.4	0.3	0.2	0.2	0.4	0.3	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
	0.016.0	0.0	0.0	0.0	0.0	0.0	1.0	0.1	0.0	0.0	4.0	0.0	1.0	0.0	0.0
YNUJ	0.3	2.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	17.3	0.0	0.0	0.0	0.0	0.0	3.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YNUJ	0.0	1.5	0.0	0.2	0.5	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.017.6	0.0	0.0	0.0	0.0	0.0	3.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YNUK	0.0	1.5	0.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.017.5	0.0	0.0	0.0	0.0	3.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YMFJ	0.2	0.5	0.1	0.1	0.3	0.3	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.016.6	0.0	0.0	0.0	1.0	0.0	0.0	4.0	0.0	0.0	0.4	0.4	0.0
YMFJ	0.0	0.2	0.1	0.2	0.5	0.4	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.016.7	0.0	0.0	1.0	0.0	0.0	4.0	0.0	0.0	0.4	0.4	0.0
YMFJ	0.0	0.2	0.1	0.1	0.3	0.3	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.017.1	1.0	0.0	0.0	0.0	4.0	0.0	0.0	0.4	0.4	0.0
YMOI	0.2	0.5	0.1	0.0	0.0	0.0	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.016.8	0.0	0.0	0.0	1.0	0.0	4.0	0.0	0.0	0.0	0.4	0.4	0.0
YMOJ	0.0	0.2	0.0	0.2	0.5	0.4	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.016.3	0.0	0.0	1.0	0.0	0.4	0.0	0.0	0.0	0.4	0.4	0.0
YMOK	0.0	0.2	0.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.016.8	1.0	0.0	0.0	4.0	0.0	0.0	0.0	0.4	0.4	0.0
YMEIL	0.5	0.9	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	0.0	0.0	0.016.0	0.0	0.0	0.0	1.0	0.1	0.0	0.0	4.0	0.0	0.4	0.4	0.0
YMEJ	0.3	0.2	0.2	0.2	0.5	0.4	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.0
	0.0	0.0	0.0	0.015.6	0.0	0.0	1.0	0.1	0.0	0.0	4.0	0.0	0.4	0.4	0.0
YMDKP	0.0	0.2	0.0	0.0	0.2	0.2	0.3	0.3	0.3	0.1	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.016.3	1.0	0.0	0.0	0.0	4.0	0.0	0.0	1.0	0.0	0.0
YMOKP	0.0	0.2	0.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.016.6	1.0	0.0	0.0	4.0	0.0	0.0	0.0	1.0	0.0	0.0
YMEJP	0.4	0.3	0.2	0.2	0.4	0.3	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
	0.0	0.0	0.0	0.016.0	0.0	0.0	1.0	0.1	0.0	0.0	4.0	0.0	1.0	0.0	0.0
YMUJ	0.3	2.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.017.3	0.0	0.0	0.0	3.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YMUJL	0.0	1.5	0.0	0.2	0.7	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.017.4	0.0	0.0	3.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
YMUJ	0.0	1.5	0.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.017.5	3.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
CNSI	1.0	2.2	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	15.7	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.0	0.0	3.0	0.0	0.3	0.3	0.0
CNSJ	0.0	0.2	0.0	1.0	1.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.015.7	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.0	3.0	0.0	0.0	0.3	0.3	0.0
CNSK	0.0	0.2	0.0	0.0	0.0	0.0	2.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.015.7	0.0	0.0	0.0	0.0	0.5	0.0	3.0	0.0	0.0	0.0	0.3	0.3	0.0
BNI	0.0	2.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20.0	0.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
BNJ	0.0	0.2	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.020.0	0.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
RNI	1.0	1.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	20.5	0.0	0.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RNJ	0.0	0.1	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.020.5	0.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RNK	0.0	0.1	0.0	0.0	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.020.5	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RNKH	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.020.5	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RMI	1.0	1.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.020.5	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RMJ	0.0	0.1	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.020.5	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RMK	0.0	0.1	0.0	0.0	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.020.5	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.2	0.0
RMKH	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.024.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
CMSI	1.0	2.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.015.7	0.0	0.0	0.5	0.0	0.0	0.0	3.0	0.0	0.0	0.3	0.3	0.0
CMSJ	0.0	0.2	0.0	1.0	1.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.015.7	0.0	0.5	0.0	0.0	0.0	3.0	0.0	0.0	0.3	0.3	0.0
CMSK	0.0	0.2	0.0	0.0	0.0	0.0	2.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.015.7	0.5	0.0	3.0	0.0	0.0	0.0	0.0	0.3	0.3	0.0
BMI	0.0	2.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.020.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
BMJ	0.0	2.2	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.020.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
BNK	0.0	0.2	0.0	0.0	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.020.0	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0
BMK	0.0	0.2	0.0	0.0	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.020.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	0.4	0.0

Biological coefficients used in model prediction (see first page in Appendix for abbreviations) for each population subgroup.

	MA	MB	MC	MD	ME	MF	BRJ	M10J	MN	FR	RF
XNFI	190.	240.	50.	120.	480.	80.	25.	55.	16979.	0.35	0.15
XNFJ	190.	240.	50.	120.	480.	80.	25.	55.	24255.	0.35	0.15
XNFK	190.	240.	50.	120.	480.	80.	25.	55.	2426.	0.35	0.15
XNOIL	180.	230.	50.	120.	420.	80.	25.	55.	23447.	0.35	0.15
XNOJ	180.	230.	50.	120.	420.	80.	25.	55.	16170.	0.35	0.15
XNOK	190.	240.	50.	120.	420.	80.	25.	55.	7277.	0.35	0.15
XNEI	190.	240.	50.	120.	420.	80.	25.	55.	8894.	0.35	0.15
XNEJ	190.	240.	50.	120.	420.	80.	25.	55.	11319.	0.35	0.15
XNFKP	190.	240.	50.	120.	420.	80.	25.	55.	7277.	0.35	0.15
XNOKP	190.	240.	50.	120.	420.	80.	25.	55.	9702.	0.35	0.15
XNEJP	190.	240.	50.	120.	420.	80.	25.	55.	9702.	0.35	0.15
XNUI	180.	250.	50.	90.	480.	80.	25.	55.	4851.	0.35	0.15
XNUJ	180.	250.	50.	90.	480.	80.	25.	55.	4851.	0.35	0.15
XNUK	180.	250.	50.	90.	480.	80.	25.	55.	4851.	0.35	0.15
XMPFI	200.	250.	50.	120.	480.	80.	25.	55.	32113.	0.55	0.15
XMFJ	200.	250.	50.	120.	480.	80.	25.	55.	46163.	0.55	0.15
XMPK	200.	250.	50.	120.	480.	80.	25.	55.	4014.	0.55	0.15
XMOI	180.	230.	50.	120.	420.	80.	25.	55.	42149.	0.55	0.15
XMOJ	180.	230.	50.	120.	420.	80.	25.	55.	30106.	0.55	0.15
XMOK	180.	230.	50.	120.	420.	80.	25.	55.	12043.	0.55	0.15
XMEI	190.	240.	50.	120.	480.	80.	25.	55.	18064.	0.55	0.15
XMEJ	190.	240.	50.	120.	480.	80.	25.	55.	22078.	0.55	0.15
XMPKP	190.	240.	50.	120.	480.	80.	25.	55.	12043.	0.55	0.15
XMOKP	180.	230.	50.	120.	480.	80.	25.	55.	20071.	0.55	0.15
XMEJP	200.	250.	50.	120.	480.	80.	25.	55.	20071.	0.55	0.15
XMUI	180.	260.	50.	90.	480.	80.	25.	55.	8028.	0.55	0.15
XMUJ	180.	260.	50.	90.	480.	80.	25.	55.	8028.	0.55	0.15
XMUK	180.	260.	50.	90.	480.	80.	25.	55.	8028.	0.55	0.15
YNFI	130.	160.	50.	90.	360.	60.	20.	40.	19951.	0.35	0.15
YNFJ	130.	160.	50.	90.	360.	60.	20.	40.	16324.	0.35	0.15
YNFK	130.	160.	50.	90.	360.	60.	20.	40.	5441.	0.35	0.15
YNOI	120.	140.	50.	90.	360.	60.	20.	40.	43529.	0.35	0.15
YNOJ	120.	140.	50.	90.	360.	60.	20.	40.	21765.	0.35	0.15
YNOK	120.	140.	50.	90.	360.	60.	20.	40.	16324.	0.35	0.15
YNEIL	130.	150.	50.	90.	360.	80.	20.	40.	10882.	0.35	0.15
YNEJ	130.	150.	50.	90.	360.	60.	20.	40.	16324.	0.35	0.15
YNFKP	130.	150.	50.	90.	360.	60.	20.	40.	10882.	0.35	0.15
YNOKP	120.	150.	50.	90.	360.	60.	20.	40.	21765.	0.35	0.15
YNEJP	140.	150.	50.	90.	360.	60.	20.	40.	10882.	0.35	0.15
YNUI	110.	150.	50.	70.	360.	60.	20.	40.	101569.	0.35	0.15
YNUJ	110.	160.	50.	70.	360.	60.	20.	40.	45343.	0.35	0.15
YNUK	110.	160.	50.	70.	360.	60.	20.	40.	29020.	0.35	0.15
YMPFI	140.	150.	50.	90.	360.	60.	20.	40.	4466.	0.55	0.15
YMFJ	140.	150.	50.	90.	360.	60.	20.	40.	2977.	0.55	0.15
YMPK	140.	150.	50.	90.	360.	60.	20.	40.	992.	0.55	0.15
YMOI	120.	140.	50.	90.	360.	60.	20.	40.	7939.	0.55	0.15
YMOJ	120.	140.	50.	90.	360.	60.	20.	40.	3969.	0.55	0.15
YMOK	120.	140.	50.	90.	360.	60.	20.	40.	2977.	0.55	0.15
YMEIL	130.	150.	50.	90.	360.	80.	20.	40.	1985.	0.55	0.15
YMEJ	130.	150.	50.	90.	360.	60.	20.	40.	2977.	0.55	0.15
YMDKP	130.	150.	50.	90.	360.	60.	20.	40.	1985.	0.55	0.15
YMOKP	130.	150.	50.	90.	360.	60.	20.	40.	3969.	0.55	0.15
YMEJP	130.	150.	50.	90.	360.	60.	20.	40.	1985.	0.55	0.15
YMUI	110.	160.	50.	90.	360.	60.	20.	40.	16870.	0.55	0.15
YMUJL	110.	160.	50.	90.	360.	60.	20.	40.	7443.	0.55	0.15
YMUJ	110.	160.	50.	90.	360.	60.	20.	40.	4466.	0.55	0.15
YMUJL	110.	160.	50.	90.	360.	60.	20.	40.	4466.	0.55	0.15
CNSI	100.	230.	30.	80.	550.	40.	25.	22.	49385.	0.35	0.15
CNSJ	100.	230.	30.	80.	550.	40.	25.	22.	41154.	0.35	0.15
CNSK	100.	230.	30.	80.	550.	40.	25.	22.	16462.	0.35	0.15
BNI	50.	100.	20.	20.	400.	30.	20.	15.	16200.	0.50	0.15
BNJ	50.	100.	20.	20.	400.	30.	20.	15.	8100.	0.50	0.15
RNI	70.	150.	30.	50.	100.	40.	16.	40.	25684.	0.35	0.15
RNJ	70.	150.	30.	50.	100.	40.	16.	40.	25684.	0.35	0.15
RNK	70.	150.	30.	50.	100.	40.	16.	40.	19263.	0.35	0.15
RNKH	70.	150.	30.	50.	100.	40.	16.	40.	51368.	0.35	0.15
RMI	70.	150.	30.	50.	100.	40.	16.	55.	2857.	0.55	0.15
RMJ	70.	150.	30.	50.	100.	40.	16.	55.	4286.	0.55	0.15
RMK	70.	150.	30.	50.	100.	40.	16.	55.	1429.	0.55	0.15
RMKH	70.	150.	30.	50.	100.	40.	16.	55.	11429.	0.55	0.15
CMSI	100.	230.	30.	80.	550.	40.	25.	22.	133846.	0.55	0.15
CMSJ	100.	230.	30.	80.	550.	40.	25.	22.	111538.	0.55	0.15
CMSK	100.	230.	30.	80.	550.	40.	25.	22.	44615.	0.55	0.15
BMI	50.	100.	20.	20.	400.	20.	20.	15.	36000.	0.65	0.20
BMJ	50.	100.	20.	20.	400.	20.	20.	15.	18000.	0.65	0.20
BNK	50.	100.	20.	20.	400.	20.	20.	15.	2700.	0.50	0.20
BMK	50.	100.	20.	20.	400.	20.	20.	15.	18000.	0.65	0.20

Appendix C

Benefit estimates for different levels of lead in gasoline

Estimates with 80 mg/l gasoline
Suburban living, working in city center

Population subgroup	Adult men	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Population numbers	435000	152250	282750	435000	369750	65250	142500	122550	19950	396000	106920	289080	99000	26730	72270	1507500
Half population		76125	141375	217500	184875	32625	71250	61275	9975	198000	53460	144540	49500	13365	36135	753750
Blood lead 1		2,00	2,00		2,00	2,00		2,00	2,00		2,00	2,00				
Blood lead 2		28,90	35,20		32,70	39,30		35,93	53,27		37,21	47,03				
DBP1		84,70	84,70		80,60	80,60		92,00	92,00		64,00	64,00				
DBP2		92,02	92,56		88,26	88,76		99,91	100,99		72,01	72,65				
Change in DBP		7,32	7,86		7,66	8,16		7,91	8,99		8,01	8,65				
Change in H		0,38063	0,41971		0,40511	0,44146		0,42377	0,50045							
Change in Pr(CHD)		0,01823	0,01971		0,01728	0,01853		0,02371	0,02729							
Change in Pr(MORT)		0,02297	0,02486		0,02148	0,02306		0,03056	0,03523							
Change in IQ points											8,8015	11,2585				

Estimates with 80 mg/l gasoline, contd.
City center living, working in city center

Population subgroup	Adult men	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Population numbers	435000	152250	282750	435000	369750	65250	142500	122550	19950	396000	106920	289080	99000	26730	72270	1507500
Half population		76125	141375	217500	184875	32625	71250	61275	9975	198000	53460	144540	49500	13365	36135	753750
Blood lead 1		2,00	2,00		2,00	2,00		2,00	2,00		2,00	2,00				
Blood lead 2		34,500	44,100		38,900	48,900		50,445	70,932		54,054	73,320				
DBP1		84,70	84,70		80,60	80,60		92,00	92,00		64,00	64,00				
DBP2		92,50	93,18		88,73	89,36		100,84	101,78		73,03	73,87				
Change in DBP		7,80	8,48		8,13	8,76		8,84	9,78		9,03	9,87				
Change in H		0,41573	0,46405		0,43945	0,48407		0,49005	0,55349							
Change in Pr(CHD)		0,01956	0,02142		0,01846	0,02005		0,02679	0,02995							
Change in Pr(MORT)		0,02466	0,02705		0,02297	0,02498		0,03458	0,03871							
Change in IQ points											13,0135	17,83				

Estimates with 80 mg/l gasoline, contd.

Population subgroup	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Suburbs living															
Hypertension reductions	28975	59336	0	74895	14403	0	25967	4992							208568
Reductions in Pr(CHD)	139	279	0	160	30	0	145	27							780
Reductions in Pr(MORT)	146	293	0	165	31	0	156	29							821
Reduction in IQ points										588160	2034129				2622290
City living numbers															
Hypertension reductions	31647	65605	0	81243	15793	0	30028	5521							229837
Reductions in Pr(CHD)	149	303	0	171	33	0	164	30							849
Reductions in Pr(MORT)	156	319	0	177	34	0	177	32							895
Reduction in IQ points										869627	3221435				4091062
Total numbers															
Hypertension reductions	60623	124941		156138	30196		55995	10513							438405
Reductions in Pr(CHD)	288	582		330	63		309	57							1629
Reductions in Pr(MORT)	302	611		342	65		333	61							1715
Reduction in IQ points										1457787	5255565				6713352

Estimates with 50 mg/l gasoline
Suburban living, working in city center

Population subgroup	Adult men	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Population numbers	435000	152250	282750	435000	369750	65250	142500	122550	19950	396000	106920	289080	99000	26730	72270	1507500
Half population		76125	141375	217500	184875	32625	71250	61275	9975	198000	53460	144540	49500	13365	36135	753750
Blood lead 1		2,00	2,00		2,00	2,00		2,00	2,00		2,00	2,00				
Blood lead 2		20,20	24,40		22,80	27,20		24,96	36,71		26,29	32,76				
DBP1		84,70	84,70		80,60	80,60		92,00	92,00		64,00	64,00				
DBP2		91,04	91,55		87,27	87,75		98,92	99,97		71,06	71,66				
Change in DBP		6,34	6,85		6,67	7,15		6,92	7,97		7,06	7,66				
Change in H		0,310579634	0,34726		0,334003	0,368643		0,351689	0,428003							
Change in Pr(CHD)		0,015586878	0,016972		0,014854	0,016032		0,020467	0,023899							
Change in Pr(MORT)		0,019606507	0,021367	0	0,018433	0,019912	0	0,026348	0,03081							
Change in IQ points											6,0715	7,69				

**Estimates with 50 mg/l gasoline, contd.
City center living, working in city center**

Population subgroup	Adult men	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Population numbers	435000	152250	282750	435000	369750	65250	142500	122550	19950	396000	106920	289080	99000	26730	72270	1507500
Half population		76125	141375	217500	184875	32625	71250	61275	9975	198000	53460	144540	49500	13365	36135	753750
Blood lead 1		2,00	2,00		2,00	2,00		2,00	2,00		2,00	2,00				
Blood lead 2		24,00	30,30		26,90	33,50		34,52	48,30		37,44	50,31				
DBP1		84,70	84,70		80,60	80,60		92,00	92,00		64,00	64,00				
DBP2		91,51	92,15		87,72	88,32		99,80	100,72		72,03	72,84				
Change in DBP		6,81	7,45		7,12	7,72		7,80	8,72		8,03	8,84				
Change in H		0,344021119	0,390001		0,366453	0,409902		0,415815	0,481694							
Change in Pr(CHD)		0,016849647	0,018581		0,015957	0,017441		0,023346	0,026391							
Change in Pr(MORT)		0,021212139	0,023417		0,019819	0,021685		0,03009	0,034058							
Change in IQ points											8,86	12,0775				

Estimates with 50 mg/l gasoline, contd.

	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Total
Suburbs living numbers												
Hypertension reductions	23643	49094	0	61749	12027	0	21550	4269				172331
Reductions in Pr(CHD)	119	240	0	137	26	0	125	24				671
Reductions in Pr(MORT)	124	252	0	142	27	0	135	26				705
Reduction in IQ points										405728	1389391	1795119
City living numbers												
Hypertension reductions	26189	55136	0	67748	13373	0	25479	4805				192730
Reductions in Pr(CHD)	128	263	0	148	28	0	143	26				736
Reductions in Pr(MORT)	135	276	0	153	29	0	154	28				775
Reduction in IQ points										592069,5	2182102	2774172
Total numbers												
Hypertension reductions	49831	104230		129497	25400		47029	9074				365061
Reductions in Pr(CHD)	247	503		285	55		268	50				1408
Reductions in Pr(MORT)	259	528		295	57		288	54				1480
Reduction in IQ points										997797,5	3571493	4569291

**Estimates with 13 mg/l gasoline
Suburban living, working in city center**

Population subgroup	Adult men	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Population numbers	435000	152250	282750	435000	369750	65250	142500	122550	19950	396000	106920	289080	99000	26730	72270	1507500
Half population		76125	141375	217500	184875	32625	71250	61275	9975	198000	53460	144540	49500	13365	36135	753750
Blood lead 1		2,00	2,00		2,00	2,00		2,00	2,00		2,00	2,00				
Blood lead 2		7,20	8,30		7,80	8,90		8,50	11,59		9,83	11,47				
DBP1		84,70	84,70		80,60	80,60		92,00	92,00		64,00	64,00				
DBP2		88,21	88,60		84,33	84,69		95,96	96,81		68,36	68,78				
Change in DBP		3,51	3,90		3,73	4,09		3,96	4,81		4,36	4,78				
Change in H		0,13505191	0,1559404		0,1466646	0,1666291		0,1594833	0,2095726							
Change in Pr(CHD)		0,00832861	0,009299		0,0079948	0,0088112		0,0113179	0,0138917							
Change in Pr(MORT)		0,01042512	0,0116477		0,0098665	0,0108814		0,0145084	0,0178297							
Change in IQ points											1,957	2,3665				

Estimates with 13 mg/l gasoline, contd.
City center living, working in city center

Population subgroup	Adult men	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Population numbers	435000	152250	282750	435000	369750	65250	142500	122550	19950	396000	106920	289080	99000	26730	72270	1507500
Half population		76125	141375	217500	184875	32625	71250	61275	9975	198000	53460	144540	49500	13365	36135	753750
Blood lead 1		2,00	2,00		2,00	2,00		2,00	2,00		2,00	2,00				
Blood lead 2		8,100	9,800		8,900	10,600		10,797	14,628		12,636	15,834				
DBP1		84,70	84,70		80,60	80,60		92,00	92,00		64,00	64,00				
DBP2		88,53	89,05		84,69	85,17		96,62	97,45		69,05	69,67				
Change in DBP		3,83	4,35		4,09	4,57		4,62	5,45		5,05	5,67				
Change in H		0,1522719	0,1818391		0,1666291	0,1946106		0,1976558	0,2503333							
Change in Pr(CHD)		0,00913187	0,0104449		0,0088112	0,0099045		0,0132986	0,0158525							
Change in Pr(MORT)		0,01143704	0,0130934		0,0108814	0,0122425		0,0170637	0,020365							
Change in IQ points											2,659	3,4585				

Estimates with 13 mg/l gasoline, contd.

Population subgroup	Non-smokers	Smokers	Adult women	Non-smokers	Smokers	Retired persons	Non-smokers	Smokers	Children	Unexposed to passive smoking	Exposed to passive smoking	Babies (0-2)	Unexposed to passive smoking	Exposed to passive smoking	Total
Suburbs living numbers															
Hypertension reductions	10281	22046	0	27115	5436	0	9772	2090							76741
Reductions in Pr(CHD)	63	131	0	74	14	0	69	14							366
Reductions in Pr(MORT)	66	137	0	76	15	0	74	15							383
Reduction in IQ points										130776,53	427567,39				558343,91
City living numbers															
Hypertension reductions	11592	25708	0	30806	6349	0	12111	2497							89062
Reductions in Pr(CHD)	70	148	0	81	16	0	81	16							412
Reductions in Pr(MORT)	73	154	0	84	17	0	87	17							431
Reduction in IQ points										177687,68	624864,49				802552,16
Total numbers															
Hypertension reductions	21873	47754		57920	11785		21884	4588							165803
Reductions in Pr(CHD)	133	279		155	31		151	30							778
Reductions in Pr(MORT)	139	291		160	31		161	32							814
Reduction in IQ points										308464,2	1052431,9				1360896,1



Norsk institutt for luftforskning (NILU)

Postboks 100, N-2007 Kjeller

RAPPORTTYPE OPPDRAKS RAPPORT	RAPPORT NR. OR 43/2000	ISBN 82-425-1207-8 ISSN 0807-7207	
DATO	ANSV. SIGN.	ANT. SIDER 49	PRIS NOK 81,-
TITTEL Blood lead concentrations in the population of Tbilisi, Georgia Results of model estimates		PROSJEKTLEDER Jocelyne Clench-Aas	
		NILU PROSJEKT NR. O-98129	
FORFATTER(E) Jocelyne Clench-Aas, Manana Juruli, and Kari Arnesen		TILGJENGELIGHET * A	
		OPPDRAKSGIVERS REF.	
OPPDRAKSGIVER United Nations Office for Project Services			
STIKKORD Blood lead	Compartment model	Socio-economic effects	
REFERAT			
TITLE			
<p>ABSTRACT This report presents the results of compartment modeling of the concentrations of lead in blood for the population of Tbilisi. The blood lead concentrations are calculated both for the air pollution situation as estimated now, and as estimated if the gasoline in reality is fully leaded. This provides the range of benefits that may be anticipated with lead reduction measures in the gasoline.</p>			

* Kategorier: *A* Åpen - kan bestilles fra NILU
 B Begrenset distribusjon
 C Kan ikke utleveres