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ABSTRACT

In the spring of 1983, the Norwegian Institute for Air Research (NILU) in cooperation with the Institute of Occupational Health, conducted a study of the effects of moderately heavy traffic on blood lead concentrations in 300 men, women and children. Two sites were chosen:

1) Holmestrand - a town traversed by a major throughway (at the time of measurement, 11 000 vehicles daily) where the traffic is stopped by a light.
2) Sørumsand - a small town having very little traffic (at the time of measurement estimated at 2 500 cars daily) and no industrial sources of airborne lead.

Individual air lead exposure was estimated by combining information on weekly activity patterns from a self-administered questionnaire with both measured and estimated ambient lead concentrations. Blood lead for each individual was measured by electrothermal atomic absorption spectroscopy. The questionnaire also included information on additional lead exposure via hobbies, occupation and smoking (both active and passive).

The findings indicate that:

1) Blood lead is correlated to individual air lead exposure in all population groups.

2) Blood lead's relationship to air lead (the amount of blood lead corresponding to a specific air lead exposure) differs with age, sex and smoking habits. Children have higher blood lead for a given air lead exposure than adults (slope of regression line steeper). Children exposed to passive smoking have higher blood lead than children not exposed to passive smoking, and female smokers have higher blood lead levels for a given air lead exposure than female non-smokers, indicating in both cases that smoking or passive smoking in children possibly increased uptake of ambient lead.

3) The estimated baseline (extrapolation of linear relationship to 0 air lead) blood lead level was ca. 6.4 μg/dl (0.31 μmoles/l) in adult men; 2.9 μg/dl (0.14 μmoles/l) in adult women; and 2.5 μg/dl (0.12 μmoles/l) in children.

Therefore, this study gives preliminary indication that under conditions of chronic exposure to low to moderate air lead stemming from traffic pollution (.03 - .25 $\mu g/m^3$), inhalation can be responsible for up to 60 to 80% of the lead found in blood in the most sensitive population subgroups. Active smoking in women and passive smoking in children can significantly increase the importance of inhalation's contribution to blood lead concentrations. Therefore, in populations of children exposed to passive smoking or female smokers, reducing ambient concentrations of lead will result in considerably larger reductions in blood lead than has been previously predicted in the literature. Guidelines for highest permissible ambient lead concentrations should consider these population subgroups.

SAMMENDRAG

Barn har et noe høyere blyinnhold i blodet enn voksne ved lik blypåvirkning fra luft. Barn utsatt for passiv røyking har høyere blyopptak fra luft enn barn som ikke er utsatt for passiv røyking. Denne forskjellen øker ved økende blyinnhold i lufta.

Tilsvarende har røykende kvinner et større blyopptak fra luft enn ikke-røykere. Også her blir forskjellen større ved økende blyinnhold i lufta.

Hos menn er forskjellen mellom røykeres og ikke-røykeres blyopptak fra luft liten.

Dette er hovedkonklusjonene i undersøkelsen som NILU, i samarbeid med Yrkeshygienisk Institutt og de lokale helseråd, gjennomførte i Holmestrand og Sørumsand våren 1983. Holmestrand ble valgt pga. trafikkomleggingen "Holmestrand-tunnelen" ville medføre, og Sørumsand ble valgt som et trafikkmessig "rent" område. Døgntrafikken var i løpet av undersøkelsen ca 11 000 kjøretøyer i Holmestrand og ca 2500 kjøretøyer i Sørumsand.

Hver enkelt deltakers blypåvirkning fra luft ble beregnet på grunnlag av de luftprøvene som ble tatt både innendørs og utendørs, samt spørreskjemaet alle måtte fylle ut. Dette skjemaet inneholdt også informasjon om blypåvirkning fra hobbyer, yrke og røyking (også passiv røyking). Gjennomsnittlig blyinnhold i blodet var:

Holmestrand: Menn 8.8 µg/dl; kvinner 6.9 µg/dl og barn 8.8 µg/dl

Sørumsand : Menn 6.7 µg/dl; kvinner 3.9 µg/dl og barn 4.3 µg/dl.

En regner ikke med negative effekter når bly-konsentrasjonen i blodet er under 30 µg/dl. Ingen av de målte blodkonsentrasjoner overskred denne verdi.

Resultatene fra Sørumsand er blandt de laveste målte i Europa, og verdiene nærmer seg de bakgrunnsverdiene som finnes i kroppen uten bly i lufta i det hele tatt.

Undersøkelsen indikerer at under vedvarende påvirkning fra lavt til moderat blyinnhold i innåndingsluften fra trafikk-kilder kan innåndingen bidra med opptil 60-80% av blyinnholdet i blodet hos de mest følsomme menneskegruppene. Røyking hos kvinner og passiv røyking hos barn synes i betydelig grad å øke luft-blyets andel av blyinnholdet i blod. Der det finnes røykende kvinner og barn utsatt for passiv røyking, vil derfor en reduksjon av blyinnholdet i luft føre til en atskillig større reduksjon av blod-bly enn de som tidligere har vært omtalt i litteraturen.

FOREWORD

This report summarizes an investigation of blood lead concentrations done by the Norwegian Institute of Air Research (NILU) with the Institute of Occupational Health under conditions of moderate to low air lead exposure. This investigation is the first phase of a two part study, and was done in May 1983. The second phase repeats blood and air measurements in the two selected towns Holmestrand and Sørumsand one year later, thus in May 1984. The second phase seeks to examine to what extent a drop in blood lead occurs in the experimental town (Holmestrand) after a tunnel has removed the majority of car traffic through it.

This report is in two parts. Part I contains the main body of the report with relevant tables and graphs. Part II contains additional information and data prints of results of detailed analyses that were considered more peripheral to the study.

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BLOOD LEAD - A FUNCTION OF VEHICULAR EMISSIONS AND SMOKING

1 INTRODUCTION

To what degree lead in air contributes to body lead levels (usually estimated by blood lead) in humans is a topic that has been frequently discussed in the literature. (EPA criteria document 1977; Hammond et al., 1981; Chamberlain, 1983; and Snee, 1981) It is the fundamental question underlying the determination of air quality standards.

The relationship between blood lead and air lead concentrations has been much discussed. The discussion centers principally around two aspects:

1) The shape of the regression if blood lead (y axis in µg/dl) is plotted against air lead (x axis in $µg/m^3$) with air lead concentrations ranging from 0 to 4 or 5 $µg/m^3$. Discussion is concerned with whether the shape of the regression is linear or curvilinear. The slope of the regression is equivalent to what is known as the blood to air lead ratio. It answers the question, what change in blood lead concentration will result from a 1 $µg/m^3$ decline in air lead level. Estimates found in the literature range from 1 (1 $µg/m^3$ decline in air lead results in a 1 µg/dl decline in blood lead concentration) to 3 (a 1 $µg/m^3$ decline in air lead results in a 3 µg/dl decline in blood lead concentration). However, these estimates have been, for the most part, calculated at air lead exposures over 2 $µg/m^3$. Should the regression be curvilinear, however, this ratio will not be constant but have higher values under conditions of lower air lead exposures. As has been discussed by Chamberlain (1983), Hammond et al. (1981) and Laxen (1983), evidence is mounting that the relationship is curvilinear, thus the blood to air lead ratio is higher at lower concentrations.

2) The intercept of the regression with the y axis which is equivalent to the blood lead level when air lead levels are 0. (If $y = a + f(x)$, then if $x = 0$ $y = \underline{a}$ or the intercept). The intercept will also depend on the shape of the curve. Much of

the previous literature has extrapolated linear relationships established under relatively higher air lead exposure (over 2 or 3 $\mu g/m^3$) to 0. The value thus obtained is said to represent the body burden of lead coming from non-air sources such as food. Air has been said to account for no more than 30% of blood lead concentrations. However, it is evident, that should the blood to air relationship be curvilinear, a straight line extrapolation from blood lead values measured at higher concentrations is inappropriate.

Exposure to air lead via vehicular traffic emissions leads to low to moderate ambient concentrations. In order to properly assess the effect of reducing lead in gasoline one needs to know the blood to air lead ratio under low air lead exposure conditions where the primary air lead source is vehicular traffic. This is especially so in Scandinavia where blood lead levels are already fairly low (Nordman, 1975; Wilder, 1979; Bach, 1979; Elinder et al., 1983; Omang, Moseng, 1974; Borenstein, et al., 1979).

This relationship needs to be established for each of the population subgroups: children, women and men. When comparing these population subgroups, it is necessary to account for differences in exposure due to lifestyle. Children are out-doors more than adults, and are usually playing thus increasing respiratory ventilation.

On May 14, 1983 a tunnel was opened near a town in southern Norway, Holmestrand, that allowed traffic from a major throughway that had been congested at a traffic light to completely bypass the town. It was thus considered a unique opportunity to measure blood lead concentrations in inhabitants before and after the opening of the tunnel. This report represents phase I of the study, before the opening of the tunnel.

Due to a relatively long half-life of lead in the blood (circa 16 to 18 days - Chamberlain et al. 1978) and seasonal dependence of both air lead and possibly blood lead (Manton, 1977) it was decided to remeasure inhabitants (phase II) one year later, that is in May 1984.

Holmestrand lies by the Oslo fjord, with a 75 m cliff behind it (Figure 1). During the month preceding blood sampling an estimated 11000 vehicles, passed daily (personal communication - S. Kålås, Vegdirektoratet). However, the number of vehicles was not uniform for every day of the week. Traffic tended to be more congested on the weekends, especially in the spring and summer associated with traffic to and from vacation homes.

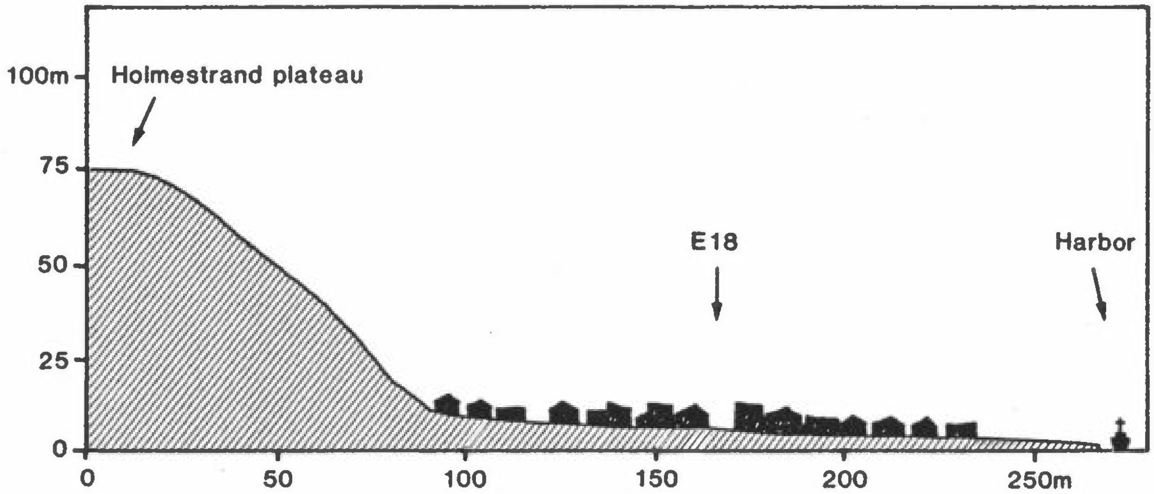

Figure 1: Cross-sectional view of Holmestrand of the traffic light.

Sørumsand was chosen as a control area. It also lies in southern Norway (Figure 2) but has no major throughway. An estimated 2500 vehicles daily crossed the largest roads in the period preceding blood sampling (personal comunication, S. Kålås, Vegdirektoratet).

In order to properly calculate the ratio of blood lead to air lead concentrations, air lead exposure estimates for each individual are necessary. However, such estimates were obtained in only one of the studies reported in the literature (Azar et al., 1975). The methods used to date to assess exposure to ambient lead in studies of blood lead levels in exposed individuals are.

1) Outdoor air samplers
2) Gasoline consumption
3) Direct measurements through portable monitors
4) The diary method - asking individuals to detail their presence in different microenvironments (e.g. inside home, outside work, inside school, etc.)

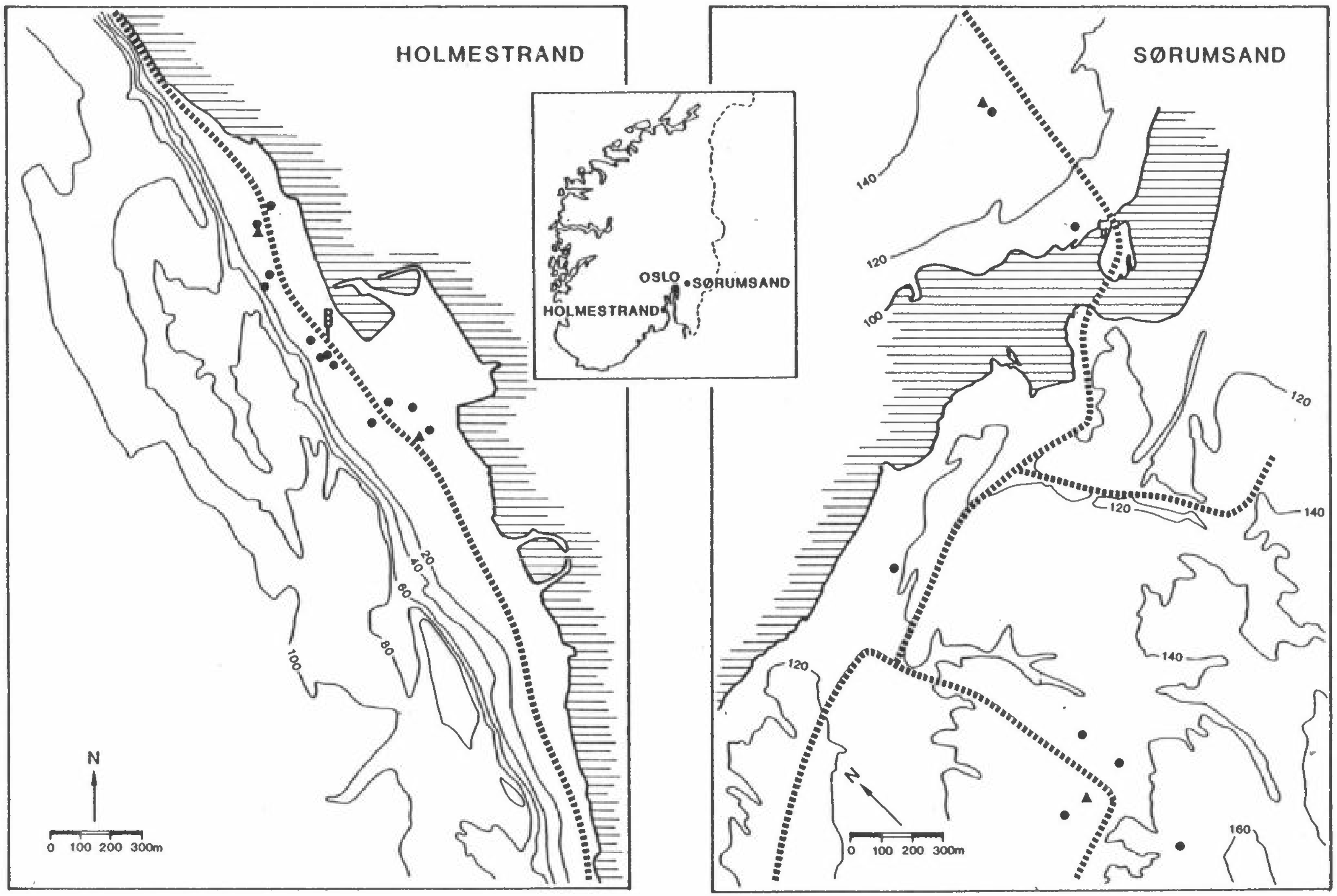

Figure 2: Topographic maps of the two study towns, Holmestrand and Sørumsand with outdoor air lead stations (▲) and indoor air lead stations (●) indicated.

Outdoor air samplers have been used in most published studies, (EPA criteria document, 1977; Hammond et al., 1981, Chamberlain, 1983, Snee, 1981). The number of stations and duration of measurement varied among studies. The use of outdoor air samplers alone can be a satisfactory method to distinguish between major differences in ambient concentrations in different regions. They are not, however, a sufficient measure of individual air lead exposure. Regional differences in the indoor to outdoor air lead ratio can result due to differences in age of buildings, use of air conditioners or season of the year with resulting changes in ventilation. Ignoring these can lead to problems in assessing the resulting actual air lead exposure in individuals. Table 1 indicates hypothetical daily average air lead concentrations children and adults are exposed to based on different indoor to outdoor air lead ratios (I/O).

Table 1: Hypothetical daily average air lead concentrations children and adults may be exposed to under differing outdoor air lead concentrations and different indoor - outdoor (I/O) ratios. Values represent an estimated hourly average of a 24 hour day.

	Outdoor Air lead level			
I/O Ratio	3 $\mu g/m^3$	2 $\mu g/m^3$	1 $\mu g/m^3$	.5 $\mu g/m^3$
Children-outside 6 hrs/day				
10% I/O	.98*[1]	.65	.32	.16
50%	1.88	1.25	.62	.31
80%	2.55	1.70	.85	.42
Adults-outside 1 hr/day				
10% I/O	.41	.30	.14	.07
50%	1.56	1.04	.52	.26
80%	2.42	1.50	.81	.40

*[1] Value calculated as: 6 hrs x outdoor value (3 $\mu g/m^3$) + 18 hrs x indoor value (=10% outdoor value) / 24 =

$$\frac{(3)(6)+(18)(.3)}{24} = .98$$

We define individual air lead exposure as the daily average air lead concentration each individual is exposed to. As can be seen Table 1 a 10% I/O (as can be found in homes with air conditioner-Stock et al., 1983) with an outdoor lead level of 3 $\mu g/m^3$ leads to similar individual exposures in children that an 80% I/O (as can be found with good ventilation in the summer - this study) does at 1 $\mu g/m^3$ ambient lead.

Consumption of leaded gasoline (measured using sales statistics of gasoline having different lead concentrations) was the principal method used by Billick et al., (1979) and more recently the series of publications resulting from analysis of the NHANES data (National Health and Nutrition Examination Survey) (Annest et al., 1982 and 1983; Pirkle, 1983; Schwartz, 1983). These studies indicate a very close correlation between changes in gasoline lead consumption and blood lead levels. This method of estimating lead exposure produced satisfactory results in these studies because of the extremely large numbers of individuals measured (e.g. 27 801 in Annest et al., 1983) However, it was impossible to calculate a blood to air ratio or to set air lead standards from the findings.

In one study of adult men (Azar et al., 1975) individual ambient lead exposure was measured with portable samplers. This study has served as a principal reference in discussions of the blood to air lead ratio.

Although the diary method of exposure has been used in epidemiological studies of other compounds (discussed in Moschandreas, 1981; Duan, 1982) it has not been used in lead studies.

Portable air samplers although very effective for measuring ambient exposure in the workplace can interfere with people's normal way of life since they make a noise, and are cumbersome. It is also uncertain whether people in reality wear monitors at all times or whether they modify their normal activities on account of them. It is especially doubtful how effective they are in measuring children's exposure.

The diary method used in conjunction with air measurements removes these problems. It is much easier for individuals to note how much time they spend indoors or outdoors and what their activities are, than to wear a portable monitor. Because of lead's comparatively long half-life in the blood (16 to 18 days - Chamberlain et al., 1978) a generalized activity pattern can be as effective as a detailed pattern in estimating exposure to air lead.

Therefore, this study has been designed differently from previous studies in several ways:

a) Air lead exposure was estimated for each individual by combining both measured ambient air lead levels (both outdoor and indoor) and patterns of activity from self-administered questionnaires.

b) This study did not focus on high exposure as is found around smelters. The two towns chosen for the study differed primarily in amount and concentration of traffic.

c) The populations of the two towns were relatively homogeneous in size, race and socio-economic status. Norwegians in general differ little in lifestyle and eating habits.

d) The study took into account such confounding factors as age, sex, socio-economic status, smoking habits, exposure to passive smoking, exposure to lead contaminated hobbies, (e.g. shooting, ceramic painting etc.) and occupational exposure.

2 MATERIALS AND METHODS

2.1 Overview

This study combined information from three main sources (summarized in Figure 3):

1) self-administered questionnaires
2) measurements of ambient air lead (both indoor and outdoor)
3) blood measurements

Combining these three sources of information enabled estimating individual air lead exposure, removing confounding factors and studying the correlation of blood lead to air lead concentrations.

2.2 Population characteristics

A total of 303 individuals volunteered for the study, 178 from the moderate lead exposure town Holmestrand and 125 from the low lead exposure town Sørumsand. The criteria used in selecting study subjects was: 1) Sørumsand - that they resided in two main living areas, near where outdoor monitors were stationed; 2) Holmestrand - that they lived or worked near the main highway. In both towns efforts were made to have as many children as possible. Table 2 compares various population characteristics of the inhabitants of the two towns.

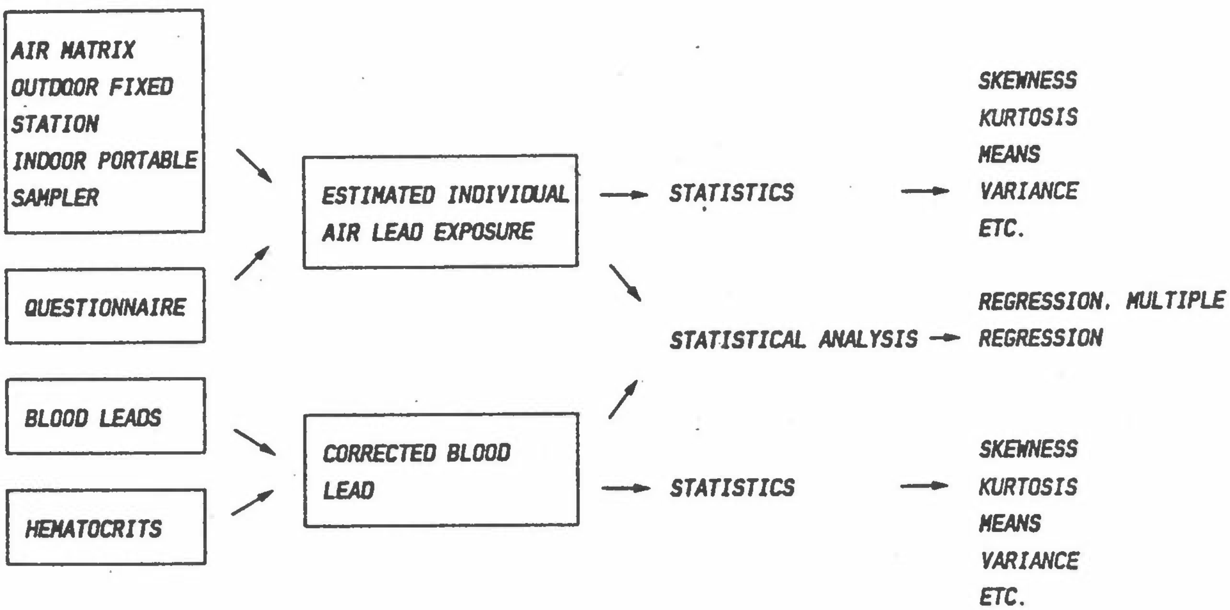

Figure 3: Schematic overview of the methodology used in this investigation.

Table 2: Population characteristics of the two towns where blood and air lead concentrations were measured - Holmestrand (moderate air lead levels) and Sørumsand (low air lead levels).

	Holmestrand		Sørumsand	
Sample size	178		125	
Age range	3 - 91 years		3 - 90 years	
Numbers of:	male	female	male	female
Children (2-9 yrs)	2	4	8	7
Children (10-15)	10	11	7	6
Adults (16-67)	41	69	30	57
Pensionists (>67)	16	28	5	6
Socio-economic factors	(Percentage of population)			
Social Class A*	15%		50%	
" " B	25%		16%	
" " C	19%		21%	
" " D	11%		4%	
" " E	0%		0%	
those on public assistance F	30%		9%	
Elderly in home for elderly G	16%		5%	

* Definition of social class divisions is given in Appendix 2.

2.3 Estimation of Air Lead Exposure

The estimate of individual air lead exposure was obtained by combining information from 3 different sources: outdoor fixed site measurements and portable indoor measurements were used to create a matrix of ambient lead concentrations. This matrix was combined with questions pertaining to time spent in each of the microenvironments (e.g. indoor home, indoor school, outdoor school) to create the individual ambient lead exposure estimate. This estimate was equal to an average (calculated over 21 days) air lead concentration (in $\mu g/m^3$) that each individual was exposed to. Figure 4 gives an idealized overview of the factors influencing individual air pollution exposure.

2.3.1 Fixed outdoor stations

A total of four fixed low volume samplers were placed, two in each town (Figure 2). Each intake was situated at a height of 2 meters. Twenty-four hour samples were collected for a minimum of thirty days at each site. The sites in Holmestrand were chosen: 1) to the north of the crosslight because of proximity to an old people's home where thirty of the participants of the study lived, and 2) to the south of the crosslight near a school that was attended by nearly all the children in the area. In Sørumsand the sites were placed in areas where most of the volunteers lived.

Particulate bound lead collected on the filters was measured at the Norwegian Institute of Air Research by electrothermal atomic absorption spectroscopy (EAAS) after extraction of the lead from the filters with 1:1 nitric acid. Analyses were made by a Perkin-Elmer 2380 atomic absorption spectrophotometer equipped with a graphite furnace 400, an AS-1 automatic sampler, a PRS-10 printer, a Model 56 recorder, a deuterium arc background corrector and a lead hollow cathode lamp. Ordinary graphite tubes were used throughout this study. A summary of the air lead method is listed in Table 3. The detection limit of the analysis is 1 μgPb/l which corresponds to 0.003 μg Pb/m^3 for the outdoor samples (10 ml extract, 3.5 m^3 of air). The precision is about 5% at the 0.2 μg Pb/m^3 level. The calibration standards used are diluted Titrisol ampoules (Merck) diluted with nitric acid to approximately the same acid concentration as in the samples.

Table 3: Summary of air lead method.

Sample preparation

To cut pieces of the filter in polyethylene centrifuge tubes is added 1:1 HNO_3 (2 ml in the case of outdoor sampler, 1 ml for indoor samples). The tubes are left in a water bath at 80 ^{0}C for 1 hour. 8 or 4 m s of distilled water is added and the tubes are shaken and centrifuged.

Instrumental parameters

Wavelength	283.3 nm
Spectral band width	0.7 nm
Lamp current	10 mA
Read time	3 sec
Signal mode	Peak height

Furnace/autosampler program

Sample volume 20 µl

	Temp ^{0}C		Ramp/hold (sec)
Dry	120		2/40
Char	500		5/30
Atomize	2300	Argon flow 20 ml/min	1/3
Clean out	2600		1/1

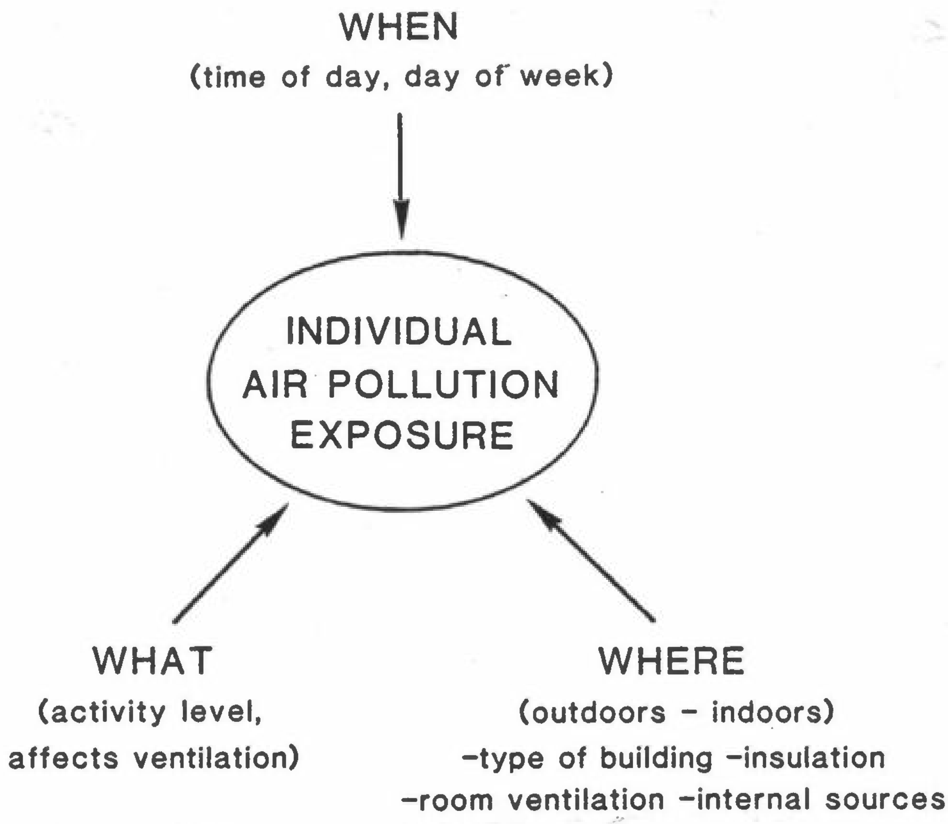

Figure 4: Individual air pollution exposure estimates should account for differences in peoples behavior patterns.

2.3.2 Indoor air samplers

Portable 8-hour samplers were distributed to shops and offices along the main highway as well as a few private individuals. Although generally 3 consecutive 8 hour samples were collected at each site (generating a full 24 hour sampling period), some measurements at some sites continued for a longer period (near the stationary sites) and others such as in shops for a shorter period, the 8 hours the shops were open. Values obtained by portable samplers were compared to those obtained by the fixed low volume samplers (in a separate study) and found to be higher. This can be partially explained by 1) slight differences in size of particles collected by the two systems, 2) deposition occurring in the tubing in the outdoor samplers connecting the intake to the filter (Vitols, 1983). Therefore the values of the indoor samples were corrected for the small difference to facilitate comparison.

The filters were analyzed for lead in the same laboratory (NILU) using the same methodology as the outdoor samples.

2.3.3 Air lead matrix

Blood lead having a 16 to 18 day half-life reflects a long accumulation period (Chamberlain et al., 1978). Thus 21 day average air lead value for each site in each town was calculated. Since in Holmestrand, traffic congestion is in fact heaviest on the weekends, one set of calculations reflected weekday averages and one set weekend values. The missing week's values in Holmestrand by the northern station were interpolated from the relationships between the two sites on the other 34 days.

The indoor-outdoor ratio (I/O) was found to be between 50-65%. This value corresponds well to the 50% I/O found in an earlier study done in Oslo in buildings of similar age using low volume samplers for both the indoor and outdoor values (S. Larssen, 1981) Therefore a value of 50% was used for calculations in Holmestrand. Values of I/O were unquestionably higher in Sørumsand (85%), possibly due to more opening of windows.

The final air lead matrix is found in Appendix 1.

Further estimates made in the drawing up of the matrix:

1) Air lead at a distance greater than 50 m from the highway was 50% of those values by the highway. This percentage was considered valid since it brought values down to very close to background (values found in Sørumsand). These were further validated by comparing indoor measurements at different geographic locations.
2) The values at the cross-light in Holmestrand were estimated from the indoor measurements.
3) A general outdoor value was calculated that was a simple mean of the two outdoor stations.
4) Values for those who lived up on the Holmestrand plateau were simply estimated to be the same values as an average of the Sørumsand concentrations.

2.3.4 Questionnaires

A series of questions in the self-administered questionnaires, aimed at enabling the estimation of exposure, asked about:

1) Location of home, school or work in the town:

   a) For Holmestrand the town was divided into 1) by the light crossing, 2) to the north, and 3) to the south of the light crossing, but less then 50 m from the highway, 4&5) north and south but more than 50 m from the highway, 6) those that lived on the Holmestrand plateau with low air lead concentrations.

   b) For Sørumsand - four general living areas were isolated. This division appeared less important since air lead levels seemed relatively uniform over the entire area.
2) Time spent indoors at home, indoors at work or school, outdoors, time spent jogging or in heavy activity and time spent traveling.

2.3.5 Individual air lead exposure estimate

The individual air lead exposure estimate was calculated as an hourly average of air lead concentrations (in $\mu g/m^3$) that individuals were exposed to. An extra factor was used if people were actively jogging to account for increased respiratory rate (X 3). Likewise, a factor (1.5) was used to account for higher activity in children when they were outdoors, since they were usually playing. (Factors found both in consultation with lung specialists and Åstrand, Rodahl,1977).

However, in the analysis phase of the study comparisons were made of the results obtained both using these extra factors and without. A comparison is given in Part II. Adding these activity factors results in increasing the range of levels of pollution exposure, thus slightly flattening the slope of the regression of blood lead to air lead.

Since we believe that these corrective factors give a more accurate estimate of air lead exposure, they are incorporated into all further analyses.

2.3.6 Measurements of dustfall and drinking water

In order to ascertain if blood lead values reflected intake from other possibly important sources such as dust in the home, playground, or from drinking water, a few extra measurements of indoor and outdoor dust and drinking water were made.

Measurements were made of outdoor dustfall using a NILU dust collector which is a long funnel shaped container holding a filter and a trap underneath to collect precipitation. It stands at a height of 1,5 m. The sampling period was 43 days in Holmestrand and 29 days in Sørumsand. Lead was analyzed both in water soluble and water insoluble dust in the same laboratory (NILU) using the same methods as for air samples.

Indoor dust samples were collected by washing a square surface (10x10 cm) in the dustiest corner of the house with a distilled water soaked filter. The filters were then stored in sterilized glass bottles. Lead was measured in the same laboratory using the same methods as described above. This indoor dust method was first described by Vostal et al., in 1974.

A 20 ml sample of drinking water was obtained in acid washed polyethylene bottles from the main faucet in the house or building. Water was allowed to run for a few minutes before sampling.

2.4 Blood measurements

2.4.1 Collection of blood samples

From each individual 3 - 10 ml whole blood was collected in green stoppered Venoject evacuated blood collection tubes (VT 100 SH - sodium heparin). Blood sampling was done in May 1983.

The blood samples were stored at 4 - 8 degrees Celcius before and during transport. The samples were mixed by inverting the tubes for 5 minutes, before 1-2 ml samples were poured into two 2 ml plastic test tubes for analysis of hemoglobin and hematocrit and separately zinc-protoporphyrin. Thereafter two drops of Triton X-100 were added to the remaining blood to measure blood lead.

2.4.2 Determination of hematocrit, hemoglobin and zinc-protoporphyrin (ZPP).

After arriving at the laboratory the day after collection, hematocrit (red blood cell volume in per cent of whole blood) was determined in duplicate using microhematocrit centrifuge (LIC HK4) at 9500 G for three minutes.

Hemoglobin was measured (by the standard cyanmethemoglobin method using photometer (Linson 3)) in all blood samples having a lower than 40% hematocrit.

Zinc-protoporphyrin was determined with a ZnP Model 4000 Hematofluorometer (Environmental Sciences Associates, Inc., U.S.A.)

The zinc-protoporphyrin values were adjusted to a standard hematocrit using 45% (by volume) of red blood cells and 14.8 g/100 ml hemoglobin as reference values.

2.4.3 Determination of lead in whole blood

Contamination is by far the most important source of error in the analysis of low blood lead levels. The contamination of lead from both collection tubes and syringes was tested by leaching with 0.2 M HNO_3 to be less than 0.01 μmol Pb/L whole blood.

Lead concentrations in whole blood were determined by electrothermal atomic absorption spectroscopy (EAAS) using a Perkin-Elmer 5000 atomic absorption spectrophotometer equipped with an AS-40 automatic sampler, a PRS-10 printer, a Model 56 recorder, a deuterium arc background corrector and a lead electrodeless discharge lamp. The summary of the whole blood lead method is listed in Table 4.

Ordinary graphite tubes were used throughout this study. The within-run precision of the method was typically 1.5 - 2-0% at the 0.4 μmol Pb/l level, and the detection limit (2x noise level) was 0.01 μmol Pb/l.

Since the majority of lead is concentrated in the erythrocytes, differences in hematocrit can influence blood lead. This would result in apparently higher blood lead levels in those individuals whose hematocrit were elevated due to other factors, e.g. smoking, and apparently false low values in anemic individuals. Therefore, blood leads were all standardized to a hematocrit of 45 (Mc Intire, Angle, 1972) using the formula:

$$\frac{(\text{Pb-B}) \times 45}{\text{actual hematocrit}}.$$

For ease of comprehension, values have been converted from μmoles/l to μg/100 ml (dl) using the formula Pb-B μg/dl = Pb-B(μmoles/l) * 20.72.

2.4.4 Quality control programs

The accuracy of the blood-lead method is confirmed twice a year through interlaboratory survey programs organized by the Swedish National Board of Occupational Safety and Health. The results of the performance of the present method from the three last years are plotted in Figure 5. Day to day variation of the method is monitored through an intra-quality control program. Vials of frozen blood-bank samples are analyzed regularly with a variation of typically ± 7%.

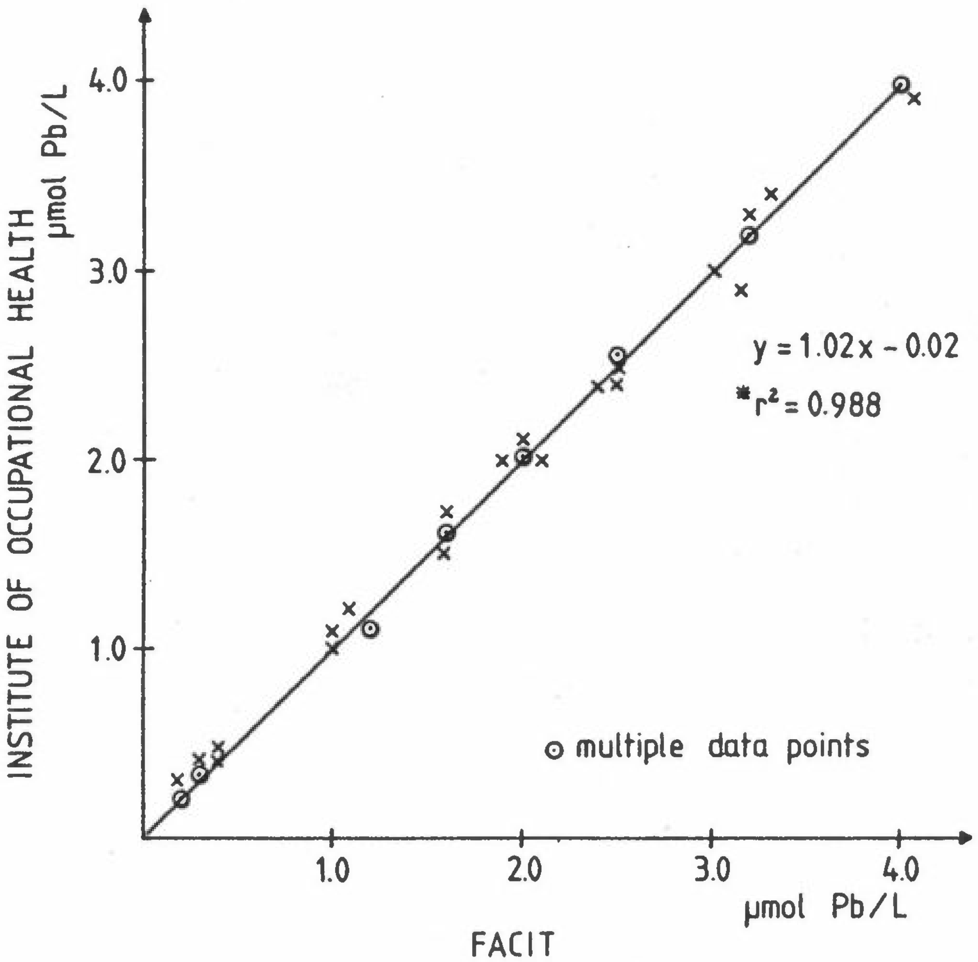

Figure 5: Results from interlaboratory surveys of lead analysis in whole blood (1980 - 1983). The linear regression is based on 47 data pairs.
r^2 = coefficient of determination.

Table 4: Summary of whole blood lead method

Sample Preparation			
Dilute whole blood 1:4 with matrix modifier* into the sampler cup. Use the method of standard addition.			
* 0.2% Triton X-100 and 0.5% $(NH_4)_2HPO_4$.			
Instrumental Parameters			
Wavelength			283.3 nm
Spectral Band Width			0.7 nm
Electrodeless Discharge Lamp			6 W
Background Corrector			On
Read Time			5 sec.
Signal Mode			Peak height
Average			2 or 3
Furnace/Autosampler Program			
Sample volume 10 µl, ordinary graphite tubes.			
	Temp. ^{0}C		Ramp/Hold sec.
Dry	120		5/15
Char	180		5/5
	230		5/2
	400		5/5
	800	Baseline 12	5/20
Atomize	2400		1/6
		Recorder	-5
		Read	-1
		Int argon flow	50 mL/min
Clean out	2700		1/2

2.5 Control for additional confounding factors

The self-administered questionnaire provided information on smoking habits, exposure to passive smoking, and exposure to lead through hobbies and occupation. In addition such information as sleeping with window open was revealed.

The smoking information was detailed, and covered number of cigarettes smoked and/or grams of tobacco for pipes and/or cigars. In addition, information was obtained about previous smoking history, time elapsed since quitting and whether or not the individual was still an occasional smoker. Children were asked if they smoked. All children, non-smokers, former smokers and occasional smokers were asked whether or not they were exposed to passive smoking and for how many hours per day. A review of the definitions inherent in each smoking category is given in Table 5.

Occupational exposure to lead covered both current and previous exposure.

Children's information was verified by comparing their information to information provided by the parents.

All individuals were classified into social category by occupation; for housewives by occupation of spouse; for children by occupation of male parents followed by female parent. The classification system used (Skrede, 1971) divides occupation into five classes (see Appendix 2).

2.6 Data analysis

Hematocrit standardized blood lead (CPbB) and estimated air lead exposure (PbA) along with the measured social and biological parameters were analyzed using conventional statistical packages (Jakobsen, 1982). Analyses included tests for skewness, kurtosis, simple regressions and multiple regression.

Table 5: Definition of subgroups used in data analysis.

1) CHILDREN 3 - 15 YRS A) NOT EXPOSED TO PASSIVE SMOKING B) EXPOSED TO PASSIVE SMOKING 2) WOMEN 16 - 90 YRS A) NON-SMOKERS - Have never smoked - Are not exposed to passive smoking - Do not occasionally smoke B) FORMER SMOKERS - Former smokers who quit 3 months ago or more C) SMOKERS - Persons who smoke more than 1 cigarette/day - Persons who have quit smoking for less than 3 months 3) MEN 16 - 90 YRS A) NON AND LIGHT SMOKERS - Catagories A and B above (Women) B) SMOKERS - Same as for Women

3 RESULTS

3.1 Individual Air Lead Exposure

3.1.1 Lead in air in Holmestrand and Sørumsand.

Figure 6 shows values of air lead ($\mu g/m^3$) at the two stations in Holmestrand and the one in Sørumsand for comparison. Measurements were continued after the opening of the tunnel to show the impact of vehicular traffic on the air lead levels in Holmestrand. Levels in Sørumsand can be considered as background. Air lead levels in Holmestrand are from 3 to 6 times as high as Sørumsand. This observation is backed up by dustfall measurements shown in Table 6, where lead in dust is 3 to 4 times higher in Holmestrand than Sørumsand.

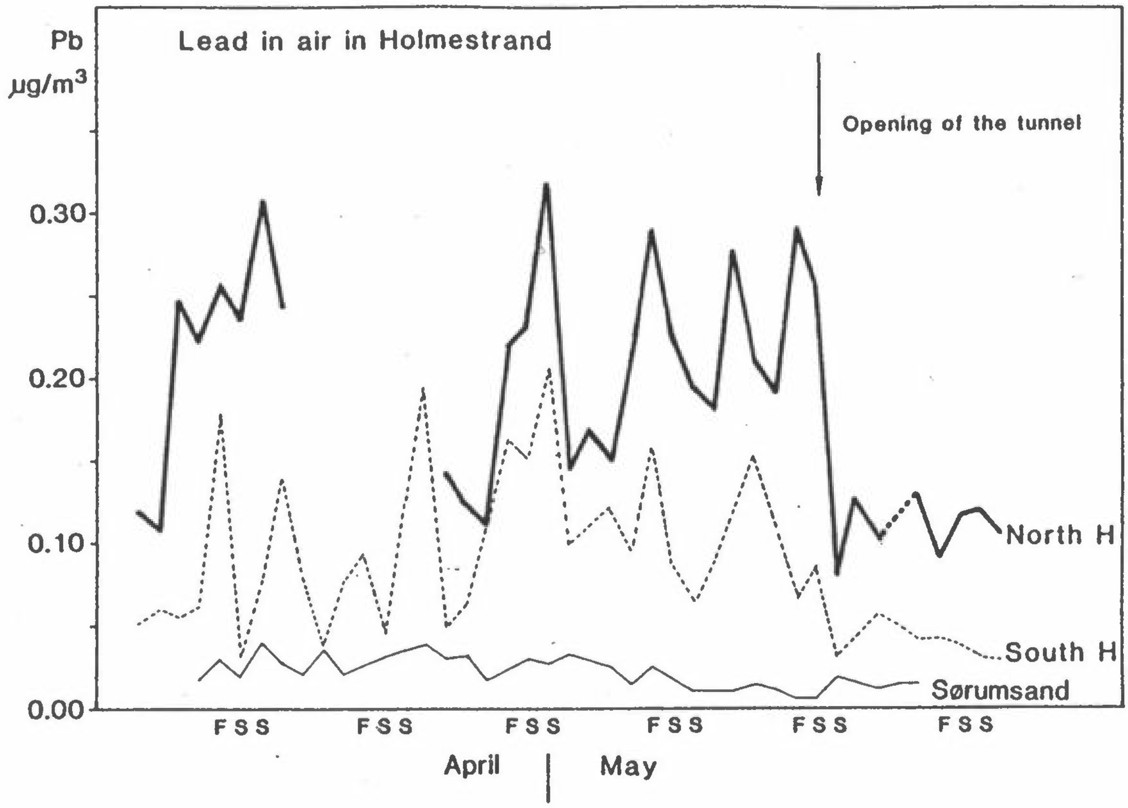

Figure 6: Concentrations of lead in air at two locations in Holmestrand and one in Sørumsand.

Table 6: Lead levels in outdoor dustfall in Holmestrand and Sørumsand during the study period - two control low exposures sites also given.

Site	Water insoluble $\mu g/m^2/day$	Water soluble $\mu g/m^2/day$	Total $\mu g/m^2/day$
Holmestrand	44,1	72,2	116,3
Sørumsand	10,4	20,8	31,2
Birkenes		49,9	
Kise (Hedmark)		18,9	

3.1.2 Indoor lead levels

In Table 7 (site placement is indicated in Figure 7) indoor air lead levels were standardized such that values measured on different days could be compared with each other. Values of the north station are roughly twice as high as those in the south (similar to ambient lead levels) and at the light crossing they are four times as high as the south station. It is obviously necessary to detail air lead concentrations in a geographic area and populations movements within that area to get a correct impression of actual exposure. As also shown in Table 7, dust in homes was very little correlated to air lead levels.

Table 7: Comparison of lead levels, air (µg/m^3) and dust (µg/m^2) in Holmestrand.

Station number	Standardized indoor air µg/m^3)*[1]	Dust µg/m^2
3*[2]	2.58	700
6	0.44	26
8	0.45	25
5	0.16	136
12	0.09	46
11	0.01	11
4	0.09	52
2	0.16	60

*[1] These values represent averages of standardized values. Standardized values were computed by comparing the same day outdoor value to a standard mean and correcting indoor air for this difference.

*[2] These station numbers refer to these numbered in Figure 7. Station 3 is a gas station. All others are shops, schools or homes.

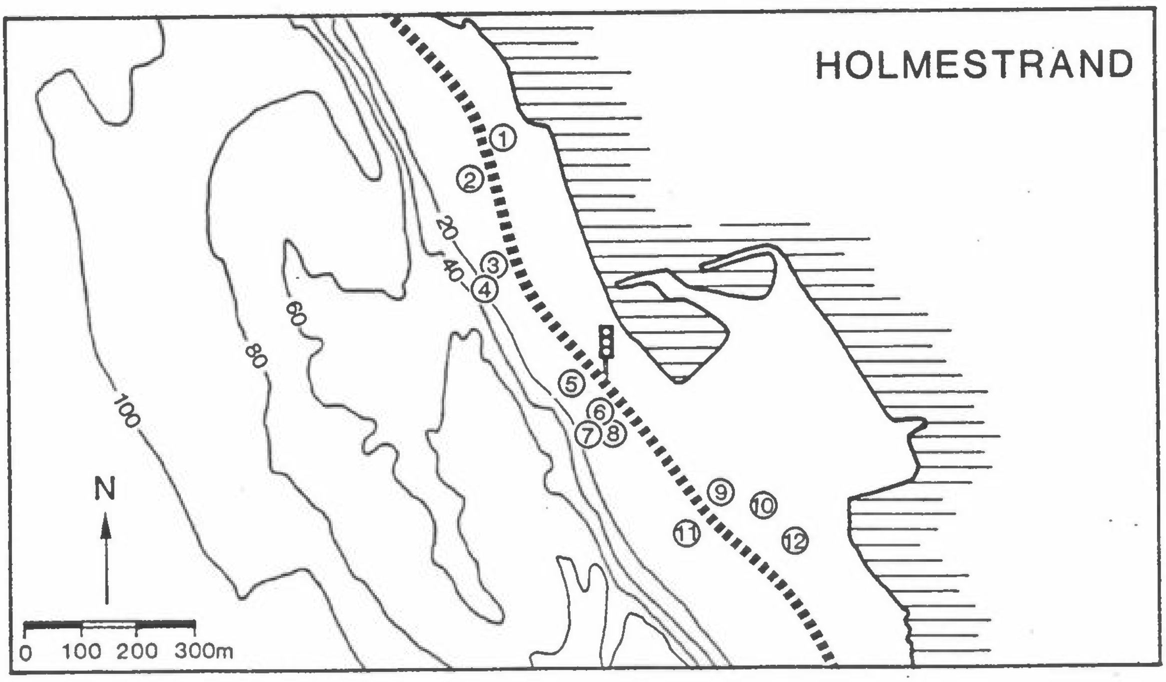

Figure 7: Location of indoor sites in Holmestrand referred to in Table 7.

3.1.3 Estimation of personal exposure

When comparing blood lead levels between population subgroups it is necessary to take into account the possibility that differences have in part to do with differences in exposure to ambient lead. The frequency distribution of the estimated individual air lead exposure is shown in Figure 8 for two population groups, children and pensionists. Geographic differences in air lead level, and individual differences in peoples' behavior patterns leads to a range in air lead exposure between the two towns whose air leads differed by a factor of at least 3 to 4.

Not only does exposure to air lead in pensionists differ substantially from those in children, but individuals from the two towns overlap. This is good evidence for the necessity of estimating individual exposure. People differed in the amount of time spent outdoors, especially important in housewives, children and pensionists. As documented in the questionnaires, people travelled on the weekends, some to low exposure areas (e.g. in the mountains for those living in Holmestrand) or to high exposure areas (e.g. Oslo for those living in Sørumsand), However, since at least 75% of time is spent indoors at home, one can say that it is the pollution level at the place of residence that is of primary importance for determining blood lead levels. Differences in individual exposure must be accounted for in examining blood lead concentration.

3.2 Blood lead levels

3.2.1 Preliminary data handling

In the main body of this report we present the findings observed based on the following data correction factors. However, alternate findings are briefly described and presented in Part II. Our correction factors were:

1) Because of concentration of lead in the erythrocyte levels of lead measured in whole blood were corrected for hematocrit (CPbB).
2) The estimate for individual exposure to air lead included a factor to account for increased respiration during high activity.
3) Both air and blood lead levels were found to be log normally distributed, and the analyses of statistical significance were run on the logarithmic values. The frequency distributions of the data can be found in Appendix 4, part II.

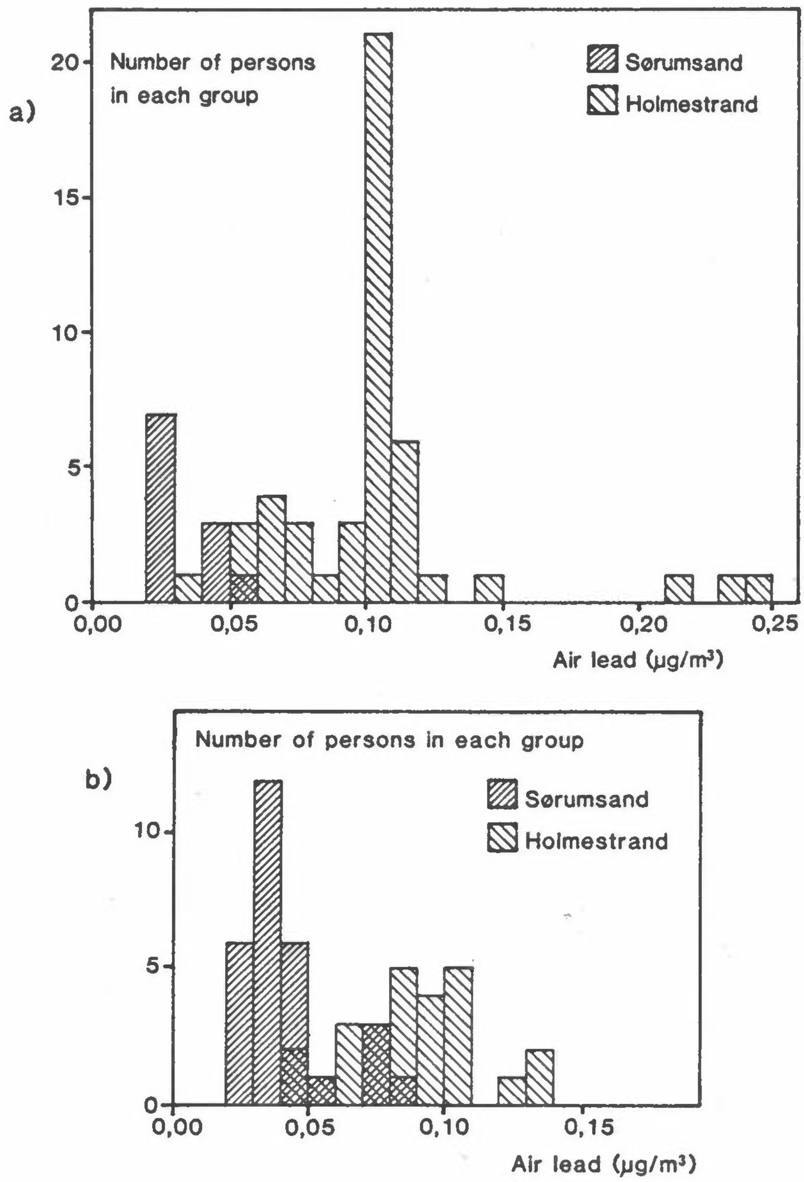

Figure 8: Frequency of exposure to different air leads in two population groups in the two towns, Holmestrand and Sørumsand.
a) Pensionists, b) Children.

As can be seen in Part II, none of the preceeding changes altered the principal findings in any way. They have been chosen to increase the accuracy of the resultant calculated coefficients.

3.2.2 The correlation of blood to air lead concentrations

Certain parameters were eliminated from further analysis either because of insufficient sample size or because of lack of significant differences. Tables of means and standard deviations of some of these parameters can be found in Part II. The variables retained for further analysis were: age, sex, smoking habits, social class and air lead exposure (PbA).

As a result of initial analyses the data were subdivided into two subgroups, children and adults (pensionists were not found to be significantly different from younger adults). A multiple regression (Tables 8 and 9) of each of these groups resulted in the elimination of social class (as defined in appendix II) and age as significant variables, and the retention of smoking and sex.

The data were subdivided into 8 groups: 1)children - unexposed to passive smoking, 2) children - exposed to passive smoking, 3) women - non smokers, 4) women - exposed to passive smoking, 5) women - former smokers, 6) women - smokers, 7) men - non-smokers and former smokers, and 8) men - smokers, categories defined in Table 4).

In a first step, means and standard deviations were compared for each town, Holmestrand (higher air lead) and Sørumsand (control) for each of the above named groups. The results are presented in Table 10. They indicate higher blood lead levels in Holmestrand for each population group. Means and standard deviations have been used here despite the fact that the data was log normally distributed so as to present the data in a clear and comprehensible manner. These values were not used to test for statistical significance. It is more correct to use the median plus possibly the standard deviation of the logarithm. However, it is doubtful that this is very comprehensible to most people. Medians are also given in Table 10 for comparison. As can be seen in Table 10 the differences between mean and median were quite small and did not significantly change the findings.

Table 8: Effects of sex, social class, exposure to passive and active smoking and log air lead on log blood lead in children and adults.

MULTIPLE REGRESSION ANALYSIS
EFFECTS OF SEX, SOCIAL CLASS, SMOKING HABITS AND LOG AIR LEAD

ON LOG BLOOD LEAD (µg/dl)

ADULTS

VARIABLES IN EQUATION (CONSTANT- 3.4937 I VARIABLES NOT IN EQUATION :

ID	B - COEFFICIENT	STD.ERROR	F TO REMOVE	P-VALUES FOR B	STANDARDIZED B (R.PART)	BETA 95% CONF.INT. UPPER	LOWER	I	ID	PARTIAL CORR.	TOLERANCE	F TO ENTER
137	0.401	0.051	60.692	0.000	0.4875	0.5028	0.2995	I	SOC.CLS	0.0596	0.9398	0.5784
129	0.036	0.012	8.618	0.004	0.1835	0.0605	0.0118					
90	-0.317	0.070	20.313	0.000	-0.2820	-0.1781	-0.4558					

SUMMARY TABLE :

STEP NR.	MULT.R	MULT.RSQ	INCREASE IN RSQ	RESIDUAL EFFECT	F-VALUE FOR E/I	VAR. NR ENTER	REMOVED	VAR. NAME
1	0.4980	0.2480	0.2480	0.8672	54.427	137		LOG AIR LEAD
2	0.5743	0.3298	0.0817	0.8187	19.997	90		SEX
3	0.6028	0.3634	0.0337	0.7979	8.618	129		SMOKING

MULTIPLE REGRESSION ANALYSIS

EFFECTS OF SEX, SOCIAL CLASS, EXPOSURE TO PASSIVE SMOKING AND THE LOG OF AIR LEAD

ON LOG BLOOD LEAD (µg/dl)

CHILDREN

VARIABLES IN EQUATION : (CONSTANT= 3.1737) I VARIABLES NOT IN EQUATION :

ID	B - COEFFICIENT	STD.ERROR	F TO REMOVE	P-VALUES FOR B	STANDARDIZED B (R.PART)	BETA 95% CONF.INT. UPPER	LOWER	I	ID	PARTIAL CORR.	TOLERANCE	F TO ENTER
137	0.500	0.085	34.556	0.000	0.5165	0.6706	0.3288	I	SOC.CLS	0.1577	0.6609	1.1987
114	0.567	0.097	33.856	0.000	0.5112	0.7623	0.3705	I	SEX	-0.1558	0.9770	1.1697

SUMMARY TABLE :

STEP NR.	MULT.R	MULT.RSQ	INCREASE IN RSQ	RESIDUAL EFFECT	F-VALUE FOR E/I	VAR. NR ENTER	REMOVERD	VAR. NAME
1	0.6333	0.4011	0.4011	0.7739	32.813	137		LOG AIR LEAD
2	0.8055	0.6488	0.2477	0.5926	33.856	114		PASSIVE SMOKING

Table 9: Effects of age, sex, passive and active smoking and log air lead on log blood lead in children and adults.

MULTIPLE REGRESSION ANALYSIS
ADULTS

EFFECTS OF AGE, SEX, AIR LEAD AND SMOKING

	REGRESSION	RESIDUAL
DEGREES OF FREEDOM:	3	224
SUM OF SQUARES :	17.3	39.7
MEAN SQUARE :	5.8	0.2
F-RATIO :	32.5	PROB= 0.000

VARIABLES IN EQUATION : (CONSTANT= 3.2215) I VARIABLES NOT IN EQUATION :

ID	B - COEFFICIENT	STD.ERROR	F TO REMOVE	P-VALUES FOR B	STANDARDIZED B (R.PART)	BETA 95% CONF.INT. UPPER	LOWER	I	ID	PARTIAL CORR.	TOLERANCE	F TO ENTER
137	0.342	0.043	62.256	0.000	0.4419	0.4280	0.2569	I	AGE	-0.0726	0.8552	1.1813
129	0.043	0.012	12.832	0.000	0.2011	0.0660	0.0192					
90	-0.292	0.060	23.866	0.000	-0.2731	-0.1745	-0.4105					

SUMMARY TABLE :

STEP NR.	MULT.R	MULT.RSQ	INCREASE IN RSQ	RESIDUAL EFFECT	F-VALUE FOR E/I	VAR. NR ENTER	REMOVED	VAR. NAME
1	0.4259	0.1814	0.1814	0.9048	50.086	137		LOG AIR LEAD INDEX
2	0.5134	0.2636	0.0822	0.8581	25.112	90		SEX
3	0.5509	0.3035	0.0399	0.8346	12.832	129		SMOKING

MULTIPLE REGRESSION ANALYSIS
CHILDREN

EFFECTS OF SEX, AGE, AIR LEAD AND PASSIVE SMOKING

	REGRESSION	RESIDUAL
DEGREES OF FREEDOM:	2	48
SUM OF SQUARES :	9.0	4.9
MEAN SQUARE :	4.5	0.1
F-RATIO :	44.3	PROB= 0.000

VARIABLES IN EQUATION : (CONSTANT= 3.1737) I VARIABLES NOT IN EQUATION :

ID	B - COEFFICIENT	STD.ERROR	F TO REMOVE	P-VALUES FOR B	STANDARDIZED B (R.PART)	BETA 95% CONF.INT. UPPER	LOWER	I	ID	PARTIAL CORR.	TOLERANCE	F TO ENTER
137	0.500	0.085	34.556	0.000	0.5165	0.6706	0.3288	I	SEX	-0.1558	0.9770	1.1697
114	0.567	0.097	33.856	0.000	0.5112	0.7623	0.3708	I	AGE	-0.1194	0.9971	0.6800

SUMMARY TABLE :

STEP NR.	MULT.R	MULT.RSQ	INCREASE IN RSQ	RESIDUAL EFFECT	F-VALUE FOR E/I	VAR. NR ENTER	REMOVED	VAR. NAME
1	0.6333	0.4011	0.4011	0.7739	32.813	137		LOG AIR LEAD INDEX
2	0.8055	0.6488	0.2477	0.5926	33.856	114		PASSIVE SMOKING

Table 10: Blood lead levels (µg/dl) in inhabitants of Holmestrand and Sørumsand.

		CHILDREN		WOMEN						MEN					
		Passive* smoking		Smoking habits						Smoking habits					
		Not Exp	Exp	Nev	Form	Occs	1-9	10+	All smok	Nev	Form	Occs	1-9	10+	All smoke
Sørum-sand	Mean	4.83	5.88	4.45	4.49	4.09	3.21	3.53	3.43	6.59	4.95	4.90	6.54	8.59	7.57
	St.dev	1.89	2.62	1.73	1.43	1.97	1.55	1.24	1.26	1.80	2.29	1.15	1.79	1.66	1.96
	(N)	23	4	25	18	8	3	6	9	6	7	3	5	5	10
	Median	4.14	4.60	4.51	4.20	3.33	3.18	3.40	3.24	7.41	4.45	4.43	6.53	7.9	7.20
Holme-strand	Mean	7.13	13.09	6.20	7.33	5.68	10.6	8.60	9.21	8.85	8.30	-	9.01	9.14	9.11
	St.dev	1.78	3.61	2.38	4.27	1.75	7.9	3.35	5.09	3.49	2.26	-	4.03	2.39	2.76
	(N)	11	13	43	15	3	9	21	30	10	16	0	5	15	20
	Median	7.16	12.25	6.06	6.96	5.51	8.81	8.08	8.26	7.76	7.53	-	10.56	9.32	9.32

*See Table 4 for further explanation of categories.
Occupationially exposed individuals not included for analysis.

The natural logarithms of blood lead levels (log CPbB) were related to the natural logs of air lead levels (log PbA) for each of these subgroups. The results of these regressions are presented in Figure 9 (values are converted back to non-logarithmic form for clearer understanding).

The results indicate an increased blood lead level in children exposed to passive smoking as opposed to unexposed children over all air lead exposures. Blood lead levels were increased in children as compared to adults. An increased blood lead level in female smokers as compared to non-smokers was also found. Removal of the four highest values did not significantly change the results. Passive smoking in women did not result in significantly higher blood lead levels at these air lead exposures (sample size of 16). The male data are more difficult to interpret. There were very few male non-smokers. The results indicate that although blood lead was significantly and positively correlated to air lead, there were no significant differences in this relationship between the two groups, non and former smokers and smokers. However, in women, former smokers were in fact quite similar to smokers (Figure 10). It must be stressed here, that since these values are hematocrit adjusted, differences are independent of differences in hematocrit.

The curves for each group of children were significantly different from each other ($p < .05$), as were those between smoking and non-smoking women ($p < .05$). Correlation coefficients, their squares, and significance for each of the above regressions are given in Table 11. As a further verification of both these findings, examining blood leads in families (Table 12) shows higher values in children than their mothers and fathers. Men have higher values than their wives.

It is very difficult to separate smoking habits including exposure to passive smoking from social class. An attempt was made in this study using occupation (or occupation of parent or sponse) as classified in Appendix II. No classification system can be considered totally satisfactory. In this study smoking habits seemed statistically more important in determining blood lead values than social class.

3.2.3 The baseline blood lead level coming from non-air sources

In order to calculate the point corresponding to 0 air lead, it was necessary to use linear regressions (not the logarithmic transformations since they automatically begin at 0). Although the relationship is curvilinear over the range 0 - 3 $\mu g/m^3$, any short segment of the curve can be approximated by a straight line. The intercept estimates blood lead coming from other, primarily nutritional sources.

Table 11: Regression coefficients of log blood lead versus log air lead exposure and their statistical significance*

Population group	β *	constant α	R	R^2	F	P
CHILDREN						
unexposed to passive smoking	.44	2.98	.60	.36	17.90	.001
exposed to passive smoking	.63	4.12	.74	.54	17.96	.001
WOMEN						
Non-smokers	.22	2.26	.32	.10	5.68	.05
Former smokers	.47	3.08	.68	.46	17.17	.001
Smokers	.56	3.51	.62	.38	22.69	.001
MEN						
Non/light smokers	.23	2.62	.32	.10	4.19	.05
Smokers	.23	2.75	.42	.17	6.54	.05

* $\ln y = \ln\alpha + \beta \ln x$ is equal to $y = \alpha x^{\beta}$ or equivalent to the power function.

Table 12: Blood lead concentrations (µg/dl) in family members in Holmestrand.

Family unit	Husband blood-lead (µg/dl)	Smoker Y/Yes N/No	Wife blood-lead (µg/dl)	Smoker Y/N	Children Blood-lead (µg/dl)	age yrs
1	11.83	Y	4.76	N	-	
2	-	-	7.46	Y	16.55	10
					15.79	9
					13.44	12
3	7.17	Y	3.90	Y	12.27	13
4	12.10	N	5,78	N	-	
5	10.50	Y	9.10	Y	-	
6	-	N	7.36	N	11.46	16
					7.33	13
7	-	N	4.27	N	10.75	12
8	8.39	N	6.22	N	8.83	14
					6.63	13
					8.58	10
9	12.49	Y	7.15	N	-	
10	7.19	N	6.15	N	-	
11	6.78	N	8.16	N	-	
12	-	N	6.15	N	6.13	11
					7.17	12
					4.60	15
13	8.91	Y	7.23	Y	9.92	7
					11.64	5
14	9.32	Y	7.50	N	14.23	5
15	8.18	Y	7.54	Y	-	
16	3.77	N	2.44	N	4.93	10
17	7.29	N	5.78	N	-	
18	5.86	Y	2.88	N	-	
19	5.37	N	3.02	N	-	
20	5.18	N	2.28	N	-	
21	6.69	N	6.28	N	-	

Multiple regressions of blood lead vs air lead exposure and smoking exposure for: 1) children, 2) women and 3) men, resulted in an intercept of: 1) 2.5 µg/dl for children, 2) 2.9 µg/dl for adult women and 3) 6.4 µg/dl for adult men (Table 13). Table 13 also indicates that the slope at lower air lead concentrations (β_1-air lead exposure) is higher than the 1, 2 or 3 to 1 ratio predicted in the literature.

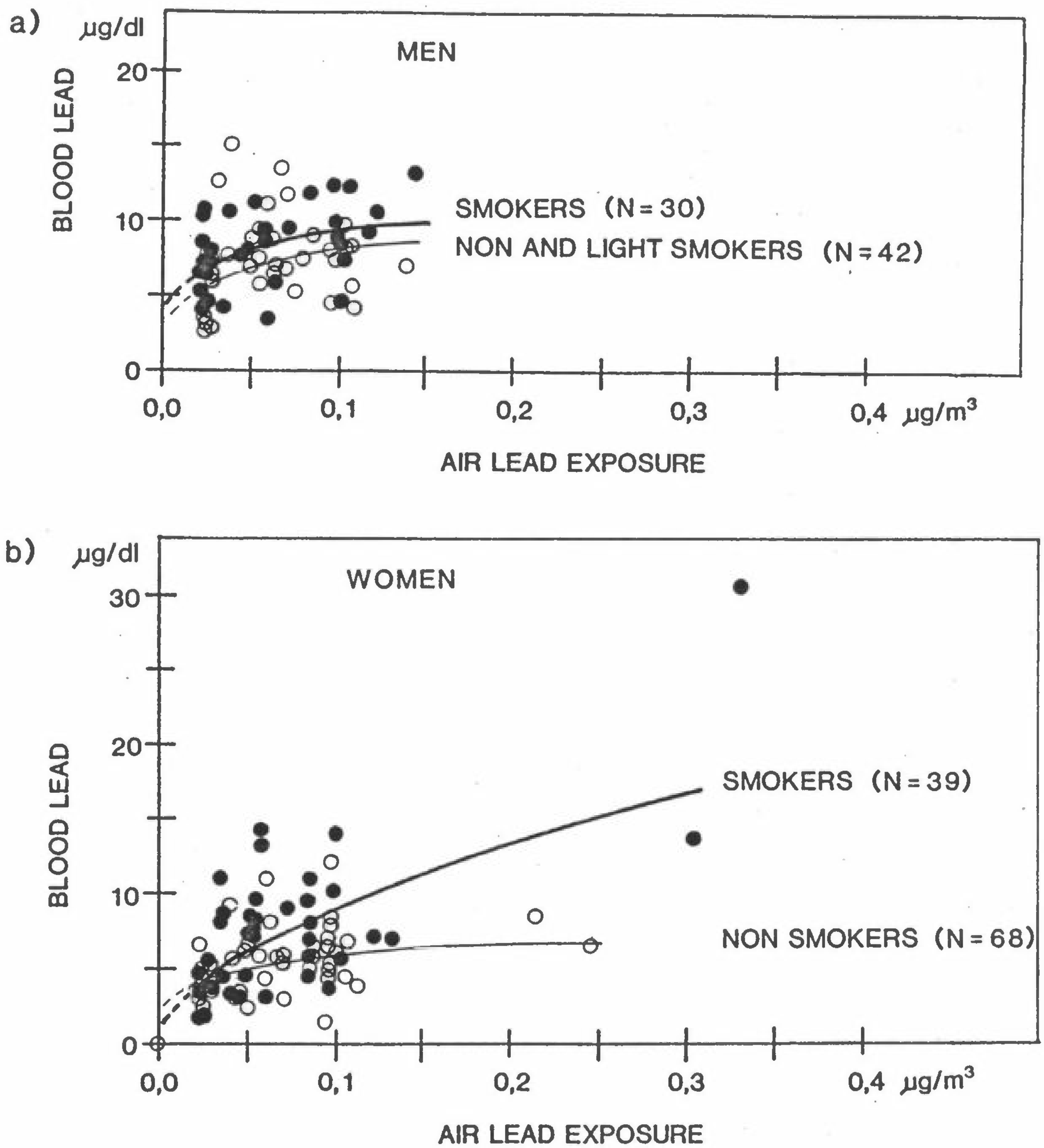

Figure 9: Blood lead levels (µg/dl) in population subgroups of the inhabitants of Sørumsand and Holmestrand as a function of individual estimated air lead exposure. ● - smokers (in men and women) and exposed to passive smoking (in children), O-non smokers and unexposed to passive smoking (in women and children), non and light smokers (in men). The data include values from both Sørumsand and Holmestrand. A log-log regression is the equivalent of the curvilinear relationship $y = \alpha x^{\beta}$ where α = intercept and β the regression coefficient. Values for α and β can be found in Table 11.

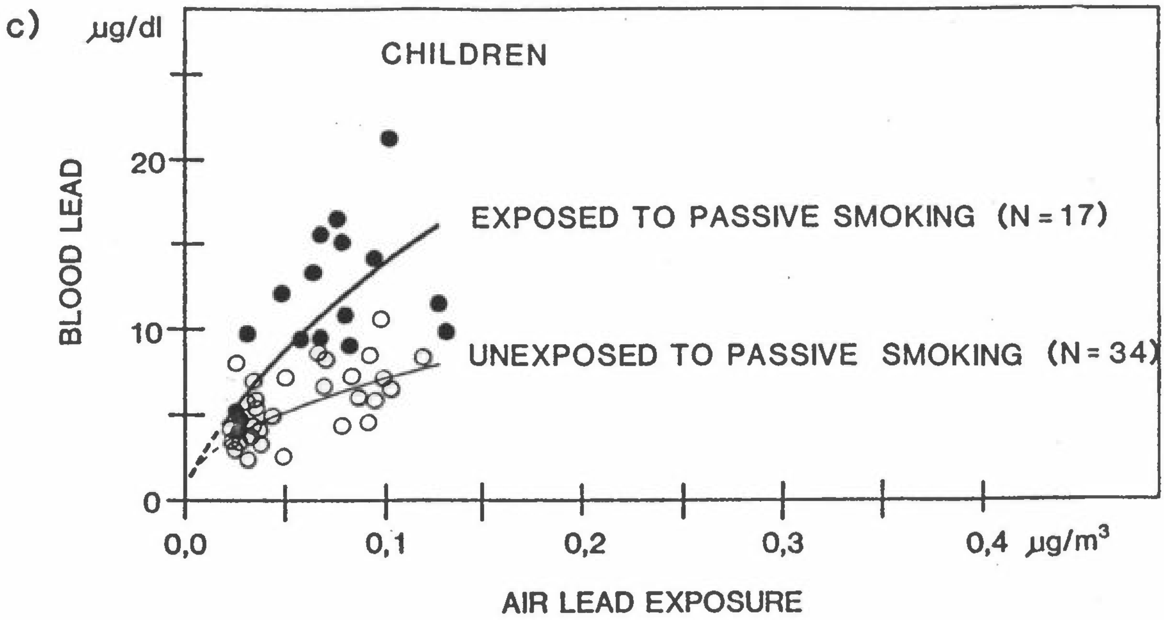

Figure 9: cont.

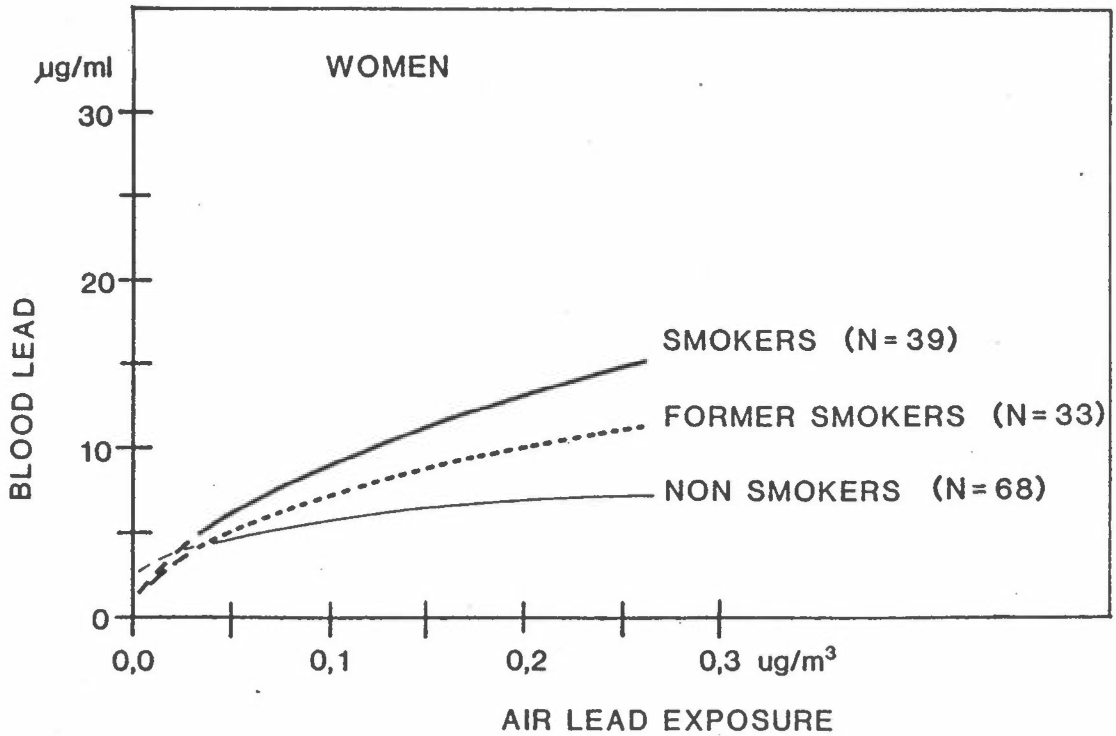

Figure 10: Calculated regression curves of blood level (µg/dl) versus air lead exposure ($\mu g/m^3$) in adult women.

Table 13: Regression coefficients for straight line regression analysis (not logarithmic transformed data) with statistical significance for data including inhabitants of Sørumsand and Holmestrand.

	α	β_1*	β_2	R	R^2	F	P
Children	2.52	56.35	4.98	.78	.61	37.2	.001
Adult Females	2.99	42.53	0.32	.62	.38	46.6	.001
Adult Males	6.49	23.53	-	.29	.08	6.4	.014

* slope β_1 = air lead exposure, β_2 = smoking or exposure to passive smoking.

The above factors correspond to $Y = \alpha + \beta_1 x_1 + \beta_2 x_2$

3.3 Hematocrit levels

The findings reviewed in this section are presented in Part II. Results of multiple regression analysis in adults revealed a well known significant difference between the sexes with females having the lower value. The other variables - social class, smoking and logarithm of blood lead produced no significant correlations.

In children, sex, social class, and logarithm of blood lead level and passive smoking did not produce a significant effect on hematocrit levels.

The inhabitants of Sørumsand had significantly lower hematocrits than those in Holmestrand ($t = 3.60$, $pH<0.001$).

3.4 Zinc protoporphyrin levels

Zinc protoporphyrin (ZPP) levels were standardized for a hematocrit of 45. Results of analyses are presented in Part II, Appendix VI.

The inhabitants of Sørumsand had lower ZPP levels than in Holmestrand.

The multiple regression analyses of the logarithm of ZPP (ZPP is log normally distributed) resulted in:

A: Adults - a significant correlation with sex with females having a higher value; and a significant correlation with hematocrit with a decrease in log ZPP with increasing hematocrit. No significant correlations with logarithm of blood lead or smoking were found.

B: Children - a significant correlation with hematocrit (a decrease in log ZPP with increasing hematocrit) and no correlation with either passive smoking or logarithm of blood lead.

These findings are not in themselves surprising. The measured blood leads in this study are low and below any reported effect on ZPP in the literature. The inverse relationship between hematocrit and zinc protoporphyrin is well known. Iron deficiency is the most common cause of elevated ZPP in a population not exposed to lead. However, redoing the analysis in men and women, using only those individuals having hematocrits over 40, did not remove the inverse correlation with hemocrit. This negative correlation of ZPP and hematocrit in the normal range of hematocrits is not reported in the literature. The difference between the towns is more difficult to explain. However, since hematocrit levels were lower in Sørumsand, they cannot be the explanation for the lower ZPP levels in Sørumsand.

4.0 DISCUSSION

This investigation revealed several important findings:

1) In certain population groups, lead in air, at least when the lead originates from gasoline combustion, has a greater influence on blood lead than predicted by the literature,
2) The values, measured under low exposure conditions, reveal a much lower dependance on food and other non-air sources (intercept at 0 air lead) than previously reported in the literature.

These two findings together lend strong support to the curvilinear relationship of lead intake and blood lead hypothesized in the literature (Chamberlain, 1983).

Estimating individual air lead exposure was a necessary addition to traditional methodology that allowed assessing the impact of air lead in a more quantified manner.

It is extremely important, in investigations of health effects of air pollution, to account for the differences in exposure to the pollutants themselves. Individual air pollution exposure should be estimated. These estimates serve:

1) to find and describe the most exposed subpopulation,

2) to get a better appreciation of individual's exposure routes,

3) to suggest realistic air quality standards that protect the most exposed populations.

The estimates should take into account: 1) differences in mobility pattern of individuals, where people live, work, go to school, etc., 2) differences in air pollution levels at different times of day,and different days of the week; and 3) differences in respiratory ventilation rate caused by varying exercise levels.

Although it has frequently been mentioned that children have a higher blood lead level than adults (Annest et al., 1982). the impact of air lead concentration on these levels has not previously been shown. By standardizing all blood leads to a hematocrit of 45, it is evident that these differences are not caused by the possible differences in number of red cells per unit blood between children and adults.

An additional interesting finding is the impact of passive smoking on these children's blood-air relationship. It is impossible in such a study to resolve the question of why. Several theories can be put forth, however. The most tempting is that passive smoking in children alters lung function such that a greater number of lead containing particles reaches the alveoli. It is important to recall that this study was concerned with air lead originating from the combustion of gasoline. This combustion is known to release very small particles (less than 0.3 μ) (Chamberlain et al., 1978). Particle size would be a very important determinant in such an explanation. Increased particular penetration into the alveoli with smoking has been seen in adults (Cohen et al., 1979; Westergaard, Olsen, 1973). It is known that passive smoking increases the risk for lung infections in children (Dutau et al., 1981; Lefcoe et al., 1983). However, one cannot rule out other theories including decreased excretion of lead (possible renal alterations) or reduced transfer of lead from blood to bone.

This study, does however, reject certain hypotheses. The regression curves do indicate that the inhalation of more lead because of tobacco smoke's lead content is not the explanation. The curves in children exposed to passive smoking would not have had a significantly different shape, but simply be parallel to the curves for non-exposed children. From the indoor measurements in smoking homes, it was evident that the increase in lead content because of smoking was not much higher than .03 - .04 $\mu g/m^3$. Although it can be said that children exposed to passive smoking generally belong to a different social class, this investigation revealed that when both passive smoking and social class were entered in multiple regressions, passive smoking was highly significant and social class was not. However, it is extremely difficult to define social class and any classification system can be considered imperfect.

Women who did not smoke had very low blood lead values (lower than men). Such a difference has previously been reported in the literature (Annest et al., 1982). However, when women smoked, air lead had a greater impact on their blood lead levels than in men. Again having blood lead values standardized to a hematocrit of 45 removed an explanation due to differences in cell numbers, both between men and women, and between women non-smokers and smokers. The statistically significant difference in shape of the curves between women smokers and non-smokers rules out a mere increased lead intake due to tobacco smoke's lead content. Explanation can only be theoretical in this study, and is similar to that given for children exposed to passive smoking. Higher lead levels in smoking women has previously been reported (Lubin et al., 1975).

The values found in men are more difficult to interpret. First, there were very few men who had never smoked. Therefore those who quit were combined with non-smokers for analysis.

However, as seen in women, former smokers in fact resemble smokers more than they do non-smokers. In addition it is known that other factors can influence blood lead levels, for example alcohol consumption (Elinder et al., 1983). Men tend to deal more with cars and boats, and are possibly more in contact with other lead sources than women. Therefore the variation in men seems to be higher than in women and children making the findings difficult to interpret.

The findings of this investigation, give preliminary support to a curvilinear relationship between blood and air lead, such that air's contribution to blood lead is much higher under lower air lead concentrations. This is especially so in children, even more so in those exposed to passive smoking, and in female smokers. The low air exposures found in Scandinavia have enabled examining the non-air contribution more closely. It can now be seen to be much lower than previously reported by the linear extrapolations used in the literature.

The findings of this study as would be expected did not indicate any measurable correlation between blood lead and zinc protoporphyrin either in children or adults at low to moderate ambient lead exposure.

Finally, these findings give an indication that the calculated air quality standards for air lead, based on linear extrapolation of adult male blood levels will not protect all population subgroups. Those most susceptible population subgroups are: children, both exposed and unexposed to passive smoking, and adult female smokers.

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APPENDIX I

Air Lead Matrix Used For Each Town.

Measured and estimated air-lead concentrations ($\mu g/m^3$) for a 21 day period in Holmestrand used in calculating individual air lead exposure.

	Mondays through Thursdays		Weekends	
	Outdoor	Indoor (50%)	Outdoor	Indoor (50%)
Up on the plateau	.030	.015	.030	.015
North >50 m from highway	.082	.041	.131	.065
South >50 m from highway	.042	.021	.060	.030
North <50 m from highway	.163	.082	.263	.132
South <50 m from highway	.085	.042	.119	.060
Light crossing	.587	.298	.947	.474
General under mtn	.100	.050	.150	.075

Measured and estimated air lead concentration ($\mu g/m^3$) for a 21 day period in Sørumsand used in calculating individual air lead exposure.

	Mondays through Thursdays		Weekends	
	Outdoor	Indoor (85%)	Outdoor	Indoor (85%)
around Sennerud feltet	.024	.020	.026	.022
Fossum/ Fynsfeltet Sentrum	.030	.026	.031	.026
Valls feltet	.030	.026	.031	.026
Center and west	.030	.026	.031	.026
Sørum kommune	.020	.017	.02	.017
General outdoor	.027	.023	.027	.023

APPENDIX II

Miscellaneous general information used in this report

1. Classification system for Social Class.
2. Eating habits of Norwegians

V e d l e g g 2
Appendix 2

SOSIALGRUPPER
SOCIAL CLASSES

	Yrkesområde i Standard for yrkesgruppering i offentlig norsk statistikk *Occupation by Standard of Occupational Classifications*
Sosialgruppe A *Social class A*	00 Teknisk arbeid, 01 Kjemiker- og fysikerarbeid, 02 Biologisk arbeid, 03 Medisinsk arbeid, 05 Annet syke- og helsevernsarbeid, 06 Pedagogisk arbeid, 07 Religiøst arbeid, 08 Juridisk arbeid, 0X Annet arbeid innen teknisk, vitenskapelig, humanistisk og kunstnerisk arbeid, 10 Offentlig administrasjons- og forvaltningsarbeid, 11 Bedrifts- og organisasjonsledelse, 31 Salg av fast eiendom, tjenester, verdipapirer, forsikringer, brukte ting m.m.
Sosialgruppe B	09 Kunstnerisk og litterært arbeid, 20 Bokførings- og kassearbeid, 21 Stenografi- og maskinskrivingsarbeid, 29 Annet kontorarbeid, 30 Grossister og detaljister, 32 Handelsreisende- og agenturarbeid, 33 Handelsarbeid fra kontor, og detaljhandelsarbeid, 60 Skipsbefalarbeid, 62 Lufttrafikkarbeid, 63 Lokomotivførerarbeid, 65 Konduktørarbeid, 66 Trafikkledelse, 67 Post- og telekommunikasjonsarbeid, 74 Finmekanisk arbeid, 90 Sivilt overvåkings- og tryggingsarbeid, 96 Sport og idrett, 97 Fotografarbeid, 98 Begravelsesservice, X1 Militært arbeid
" C	04 Sykepleie- og annet pleiearbeid, 64 Vegtrafikkarbeid, 69 Annet transport- og kommunikasjonsarbeid, 71 Tilskjærings- og sømarbeid, 72 Skotøy- og lærvarearbeid, 75 Jern- og metallvarearbeid, 76 Elektroarbeid, 77 Trearbeid, 78 Malings- og bygningstapetseringsarbeid, 80 Grafisk arbeid, 82 Næringsmiddelarbeid, 87 Maskin- og motordrift, 92 Serveringsarbeid, 93 Vaktmester- og rengjøringsarbeid, 94 Hygiene og skjønnhetspleie, 99 Annet servicearbeid
" D	42 Viltstell og jakt, 43 Fiske- og fangstarbeid, 44 Skogsarbeid, 50 Gruve- og sprengningsarbeid, 51 Brønnborings- og diamantboringsarbeid, 52 Oppredningsarbeid, 59 Annet gruve- og sprengningsarbeid, 61 Dekks- og maskinmannskapsarbeid, 68 Postalt og annet budarbeid, 70 Tekstilarbeid, 73 Smelteverk-, metallverk- og støperiarbeid, 79 Annet bygge- og anleggsarbeid, 81 Glass-, keramikk- og teglarbeid, 83 Kjemisk prosessarbeid, treforedlings- og papirarbeid, 84 Tobakkarbeid, 85 Annet tilvirkingsarbeid, 86 Pakke- og emballeringsarbeid, 88 Laste-, losse- og lagerarbeid, 89 Diversearbeid innen industri-, bygge- og anleggsarbeid, 91 Hotell- og restaurantarbeid, husarbeid, 95 Vaske-, rense- og strykearbeid
" E	40 Arbeidsledelse i jord- og skogbruk, 41 Jordbruksarbeid, dyrerøkt

From: Yrke-Dødelighet 1970-1973,

35B. 1976

YRKESOMRADER PA ENGELSK

OCCUPATIONAL CLASSIFICATION IN ENGLISH

00 Technical work
01 Chemical and physical work
02 Biological work
03 Medical work
04 Nursing care
05 Other professional health and medical work
06 Pedagogical work
07 Religious work
08 Juridical work
09 Artistic and literary work
0X Other work in major group 0

10 Public administration
11 Administration of private enterprises and organizations

20 Book-keeping and cashier work
21 Stenography and typing work
29 Other clerical work

30 Working proprietors
31 Salesmen of real estate, securities, business-services, insurance etc.
32 Commercial travellers and manufacturers' agents work
33 Sales work from offices and retail sales work

40 Management in agriculture and forestry
41 Farmwork and livestock work
42 Game supervisors and game hunters
43 Fishing, whaling and sealing work
44 Forestry work

50 Mining and quarrying work
51 Well drilling and related work
52 Mineral treating work
59 Other mining and quarrying work

60 Ship officers and pilots
61 Deck and engine-room crew work
62 Air transport work
63 Railway engine drivers and firemen
64 Road transport work
65 Conductors, dispatchers and freight assistant work
66 Traffic supervising work
67 Postal and telecommunication work
68 Postal and other messenger work
69 Other transport and communication work

70 Textile work
71 Cutting and seam work
72 Shoe and leather work
73 Smelting, metallurgical and foundry work
74 Precision mechanical work
75 Iron and metalware work
76 Electrical work
77 Wood work
78 Painting and paperhanging work
79 Construction work not elsewhere classified

80 Graphic work
81 Glass, ceramic and clay work
82 Food and beverage work
83 Chemical and related process work
84 Tobacco work
85 Other production-process work
86 Packing and wrapping work
87 Stationary engine and motor-power work
88 Longshoremen and related freighthandlers
89 Labouring work not elsewhere classified

90 Public safety and protection work
91 Hotel, restaurant and domestic work
92 Waiting work
93 Building caretaking and charwork
94 Hygienical and beauty treatment work
95 Laundering, dry-cleaning and pressing work
96 Professional athletes and sportsmen etc.
97 Photographical work
98 Funeral service
99 Other service work
X1 Military work

Tabell 7.3. **Sammensetningen av matvareforbruket for gjennomsnittshusholdningen og pr. person. 1974—76. 1979-priser** *Consumption of food for the average household and per person. 1974–76. 1979 prices*

	Verdi *Value*		Mengde *Quantity*		Mengde pr. person pr. dag *Quantity per person per day*
	Kr *Kroner*	Prosent *Per cent*	Mengde-enhet *Quantity unit*	Kg/l *Kg/litre*	Gram/ml.
Matvarer i alt *Food, total*	13 275	100,0			
00 Mjøl, gryn og bakervarer *Flour, meal and bakery products*	1 193	9,0	kg	225,3	220,3
001 Mjøl og gryn *Flour and meal*	194	1,5	"	106,4	104,1
002 Kjeks, flatbrød og knekkebrød *Crispbread, biscuits etc.*	136	1,0	"	8,9	8,7
003 Brød *Bread*	534	4,0	"	93,4	91,3
004 Kaker *Cakes*	269	2,0	"	12,5	12,2
005 Makaroni og cornflakes *Macaroni and cornflakes*	60	0,5	"	4,1	4,0
01 Kjøtt, kjøttvarer og flesk *Meat, meat products and pork*	3 685	27,8	"	127,2	124,4
011 Ferskt kjøtt og flesk *Fresh meat and pork*	1 684	12,7	"	68,9	67,4
012 Saltet, røykt og tørket kjøtt og flesk *Salted, smoked and dried meat*	522	3,9	"	11,3	11,1
013 Kjøtthermetikk *Canned meat*	150	1,1	"	5,8	5,7
014 Andre kjøtt- og fleskevarer *Other meat and pork products*	1 253	9,4	"	39,1	38,2
015 Fryst kjøtt og kjøttvarer *Frozen meat and meat products*	76	0,6	"	2,1	2,1
02 Fisk og fiskevarer *Fish and fish products*	948	7,1	"	68,0	66,5
021 Fersk fisk *Fresh fish*	276	2,1	"	28,8	28,2
022 Fryst fisk *Frozen fish*	118	0,9	"	7,3	7,1
023 Saltet, røykt og tørket fisk og skalldyr *Salted, dried and smoked fish and shellfish*	198	1,5	"	11,8	11,5
024 Middagshermetikk *Canned dinner goods*	60	0,5	"	6,7	6,6
025 Småhermetikk *Sardines etc.*	125	0,9	"	3,4	3,3
026 Andre fiskevarer *Others fish products*	171	1,3	"	10,1	9,9
03 Mjølk, fløte, ost og egg *Milk, cream, cheese and eggs*	2 165	16,3	"	545,9[1]	533,9[1]
031 Mjølk *Milk*	953	7,2	liter	464,0	453,8
032 Fløte *Cream*	294	2,2	"	15,1	14,8
034 Ost *Cheese*	492	3,7	kg	25,2	24,6
035 Egg *Eggs*	426	3,2	"	27,2	26,6
04 Spisefett og -oljer *Edible oils and fats*	461	3,5	"	57,2	55,9
041 Smør *Butter*	145	1,1	"	9,2	9,0
042 Margarin og spiseolje *Margarine and edible oils*	316	2,4	"	47,9	46,8
05 Grønnsaker, frukt og bær *Vegetables, fruits and berries*	2 226	16,8	"	269,8	263,9
051 Kål og gulrøtter *Cabbage and carrots*	225	1,7	"	48,3	47,2
052 Andre friske grønnsaker *Other fresh vegetables*	304	2,3	"	28,6	28,0
053 Epler, pærer, plommer *Apples, pears and plums*	360	2,7	"	55,2	54,0
054 Sitrusfrukter, bananer og druer *Citrus fruits, bananas and grapes*	423	3,2	"	63,8	62,4
055 Tørket frukt og nøtter *Dried fruits and nuts*	116	0,9	"	6,4	6,3
056 Bær *Berries*	183	1,4	"	17,4	17,0
057 Konserverte grønnsaker *Preserved vegetables*	240	1,8	"	15,2	14,9
058 Konserverte frukt og bær *Preserved fruits and berries*	375	2,8	"	34,8	34,0
06 Poteter og varer av poteter *Potatoes and potato products*	469	3,5	"	204,8	200,3
061 Poteter *Potatoes*	375	2,8	"	200,0	195,6
062 Varer av poteter *Potato products*	94	0,7	"	4,8	4,7
07 Sukker *Sugar*	146	1,1	"	44,3	43,3
08 Kaffe, te, kakao og kokesjokolade *Coffee, tea, cocoa and chocolate*	970	7,3	"	22,6	22,1
081 Kaffe *Coffee*	862	6,5	"	20,3	19,9
082 Te *Tea*	50	0,4	"	0,5	0,5
083 Kakao og kokesjokolade *Cocoa and chocolate*	58	0,4	"	1,8	1,8
09 Andre matvarer *Other foods*	1 012	7,6	"	27,7	27,1
091 Spisesjokolade og drops *Chocolate and sugar confectionery*	362	2,7	"	9,1	8,9
092 Iskrem *Ice-cream*	166	1,3	"	8,3	8,1
093 Annet *Other*	484	3,6	"	10,2	10,0

[1] 1 l mjølk er regnet lik 1,03 kg.

From: Sosialt Utsyn 1980-SSB

NORSK INSTITUTT FOR LUFTFORSKNING (NILU)
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3 STIKKORD (à maks. 20 anslag) Lead	Traffic pollution	Smoking	
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TITLE Blood lead - a function of vehicular emissions and smoking. Part I.

ABSTRACT (max. 300 characters, 7 lines)
Blood lead was measured in the populations of 2 towns - one exposed to moderately heavy traffic pollution and a control low exposure town. Blood leads were significantly higher in the traffic exposed town. Children exposed to passive smoking and female smokers had the highest blood lead values.

*Kategorier:	Åpen - kan bestilles fra NILU	A
	Må bestilles gjennom oppdragsgiver	B
	Kan ikke utleveres	C