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A COHORT STUDY

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SUMMARY

INTRODUCTION

In the spring of 1983, the Norwegian Institute for Air Research (NILU) in cooperation with the National Institute of Occupational Health and the local city health departments studied blood lead concentrations in the inhabitants of Holmestrand (moderate traffic pollution) and Sørumsand (control town). Despite low to moderate blood lead concentrations, the inhabitants of Holmestrand had higher concentrations of blood lead than were expected, especially among those children who are exposed to passive smoking at home, and in smoking women. Immediately after the study in 1983, a tunnel was opened that removed traffic from Holmestrand reducing concentrations of lead in air. In 1984, the same study team repeated the investigation in the same individuals to measure the possible reductions in blood lead concentrations that resulted from the reduced air pollution.

METHODOLOGY

The study was conducted at two sites:

1) Holmestrand - a town traversed by a major throughway (at the time of measurement in Phase I, 11 000 vehicles daily). The traffic is stopped by a light. Immediately after Phase I of the study a tunnel was opened that caused traffic to bypass the town, reducing the amount of traffic on the main throughway to circa 2 000 vehicles daily.

2) Sørumsand - a small town having very little traffic (at the time of measurement estimated at 3 000 cars daily) and no industrial sources of airborne lead.

The study was designed such that for each individual a specific blood lead concentration was related to an estimate of that individual's exposure to ambient lead during the two weeks immediately prior to blood sampling. The same method was used both years.

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 hematologic variables, hematocrit and zinc protoporphyrin, were also measured. The questionnaire included information on additional lead exposure via hobbies and occupation, and other socio-economic parameters such as smoking (both active and passive), alcohol consumption, and use of vitamins and iron supplements.

RESULTS

Concentrations of lead in blood declined in the measured cohort at the rate of 4 μg/dl blood lead per 1 $\mu g/m^3$ air lead. This is higher than values reported in the literature and seems to confirm findings, previously reported by the same study team, that indicate higher absorption of lead originating from traffic pollution than lead originating from smelters.

Children exposed to passive smoking and adult smokers had higher concentrations of blood lead in 1984, similarly to the results observed in 1983. These differences may reflect changes in absorption and retention of lead particles caused by active/passive smoking. The amount of the decline in blood lead concentrations, however, was not different between non-smokers and smokers.

Alcohol consumption was negatively correlated with the decrease in blood lead concentrations. After the opening of the tunnel, the decrease in blood lead concentration was greater in non-drinkers than in individuals who drank small to moderate amounts of alcohol.

Zinc protoporphyrin concentrations in blood measured in 1984 were significantly related to passive smoking in children and smoking in adults.

SAMMENDRAG

INNLEDNING

Våren 1983 gjennomførte Norsk institutt for luftforskning (NILU) i samarbeid med Statens arbeidsmiljøinstitutt, tidligere Arbeidsforskningsinstituttene (AMY), og de lokale helseråd en undersøkelse av blykonsentrasjoner i blodet hos innbyggere i Holmestrand og Sørumsand. Holmestrand ble valgt på grunn av trafikkbelastning, og Sørumsand fungerte som kontrollområde. På tross av lave til moderate konsentrasjoner av bly i blod, hadde innbyggerne i Holmestrand høyere nivåer enn ventet. Spesielt gjaldt dette røykende kvinner og barn utsatt for passiv røyking. Rett etter undersøkelsen åpnet veitunnelen som fjernet trafikken fra Holmestrand. Den samme forskningsgruppen gjentok derfor i 1984 undersøkelsen hos de samme personene målt i 1983, for å se om reduksjon av bly i luft førte til tilsvarende reduksjon av bly i blod.

METODIKK

Undersøkelsen ble utført på to steder i Sør-Norge:

1) Holmestrand - Et tettsted med moderat trafikk (ca. 11 000 kjøretøyer i døgnet før åpningen av tunnelen) og 2 000 kjøretøyer etter åpningen.

2) Sørumsand - Et tettsted med svært lite trafikk (ca. 3 000 kjøretøyer i døgnet i undersøkelsesperioden) og ingen industrielle kilder til blyutslipp.

I undersøkelsen ble blodbly-konsentrasjon hos hver enkelt deltager sammenstilt med den beregnede eksponering for bly i luft for de samme deltagere de siste to ukene før blodprøven ble avgitt. Den samme metodikk ble brukt begge årene.

Hver deltager besvarte et skjema med spørsmål om aktiviteter og aktivitetsmønstre de siste to ukene før prøvetaking av blod. Disse opplysningene sammen med en rekke målinger av blykonsentrasjoner i luft, dannet grunnlaget for beregningene av individuell eksponering for bly

i luft. Blyinnholdet i blod ble målt ved hjelp av elektrotermisk atomabsorpsjonspektroskopi. Blodparametrene hematokrit (volum røde blodceller) og sink protoporphyrin ble målt. Spørreskjemaet gav også informasjon om annen eksponering for bly gjennom arbeid, hobbyer, røyking (både aktiv og passiv) og alkoholforbruk, samt om inntak av vitaminer og jerntilskudd.

RESULTATER

Resultatene viste at nivået av bly i blod i gjennomsnitt synker med 4 µg/dl ved en reduksjon av bly i luft på 1 $\mu g/m^3$. Dette er høyere enn verdier på mellom 1 og 2 µg/dl tidligere rapportert i litteraturen. Det er imidlertid i samsvar med tidligere rapporterte resultater fra den samme forskningsgruppen og tyder på at det tas opp mer i blodet av bly fra trafikkforurensning enn av blyforurensning fra smelteverk.

Det var funnet en noe overraskende statistisk signifikant sammenheng mellom alkoholforbruk og bly i blod, slik at de som ikke drikker alkohol gikk mer ned i bly i blod enn de som har et lite eller moderat alkoholforbruk.

Barn som var eksponert for passiv røyking og voksne som røykte, hadde høyere konsentrasjoner enn andre av bly i blod både i 1983 og i 1984. Endringer i retensjon eller absorpsjon av bly på partikler av aktiv/passiv røyking er en mulig forklaring.

Det ble funnet en signifikant sammenheng mellom nivåer av sink protoporfyrin og eksponering for passiv røyking hos barn og røyking hos voksne.

FOREWORD

This report summarizes an investigation performed in 1983 and 1984 by the Norwegian Institute of Air Research (NILU) in collaboration with the National Institute of Occupational Health and City Health Departments in Holmestrand and Sørumsand. The study was partially financed by the Royal Norwegian Council for Scientific and Industrial Research and the State Pollution Control Authority.

CONTENTS

THE EFFECT OF REDUCING AIR LEAD FROM VEHICULAR SOURCES ON THE BLOOD LEAD CONCENTRATIONS IN TWO NORWEGIAN TOWNS

A COHORT STUDY

1 INTRODUCTION

There has been much discussion in the literature of the influence of inhalation of air lead on human blood lead concentrations. This relationship is of primary importance when attempting to set air quality standards and evaluate the possible influence of measures enacted to protect the population from unduly high concentrations of lead in blood.

Therefore, in 1983, a study was organized by the Norwegian Institute for Air Research in collaboration with the National Institute for Occupational Health and the local health departments of two towns, Holmestrand and Sørumsand, to investigate this relationship (Clench-Aas et al., 1984). Sørumsand was chosen as a control, low lead town, having very little through-going vehicular traffic (3 000 cars daily) and no industrial sources. Holmestrand was a town of relatively similar size and socio-economic conditions where a principal highway (at the time of the study, 11 000 vehicles daily) was stopped by a traffic light (Figure 1). The light caused traffic to back up, especially noticeable during the summer weekends because of vacation travel leaving Oslo.

Holmestrand was chosen for investigation, because a tunnel was to be opened in 1983 that would remove a substantial amount of traffic from the town. Therefore, a two phased study was planned where blood lead levels were measured in the inhabitants of the two towns just before the opening of the tunnel in May 1983 and again (phase II) one year later in May 1984. During that same time period the lead concentration in high octane gasoline was reduced from 0.40 to 0.15 g/l.

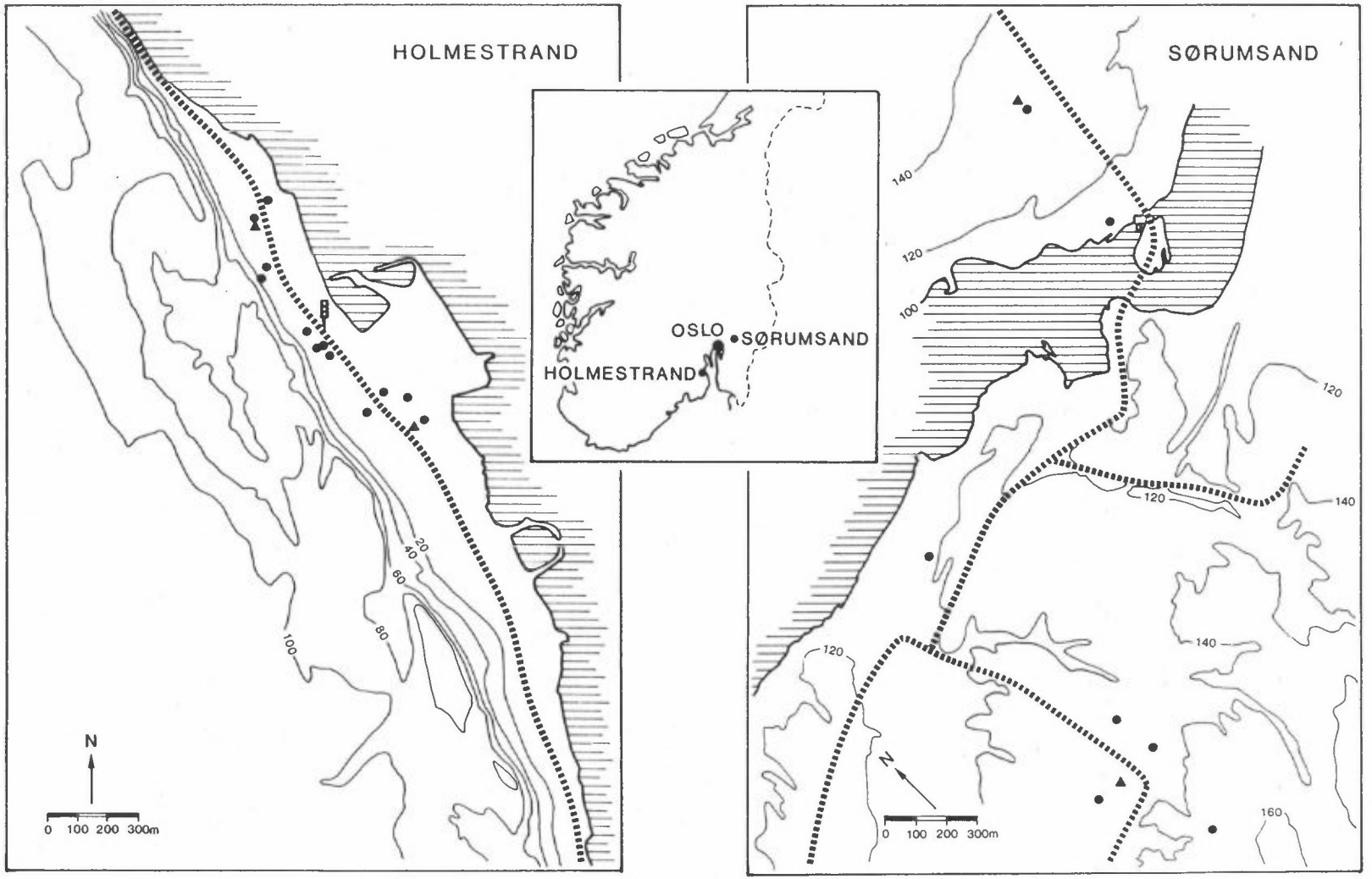

Figure 1: Topographical maps of the two study towns, Holmestrand and Sørumsand with outdoor lead air lead stations (▲) and indoor air lead stations (●), major roads indicated (▪▪▪▪).

The goal of the study was to provide coefficients that would allow predicting the magnitude of decrease in blood lead concentrations in exposed populations after measures have been enacted to reduce concentrations of lead in air. The literature predicts between 1 and 2 µg/100 ml decline with a reduction of 1 $\mu g/m^3$ of lead in air. There are two different ways of determining this: 1) by comparing different sites having different exposure to ambient lead (cross-sectional design), and 2) by comparing the same people before and after a change in exposure (cohort study design). This study was designed such that it could examine the problem both ways. Phase I of the study is equivalent to method 1 above and Phase II equivalent to method 2. Individual air lead exposure was estimated for each individual, by combining information on weekly activity patterns from a self-administered questionnaire with both measured and estimated ambient lead concentrations. The questionnaire also included information on additional lead exposure via hobbies, occupation and smoking (both active and passive).

The findings of Phase I indicated that:

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

2) The relationship of blood to air lead concentration (the amount of blood lead corresponding to a specific air lead exposure) differed with age, sex and smoking habits. Children had higher concentrations of blood lead for a given air lead exposure than adults (slope of regression line steeper). Children exposed to passive smoking had higher blood lead concentrations than children not exposed to passive smoking, and female smokers had higher values of blood lead 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 concentrations were about 6.4 µg/100 ml (0.31 µmoles/l) in adult men; 2.9 µg/100ml (0.14 µmoles/l) in adult women; and 2.5 µg/100ml (0.12 µmoles/l) in children.

The first study in Holmestrand gave preliminary indication that under conditions of chronic exposure to low to moderate air lead stemming from traffic pollution (0.03-0.25 $\mu g/m^3$), inhalation could be responsible from 10 to as high as 60 to 80% of the lead concentrations found in blood in the most sensitive population subgroups. Active smoking by women and passive smoking by children could significantly increase the importance of inhalation's contribution to blood lead concentrations. Therefore, in populations of children exposed to passive smoking or in female smokers, reducing ambient concentrations of lead would result in considerably larger reductions in blood lead than had been previously predicted in the literature.

Outdoor air samplers have been used to measure exposure to outdoor lead in most published lead exposure studies, (EPA criteria document, 1977; Hammond et al., 1981, Chamberlain, 1983, Snee, 1981). The number of stations and duration of measurement varied among studies. Using outdoor air samplers alone can satisfactorily distinguish between major regional differences in ambient concentrations. These samplers, however, are not sufficent to measure individual air lead exposure. Regional differences in the indoor to outdoor air lead ratio can occur due to differences in age of buildings, use of air conditioners, or season of the year with resulting changes in ventilation. Ignoring these differences can lead to problems in assessing the resulting actual air lead exposure in individuals.

We define individual air lead exposure as the average daily air lead concentration each individual is exposed to. A 10% indoor/outdoor ratio (f.ex. 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% indoor/outdoor ratio (f.ex. 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 in 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 relation between changes in gasoline lead consumption and blood lead levels. However, it was impossible in these studies to calculate a blood to air lead 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.

This study reports the changes in blood lead concentrations measured in the same individuals immediately before and one year after the opening of the tunnel in Holmestrand. The individuals from Sørumsand were also remeasured one year later as a control. The study has a relatively different study design than previously reported investigations since it estimates individual exposure to ambient lead for all participants both years using the same methodology. Confounding factors such as socio-economic status, smoking habits, occupational exposure to lead or exposure to lead via lead contaminated hobbies was accounted for using a questionnaire that was administered both years.

2 MATERIALS AND METHODS

2.1 OVERVIEW

The study used information from three main sources

1) self-administered questionnaires, that is each individual fills out a detailed questionnaire himself
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. See Table 1.

Table 1: A brief summary of the methods used in the study of differences in blood and air lead concentrations in the inhabitants of Holmestrand and Sørumsand in 1983 and 1984.

METHODOLOGY	
AIR	BLOOD
1) OUTDOOR AIR - 2 stations/town Circa 30 days - Low Volume Automatic Samplers - Filters measured by atomic absorption - Daily averages	- 10 ml Venous blood - Heparinized vacutainer - Atomic absorption
2) INDOOR AIR - Portable Air Samplers - Filters measured by atomic absorption	
QUESTIONNAIRE	
USED FOR ESTIMATION OF AIR LEAD EXPOSURE	CONTROLLED FOR CONFOUNDING FACTORS
- Site of home - Site of work/school - Time spent indoors/outdoors - Travel time - Location previous weekend	- Age - Sex - Occupation - Smoking - Exposure to passive smoking - Hobbies - Alcohol Consumption - Consumption of iron supplements - Consumption of medication

2.2 CHOICE OF SUBJECTS

2.2.1 Subject selection

Holmestrand

In 1983, individuals were selected to participate in a study on blood lead concentrations in the population before the tunnel opened. Criterion for subject inclusion was that the individual either lived or worked near the main highway. Efforts were made to include as many children as possible, therefore, all children and their families living in the area of interest were contacted. After the tunnel had opened in 1984, all individuals who participated in 1983 were recontacted and asked to participate again. Of the original population sample, 84% participated in 1984. In 1984, the participants were also asked to collect samples of urine to be analysed for cadmium.

Sørumsand

In Sørumsand, letters were sent to families with children living near the outdoor samplers. As in Holmestrand, the sample population was first recruited in 1983, and then recontacted in 1984 (86% of the original population took part).

In this kind of study, the selection of participants is not random since it is of course based on voluntary participation. More participants are likely to be recruited from people working in the health fields, people possibly more interested in their own health (e.g. joggers) and on the contrary from people having been quite sick and therefore used to blood sampling.

2.2.2 Population characteristics

Approximately 80% of the 1983 study population participated in the study in 1984. The two primary reasons for not participating in 1984 were death, or moving out of the area. Population characteristics are summarized in Table 2.

Table 2: Population characteristics of participants from the two towns where blood and air lead concentrations were measured - Holmestrand (moderate air lead levels) and Sørumsand (low air lead levels) in 1983 and in 1984.

	1983				1984			
	Holmestrand		Sørumsand		Holmestrand		Sørumsand	
Sample size	178		125		149		107	
Age range	3-91 years		3-90 years		3-92 years		3-91 years	
Numbers of:	male	female	male	female	male	female	male	female
Children (2-15 yrs)	12	15	15	13	10	11	14	10
Adults (16-66 yrs)	41	69	30	57	34	61	25	50
Pensionists (>66 yrs)	16	28	5	6	14	19	3	5
Socio-economic composition* (Percentage of population)								
Social Class A	15%		50%		18%		52%	
" " B	25%		16%		29%		17%	
" " C	19%		21%		20%		20%	
" " D	11%		4%		8%		4%	
" " E	0%		0%		0%		5%	
those on public assistance F	9%		9%		9%		3%	

* Skrede (1971).

2.3 ESTIMATION OF AIR LEAD EXPOSURE

An unusual feature of this study relative to most investigations of the influence of air lead on blood lead concentrations, is the attempt to estimate individual air lead exposure that accounts for time spent both indoors and outdoors.

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 information pertaining to time spent in each of several microenvironments (e.g. indoor home, indoor school, outdoor school) to create the individual ambient lead exposure estimate. This estimate was determined as an average air lead concentration over a 14-day period (in $\mu g/m^3$) that each individual was exposed to prior to blood sampling. This method of exposure estimating is called the "diary method", and was improved in 1984 to account for open window during the night, and an improved estimation of indoor values based on building type. The 1983 data were

then similarly revised, so that some of the exposure values reported in this report are different from those reported in the first report of the series (Clench-Aas et al., 1984). For the comparison of 1983 and 1984 data, it was important that both pre and post exposure was estimated using the same model.

All other features of the exposure estimation were the same in the two years and are summarized in Table 1. Figure 1 shows location of outdoor and indoor air lead stations. The stations were situated identically the two years.

2.3.1 Fixed outdoor stations

Holmestrand

Two fixed low volume samplers were placed with air intake at a height of 2 meters. Twenty-four hour samples were collected over a 30 day period. The sites in Holmestrand were (Figure 1): 1) to the north of the crosslight near an old people´s home where thirty of the study participants lived, and 2) to the south of the crosslight near a school that was attended by nearly all the children in the area. The air lead values used for this study are found in Appendix 1.

Sørumsand

Two fixed low volume samplers, of the same type as used in Holmestrand were placed with air intake at a height of 2 meters. Twenty-four hour samples were collected for 29 days. The sites were chosen in areas where most of the volunteers lived (Figure 1).

2.3.2 Indoor air samples

Portable 8-hour samplers were distributed to shops, schools and private individuals living in the experimental area (Figure 1 and Table 8). Generally, 3 consecutive 8-hour samples were collected at

each site (generating a full 24-hour sampling period). Some samples, such as in shops and schools, were collected for a shorter period.

Indoor values were found to vary by house-type (new apartment, old apartment, house, etc.). Coefficients for I/O (percentage of outdoor air concentrations found indoors) ranged from 35% to 60%. These coefficients were used dependent on information furnished by the volunteers on their homes. In addition, indoor samplers were used inside cars to estimate the amount of lead absorbed during car transit in the city.

2.3.3 Chemical analysis of ambient lead

Inhalable particulate bound lead (particle size <10 µm) was collected on Whatman 40 cellulose fiber filters by the low-volume sampler. The diameter of particles collected by the low-volume sampler varies from 10 to 20 µm dependent on wind velocity, although 10 µm seems a more reliable figure given the construction of the sampler. The filter determines the smallest diameter. The filter used allows at least 80% efficiency of recovery for particles of a diameter of 0.1 µm or less.

Lead on the filters was determined 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 atomizer 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 µg Pb/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 determination 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 ml 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

Atomizer/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/

The filters from the portable 8-hour samplers used for measuring indoor air lead concentrations (Millipore AAwp 0.8 µm mixed cellulose ester membrane filter) were analyzed for lead in the same laboratory (NILU) and using the same methodology as the outdoor samples. These portable samplers collect particles whose diameter is less than 15-20 µm but generally larger than the low-volume sampler since the air impacts the filter directly, not (as in the case with the low-volume sampler) having to travel through tubing. The filters used in these samplers have 99.999% recovery efficiency for particles 0.035 µm in diameter.

2.3.4 Diary information

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

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

   a) In Holmestrand, the area was divided into 6 regions, where people either worked or lived. The divisions were made in 1983 to account for regions with differing amounts of traffic.

   b) In Sørumsand - four general living areas were singled out. The division here appeared less important since air lead levels appeared relatively uniform over the entire area.

2) Overviews were acquired of time spent indoors at home, indoors at work or school, outdoors, time spent jogging or in physical activity and time spent travelling for the 14 days prior to blood sampling.

2.3.5 Individual air lead exposure estimate

Lead concentrations in blood have a 16 to 18 day half-life and thus reflect a relatively long accumulation period (Chamberlain et al., 1978). The individual air lead exposure estimate reflects an average air lead concentration (in $\mu g/m^3$) that individuals were exposed to for the 14 days prior to blood sampling. It takes into account time spent indoors and travelling. A factor of 2 was used for the air exposure if people were jogging to account for increased respiratory rate. Likewise, a factor (1.2) was used to account for higher activity in children when they were outdoors. (Factors found both in consultation with lung specialists and Åstrand and Rodahl, 1977.) Use of 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 (Clench-Aas et al., 1984).

An overview of the method of air lead exposure estimating is given in Table 4.

Table 4: Overview over information used for estimating short term exposure to lead.

Geographic area	Lead concentration Day 1	Day 2	Day 3	Day 30
Holmestrand				
Area A				
Area B				
Area C				
Area D etc.				
Car				
Sørumsand				
Area A				
Area B etc.				

+

INDOORS/OUTDOORS COEFFICIENT MATRIX

Building Type	Window/Vent open/closed
Apartment old	
Apartment new	
Individual house, old	
Individual house, new	
etc.	

+

+

DIARY

Microenviron	DAY Day 1	Day 2	Day 3	Day 30
Home				
inside				
outside				
Work/School				
inside				
outside				
In car				
etc.				

COEFFICIENT MATRIX TO ACCOUNT FOR LUNG VENTILATION FROM ACTIVITY LEVEL

Activity Type	Coefficient
Sleeping	
Playing	
Jogging	
etc.	

In order to obtain a more accurate estimate of individual air lead exposure, outdoor concentrations were modified according to building type, if the window was closed.

2.3.6 Measurements of indoor and outdoor dust and drinking water

In order to ascertain if blood lead concentrations 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, whose opening lies at a height of 1.5 m. The sampling period was

43 days in 1983 and circa 30 days in 1984 in Holmestrand and 29 days in Sørumsand both years. Lead in dust was analyzed both in the water soluble and water insoluble fractions in the same laboratory (NILU) using the same methods as for air samples.

Indoor dust samples were collected by washing a square surface (10 x 10 cm) in the dustiest corner of the house with a filter soaked in distilled water. 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 sampling method was first described by Vostal et al., in 1974. Sampling sites were as similar as possible both years.

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. Nitric acid is added to stabilize the sampler (Clench-Aas et al., 1984).

2.4 BLOOD MEASUREMENTS

2.4.1 Collection of blood samples

Whole blood (3-10 ml), was collected from each individual in green stoppered Venoject evacuated blood collection tubes (VT 100 SH - sodium heparin). Blood sampling was done in May 1983 and 1984.

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. 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

Hematocrit (red blood cell volume in per cent of whole blood) was measured in order to standardize blood lead concentrations. As a

further control of hematocrit values, hemoglobin values were also regularly measured in 1984. All determinations were made by the National Institute of Occupational Health.

Hematocrit (Ht) was determined in duplicate using microhematocrit centrifuge (LIC HK4) at 9500 G for three minutes.

Hemoglobin (Hb) was measured (by the standard cyanmethemoglobin method using photometer (Linson 3)).

Zinc-protoporphyrin (ZPP) 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 of 45%.

In 1983 the measurements were made after the samples were brought back to the laboratory. In this way, some samples were lost due to hemolysis of the samples prior to measurement. Therefore, in 1984 the blood parameters were measured on site immediately after blood sampling to increase the number of valid samples.

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 concentrations. The contamination of lead from both collection tubes and syringes was tested by leaching with 0.2 molar HNO_3. It was found 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 a graphite atomizer Model 500, an AS-40 automatic sampler, a PRS-10 printer, a Model 56 recorder, a deuterium arc background corrector and a lead electrodeless discharge lamp. A summary of the whole blood lead method is listed in Table 5. All measurements were made by the National Institute of Occupational Health and were done identically both years by the same technicians.

Table 5: Summary of whole blood lead determination method.

Sample Preparation

Dilute whole blood 1:4 with chemical 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

Atomizer/Autosampler Program

Sample volume 10 µl, ordinary graphite tubes.

	Temp. ^0C		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

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 or amount of hemoglobin can influence blood lead concentration. This would result in apparently higher blood lead levels in those individuals whose hematocrit values were elevated due to other factors, e.g. smoking, and apparently false low values in anemic individuals. Therefore, blood lead concentrations were all standardized to a hematocrit of 45% (McIntire and Angle, 1972) using the formula:

$$\text{Standardized B-Pb} = \frac{\text{B-Pb} \times 45.0}{\text{measured hematocrit}}$$

Since the intention of this study was to compare values of lead in blood (B-Pb) with lead in air, and air is measured in µg/m^3, blood lead values have been converted from µmoles/l to µg/100 ml (dl) using the formula B-Pb µg/100ml = B-Pb(µmoles/l) x 20.72.

2.4.4 Quality control programs

The accuracy of the blood-lead determination method is confirmed twice a year through interlaboratory survey programs organized by the Swedish National Board of Occupational Safety and Health. The performance of the present method from the three last years are plotted in Figure 2. 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% at the normal concentration level.

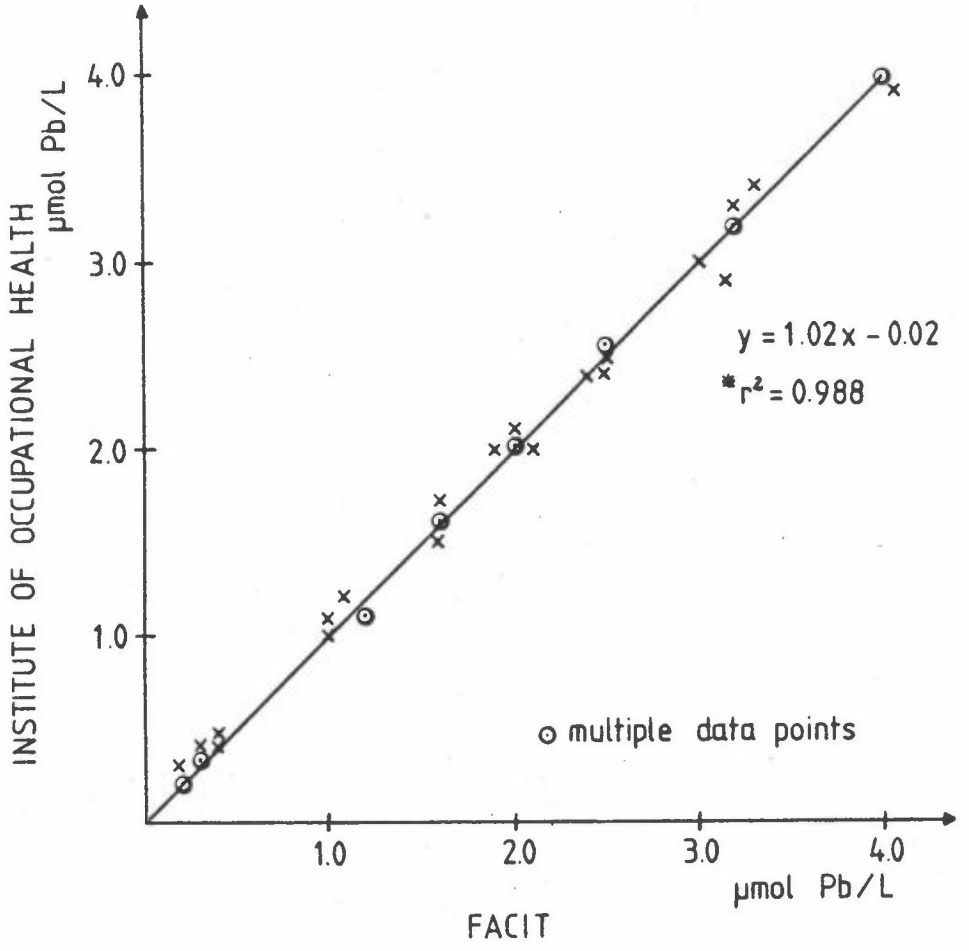

Figure 2: Results from interlaboratory surveys of lead analysis in whole blood (1980-1983). The linear regression is based on 47 data pairs. r^2 = coefficient of determination.

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, eating of snow, etc. was revealed.

The questionnaire was very similar in the two years. However, in 1984 two questions were added on: 1) consumption of alcohol, and 2) medication usage, especially iron supplements. The items covered are summarized in Table 1.

The smoking information was detailed covering 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.

Information on occupational exposure to lead covered both current and previous exposure.

All individuals were classified into social category by occupation; for housewives by occupation of spouse; for children by occupation of male parent followed by female parent. The classification system used (Skrede, 1971) divides occupation into five classes (see Clench-Aas et al., 1984) (Table 2).

Information provided by children was verified by comparing that given by the parents where possible.

2.6 DATA ANALYSIS

The complete 1983 and 1984 data set was reduced to include only the individuals who participated in both years. For each individual, the blood parameter measurements from 1984 were analysed along with the

differences in them between 1983 and 1984, and with the items covered by the questionnaire. The data analysis was performed using the SPSS/ PC+ statistical package (Nie et al., 1975).

3 RESULTS

3.1 DIFFERENCES IN AIR LEAD EXPOSURE BETWEEN 1983 AND 1984

3.1.1 Concentrations of lead in outdoor air in Holmestrand and Sørumsand in 1983 and 1984

As can be seen in Figures 3 and 4, Table 6 and Appendix 1, concentrations of lead in air dropped substantially in Holmestrand from 1983 to 1984. At station 1 (north of the crosslight) values measured during the weekends (peak times due to vacation traffic through the town) decreased from 0.263 to 0.055 $\mu g/m^3$. It is also interesting to note that concentrations of lead in air measured in Sørumsand also decreased slightly between 1983 and 1984. Traffic counts there indicated approximately the same amount of traffic both years, therefore the decline is probably due to the gradual decrease in lead content of high octane gasoline (from 0.4 to 0.15 g/l). (More details of the results of the 1983 investigation are reported in Clench-Aas et al., 1984.)

3.1.2 Concentrations of lead in outdoor dust in 1983 and 1984

As can be seen in Table 7, concentrations of lead in outdoor dust decreased from 1983 to 1984 by approximately 60% in both Holmestrand and Sørumsand. The decrease was only visible in the water soluble fraction.

3.1.3 Concentrations of indoor air lead levels in 1983 and 1984

Changes in concentrations of lead in indoor air (Table 8) also decreased in Holmestrand, especially in commercial and public buildings where concentrations tended to be highest. This is as expected since concentrations measured indoors reflect values measured outdoors.

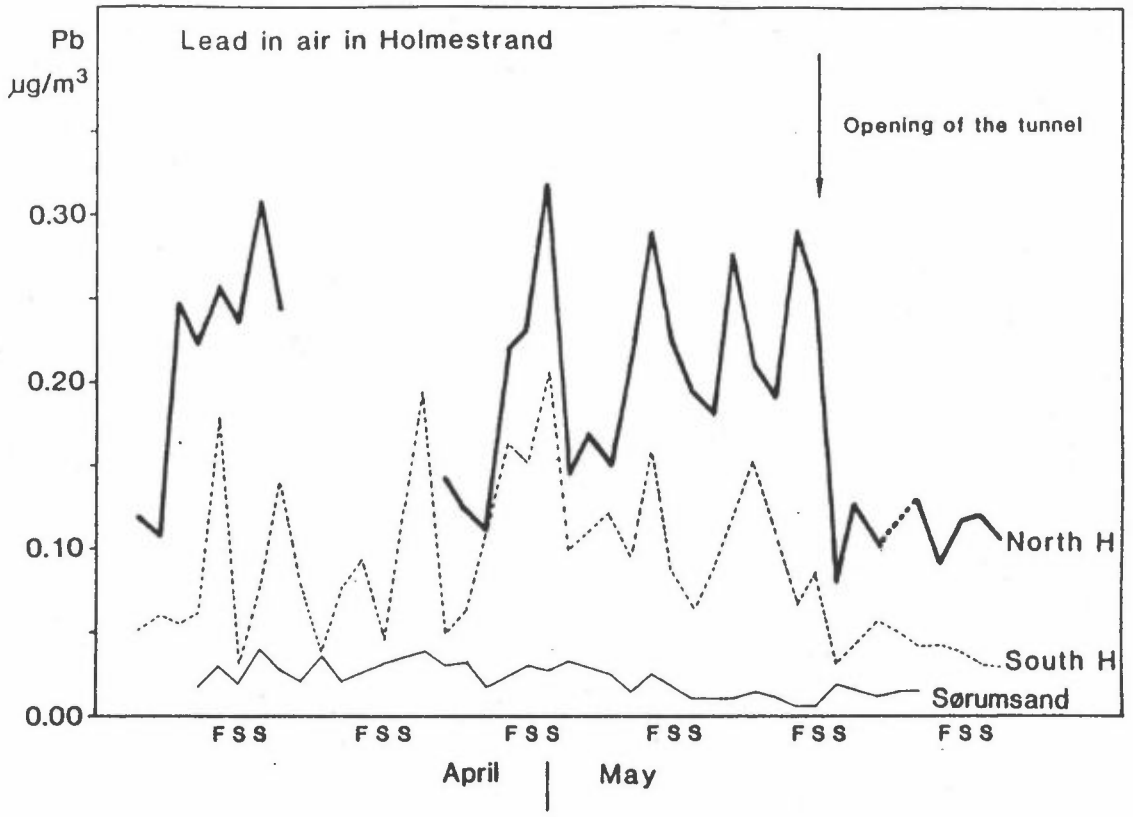

Figure 3: Concentrations of lead in air in the spring of 1983 at two locations in Holmestrand and one in Sørumsand.

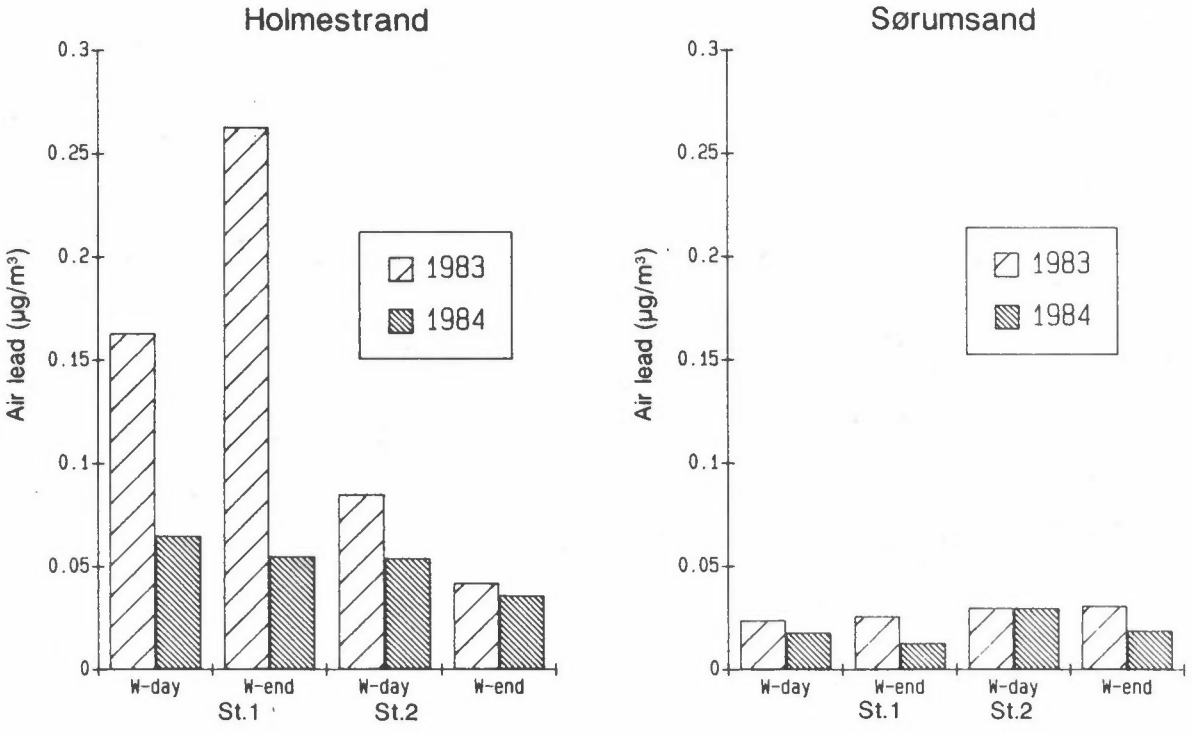

Figure 4: Concentrations of ambient lead in Holmestrand and Sørumsand (2 stations each) in 1983-1984.

Table 6: Measured air lead ($\mu g/m^3$) (daily means) for the 30 days preceeding blood sampling in Sørumsand and Holmestrand in 1983 and 1984.

		1983		1984	
		Weekday	Weekend	Weekday	Weekend
Sørumsand	St. 1	0.024	0.026	0.018	0.013
	St. 2	0.030	0.031	0.030	0.019
Holmestrand	St. 1	0.163	0.263	0.065	0.055
	St. 2	0.085	0.042	0.054	0.036

Table 7: Lead concentrations (in $\mu g/m^2/day$) in outdoor dustfall in Holmestrand and Sørumsand during 1983 and 1984. Samples were collected over a 1 month period. Total volume of precipitation collected indicated in ml in parentheses.

	1983			1984		
Site	H_2O insol $\mu g/m^2/day$	H_2O sol $\mu g/m^2/day$	Total $\mu g/m^2/day$	H_2O insol $\mu g/m^2/day$	H_2O sol $\mu g/m^2/day$	Total $\mu g/m^2/day$
Holmestrand	44.1	72.2 (3250 ml)	116.3	40.9	1.3 (1205 ml)	42.2
Sørumsand	10.4	20.8 (525 ml)	31.2	11.9	1.3 (480 ml)	13.2

3.1.4 Concentrations of lead in indoor dust and drinking water in 1983 and 1984

Values of lead measured in indoor dust are more difficult to interpret. As can be seen in Table 8, some values did decrease substantially. The sampling method used, that of choosing the dustiest area in the building, was very subjective and could reflect simply changes in hygiene or cleanliness. An attempt was made to make the two measurements (one year apart) in the same place.

Drinking water samples were taken in 1983 and found not to include measurable (< 1 µg Pb/l) concentrations of lead. Therefore, they were not resampled in 1984.

Table 8: Average 24 hour indoor air lead concentrations in Holmestrand and Sørumsand in May 1983 and 1984, with corresponding measured outdoor values at the fixed stations. Indoor dust for the same time periods is also given.

Site	Indoor air* ($\mu g/m^3$) 1983	1984	Indoor dust ($\mu g/m^2$) 1983	1984
HOLMESTRAND				
Commercial estb.				
1	1.66	0.55	595	530
2	0.35	0.13	22	420
3	0.29	0.09	21	47
Public Bldg.				
1	0.09	0.04	116	12
2	0.11	0.04	-	-
3	0.18	0.05	51	20
Schools				
1	0.08	0.01	39	18
2	0.02	0.04	-	-
3	0.04	0.04	-	-
Homes				
1(N.S.)**	0.07	0.07	9	-
2(S.)	0.06	0.05	44	-
Car	-	1.82	-	-
Outdoor val. for same day as indoor ranged	0.03 to 0.15	0.01 to 0.04		
SØRUMSAND				
Public Bldg.	0.004	0.03	No samples taken	
Homes				
1(N.S.)	0.01	0.02		
2(S.)	0.01	0.02		
3(N.S.)	-	0.01		
4(S.)	-	0.02		
Outdoor val. for same day as indoor ranged	0.016 to 0.011	0.01 to 0.04		

* Indoor values were measured using portable pumps and a slightly different filter system than fixed outdoor samplers. In addition, the intake is different between the two systems, therefore, comparisons should be made with caution.

** N.S.: non-smokers only, S: smokers present.

3.1.5 Estimates of personal air lead exposure in 1983 and 1984

As can be seen in Table 9, personal exposure to air lead as measured with aid of the diary method declined markedly both in children and adults in Holmestrand. A decrease was also seen in Sørumsand, although less marked.

Table 9: Exposure estimates for air lead concentrations ($\mu g/m^3$) in individuals from Holmestrand and Sørumsand by age groups for 1983 and 1984.

CHILDREN				ADULTS			
	Age group (yrs)					Age group (yrs)	
	0-4	5-10	11-15			16-66	67+
SØRUMSAND 1983							
					Mean	0.11	0.03
				Male	St.dev.	0.11	0.01
Mean	0.06	0.06	0.06		(N)	18	3
St.dev.	0.06	0.04	0.02		Median	0.07	0.02
(N)	2	10	8				
Median	0.02	0.05	0.06		Mean	0.05	0.11
				Female	St.dev.	0.03	0.11
					(N)	48	3
					Median	0.05	0.04
SØRUMSAND 1984							
					Mean	0.09	0.01
				Male	St.dev	0.08	0.00
Mean	0.03	0.03	0.04		(N)	18	3
St.dev.	0.02	0.02	0.02		Median	0.08	0.01
(N)	2	10	8				
Median	0.02	0.02	0.03		Mean	0.06	0.06
				Female	St.dev.	0.06	0.08
					(N)	48	3
					Median	0.05	0.01
HOLMESTRAND 1983							
					Mean	0.22	0.14
				Male	St.dev.	0.17	0.04
Mean	0.58	0.15	0.11		(N)	23	12
St.dev.	-	0.06	0.04		Median	0.13	0.14
(N)	1	6	15				
Median	0.58	0.16	0.11		Mean	0.22	0.15
				Female	St.dev.	0.18	0.06
					(N)	52	17
					Median	0.13	0.16
HOLMESTRAND 1984							
					Mean	0.10	0.04
				Male	St.dev.	0.04	0.02
Mean	0.03	0.06	0.06		(N)	23	12
St.dev.	-	0.02	0.04		Median	0.09	0.04
(N)	1	6	15				
Median	0.03	0.06	0.05		Mean	0.08	0.04
				Female	St.dev.	0.05	0.02
					(N)	52	17
					Median	0.07	0.04

Blood lead was hematocrit adjusted. Occupationally exposed individuals as well as those possibly exposed through hobbies not included.

3.2 BLOOD LEAD CONCENTRATIONS MEASURED IN 1984

The natural logarithm of the concentrations of lead measured in children (over 3 years) and adults in 1984 were analysed by analysis of variance with covariates. The analysis included a set of covariates (the natural logarithm of air and/or blood lead concentrations, age and hematocrit) and main factors (sex, passive smoking in children and smoking in adults, and alcohol consumption in adults). The effect of the factor was determined after removing the effect of the covariates. The results of the statistical analysis are summarized in Table 10.

Table 10: Results of analysis of variance of values measured in 1984 of natural logarithm of blood lead concentrations (ln PbB), hematocrit (Ht) and natural logarithm of zinc protoporphyrin (ln ZPP) in children over 3 years and in adults.

		CHILDREN				ADULTS		
Dependent variable	N	ln PbB	Ht	ln ZPP	N	ln PbB	Ht	ln ZPP
COVARIATES*								
ln PbA	38	0.43	N.S.	N.S.	190	0.17	N.S.	N.S.
ln PbB		N.A.	1.72	N.S.		N.A.	2.69	N.S.
Age		N.S.	0.571	N.S.		N.S.	N.S.	N.S.
Ht		N.A.	N.A.	-0.17		N.A.	N.A.	-0.09
MAIN FACTORS**								
Sex								
Male	21	+0.15	N.S.	N.S.	71	+0.21	+1.74	N.S.
Female	17	-0.16	N.S.	N.S.	119	-0.12	-1.04	N.S.
Passive smoking								
Not exposed	27	-0.15	N.S.	-0.28		N.A.	N.A.	N.A.
Exposed	11	+0.36	N.S.	+0.68		N.A.	N.A.	N.A.
Smoking								
Non-smokers		N.A.	N.A.	N.A.	110	-0.06	N.S.	-0.10
Smokers		N.A.	N.A.	N.A.	80	+0.08	N.S.	+0.14
Alcohol consumption								
Total non-drinkers and occasional drinkers		N.A.	N.A.	N.A.	138	-0.07	N.S.	N.S.
Drink weekly		N.A.	N.A.	N.A.	52	+0.17	N.S.	N.S.

* N.S. = Not Significant; N.A. = Not Applicable.
Values reported are regression coefficients when covariate is significant at least at the 5% level. The entire analysis must be significant at least at the 5% level to be reported.

** Values are only reported when entire analysis is significant (5% level) and the individual factor is significant at least at the 5% level. Then the reported values are differences with respect to a grand mean after adjusting for covariates.

Children

Blood lead concentrations are positively correlated to air lead exposure, but not correlated to age. In addition, there are significant differences between boys and girls, boys having higher values. Passive smoking is significantly correlated to blood lead concentrations, as was also found in 1983 (Clench-Aas et al., 1984) with those children exposed for passive smoking having the higher values.

Adults

A significant correlation between blood and air lead exposure is also found for adults. Adult men have higher values than women. Smokers have higher values than non-smokers. Those who drink alcohol at least once a week have higher values than those who do not drink or drink occasionally.

3.3 DIFFERENCES IN BLOOD LEAD CONCENTRATIONS BETWEEN 1983 AND 1984

The data were analyzed using analysis of variance with covariates. The dependent variable was ΔPbB (difference in blood lead measurements between the two years). The covariates entered into the analysis were ΔPbA (difference in exposure estimate for air lead), and age. The factors entered into the analysis were sex, current smoking and alcohol consumption in adults and passive smoking in children. These variables were chosen partially based on previous findings (Clench-Aas et al., 1984). The analysis was performed by first removing the effect of the covariates then computing the effect on the factors.

Frequency distribution of ΔPbB is close to a normal distribution, with one outlying observation. This observation was not removed prior to the analysis, but the value was checked and is considered correct. For the histogram of ΔPbB see Appendix 2.

Children

As can be seen in Table 11 there are no significant relations between the differences in blood lead concentrations and differences in

exposure to air lead or age. There are no significant sex differences. However, there are differences between passive smoking exposed children and those not exposed to passive smoking (at the 6% level). Those that are exposed to passive smoking show a larger decline in blood lead concentrations than those not exposed (Table 12). This seems to indicate that even though we measure higher uptake of air lead by children exposed to passive smoking both in 1983 and in 1984, the exposed children have a greater decrease in blood lead values despite similar declines in air lead exposure.

Table 11: Results of analysis of variance of measured differences (1984 values - 1983 values) in blood lead concentrations (Δ PbB), in hematocrit (Δ Ht) and in zinc protoporphyrin (Δ ZPP) in children over 3 years and adults.

	CHILDREN				ADULTS			
	N	Δ PbB	Δ Ht	Δ ZPP	N	Δ PbB	Δ Ht	Δ ZPP
COVARIATES[1]								
Δ PbB	38	N.A.	N.S.	N.S.	188	N.A.	-0.23	N.S.
Δ PbA		N.S.	N.S.	N.S.		3.76	+4.05	+0.22
Age		N.S.	N.S.	N.S.		N.S.	+0.05	N.S.
Δ Ht		N.A.	N.A.	N.S.		N.A.	N.A.	-0.01
MAIN FACTORS[2]								
Grand mean		1.58				1.09		0.221
Sex								
Male	21	N.S.	N.S.	N.S.	118	N.S.	N.S.	-0.04
Female	17	N.S.	N.S.	N.S.	71	N.S.	N.S.	+0.03
Passive smoking								
Not exposed	27	-0.50[3]	N.S.	N.S.		N.A.	N.A.	N.A.
Exposed	11	+1.34	N.S.	N.S.		N.A.	N.A.	N.A.
Smoking								
Non-smokers		N.A.	N.A.	N.A.	109	N.S.	N.S.	N.S.
Smokers		N.A.	N.A.	N.A.	80	N.S.	N.S.	N.S.
Alcohol consumption								
Total non-drinkers and occasional drinkers		N.A.	N.A.	N.A.	137	+0.23	N.S.	N.S.
Drink weekly		N.A.	N.A.	N.A.	52	-0.61	N.S.	N.S.

1) N.S. = Not Significant; N.A. = Not Applicable. Values reported are regression coefficients when covariate is significantly (at least of the 5% level) related. The entire analysis must be significant to report individual differences.
2) Values are only reported when the entire analysis of variance is significant and the individual factor is significant at least at the 5% level. The values reported are differences from the grand mean after adjusting for covariates.
3) Significant at the 0.06 level.

Table 12: Differences in air lead concentration and measured blood parameters between 1983 and 1984 with respect to passive smoking in children.

	Holmestrand		Sørumsand	
	passive smoking		passive smoking	
	no	yes	no	yes
Δ PbA ($\mu g/m^3$)				
Valid N	11	13	21	3
Mean	0.05	0.11	0.02	0.02
Median	0.04	0.08	0.02	0.04
Std.dev.	0.03	0.14	0.03	0.04
Δ PbB ($\mu g/m^3$)				
Valid N	10	11	17	3
Mean	1.64	2.60	0.60	0.40
Median	1.67	2.81	0.73	1.29
Std.dev.	2.51	3.30	1.94	1.55
Δ Ht (%)				
Valid N	10	12	20	3
Mean	-1.10	-1.10	0.08	-2.17
Median	-0.50	-1.00	0.25	-1.00
Std.dev.	1.93	2.33	2.07	2.02
Δ ZPP ($\mu mol/l$)				
Valid N	10	12	19	3
Mean	0.34	0.33	0.22	0.25
Median	0.37	0.33	0.21	0.23
Std.dev.	0.10	0.13	0.10	0.04

Adults

As can be seen in Table 11, the analysis of variance with covariates indicates that age, sex and smoking habits do not exert a significant effect on ΔPbB, whereas ΔPbA and alcohol consumption do ($P<0.01$). The regression coefficient for the relation between ΔPbB and ΔPbA was 3.8 indicating that blood lead concentrations decrease around 4 µg/dl for a decline of 1 $\mu g/m^3$ air lead concentration (Figure 5). This value is higher than the values of between 1 to 2 reported in the literature. This result was already indicated in phase I of this study (Clench-Aas et al., 1984). The results of the regression without the high exposure point are shown in Figure 5. Since the values reported in the literature were primarily derived from studies surrounding iron smelters, the indication seems to be that lead originating from traffic pollution is more completely absorbed into the body than ambient lead originating from smelters. Several explanations may be offered.

Differences in particle size may lead to differential absorption. Differences in chemical forms of Pb (elemental Pb from traffic, pollution and PbO from smelters) may also explain differential absorption. A fuller discussion of this subject can be found in a similar study done by the same research team in an area in Norway surrounding a smelter (Clench-Aas et al., 1986).

The more interesting finding in this study was the significant negative correlation between ΔPbB and alcohol consumption (Figure 6). Those individuals drinking alcohol at least once a week decreased their blood lead concentration by 0.5 μg/dl, whereas those who never or only occasionally consumed alcohol decreased their blood lead concentration by 1.33 μg/dl. The data are presented in the form of two way tables in Tables 14 to 17. Total non drinkers have higher values than both those that drink occasionally (less than once a week), and those that drink on a weekly basis. Phrased differently, those individuals that do not drink alcohol at all had a higher decline in blood lead concentrations with a decline in air lead concentrations than those that consumed alcohol. This seems to indicate metabolic changes induced by the consumption of alcohol that exert an influence on metal metabolism in the organism. Changes in the intestinal barrier that increase lead absorption from the gut have been reported (Moore, 1986).

The preliminary findings reported in Phase I of the study (Clench-Aas et al., 1984) indicated that adult women smokers had substantially higher ratios of blood to air lead concentrations. This possibly indicated a higher uptake in the lungs. Children exposed to passive smoking as opposed to children not exposed revealed the same phenomenon. The data for adult men and women were therefore divided into groups of smokers and non-smokers. The multiple regression of differences in blood lead concentrations on differences in exposure to air lead were significant only in women (Table 13). Regression coefficients were 6.2 for non-smoking women and 7.0 for smoking women. These values are substantially higher than the value of 3.8 measured on the entire undivided population. These values do not indicate a difference between smoking and non-smoking women, however, since levels of lead in blood were higher in smoking women both in 1983 and 1984, it is not surprising.

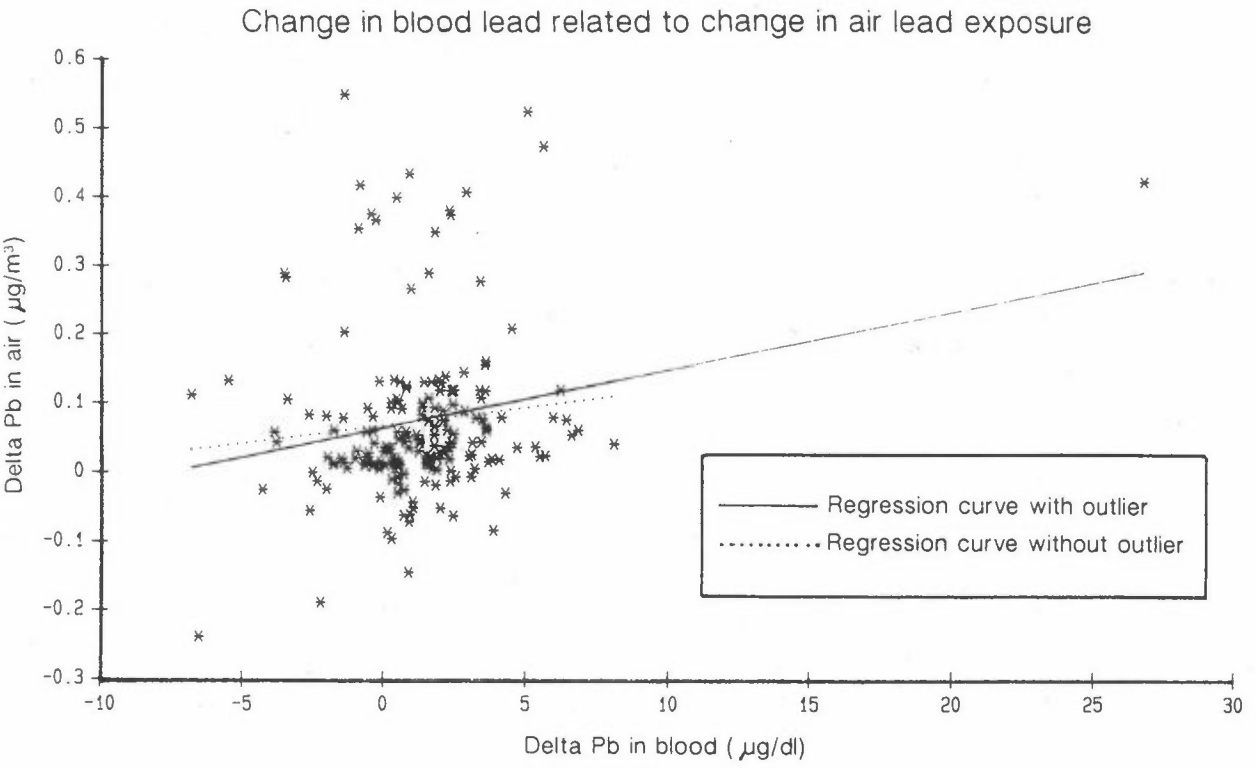

Figure 5: Regression of changes in blood lead concentrations in the same individuals between 1983 and 1984 as opposed to changes in estimated air lead exposure in the same individuals. The isolated point in the graph represents an individual living and working right at the cross light and is therefore not an outlier.

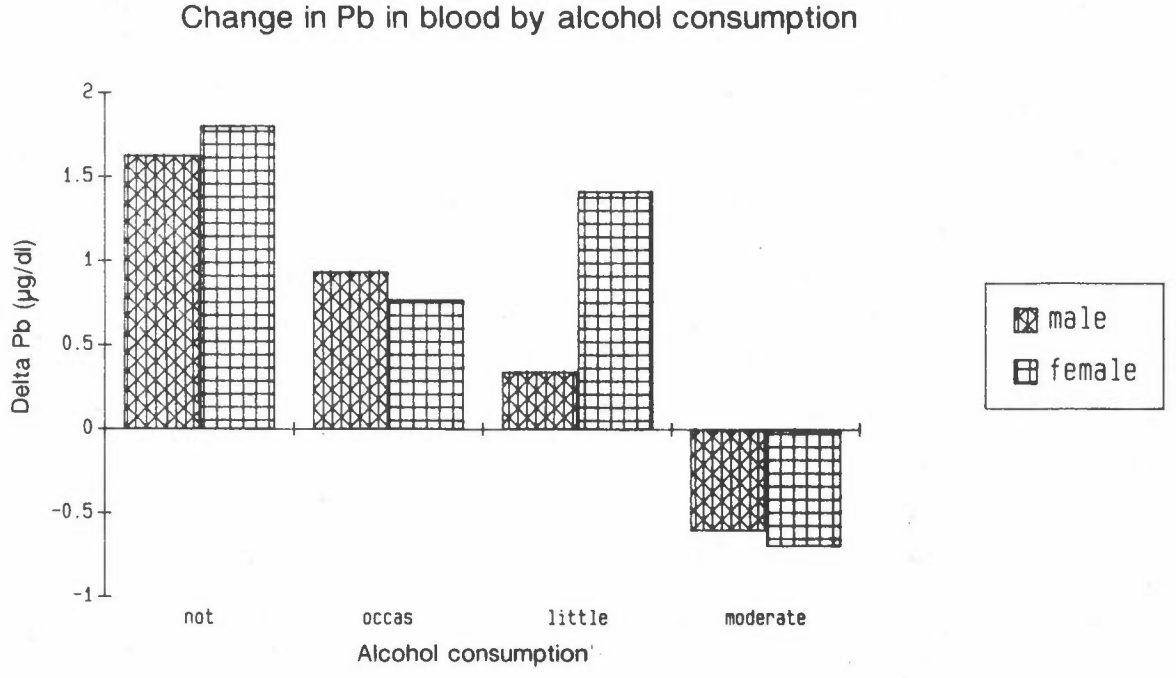

Figure 6: Changes in concentrations of lead in blood (µg/dl) in the inhabitants of Holmestrand and Sørumsand as a function of alcohol consumption in adult men and women.

Table 13: Results of multiple regression of differences between 1983 and 1984 in blood lead concentrations (Del PbB) on differences in air lead exposure (Del PbA) and alcohol consumption. Subjects were divided into 4 subgroups, by sex and smoking habits.

	N	Del PbA		Alc. cons.		Constant	
		B	Sign.	B	Sign	B	Sign.
Males							
non-smokers	34	6.74	0.28	-0.43	0.47	1.54	<0.01
smokers	38	-0.15	0.96	-0.85	0.13	1.07	0.12
Females							
non-smokers	81	6.21	<0.01	-0.40	0.11	0.69	<0.01
smokers	41	7.02	0.03	-0.91	0.11	0.88	0.11

Table 14: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Holmestrand with respect to alcohol consumption in men.

Holmestrand	Alcohol consumption*			
	not	occas	little	moderate
Δ PbA ($\mu g/m^3$)				
Valid N	17	25	8	6
Mean	0.12	0.12	0.15	0.10
Median	0.08	0.09	0.11	0.03
Std.dev.	0.16	0.10	0.16	0.18
Δ PbB ($\mu g/m^3$)				
Valid N	15	25	8	6
Mean	1.62	1.60	0.47	0.07
Median	1.78	2.06	1.34	
Std.dev.	3.51	3.12	2.37	2.17
Δ Ht (%)				
Valid N	15	25	8	6
Mean	-1.01	0.46	-0.13	0.08
Median	-0.50			0.50
Std.dev.	2.56	3.85	5.19	1.56
Δ ZPP ($\mu mol/l$)				
Valid N	15	25	8	6
Mean	0.28	0.18	0.24	0.12
Median	0.32	0.15	0.20	0.12
Std.dev.	0.12	0.14	0.20	0.14

* not : never drinks
occas : drinks occasionally but less than once a week
little : drinks weekly, but little
moderate: drinks weekly but moderate amounts

Table 15: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Holmestrand with respect to alcohol consumption in women.

Holmestrand	Alcohol consumption*			
	not	occas	little	moderate
Δ PbA ($\mu g/m^3$)				
Valid N	31	33	18	4
Mean	0.08	0.10	0.21	0.16
Median	0.08	0.07	0.13	0.08
Std.dev.	0.08	0.13	0.15	0.18
Δ PbB ($\mu g/m^3$)				
Valid N	29	29	17	4
Mean	2.02	2.06	1.17	-0.21
Median	2.09	1.64	0.79	-0.44
Std.dev.	1.68	2.40	2.75	3.02
Δ Ht (%)				
Valid N	30	31	17	4
Mean	1.02	0.06	0.38	2.38
Median	0.50	1.00	0.50	2.25
Std.dev.	3.56	3.43	4.27	1.25
Δ ZPP ($\mu mol/l$)				
Valid N	30	31	17	4
Mean	0.31	0.29	0.23	0.25
Median	0.26	0.26	0.26	0.19
Std.dev.	0.27	0.21	0.13	0.15

* not : never drinks
occas : drinks occasionally but less than once a week
little : drinks weekly, but little
moderate: drinks weekly but moderate amounts

Table 16: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Sørumsand with respect to alcohol consumption in men.

Sørumsand	Alcohol consumption*			
	not	occas	little	moderate
Δ PbA ($\mu g/m^3$)				
Valid N	10	11	9	2
Mean	-0.00	0.03	-0.00	0.02
Median	-0.00	0.02	-0.01	0.02
Std.dev.	0.03	0.04	0.06	0.00
Δ PbB ($\mu g/m^3$)				
Valid N	10	11	9	2
Mean	1.16	0.88	0.18	-1.47
Median	0.83	0.37	0.91	-1.47
Std.dev.	3.16	2.04	2.94	0.06
Δ Ht (%)				
Valid N	10	11	9	2
Mean	-2.05	-1.14	-1.28	0.75
Median	-0.75	-1.50	-1.50	0.75
Std.dev.	3.33	2.58	2.51	2.47
Δ ZPP ($\mu mol/l$)				
Valid N	10	11	9	2
Mean	0.22	0.19	0.10	0.16
Median	0.23	0.20	0.12	0.16
Std.dev.	0.11	0.07	0.08	0.05

* not : never drinks
occas : drinks occasionally but less than once a week
little : drinks weekly, but little
moderate: drinks weekly but moderate amounts

Table 17: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Sørumsand with respect to alcohol consumption in women.

Sørumsand	Alcohol consumption*			
	not	occas	little	moderate
Δ PbA ($\mu g/m^3$)				
Valid N	15	35	4	2
Mean	0.03	-0.02	0.02	0.06
Median	0.02	0.00	0.02	0.06
Std.dev.	0.02	0.06	0.02	0.04
Δ PbB ($\mu g/m^3$)				
Valid N	14	35	4	2
Mean	0.95	0.03	2.47	-1.16
Median	0.80	0.21	2.42	-1.16
Std.dev.	0.82	1.78	0.53	2.09
Δ Ht (%)				
Valid N	15	35	4	2
Mean	-0.23	-0.93	-3.63	1.50
Median	-0.50		-3.00	1.50
Std.dev.	3.45	3.39	2.66	0.71
Δ ZPP ($\mu mol/l$)				
Valid N	15	35	4	2
Mean	0.24	0.22	0.28	0.10
Median	0.19	0.18	0.28	0.10
Std.dev.	0.19	0.14	0.03	0.04

* not : never drinks
occas : drinks occasionally but less than once a week
little : drinks weekly, but little
moderate: drinks weekly but moderate amounts

3.4 HEMATOCRIT LEVELS MEASURED IN 1984

Hematocrit was measured both years of the study in order to standardize blood lead concentrations for the number of red blood cells. Hematocrit can also be viewed as an effect variable, since high blood lead concentrations have been reported to cause anemia, thus reduce hematocrit (National Academy of Sciences, 1972). However, it is necessary to point out that in Holmestrand the decline in air lead concentrations was due to a rerouting of traffic that decreased not only lead, but also other pollutants emitted by vehicular traffic. For example, reduced concentrations of carbon monoxide can on the contrary increase hematocrit. Figure 7 summarizes the expected interrelationships between air pollutants and blood parameters.

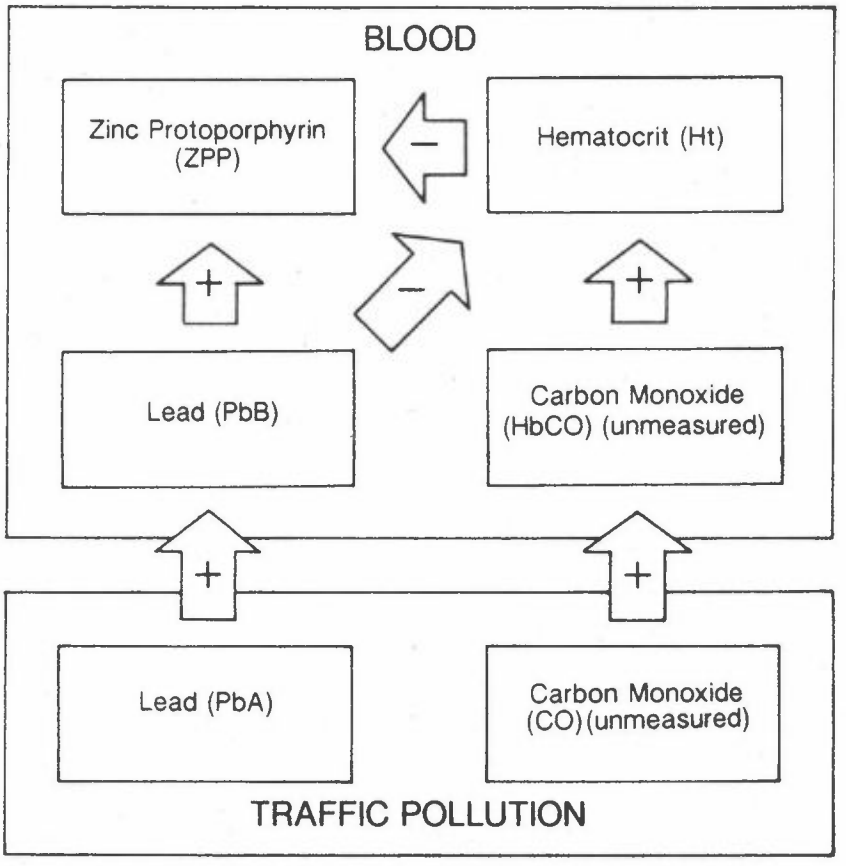

Figure 7: Path diagram of impact of traffic pollution on blood parameters. Direction of influence indicated by + or -. Carbon monoxide was not measured in this study, but since traffic pollution was removed from the air, it can be expected that carbon monoxide decreased by corresponding amounts.

Iron deficiency anemia can in turn, result in increased concentration of zinc protoporphyrin.

Children

In children over 3 years of age, both age and the natural logarithm of blood lead concentrations are significantly and positively correlated to hematocrit levels. We find a significant and positive relationship between hematocrit and blood lead instead of the negative relationship expected by the biological effects of blood lead. Since lead in blood originates from traffic pollution it is probably correct to assume that levels of carbon monoxide are also elevated at times. Carbon monoxide can increase hematocrit and therefore, the relationship observed between blood lead and hematocrit may in reality be a relationship between carbon monoxide and hematocrit.

There were no measured differences in hematocrit between boys and girls or between children exposed to passive smoking and those not exposed to passive smoking.

Adult

The same positive relationship between hematocrit and blood lead is observed in adults. The discussion above is probably relevant here as well. The well known sexual differences in hematocrit are observed in adults.

3.5 DIFFERENCES IN LEVELS OF HEMATOCRIT

The histogram of measured differences in hematocrit between 1983 and 1984 is shown in Appendix 2.

Children

Differences in hematocrit levels between 1983 and 1984 are not significantly correlated to either the covariates or the main factors.

Adults

Table 11 shows the result of an analysis of variance of ΔHt (difference in hematocrit between the two years) by the factors sex, smoking and alcohol consumption, and with the covariates ΔPbB, ΔPbA, and age. No factors were found to significantly influence ΔHt. ΔPbA and age were found to be significantly and positively related to ΔHt ($p \leq 0.05$), whereas ΔPbB was significantly ($p \leq 0.05$) but negatively correlated to ΔHt.

If the effect of higher concentrations of lead in blood was to provoke a decline in hematocrit, then hematocrit levels would increase from 1983 to 1984 with the decrease in concentrations of lead in blood. This would result in small to negative values of ΔHt with increased ΔPbB and ΔPbA, thus a negative regression coefficient. If, on the other hand, levels of CO in 1983 (with greater amounts of traffic) were high enough to increase hematocrit, then the reverse would be expected to be found. One would expect to find a high ΔHt (greater differences between 1983 and 1984) correlated to high ΔPbA or ΔPbB. The results show a positive relation of ΔHt with ΔPbA (regression coefficient = 4.05) and an inverse relation with ΔPbB (regression coefficient = -0.22), thus indicating that the primary effect measured could be an effect of reduced concentrations of ambient CO. However, the expected inverse relation of ΔHt with a decrease in the concentration of lead in blood was also found. It is surprising if it is so, that CO should be so dominating, since levels of CO (not measured) would only have been high during weekends and mostly during the summer season. The suggestion that sporadic high concentrations of CO may influence hematocrit is unexpected. It is equally unexpected to find a negative relation of blood lead with hematocrit at such low blood lead concentrations as measured in this study.

The positive correlation between age and ΔHt indicates that there were larger declines in hematocrit in the elderly than in the young (see Figure 8). It is possible that the elderly were more exposed to CO than the young. The home for the elderly in Holmestrand was situated adjacent to the freeway in a rather pronounced city canyon.

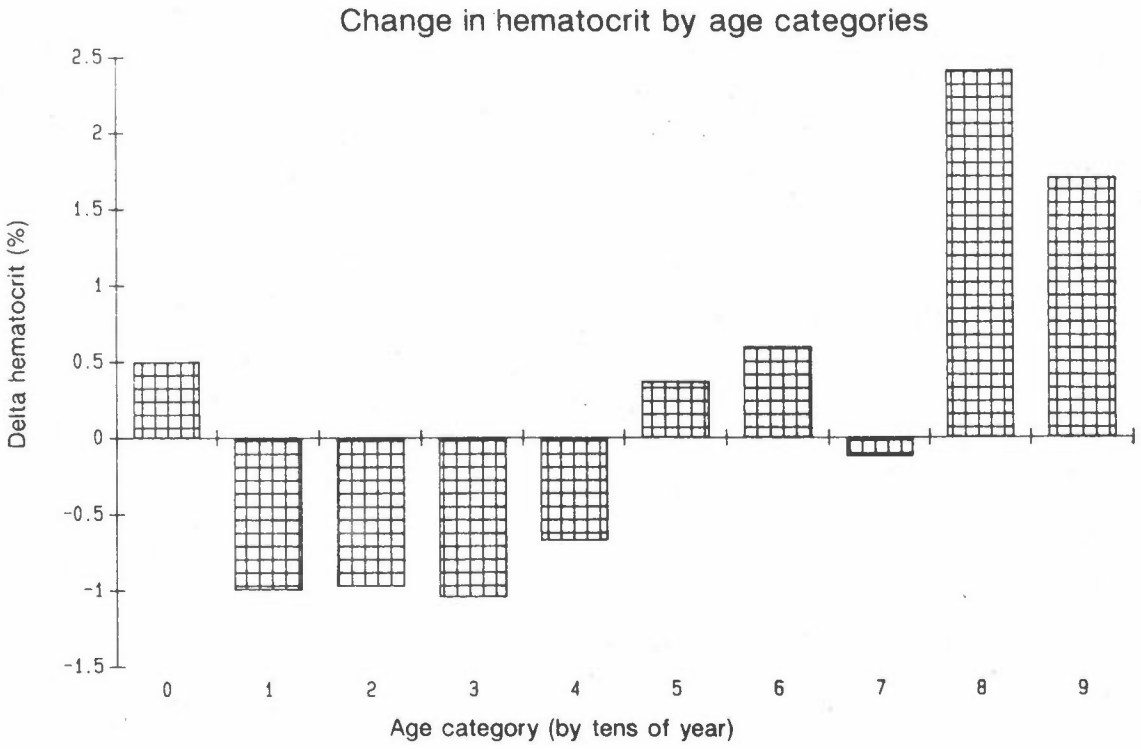

Figure 8: Changes in hematocrit (%) as a function of age (10 year age categories) in the inhabitants of Holmestrand and Sørumsand.

3.6 VALUES OF ZINC PROTOPORPHYRIN MEASURED IN 1984

High concentrations of lead in blood are known to influence heme metabolism and thus levels of zinc protoporphyrin (National Academy of Sciences, 1972). Therefore, zinc protoporphyrin (ZPP) was measured in this study, both in 1983 and 1984. The resulting differences (ΔZPP) should reveal if blood lead concentrations in 1983 were high enough to cause a measurable effect on the heme system. It is important to note, however, that ZPP levels also increase when hematocrit is very low (see Figure 7).

Children

A significant and negative relationship between zinc protoporphyrin (ZPP) and hematocrit is observed and is related to iron deficiency. This is fairly well known. There is, however, no indication of an

effect of blood lead at the concentrations measured in 1984 on ZPP concentrations or thus on the heme system.

An interesting and unreported significant difference between children exposed to passive smoking and children not exposed to passive smoking in ZPP concentrations is observed. Children exposed to passive smoking have higher levels of ZPP. This relationship should be examined more carefully (Table 10).

Adult

The same negative and known relationship between hematocrit and ZPP seen in children is also observed in adults (Table 10).

Again, not previously reported, are significantly higher values of ZPP in smokers than in non-smokers.

3.7 DIFFERENCES IN ZINC PROTOPORPHYRIN CONCENTRATIONS

The histogram of differences in zinc protoporphyrin concentrations between 1983 and 1984 is shown in Appendix 2.

Children

There were no significant relationships found between differences in ZPP concentrations measured in 1983 and 1984 and either covariates or factors (Table 11).

Adult

A significant effect ($p \leq 0.01$) of sex is found with concentrations of ZPP in males lower than those measured in females. This, however, may simply reflect that ZPP values were standardized to a hematocrit of 45%. Men have higher levels of hematocrit than women and children.

Among covariates, ΔPbA and ΔHt were significantly ($p \leq 0.05$) related to ΔZPP, but ΔPbA positively and ΔHt negatively. The correlation between ΔZPP and ΔHt is as expected. It seems plausible that the correlations between ΔZPP and ΔPbA in fact reflects the correlation of ΔZPP with hematocrit (as mentioned above, values of ZPP are standardized to a Ht of 45%) since ΔPbA is also positively correlated with ΔHt.

4 DISCUSSION

The primary aim of this investigation was to quantify the relationship between blood lead concentration and air lead concentration such that the question "How much can we expect blood lead concentrations to decrease if we decrease air lead concentrations by 1 $\mu g/m^3$?" was answered. To do this it was necessary to refine and improve the measurement of exposure to air lead to account for individual differences in time spent in microenvironments that can vary significantly in concentrations of lead. For example, some people spend much more time indoors where concentrations are generally low, whereas other people are more out walking near roads with heavy traffic or driving cars in rush hour traffic.

Therefore, this study was designed as a cohort study where each individual was measured twice, once before the opening of a tunnel that removed most of the traffic from a town that up until then could be considered moderately exposed, and again one year later when the air was considered relatively clean. Each individual´s personal exposure to air lead was quantified each year using information provided through a diary together with concentrations of lead in air measured or estimated in different geographic areas of the town. Measurements in a control town were performed to eliminate possible differences in methodology between the two years. Results from the studies of concentrations of lead in blood and air before the tunnel was opened have previously been reported (Clench-Aas et al., 1984).

This investigation confirmed the finding earlier reported by the same research team (Clench-Aas et al., 1984) that the ratio of blood lead to air lead concentrations is greater than the 1 to 2 $\mu g/dl$ to 1 $\mu g/m^3$

reported in the literature. We found a ratio of 4 to 1 for the entire population.

In 1984, similar to the findings of 1983, we found higher concentrations of lead in blood in adult women than men and slightly higher in women smokers than in non-smokers. We did confirm the earlier reported expectations of a greater decline in blood lead concentrations in children exposed for passive smoking. After we reported the indicated relationship between blood lead concentrations and passive smoking in children in 1984, a Swedish team found the same phenomenon in areas surrounding a lead smelter (Willers et al., 1988). We earlier reported that the relationship between smoking in adults and blood lead concentrations was possibly reflecting higher uptake through the lung by smokers as a results of damage by cigarette smoke to the lung. This idea has been reinforced by the reported higher uptake of CO through the lungs of smokers due to altered pulmonary epithelial permeability in man (Jones et al., 1983), and altered tracheal mucous transport velocity in smokers (Goodman et al., 1978). It would be of interest whether the same mechanism is in effect in children exposed to passive smoking. Since we had measured higher absorption (steeper slope between blood and air lead concentrations) in these two population subgroups, we expected them to decline more after the removal of the air lead source.

An interesting finding is that those individuals who consume alcohol at least once a week did not reduce their blood lead concentrations as much as non-drinkers. Alcohol consumption was also positively related with blood lead concentrations in 1984 (alcohol consumption not measured in 1983). This seems to indicate metabolic changes connected to alcohol consumption. Increased lead absorption has been reported with increased alcohol consumption, and is related to alterations in the intestinal barrier caused by ethanol (Moore, 1986).

It was surprising that blood lead concentration had a negative relationship with hematocrit since the concentrations of blood lead measured in this study could be considered rather low.

An interesting relationship between passive smoking in children and smoking in adults on concentrations of zinc protoporphyrin are

described (higher enzyme levels in smokers or passive smoking exposed children). This result should be further studied, possibly in animals.

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We mostly, however, want to thank our volunteers - people of all ages, from little children of 2 to the elderly up to 98 years old. They willingly came, filled out a long and cumbersome questionnaire two years in a row, offered their blood and were always pleasant and smiling.

APPENDIX 1

Air lead values in Holmestrand and Sørumsand
in 1983 and 1984

Table 1-1: Measured and estimated air lead concentrations ($\mu g/m^3$) in Holmestrand used in calculating individual air lead exposure in 1983 and 1984.

Date	HOLMESTRAND 1983 Station 1	Station 2	HOLMESTRAND 1984 Station 1	Station 2
April				
11	0.03	0.10		
12	0.08	0.14		
13	0.07	0.32		
14	0.08	0.29		
15	0.23	0.33		
16	0.05	0.31		
17	0.10	0.42		
18	0.18	0.33	0.04	0.04
19	0.11	-	0.05	0.07
20	0.05	-	0.10	0.03
21	0.10	-	0.06	0.09
22	0.12	-	0.04	0.02
23	0.06	-	0.06	0.07
24	0.16	-	0.07	0.07
25	0.25	-	0.06	0.08
26	0.06	0.19	0.07	0.05
27	0.08	0.16	0.11	0.10
28	0.15	0.14	0.04	0.06
29	0.21	0.29	0.05	0.07
30	0.20	0.30	0.12	0.09
May				
1	0.27	0.42	0.03	0.06
2	0.15	0.23	0.03	0.04
3	0.15	0.20	0.05	0.09
4	0.16	0.25	0.06	0.09
5	0.12	0.38	0.02	0.04
6	0.20	0.29	0.01	0.03
7	0.11	0.25	0.01	0.01
8	0.08	0.23	0.03	0.05
9	0.12	0.35	0.02	0.05
10	0.15	0.21	0.04	0.06
11	0.20	0.27	0.04	0.06
12	0.14	0.38	0.02	0.06
13	0.08	0.34	0.03	0.05
14	0.11	0.10	0.07	0.07
15	0.04	0.17	0.03	0.05
16	0.06	-	0.05	0.09
17	0.08	0.19	0.05	0.08
18	0.06	0.09	0.04	0.09
19	0.05	0.14	0.04	0.07
20	0.05	0.18	0.05	0.06
21	0.05	0.11	0.06	0.04
22	0.04	0.18	0.07	0.01
23	-	-	-	0.03

Table 1-2: Measured and estimated air lead concentrations ($\mu g/m^3$) in Sørumsand used in calculating individual air lead exposure in 1983 and 1984.

	SØRUMSAND 1983		SØRUMSAND 1984	
Date	Station 1	Station 2	Station 1	Station 2
April				
14	0.01	0.01		
15	0.04	0.03		
16	0.02	0.02		
17	0.05	0.06	0.02	0.02
18	0.04	0.04	0.03	0.01
19	0.03	0.02	0.05	0.02
20	0.05	0.04	0.74	0.03
21	0.03	0.02	0.04	0.02
22	0.03	0.04	0.02	0.01
23	0.03	0.04	0.04	0.02
24	0.04	0.06	0.04	-
25	0.04	0.09	0.04	-
26	0.04	0.03	0.03	-
27	0.04	0.04	0.03	-
28	0.02	0.03	0.02	-
29	0.03	0.03	0.02	-
30	0.04	0.04	0.03	-
May				
1	0.04	0.06	0.04	0.03
2	0.04	0.04	0.03	0.01
3	0.03	0.05	0.04	0.01
4	0.03	0.04	0.12	0.01
5	0.02	0.05	0.01	0.01
6	0.04	-	0.01	0.01
7	0.02	-	0.01	0.01
8	0.01	-	0.02	0.01
9	0.02	-	0.01	0.01
10	0.01	-	0.02	0.03
11	0.02	-	0.02	0.01
12	0.02	-	0.02	0.01
13	0.01	0.02	0.01	0.02
14	0.01	0.01	0.03	0.04
15	0.03	0.02	0.03	0.01
16	0.02	0.02	-	-
17	0.02	0.02	-	-
18	0.02	0.02	-	-
19	0.01	0.02	-	-

APPENDIX 2

Frequency distributions of
ΔPbB, ΔPbA, ΔZPP and ΔHt

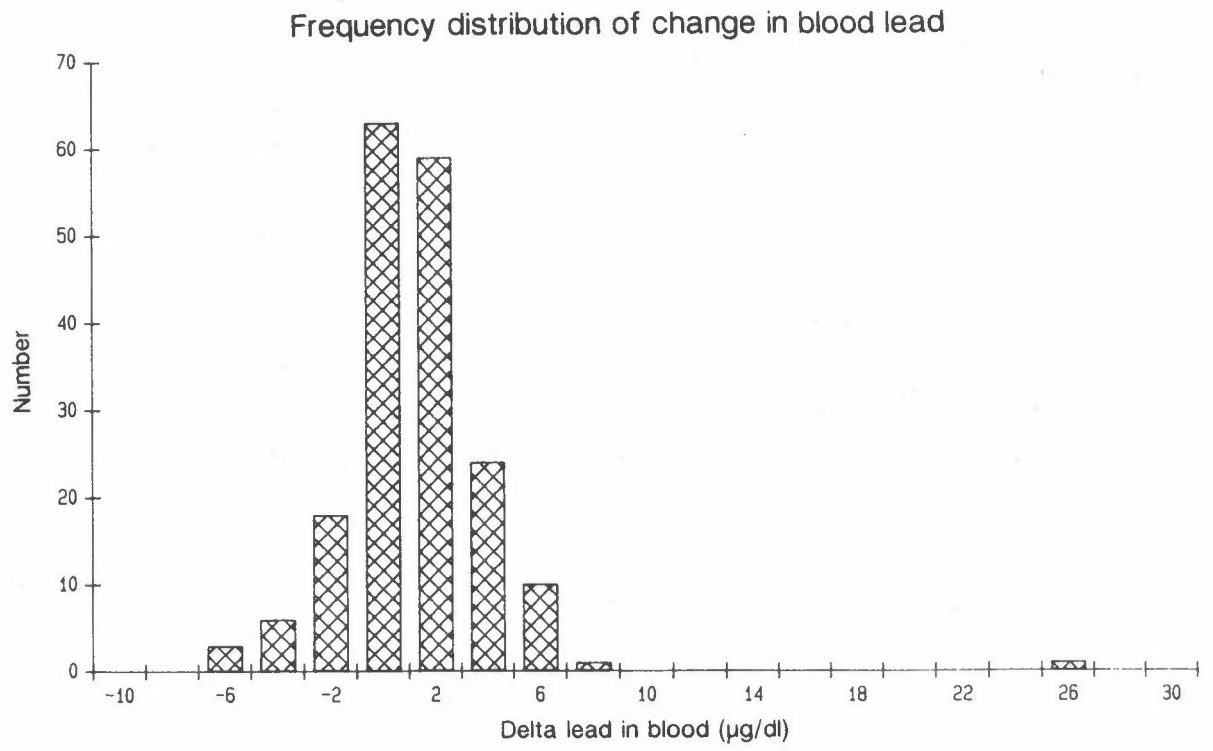

Figure 2-1: The frequency distribution of the changes in blood lead concentration (Δ lead in blood in µg/dl).

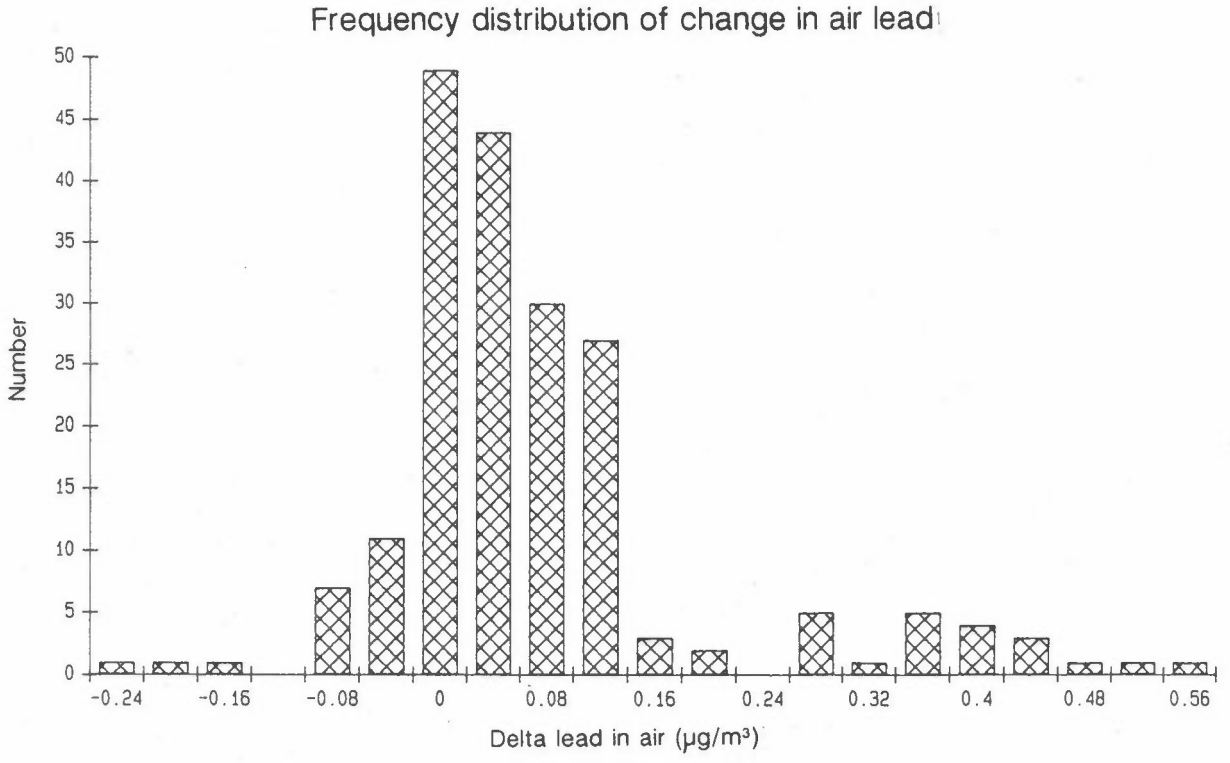

Figure 2-2: The frequency distribution of the changes in exposure to air lead concentrations (Δ lead in air in $\mu g/m^3$).

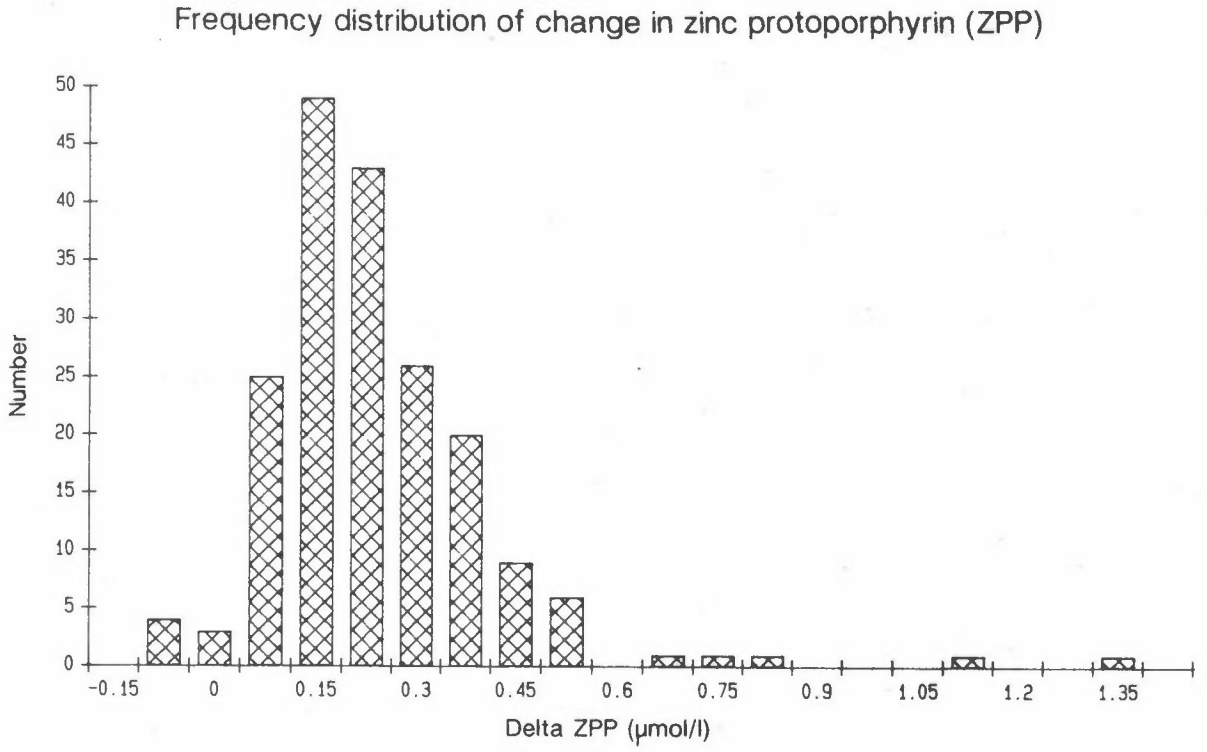

Figure 2-3: The frequency distribution of the changes in zinc protoporphyrin (ΔZPP in µmols/l).

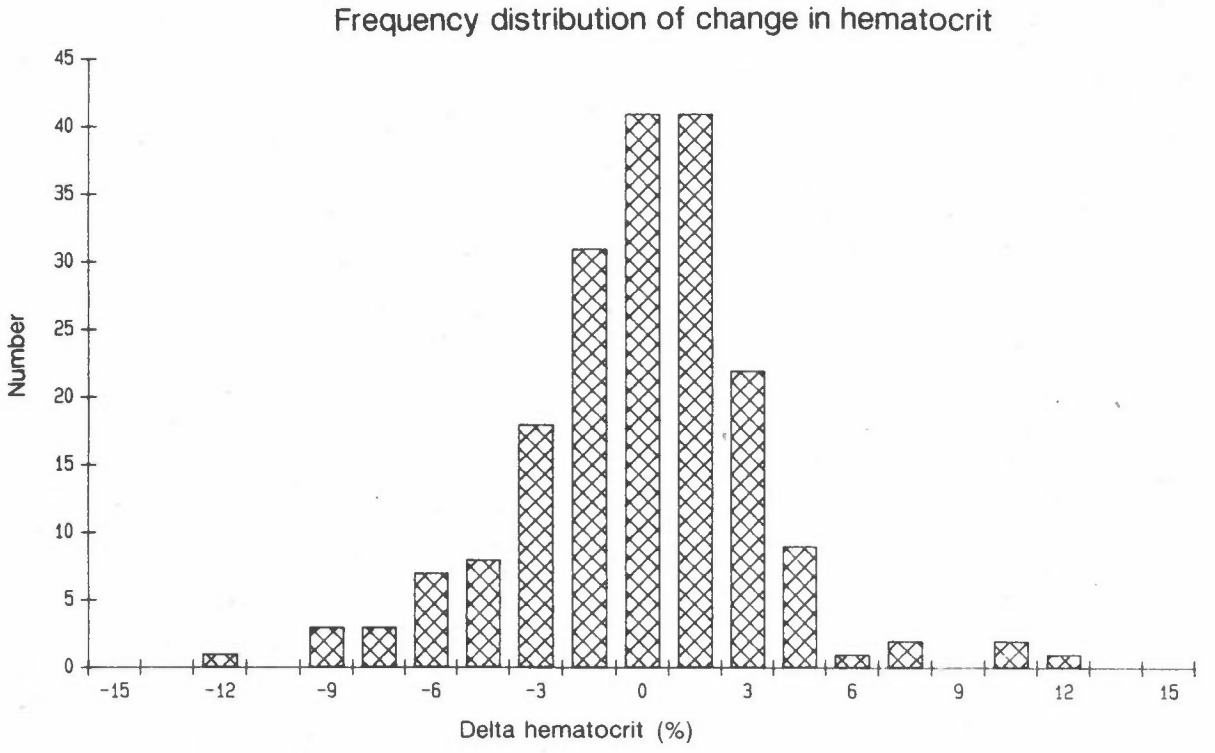

Figure 2-4: The frequency distribution of the changes in measured hematocrit (ΔHt in %).

APPENDIX 3

Tables showing
Δ PbB, Δ PbA, Δ ZPP and Δ Ht
as a function of socio-economic parameters

Table 3-1: Differences in air lead concentration and measured blood parameters between 1983 and 1984 with respect to sex.

	Holmestrand		Sørumsand	
	men	women	men	women
Δ PbA ($\mu g/m^3$)				
Valid N	58	90	41	63
Mean	0.12	0.12	0.01	0.00
Median	0.08	0.09	0.01	0.01
Std.dev.	0.13	0.13	0.04	0.06
Δ PbB ($\mu g/m^3$)				
Valid N	56	83	39	59
Mean	1.35	2.08	0.66	0.37
Median	1.80	1.66	0.47	0.52
Std.dev.	3.01	3.56	2.40	1.67
Δ Ht (%)				
Valid N	56	86	40	62
Mean	0.00	0.55	-1.11	-0.70
Median		0.75	-1.00	
Std.dev.	3.51	3.54	2.68	3.29
Δ ZPP (µmol/l)				
Valid N	56	86	39	62
Mean	0.21	0.29	0.18	0.22
Median	0.21	0.26	0.17	0.18
Std.dev.	0.15	0.21	0.10	0.14

Calculations for all parameters were made as 1984-1983 values.
PbA = Air exposure to lead; PbB = Blood lead concentrations;
Ht = Hematocrit; ZPP = Zinc protoporphyrin.

Table 3-2: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Holmestrand with respect to smoking habits in women.

Holmestrand	Current smoking			
	never	prev	occas	smoke
Δ PbA ($\mu g/m^3$)				
Valid N	52	7	2	29
Mean	0.10	0.09	0.11	0.16
Median	0.08	0.06	0.11	0.09
Std.dev.	0.11	0.16	0.01	0.15
Δ PbB ($\mu g/m^3$)				
Valid N	48	6	2	27
Mean	1.95	0.20	-0.72	2.92
Median	1.72	0.83	-0.72	1.99
Std.dev.	1.80	1.14	3.81	5.57
Δ Ht (%)				
Valid N	50	7	2	27
Mean	0.55	1.36	2.00	0.24
Median	0.50	2.00	2.00	
Std.dev.	2.71	2.56	0.71	5.03
Δ ZPP ($\mu mol/l$)				
Valid N	50	7	2	27
Mean	0.33	0.20	0.14	0.26
Median	0.30	0.16	0.14	0.24
Std.dev.	0.21	0.15	0.11	0.22

Table 3-3: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Holmestrand with respect to smoking habits in men.

Holmestrand	Current smoking		
	never	prev	smoke
Δ PbA ($\mu g/m^3$)			
Valid N	20	13	25
Mean	0.11	0.07	0.14
Median	0.07	0.08	0.11
Std.dev.	0.14	0.08	0.14
Δ PbB ($\mu g/m^3$)			
Valid N	18	13	25
Mean	2.25	1.70	0.52
Median	1.87	2.24	1.70
Std.dev.	3.02	2.67	3.06
Δ Ht (%)			
Valid N	18	13	25
Mean	0.03	0.35	-0.20
Median		0.50	-0.50
Std.dev.	4.67	1.13	3.47
Δ ZPP (µmol/l)			
Valid N	18	13	25
Mean	0.25	0.19	0.19
Median	0.24	0.15	0.17
Std.dev.	0.12	0.16	0.17

Table 3-4: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Sørumsand with respect to smoking habits in women.

Sørumsand	Current smoking			
	never	prev	occas	smoke
Δ PbA ($\mu g/m^3$)				
Valid N	35	15	6	7
Mean	-0.00	-0.00	0.01	0.03
Median	0.01	0.01	0.02	0.02
Std.dev.	0.07	0.05	0.04	0.03
Δ PbB ($\mu g/m^3$)				
Valid N	31	15	6	7
Mean	0.32	0.69	0.45	-0.16
Median	0.55	0.65	-.09	0.20
Std.dev.	1.91	1.30	1.26	1.64
Δ Ht (%)				
Valid N	34	15	6	7
Mean	-0.77	-1.83	1.92	-0.14
Median	-0.50	-1.00	2.25	1.00
Std.dev.	2.65	3.12	2.08	5.89
Δ ZPP (µmol/l)				
Valid N	34	15	6	7
Mean	0.23	0.23	0.18	0.19
Median	0.19	0.16	0.21	0.13
Std.dev.	0.15	0.15	0.12	0.15

Table 3-5: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Sørumsand with respect to smoking habits in men.

Sørumsand	Current smoking			
	never	prev	occas	smoke
Δ PbA ($\mu g/m^3$)				
Valid N	20	7	4	10
Mean	0.01	0.00	-0.03	0.04
Median	0.01	0.01	-0.02	0.02
Std.dev.	0.03	0.04	0.05	0.05
Δ PbB ($\mu g/m^3$)				
Valid N	18	7	4	10
Mean	1.03	0.70	-1.32	0.74
Median	0.82	0.85	-0.69	0.48
Std.dev.	2.55	1.54	3.39	2.14
Δ Ht (%)				
Valid N	19	7	4	10
Mean	-0.87	-1.00	0.13	-2.15
Median	-1.00	-0.50	-0.25	-2.00
Std.dev.	3.17	1.87	1.80	2.37
Δ ZPP ($\mu mol/l$)				
Valid N	18	7	4	10
Mean	0.19	0.18	0.13	0.17
Median	0.21	0.16	0.13	0.17
Std.dev.	0.08	0.17	0.02	0.07

Table 3-6: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Holmestrand in non-smokers with respect to passive smoking.

Holmestrand	men		women	
	passive smoking		passive smoking	
	no	yes	no	yes
Δ PbA ($\mu g/m^3$)				
Valid N	8	12	37	13
Mean	0.11	0.11	0.09	0.15
Median	0.07	0.07	0.08	0.10
Std.dev.	0.15	0.15	0.08	0.16
Δ PbB ($\mu g/m^3$)				
Valid N	7	11	35	11
Mean	1.61	2.66	1.89	2.35
Median	1.81	2.06	1.68	2.32
Std.dev.	1.88	3.59	1.79	1.97
Δ Ht (%)				
Valid N	7	11	35	13
Mean	1.76	-1.06	0.89	-0.50
Median	1.00	-1.00	1.00	-1.00
Std.dev.	6.17	3.30	2.86	2.26
Δ ZPP ($\mu mol/l$)				
Valid N	7	11	35	13
Mean	0.27	0.24	0.32	0.35
Median	0.27	0.21	0.27	0.37
Std.dev.	0.11	0.12	0.24	0.14

Table 3-7: Differences in air lead concentration and measured blood parameters between 1983 and 1984 in Sørumsand in non-smokers with respect to passive smoking.

Sørumsand	men		women	
	passive smoking		passive smoking	
	no	yes	no	yes
Δ PbA ($\mu g/m^3$)				
Valid N	16	3	21	11
Mean	0.02	-0.02	0.01	-0.02
Median	0.02	-0.02	0.01	0.01
Std.dev.	0.03	0.03	0.04	0.08
Δ PbB ($\mu g/m^3$)				
Valid N	14	3	17	11
Mean	0.67	2.28	0.34	0.25
Median	0.40	1.47	0.55	0.58
Std.dev.	2.28	4.13	1.37	2.45
Δ Ht (%)				
Valid N	15	3	20	11
Mean	-0.33	-4.00	-0.48	-0.65
Median	-1.00	-1.50		-0.50
Std.dev.	2.68	4.77	2.67	2.79
Δ ZPP ($\mu mol/l$)				
Valid N	14	3	20	11
Mean	0.22	0.12	0.23	0.22
Median	0.22	0.12	0.16	0.19
Std.dev.	0.07	0.11	0.18	0.09

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TITTEL
The effect of reducing air lead from vehicular sources on the blood lead concentrations in two Norwegian towns - A cohort study

PROSJEKTLEDER
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3 STIKKORD (à maks. 20 anslag)
Blood lead | air lead exposure | hematologic parameters

REFERAT (maks. 300 anslag, 7 linjer)
Bly i blod ble målt hos to befolkningsgrupper, den ene var eksponert for bly fra bilforurensninger mens den andre var eksponert for lave konsentrasjoner av bly i luft. Bly i blod ble målt hos den samme befolkningsgruppen både i 1983 før tunnelen i Holmestrand åpnet og igjen i 1984, ett år etter åpningen. Bly i blod var redusert mer enn antatt i litteraturen. Korrelasjonen mellom reduksjon i eksponering for bly fra luften og reduksjon i konsentrasjon av bly i blod var signifikant.

TITLE The effect of reducing air lead from vehicular sources on the blood lead concentrations in two Norwegian towns - A cohort study

ABSTRACT (max. 300 characters, 7 lines)
Blood lead was measured in two populations - one exposed to moderate lead emissions from traffic pollution and another a control low exposure town. Blood lead concentrations were measured in 1983 before the opening of a tunnel that removed the bulk of the traffic from the town and again one year later. Reductions in the concentrations of lead in blood were larger than predicted by factors given in the literature. There was a significant correlation between the reduction in exposure to lead in air and reductions in blood lead concentrations.

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