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LEAD IN BLOOD IN INHABITANTS OF OSLO-NYDALEN EXPOSED TO AIR LEAD FROM INDUSTRIAL AND VEHICULAR SOURCES

PART I

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SUMMARY

INTRODUCTION

In the spring of 1984, the Norwegian Institute for Air Research (NILU) in cooperation with the Institute of Occupational Health (YHI) studied blood lead concentrations in the inhabitants of Holmestrand (moderate traffic pollution) and Sørumsand (control town). Despite blood lead concentrations that are low to moderate as compared to values reported internationally, inhabitants of Holmestrand had higher blood lead values than were expected, especially in children exposed to passive smoking and smoking women (Clench-Aas et al., 1984). Therefore in 1984, NILU, YHI and the City Health Department of Oslo, conducted a study of the effects of higher ambient lead exposure on the blood lead concentrations of the inhabitants of the Oslo-Nydalen area. This area is one of the more highly lead exposed regions in Norway having two industrial point sources and crossed by a major traffic throughway. It was necessary to compare blood lead concentrations in the inhabitants of Oslo-Nydalen, with individuals that are not exposed to high amounts of ambient lead. Therefore, values from a similar study in Sørumsand, done in May 1984, were used as a control.

METHODOLOGY

The study was conducted in two sites:

- Oslo-Nydalen a part of Oslo traversed by a major throughway (ca. 30 000 vehicles daily) and having two point sources of industrial lead emissions.
- 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.

In Oslo-Nydalen, 470 people (ranging in age from 2 to 98 years; 186 children: 125 men; and 159 women) volunteered for the study. In Sørumsand, 118 (ranging in age from 3 to 91 years; 24 children; 28 men; and 55 women) volunteered.

One of the unique features of this study was its experimental design. For each individual a specific blood lead concentration was related to an estimate of that individual's own exposure to ambient lead during the two weeks prior to blood sampling.

Individual air lead exposure was estimated by combining information from diaries of weekly patterns of activity (hours per day for each day of the week, spent in each of several microenvironments such as indoor at home, indoor at work or school, outdoors at home, etc.) with both measured and estimated ambient lead concentrations. Blood and air lead for each individual was measured by electrothermal atomic absorption spectrometry. Zinc protoporphyrin concentrations were measured since they have been reported to be increased by higher concentrations of blood lead. In addition, the hematologic variables hemoglobin, hematocrit and mean cell hemoglobin concentration were also measured. The questionnaire included information on: 1) additional lead exposure via hobbies, occupation, and smoking (both active and passive), and 2) other socio-economic parameters such as alcohol consumption, use of vitamins and iron supplements, etc. that could influence metabolism.

RESULTS AND DISCUSSION

Measured blood lead concentrations in Oslo-Nydalen were low when compared to those reported in the international literature. Despite higher amounts of lead in air in the Oslo-Nydalen area than those measured in Holmestrand in 1983 before the opening of the tunnel, concentrations of lead in blood were lower.

Ambient lead in the Oslo-Nydalen area ranged from 0.02 μ g/m³ to 5 μ g/m³ during the measuring period. Ambient lead at the five stations, situated where people who participated in the study lived, averaged for the month of

February: 0.2, 0.3, 0.3, 0.3, and 0.6 μ g/m³. These values can be compared to Sørumsand where ambient lead ranged from 0.01 to 0.04 μ g/m³ during the sampling period (May 1984).

Concentrations of lead in blood in Oslo-Nydalen averaged 6.0 μ g/100 ml in children, 5.2 μ g/100 ml in women and 5.7 μ g/100 ml in men. These compare to those values measured in Sørumsand, 3.8 μ g/100 ml in children, 3.4 μ g/100 ml in women and 5.9 μ g/100 ml in men.

The following table summarizes the principal findings of the study by comparing the median blood lead concentration in children, women and men in Oslo-Nydalen and Sørumsand. The median value of the air lead exposure estimate is also given.

1	CHILD	DREN	WON	MEN	MEN			
		Blood lead concentration		Blood lead concentration		Blood lead concentration		
ю 4	µg/m ³	µg/100ml	µg/m ³	µg/100ml	µg/m ³	µg/100ml		
Oslo-Nydalen	0.21	6.0	0.19	5.2	0.21	5.7		
Sørumsand	0.03	3.8	0.04	3.4	0.05	5 9		

Single regression analysis between the logarithms of blood lead versus air lead exposure estimates, resulted in significant correlations in women and children (both boys and girls), but not in men. The measured slope (β) of the regression agreed quite closely with those values of β reported in the literature. It has been reported in the literature that a decrease in the ambient concentrations of lead of 1 µg/m³ would result in decreases in blood lead concentrations between 1 and 2 µg/100 ml. The regressions done in this study predict decreases in blood lead concentrations of 1.2 µg/100 ml in boys, 1.3 µg/100 ml in girls, and 0.9 in women with a 1 µg/m³ decline in ambient lead levels.

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The results found in this study in Oslo-Nydalen are different from those reported earlier for Holmestrand in 1983. Despite exposure to higher air lead concentrations in the Oslo-Nydalen area than those found in Holmestrand, blood lead concentrations were lower. For example, whereas children in Oslo-Nydalen had an average of 6.0 µg/100 ml lead in blood, in Holmestrand the average was 9.6 µg/100 ml. Similarly in women, the average in Oslo-Nydalen was 5.2 µg/100 ml whereas in Holmestrand the average was 6.7 μ g/100 ml. Even in men, averages were 8.4 μ g/100 ml in Holmestrand as opposed to 5.7 µg/100 ml in Oslo-Nydalen. Calculated regression coefficients in the earlier Holmestrand study indicated greater reductions in blood lead with a $1 \mu g/m^3$ reduction in air lead than those measured in Oslo-Nydalen or those reported in the literature.

The hypothesis is put forth in this report, that the findings in the two studies differ due to differences in the sources of ambient lead in the two towns. In the Oslo-Nydalen area, the primary source of high ambient lead are the industrial sources, while in Holmestrand the only source is traffic pollution. This could be verified by comparing ambient lead and cadmium values in Oslo-Nydalen with those values found in Sørumsand and Holmestrand. It is suggested that lead is concentrated on smaller size particles when the lead source is traffic emissions than when lead is emitted from the industrial sources in the area. The result could then be that smaller size particles can penetrate further into the alveolar region of the lung leading to greater absorption. Another hypothesis is that the chemical form of the lead may be different in the two sources leading to different absorption factors in the two geographic areas. Yet a third hypothesis, is that the pattern of emissions when the sources are industrial or traffic are different. For example, lead concentrations from industrial sources can be very high, but only over a short time-span, whereas vehicle exhaust may result in lower ambient lead concentrations but over a longer time-span. It is possible that such differences in emission patterns can in some way affect absorption.

In the earlier Holmestrand study, passive smoking in children and smoking in adult women seemed to increase the uptake of lead in air by these two population subgroups. These findings were not confirmed in this study.

In addition to directly examining the relationship between the concentrations of air lead and blood lead, this study also studied the relationship of other socio-economic parameters to blood lead concentrations using multiple step-wise regression. In children there was a significant difference between the two sexes, with female children having the lower blood lead concentrations. In adults, sex, age, social class, and alcohol consumption were significantly related to blood lead concentration. These relationships were such that age and alcohol consumption increased the impact of air lead on concentrations of lead in blood.

Blood lead concentration, if high enough, has been shown to interfere with heme synthesis, altering such parameters as hematocrit, hemoglobin, and the enzyme zinc protoporphyrin. Since measured blood lead concentrations were not high, it was not surprising that there was no measurable effect of lead in blood on the parameters hemoglobin, hematocrit, mean cell hemoglobin concentration or zinc protoporphyrin.

SAMMENDRAG

INNLEDNING

Våren 1983 gjennomførte Norsk Institutt for Luftforskning (NILU) i samarbeid med Yrkeshygienisk Institutt (YHI) en undersøkelse av blykonsentrasjoner i blodet hos innbyggere i Holmestrand og Sørumsand. Holmestrand ble valgt pga trafikkbelastning, og Sørumsand fungerte som kontrollområde. Konsentrasjonene av bly i blodet hos befolkningen i Holmestrand var lave til moderate når man sammenligner disse med resultater fra andre undersøkelser utenfor Norge. Blykonsentrasjonene var likevel høyere enn forventet ut fra belastningen. Spesielt gjaldt dette røykende kvinner og barn som var utsatt for passiv røyking (Clench-Aas et al., 1984). Med bakgrunn i denne undersøkelsen gjorde derfor NILU og YHI sammen med Oslo helseråd en ny undersøkelse våren 1984. Denne gangen ble effekten av en høyere blyeksponering enn i Holmestrand undersøkt hos innbyggere i Oslo-Nydalen området. Området er blant de mer belastede i Norge på grunn av to industrielle punktkilder for bly samt en gjennomfartsvei med stor trafikk. Det ble også utført målinger i Sørumsand i mai 1984 og resultatene derfra er brukt som kontroll.

METODIKK

Undersøkelsen ble utført på to lokaliteter:

- Oslo-Nydalen En bydel i Oslo med en gjennomfartsvei med stor trafikk (ca 30 000 kjøretøyer i døgnet). I området finnes også to punktkilder med industrielle blyutslipp.
- Sørumsand Et tettsted med lite trafikk (ca 3000 kjøretøyer i døgnet i undersøkelsesperioden) og ingen industrielle kilder til bly.

I Oslo-Nydalen og i Sørumsand deltok henholdsvis 470 personer (alder fra 2 til 98 år; 186 barn, 125 menn og 159 kvinner) og 118 (alder fra 3 til 91 år; 24 barn, 28 menn og 55 kvinner) i undersøkelsen.

Et svært spesielt trekk ved denne undersøkelsen er at blykonsentrasjon i blodet hos hver enkelt deltager ble sammenstilt med samme deltagers beregnede eksponering for bly i luft de siste to ukene før blodprøven ble avgitt.

Den siste uken før blodprøven besvarte hver deltager et skjema med spørsmål om aktiviteter og livsførsel. Den individuelle eksponering for bly i luft ble beregnet på grunnlag av denne informasjon om aktivitetsmønsteret samt målte og estimerte mengder av bly i uteluft. Blyinnholdet i blod og luft ble målt ved hjelp av atomabsorpsjonspektrometri. Blodparametrene hemoglobin, hematokrit, gjennomsnittlig hemoglobinkonsentrasjon i røde blodlegemer og sink protoporphyrin ble målt. Spørreundersøkelsen gav også informasjon om annen eksponering for bly gjennom arbeid, hobbyer og røyking (både aktiv og passiv). I tillegg var det samlet informasjon om alkoholforbruk, inntak av vitaminer og jernsupplementer som kan influere på metabolismen av bly.

RESULTATER OG DISKUSJON

Alle de målte blykonsentrasjonene i blod var lave til moderate når de sammenlignes med internasjonale verdier. På tross av at nivåene av bly i luften i Oslo-Nydalen området var høyere enn i Holmestrand i 1983 før åpningen av tunnelen, var konsentrasjonene av bly i blod lavere.

Bly-nivået i luft i Oslo-Nydalen området varierte fra 0.02 μ g/m³ til 5 μ g/m³ i måleperioden. Gjennomsnittverdier for de 5 stasjonene i området hvor deltagerne bodde var i februar måned: 0.2, 0.3, 0.3 og 0.6 μ g/m³. Disse verdiene kan sammenlignes med gjennomsnittverdiene i Sørumsand, som var fra 0.01 til 0.04 μ g/m³ i mai 1984.

Konsentrasjonene av bly i blod i Oslo-Nydalen var i gjennomsnitt 6.0 μ g/100 ml hos barn, 5.2 μ g/100 ml hos kvinner og 5.7 μ g/100 ml hos menn. Til sammenligning var de målte verdier i Sørumsand: 3.8 μ g/100 ml hos barn, 3.4 μ g/100 ml hos kvinner og 5.9 μ g/100 ml hos menn.

En oppsummering av resultatene er gitt i den etterfølgende tabell. Tabellen viser en sammenstilling av medianverdiene for blykonsentrasjonene i blod hos barn, kvinner og menn i Oslo-Nydalen og i Sørumsand i 1984. I tillegg er medianverdiene for lufteksponering også gitt.

	BAR	N	KVINNE	R	MENN		
1	Luft bly eksponerin	8lod bly g kons.	Luft bly eksponering	Blod bly kons.	Luft bly eksponering	Blod bly kons.	
	µg/m ³	µg/100ml	µg/m ³	µg/100ml	µg/m ³	µg/100ml	
Oslo-Nydalen	0.21	6.0	0.19	5.2	0.21	5.7	
Sørumsand	0.03	3.8	0.04	3.4	0.05	5.9	

Enkel regresjonsanalyse mellom logaritmene av blykonsentrasjoner i blod og de estimerte eksponeringene for bly i luft, gav signifikante korrelasjoner hos kvinner og barn (både gutter og jenter), men ikke hos menn. Den målte vinkelkoeffisient (β), stemte godt med den som er rapportert i litteraturen fra tilsvarende undersøkelser. I litteraturen har det vært rapportert at en reduksjon i luftkonsentrasjonen av bly på 1 µg/m³ fører til en nedgang på mellom 1 og 2 µg/100 ml i blodet. Denne undersøkelsen viser en nedgang på 1.2 µg/100 ml hos gutter, 1.3 µg/100 ml hos jenter og 0.9 µg/100 ml hos kvinner når bly i luft senkes med 1 µg/m³.

Resultatene i denne undersøkelsen avviker imidlertid fra Holmestrandresultatene, 1983. Eksponering for bly i luft i Oslo-Nydalen er høyere enn i Holmestrand, men likevel var blyinnholdet i blodet lavere. Mens barna i Oslo-Nydalen hadde gjennomsnittlig 6.0 µg/100 ml i blodet, var tilsvarende verdi i Holmestrand 9.6 µg/100 ml, hos kvinner var gjennomsnittet i Oslo-Nydalen 5.2 µg/100 ml mot 6.7 µg/100 ml i Holmestrand. Selv hos menn var gjennomsnittsverdiene henholdsvis 5.7 og 8.4 µg/100 ml. Resultatene av regresjonsanalysen fra Holmestrandundersøkelsen tyder på at en reduksjon av blyinnholdet i luft på 1 µg/m³ vil resultere i en større senkning av blykonsentrasjonen i blodet enn den man har beregnet i Oslo-Nydalen eller som er beskrevet i litteraturen tidligere.

En mulig forklaring på de forskjeller man har funnet mellom resultatene i de to områder, er at kildene til bly i luft er forskjellige. I Oslo-Nydalen kommer en stor del fra industrielle kilder, mens bileksos er eneste betydelige kilde til bly i Holmestrand. Dette kunne bekreftes ved å. sammenligne forholdet mellom nivåene av bly og kadmium i luften i Oslo-Nydalen, med de samme forholdene i Sørumsand og Holmestrand. Kadmium er hovedsakelig en industriforurensning og vil være en god indikator for bidraget av bly fra industrien i forhold til trafikken. Det er mulig at bly befinner seg på langt mindre partikler i utslipp fra bileksos enn når det stammer fra industrielle kilder. Dersom dette er riktig vil en få forskjeller i opptaket. Små partikler har større penetrasjon helt inn i alveoli i lungene og kan derfor ha større opptak i blodet. Det kan også være mulig at den kjemiske form av bly er forskjellig i de to typer kilder, og at dette fører til forskjell i absorpsjon. Enda en tredje hypotese er at utslippene fra de forskjellige kilder ikke varierer etter samme mønster. Blykonsentrasjonene kan for eksempel i industrielle utslipp være svært høye, men bare over kort tid, mens bileksos fører til lavere blynivå i luften, men over et lengre tidsrom. Det er mulig at slike variasjoner i utslippsmønstrene kan ha en effekt på blyopptak i lungene.

I den tidligere Holmestrand-undersøkelsen syntes passiv røyking hos barn og røyking hos voksne kvinner å øke i blyopptaket fra luft. En slik effekt ble ikke bekreftet av denne undersøkelsen.

I tillegg til en direkte sammenstilling av bly i blod og bly i luft ble virkningen av sosio-økonomiske variable analysert ved å gjøre en trinnvis multippel regresjonsanalyse. Resultatene fra gruppen barn viste en signifikant forskjell mellom kjønnene. Jenter hadde de laveste blykonsentrasjonene i blodet. Hos voksne ble det funnet at kjønn, alder, sosial klasse, alkoholforbruk og graden av eksponering for bly i luft, hadde signifikant sammenheng med blod/bly-konsentrasjonen. Opptak av bly fra luften syntes å øke med alder og alkoholforbruk.

Det er vist at dersom konsentrasjonen av bly i blod blir tilstrekkelig høy, vil blyet virke inn på kroppens hem-synteser. Dette gjelder variable som hemoglobin, hematokrit og sink protoporphyrin. Alle de målte konsentrasjonene av bly i blod var lave sammenlignet med resultatene fra andre undersøkelser, og det var derfor ikke overraskende at det ikke ble påvist effekter av bly i blod på parametre som hemoglobin, hematokrit og sink protoporphyrin i denne undersøkelsen.

FOREWORD

This report summarizes an investigation done in 1984 by the Norwegian Institute of Air Research (NILU) in collaboration with the Institute of Occupational Health and City Health Department, Oslo, of blood lead concentrations in the inhabitants of the Nydalen area in Oslo. Concentrations of blood lead for the control town, Sørumsand, were taken from a study done in 1984.

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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LEAD IN BLOOD IN INHABITANTS OF OSLO-NYDALEN EXPOSED TO AIR LEAD FROM INDUSTRIAL AND VEHICULAR SOURCES

1 INTRODUCTION

There has been much recent 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 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 (2500 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, 11000 vehicles daily) was stopped by a traffic light. The light caused traffic to back up, especially noticeable during the summer and 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 concentrations 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.

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 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 (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 in women 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 values was ca 6.4 μ g/100 ml (0.31 μ mol/l) in adult men; 2.9 μ g/100 ml (0.14 μ mol/l) in adult women; and 2.5 μ g/100 ml (0.12 μ mol/l) in children.

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

The results of this first study suggested that it might be beneficial, using the exact same methodology, to measure blood lead concentrations in a central area of Oslo - Nydalen (Figure 1). This area has a major traffic throughway (ca. 30 000 vehicles daily) and two important industrial lead sources, a scrap iron smelter and a car battery factory. Earlier studies in the downtown Oslo area (Omang and Moseng, 1974) did not indicate unusually high blood lead levels.

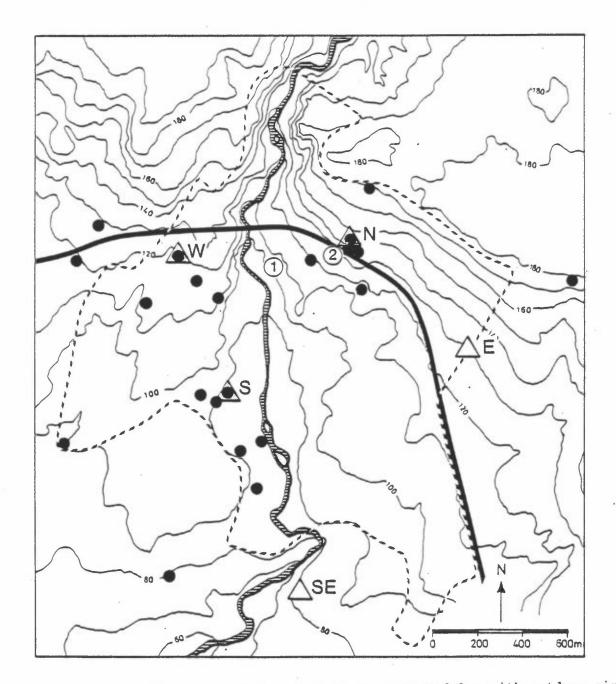


Figure 1: Topographical map of the study area, Oslo-Nydalen with outdoor air lead stations (△) and indoor air lead stations (●) indicated. Additional outdoor stations were operating in downtown Oslo as well. ① ② Point industrial lead sources - Highway 160 with approx.

(1) (2) Point industrial lead sources - highway too with appion. 30 000 vehicles daily.

-- Geographical limits within which volunteers' lived.

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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. 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 geographic differences in housing construction, culture and ambient temperature can result in large differences in 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 interior ventilation. Ignoring these can lead to problems in assessing the resulting actual air lead exposure of 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 average of a 24 hour day.

	Outdoor air lead concentration							
I/O Ratio	3 µg/m ³	2 µg/m ³	1 µg/m ³	.5 µg/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				0.7				
10% I/O	. 41	. 30	. 14	.07				
50%	1.56	1.04	.52	.26				
80%	2.42	1.50	.81	. 40				

 \star^1 Value calculated as: 6 hrs x outdoor value (3 $\mu\text{g/m}^3$) + 18 hrs x indoor value (=10% outdoor value) / 24 =

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

We define individual air lead exposure as the average daily air lead concentration each individual is exposed to. As can be seen in Table 1 a 10% I/O (as can be found in homes with air conditioner (Stock et al., 1983) with an outdoor lead concentration of 3 μ g/m³ 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 μ g/m³ 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 concentrations. 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 as close as possible to our previous 1983 Holmestrand study, but differently from previous studies in several ways:

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

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

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

2.2 <u>CHOICE OF SUBJECTS</u>

2.2.1 Subject selection

Based on the interesting findings of the earlier study the intention in the Oslo-Nydalen study, was to maximize participation of children. The local schools provided lists of all children living in the chosen study area.

Families were contacted by mail and requested to participate as a family. A first recruiting letter was sent to 454 families. Of these 94 (21%) responded to the first letter. A second reminder letter, was sent to the remaining 360 families who had not answered during the first round. Of these 104 (28%) answered. Of the total 198 (44%) families answering 166 (37%) said yes and 32 (7%) no.

In addition three pre-school facilities in the area were contacted and three clinics for the elderly (sykehjem).

The total number of participants was 470, grouped as shown in Table 2.

Typical for this kind of study, the selection was not random since it was of course based on voluntary participation. One becomes aware most notably of a prevalence of people in the health fields, people possibly more interested in their own health (joggers and so forth) and to the contrary people having been quite sick at some point in their lives and therefore used to blood sampling. There was a noticeable lack of families employed in the local industries despite local industrial support of the study.

The 1984 Sørumsand study is used as a control. The method of subject selection in Sørumsand was similar. Letters were sent to families having children, living near the outdoor samplers.

2.2.2 Population characteristics

A total of 470 individuals volunteered for the study from Oslo-Nydalen, 182 children (105 boys and 77 girls), and 288 adults (129 men and 159 women).

The control (low lead exposure) town Sørumsand measured in 1984 had a total of 107 individuals, 24 children (14 boys, 10 girls) and 83 adults (28 men and 55 women). Table 2 compares various population characteristics of the inhabitants of the two regions.

Table 2: Population characteristics of the two towns where blood and air lead concentrations were measured - Oslo-Nydalen (moderate air lead levels originating primarily from industrial sources) and Sørumsand (low air lead concentrations - control area).

			Oslo-Nydalen	Sørumsand
Sample	size		470	107
Age rar	ige		2 - 98 years	3 - 91 years
Numbers	of:		male, female	male, female
Childre	en (2-	-9 yrs)	57 42	7 6
Childre		-	52 35	7 4
Adults	(16-6	57)	109 136	26 50
Pension	ists	(>67)	16 23	2 5
Socio-e	conor	nic factor	5 (Percentage of	population)
Social	Class	5 A*	27%	52%
60		В	29%	17%
88	88	C	16%	20%
н	86	D	0.05%	4%
88	84	E	0%	5%
those o	n puk	olic		
assista	nce	F	-	3%

* Definition of social class divisions is given in Appendix II. People in homes for the elderly excluded because of unknown occupation.

2.3 ESTIMATION OF AIR LEAD EXPOSURE

The diary method was used to estimate individual air lead exposure. This method combines information from 3 different sources. Outdoor fixed site measurements, portable indoor measurements and a diary where questions are asked pertaining to time spent in each of the microenvironments (e.g. indoor home, indoor school, outdoor school). An estimate of air lead exposure was calculated for each of the 14 days prior to blood sampling. A mean of these 14 exposure estimates of air lead concentration (in μ g/m³) was calculated for each individual.

2.3.1 Fixed outdoor stations

A total of four fixed low volume samplers were placed for this study in the Nydalen region, in addition to using four already existing stations in Oslo (one near the study area and three downtown). Thus, 8 outdoor stations were used for estimating air lead exposure during the experimental period, February 1984, in Oslo, (Figure 1). In addition, two fixed low volume samplers were used in Sørumsand in May 1984. 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 five Oslo-Nydalen stations were placed in different directions from both the industrial sources and the highway, yet in areas where people lived. For simplicity they are referred to as N, W, S, E and SE (Figure 1). The other three sites were in downtown Oslo (two with high traffic, and one city background station). In Sørumsand, the sites were placed in areas where most of the volunteers lived.

Inhalable particulate bound-lead (< 10μ m) was collected on Whatman 40 cellulose fiber filters by the low-volume sampler. The low-volume sampler collects particles whose largest diameter varies from 10 to 20 μ m dependent on wind velocity. However 10 μ m is a more reliable figure based on the construction of the sampler. The filter determines the smallest diameter. The filter used allows 80% efficiency of recovery for particles 0.1 μ m or less.

Lead on the filters was determined at the Norwegian Institute of Air Research by electrothermal atomic absorption spectrometry (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 μ gPb/l which corresponds to 0.003 μ g Pb/m³ for the outdoor samples (10 ml extract, 3.5 m³ of air). The precision is about 5% at the 0.2 μ g Pb/m³ 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 analysis method.

Sample preparation

To cut pieces of the filter in polyethylene centrifuge tubes is added 1:1 HNO, (2 ml in the case of outdoor sampler, 1 ml for indoor samples). The tubes are left in a water bath at 80°C for 1 hour. 8 or 4 mls of distilled water is added and the tubes are shaken and centrifuged. Instrumental parameters 283.3 nm Wavelength 0.7 nm Spectral band width 10 mA Lamp current Read time 3 sec Signal mode Peak height Furnace/autosampler program Sample volume 20 µl Temp⁰C Ramp/hold (sec) 2/40 120 Dry 500 5/30 Char 2300 Argon flow 1/3 Atomize 20 ml/min 2600 1/1 Clean out

2.3.2 Indoor air samplers

Portable 8-hour samplers were distributed to shops, schools and private individuals living in the experimental area (Figure 1). Generally 3 consecutive 8 hour samples were collected at each site (generating a full 24 hour sampling period). Some, 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 lead concentrations found indoor) were characterized and 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.

The filters (Millipore AAwp 0.8 μ m mixed cellulose ester membrane filter) were analyzed for lead in the same laboratory (NILU) using the same methodology as the outdoor samples. These portable samplers collect particles less than 15-20 μ , 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 of 0.035 μ m diameter.

2.3.3 Diary information

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

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

- a) The area surrounding Oslo-Nydalen was arbitrarily divided into 12 sub-regions where air lead was either directly measured or estimated. In addition, downtown Oslo was divided into subdivisions based on degree of traffic. These divisions were determined using results of modelling within the city of Oslo (Grønskei et al., 1982) coupled with direct measurement of outdoor lead during the sampling period.
- 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) Overviews were acquired of time spent indoors at home, indoors at work or school, outdoors, time spent jogging or in heavy activity and time spent travelling for the 14 days prior to blood sampling.

2.3.4 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). It was therefore decided to calculate air lead exposure estimates for the 14 days prior to each individual blood sampling. Since, blood sampling of the entire population sample took place over a 14 day period, daily lead measurements (24 hour means) were collected for each station over a 30 day period. The air lead values used in this study are found in Appendix I.

The individual air lead exposure estimate takes into account time spent indoors and travelling. An extra factor was used if people were actively jogging to account for increased respiratory rate (X 2). Likewise, a factor (1.2) was used to account for higher activity in children when they were outdoors, since they were usually playing. (Factors recommended by lung specialists and Astrand and Rodahl, 1977).

Adding these activity factors results in increasing the total range of pollution exposure, thus slightly flattening the slope of the regression of blood lead to air lead. (Clench-Aas et al., 1984).

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

2.3.5 Measurements of lead in snow and drinking water

In order to assure that blood lead values in the study area did not reflect intake from other possibly important sources such as lead in snow in playgrounds, or from drinking water, a few extra measurements of snow and drinking water were made.

Surface snow samples were gathered in fields where children were most likely to play, at all the schools and kindergartens. In addition, one sample was collected within the industrial area. Measurements of lead in snow reflect deposition of lead from the air. These values can give an indication of relative concentration of lead in these different geographical areas. This information was used to help estimate air lead values in those subdivisions where air was not directly measured (subdivisions - described in 2.3.3).

The samples represent several centimeters depth and were collected on the 29th of February. A small amount of snow fell on the 24th (0.1 mm precipitation) and 22nd (0.3 mm precipitation) that did not considerably increase snow depth. It is necessary to go back to the 16th and 17th (13 days prior to sampling) where 3 centimeters of snow fell to find a more substantial snowfall (Meteorological Institute - personal communication).

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 February 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.

In Sørumsand, blood samples were collected in May 1984 and analyzed by the same methodology.

2.4.2 Determination of hematocrit, hemoglobin and zinc-protoporphyrin

In order to standardize blood lead concentrations, hematocrit was measured. As a further control of hematocrit values, hemoglobin values were also measured. 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)).

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 of 45%.

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 M HNO_3 to be less than 0.01 μ mol Pb/l whole blood.

Lead concentrations in whole blood were determined by electrothermal atomic absorption spectrometry (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. A 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 $0.4 \ \mu mol \ Pb/l$, and the detection limit (2x noise level) was $0.01 \ \mu mol \ Pb/l$.

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:

B-Pbx45.0

measured hematocrit

Since the intension of this study was to compare values of lead in blood with lead in air, and air is measured in $\mu g/m^3$, blood lead values have been converted from $\mu mol/l$ to $\mu g/100$ ml (dl) using the formula B-Pb $\mu g/100$ ml = B-Pb($\mu mol/l$) x 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 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 \pm 7%.

2.5 INFORMATION ON SOCIO-ECONOMIC PARAMETERS

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

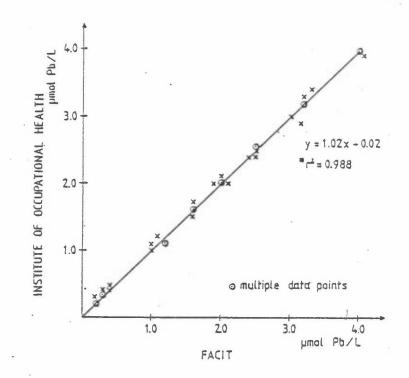


Figure 2: Results from interlaboratory surveys of lead analysis in whole blood (1980 - 1983). The linear regression is based on 47 data pairs. $r^2 = c$

= coefficient of determination.

Table 4: Summary of whole blood lead method.

sampler cup. U	lood 1:4 with matri se the method of st X-100 and 0.5% (NH	andard addition	
- 0.2% IFICON	A-100 and 0.5% (MH	Jaro.	
Instrumental P	arameters		
Wavelength		283.3	1778
Spectral Band	Width	0.7	
Electrodeless			W
Background Cor.		On	
Read Time		5	sec.
Signal Mode		Peak	neight
Average		2 0:	c 3
Furnace/Autosa	mpler Program		
	mpler Program 10 µl, ordinary gra	phite tubes.	
		phite tubes.	Ramp/Hold
	10 µl, ordinary gra	phite tubes.	Ramp/Hold sec.
Sample volume	10 µl, ordinary gra Temp. C	phite tubes.	sec.
Sample volume Dry	10 µl, ordinary gra Temp. C 120	phite tubes.	sec. 5/15
Sample volume Dry	10 µl, ordinary gra Temp. C 120 180	phite tubes.	sec. 5/15 5/5
Sample volume Dry	10 µl, ordinary gra Temp C 120 180 230	phite tubes.	sec. 5/15 5/5 5/2
Sample volume Dry	10 µl, ordinary gra Temp. C 120 180	phite tubes. Baseline 12	sec. 5/15 5/5
Sample volume Dry Char	10 µl, ordinary gra Temp C 120 180 230 400		sec. 5/15 5/5 5/2 5/5
Sample volume Dry Char	10 µl, ordinary gra Temp. C 120 180 230 400 800		sec. 5/15 5/5 5/2 5/5 5/20
Sample volume Dry Char	10 µl, ordinary gra Temp. C 120 180 230 400 800	Baseline 12	sec. 5/15 5/5 5/2 5/5 5/20 1/6
Furnace/Autosa Sample volume Dry Char Atomize	10 µl, ordinary gra Temp. C 120 180 230 400 800 2400	Baseline 12 Recorder	sec. 5/15 5/5 5/2 5/5 5/20 1/6 -5

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.

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

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 Appendix 2).

2.6 DATA ANALYSIS

Hematocrit adjusted blood lead (CPbB) and estimated air lead exposure (PbA) along with the measured social and biological parameters were analyzed using conventional statistical packages DDPP (Jakobsen, 1982). Analyses included tests for skewness, kurtosis, factor analysis, simple regressions and multiple step-wise regression. Table 5: Definition of subgroups used in data analysis.

2 - 15 YRS 1) CHILDREN A) NOT EXPOSED TO PASSIVE SMOKING B) EXPOSED TO PASSIVE SMOKING 2) WOMEN 16 - 97 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 16 - 97 YRS3) MEN - Same as for Women

3 RESULTS

3.1 INDIVIDUAL AIR LEAD EXPOSURE

3.1.1 Lead in outdoor air in Oslo-Nydalen and Sørumsand.

Figure 3 shows values of air lead $(\mu g/m^3)$ at three fixed outdoor stations in Oslo. Values in Sørumsand can be considered as background and averaged 0.03 $(\mu g/m^3)$. Air lead concentrations in Oslo are much more variable and under the influence of meteorological conditions than those in Sørumsand. Mean values in Oslo for the observation period (February, 1984) as indicated in Appendix I, were 8 to 20 times higher than for those measured in Sørumsand and 2 to 6 times higher than average values in Holmestrand (Table 6).

In an attempt to evaluate the relative importance of the two principal types of lead sources - industrial emissions and vehicular traffic - it is necessary to examine air lead values day by day taking wind direction into account. Wind direction is also indicated in Table 2.2 in Appendix I. The given wind direction and wind speeds reflect averages of 24 values collected hourly at one station - Blindern. If wind direction shifted during the day, the primary directions are indicated. One must be cautious using the meteorological values given since Blindern is west of the study area. The experimental area - Nydalen - lies in a valley (see Figure 1) with a slight northwest - southwest axis that could result in slightly different wind directions than measured at the meteorological station. It can be roughly estimated that when winds come from the north-northeast the Bakkehaugveien station (w) receives lead mainly from traffic on the highway whereas the Nydalsveien station (S) receives air lead from both traffic and the industrial sources. Likewise when winds come from the south-southwest, then Grefsen skole (E) gets air lead mainly from traffic and OT. vei (N) gets air lead both from traffic and industrial sources.

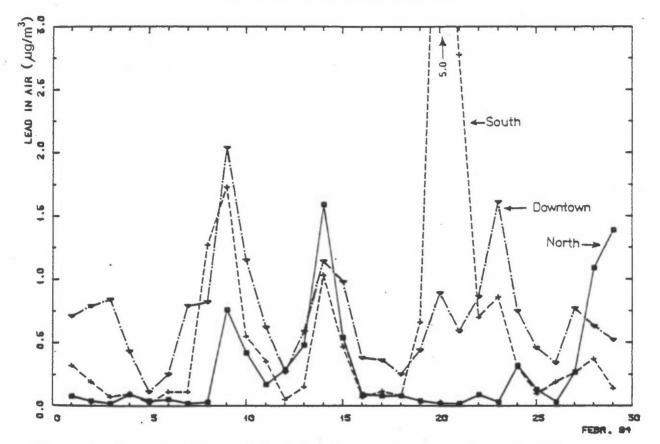
There were very few days where wind originated from only one direction. Table 7 uses a restrictive definition of north and south and shows values for the above named station. Pure southerly winds were not associated with high values in any of the stations. Pure northerly winds resulted in values a factor of 10 higher in Nydalsveien (thus both industrial and traffic sources) in 4 out of 5 days.

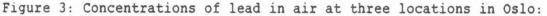
0:	510	S	ørumsand	Holmestrand			
Station	Average yalue (µg/m ³)	Station	Average yalue (µg/m ³)	Station	Average yalue (µg/m ³)		
North East South West Southeast DowntownOslo """	0.29 0.24 0.63 0.34 0.27 0.70 0.45	1 2	0.02 0.02	Crosslight* N near hiway S near hiway N living area* S living area*	0.69 0.19 0.09 0.10 0.05		

Table 6: Average outdoor air lead values $(\mu g/m^3)$ in several fixed stations in Oslo (February 1984) and the control area Sørumsand (May 1984) with Holmestrand (May 1983) given for comparison.

*Values estimated using indoor portable samples. All other unmarked values represents actually measured samples.







1) One to the north of primary lead sources (-----).

2) One to the south of the primary lead sources (----).

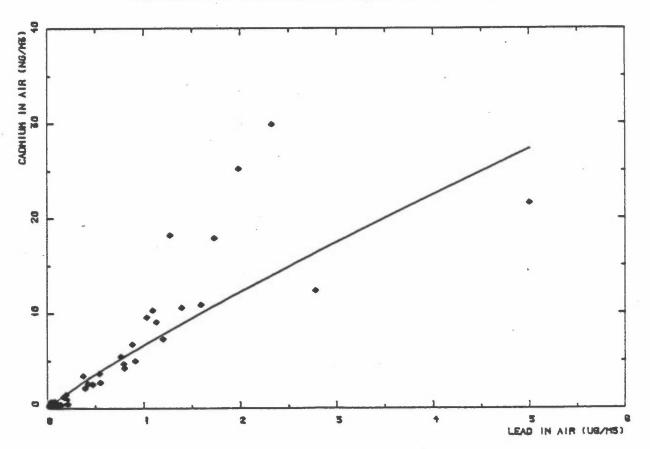
3) Downtown Oslo in a street canyon (-----).

Table 7: Air lead values as a function of wind direction and measuring station. Values gathered from Appendix I.

	Winds from the n (310 to 50 degre	Winds from the south (140 to 230 degrees)						
Date	Bakkehaugveien (W)	Nydalsveien (S)	Date	Grefsen skóle (E)	OT vei (N)			
24/1	0.08	0.84	5/2	0.09	0.04			
28/1	0.07	0.65	6/2	0.05	0.07			
29/1	0.06	0.80	12/2	0.11	0.29			
3/2	1.13	0.07	17/2	0.12	0.08			
8/2	0.11	1.27	18/2	0.14	0.08			

The air pollution exposure estimate was, of course, most affected by high air lead values on cold winter days with climatic inversion. Using values for February (only February values were used in calculating the estimate) one can examine air lead values at all stations if and when one of the values was over $1 \mu g/m^3$. This is done in Table 8. Wind direction varies a good deal during the day. Thus it is not easy to compare 24 hour air lead values to wind direction. Traffic contributes most lead to the air during rush hour, therefore the wind direction at that time is most interesting. Industrial sources do not have continuous emissions.

Air cadmium was measured during the same time period and at four of the stations. Cadmium is released from the main industrial source but not from traffic emissions. These results are further discussed and summarized, in a separate report concerning air concentration and urinary excretion of cadmium (Clench-Aas, et al., 1986).



CORRELATION BETWEEN AIR-LEAD AND AIR-CADMIUM

Figure 4: Correlation between lead and cadmium in air. The points in the graph are those given in Tables 8 and 9.

Date	N* OT Vei	E Grefsen skole	S Nydals- veien	W Bakkehaug- veien	SE Sagene brannst.	Wind* direct
2/12	0.04	0.07	0.19	2.32	0.05	60
3/12	0.02	0.03	0.07	1.13	0.07	50
8/2	0.03	0.06	1.27	0.11	0.57	15 (11h) 50 (13h)
9/2	0.76	0.91	1.73	0.79	1.42	90 (3h) 230 (14h) 60 (7h)
14/2	1.59	0.88	1.03	0.80	0.88	very unruly
15/2	0.54	0.55	0.47	1.20	0.38	90 (6h) 200 (18h)
20/2	0.02	0.04	5.00	0.06	0.08	70
21/2	0.02	-	2.78	0.16	0.06	70
28/2	1.09	0.39	0.37	0.20	0.37	260
29/2	1.39	0.42	0.14	0.21	0.42	240
2/3	0.02	0.04	1.98	0.11	-	250 (20h) 50 (4h)

Table 8: Examination of air lead values at all 5 stations in the study area on those days where air lead concentrations exceeded 1 μ g/m³ for at least one site.

Table 9: Air cadmium values (ng/m^3) for the same days as mentioned in Table 8.

Date	N* OT Vei	E Grefsen skole	S Nydals- veien	W Bakkehaug- veien	Wind* direct
2/12	0.72	0.39	1.54	29.90	60
3/12	0.38	0.50	0.78	9.06	50
8/2	0.27	0.31	18.20	0.33	15 (11h) 50 (13h)
9/2	5.51	5.00	17.90	4.67	90 (3h) 230 (14h) 60 (7h
14/2	10.87	6.73	9.56	4.22	very unruly
15/2	3.73	2.72	2.58	7.27	90 (6h) 200 (18h)
20/2	0.43	0.74	21.60	0.60	70
21/2	0.32	-	12.35	1.25	70
28/2	10.30	2.20	3.40	1.84	260
29/2	10.30	2.64	0.37	0.51	240
2/3	0.30	0.61	25.20	0.40	250 (20h) 50 (4h)

*Site locations are given in Figure 1. See section 3.1.1 for description of meteorological information.

Values of ambient cadmium can be used to assess the relative importance of industrial versus traffic emissions as primary source of lead in air. Table 9 gives air cadmium for the four stations on the same days air lead concentrations were high as given in Table 8. Since both air lead and air cadmium are log normally distributed the regression of the natural logarithm of air cadmium versus the natural logarithm of air lead gives an r^2 of 0.89. All stations and days with values of air lead over 1 μ g/m³ in Table 8 have values of air cadmium over 7 ng/m³. Therefore, indications are that the main source of the highest air lead concentrations in the study area is industrial emissions. Correlation coefficients between air lead and air cadmium in St. Olavs gate, downtown Oslo was 0.28 and in Holmestrand, 0.06. Thus only in the area immediately surrounding Nydalen were concentrations of cadmium in air correlated to lead in air.

3.1.2 Indoor air lead concentrations

Table 10 gives average 24 hour indoor air lead concentrations $(\mu g/m^3)$ of various indoor sites during the measuring period with the outdoor value of the nearest fixed station for the same day (sites of indoor samplers as indicated in Figure 1).

It is evident by examining the values that other local sources (for example proximity to a parking lot or road) may cause locally higher values than were measured at the outdoor station. Table 11 compares indoor samples collected morning, afternoon and night, to give some idea of the variability during the course of a 24 hour day. Although air lead concentrations vary significantly during the day, (as Table 11 indicates) they do not vary predictably. Therefore no correction was made for 24 hour variations.

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.

Table 10: Average 24 hour indoor air lead concentrations $(\mu g/m^3)$ in Oslo-Nydalen, February 1984, with corresponding measured outdoor value $(\mu g/m^3)$ at nearest fixed station for the same measuring day. Indoor values were measured using portable pumps and a slightly different filter system than fixed outdoor samplers. This has led to slightly higher values for the indoor sampler than the outdoor sampler when the two have been directly compared.

Northeast of Indoor	point sources Outdoor	<u>South of r</u> Indoor	oint sources Outdoor
0.02 0.00 0.27* 0.04 0.02 0.02 0.02 0.02 0.02	0.03 0.03 0.32 0.32 0.03 0.03 0.03 0.03	0.04 0.07 0.11* 0.03 3.19 0.04 0.05* 0.82 0.15 0.16 0.06 0.04 0.31	0.07 0.34 0.27 0.07 5.00 0.07 2.78 0.37 0.21 0.27 0.11 0.11 0.34
East of poir Indoor Ou	nt sources utdoor	West of po Indoor	o <u>int sources</u> Outdoor
0.06 (). 4 3). 06). 04	0.05 0.04 0.17* 0.03* 0.02* 0.05* 0.07*	0.10 0.14 0.07 0.10 0.07 1.20 0.10
Avera	ge of 1 to 2 wee) 1.35 1.1	5	.ving

*Measurements represent less than 24 hours, for example in schools.

Table 11: Comparison of 8 hour indoor air lead concentrations $(\mu g/m^3)$ in the Oslo-Nydalen area in February, 1984, by time of day of collection: morning (0600-1500), afternoon (1500-2300) and night (2300-0700).

Corresponding 24 hour outdoor value of nearest fixed station	Morning 0600-1500	Afternoon 1500-2300	Night 2300-0700
0.02 0.10 0.10 0.07 0.04 0.14 0.43 0.34 0.34 0.06 0.03 0.03	0.02 0.04 0.04 0.05 0.03 0.28 0.06 0.29 0.11 0.02 0.02	0.13 0.02 0.02 0.00 0.05 0.05 0.05 0.08 0.06 0.05 0.02 0.02	0.04 0.08 0.02 0.07 0.03 0.05 0.27 0.06 0.58 0.02 0.00 0.01
0.03 0.03	0.03 0.04	0.01 0.01	0.03 0.01

3.1.3 Estimation of personal exposure

When comparing blood lead concentrations 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 5 for the inhabitants of Oslo-Nydalen and Sørumsand.

Differences in:

- 1) geographic distribution of values of air lead,
- individual behavior patterns (e.g. time spent indoors or outdoors, vacationing outside the area, sleeping with open or closed windows),
- 3) time spent in active sport (e.g. jogging),
- 4) travel time.

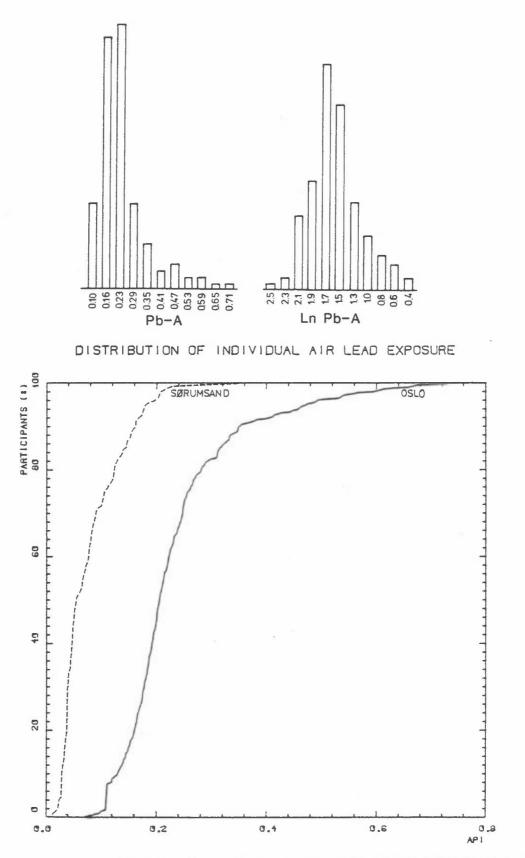


Figure 5: Frequency distributions of exposure estimates to air lead $(\mu g/m^3)$ and their natural logarithms in the population living in the Oslo-Nydalen area. See text for description of exposure estimating method.

led to a range in air lead exposure from 0.07 to 0.74 or equivalent to a 10 fold range of exposure to air lead concentrations within a fairly limited geographical area in the city of Oslo. Exposure estimates in Sørumsand ranged from 0.01 to 0.27 or an even greater range. Differences in individual exposure to lead must be accounted for in evaluating blood lead concentration.

As can be seen in Figure 5, air lead exposure estimates are log-normally distributed. Therefore, the natural logarithm of the estimate should be used in statistical analysis.

3.1.4 Lead in snow

As shown in Figure 6, lead in snow (measured as $\mu g/liter$) varied considerably in the geographic area. Snow samples were mostly collected in areas where children might eat snow; in fields near schools and kindergartens relatively far from roads. One sample was collected within the industrial zone.

Generally, concentrations decline with distance from the industrial sources.

3.2 GENERAL STATISTICAL OVERVIEW OF DATA SET

In order to get a preliminary overview of variable interactions, a correlation matrix (Pearson's rho) of pertinant variables in the Oslo-Nydalen data set was calculated. Since questions asked of children were not the same as of adults the data set was divided into two groups, children (15 years or less) and adults (16 years or older). The results are presented in Appendix III.

The continuous variables entered into the matrices were: mean cell hemoglobin concentration (MCHC), hemoglobin (Hb), hematocrit (Ht), age, and the natural logarithms of standardized blood lead concentrations (Log Pb-B), standardized zinc protoporphyrin (Log ZPP), and air lead exposure (Log Pb-A). A fuller description and validation of the choice of these variables (for example use of natural logarithm), is found in the following chapters concerning detailed analysis of each parameter.

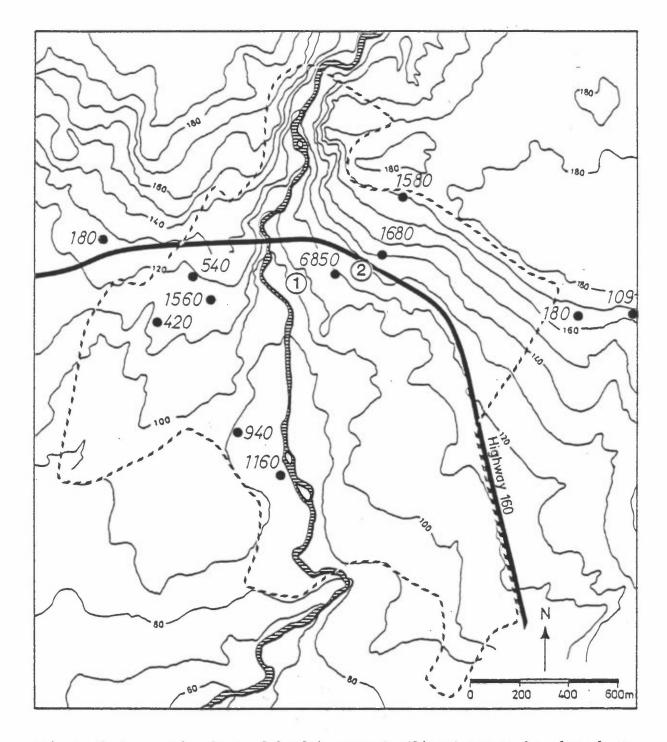


Figure 6: Measured values of lead in snow (µg/liter) two weeks after last snowfall. (1) and (2) industrial point source; • snow sample sites; — Highway 160 with 30.000 vehicles daily. The categorical parameters entered into the matrix for children were: sex, passive smoking, social class, use of vitamin supplements, use of iron supplements, and eating of snow. For adults the categorical parameters were: sex, social class, alcohol consumption, use of vitamin supplements, use of iron supplements and smoking.

Examination of Appendix III reveals most strikingly a greater number of intercorrelations in the adult population than the children. Correlations between Hb, Ht and MCHC are of course not surprising since MCHC = Hb/Ht x 100. But it is evident that there exists a great deal of intercorrelation between explanatory variables. For example social class, alcohol consumption, use of iron supplements, log Pb-Air and age. Most importantly for this study is that the air lead exposure estimate is of course, a function of residential area. Residential area, however, can also be correlated to social class, alcohol consumption, age and sex in addition to value of blood lead.

In an attempt to get a better overview over the independent variables and possible interactions between them factor analysis was used. The results of the independent variables are presented in Table 12. Factor analysis is one technique that allows variables that are strongly intercorrelated to be identified such that in the interpretation of the results it is understood that significance of one of these variables can in reality, mean that any of the intercorrelated variables is significant. In children, eating of snow is strongly associated with younger children. Although this is not surprisit should be noted that the variable, eating snow, may in fact be a ing, surrogate for very young children. There is greater exposure to passive smoking in those social categories with the least medical knowledge (description of social classes in Appendix II). Female children take more vitamins than male children. In adults, increased use of vitamins and iron in the adult woman with increasing age are correlated. Smoking and alcohol consumption are positively related. Log Pb-A and social class came out as unique significant variables.

Table 12: Results of factor analysis with variance rotation of the independent variables in children and adults.

	Factor 1	Factor 2	Factor 3	Factor 4
CHILDREN* ²	Eating snow (-)* ¹ Age (+)	Passive smoking (+) Social class (+)	Sex (+) Use of vitamins (+)	x
% of variance explained by factor	19.4%	15.3%	13.7%	X
ADULTS* ³	Age (-) Sex (+) Use of vitamins (+) Use of iron (+)	Smoking (+) Alcohol consumption (+)	Social Class (+)	Log Pb-A(+)
% of variance explained by factor	20.0%	15.5%	14.4%	13.2%

 \star^1 (+) (-) indicates direction of association, see text for direction of variables. *² Children who admitted to smoking removed from analysis.

*3 Occupationally exposed individuals removed from analysis, as well the elderly living in a clinic.

3.3 BLOOD LEAD CONCENTRATIONS

3.3.1 Preliminary data handling

The findings in this report have used the following data correction factors. A further discussion of these assumptions is given earlier in the Holmestrand report (Clench-Aas, et al., 1984):

- 1) Because of concentration of lead in the erythrocyte, values of lead measured in whole blood were standardized for hematocrit (CPbB),
- 2) The estimate for individual exposure to air lead included a factor to account for increased respiration during high activity.
- 3) Values of both air and blood lead were found to be log normally distributed, and the analyses of statistical significance were run on the natural logarithmic values.

As was seen in the earlier report 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.

3.3.2 The frequency distribution of standardized blood lead

Figure 7 shows the frequency distribution with accompanying statistics of standardized blood lead concentrations and its natural logarithm. As is evident and often discussed in the literature, the distribution is better approximated by a log normal distribution.

The values of blood lead concentrations ranged from 1.4 μ g/100 ml to 36.9 μ g/100 ml in the Oslo-Nydalen population. The higher values, however, were in 15 occupationally exposed men who worked at either of the two point sources. Excluding occupationally exposed individuals resulted in a total range of blood lead concentrations from 1.4 to 23.6 μ g/100 ml. Occupationally exposed individuals were removed from the data set during analysis of the data.

3.3.3 The correlation of blood lead concentrations to air lead concentrations and other biological and socio-economic parameters

As a result of initial analyses and the previous years findings, the data were subdivided into two subgroups, children and adults, and included both the Oslo-Nydalen data and the control low lead values of Sørumsand measured in 1984. Step-wise multiple regression was used to analyze each sub-group children and adults. Analysis proceeded in several steps. The first step was to relate the natural logarithm of blood lead concentrations (Log B-Pb) against as many independent variables as possible. However, this often resulted in the removal of too many cases due to missing variables. Therefore the results were run against a limited number of independent variables where data was complete. When a categorical parameter was found significant, data was subdivided into each group and the regressions rerun with the continuous variables, age and the natural logarithm of air lead exposure (Log Pb-A). In children the first step consisted of relating Log B-Pb against age, sex, Log Pb-A, social class, passive smoking and snoweating. The result of the first step was that none of the variables were significantly correlated with log B-Pb. However, when the variables social class and snoweating were removed from the analysis, sex was found to be significantly correlated to log B-Pb (Table 13). Splitting the data set into two groups males and females resulted in the regression coefficients for log B-Pb versus log Pb-A given in Table 17. Age was not significantly correlated to log B-Pb. These are visually portrayed in Figures 10 and 11.

In adults the result of the first step was that age, sex, social class, alcohol consumption and the natural logarithm of air lead concentration was significantly correlated to log B-Pb (Table 13). Only smoking was not significant. The data was then divided into men and women with the results given in Table 17 and Figures 10 and 11.

As a result of the step-wise multiple regression analysis, means, standard deviations and medians were tabulated for easier comprehension: by sex and age (Table 14 and Figure 8) and by alcohol consumption (Table 15). Values from Holmestrand in 1983 are given for comparison in Table 14. Because of the interesting finding in the previous study concerning smoking (Clench-Aas et al., 1984) the values have been tabulated in Table 16 and Figure 9, with Holmestrand and Sørumsand given as a comparison.

The results for the Oslo-Nydalen data indicate a weak but positive increase in blood lead concentrations in children and women with increasing air lead exposure. An increased blood lead concentration in adult men and women with increasing age was also found.

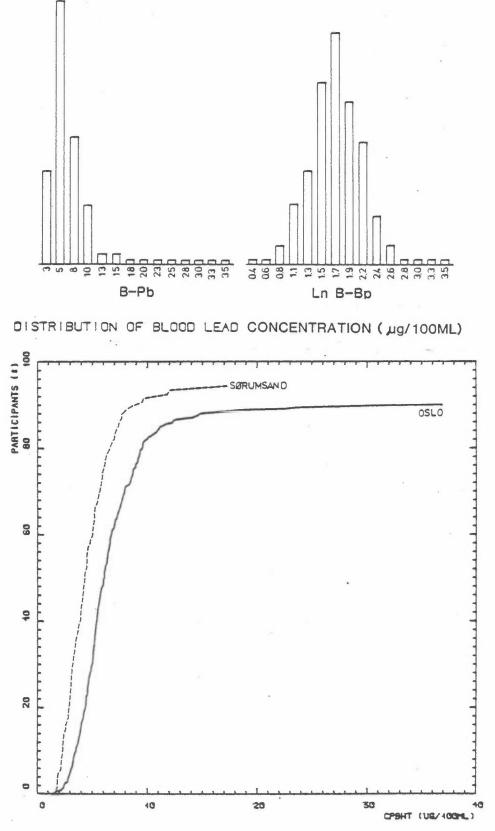
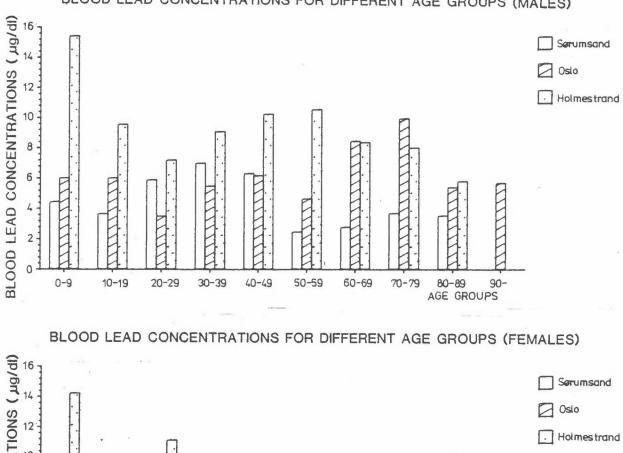


Figure 7: Frequency distributions of standardized blood lead concentrations in $\mu g/100$ ml and the natural logarithm of these values in Sørumsand and Oslo-Nydalen.



BLOOD LEAD CONCENTRATIONS FOR DIFFERENT AGE GROUPS (MALES)

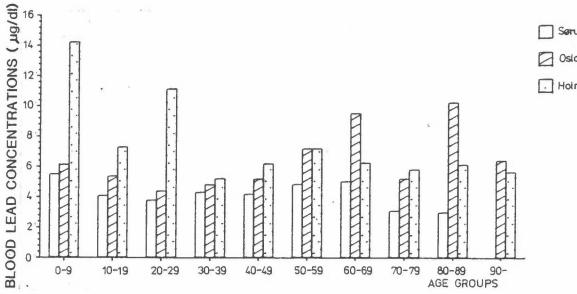


Figure 8: Median values of standardized blood lead concentrations (µg/100 ml) by 10 year age groups in males and females living in Sørumsand, Holmestrand and Oslo-Nydalen.

Table 13: Results of step-wise multiple regression analysis of the natural logarithm of blood lead concentrations: a) in children with sex, age, passive smoking and the natural logarithm of air lead exposure as independent variables; b) in adults, with sex social class, alcohol consumption, age, smoking and the natural logarithm of air lead exposure as independant variables.

a) CHILD	OBEN										
ar chicl											
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sex -	0.131	0.001	3.673	0.025	-1.001	-0.0101 -1		logPb-A		0.9774	0.0063
								pas smok		0.9933	0.0216
								pee smok	0.0122	0.0000	0.0210
) * ****	F-LEVEL:	s (4.000,	3.900) OR	TOLERANCE	INSUFFICIENT	FOR FURTHE	R STEPPING				
UMMARY	TARIE										
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SI AUUL	_15										
b) ADUL Variable	_TS Es in equ	iation:	*	(1	CONSTANT=	1.6598		I VARIA	BLES NOT I	N EQUATION:	÷
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Variable 8- ID COEFF	es in equ - FICIENT -0.174	STD.ERR	OR REMOV	P-VALUES E FOR B	STANDARDIZE B (R.PART)	D BETA 95% UPPER	LOWER	I I IO	PARTIAL CORR.	TOLERANCE	ENTE
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Variable 8- 10 COEFF Soc.cl. Soc.cl.	-0.174 0.055 0.006	STD.ERR 0.055 0.021 0.002	OR REMOV 9.825 6.715 7.763	P-VALUES E FOR B 0.002 0.010 0.006	STANDARDIZE B (R.PART) -0.1916 0.2245 0.2447	0 BETA 95% UPPER -0.0645 0.0960 0.0105	LOWER -0.2829 0.0131 0.0018	I I IO	PARTIAL CORR.	TOLERANCE	ENTE
Variable 8- ID COEFF Sex Soc.cl. Age Alc.co. .og Pb-A	-0.174 -0.174 0.055 0.006 0.072 0.087	STD.EAR 0.055 0.021 0.002 0.030 0.031	0R REMOV 9.825 6.715 7.763 5.655 7.585	P-VALUES FOR 8 0.002 0.010 0.006 0.018 0.006	STANDARDIZE B (R.PART) -0.1916 0.2245 0.2447 0.1482 0.1726	0 BETA 95% UPPER -0.0645 0.0960 0.0105 0.1312 0.1487	LOWER -0.2829 0.0131 0.0018 0.0123 0.0246	I I IO	PARTIAL CORR.	TOLERANCE	ENTE
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Variable B- ID COEFF Sex Soc.cl. Age Alc.co. .og Pb-A Alc.co. .og Pb-A SuMMAR' STEP NR. 1 2	-0.174 -0.055 0.005 0.072 0.087 F-LEVELS Y TABLE: MULT.R. 0.1857 0.4183	STD.EAR 0.055 0.021 0.002 0.030 0.031 5 (4.000, MULT.RSQ 0.0345 0.1750	OR REMOV 9.825 6.715 7.763 5.655 7.565 3.900) OR INCREASE IN RS9. 0.0345 0.0554	P-VALUES FOR 8 0.002 0.010 0.006 0.018 0.006 TOLERANCE RESIDUA EFFECT 0.9826 0.9083	STANDARDIZE B (R.PART) -0.1916 0.2245 0.2447 0.1482 0.1726 INSUFFICIENT L F-VALUE FOR E/I 5.249 14.714	0 8ETA 95% UPPER -0.0645 0.0960 0.0105 0.1312 0.1487 FOR FURTHER VAR.NAME Age Log Pb-A	LOWER -0.2829 0.0131 0.0018 0.0123 0.0246	I I IO	PARTIAL CORR.	TOLERANCE	ENTE
Variable B- ID COEFF Sex Sac.cl. Age Alc.co. .og Pb-A Alc.co. .og Pb-A SuMMAR' STEP NR. 1 2 3	-0.174 0.055 0.006 0.072 0.087 F-LEVELS Y TABLE: MULT.R. 0.1857 0.4183 0.4548	STD.EAR 0.055 0.021 0.002 0.030 0.031 5 (4.000, MULT.RSQ 0.0345 0.1750 0.2068	OR REMOV 9.825 6.715 7.763 5.655 7.565 3.900) OR INCREASE IN RS9. 0.0345 0.0355 0.0554 0.0318	P-VALUES FOR 8 0.002 0.010 0.006 0.018 0.006 TOLERANCE RESIDUA EFFECT 0.9826 0.9083 0.8906	STANDARDIZE B (R.PART) -0.1916 0.2245 0.2447 0.1482 0.1726 INSUFFICIENT L F-VALUE FOR E/I 5.249 14.714 8.744	D BETA 95% UPPER -0.0645 0.0960 0.0105 0.1312 0.1487 FOR FURTHER VAR.NAME Age Log Pb-A Sex	LOWER -0.2829 0.0131 0.0018 0.0123 0.0246	I I IO	PARTIAL CORR.	TOLERANCE	ENTE
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Occupationally exposed individuals not included for analysis. Data includes inhabitants of Sørumsand (low air lead control) and Oslo-Nydalen.

							Age G	roup (yr	z)			
			0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	> 90
		Mean	6.3	6.5	4.2	6.7	6.6	5.7	8.5	11.1	8.5	5.7
	Male	St.dev.	1.9	2.5	0.9	3.5	2.1	1.7	-	4.0	5.9	-
		(N)	(39)	(56)	(2)	(43)	(36)	(5)	(1)	(5)	(8)	(1)
		Median	6.0	6.0	3.5	5.5	6.2	4.7	8.5	10.0	5.4	5.1
Oslo-Nydaler	r											
1984		Mean	6.0	5.6	4.3	5.0	5.3	7.4	12.1	8.4	11.0	7.1
	Female	St.dev.	1.8.	2.3	1.3	2.2	1.8	1.1	3.6	1.7	2.8	4.8
		(N)	(28)	(43)	(4)	(83)	(33)	(3)	(2)	(2)	(12)	(6)
		Median	6.1	5.4	4.4	4.8	5.2	7.2	9.6	5.2	10.3	6.4
		Hean	16.2	9.8	8.0	9.0	9.9	11.1	8.7	7.7	5.9	
	Male	St.dev.	5.0	3.3	3.4	3.2	2.8	4.1	2.3	1.8	1.2	-
		(N)	(3)	(10)	(4)	(6)	(7)	(3)	(16)	(6)	(3)	
		Median	15.4	9.6	7.2	9.1	10.3	10.6	8.4	8.0	5.8	
Holmestrand												
1983		Mean	13.3	7.7	13.0	7.0	6.6	7.0	8.4	7.4	6.8	6.0
	Female	St.dev.	3.0	1.8	7.7	3.4	2.4	2.2	2.5	4.5.	2.5	Q.,
		(N)	(3)	(11)	(9)	(14)	(17)	(9)	(17)	(7)	(16)	(2)
		Median	14.2	7.3	11.1	5.2	6.2	7.2 .	6.3	5.8	5.1	5.3
		Mean	4.8	4.7	5.8	6.2	7.2	5.9	6.5	-	6.0	-
	Male	St.dev.	1.8	1.7	-	2.0	4.5	1.9	4.8	-	3.9	-
		(N)	(6)	(9)	(1)	(9)	(7)	. (4)	(3)	-	(2)	-
		Median	4.5	4.3	5.8	5.8	5.8	4.8	5.2	-	3.9	-
Serumsand		Mean	3.7	3.4	2.9	3.8	3.8	5.5	3.7	2.2	4.0	2
	Female	St.dev.	1.7	1.3	0.5	2.0	1.2	3.5	1.9	-	-	-
	L. AWYT &	St.dev.	(3)	(7)	(2)	(18)	(18)	(8)	(2)	(1)	(1)	_
		(N) Median	131	3.3	121	3.5	3.4	4.6	2.4	2.2	117	-

Table 14: Standardized blood lead concentrations (μ g/100 ml) in individuals from Oslo-Nydalen, Holmestrand and Sørumsand (control area) by 10 year age groups.

81ood lead concentrations are hematocrit adjusted. Occupationally exposed individuals not included. Values for Holmestrand originate from the 1983 study.

		Never drink	Drink less than 1	Drink at	least one	ce a week
		ditik	per week	Seldom	Often	Daily
Women	Mean St.dev. (N) Median	5.7 3.5 (31) 4.9	6.2 3.0 (32) 6.0	5.6 2.3 (73) 5.2	5.2 2.0 (12) 5.2	- - -
Men	Mean St.dev. (N) Median	5.5 1.5 (15) 5.2	7.3 4.8 (20) 5.9	6.7 3.1 (53) 5.5	8.3 2.6 (13) 8.3	5.3 - (1)

Table 15: Standardized blood lead concentrations (μ g/100 ml) in individuals from Oslo-Nydalen by alcohol consumption.

Blood lead concentrations are hematocrit adjusted.

Occupationally exposed individuals not included for analysis.

Table 16: Standardized blood lead concentrations (µg/100 ml) in inhabitants of Oslo-Nydalen, Holmestrand and Sørumsand.

		CHI	LDREN			WO	MEN						MEN		
		1	sive* king			Smokin	g habit	\$				Smokin	g habit	S	
		Not exp	Exp	Nev	Form	.0ccs	1-9	10+	All smoke	Nev	Form	0ccs	1-9	10+	All smoke
Sørum-	Mean	4.8	5.9	4.4	4.5	4.1	3.2	3.5	3.4	6.6	5.0	4.9	6.5	8.6	. 7.6
sand	St.dev	1.9	2.6	1.7	1.4	2.0	1.6	1.2	1.3	1.8	2.3	1.2	1.8	1.7	2.0
	(N)	23	4	25	18	8	3	6	9	6	7	3	5	5	10
	Median	4.1	4.6	4.5	4.2	3.3	3.2	3.4	3.2	7.4	4.4	4.4	6.5	7.9	7.2
Holme-	Mean	7.1	13.1	6.2	7.3	5.7	10.6	8.6	9.2	8.8	8.3	-	9.0	9.1	9.1
strand	St.dev	1.8	3.6	2.4	4.3	1.8	7.9	3.4	5.1	3.5	2.3	-	4.0	2.4	2.8
	(N)	11	13	43	15	3	9	21	30	10	16		5	15	20
	Median	7.7	12.2	6.1	7.0	5.5	8.8	8.1	8.3 .	7.8	7.5	-	10.6	9.3	9.3
Oslo-	Mean	6.2	6.3	5.9	4.3	5.4	5.3	6.5	6.2	6.0	6.8	6.9	7.9	7.8	7.8
Nydalen	St.dev	2.3	2.0	2.8	1.5	2.5	1.5	3.4	3.0	1.8	3.9	2.0	3.7	4.7	4.4
	(N)	82	66	75	20	17	9	32	43	37	23	12	12	21	33
	Median	6.0	6.0	5.4	4.3	4.8	5.2	5.6	5.7	5.6	. 5.5	7.6	6.9	5.9	6.0

See Table 5 for explanation of categories. Blood lead concentrations are hematocrit adjusted. Occupationally exposed individuals are not included. Values for Holmestrand originate from the 1983 study.

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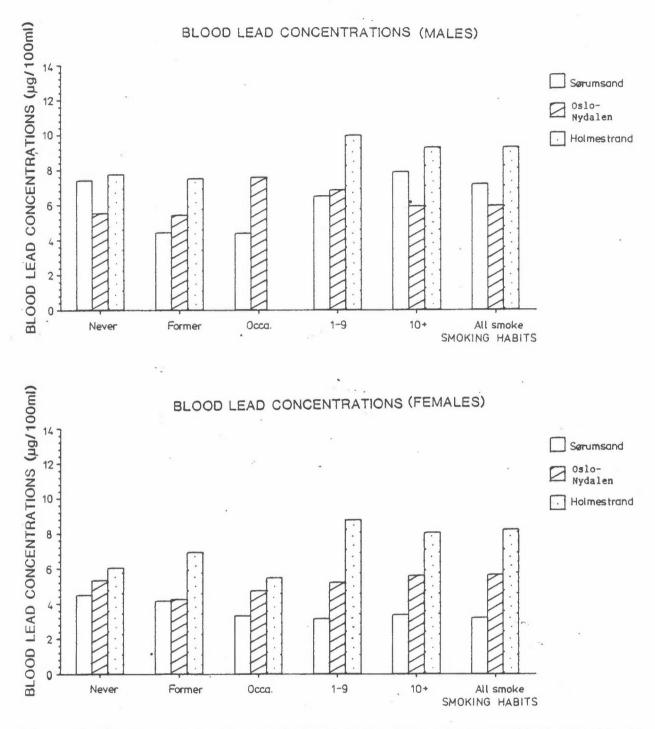


Figure 9: Median values of standardized blood lead concentrations (µg/100 ml) by smoking habits in adults and exposure to passive smoking in children living in Sørumsand, Holmestrand and Oslo-Nydalen.

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Figure 9: Continued

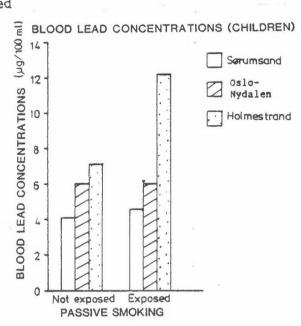
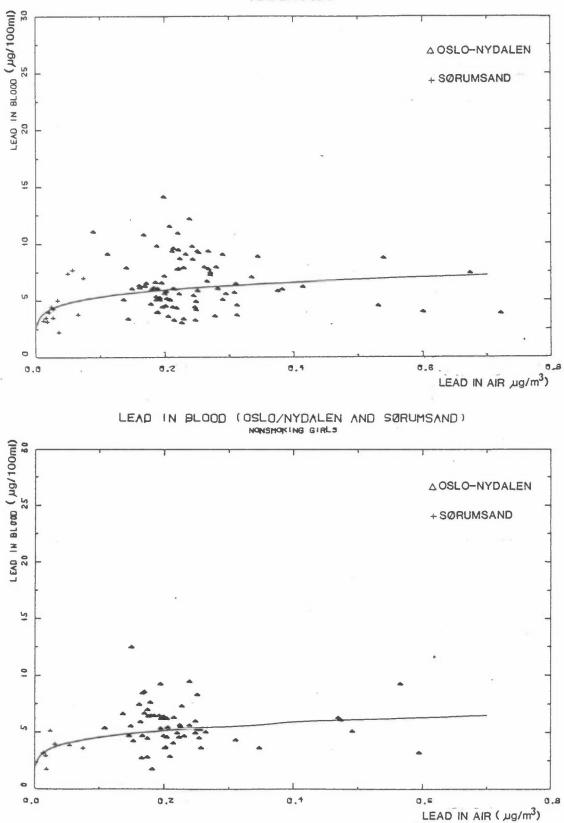


Table 17: Regression coefficients of log blood lead versus log air lead exposure (Log Pb-A) and age with statistical significance in children, women and men.

Population group	β	constant a	R	R ²	F	P
CHILDREN BOYS vs. Log Pb-A* ¹ vs. age* ²	0.15	7.61	0.31	0.10	11.00	.05 N.S.
GIRLS vs. Log Pb-A vs. age	0.17	6.96 -	0.34	0.12	9.0	.05 N.S.
WOMEN						
vs. Log Pb-A vs. age	0.139	6.23 3.22	0.27 0.30	0.07 0.09	15.72 20.24	.01 .01
MEN						
vs. Log Pb-A vs. age	0.004	- 5.05	0.19	0.03	- 4.697	N.S. .05

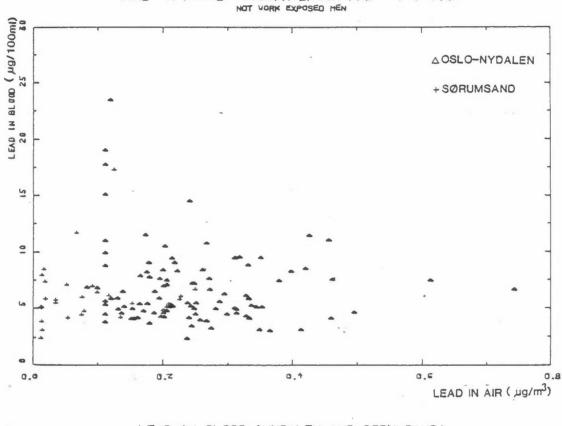
*¹Lny = $\ln\alpha + \beta \ln x$ which is equivalent to $y = \alpha x^{\beta}$.

 $*^{2}$ Lny = ln α + β x which is equivalent to y = $\alpha e^{\beta X}$. Blood lead is hematocrit adjusted. Occupationally exposed individuals not included for analysis. Data includes inhabitants of Sørumsand (low air lead control) and Oslo-Nydalen. 55



LEAD IN BLOOD (OSLO/NYDALEN AND SØRUMSAND)

Figure 10: Blood lead concentrations ($\mu g/100 \text{ ml}$) in children, women and men as a function of individual estimated air lead exposure. The data include values from both Sørumsand and Oslo-Nydalen. 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 17. + Sørumsand Δ Oslo-Nydalen.



LEAD IN BLOOD (OSLO/NYDALEN AND SØRUMSAND)

LEAD IN BLOOD (NYDALEN AND SØRUMSAND) NOT VORK EXPOSED VOMEN

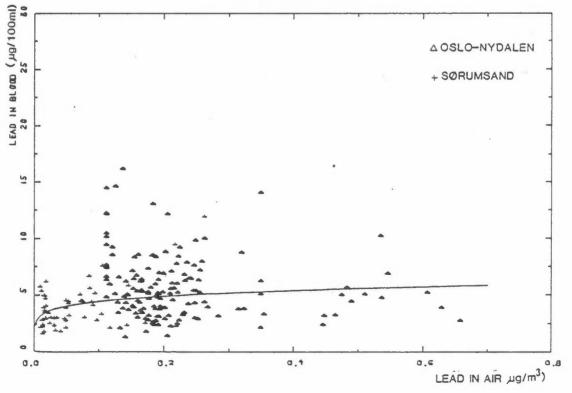
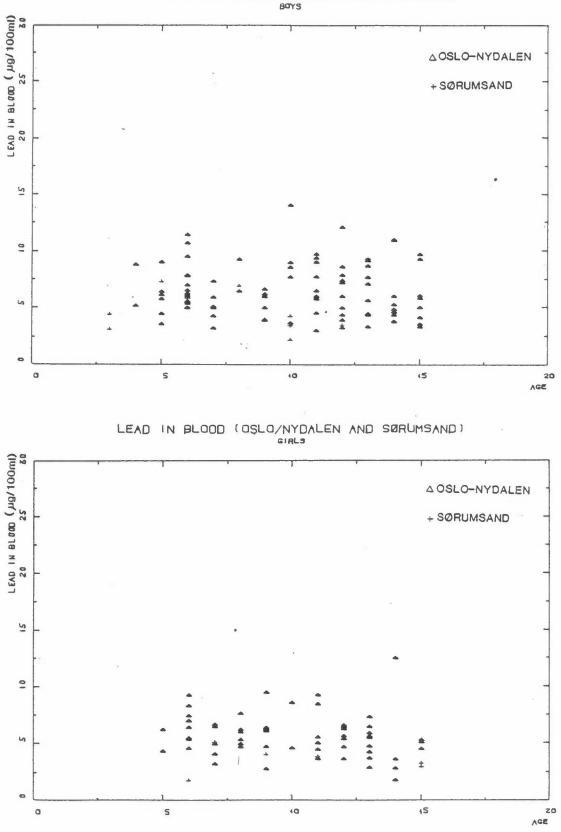
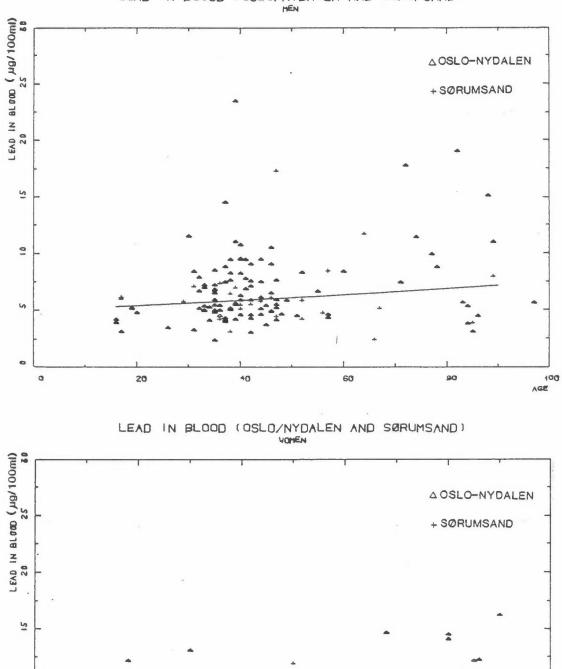


Figure 10: Continued



LEAD IN BLOOD (OSLO/NYDALEN AND SØRUMSAND)

Figure 11: Blood lead concentrations (μg/100 ml) in children, women and men as a function of age. The data include values from both Sørumsand and Oslo-Nydalen. + Sørumsand Δ Oslo-Nydalen.



LEAD IN BLOOD (OSLO/NYDALEN AND SØRUMSAND)

Figure 11: Continued

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3.4 HEMOGLOBIN CONCENTRATIONS

Hemoglobin values were examined for children and adults in Oslo-Nydalen and Sørumsand using step-wise multiple regression. Prior analysis revealed them to be normally distributed. Results of the analyses are summarized in Part II. There was no effect of blood lead on hemoglobin values in children or adults. However, in children there was a positive correlation between age and hemoglobin. In adults there was a positive correlation between alcohol consumption and hemoglobin. Not unexpectedly, females had lower hemoglobin values than males. There was no effect of smoking or social class on hemoglobin concentrations.

3.5 <u>HEMATOCRIT LEVELS</u>

The findings reviewed in this section are presented in Part II. Results of step-wise multiple regression analysis in adults of Sørumsand and Oslo-Nydalen only supported the well known difference between the sexes with females having the lower value as for hemoglobin. The other variables - social class, smoking, logarithm of blood lead, age and alcohol consumption - produced no significant correlation.

In children, sex, use of iron supplements, logarithm of blood lead and passive smoking were not significantly correlated to hematocrit levels, whereas age was posivitely associated with hematocrit.

As with hemoglobin the values cannot be considered surprising.

3.6 MEAN CELL HEMOGLOBIN CONCENTRATIONS (MCHC)

Mean cell hemoglobin concentration (MCHC) is the hemoglobin divided by the hematocrit x 100. It reflects the density of hemoglobin within the red blood cell. Elevation of this parameter therefore indicates either increased concentrations of hemoglobin within the cell, or cell shrinking.

A rather strong association between ingested lithium levels and MCHC has been observed earlier (Clench et al., 1981). Therefore it was of interest to explore the relationship of MCHC with lead and its possible usefulness as an "effect" variable.

Step-wise multiple regression in children in the Oslo-Nydalen area with sex, age, use of iron supplements, passive smoking, social class and natural logarithm of blood lead concentration entered as independent variables revealed only a significant correlation with passive smoking. Children exposed to passive smoking had higher MCHCs. In adults, MCHC increased with smoking. In other words, the blood cells were smaller or the concentration of hemoglobin within the cells greater with increasing smoking. Women had lower MCHC than men and MCHC dropped with increasing age (Table 16). Social class, alcohol consumption, use of iron supplements and log of blood lead concentrations were not significantly related to MCHC.

3.7 ZINC PROTOPORPHYRIN CONCENTRATIONS

Zinc protoporphyrin (ZPP) concentrations were standardized for a hematocrit of 45%. Results of analyses are presented in Part II.

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

1: Adults - a significant relationship with hemoglobin was found, with a decrease in log ZPP with increasing hemoglobin. Increased alcohol consumption led to increased ZPP. No significant relationships were found with logarithm of blood lead, smoking, sex, age, social class, or use of iron supplements.

2: Children - no significant correlations.

These findings were expected. The measured blood leads in this study are low and below any reported effect on ZPP in the literature. The findings are similar to those reported for Holmestrand and Sørumsand (Clench-Aas et al., 1984).

4 DISCUSSION

Concentrations of lead in blood were low in the Oslo-Nydalen area relative to values reported internationally or measured in Holmestrand in 1983, although values were higher than the control area Sørumsand.

The comparison between the values measured in this study and those measured in Holmestrand are especially interesting since: 1) methodology in the two studies was identical and 2) air lead values were higher in Oslo-Nydalen.

However, air lead exposure was shown to have a significant relationship with measured blood lead concentrations in women and children, a relationship that was, as in Holmestrand, lacking in men. Earlier studies have indicated that a $1 \ \mu g/m^3$ decrease in the concentration of air lead will lead to a 1 to 2 $\mu g/100$ ml decrease in blood lead concentrations (see discussion Clench-Aas et al., 1984) or a 1 or 2 to 1 ratio. In the children measured in this study, the calculated regression coefficients (Table 17) indicate a drop of 1.2 $\mu g/100$ ml lead in blood with a drop of 1 $\mu g/m^3$ (from 1.5 to 0.5 $\mu g/m^3$) in air for boys, and a drop of 1.3 $\mu g/100$ ml for girls. In women the same decrease in air lead yields a drop of 0.9 $\mu g/100$ ml.

The passive smoking effect, found so clearly in Holmestrand in 1983 were totally lacking in Oslo-Nydalen.

The findings of this study did not indicate any measurable correlation between blood lead concentrations and zinc protoporphyrin, hematocrit or hemoglobin either in children or adults at the existing low levels of lead in blood.

One can, at this point, only hypothesize as to why the values for blood lead measured in Oslo-Nydalen are lower than those measured in Holmestrand despite higher air lead values.

It is possible, that lead in industrial emissions is bound to larger size particles than lead emissions from car exhaust, thus leading to less penetration into the alveoli of the lung. This would also explain why passive smoking was so important in the Holmestrand study and not in the Oslo-Nydalen study since passive smoking impairs the function of the respiratory cilia, thus decreasing respiratory clearance allowing more particles to descend into the alveoli. It is also possible that lead from the two sources exist in different chemical forms having different uptake by the body. Work done by Chamberlain et al. (1978) seems to indicate the latter to be an unlikely explanation. Yet a third possibility is that air lead values stemming from the industrial exposure in Oslo-Nydalen, because of both geography and weather conditions, showed rather large daily variations. Values were, in fact, high for rather short periods of time. It is possible that the Holmestrand values have been higher over longer periods of time during the 24 hours. This may impact lead uptake by the lung.

It is important to pursue these findings further. They do give a possible explanation for major inconsistencies in the literature. For example, the studies of the NHANES data (Annest et al., 1982, 1983, Pirkle, 1983 and Schwartz, 1983) found a larger drop in blood lead concentrations with a decline of lead in gasoline than predicted by the literature. Chamberlain et al. (1978) found higher blood to air lead ratios when examining individuals exposed to air lead from motor vehicles than predicted. He also found higher uptake from individuals breathing air near a roundabout or motor way than general urban air. The coefficients we have found in Oslo-Nydalen under low to moderate exposure correspond fairly well to those described in the literature. Most studies sited in the literature have been done in regions of high lead exposure where the source has been high industral emissions (2 to 3 μ g/m³ air lead concentrations).

Not only is lead a very important pollutant of its own, but it can also be considered an excellent tracer for other particulate-bound pollutants. The findings suggested by these two studies need to be examined in depth, preferably in the animal model. Should the first hypothesis (based on differences in particle size) be confirmed, the possibility exists that uptake of other particulate-bound pollutants differ when pollution sources vary in the size of the emitted particles.

The relationship of alcohol consumption and smoking with blood lead was reported earlier in Sweden (Elinder et al., 1983). The authors of that study indicated high lead content in wine and home-made spirit as the possible source. Contrary to the Swedish study, this study did find an age effect that was lacking in Holmestrand. In Holmestrand the elderly lived in a home for the elderly situated by the main road. However, they had not lived by this road their whole lives. They came from all over the district, a district which can be considered to have very low values of ambient lead. Here we found no higher blood lead concentrations among the elderly. In Oslo-Nydalen, ambient lead concentrations have been higher in the past than current levels. Lead emission from one of the industrial sources has decreased in the later years. In addition, lead content in gasoline has been reduced from 0.45 to 0.15 g/l, resulting in reductions of values of lead in air of ca 50%. The homes for the elderly in Oslo take preferentially people who have lived locally for a long time. Therefore, although the homes for the elderly are situated in low exposure areas, the population in them could have been exposed to higher concentrations over their life-time. In this population we found substantially higher blood lead concentrations in the elderly, especially elderly women, than predicted by current air lead exposure alone (see Table 14, Figure 8 and 11). In the previous Holmestrand study, no age effect was measured despite high current ambient lead exposure but where lifetime exposure had been low. These high values of blood lead can represent, therefore, a possible resorption of lead deposition in bone. If we assume that all of the increase attributed to age comes from bone resorption, then the difference between an 80 year old and a 20 year old woman is 2.3 μ g/100 ml (using the coefficients presented in Table 17). This represents the possibility that 30% of an average of 9.4 μ g/100 ml for women over 70 is coming from resorption from bone.

Although age in children has frequently been discussed as being very important and leading to much higher blood lead values, one must remember that these statements have generally concerned 2-3 year olds. The children in both this and the Holmestrand study ranged from 2 upwards, with an average age of 10 years. However, the calculated regression coefficients (Table 17) do indicate a higher uptake of air lead by children than adult women.

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5 REFERENCES

- Annest, J.L., Pirkle, J.L., Makuc, D., Neese, J.W., Bayse, D.B., Kovar, M.G. (1983) Chronological trend in blood lead levels between 1976 and 1980. <u>N.</u> <u>Eng. J. Med.</u>, <u>308</u>, 1373-7.
- Annest, J.L., Mahaffey, K.R., Cox, D.H., Roberts, J. (1982) Blood lead levels for persons 6 months - 74 years of age. United States, 1976 - 80. <u>Advance Data</u>, <u>79</u>, 1 - 18.
- Azar A., Snee, E.O., Habibi, K. (1975) An epidemiological approach to community air lead exposure using personal air samples. In: <u>Lead</u>. Ed by T.B. Griffin and J.H. Knelson. London, Acad. Press. pp 254-290.
- Billick, I.H., Curran, A.S., Shier, D.R. (1979) Analysis of pediatric blood lead levels in New York city for 1970 - 1976. <u>Environ. Health Perspec.</u>, <u>31</u>, 183 - 190.
- Chamberlain, A.C. (1983) Effect of airborne lead on blood lead. <u>Atmos.</u> <u>Environ.</u>, <u>17</u>, 677-692.
- Chamberlain, A.C., Heard, M.J., Little, P., Newton, D., Wells, A.C., Witten, R.D. (1978) Investigations into lead from motor vehicles. Harwell, H.M. Stationary Office. (AERE-R 9198).
- Clench, J., Ferrel, R.E., Schull, W.J., Barton, S.A. (1981) Hematocrit and hemoglobin, ATP and DPG concentrations in Andean Man; the interaction of altitude and trace metals with glycolytic and hematologic parameters in man. In: <u>The Red Cell: Fifth Ann Arbor Conference</u>. Ed. by G.J. Brewer. N.Y., Alan R. Liss.
- Clench-Aas, J., Thomassen, Y., Levy, Y., Skaug, K. (1984) Blood lead a function of vehicular emissions and smoking. Parts I and II. Lillestrøm (NILU OR 44/84).
- Duan, N. (1982) Models for human exposure to air pollution. <u>Environ</u>. <u>Int.</u>, <u>8</u>, 305 - 309.
- Elinder, C.G., Friberg, L., Lind, B., Jawaid, M. (1983) Lead and cadmium levels in blood samples from the general population of Sweden. <u>Environ. Research</u>, <u>30</u>, 233-253.

- Grønskei, K.E., Gram, F., Larssen, S. (1982) Beregning av sprednings- og eksponeringsforhold for visse luftforurensningskomponenter i Oslo. Lillestrøm (NILU OR 8/82).
- Hammond, P.B., O'Flaherty, E.J., Gartside, P.S. (1981) The impact of air-lead on blood-lead in man - a critique of the recent literature. <u>Ed.</u> <u>Cosmet Toxicol.</u>, <u>19</u>, 631-638.

Jacobsen, P.H. (1982) Håndbok i DDPP. Oslo, Univ. forl.

- McIntire, M.S., Angle, C.R. (1979) Air lead/Blood lead in G-6-PD deficient black school children. In: <u>Environmental Health Aspects of Lead</u>, proceedings International Symposium, Amsterdam Oct. 2-6.
- Moschandreas, D.J. (1981) Exposure to pollutants and daily time budgets of people. <u>Bull. N.Y. Acad. Med.</u>, <u>57</u>, (10), 845 859.
- Omang, S.H., Moseng, J. (1974) Bestemmelse av bly i blod fra barn. <u>T.</u> norske lægeforening, <u>94</u>, 1074 - 1075.
- Pirkle, J.C. (1983) Chronological trend in blood lead levels in the United States 1976 and 1980. Presented at the International Conference on Heavy Metals in the Environment. Heidelberg, West-Germany. September, 1983.
- Schwartz, J. (1983) The relationship between gasoline lead and blood lead in Americans. Presented at the International Conference on Heavy Metals in the Environment. Heidelberg, West Germany. September, 1983.
- Skrede, K. (1971) Sosioøkonomisk klassifisering av yrker i Norge, 1960. Institutt for anvendt sosialvitenskapelig forskning. (Rapport nr. 71-1).
- Snee, R.D. (1981) Evaluation of studies of the relationship between blood lead and air lead. Int. Arch. Occup. Environ. Health, <u>48</u>, 219-242.

Statistisk Sentralbyrå. (1980) Sosialt utsyn.

Statistisk Sentralbyrå (1976) Yrke og dødelighet. 1970-1973.

- Stock T.H., Holguin, A.H., Selwyn, B.J., Hsi, B.P., Contant, C.F., Buffler, P.A., Kotchmar, D. (1983) Exposure estimates for the Houston area. Asthma and runners studies. In: Int. Symp. on the Biomedical Effects of Ozone and other Photochemical Oxidants, 1982. Proceedings. Princeton, Princeton Sci.Publ. (Advances in modern environmental toxicology. Vol. V), pp. 527-536.
- United States Environmental Protection (1977) Air quality criteria for lead. Wash. D.C.
- Vitols, V. (1983) Ambient particle losses in the SK sampling system. Lillestrøm (NILU TR 14/83).
- Astrand, P.O., Rodahl, D. (1977) Textbook of Work Physiology. N.Y., McGraw Hill.

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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, offered their blood and were always pleasant and smiling.

APPENDIX I

Air Lead Values Used For Oslo-Nydalen and Sørumsand

		0:	slo-Nyda.	len		Down-	Down-	Back-	Wind*	Wind*
	N	Ε	S	W	SE	town Oslo	town Oslo	ground Oslo	direct	speed
Date	O.T. vei	Grefsen skole	Nydals- veien	Bakke- haug- veien	Sagene brann- stasjon	St. Olavs plass		Nordahl Bruns gate	Degrees measured Blindern	
Jan. 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 30 31	0.11 0.08 0.38 0.09 0.08 0.02 0.09 0.06 0.20 0.20 0.20 0.20 0.02 0.01 0.06 0.10 0.13 0.02 0.01 0.08 0.04 0.04	0.11 0.32 0.04 0.02 0.05 0.25 0.21 0.02 0.02 0.03 0.06 0.07	0.04 0.21 0.04 0.03 0.05 0.33 0.20 0.54 1.08 1.01 1.13 0.08 1.06 0.84 2.04 1.38 1.33 0.65 0.80 1.21 0.41	0.31 0.29 0.08 0.07 0.08 0.15 0.21 0.05 0.07 0.06 0.85 0.60					50 80 80 70 40 50 70 70 70	4.0 3.5 3.5 4.5 4.0 3.5 3.5
Febr. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29	0.08 0.04 0.02 0.09 0.04 0.05 0.02 0.03 0.76 0.42 0.17 0.29 0.48 1.59 0.54 0.54 0.09 0.54 0.09 0.08 0.04 0.02 0.02 0.02 0.02 0.03 0.04 0.03 0.04 0.05 0.15 0.02 0.15 0.15 0.15 0.15 0.15 0.15 0.15 0.15	0.12 0.07 0.03 0.12 0.09 0.07 0.04 0.06 0.91 0.41 0.22 0.11 0.22 0.88 0.55 0.11 0.12 0.14 0.09 0.04	0.32 0.19 0.07 0.10 0.02 0.11 1.27 1.73 0.55 0.35 0.05 0.15 1.03 0.47 0.07 0.12 0.08 0.66 5.00 2.78 0.70 0.86 0.31 0.10 0.19 0.27 0.37 0.14	0.13 2.32 1.13 0.04 0.05 0.20 0.11 0.79 0.40 0.21 0.20 0.21 0.20 0.12 0.20 0.12 0.12	0.12 0.05 0.07 0.14 0.03 0.05 0.38 0.57 1.42 0.49 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.04 0.23 0.05 0.11 0.11 0.11 0.11 0.11 0.11 0.11	0.47 0.31 0.30 0.40 0.22 0.37 0.35 0.27 1.48 0.72 0.52 0.49 0.72 0.52 0.43 0.33 0.38 0.31 0.36 0.24 0.53 0.39 0.53 0.37 0.22 0.46 0.49 0.83	0.71 0.79 0.84 0.11 0.25 0.79 0.82 2.04 1.15 0.62 0.27 0.59 1.14 0.98 0.38 0.38 0.25 0.44 0.89 0.59 0.861 0.75 0.461 0.75 0.46 0.34 0.77 0.63 0.52	0.39 0.16 0.24 0.07 0.24 0.22 1.13 0.49 0.25 0.49 0.25 0.49 0.27 0.02 0.11 0.10 0.25 0.24 0.10 0.45 0.30 0.21 0.14 0.15 0.23 0.10 0.10	45/190/80 60 50 60/200 210 200/140 80/0/60 15/50 230/90/60 190/70 70/260 230. 250/40 unruly 90/200 190/70 200 210/60 190/70 200 210/60 70 70 60 60 230 230/60 50/100/70 50/90/210 260 240	3.5 4.0 3.0 2.0 2.0 2.0 2.0 2.0 2.0 2.0 2.0 2.5 6.0 3.0 2.5 6.5 5.0 3.0 3.0 3.0 3.0 3.0
March 1 2 3 Febr.	0.17 0.02 0.68	0.43 0.04 0.49	0.34 1.98 0.36	0.14 0.11 0.36			0.94	1.21	250 250/50 250	6.5 7.5 4.0
Stand. dev.	0.29	0.24**	0.63	0.34	0.27	0.45	0.70	0.24		

Table 1.1: Lead in air $(\mu g/m^3)$ in Oslo - winter 1984. Outdoor lead values used in estimating personal exposure.

* Wind direction and wind speed reflect averages of 24 (hourly) measurements made at a single site to the west of the area. Where wind direction has shifted during the day, the main directions are indicated. **missing values excluded.

-2: Measured and estimated air lead concent day period in Sørumsand used in calcu lead exposure. Measurements made in May	a 16	air	
-2: Measured and estimated air lead concentration day period in Sørumsand used in calculating lead exposure. Measurements made in Mav 1984.	-	indivi	
-	: Measured and estimated air lead concentration	day period in Sørumsand used in calculating	lead exposure. Measurements made in May 1984.

Car rum travelling
Oslo Bærum
Lillestrøm
In the country
Station 4
Station 3
Station 2
Station 1
Date

APPENDIX II

Miscellaneous general information used in this report

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

SOSIALGRUPPER

SOCIAL CLASSES

Yrkesområde i Standard for yrkesgruppering i offentlig norsk statistikk Occupation by Standard of Occupational Classifications

Sosialgruppe Social class		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, viten- skapelig, humanistisk og kunstnerisk arbeid, 10 Offentlig administrasjons- og for- valtningsarbeid, 11 Bedrifts- og organisasjonsledelse, 31 Salg av fast eiendom, tjenester, verdipapirer, forsikringer, brukte ting m.m.
Sosialgruppe	В	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 detaljhandels- arbeid, 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
u	С	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
u	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 tilvirkings- arbeid, 86 Pakke- og emballeringsarbeid, 88 Laste-, losse- og lagerarbeid, 89 Diversearbeid innen industri-, bygge- og anleggsarbeid, 91 Hotell- og restaurant- arbeid, husarbeid, 95 Vaske-, rense- og strykearbeid
u	Ε	40 Arbeidsledelse i jord- og skogbruk, 41 Jordbruksarbeid, dyrerøkt

From: Yrke og dødelighet.

YRKESOMRÅDER PÅ 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
- OX Other work in major group O
- 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

From: Yrke og dødelighet.

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 Va	luc	Mengde	Quantity	Mengde pr. person
	Kr Kroner	Prosent Per cent	Mengde- enhet Quantity unit	Kg/1 Kg/litre	pr. dag Quantity per person per day
					Gram/ml.
Aatvarer i alt Food, total	13 275	100.0			
0 Mjøl, gryn og bakervarer Flour, meal					
and bakery products	1 193	9,0	kg	225.3	220.
001 Mjøl og grvn Flour and meal	194	1,5	**	106,4	104.
002 Kjeks, flatbrød og knekkebrød	1.24	1.0		0.0	0
Crisphread, biscuits etc	136 534	1,0 4,0	89	8,9 93,4	8. 91.
004 Kaker <i>Cakes</i>	269	2,0	20	12.5	12.
005 Makaroni og cornflakes					
Macuroni and cornflakes	60	0.5	30	4,1	4.
I Kjøtt, kjøttvarer og flesk					
Meat. meat products and pork	3 685	27.8	44	127,2	- 124
011 Ferskt kjøtt og flesk Fresh meat and pork	1 684	12,7	20	68.9	67
012 Saltet, røykt og tørket kjøtt og flesk Salted, smoked and dried meat	522	3.9	55	11.3	11
013 Kjøtthermetikk Canned meat	150	1,1	10	5,8	5
014 Andre kjøtt- og fleskevarer Other				5,5	2
meat and pork products	1 253	9,4	30	39,1	38
015 Fryst kjøtt og kjøttvarer Frozen meat					
and meat products	76	0.6	34	2,1	2
2 Fisk og fiskevarer Fish and fish products	948	7,1	54	68,0	66
021 Fersk fisk Fresh fish	276	2.1	24	28.8	28
022 Fryst fisk Frozen fish	118	0,9		7.3	7
023 Saltet, røykt og tørket fisk og skalldyr	198	1,5	10	11.8	11
Salted, dried and smoked fish and shellfish	60	0.5	*	6,7	6
025 Småhermetikk Sardines etc.	125	0,9	10	3.4	3
026 Andre fiskevarer Others fish products	171	1.3	66	10,1	9
3 Mjølk, fløte, ost og egg					
Milk. cream. cheese and eggs	2 165	16,3	10	545.9'	533
031 Mjølk <i>Milk</i>	953	7.2	liter	464.0	453
032 Flate Cream	294	2.2	56 T	15.1	14
034 Ost Cheese	492 426	3.7	kg	25.2 27,2	24 26
035 Egg Eggs 4 Spisefett og -oljer Edible oils and fats	461	3.5	24	57.2	55
041 Smør Butter	145	1,1	14	9,2	9
042 Margarin og spiseolje					
Margarine and edible oils	316	2.4	34	47,9	46
5 Grønnsaker, frukt og bær Vegetables.					
fruits and herries	2 226	16.8	10	269.8	263
051 Kal og gulrøtter Cabhage and carrots	225	. 1.7	20	48.3	47
052 Andre friske grønnsaker Other fresh vegetables	304	2.3	18	28.6	28
053 Epler, pærer, plommer Apples, pears and plums	360	2.7	58	55.2	54
054 Sitrusfrukter, bananer og druer Citrus fruits, bananas and grapes	423	3,2	10	63,8	62
055 Tarket frukt og nøtter Dried fruits and nuts	116	0.9	3.0	6,4	6
056 Bær Berries.	183	1.4	**	17.4	17
057 Konserverte grønnsaker Preserved vegetables	240	1.8	40	15.2	14
058 Konserverte frukt og bær Preserved fruits					
and herries	375	2,8	**	34.8	34
Poteter og varer av poteter	140	3.6		204 9	200
Potatoes and potato products	469 375	3.5 2,8	58	204,8 200,0	195
061 Poteter Polatoes	94	0,7	18	4,8	4
Sukker Sugar	146	1.1	24	44,3	43
Kaffe, te, kakao og kokesjokolade					
Coffee, tea, cocoa and chocolate	970	7.3		22.6	22
081 Kaffe Coffee	862	6,5		20.3	19
082 Te Tea	50	0.4	**	0.5	C
083 Kakao og kokesjokolade Cocoa and chocolate	58	0.4	**	1.8	1
Andre matvarer Other foods	1 012	7.6	99	27.7	27
091 Spisesjokolade og drops Chocolate and sugar confectionerv	362	2,7	55	9.1	8
092 lskrem <i>lcc-cream</i>	166	1.3	**	8.3	8
093 Annet Other	484	3.6		10.2	10

t Emolk er regnet fik 1.03 kg.

From: Sosialt utsyn.

APPENDIX III

Correlation coefficients between measured variables in Oslo-Nydalen, in children and in adults Table III-1: Correlation matrix of pertinent data variables a) in children, b) in adults in Oslo-Nydalen.

al Children Correlation matrix	n n matrix												
Variables Log B-Pb	Log 8-Pb	Log ZPP	MCHC	Н	НВ	LogPb-A	Sex	Pass. smok.	Soc.cl	Vitam.	Iron	Snoweat	Age
Log B-Pb Log ZPP	1.000-	1.000											
MCHC	-0.136 -0.049	0.221**	1.000 -0.292***	1.000									
HB	-0.129	0.032	0.313***	c **	1.000								
Log Pb-A	-0.068	0.121	-0.015		0.078	1.000							
Sex	-0.175*	0.183*	-0.092	-0.036	-0,092	-0.132	1.000						
Pass.smok.	0.016	0.030	0.189**	-0.049	0.063	0.073	-0.091	1.000					
Soc.cl.	0,049	-0.349***	-0.254***	0.146	-0.008	-0.000	-0.079	0.195**	1.000				
Vit.	-0.187**	0.127	-0.085	0.047	-0.009	0.114	0.083	0.079	-0.166**	1.000			
Iron	-0.168*	0.028	0.050	0.002	0.026	0.133	0.038	0.016	0.030	0.180**	1,000		
Snoweat	0.072	-0.105	-0.130	0.015	-0.066	-0.033	0.052	-0.028	0.097	0.068	-0.059	1.000	
Age	-0.128	0.084	0.006	0.338***	0.339***	0.037	0.042	-0.027	-0.248***	0.242***	0.233**	0.233*** -0.152*	1.000
Analyses d Probabilit Sample siz Children w	lone using U y values in es are vari ho admitted	Analyses done using ODPP (Jakobsen, 1982). Probability values indicated: *<0.05; **<0.025, ***<0.001. Sample sizes are variable and range from 121 to 183. Children who admitted to smoking were removed from analysis.	en, 1982). 0.05; **<0. nge from 12 were remov	025, ***<0 1 to 183. ed from an	.001. alysis.								

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Table III-1: continues

b) Adults.						3							
Correlation matrix	n matrix												
Variables Log B-Pb	Log B-Pb	Log ZPP	MCHC	HT	HB	LogPb-A	Sex Sin	Sınoking	Soc.cl	Vitam.	Iron	Alcohol	Age
												.dwnsuoo	
l.og 8-Pb	1.000												
Log ZPP	-0.083	1.000											
MCHC	-0.007	-0.103	1.000										
HT	0.057	-0.477***	0.062	1.000									
HB	0.049	-0.462***	0.470***	0.909***	1.000								
Log Pb-A	-0.136*	-0.036	0.107	0.080	0.118	1.000							
Sex	-0.211***	0.344***	-0.155**	-0.556***	-0.560***	-0.120	1.000						
Smoking	-0.083	0.064	0.152**	0.023	0.088	0.012	-0.095	1.000					
Soc.class	0.056	-0.045	-0.194***	-0.026	-0.105	-0.156**	0.116	-0.051	1.000				
Vitan.	-0.014	0.051	0.165**	-0.102	-0.024	-0.039	0.236***	0.070	0.036	1.000			
Iron	-0.143**	0.106	-0.035	-0.176**	-0.176**	0.071	0.225***	0.023	-0.010	0.283***	1.000		
Alch.cons.	0.025	-0.118	-0.177**	0.062	-0.019	0.140**	0.017	0.028	0.178**	-0.076	-0.085	1.000	
Age	0.328***	-0.078	-0.309*** -0.003	-0.003	-0.128*	-0.314*** -0.045	-0.045	-0.096	0.364*** -0.054	-0.054	-0.241***	0.231	1.000
Analyses do	one using D	Analyses done using DDPP (Jakobsen, 1982).	en, 1982).										
Probability	y values in	dicated: *<	0.05; xx<[Probability values indicated: *<0.05; **<0.025; ***<0.001	1.00.								
Sample size	es are vari	Sample sizes are variable and range from 251 to 271.	nge from 2	51 to 271.									
Uccupation	aliy expose	d individua	ils are ren	Uccupationally exposed individuals are removed from analysis.	nalysis.								2

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TITTEL Lead in blood in inhabi exposed to air lead fr	tants in Oslo-Nydalen	PROSJEKTLE J. Clench-	
vehicular sources	UN INGUSCIIAI ANG	NILU PROSJI 0-8372	EKT NR.
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3 STIKKORD (à maks. 20 Blood lead		ematologic j	parameters
industri (Nydalen i Oslo renset sted (Sørumsand) Nydalen. Korrelasjonen regnet eksponering til som alder eller alkohol	ag, 7 linjer). o befolkningsgrupper – e: o), og en kontrollgruppe . Konsentrasjonen av bly mellom konsentrasjonen a bly i luft var signifika: forbruk, var imidlertid onsentrasjonen av bly i	fra et lit i blod var v bly i bloc nt. Andre fa like viktige	te foru- høyest i l og be- aktorer,
TITLE			
ABSTRACT Blood lead was to moderate lead emission	s measured in two popula ons from traffic polluti	tions - one on and indu	exposed strialized

ABSTRACT Blood fead was measured in two populations one exposed to moderate lead emissions from traffic pollution and industrialized sources (Oslo-Nydalen), and a control low exposure town (Sørumsand). Blood lead concentrations were higher in Oslo-Nydalen. Blood lead values were significantly correlated to exposure to air lead. Other factors such as age or alcohol consumption seemed as important as their lead in determining blood lead concentrations.

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