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PREFACE

In 1987, the Norwegian Institute for Air Research (NILU), in co-operation with the Institute for Aviation Medicine and the National Institute of Occupational Health, planned an investigation of the health effects of air pollution from vehicular traffic on the inhabitants of the Vålerenga region of Oslo. This investigation was designed as a set of studies on different facets of the health effects of exposure to air pollution. The investigation was carried out within the "Traffic and Environment" Research Program under the auspices of the Royal Norwegian Council for Scientific and Industrial Research (NTNF).

CONTENTS

SUMMARY

Though it is often stated that traffic pollution has negative effects on health and well being (e.g. nervousness, coughing), there is very little quantitative information available on just how severe this effect may be in comparison to other known factors, such as age or smoking.

In the fall of 1987, circa 1000 randomly chosen adults participated in a cross-sectional study on the relationship between traffic pollution exposure and symptoms of health effects in Norway. The study was performed within a 2x2 km area in Oslo, where traffic is the single most important pollution source. It included a large questionnaire administered by trained personnel, and measurements and modelling of traffic air and noise pollution.

Air pollution modelling was based on extensive meteorological and pollution measurements. For each participant an index of air pollution due to traffic was calculated for his/her home address. Pollution levels measured ca. 40 meters from heavy traffic, were for most typical poor-dispersion situations under air quality guideline values. The maximum 1-hr CO concentrations measured were 25-30 mg/m^3, 8-hr maximum 15-20 mg/m^3, and 1-hr NO_2 maximum 250-300 $\mu g/m^3$.

The symptoms of health effects and well-being were reported as: "not", "sometimes" or "often bothered", based on symptoms experienced during the preceeding 6 months. To relate the symptoms to the air pollution index we used logistic regression, with explanatory factors such as age, sex, smoking habits, education level and marital status.

The most important explaining factors were sex and age. The results showed, however, that for some symptoms (cough, chronic bronchitis, muscle pains, tiredness, eye irritation, and headache) the air pollution index at values usually under guideline concentrations was an important explaining factor. For upper

airways symptoms the effect of air pollution at low pollution levels is comparable to the effect of current smoking.

In an attempt to evaluate at what level traffic pollution reduces health and well being, a cohort study was designed that associated air pollution exposure and reporting of health symptoms and the measurements of peak expiratory flow (PEF) for 160 individuals hour for hour for two weeks.

Air pollution exposure was estimated for each hour and each individual using known vehicular emission rates, estimated traffic counts for each road segment, and meteorological conditions. NO_2 was used as an indicator substance for traffic pollution in general.

Fatigue, sneezing, sore/irritated throat, tight chest, annoying smell and annoying noise were significantly associated with traffic pollution. At the hourly air quality guidelines of 200 $\mu g/m^3$ NO_2, the increased risk of having each of these symptoms compared to the risk at at an exposure of 10 $\mu g/m^3$, (low to moderate exposure) is indicated in the following table (a value of 1.84 for sneezing, for example means a 84% increased risk):

	Relative risk at 200 $\mu g/m^3$ NO_2	100 $\mu g/m^3$ NO_2
Fatigue	1.23	1.17
Sneezing	1.84	1.60
Sore/irritated throat	1.67	1.48
Tight chest	1.50	1.37
Annoying noise	3.22	2.45
Annoying smell	2.94	2.29

Variations in PEF did not show significant relationships to air pollution exposure.

In addition, CO was measured in blood and breath, lead in blood and a full lung function test was done for each of the 160 participants. Body burdens of lead or CO did not show high levels that could be attributed to excessive exposure to air pollution. Increased lung function was correlated to concentrations of CO in blood, however, the results should not necessarily be interpreted as a cause-effect relationship.

SUMMARY IN NORWEGIAN

Luftforurensning ansees ofte å være en årsak til virkninger på helse og velvære, men det er svært lite kvantitativ informasjon om omfanget av effekten sammenlignet med andre faktorer, som alder og røyking.

Høsten 1987 deltok ca. 1000 tilfeldig utvalgte voksne individer i en tverrsnittsundersøkelse av effekten av trafikkforurensninger på symptomer på redusert helse i Norge. Undersøkelsen ble utført i et 2x2 km^2 stort område i Oslo, hvor trafikk er hovedkilden til luftforurensning. Et omfattende spørreskjema ble fylt ut under ledelse av trenet personell. Målinger og modellberegninger av eksponering for luft- og støyforurensninger var også inkludert i undersøkelsen.

Til modellberegninger av luftforurensninger ble det brukt data fra målinger av meteorologi, trafikk og luftforurensninger. En luftforurensningsindeks ble beregnet som representerte boligmiljøet for hver deltaker. Aktuelle målinger av luftforurensninger ga nivåer som ofte lå under nåværende retningslinjer for luftkvalitet. Maksimale 1-timers verdier som ble målt var for CO: 25-30 mg/m^3 (8-timers CO: 15-20 mg/m^3), og for NO_2: 250-300 $\mu g/m^3$.

Deltakerne rapporterte om de var "ofte", "av og til" eller "ikke" plaget av en rekke helsesymptomer de siste 6 månedene. Logistisk regresjon ble benyttet for å studere samvariasjon mellom boligindeksen for luftforurensning for den enkelte og rapportering av helsesymptomer. Kompliserende faktorer (kjønn, alder, røykevaner, utdannelsesnivå og ekteskapelig status) ble tatt med i analysen.

De viktigste faktorene som forklarte variasjonen i resultater, var kjønn og alder. Resultatene viste også at for noen parametere (hoste, kronisk bronkitt, muskelsmerter, tretthet, øyeirritasjon og hodepine) var indeksen for luftforurensning i bolig en viktig forklarende parameter selv med verdier under

nåværende retningslinjer for luftkvalitet. For noen av de øvre luftveissymptomer var effekten av luftforurensning av samme størrelsesorden som effekten av røykevaner.

For å kunne vurdere på hvilket nivå trafikkforurensningen påvirket helse og velvære, ble det gjennomført en kohortundersøkelse (panelundersøkelse) hvor 160 deltakere ble fulgt i 2 uker, time for time. Undersøkelsen sammenholdt beregnet eksponering for luftforurensning og rapportering av symptomer og registrering av toppstrømshastighet (PEF) på timesbasis.

Eksponering for luftforurensning ble beregnet for hver time ved å bruke informasjon om utslipp og antall biler for hver veilenke, samt informasjon om meteorologi. NO_2 ble brukt som indikator for trafikkforurensning generelt.

Tretthet, nysing, halsirritasjon, tetthet i brystet, lukt- og støyplager hadde betydelig samvariasjon med trafikkforurensning. På nivåer av NO_2 som tilsvarer nåværende retningslinjer for luftkvalitet for timesmiddel (200 $\mu g/m^3$), var risikoen for å bli plaget av disse symptomene (i forhold til et lavt/moderat eksponeringsnivå på 10 $\mu g/m^3$) følgende:

	Relativ risiko	
	200 $\mu g/m^3$ NO_2	100 $\mu g/m^3$ NO_2
Tretthet	1,23	1,17
Nysing	1,84	1,60
Halsirritasjon	1,67	1,48
Tetthet i brystet	1,50	1,37
Luktplager	3,22	2,45
Støyplager	2,94	2,29

Variasjoner i PEF viste ingen signifikant sammenheng med eksponering til trafikkforurensning.

I tillegg ble det målt bly i blod, CO i blod og pusteluft og det ble gjort en full lungefunksjonstest hos de 160 deltakerne i Trinn 2. Konsentrasjoner av bly og CO viste ingen tegn på forhøyede verdier som funksjon av eksponering til høye konsentrasjoner av luftforurensning. Det ble påvist en signifikant sammenheng mellom redusert lungefunksjon og CO i blod, uten at den sammenhengen kunne påvises å være en årsak- virkningsammenheng.

THE HEALTH EFFECTS OF TRAFFIC POLLUTION AS MEASURED IN THE VÅLERENGA AREA OF OSLO

SUMMARY REPORT

1 INTRODUCTION

As the number of vehicles on the road increases, concern for traffic pollution and its effects grows. Concern focuses on the feared, but largely unknown effects of traffic pollution on human health and well being.

Traffic pollution includes both air and noise pollution. Air pollution components include carbon monoxide (CO), nitrogen oxides (NO_x), particles (both from exhaust and studded tires), lead (Pb) and various organic compounds.

Through animal studies it is known that exposure to NO_2 and particles can result in morphological changes in the lung. The results of human experimental chamber studies have been unclear as to what exposure levels can precipitate reactions in the form of increased pulmonary sensitivity (WHO, 1987).

The Norwegian Institute for Air Research (NILU) has designed an investigation program to explore the health effects of air pollution from vehicular traffic. This investigation was done in co-operation with the Institute of Aviation Medicine and the Institute of Occupational Health and was integrated in a study of environmental effects of traffic in the Vålerenga area, a study within the "Traffic and Environment" Research Program. In the Vålerenga area in Oslo, a major highway (35 000 vehicles per day) passed through a residential area. A tunnel was built that led to major changes in traffic distribution in the area. This allows studying individuals' health before and after these changes.

The goals of the investigation were to answer the following questions:

1) Does air pollution from vehicular traffic have an effect on human health?

2) Do the current air quality guidelines protect the population from health effects?

3) Are some population subgroups more susceptible to the undesirable effects of air pollution than others?

The definition of health used here is that given by the World Health Organization (WHO) that includes "a state of physical, mental and social well being, and not only the absence of disease and disability" (WHO, 1985). In order to measure the effects of air pollution on well being, a set of symptoms reflecting health and well being were listed in a diary so that each individual could report on these symptoms continuously during the investigation period.

There are certain confounders that must be accounted for in investigating the health effects of air pollution from vehicular traffic. Vehicular traffic leads to both noise and a variety of air pollutants. Thus, it is essentially impossible to distinguish between the effects of each pollutant. However, since the compounds usually covary with each other, one compound can be used as an indicator substance for the others.

In order that epidemiological studies can be of help in defining air quality guidelines, it is necessary to adequately quantify air pollution exposure. This study estimated exposure using modelling together with air quality measurements.

2 STUDY DESIGN AND METHODS

2.1 DESCRIPTION OF AIR POLLUTION CONCENTRATIONS IN THE STUDY AREA - VÅLERENGA/GAMLEBYEN IN OSLO

The area of investigation is a relatively heavily populated area in the city of Oslo (Figure 1).

A major highway crossed the area (35 000 vehicles daily). The opening of a tunnel altered the traffic distribution and led to substantial reductions in the amount of traffic through the area.

Several smaller regions were selected within the area for closer investigation. The subregions were selected based on their different exposures to traffic. Some were representative of areas with decreasing amounts of traffic after the opening of the tunnel whereas others were indicative of areas of increased amounts of traffic. The sub-areas are shown in Figure 1.

The heavy traffic on the main roads of the area reults in a high air pollution level which, during periods of poor dispersion, exceeds recommended air quality guidelines.

The location of air pollution monitoring stations is shown in Figure 1.

Table 1 shows maximum short term and average concentrations of CO, NO_2, soot ("black smoke") and PM_{10} (inhalable particles) measured at the stations along the main road through the area. The maximum short term concentrations (1-24 hour averages) exceed air quality guidelines in a belt along this and the other main roads. The highest measured NO_2 concentration exceeded the guideline by a factor of almost 3, while the highest PM_{10} concentrations exeeded the WHO guideline by a factor of about 7, and the US Primary Standard by a factor of 3.

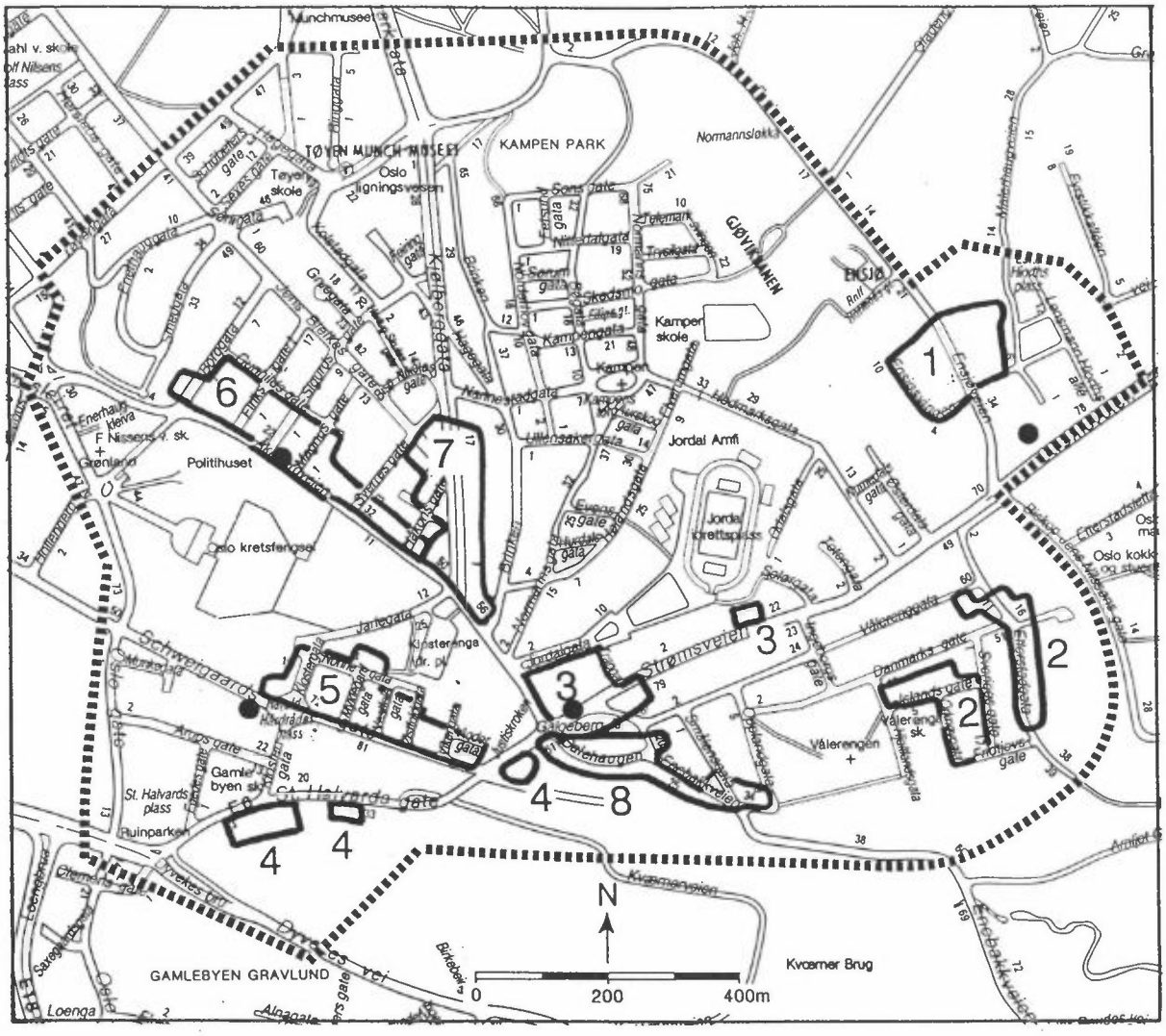


Figure 1: Area of investigation in Vålerenga in Oslo with sub-regions indicated. (●) indicate placement of air quality monitoring stations.

Table 1: Maximum and average air pollutant concentrations measured near the main road (Strømsveien) through the study area.

	Galgeberg (Curbside station)	Malerhaugen (25 m from road)	Etterstadgt. (50 m from road)	Air quality guideline[1]
CO (mg/m^3)				
Max. hourly average	29		19	25
Max. 8-hour average	20		15	10
NO_2 ($\mu g/m^3$)				
Max. hourly average		211	263	200
Max. 24-hour average	220	196	156	100
3 month average		63	56	75[2]
Black smoke (soot)($\mu g/m^3$)				
Max. 24 hour average		289	181	100
3 month average		61	42	40
PM_{10} ($\mu g/m^3$)				
Max. 24 hour average	450		156	70[3]
3 month average			49	150[4]

[1]) Recommended air quality guideline for Norway
[2]) 6 month average guideline
[3]) WHO preliminary recommended guideline
[4]) US Primary Air Quality Standard

Results of continuous and integrated measurements at Galgeberg (CO, NO_2 and PM_{10}) and at Malerhaugen (NO_x) are shown in Figures 2 and 3, respectively, for the field study period (20 October-20 November 1987). The figures show the typical variations in air pollutant concentrations due to daily traffic variation and variation in dispersion conditions. The study period included distinct air pollution episodes with very poor dispersion and high concentrations in the entire area.

Simultaneous outdoor/indoor measurements were also made of NO_2 and PM_{10} at two locations, outside and inside buildings with facades facing streets. The two apartments were uninhabited, and the results, shown in Figures 4 and 5 may not be entirely representative of typical inhabited apartments.

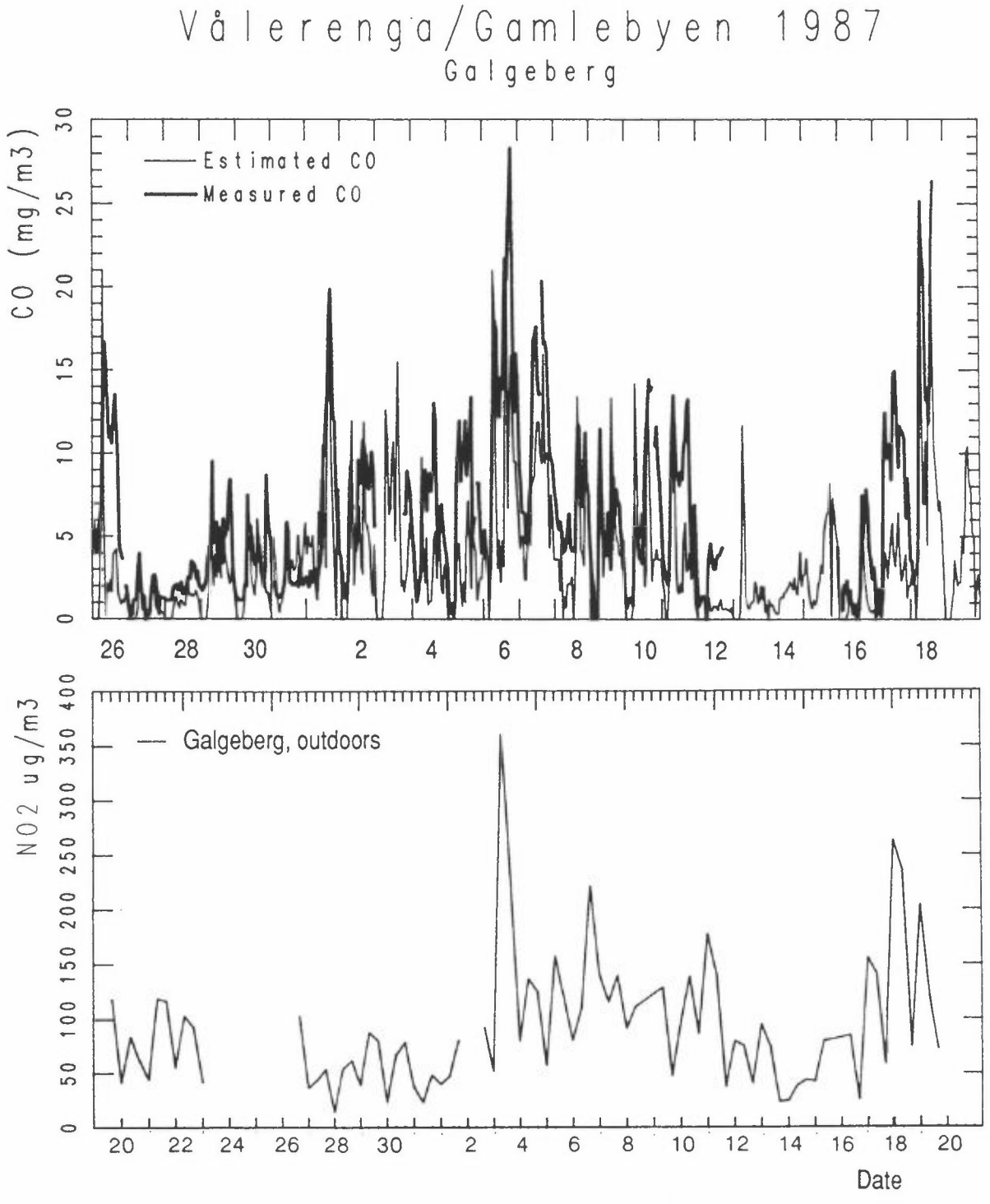


Figure 2: Measured concentrations of CO (1 hour average values) and NO_2 and suspended particles (8-hour average values) at station "Galgeberg".
Period: 26. October - 19. November, 1987.

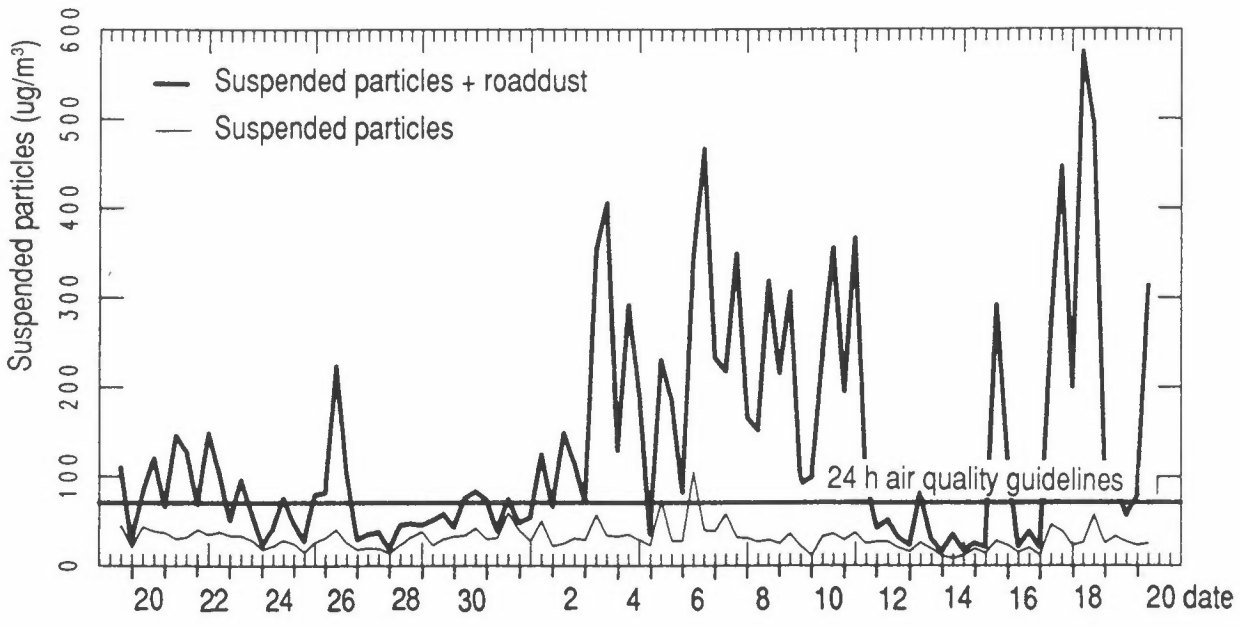


Figure 2: cont.

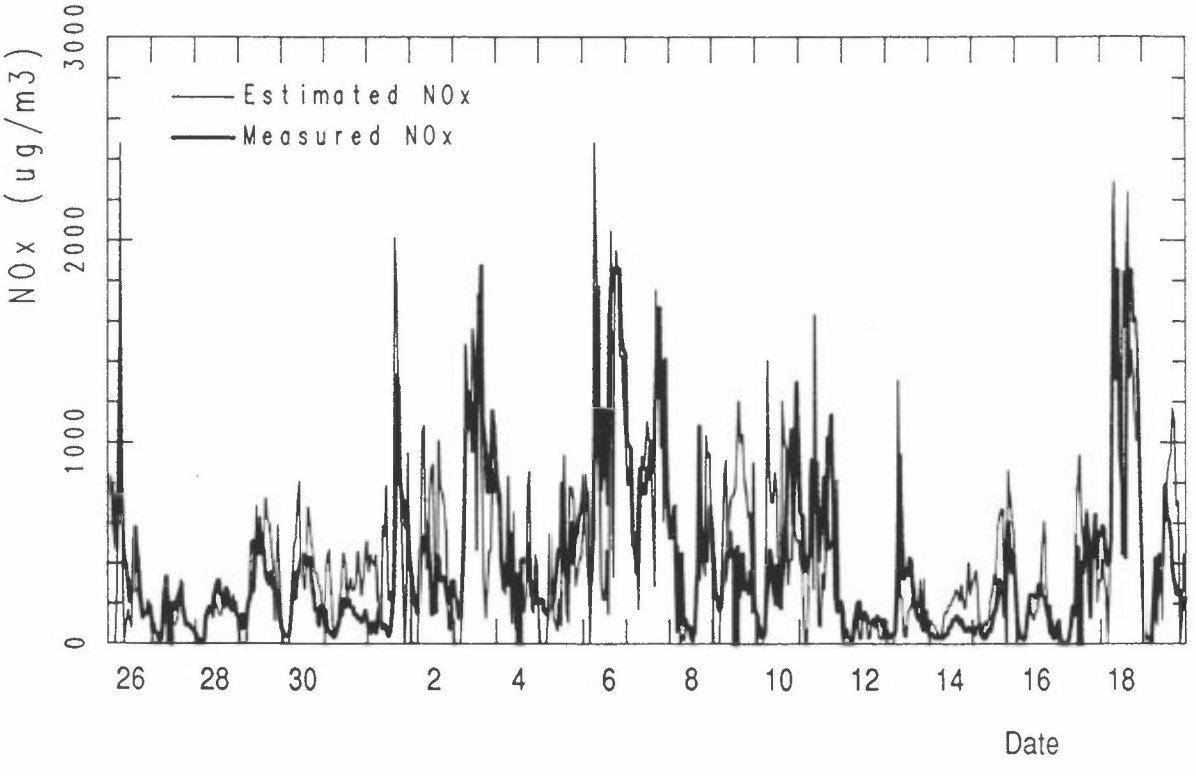
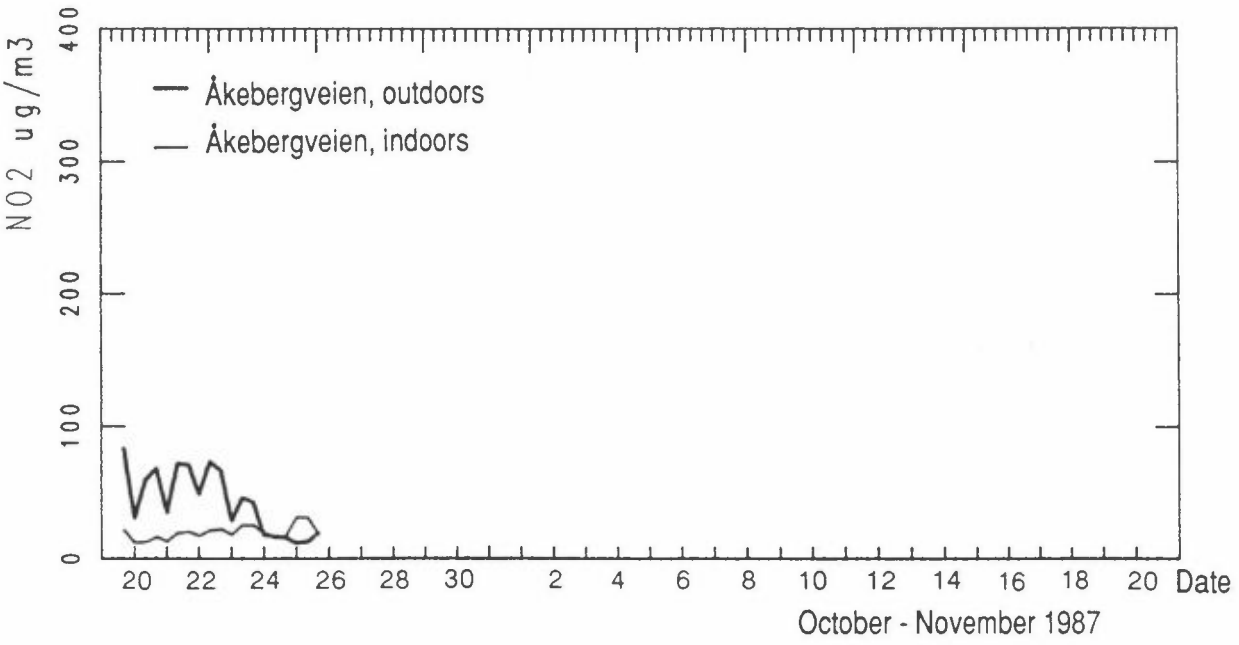


Figure 3: Measured NO_x concentrations (1-hour average values) at "Malerhaugen" station.
Period: 26. October - 19. November, 1987.



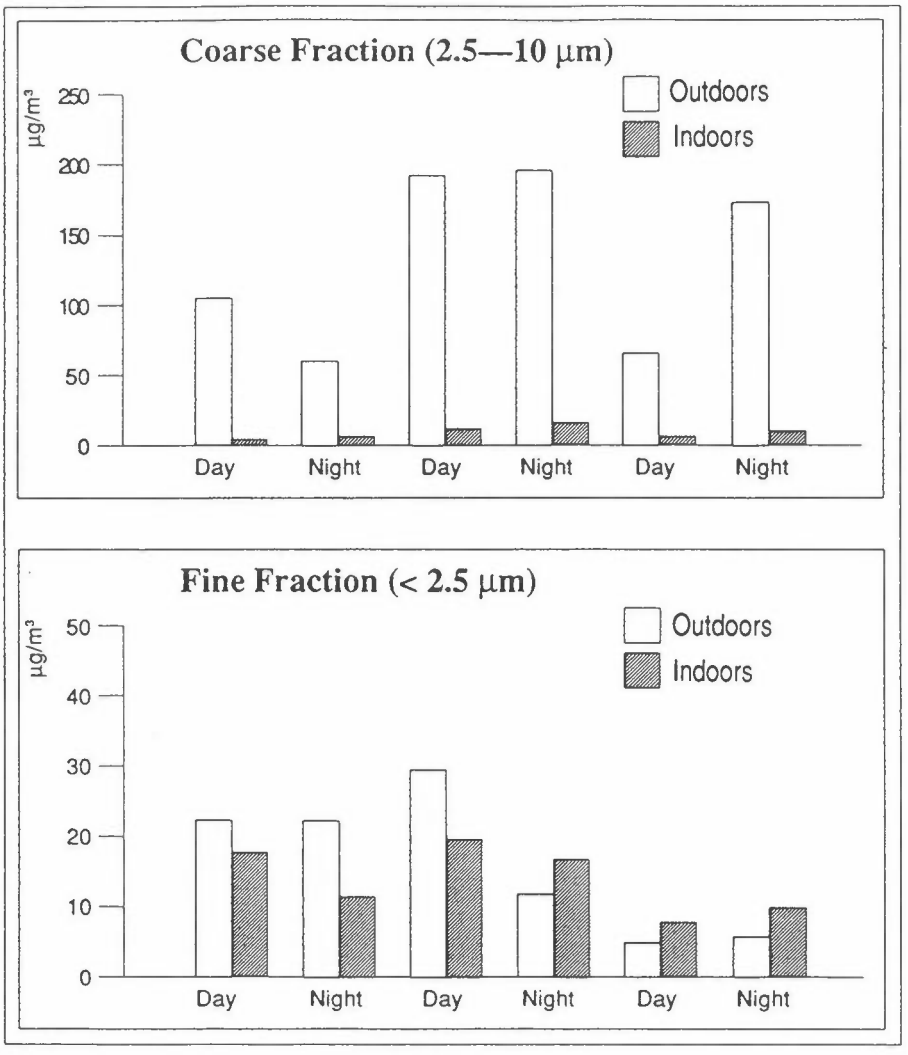
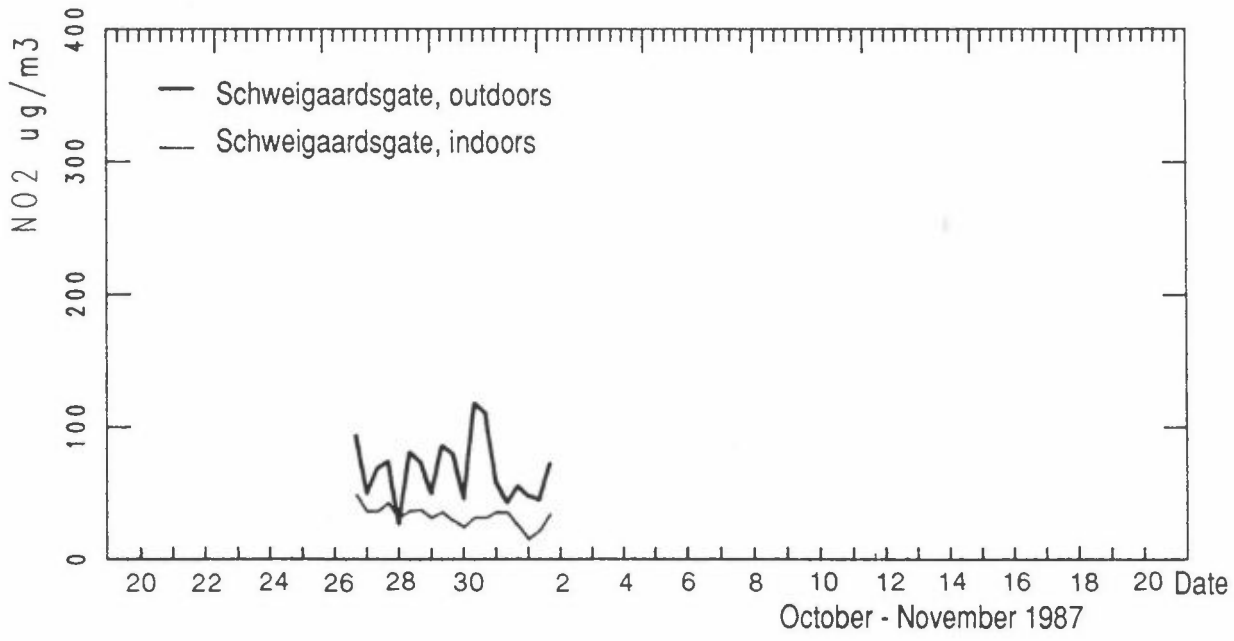


Figure 4: Measured concentrations of NO_2 (8-hour average values) and suspended particles (12-hour average values) at station "Åkerbergveien". Measurements were performed simultaneously outdoors and indoors.



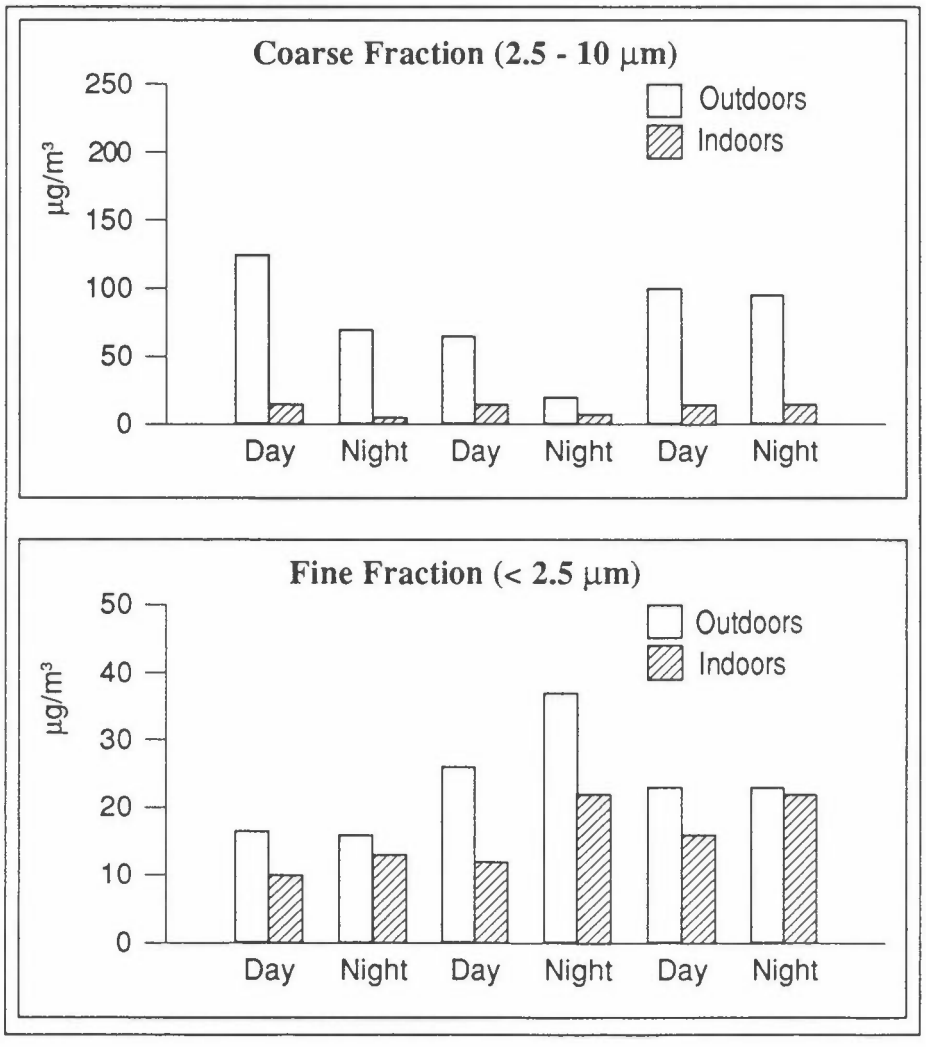


Figure 5: Measured concentrations of NO_2 (8-hour average values) and suspended particles (12-hour average values) at station "Schweigaards gate". Measurements were performed simultaneously outdoors and indoors.

The indoor NO_2 concentrations were less then half the outdoor concentrations. The indoor concentration of the fine fraction of PM_{10} (particles of diameter less than 2 μm) where 50-120% of the simultaneous outdoor concentrations, while the indoor coarse fraction (2-10 μm) was very small compared to outdoors, due to sedimentation and impaction of particles as air enters the building.

2.2 EPIDEMIOLOGICAL STUDY DESIGN

The investigation was designed as a three level study (Figure 6). The first level was a cross-sectional study of self-reported symptoms of health effects and chronic diseases of 1028 individuals living in the Vålerenga/Gamlebyen area. Each individual answered a comprehensive questionnaire concerning several aspects of traffic annoyance and health. All participants reported whether or not they were bothered by a set of health symptoms over the past 6 months and how often ("sometimes" or "often"). In addition, they described which chronic diseases they had and if they had been sick at home during the last 14 days (Hjorthol et al., 1990). This information was related to an estimated air pollution exposure index at their home address.

Approximately 150 individuals from the original sample of 1028, participated in a sub-study of the daily variation in health over a two week period. Each individual was followed hourly over a two week period through a diary. Each participant provided information on his/her whereabouts (including address, floor, type of windows and whether or not the windows faced a street), activity and health for each hour (Figure 7). Air pollution exposure was estimated for each hour for each individual, by means of an exposure model.

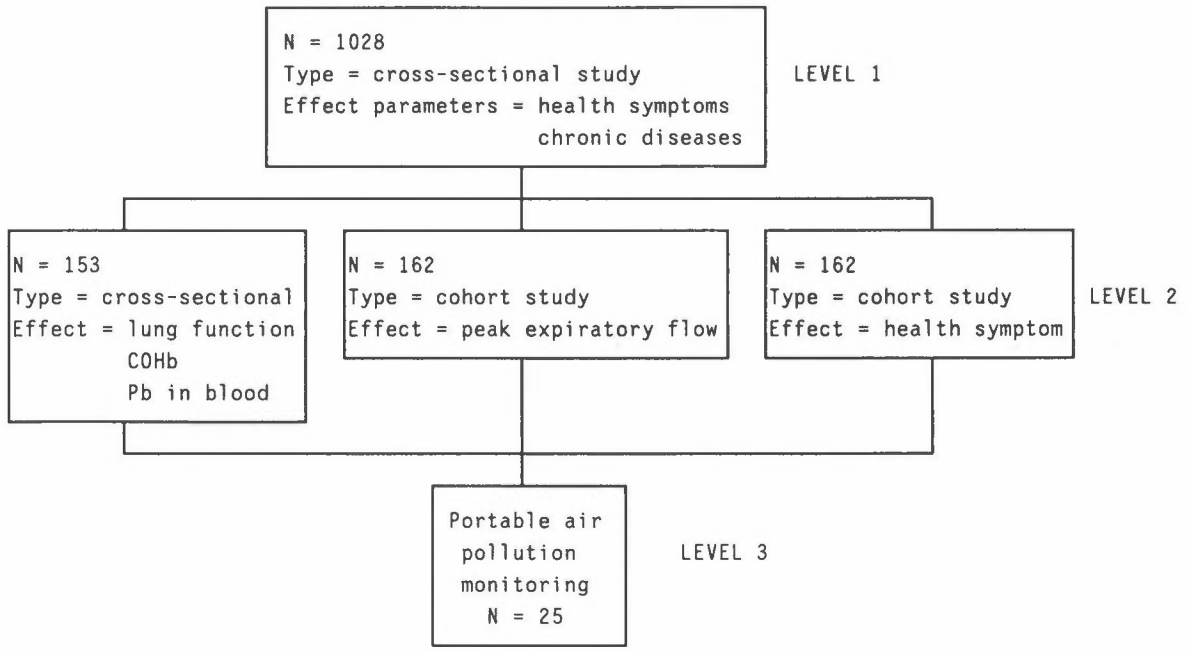


Figure 6: Description of the three levels of investigation of the health effects of air pollution from vehicular traffic.

Carbon monoxide in the blood (COHb) and breath, lead in blood and lung function were also measured in all 153 participants.

Of the 153 participants, 10 carried portable CO monitors for 1 day that continuously measured CO exposure, and 15 carried passive NO_2 monitors (Palmes tubes) for 1 week. These measurements were compared to the exposure estimates.

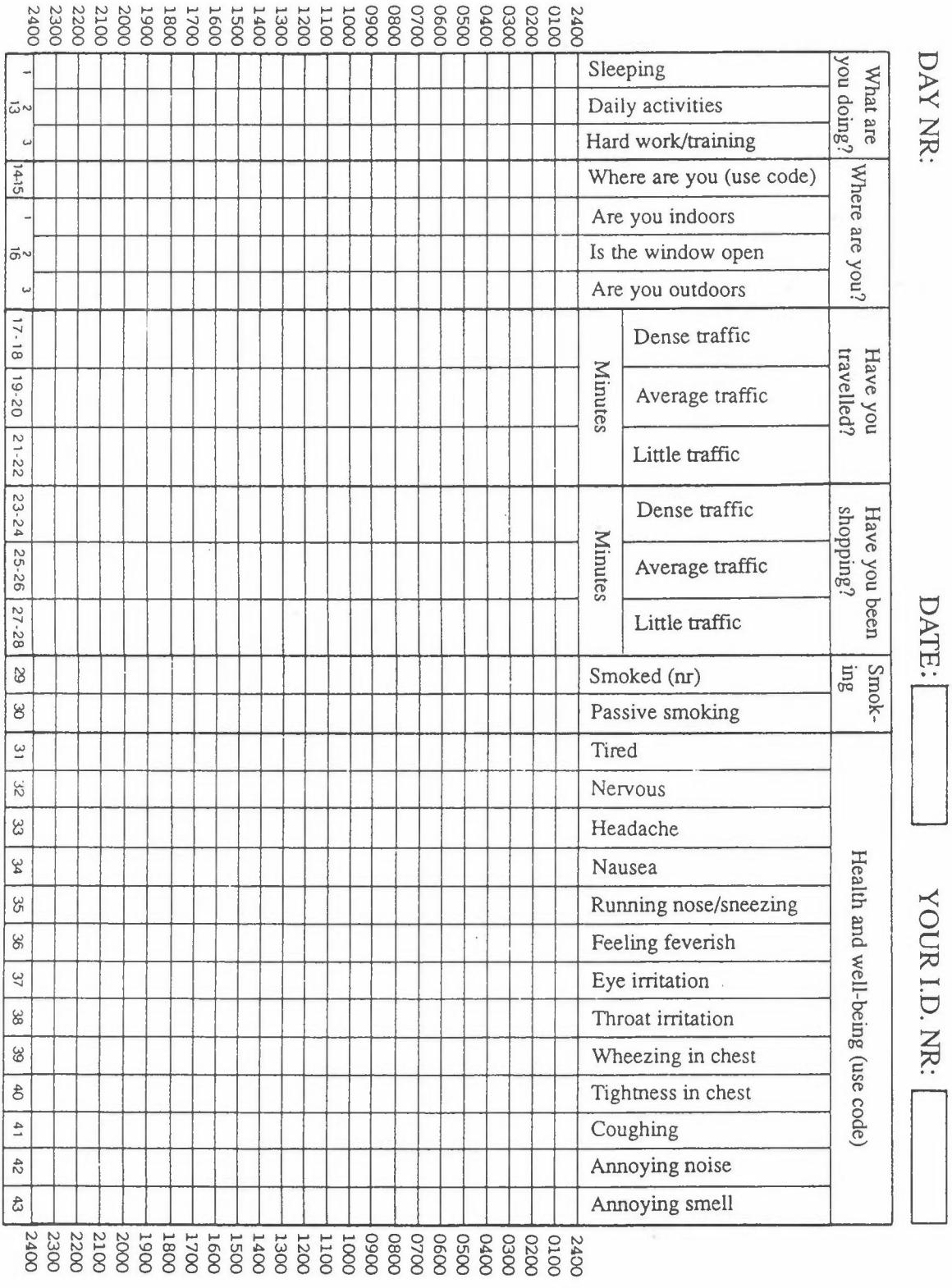


Figure 7: Diary used by participants to note on an hourly and daily basis where they were and which health symptoms were bothersome.

2.3 STUDY POPULATION

A random sample of the population living in the eight selected areas of Vålerenga/Gamlebyen were chosen for the study (1028 people). The areas were selected to represent particular traffic situations, such as: currently subject to heavy traffic, but where the traffic situation would be improved; currently subject to light traffic which would be worsened; and subject to light traffic both before and after the changes.

The 153 participants in Level 2 were volunteers from the original sample of 1028. Comparing the types of answers to the 75 questions in the original questionnaire revealed that the subsample represented very well the original random sample. The participants were, however, more educated and a greater percentage were working outside their home. The participants were generally somewhat more positive about their home environment.

2.4 CHOICE OF HEALTH PARAMETERS

In Level 1 participants were asked whether they were "sometimes" or "often" bothered during the last 6 months by the following health parameters:

- Headache
- Nausea
- Coughing, airway irritation, sore throat
- Pains in the neck, back, arms or shoulders
- Palpitation of the heart or chest pains
- Indigestion
- Fatigue
- High blood pressure
- Dizziness
- Itching, rash or allergy

- Nervousness, anxiety or restlessness
- Feeling depressed
- Problems sleeping
- Eye irritation
- Having a cold or the flu
- Respiratory trouble, problems breathing.

They were also asked if they had the following chronic illnesses:

- Asthma
- Allergy
- Chronic Bronchitis
- Lung Disease (unspecified)
- Heart Disease.

In addition, they were asked if they were sick enough to be in bed or had reduced daily activities the last 14 days.

In Level 2 the participants were asked for each hour if they had the following health symptoms (Figure 7):

- Fatigue/weak
- Nervous, restless
- Headache
- Nausea, not feeling well
- Running nose or sneezing
- Feeling feverish
- Eye irritation
- Throat irritation
- Wheezing in the chest
- Tightness of the chest
- Fits of coughing
- Annoyed by noise
- Annoyed by bad smell

The participants were provided with a Mini-Wright Peak Expiratory Flow Meter with which they were to test their lung function four times a day.

The participants were also asked how they felt generally for the entire day.

At the end of the two week period, each participant came back to a central meeting place to return their diaries, give a blood test for the measurement of COHb and lead, give a breath sample for the measurement of CO and take a full lung function test.

2.5 FIELD STUDY PROGRAM

The cross-sectional investigation was carried out by a professional team of pre-trained investigators accustomed to handling such questionnaires. The participants were randomly picked within each chosen geographic area. If the randomly chosen individual was no longer living at the given address, a person of the same sex currently living at the address was chosen. The information was obtained from oral interviews. The investigation was carried out in the fall of 1987 from October to November.

Each individual that participated in the first level of the investigation was given an information package at the end of the interview explaining the more detailed investigation in Level 2. Each participant that was willing to participate in a more detailed investigation was asked to contact the Norwegian Institute for Air Research (NILU).

Each participant then came to a central meeting place and was explained the study in more detail. Those that were still interested in participating were then given the diary and explained how to complete it. They returned to the same place

14 days later to return the completed diary forms, take a lung function test, give a blood and breath sample.

Of the nearly 500 individuals that contacted NILU, 153 completed the two week detailed investigation. The investigation period began at the end of October 1987 and was completed in November.

Ten of the participants of the Level 2 study were willing to carry a CO monitor for 24 hours and 15 to wear a Palmes tube for a week as a control of the air pollution exposure estimates.

2.6 METHODOLOGY USED TO MEASURE THE BIOLOGICAL/CLINICAL PARAMETERS

2.6.1 Blood measurements

From each individual, 3-10 ml whole blood was collected in two green stoppered Venoject evacuated blood collection tubes (VT 100 SH - sodium heparin). Blood sampling was done in October-November 1987.

The blood samples for lead analysis were stored at 4-8°C 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. Two drops of Triton X-100 were added to the remaining blood to measure blood lead.

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 µmol Pb/l whole blood.

Lead concentrations in whole blood (B-Pb) 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.

Ordinary graphite tubes were used throughout this study. The within-run precision of the method was typically 1,5-2,0% at 0,4 µmol Pb/l, and the detection limit (2x noise level) was 0,01 µmol Pl/l.

Since most lead is concentrated in the erythrocytes, differences in hematocrit can influence concentrations of lead in blood. This would result in apparently higher blood lead concentrations in those individuals whose hematocrit was elevated due to other factors, e.g. smoking, and apparently false low values in anemic individuals. Therefore, blood lead concentrations were all standardized to a hematocrit of 45% (McIntire and Angle, 1979) using the formula:

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

To facilitate comparison with earlier studies, blood lead values have been converted from µmol/l to µg/100 ml (dl) using the formula B-Pb µg/100 ml = B-Pb (µmol/l) x 20.72.

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. Day to day variation of the method is monitored through an intra-quality control program. Vials of frozen blood-bank samples are analyzed regularly with a variation of typically ± 7%.

Hemoglobin and carboxyhemoglobin (HbCO) was measured by a "Hemoximeter OSM3" built by Radiometer, Copenhagen. The instrument uses a photometric method to measure the different hemoglobin derivatives. Each derivative absorbs at a different

maximum wavelength. HbCO absorbs at 535 nm. The blood is hemolyzed in a thermally controlled cuvette by vibration at a frequency of about 40 kHz.

Each sample was measured at least twice, and the average of the measurements used. Variation between samples was minimal. The instrument was calibrated before and after each series of analyses with three different known levels of hemoglobin and HbCO. The samples were measured immediately after sampling.

2.6.2 Measurement of carbon monoxide in end-expired breath samples

Each individual was first asked to breathe normally, then to take a deep breath and blow out. They were to then take another deep breath, hold it for 20 seconds, blow half out and blow the rest into a plastic bag. The plastic bag was a special 3 liter bag with a valve opening. The bags were pumped empty between each trial. The mouthpiece was sterilized and replaced for each individual.

Each sample was analyzed immediately after sampling by connecting the bag to a portable CO monitor. The same monitor was used for each field day and for all the sample of end-expired breath.

2.6.3 Measurement of lung function

Lung function for each individual was measured using a Vitalograph-compact. The same technician tested all individuals. Some tests were considered unsuccessful and removed from data analysis. The results were studied by a lung specialist and categorized as indicative of normal or clinically reduced lung function. The values were standardized for age and height using Norwegian standards (Gulsvik, 1979) and expressed as per cent of expected. The formulas used are:

For men:

VC (Vital Capacity) =

7.40 x height - 0.029 x age - 6.68

FEV_1 (Forced Expiratory Volume) =

5.74 x height - 0.032 x age - 4.54

For women:

VC = 5.22 x height - 0.021 x age - 4.10

FEV_1 = 3.28 x height - 0.027 x age - 1.22

In addition, each of the 160 individuals in the cohort study measured peak expiratory flow (PEF) four times a day for each day using a Mini-Wright Peak Flow Meter. Three readings were taken at each time. The highest value was then considered to be the measurement. The measurements were made close to the hours of 0800, 1200, 1600 and 2000.

2.6.4 Methods for measuring exposure to CO and NO_2 using personal monitors

Measurement of carbon monoxide by personal monitors

Continuous measurements of carbon monoxide concentrations for the control of exposure estimates were done by using portable CO monitors. The portable CO monitor used in this study was developed early in the 1980s by the Environmental Protection Agency that loaned us the monitors. It is a light yet accurate continuous personal monitor equipped with a data logger. It runs on a battery and has a running time of 24 hours. The measuring unit was developed by General Electric and the logger by Magus. The measuring system involves a chemical reaction between CO and H_2O yielding $CO_2 + 2H^+ + 2e^-$. The hydrogen ions and the electrons traverse the membrane creating an electric current which is directly proportional to the amount of CO. The reaction is thermally regulated.

Passive sampling for nitrogen dioxide, using Palmes tubes

The passive sampler for nitrogen dioxide is based on molecular diffusion to a sorbent for the gas. The diffusion tube consists of an acrylic tube (7,1 cm long and 1,2 cm in diameter). Two stainless steel meshes coated with triethanolanine are placed at one end of the tube held in place by a plastic cap. The other end is stopped with another cap until exposure starts. The samplers are exposed with the open end facing down. After exposure the tubes are extracted with deionized water and the resulting nitrite ion concentration in the extraction solution is measured by ion chromatography (Palmes, 1981).

Average air concentrations for the exposed time period, are calculated by means of the measured NO_2 concentrations and a constant given by the area and length of the tube and the diffusion coefficient for NO_2 in air.

2.7 AIR POLLUTION EXPOSURE ESTIMATION

2.7.1 Exposure model

A critical element in any study of the health effects of air pollution is the assessment of air pollution exposure. The traditional method of exposure assessment has been to measure pollution at one to several air quality stations outdoors. This has its obvious shortcomings. People move around in the area and differ in the time spent indoors and outdoors. Some apartments are facing the street, others are not. Apartments are located at various distances from the roads and at different floors.

A focus of this study was to improve the air pollution exposure estimate (for each of the investigation levels) relative to that normally used in similar studies. More accurate exposure estimates should facilitate the interpretation of measured

health effects and enable relating them to air quality guidelines. The components estimated were carbon monoxide (CO) and nitrogen dioxide (NO_2). Since the area had only one main air pollution source, traffic, all pollutants including CO and NO_2 emitted by vehicular traffic will be correlated. Therefore, to simplify presentation, the results were confined to those for CO in Level 1 and NO_2 in Level 2. Many of the health symptoms described in Level 2 are more likely caused by NO_2 than CO. The methods used to estimate air pollution exposure are summarized in Figure 8.

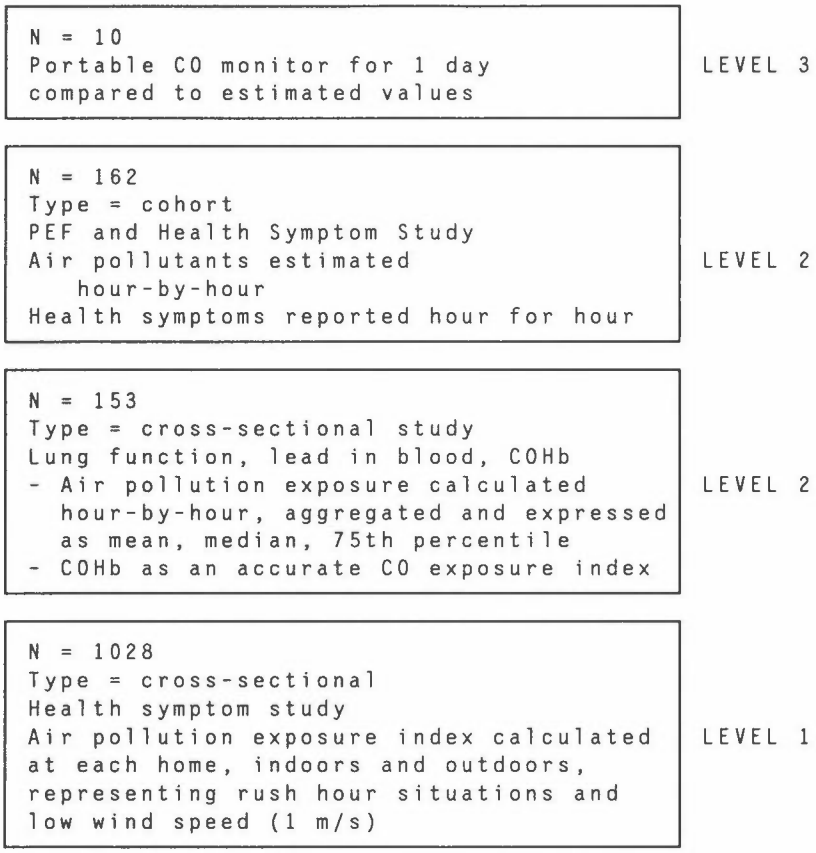


Figure 8: Summary of methods used to estimate air pollution exposure at all levels of the investigation.

The exposure estimates were based on the calculation of air pollution concentrations in time and space by means of an air pollution dispersion model (Tønnesen, 1990). This was the only

way to be able to assign an exposure value to each person in the study, either as an index for his home (Level 1), or as actual concentrations hour by hour (Level 2). The estimates given by the model were compared to measured air pollution at a few points in the area where CO, NO_x and NO_2 concentrations were measured continuously. In Level 3, personal CO exposure estimates were compared to actual exposure, measured by means of personal CO monitors carried for 24 hours by 10 participants.

The dispersion model used was a modified version of the US EPA HIWAY 2 model, modified to account for the dispersion effects of the relatively low traffic speed on the roads of the area (generally less than 60 km/h).

The air pollution concentration estimates were carried out as follows (see Figure 9):

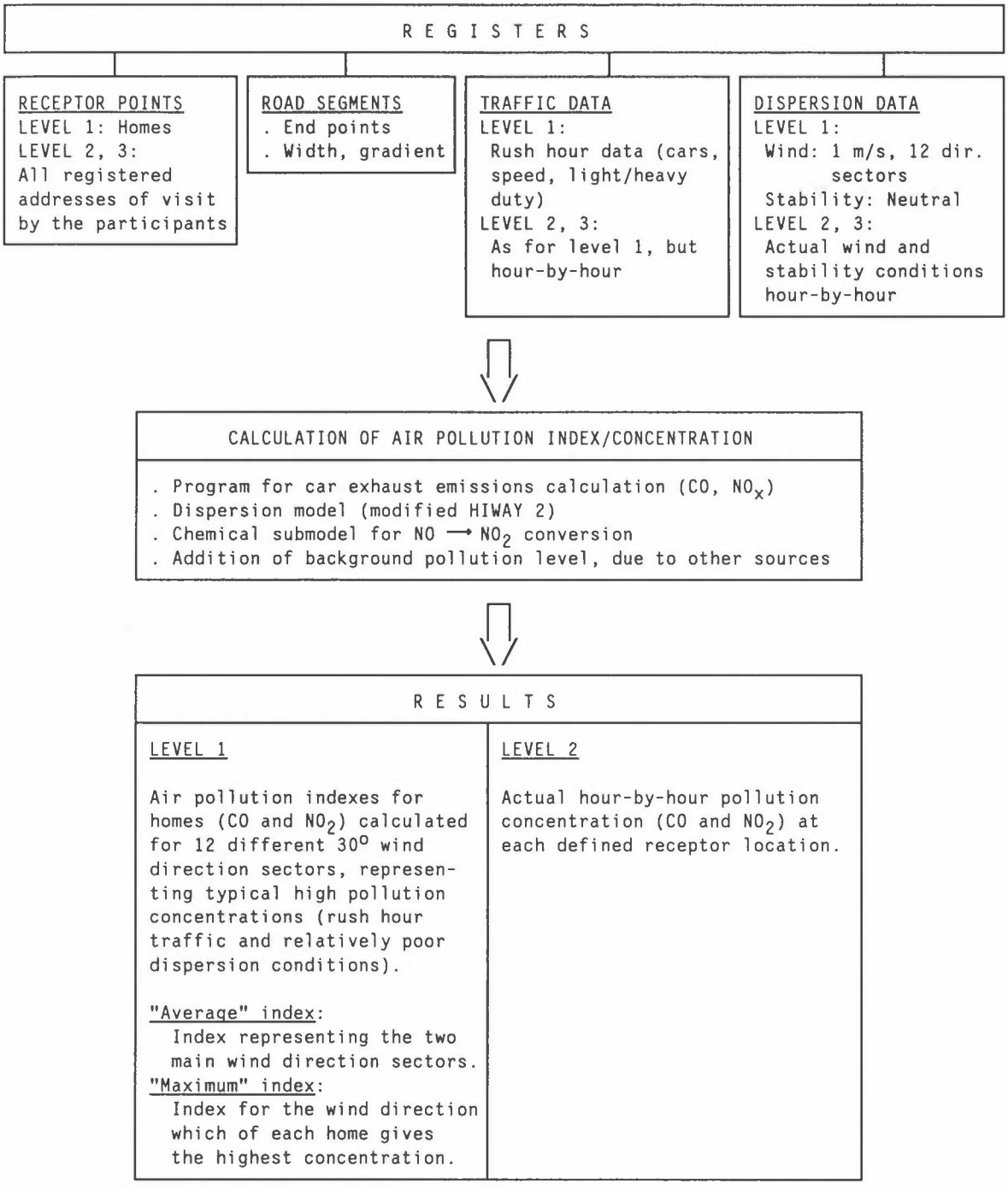


Figure 9: Diagram showing the principles of the calculation of air pollution exposure (outdoor concentrations) at Levels 1 and 2 of the health study.

- The receptor points (participant's home address and other places in the area participants visited during the study, as indicated in the diary) were positioned within a grid system (± 2 m accuracy). The receptor point for each address was defined as the mid-point of the facade facing the nearest street, 2 m above ground.

- The road system was portioned into straight line road segments, and each segment's endpoints were positioned within the same grid system.

- Exhaust emission for each road segment (g/m s) was calculated from traffic data (number of cars, speed, light/heavy duty) and emission factor data.

- The dispersion model then calculates concentrations at each receptor point, either as index values for certain defined traffic and wind/dispersion conditions (as in Level 1), or as actual hourly concentrations, based on actual traffic and dispersion data hour-by-hour (as in Levels 2 and 3).

- The chemical submodel for NO-to-NO_2 conversion takes account of the NO-NO_2-O_3 reaction scheme.

- The background pollution level, representing other sources inside and outside the area, was estimated. For NO_2, the estimate is based on regional ozone measurements.

- To calculate indoor exposure, a separate outdoor/indoor transfer model was devised, taking account of height above ground, whether the apartment faced the street or the courtyard and the quality of the windows (see Figure 10 giving the outdoor/indoor CO model for the "average" index).

At Level 1 the calculated air pollution indices for CO and NO_2 represented rush hour conditions and relatively poor atmospheric dispersion conditions (Larssen et al., 1990). Thus, the

indices represent typical high rush hour concentrations. The highest expected actual concentrations will occur during much poorer dispersion conditions, and will be a factor 2-3 larger than the calculated indices.

Indices were calculated for 12 30° wind direction sectors. "Average" indices were then calculated as the average of the indices representing the two main wind sectors of the area (which is 90° and 210°), and the "maximum" index is the largest index calculated for each home, for a given wind sector, dependent upon the location of the home relative to the road system. These indices ("average" and "max") were used in the subsequent regression analysis in Level 1 with health symptom data.

The calculated CO and NO_2 concentrations in Level 2 represent the actual pollution level experienced at each defined receptor location, hour-by-hour throughout the diary period of each participant. The hourly values were aggregated to also provide an average and maximum exposure for each participant in addition to each individual's 75th and 90th percentile of air pollution exposure.

The correlation between the NO_2 and CO estimates inherent in a method that considers only one major pollutant source, is seen in Figures 11 and 12. A comparison of the index of indoor CO exposure as estimated in Level 1 and the median of the NO_2 exposure as calculated in Level 2 is given in Figure 12. The discrepancies between the two estimates may be a measure of differences in lifestyles, f.ex. that some people are more at home (thus an index is more representative for this group) than others.

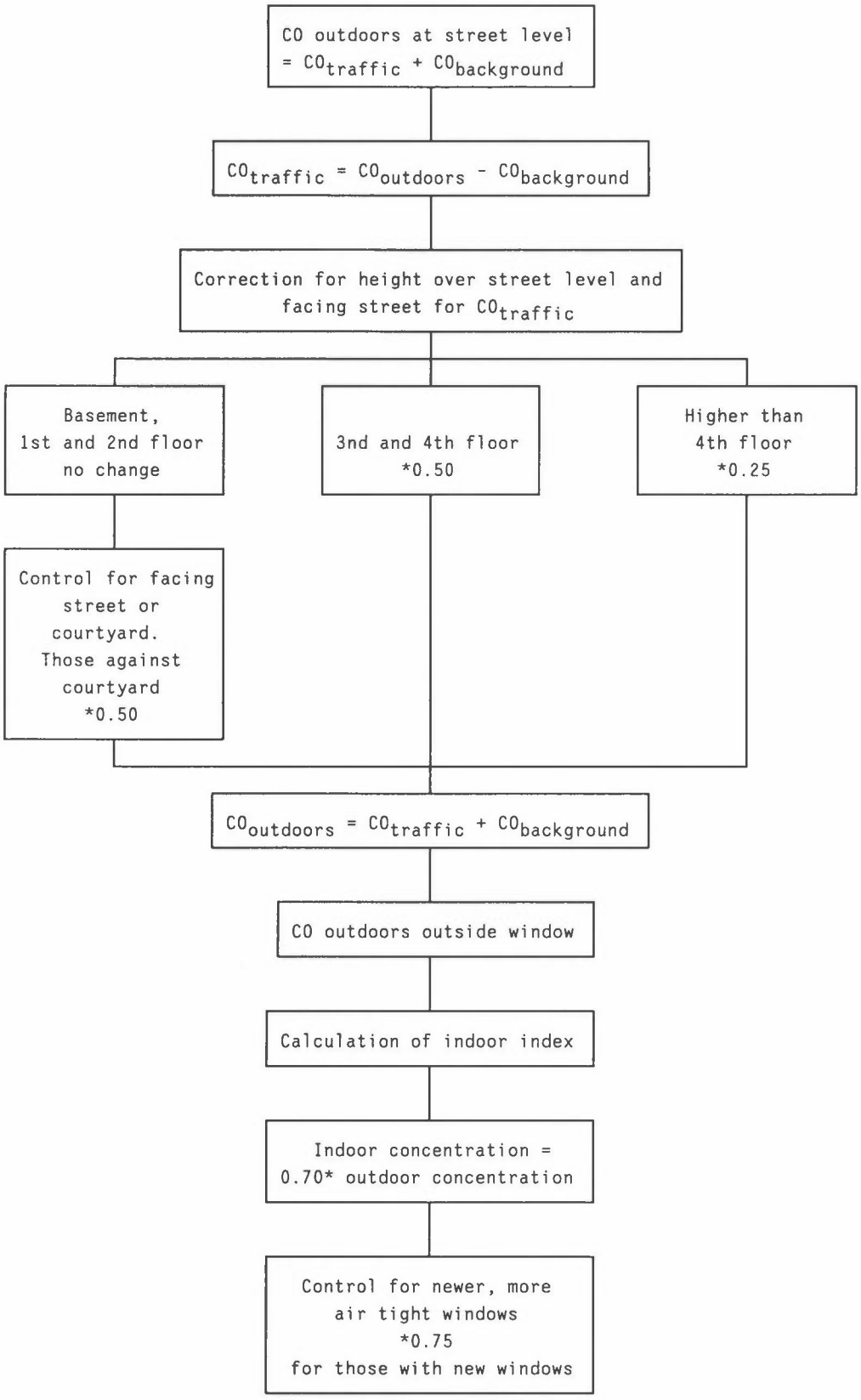


Figure 10: Flowchart of calculation of indoor index for CO (average of values calculated for the two main wind directions (90° and 210°).

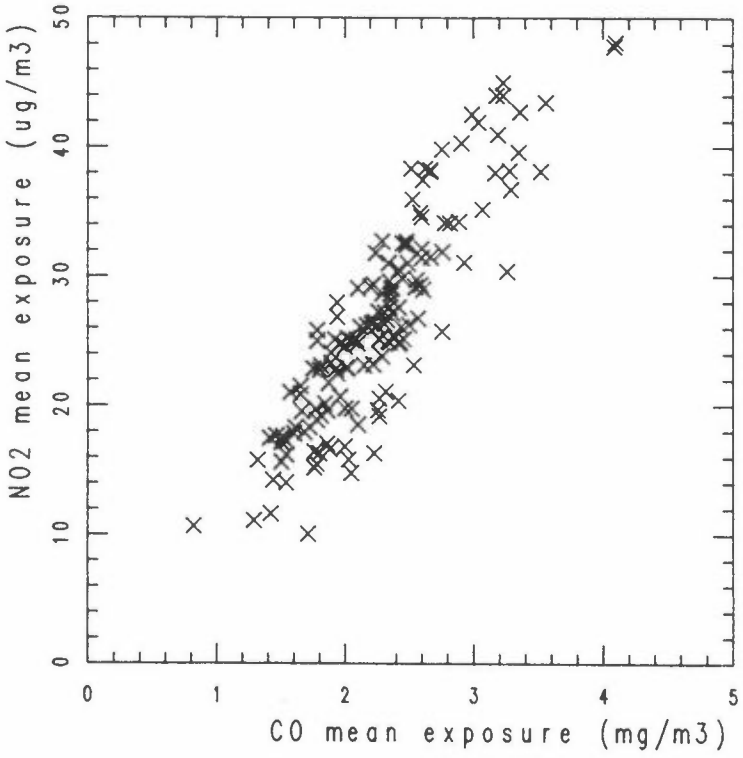


Figure 11: The relationship between mean estimated exposures to NO_2 and CO.

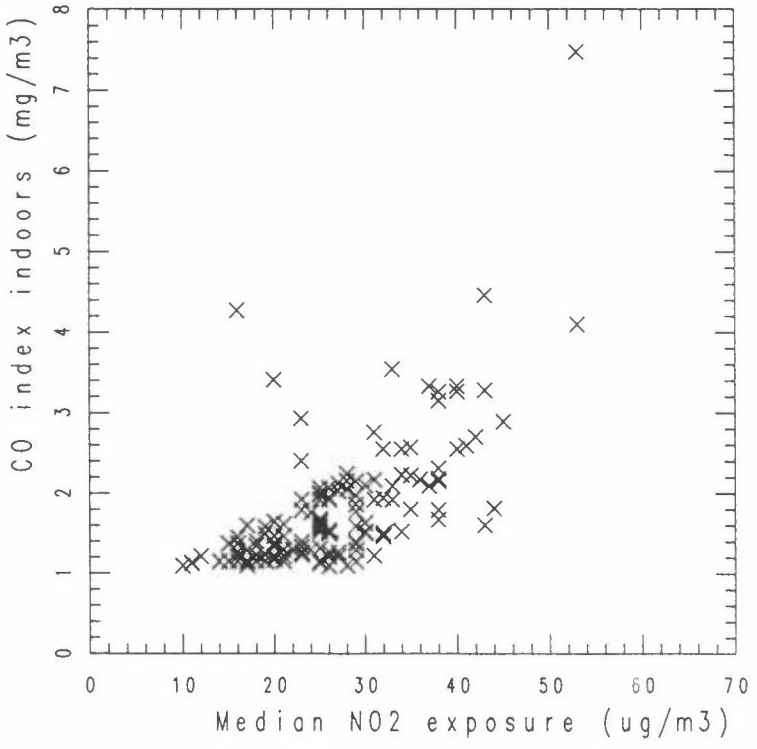


Figure 12: The relationship between the estimated indoor CO index representative for each home used in Level 1 and the median NO_2 estimated exposure based on hourly information provided by the diary used in Level 2.

2.7.2 Statistics on exposure calculations

Analysis of the information provided by the diary is summarized in Appendix 1. The elderly are more at home than other population subgroups. The elderly have their windows open more often than other groups, and children are seldom in rooms with window open. Children are more outdoors and spend more time sleeping than adults. Adult men spend more time exercising and sleep less than the other population subgroups. Adult women smoke the most and spend the most hours smoking. Adult men, followed by children, spend the most time travelling. Adult women, followed by the elderly, spend the most time shopping. The Vålerenga population spent approximately 1 hour per day travelling with approximately 15 minutes of that time spent in heavy traffic.

As a result of the lifestyle patterns described in the previous paragraph, air pollution exposure is highest for children during the day time (outdoors) and lowest at night (sleeping with windows closed) whereas the elderly have the highest exposure at night (sleeping with window open). In addition, children and the elderly have higher exposure during the middle of the day since they remain more in the area than adult men and women (Figure 13). As much as 40% of the population were in the area during the middle of the day (Figure 14).

Exposure to NO_2 as a function of time spent in different microenvironments is depicted in Figures 15 and 16 for each population subgroup.

Examining air pollution exposure hour for hour (Tables 2 and 3) showed that during the study period, 6 children and 4 pensionists, 29 adult women and 15 adult men were exposed to concentrations of NO_2 that exceeded the hourly recommended air quality guideline. The CO air quality guidelines were exceeded less often.

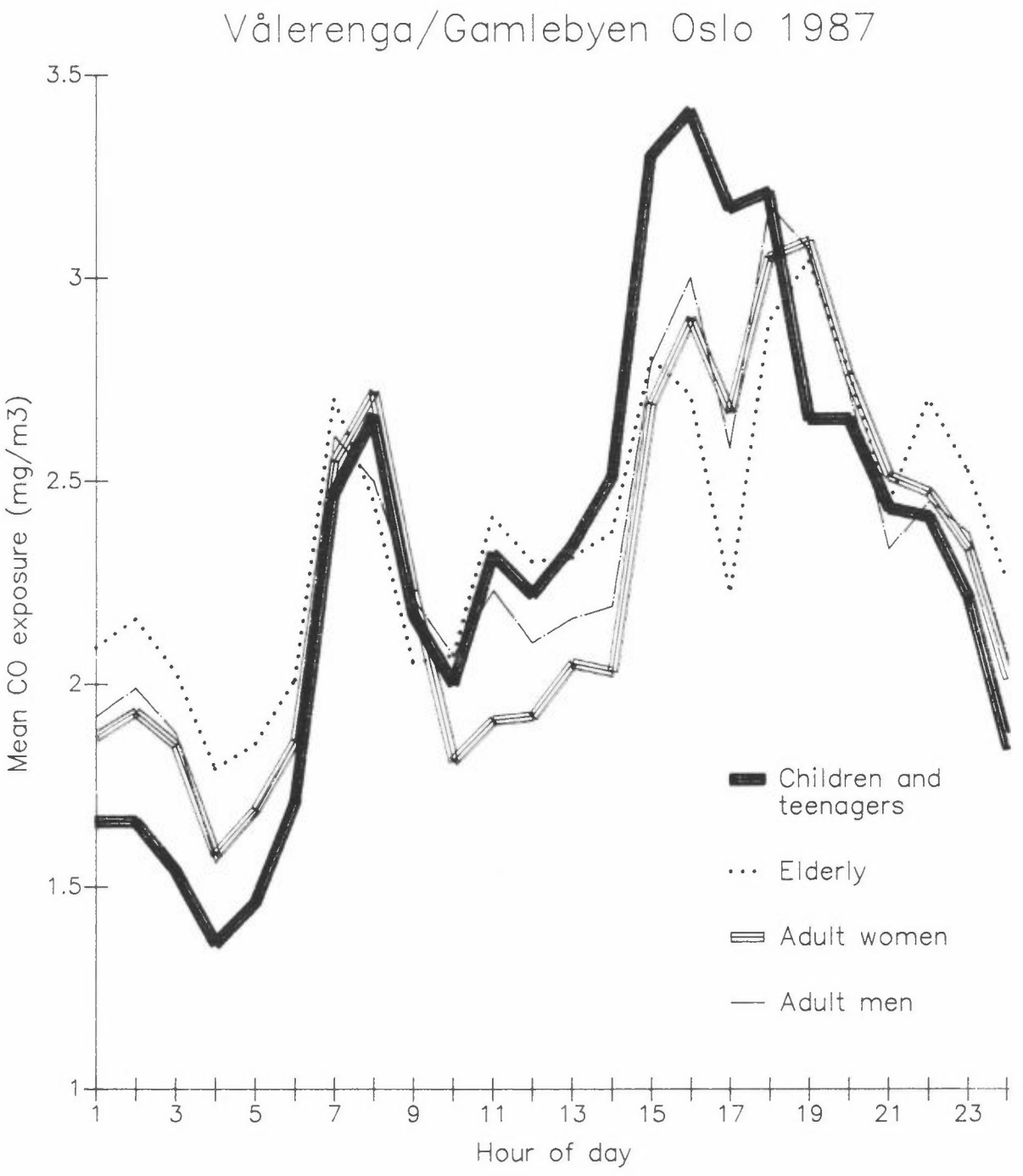


Figure 13: Average hourly estimated exposure to CO in four population subgroups. Calculations are based on information from the diary.

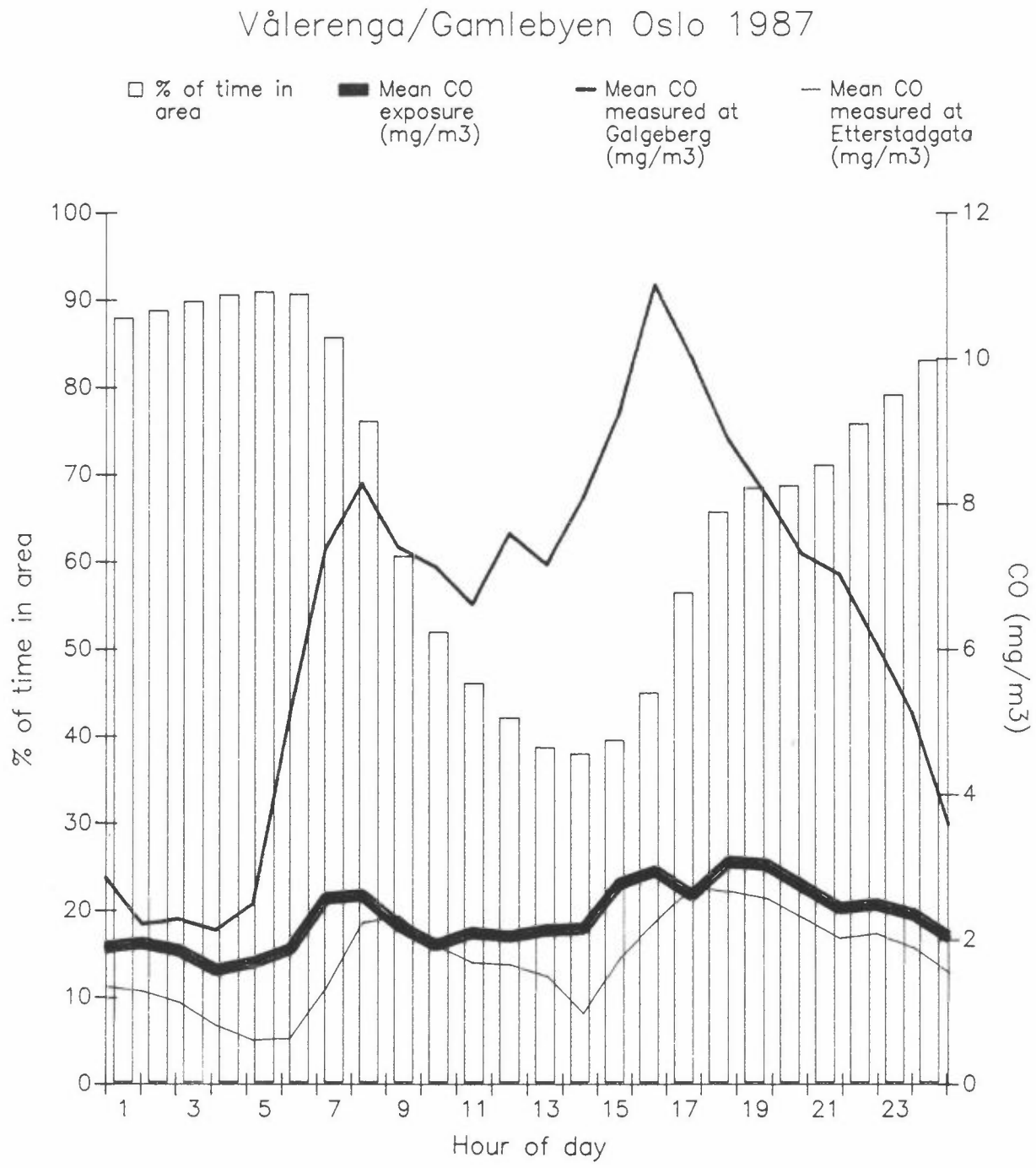


Figure 14: Outdoor concentrations of CO measured at Etterstad and Galgeberg as compared to hourly exposure estimates for the entire population. The percent of the population present at a given time in the Vålerenga area is depicted in the background.

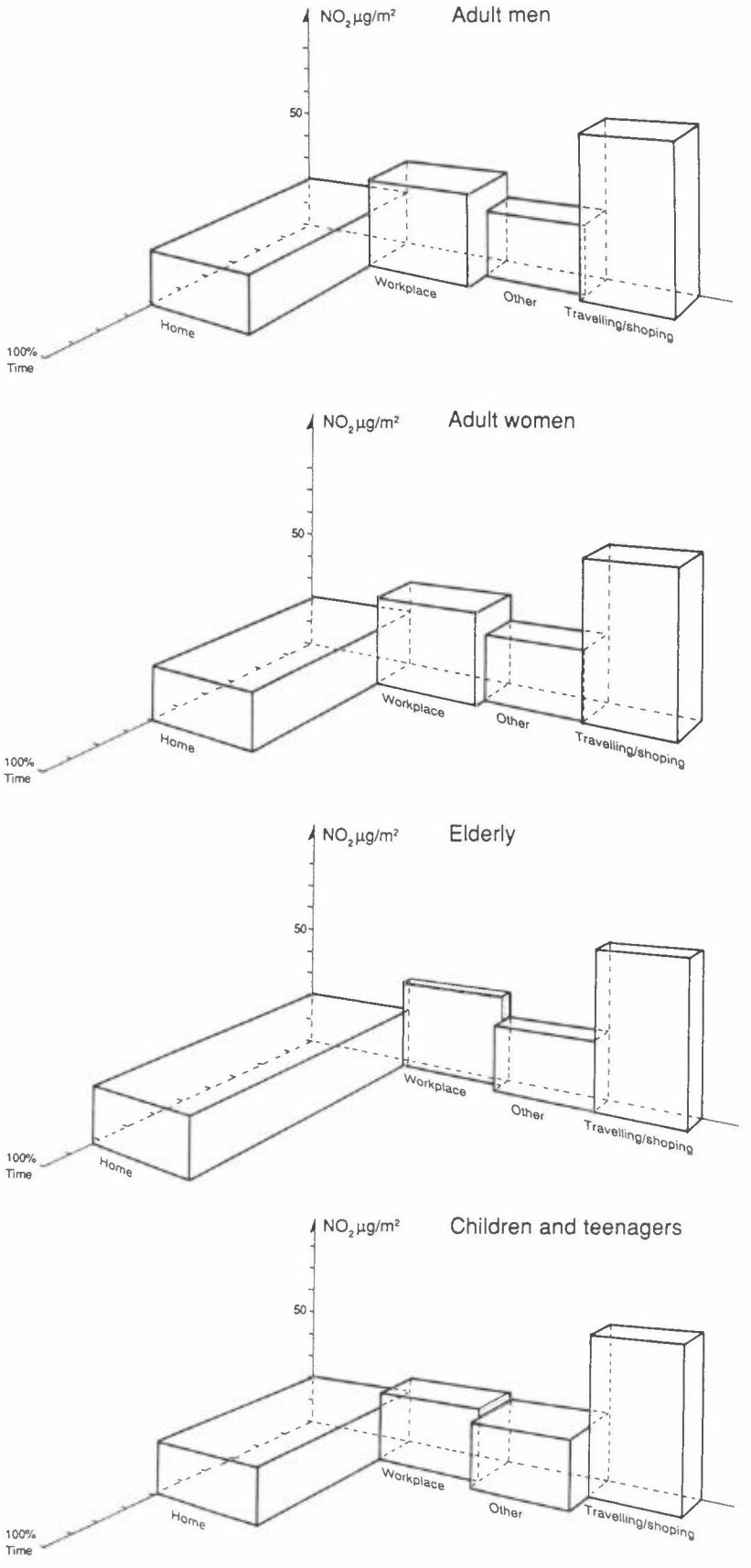


Figure 15: Assessment of exposure to NO_2 based on time spent in different micro-environments (home, workplace and travelling) for each population subgroup.

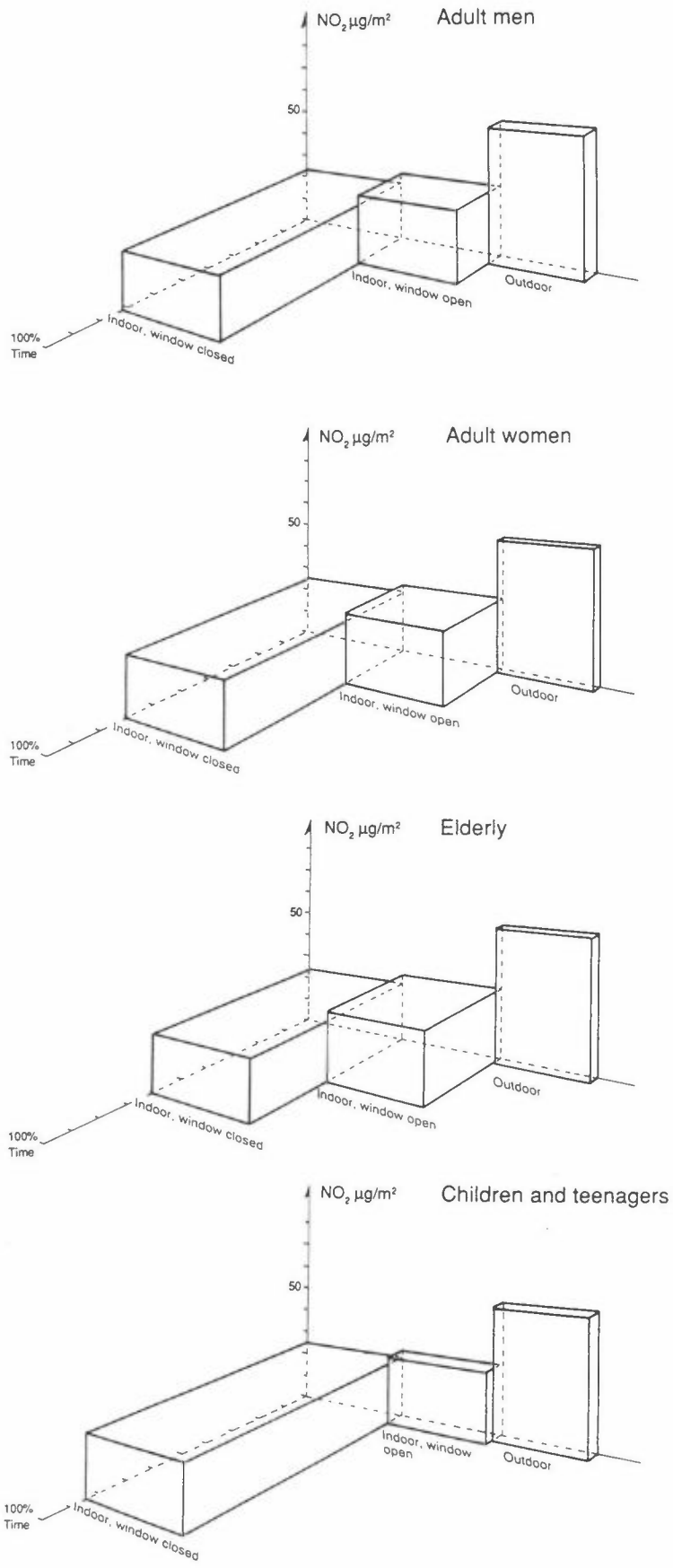


Figure 16: Assessment of exposure to NO_2 based on time spent in different micro-environments (indoors with and without open window and outdoors) for each population subgroup.

Table 2: Number of hours air quality guidelines were exceeded for CO based on exposure estimates, by population group.

	No. of participants	Hours exceeding 1h guideline for CO	Participants exceeding 1h CO guideline	Hours exceeding 8h guideline for CO	Participants exceeding 8h CO guideline
Population group					
Children and teenagers	12			5	3
Adult women	77	1	1	30	23
Adult men	48	1	1	18	16
Elderly	20			4	3

Table 3: Number of hours air quality guidelines were exceeded for NO_2 based on exposure estimates, by population group.

	No. of participants	Hours exceeding 1h guideline for NO_2	Participants exceeding 1h NO_2 guideline	Hours exceeding 24h guideline for NO_2	Participants exceeding 24h NO_2 guideline
Population group					
Children and teenagers	12	15	6		
Adult women	77	55	29	1	1
Adult men	48	35	15	2	2
Elderly	20	4	4	1	1

2.7.3 Comparison of calculated and measured exposure

During the field campaign of the health study, continuous measurements of air pollution were carried out at two fixed monitoring stations (Figure 1):

CO at "Galgeberg", a curbside site of the main road through the area (Strømsveien, about 35 000 annual daily traffic, about 15% heavy duty diesel), about 75 m away from a busy street crossing with traffic lights.

NO_x and NO_2 at "Malerhaugen", a site about 25 m west of the main road, with traffic parameters as above.

A further possibility of checking the quality of the exposure estimates was provided by measurements using personal CO monitors carried by 10 of the participants for 24 hours each (Level 3).

Figures 2, 3, 17 and 18 show measured versus calculated concentrations (1-hour averages) at Galgeberg and Malerhaugen (frequency distributions are given in Appendix 3).

Figure 19 shows measured versus calculated CO concentrations (1-hour averages) for 7 participants carrying personal monitors. For three of the participants, the measurements were discarded for comparison purposes, since there were discrepancies between the data in the 2-week diary and the data in the special diary the participants filled out for the one day they carried their monitor.

There is agreement in level and variation between measured and estimated exposure. Discrepencies are found due to 1) smoking and exposure to passive smoking, which is not taken into account in the calculations, 2) periods of movement in the area, where accurate filling out of diary is difficult, and 3) work place exposure not accounted for in the calculations (for person E and person G). Figure 20 shows the relationship between estimated values of NO_2 exposure and values measured using a passive sampling tube (same averaging time for both).

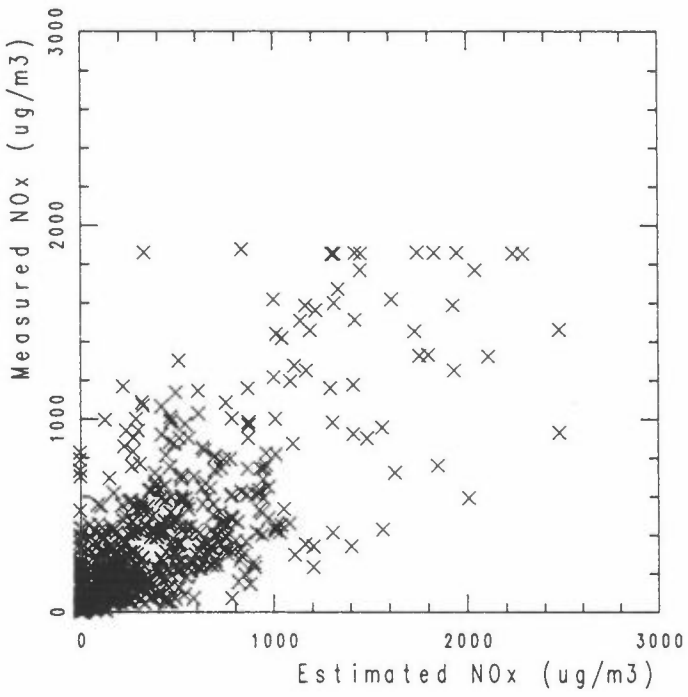


Figure 17: NO_x at "Malerhaugen" station. Comparison between measured values (1-hour averages) and values estimated by the exposure model. (The range of the measuring instrument was 0-1850 $\mu g/m^3$. The instrument "peaked" during the strongest air pollution episodes).

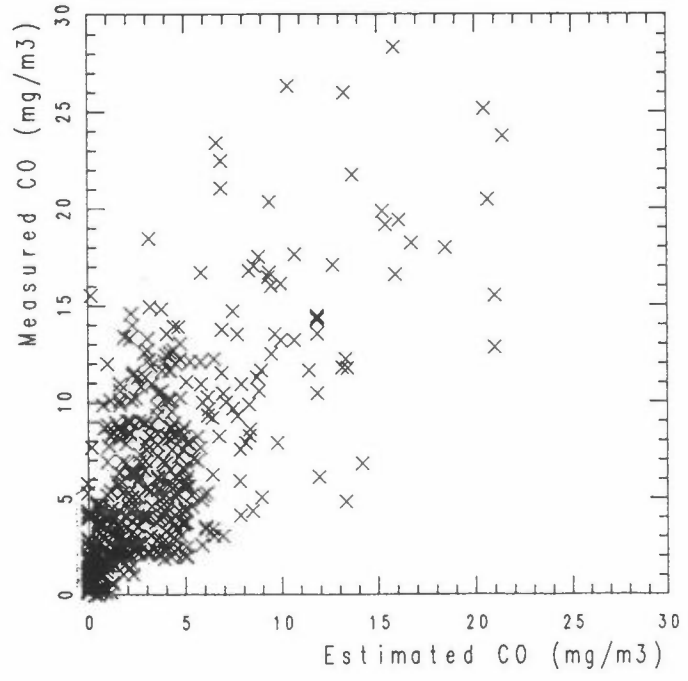


Figure 18: CO at "Galgeberg" station. Comparison between measured values (1-hour averages) and values estimated by the exposure model.

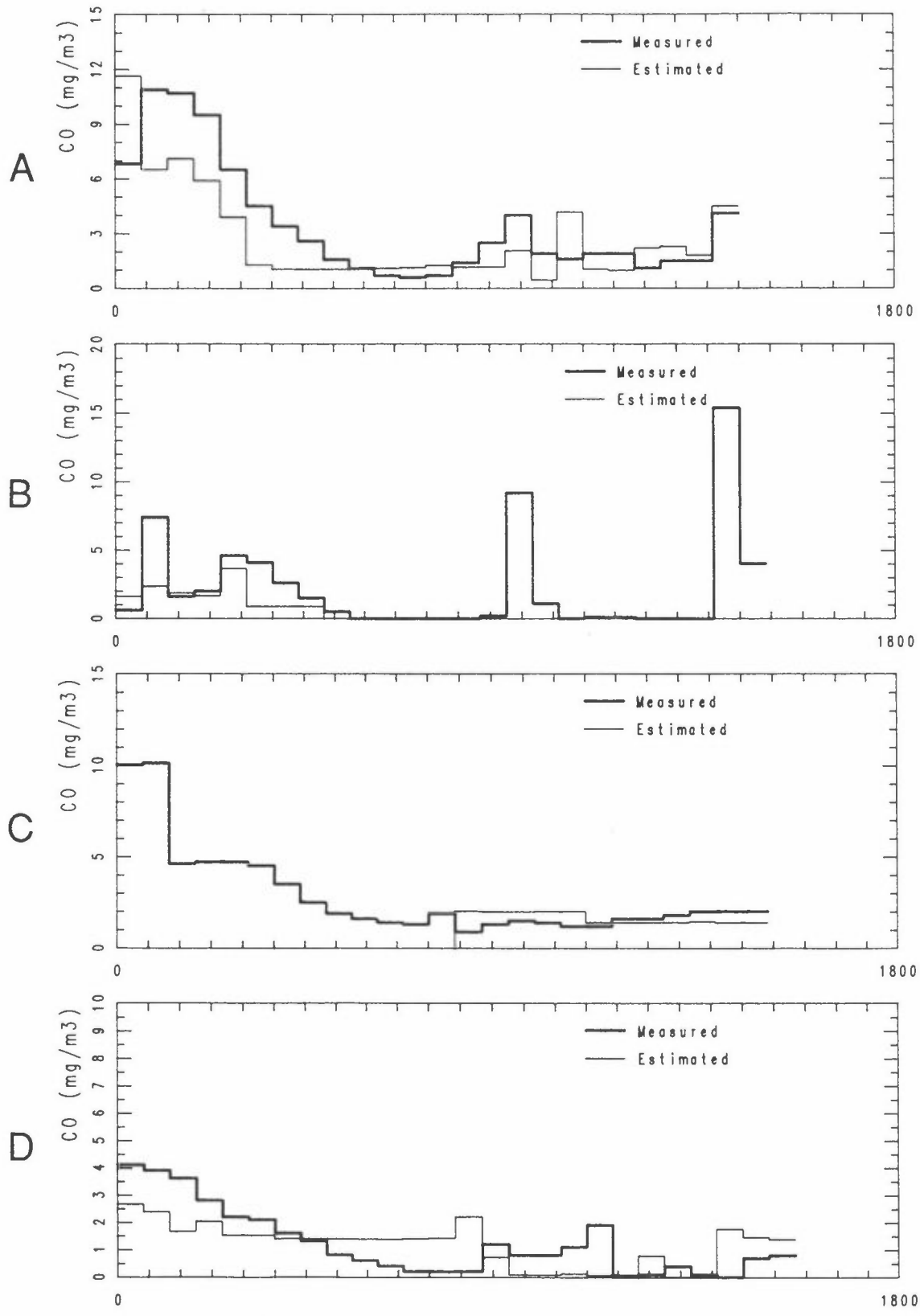


Figure 19: Comparison between measured CO exposure and exposure estimated by the exposure model, for 8 individuals carrying a personal CO monitor for 24 hours.

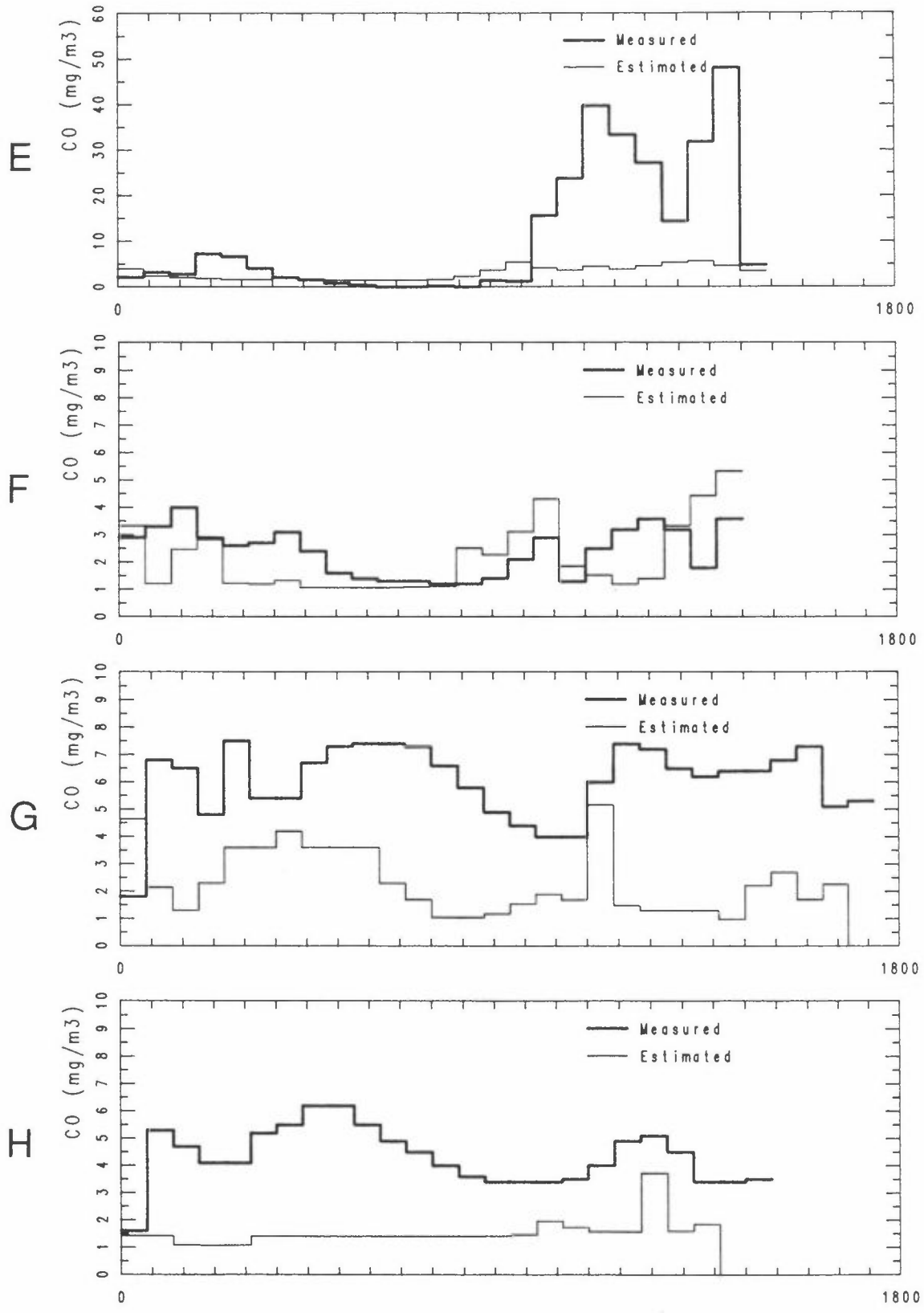


Figure 19: Cont.

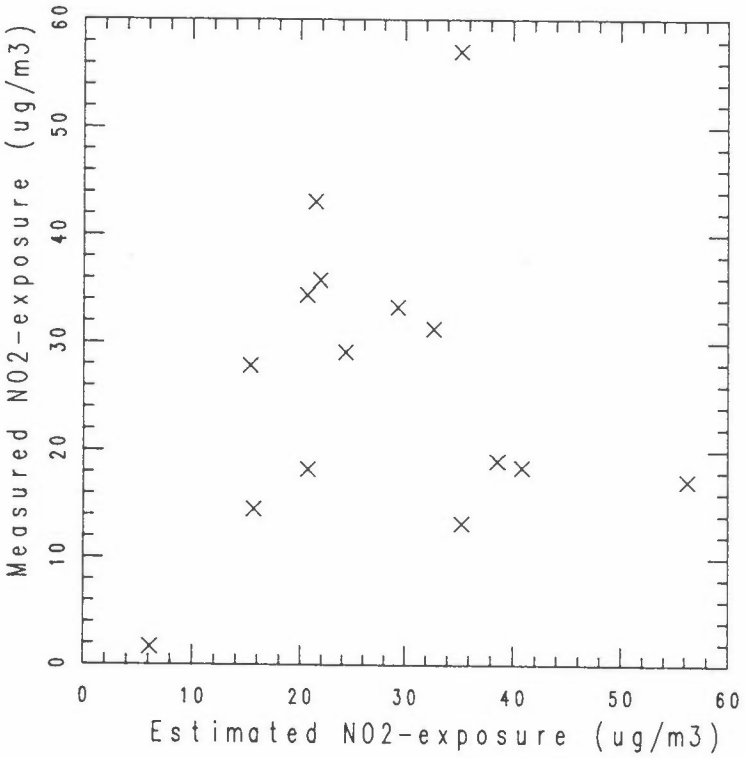


Figure 20: Comparison between measured NO_2 exposure and exposure estimated by the exposure model, for 15 individuals carrying personal NO_2 samplers ("Palmes tubes") for 24 hours.

3 RESULTS OF THE CROSS-SECTIONAL STUDY - LEVEL 1

The cross-sectional study in Level 1 attempted to answer the following question: Does air pollution cause individuals to have specified health complaints? The data from the study in Level 1 was analyzed in several phases. First, health complaints the last 6 months were recoded such that "sometimes bothered" and "often bothered" were handled similarly for each individual as a yes/no answer for each health symptom. The answers were analyzed by logistic regression (Clench-Aas et. al., 1989a). The Level 1 analysis has the advantage that the effects of air pollution can be quantitatively compared to the effects of other socio-demographic parameters such as age, sex,

education, marital status and current smoking. It is known that women are more bothered by certain health symptoms whereas men are more bothered by others. However, it has not previously been shown that air pollution is as important as current smoking in explaining the variance of some parameters. The values presented in Figure 21 are the Wald Test Statistics or Wald Score (Hosmer and Lemeshow, 1989), which are the regression coefficients divided by the standard error. Values over approximately 2.0 in absolute value are considered significantly different from zero at the 5% level and values over approximately 1.6 in absolute value are considered significant at the 10% level (in two-tailed significance tests).

As can be seen in Figure 21, headache, coughing, muscle pains, fatigue, depression, and eye irritation are significantly related to the air pollution exposure index at the 5% level. Fatigue is the only response type where the correlation to traffic pollution is as strong as or stronger than those for other explanatory factors. It must be emphasized here, that these relationships with the air pollution exposure index may be indicate effects of exposure to noise pollution. This is a possible explanation for muscle pains. Among the chronic diseases, chronic bronchitis was significantly correlated to the air pollution index, with a strength almost equal to that of current smoking.

One method of visualizing the results of logistic regression is to examine the relative risks (R.R.) or more precisely, odds ratio. Relative risk reflects the increased risk of having a health complaint, when air pollution increases from one reference level to another. The calculation of relative risk makes use of the regression coefficient. At a certain pollution level, a value of 1.5 is interpreted as a 50% increased risk. In the calculations it is possible to account for other significant factors. For example, chronic bronchitis is significantly related to air pollution. Current smoking is also a significant factor that explains the variation in the data. One can calculate the relative risk for smokers and non-smokers as

is shown in Figure 22. As can be seen in the figure, a smoker that is not exposed to air pollution has a 50% increased risk for having chronic bronchitis relative to a non-smoker. A non-smoker (may be former smoker) that is exposed to an indoor CO index of 1.8 mg/m^3 (50% of the population was exposed to a value of 1.8 mg/m^3 or higher) also had an increased risk of 50% for having chronic bronchitis. The range of air pollution exposure estimates used in the graphs represents the real range of estimates the Vålerenga population was exposed to.

The same type of comparison of relative risks can be done for being bothered "often" as opposed to only "sometimes" of the different health parameters. These are shown in Figures 23 to 28. The group that was not bothered by a health symptom was randomly split into two groups functioning as controls for each situation. Results of this analysis indicate whether or not air pollution exposure affects the degree of response of these symptoms. At a CO index value of 1.8 mg/m^3, the relative risk of having a headache "sometimes" is 1.2 (20% increased risk) whereas it is 1.4 (40%) for having a headache "often". The relative risk of having eye irritation "often" at 1.8 mg/m^3 is approximately 50% higher than at 1 mg/m^3 as opposed to circa 25% for only "sometimes" bothered. In general, this indicates that being often bothered is more closely correlated to air pollution exposure than "sometimes" bothered. The coefficents for all variables in the logistic regression for each health parameter, both for "often" and "sometimes" bothered are given in Appendix 4.

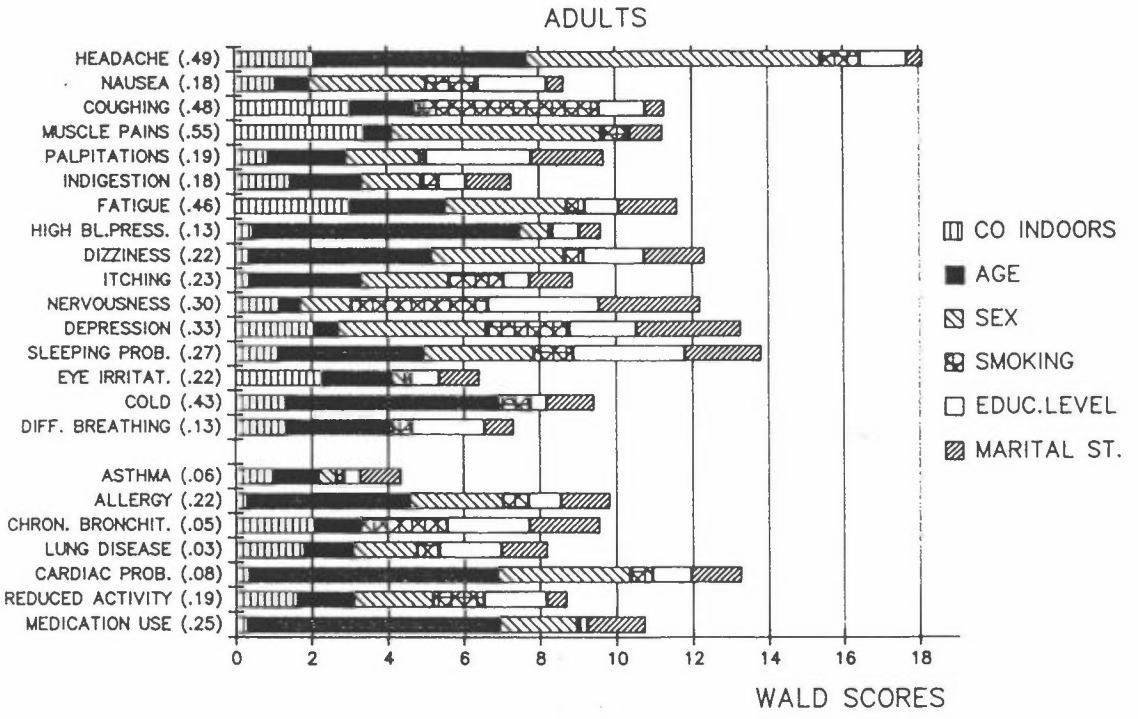


Figure 21: Effect of concentrations of air pollution indoors, age, sex, smoking, marital status and educational level on being bothered by a set of health parameters the last 6 months, for adults. The Wald test statistic (absolute value) is representative of the relative importance of the various factors. The direction of the relation between health parameter and explanatory variable is not indicated. Number in parenthesis gives the percentage of the population reporting the health parameter.

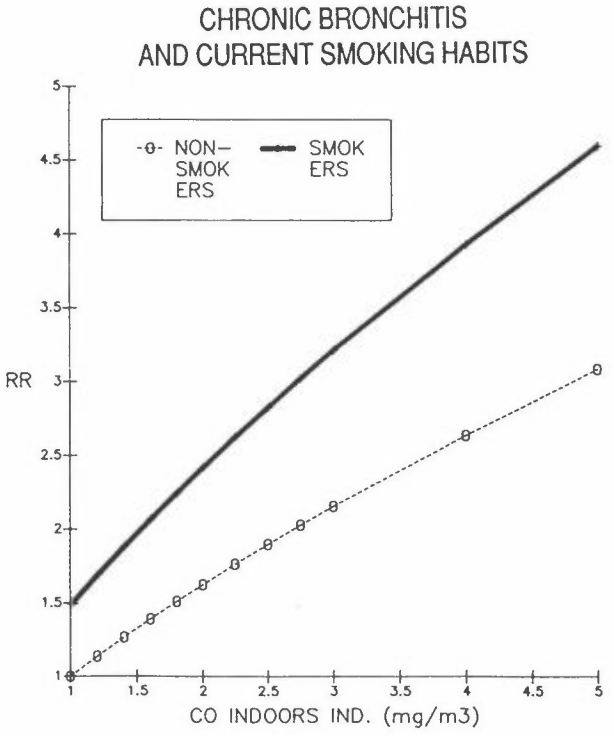


Figure 22: The calculated relative risks of self-reporting of having Chronic Bronchitis at a span of CO exposure indices indoors, reflective of those levels estimated in Vålerenga for non-smokers and smokers (current smoking habits).

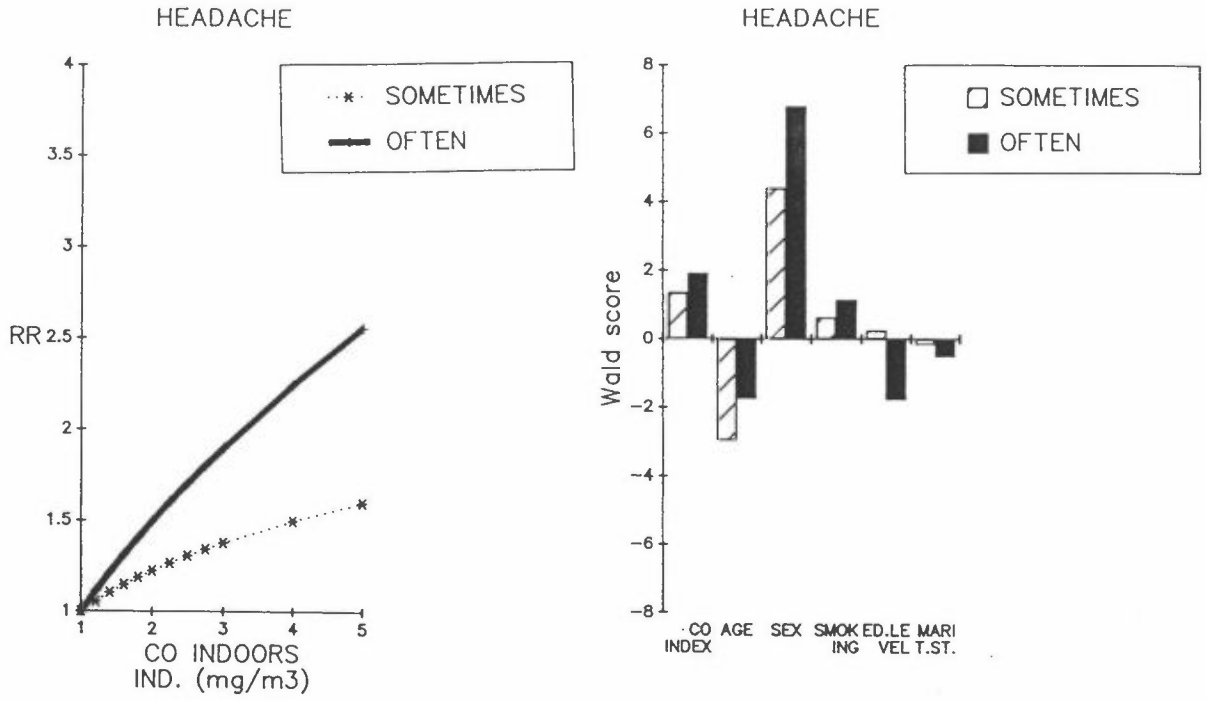


Figure 23: The relative risks of "sometimes" bothered as opposed to "often" bothered of headache at CO exposures indoors representative for the range estimated in Vålerenga. The Wald test statistic is given for comparison.

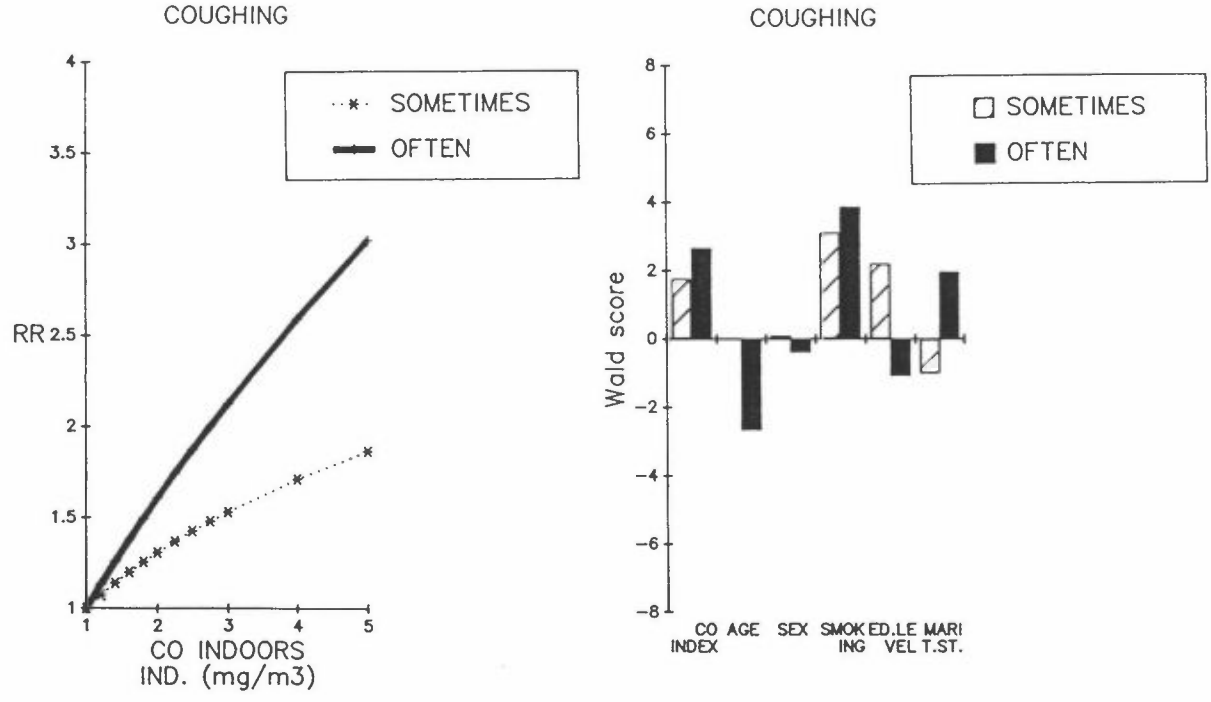


Figure 24: The relative risks of "sometimes" bothered as opposed to "often" bothered of coughing at CO exposures indoors representative for the range estimated in Vålerenga. The Wald test statistic is given for comparison.

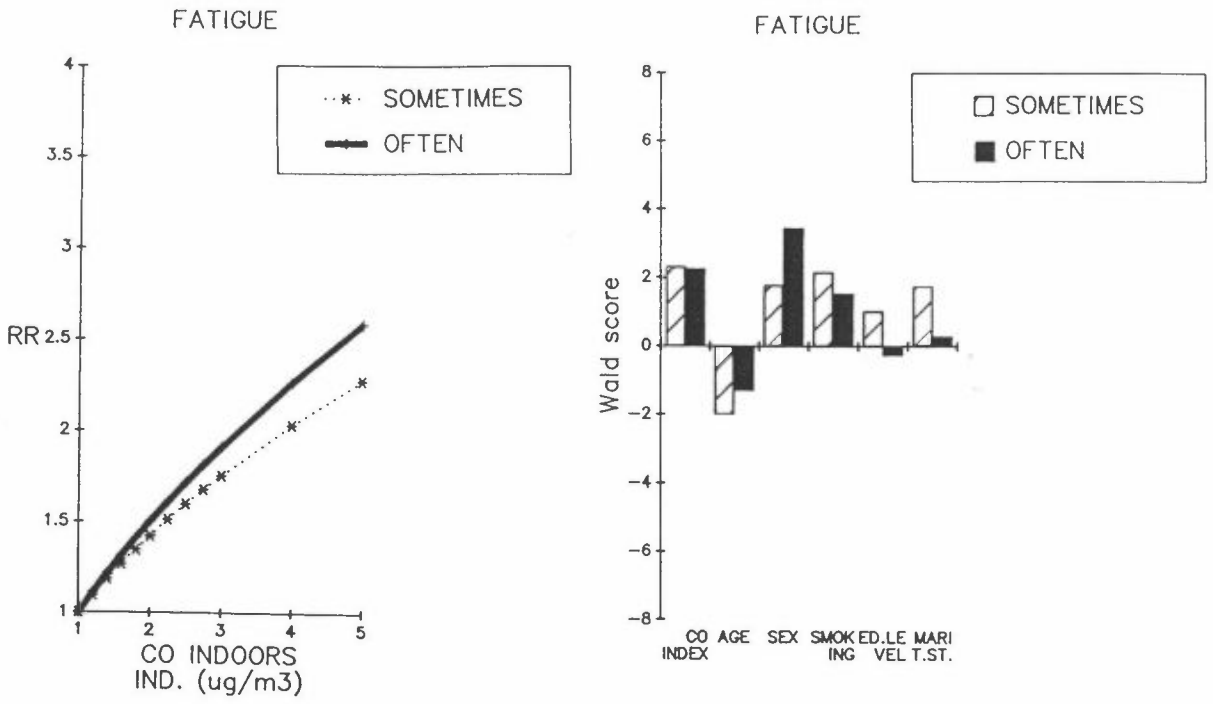


Figure 25: The relative risks of "sometimes" bothered as opposed to "often" bothered of <u>fatigue</u> at CO exposures indoors representative for the range estimated in Vålerenga. The Wald test statistic is given for comparison.

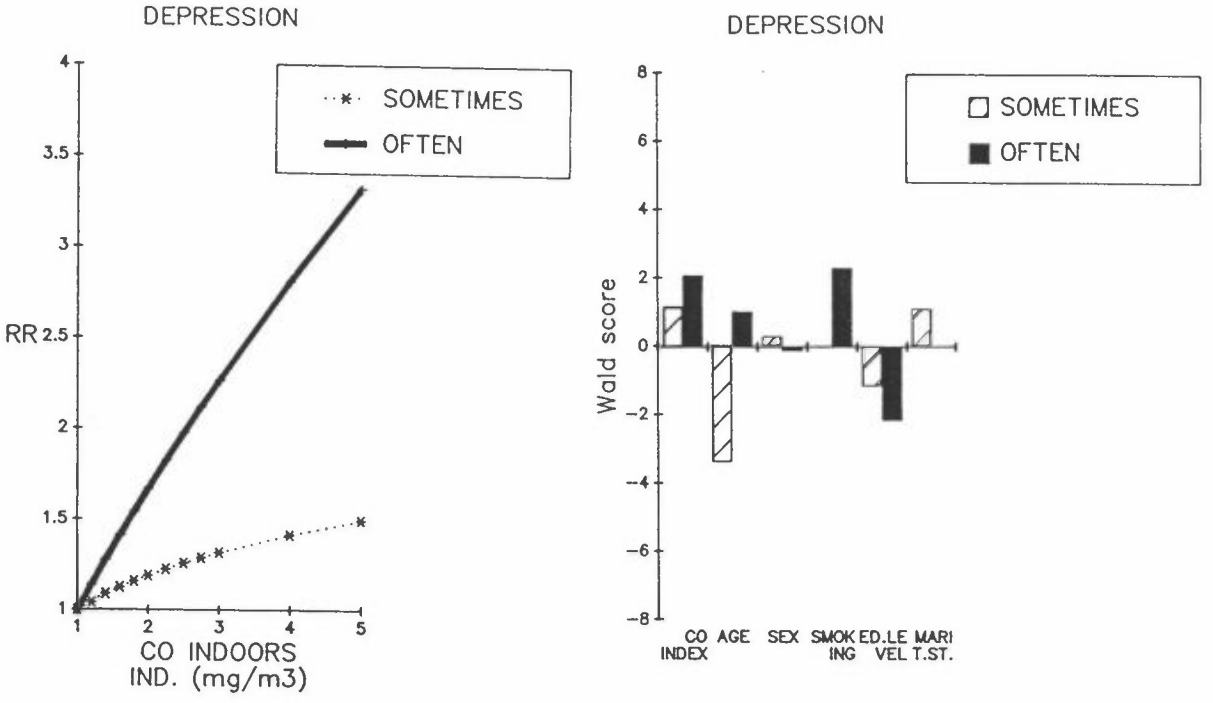


Figure 26: The relative risks of "sometimes" bothered as opposed to "often" bothered of being <u>depressed</u> at CO exposures indoors representative for the range estimated in Vålerenga. The Wald test statistic is given for comparison.

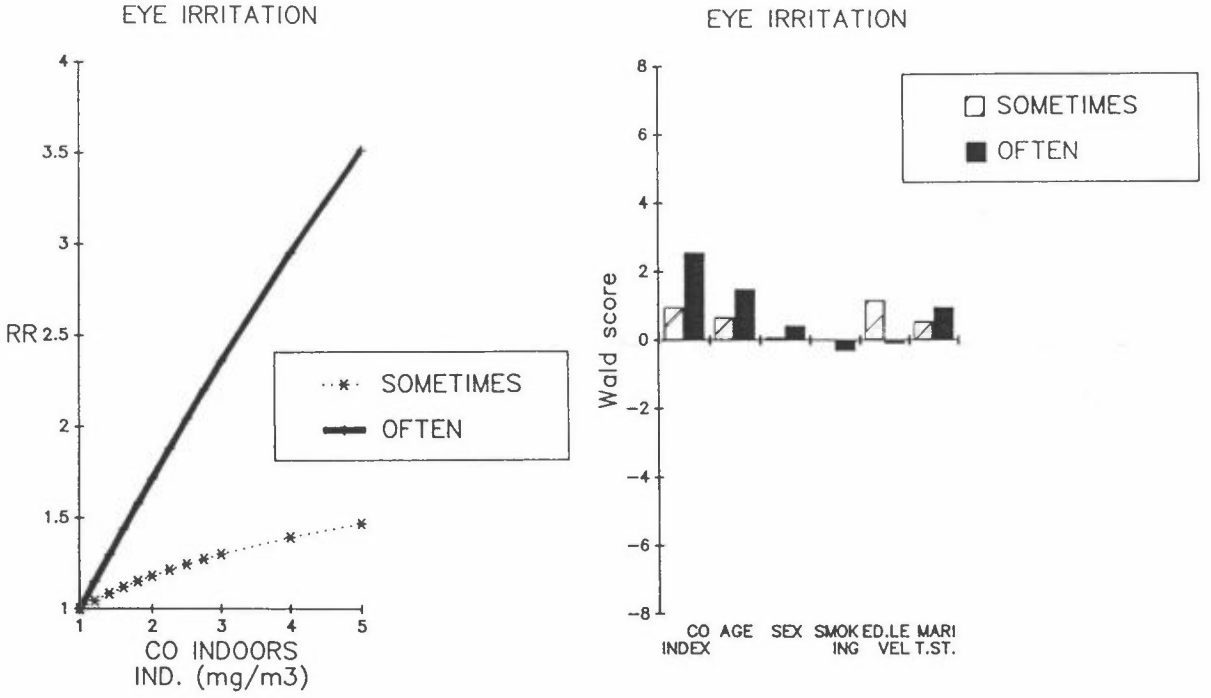


Figure 27: The relative risks of "sometimes" bothered as opposed to "often" bothered of having eye irritation at CO exposures indoors representative for the range estimated in Vålerenga. The Wald test statistic is given for comparison.

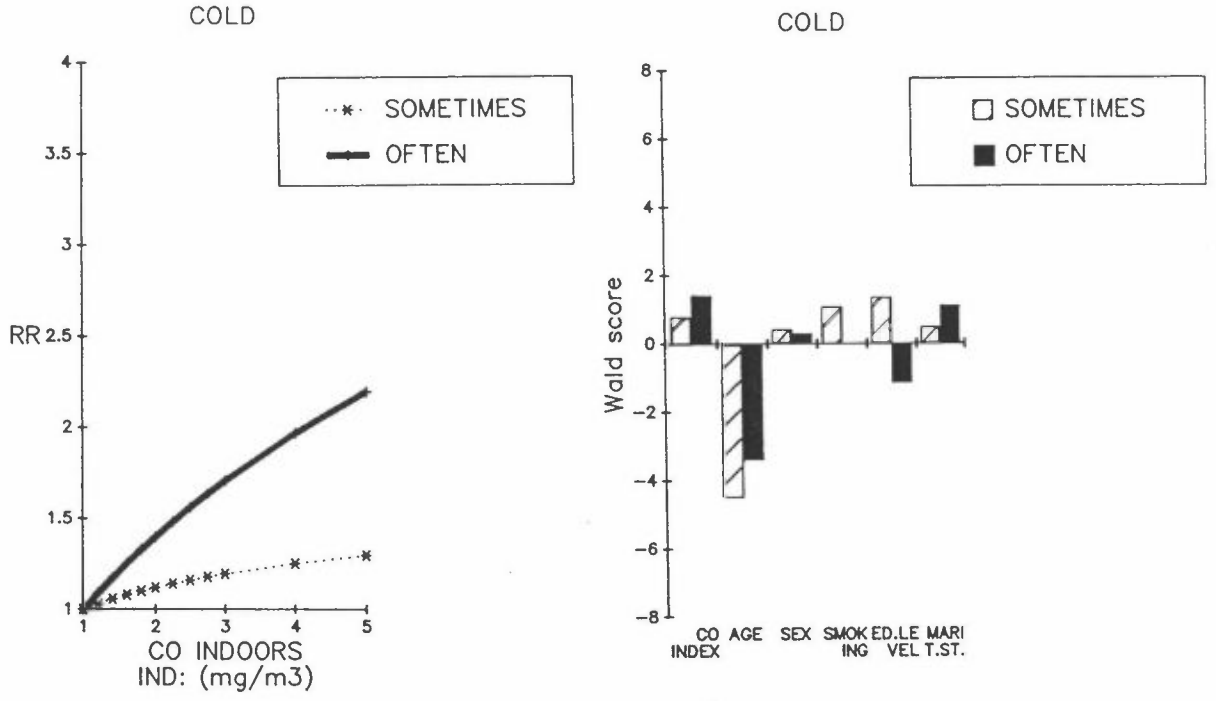


Figure 28: The relative risks of "sometimes" bothered as opposed to "often" bothered of having colds at CO exposures indoors representative for the range estimated in Vålerenga. The Wald test statistic is given for comparison.

4 RESULTS OF THE CROSS-SECTIONAL STUDY - LEVEL 2

In an attempt to answer the question of whether or not air pollution from vehicular traffic has an effect on human health, and to what extent, it was necessary to design the study in several levels.

In order to investigate thoroughly the exposure and health effects of traffic pollution in the Vålerenga area of Oslo, a subsample was chosen for indepth analysis. The entire population of 1028 was informed about the project and asked to volunteer. Of the original sample, 162 individuals participated in the study. Each participant came to the field center to be informed of the study, given the diary and asked to come back at the end of two weeks. During that period they were to fill out a two part diary concerning 1) where they were for each hour, information used to estimate exposure to air pollution, and 2) whether or not they were bothered by a set of health symptoms. Upon their return, each participant took a complete lung function test, a blood test where CO content was measured immediately and another blood sample for blood lead determination. In addition, each participant provided a breath sample where CO was also immediately measured. From previous experience (Clench-Aas et al.,1989b) it was known that the two methods for measuring the body burden of CO are very comparable, and the known correlations were then used in cleaning the data. These biological tests provided the basis for yet another cross-sectional study where the exposure information was now refined to include an average of the previous two weeks estimated exposure. So few children provided blood samples and lung function tests that they were excluded from further analysis.

4.1 CARBON MONOXIDE IN BLOOD AND BREATH

The carbon monoxide content of both blood (COHb) and breath was measured. Measurements of CO in blood were made immediately following blood sampling to increase accuracy. COHb is known

to depend on exposure to ambient CO (Ewetz and Camner, 1983) and to active smoking. In a smoker, the CO content of the blood is primarily a result of smoking. The results of CO in blood as a function of smoking (average number of cigarettes per day) and the relationships between CO in breath and blood, are shown in Figures 29 to 32.

Analysis of variance with covariates of COHb against smoking, (passive smoking in non-smokers) and ambient CO exposure the previous 4 hours revealed a significant correlation ($p<.01$) in non-smokers with a regression coefficient of .07 (ambient CO in mg/m^3, COHb in percent). In a previous cohort study done by NILU (Clench-Aas et al., 1989b) the regression coefficient between CO in blood and CO exposure the last 4 hours (as measured by portable CO monitors) was 0.08. These results indicate that COHb increases by .7% to .8% with an average increased exposure over 4 hours of 10 mg/m^3 (the recommended 8-hour air quality guideline). In Norway it is recommended that COHb concentrations not exceed 1.5%. CO is generated by the body itself, with baseline levels lying around 0.5%. Therefore exposure of 4 hours to current air quality guidelines should lead to COHb concentrations of 1.2%, under the suggested guidelines.

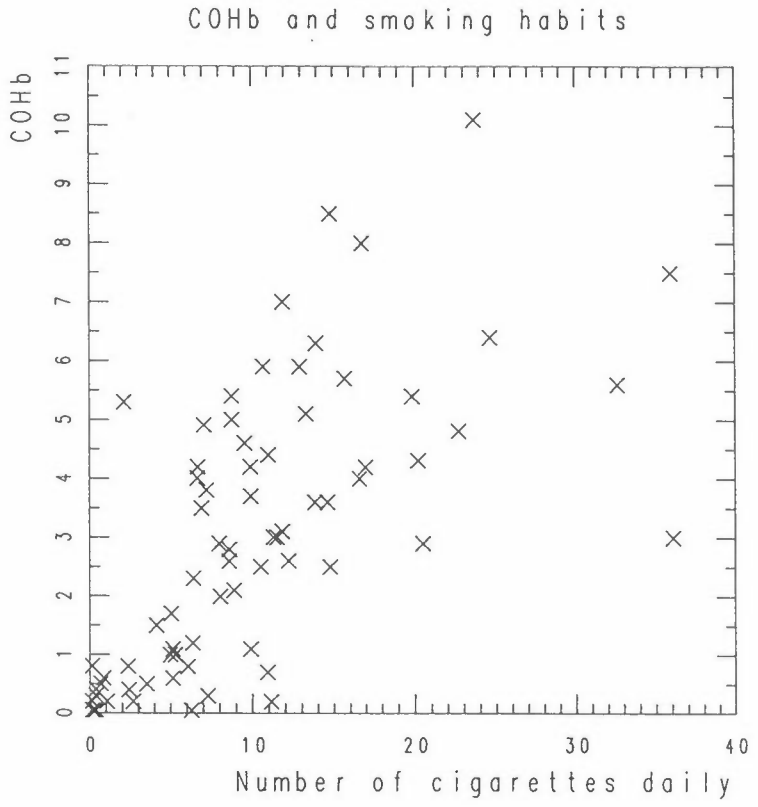


Figure 29: The relationship between COHb and the number of cigarettes smoked per day in smokers.

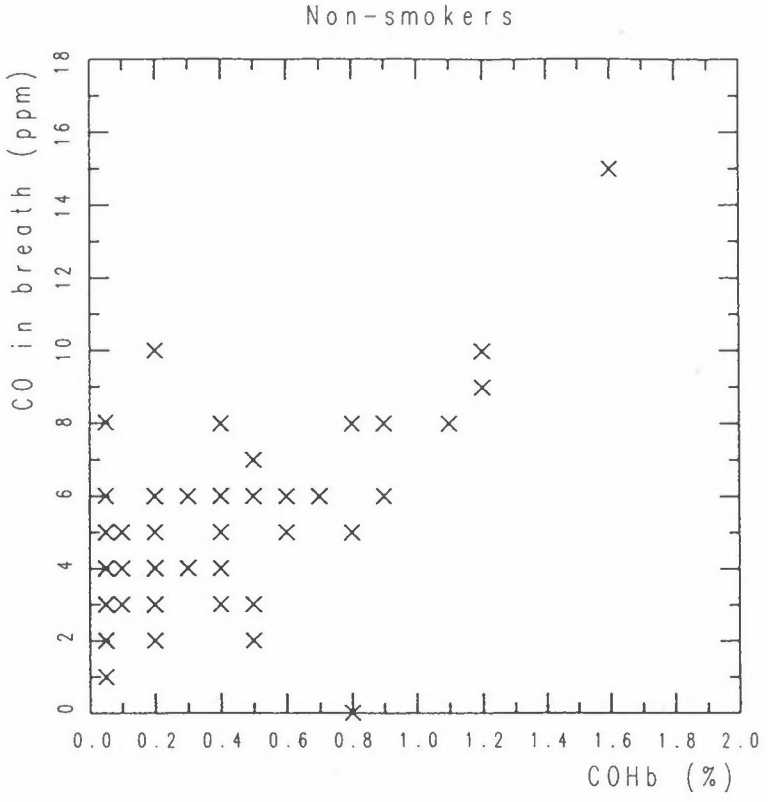


Figure 30: The relationship between COHb and CO in exhaled breath in non-smokers.

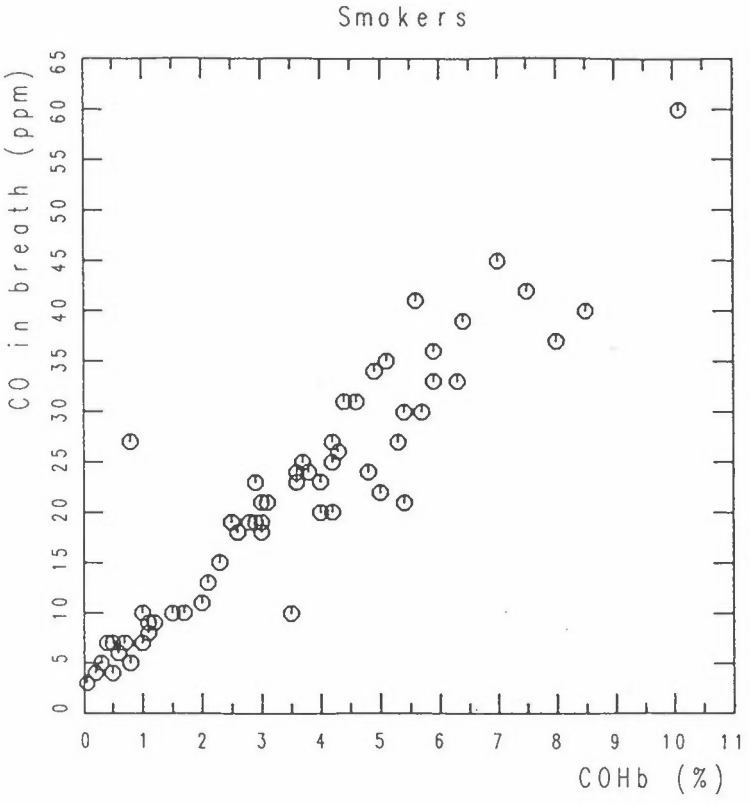


Figure 31: The relationship between COHb and CO in exhaled breath in smokers.

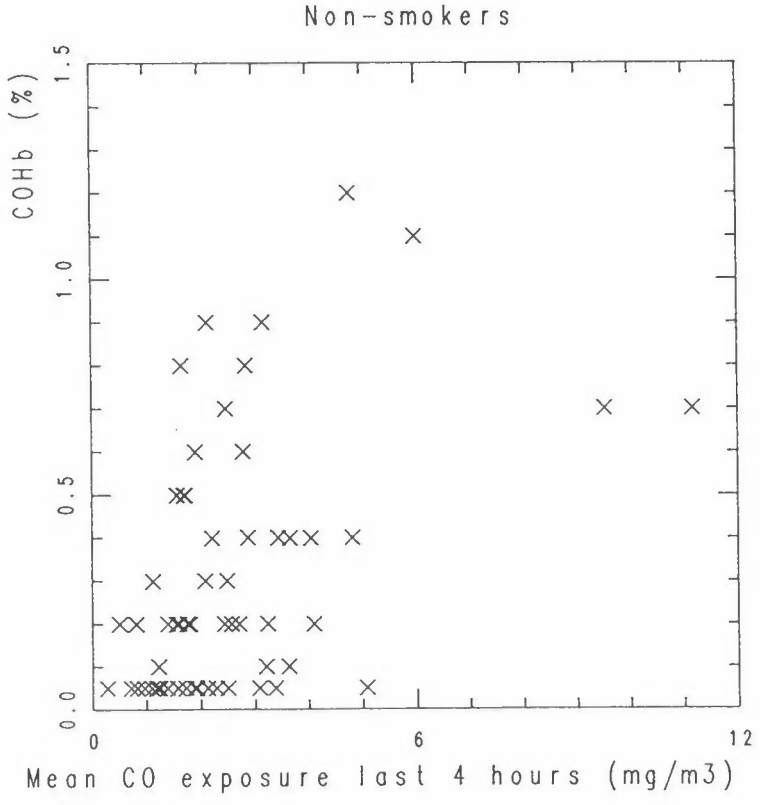


Figure 32: The relationship between COHb and CO exposure the last 4 hours in non-smokers.

4.2 LEAD IN BLOOD

Despite reductions of lead in gasoline and the introduction of lead free gasoline, it was decided in this study to control blood lead concentrations in the inhabitants of Vålerenga. Blood lead has a half-life of 16 days. It may also reflect lifetime accumulation in the bone since the blood concentration represents an equilibrium between the ingested and inspired lead on the one hand, and bone lead on the other. Concentrations of lead in blood in several Norwegian cities and towns have been previously reported (Clench-Aas et al., 1984; 1986; 1990).

Figure 33 compares blood lead concentrations in all these towns as a function of sex and age. The blood lead concentrations were all measured by the same laboratory and the same technicians. At the time of measurements in Holmestrand, there was heavy traffic through the town. Oslo-Nydalen represented an area that was exposed to industrial lead from an iron smelter. Sørumsand was a control town with no industrial and little vehicular lead exposure. No groups had mean values that approached the suggested limit for children (the most restrictive limit) of 15 µg/dl. However, middle aged men from Vålerenga had higher values than all other groups. These values were not correlated to either current air pollution exposure or time spent travelling.

Analysis of variance with covariates of Pb in blood with smoking, sex, age and ambient CO exposure revealed significant ($p \leq 0.01$) relations between Pb in blood, smoking and sex (with men and smokers having the higher values) and age.

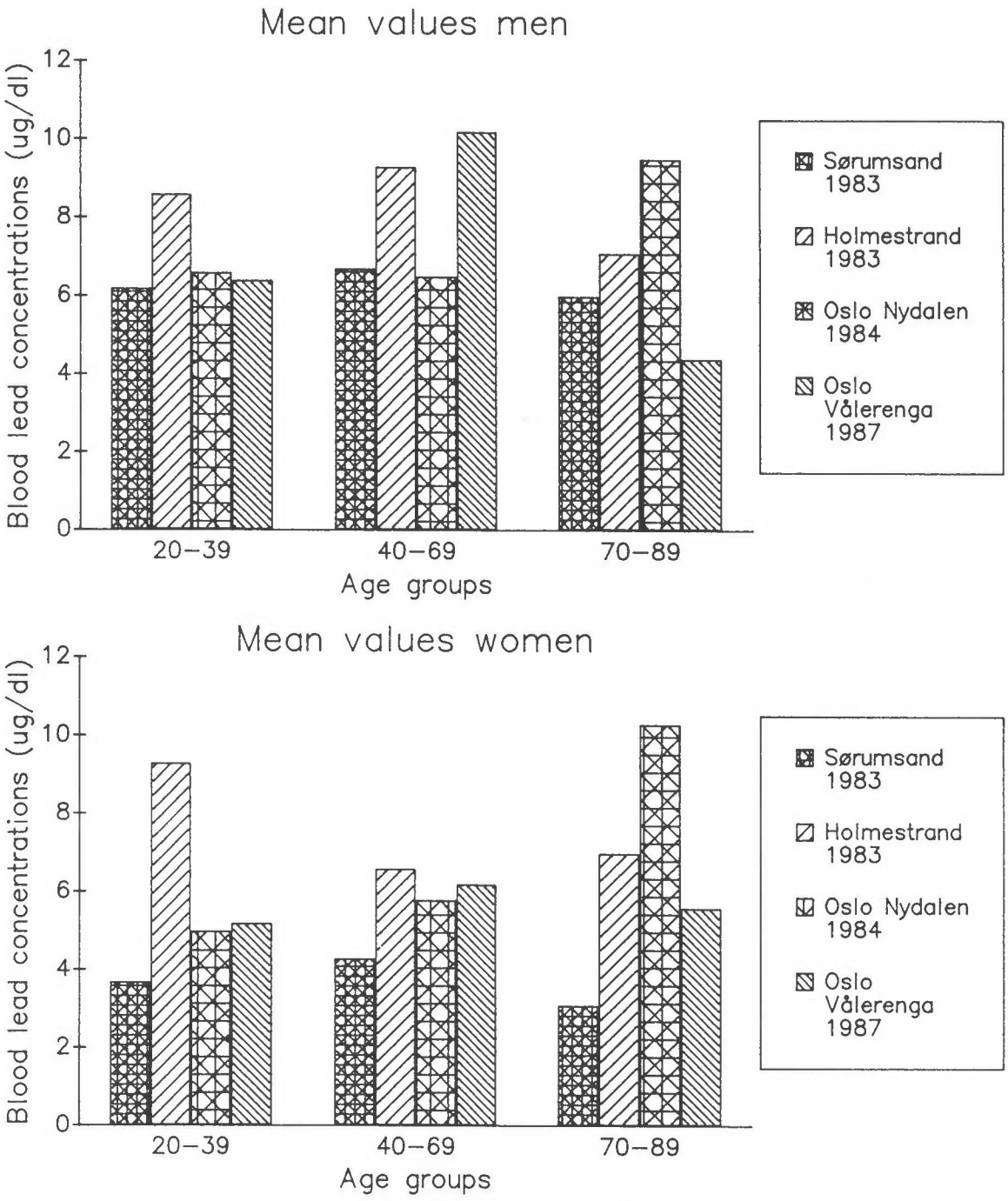


Figure 33: Mean concentration of lead in blood in men and women living in Norwegian towns as a function of age.

4.3 LUNG FUNCTION

A full lung function test was performed on each individual. The results of each individual's test results were compared to Norwegian standards developed by A. Gulsvik (1979), and then expressed as a percentage of these standards. The standards account for sex, age and height. It was desirable to correct for these biological features since it was possible that the elderly and women were more exposed to air pollution than other groups. This could have been manifested as a sex or age difference if the correction by standards was not performed.

Relating lung function to COHb resulted in highly significant relationships between reduced lung function in non-smokers and higher COHb. These are summarized in Figure 34. As can be seen in this figure, an increase of COHb of 1% decreases vital capacity by 11%, forced vital capacity by 17% and forced expiratory volume by 13%. These reductions are significant and indicate surprisingly marked reductions in lung function with CO exposure at CO values less than air quality guidelines. It should be reemphasized here that traffic pollution consists of mixture of many compounds and that CO can only be considered an indicator substance. COHb was used as a measure of CO exposure (see chapter 4.1). However, the possibility does exist that reduced lung function leads to increased retention of CO in blood.

Studying the relationship between lung function and air pollution exposure revealed a significant increase in FEV_1/FVC (regression coefficient = .167 with NO_2 exposure) and a positive relationship of FEF_{25-75}. The increase in values of FEV_1/FVC may be indicative of constrictive as opposed to obstructive changes in lung function.

The results of this investigation are provocative and interesting to pursue, but a larger sample size is necessary in future investigations.

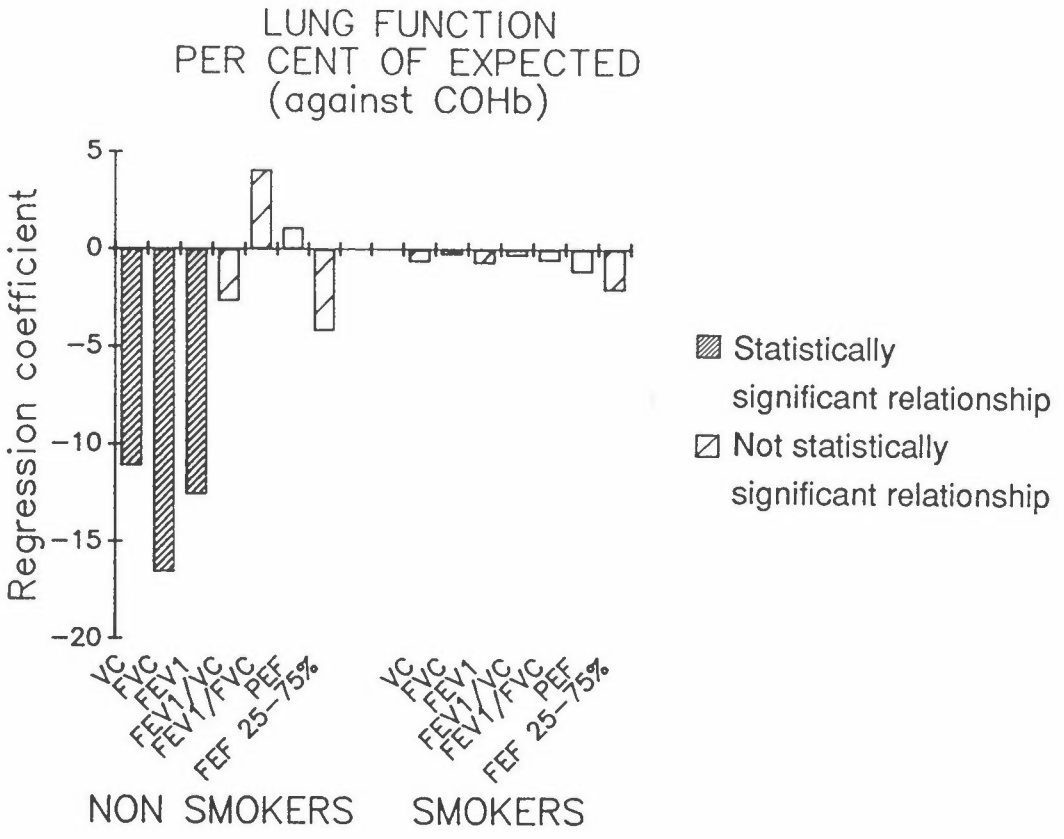


Figure 34: Calculated regression coefficients in non-smokers and smokers of various lung function parameters and levels of COHb.

5 RESULTS OF THE COHORT STUDY - LEVEL 2

A cohort of 162 individuals provided information on an hour by hour basis. A cohort study follows an individual over time. It has the advantage that each individual is his own control and therefore, problems of confounding factors are minimized. Since individuals are biologically different and have different sensitivities, a cohort study increases the possibility of identifying these sensitive individuals.

In this study, not only did the individuals fill out a diary for each hour indicating whether or not they were bothered by any of a set of health symptoms, but they took peak expiratory flow measurements (PEF) four times daily using portable Mini-Wright PEF monitors.

5.1 HEALTH SYMPTOMS

It was of interest to compare what per cent of the population was sensitive to traffic pollution exposure. Each individual and each of the 13 health symptoms were analyzed.

The first step in analysis of the information in the diaries was to compare the percentage of time that individuals were bothered by the various symptoms while in different micro-environments or while doing different activities. This information is summarized in Tables 4 to 7. There seems to be a higher percentage of hours being bothered while people are shopping or at work than at home; a slight increase with increase in pollution exposure; increased percentages while travelling or outdoors; and increased percentages while smoking themselves or being exposed to passive smoking.

As indicated in Table 5 and Figure 35 there is a tendency to increased per cent of time being bothered by certain symptoms with increased exposure to air pollution.

Table 4: Mean number of hours in different locations, and mean % of hours with reporting of symptoms in different locations.

	Location				
	Home	Work-place	School/ day-care centre	Other places	Travel-* ling or shopping within hour
Number of hours	218.2	60.9	37.1	45.6	37.0
Fatigue	3.6	6.6	3.3	1.7	5.6
Nervous/restless	0.6	0.6	0.5	0.3	1.0
Headache	2.0	4.4	1.9	1.7	3.0
Nausea	0.8	0.8	0.5	0.7	1.0
Sneezing/running nose	5.4	9.2	5.5	5.8	8.0
Feeling feverish	1.4	2.1	0.1	1.6	1.2
Eye irritation	1.8	2.3	0.0	2.1	2.1
Throat irritation	4.5	8.0	6.0	4.2	6.7
Wheezing	0.3	0.3	0.0	0.0	0.3
Tightness in chest	2.2	4.1	0.0	1.6	3.0
Coughing	3.0	2.7	4.1	2.2	3.8
Bothersome noise	3.3	7.5	1.1	2.3	9.5
Bothersome smell	1.9	3.5	1.2	1.6	5.4

* The individual has travelled or shopped at last 5 minutes within the hour.

Table 5: Mean per cent of time with reporting of symptoms by level of exposure to NO_2 (NO_2 is used as an index for exposure to traffic pollution).

	Level of NO_2 exposure									
	0-5 µg/m³	5-10 µg/m³	10-15 µg/m³	15-20 µg/m³	20-30 µg/m³	30-40 µg/m³	40-60 µg/m³	60-100 µg/m³	100-200 µg/m³	>200 µg/m³
Mean no. of hours per indiv.	76.7	23.8	24.0	28.2	50.8	33.9	41.0	27.7	7.8	2.1
Nr. of individuals	157	154	157	157	157	157	157	156	142	54
Fatigue	1.9	3.8	4.4	4.1	5.2	4.5	4.6	4.3	5.8	6.8
Nervous/restless	0.4	0.7	0.6	0.5	0.7	0.8	0.7	0.6	0.2	
Headache	1.1	2.2	2.9	2.7	2.8	3.1	3.1	2.5	1.3	1.9
Nausea	0.6	0.7	0.9	0.9	0.8	1.1	0.9	0.7	0.2	
Sneezing/running nose	3.7	6.1	5.8	6.3	6.5	6.4	7.8	6.5	8.0	17.8
Feeling feverish	0.9	0.9	1.5	1.6	1.6	1.5	1.3	0.8	1.7	
Eye irritation	1.3	2.7	2.5	2.1	2.3	2.0	2.7	2.1	2.2	2.8
Throat irritation	3.1	4.7	5.0	5.6	5.4	6.3	5.8	6.3	5.5	4.9
Wheezing	0.2	0.3	0.3	0.2	0.2	0.4	0.5	0.4	0.2	
Tightness in chest	1.6	2.4	2.4	2.5	2.6	2.4	2.4	2.6	1.8	3.7
Coughing	2.1	3.6	3.1	3.1	3.5	3.3	3.9	3.4	4.1	7.4
Bothersome noise	2.6	3.6	4.0	4.0	4.4	5.0	5.6	6.7	7.0	5.7
Bothersome smell	1.6	2.2	2.0	2.7	2.6	3.1	3.7	4.0	4.5	2.6

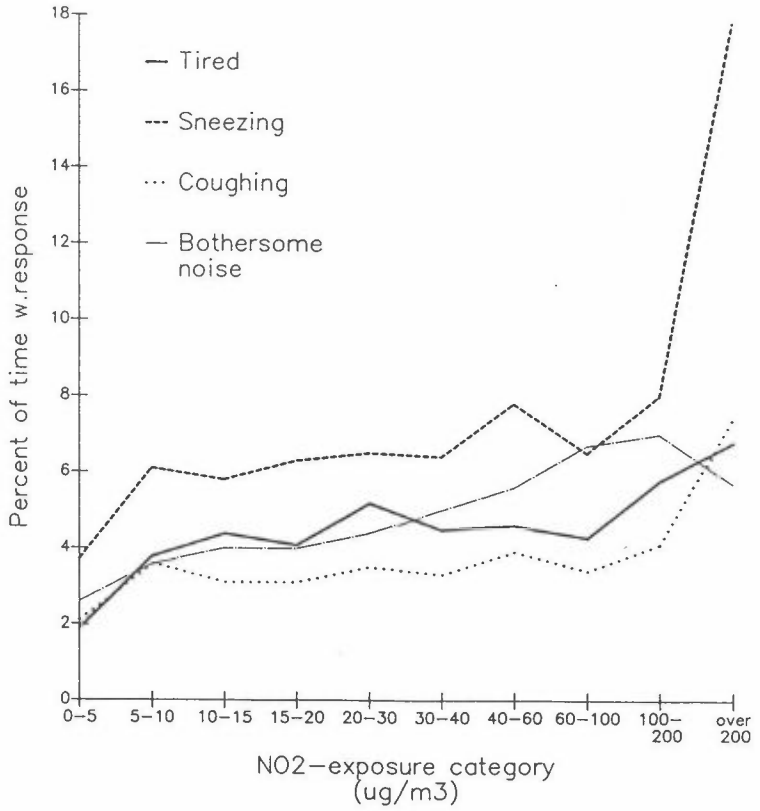


Figure 35: Mean per cent of time with reporting of symptoms by level of exposure to NO_2 (an index for traffic pollution) for selected health symptoms that showed statistical significance using the Korn-Whittemore approach.

Table 6: Mean number of hours in different locations, and mean % of hours with reporting of symptoms in different locations.

	Travelling for whole hour	Indoors	Indoors with open window	Outdoors
Number of hours	10.6	241.7	84.5	13.9
Fatigue	5.2	4.2	3.0	2.7
Nervous/restless	0.9	0.7	0.6	0.1
Headache	2.2	2.8	2.0	2.7
Nausea	0.7	0.9	0.7	1.0
Sneezing/running nose	7.7	6.8	5.1	7.5
Feeling feverish	1.5	1.2	1.4	1.3
Eye irritation	2.0	2.0	2.4	3.2
Throat irritation	5.5	5.5	4.9	6.2
Wheezing	0.3	0.2	0.5	0.2
Tightness in chest	3.3	2.4	2.5	2.5
Coughing	3.6	3.1	3.5	3.1
Bothersome noise	9.5	4.2	5.0	8.6
Bothersome smell	5.4	2.5	2.7	4.0

Table 7: Mean number of hours at different tobacco smoke categories, and mean % of hours with reporting of symptoms in different categories.

	Smokers while smoking	Smokers while not smoking	Non-smoker while exposed to passive smoking	Non-smokers while not exposed to passive smoking
Number of hours	112.6	237.1	25.7	328.8
Fatigue	4.2	1.8	4.0	4.4
Nervous/restless	0.9	0.1	0.3	0.6
Headache	3.6	1.3	3.4	2.2
Nausea	0.9	0.7	1.0	0.8
Sneezing/running nose	7.0	3.9	8.2	6.6
Feeling feverish	1.3	1.0	1.2	1.2
Eye irritation	2.0	0.8	5.6	2.2
Throat irritation	9.5	4.4	7.9	4.3
Wheezing	0.8	0.5	1.3	0.0
Tightness in chest	4.8	2.7	2.1	1.5
Coughing	5.3	2.0	3.0	2.9
Bothersome noise	4.7	2.7	9.5	5.3
Bothersome smell	2.1	1.5	7.2	3.0

Health symptom reporting was related to air pollution exposure hour by hour. In order to make the response variables uniform in quality, the data were compressed over sleep so that the entire sleep time was equal to one hour. The reasoning was that the quality of reporting of health symptoms was not reliable at night. People tend to report having a headache all night if they have a headache when going to bed or waking up. In a first step the numbers of individuals having significant logistic regression coefficients for the relationships of health symptom reporting and exposure to traffic pollution (the natural logarithm of the NO_2 estimates) are given in Table 8. As is evident in Table 8, between 1 to 13% of the entire study population for each health symptom had significant relationships to traffic pollution exposure. However, up to 36% of those who complained at least once of a particular health symptom had significant relationships between health symptom and air pollution exposure.

Table 8: The percentages of individuals having positive significant relationships between each of the health symptom parameters and air pollution estimates. Expressed as % of total population and % of population complaining at least once of a health symptom.

Health symptom	N with symp.	% of population having symptom	% of total population
Fatigue	15	17	9
Nervous/restless	2	8	1
Headache	8	11	5
Nausea	2	2	1
Sneezing/running nose	15	24	9
Feeling feverish	2	7	1
Eye irritation	2	6	1
Throat irritation	13	19	8
Wheezing	1	2	0
Tightness in chest	3	13	2
Coughing	4	10	3
Bothersome noise	21	31	13
Bothersome smell	20	36	13

It was not the same people who suffered all the health symptoms. It was possible to count the number of individuals who had a positive significant relationship between at least one health parameter and air pollution exposure estimates. Using this approach, 57 individuals or 40% of the population had at least one health symptom positively significantly related to air pollution exposure. Of these, 9% complained only of annoying noise or smell. This is substantially higher than the 10% reported here when looking at each symptom individually.

Table 9 provides the regression coefficients for the health parameters against the natural logarithm of NO_2 (statistical significance indicated by the Z value).

Table 9: Weighted mean regression coefficients of individual logistic regression analysis of health symptom versus NO_2 exposure. Multiple regressions were run with the following parameters: "Markov" (the value of the health parameter the preceeding hour), outdoor temperature, relative humidity, passive smoking, smoking, exposure to extra pollution through other activities, hard exercise, NO_2 exposure (logarithmic). These weighted means were calculated using the random effects model of the Korn and Whittemore 1979 model. Odds ratios at 150 and 200 $\mu g/m^3$ NO_2 (compared to a base value of 10 $\mu g/m^3$) are indicated for the significant regressions ($p<0.10$).

Name	No. with symptom (pos.sign)	Coeff. NO_2	Std. err.	Z*	Odds ratio NO_2 ($\mu g/m^3$) 200	150
Fatigue	87 (15)	.0350	.0204	1.718	1.233	1.209
Nervous/restless	26 (2)	-.0088	.0496	- .178		
Headache	76 (8)	-.0102	.0253	- .405		
Nausea	36 (2)	-.0552	.0380	-1.451		
Sneezing/running nose	62 (15)	.1012	.0226	4.476	1.843	1.737
Feeling feverish	30 (2)	-.0667	.0590	-1.131		
Eye irritation	33 (2)	.0367	.0358	1.025		
Throat irritation	69 (13)	.0857	.0274	3.128	1.671	1.591
Wheezing	6 (1)	.0051	.0471	.107		
Tightness in chest	23 (3)	.0684	.0397	1.721	1.503	1.445
Coughing	41 (4)	.0288	.0280	1.028		
Bothersome noise	60 (21)	.1952	.0242	8.064	3.216	2.875
Bothersome smell	55 (20)	.1800	.0297	6.053	2.940	2.651

Z = coefficient/std.error. Z over 2 in absolute value can be roughly considered significant on 5% level.

From these regression coefficients the odds ratio can be calculated. The results are shown in Figure 36. From these graphs one can read directly the odds ratio, interpreted as an increased risk of having a health symptom at different concentrations of NO_2 (again, NO_2 exposure is an indicator reflecting exposure to traffic pollution) relative to a base NO_2 exposure level. For example, for NO_2 at the hourly air quality guideline of 200 $\mu g/m^3$ the odds ratio for being tired is 1.2 or 20% higher than at a level of 10 (base level). The relative risk of having a sore throat, sneezing or having a tight chest is approximately between 1.5 and 1.81, or 50 to 80% higher than at a level of 10. Estimated exposure was less than 10 $\mu g/m^3$, 32 percent of the time. This level does not therefore represent the lowest exposure concentrations.

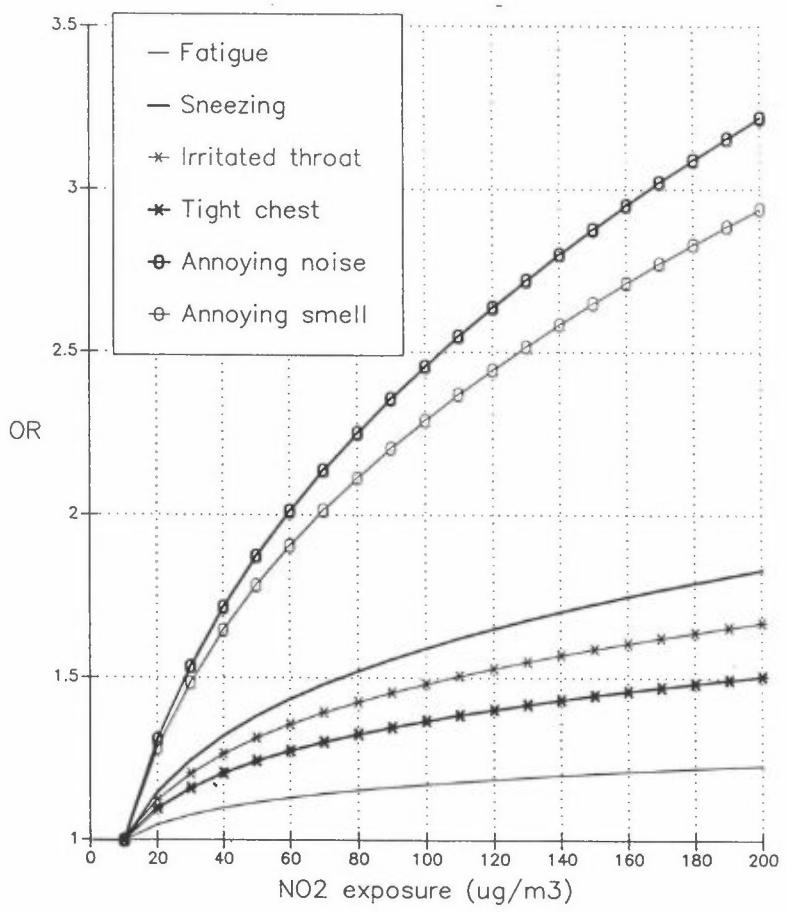


Figure 36: The odds ratio of a set of health parameters as a function of estimated air pollution exposure.

The odds ratio at a higher level A is calculated as

$$OR(A) = \exp \{\beta \ (\ln(NO_2 \text{ at level A}) - \ln(NO_2 \text{ at reference level}))\}$$

5.2 DAILY MEASUREMENTS OF PEAK EXPIRATORY FLOW (PEF)

5.2.1 Comparison of PEF measured by Mini-Wright peak expiratory flow meter and by spirometric tests

PEF measurements were recorded during the study by each individual four times per day using a Mini-Wright Peak Flow Meter. In addition, each individual's lung function was tested once during the field study using a Vitalograph-compact, the approximate hour and date of this test being recorded as well.

The Mini-Wright recordings used in the comparison were those taken each day within 3 hours of the hour when the full spirometric test was performed. After this screening only those individuals that had more than 5 PEF measurements were included in the comparison (132 individuals).

Based on the Mini Wright readings, a 5% tolerance interval for the PEF readings was constructed for each individual. The results of PEF-measurement taken by the spirometric test apparatus were then related to this interval; for 52% of individuals, these were within the indicated individual's tolerance interval and 7% were under the lower limit of the tolerance interval. Mean difference between the mean value of PEF taken by Mini-Wright and the PEF from the full test was -26 l/min with standard error of 9.3; mean standardised difference, that is

$$(\text{mean } PEF_{MINI\text{-}WRIGHT} - PEF_{FULL\ TEST})/\text{STD.DEV. of } PEF_{MINI\text{-}WRIGHT}$$

was 0,61 (median value at -1.48). For a frequency distribution of the standardized differences, see Figure 37.

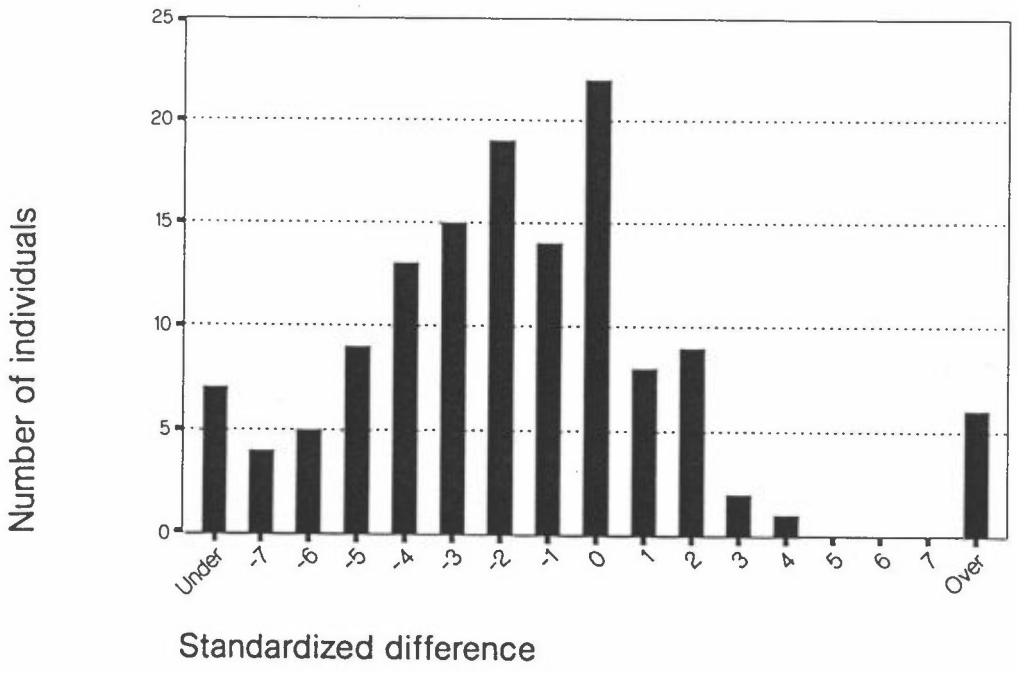


Figure 37: Standardized difference between individual's PEF taken by Mini-Wright Peak Flow Meter and PEF taken by a spirometric apparatus. Values below zero indicate that PEF taken by Mini-Wright is lower than the test apparatus.

5.2.2 Method of analysis of daily measurements of PEF

Peak expiratory flow varies during the day with the highest values occurring during the afternoon in individuals synchronized with time of awakening in the morning and sleep at night. Variations in peak flow reflect changes in the patency of the airways, the highest peak flow values corresponding to the largest patency. When examining the effects of air pollution on

peak flow, it is necessary to account for these daily (circadian) variations in lung function, and to take into account that effects of environmental agents need not be uniform during the day.

A fall in PEF in the middle of the night is a natural phenomenon that does not in itself indicate a negative effect of an environmental factor. If, however, this fall is more than average, the question can arise as to what caused PEF to decrease so. On the other hand, if the effect of an agent is to contract the airways, it is likely that the constriction will be most noticeable at the time of day when the airways are the most open, thus when patency is at a maximum. Therefore, it is not evident that effects of environmental agents are uniform during the day.

To handle these problems, it was decided to first correct the PEF values for their natural rhythm. This was done on a group as well as on an individual basis. For the group, three days worth of data were selected (each participant's 7th to 9th day). When correcting PEFs on an individual basis, all registered data was used. Parameters of the circadian variation of PEF were thus estimated for the entire group and for each individual using a cosinor function (Bingham et al., 1982).

The cosinor function considered for the PEF was the following:

$$y_i = M + A\cos(\omega t_i + \theta) + e_i, \qquad (1)$$

where Y_i is the PEF measurement at time t_i, M is the mesor (24-hour average), A the amplitude (half the estimated difference between the estimated daily minimum and maximum), θ the acrophase (the time of day that the peak values occur), and ω angular frequency (in radians). The angular frequency is inversely proportional to the period, the time interval between successive maxima. We assume the period to be 24 hours.

Equation (1) can be linearized using rules for calculating with trigonometric functions as

$$y_i = M + \beta x_i + \gamma z_i + e_i , \qquad (2)$$

where

$$x_i = \cos(\omega t_i) \qquad \beta = A \cos(\theta)$$
$$z_i = \sin(\omega t_i) \qquad \gamma = -A \sin(\theta).$$

For each time point, a difference was calculated between the measured PEF-value and the corrected value. The differences were expressed as a per cent of the individual mean PEFs to enable interindividual comparisons. The analyses were done separately for the morning (minimum) and for the afternoon (maximum) data.

5.2.3 Description of the PEF values

Each individual's PEF values are described by four parameters: the individual mean, range (difference between the observed maximum and minimum), amplitude (half of the mean range estimated from the individual cosinor), and acrophase (time of day of the daily peak estimated from the individual cosinor function). Using multiple regression analysis, including as independent variables sex, age and smoking (smoker/non-smoker), a significant increase in acrophase was found with increasing age (0.07% of the mean per year).

The mean of the individual amplitudes of PEF expressed as per cent of mean PEF was 3.4 (see Figure 38), although there were much higher values. For a person with PEF-mean of 600 l/min (male, approx.180 cm), the estimated difference between daily minimum and maximum based on this estimate would be 40 l/min. However, the group cosinor estimated the mean amplitude to be 1.3 %, yielding a much lower difference of 15 l/min. This suggests large intra-individual variations. These results are in

agreement with results of Bjerknes-Haugen et al. (1991) on a general adult Norwegian population.

It was suggested that PEF measurements be taken at 800, 1200, 1600 and 2000, and most of the readings were taken around these hours (see Table 12). The individual acrophase estimates for the daily PEF values were primarily between 1400 and 1700 with a mean around 1500 (see Figure 39). The estimated common acrophase was at 1600. As a result, in the analysis the minimal (morning) PEF value was taken to be between 600 and 1100, and the maximal (afternoon) between 1400 and 1900. No physiological variability (sex, age, height) was found for acrophase.

Multiple regression was used in an attempt to assess the impact of smoking, sex and age on both individual mean PEF values and individual PEF ranges. The results are summarized in Tables 10 and 11.

Mean individual PEFs ranged from 120 to 780 l/min. Women had lower mean PEF than men (100 l/min) (see Figure 40), and mean PEF was found to be dependent on height (ca 4 l/min per cm height). For smokers, the differences between sexes seemed a little lower (87 l/min). These results are in agreement with results found by Bjerknes-Haugen et al (1991) in a general Norwegian population.

The highest range of individual PEF values found was 320 l/min, with typical values between 40 and 120 (see Figure 41). No significant difference in the range of the PEFs between the sexes or with height or age was confirmed. However, there is an indication of a little larger range in smokers (with 1.3 l/min per each cigarette smoked), and in smokers there seems to be higher range found with increased age.

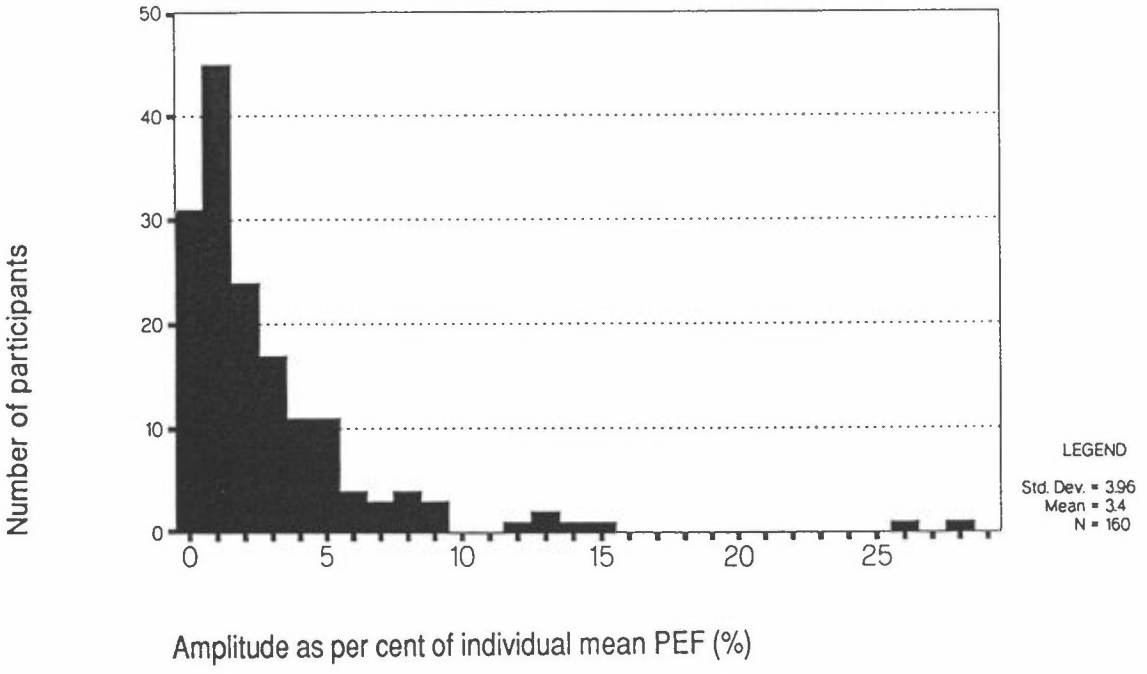


Figure 38: Frequency distribution of individual amplitudes of PEFs as measured by the Mini-Wright.

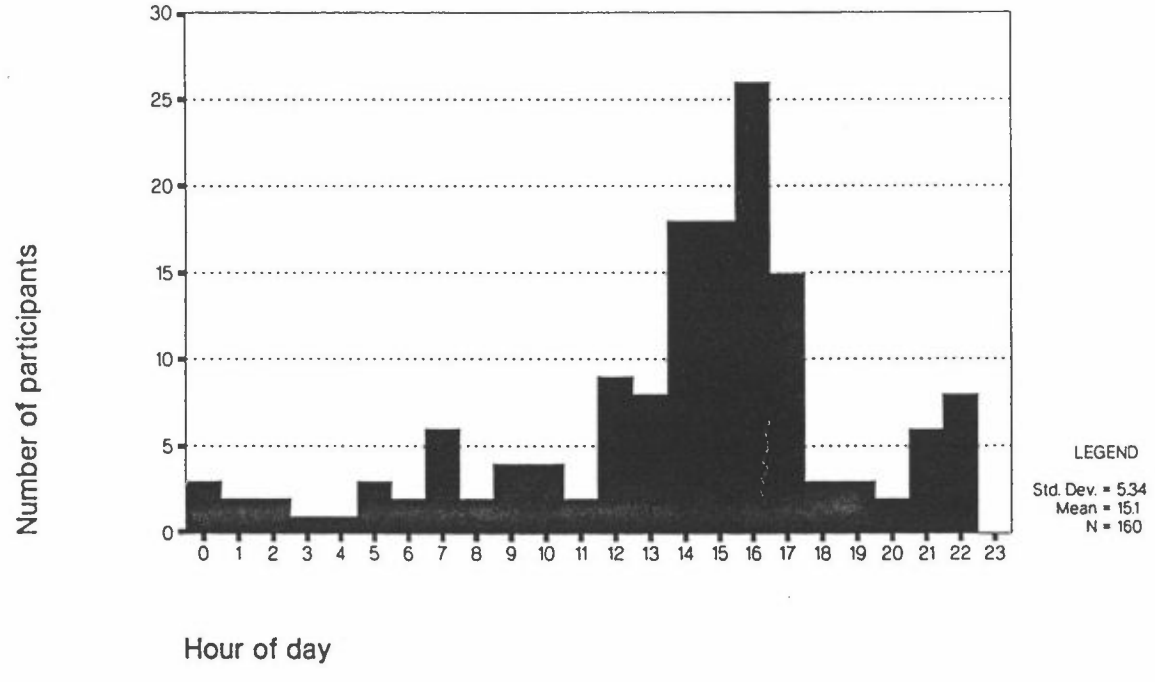


Figure 39: Frequency distribution of the mean individual acrophases (time of peak) for PEFs as measured by the Mini-Wright.

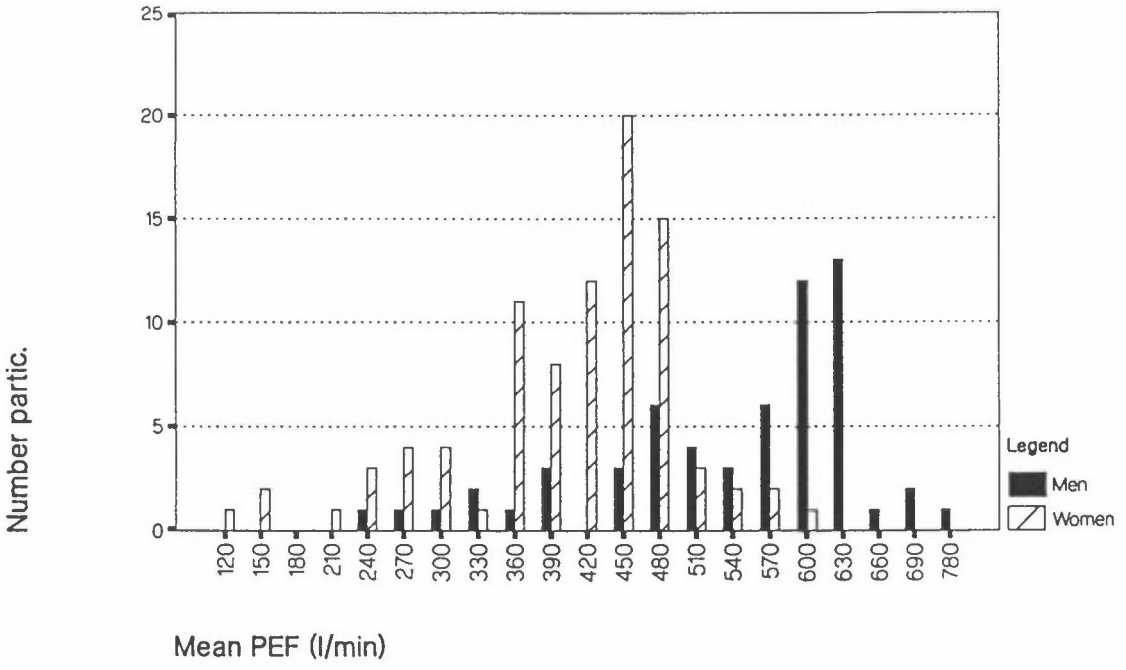


Figure 40: Frequency distribution of the mean of PEF values measured by the Mini-Wright for men and women.

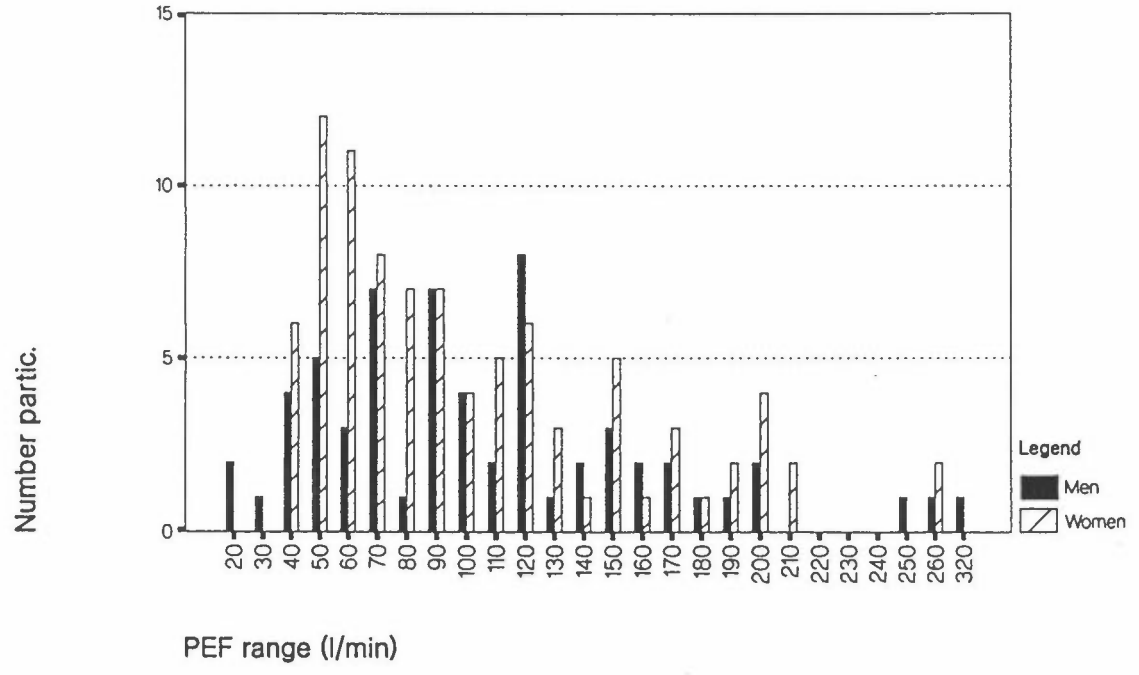


Figure 41: Frequency distribution of the range of PEFs as measured by the Mini-Wright for men and women.

Table 10: Results of the multiple regression of the mean PEF values against smoking (number of cigarettes smoked per day), age, sex and height. Regression coefficients (b) are presented with their t-values.

PARAMETERS	ALL N = 130		NON-SMOKERS N = 68		SMOKERS N = 62	
	b	t	b	t	b	t
Smoke*1	-1.66	-1.67	-	-	-0.58	-0.46
Age	1.38	0.48	6.28	1.47	-5.90	-1.52
Age squared	-0.03	-1.18	-0.08	-1.87	0.03	0.90
Sex*2	-99.7	-5.27	-109.4	-3.89	-86.71	-3.57
Height	4.36	4.24	3.79	2.36	5.21	4.01
Constant	-80.40	-0.40	-74.40	-0.24	-91.40	-0.36
% explained variability	53		54		56	

*1 Mean number of cigarettes per day
*2 Men=1; Women=2.

Table 11: Results of the multiple regression of the range of PEF values against smoking (number of cigarettes smoked per day), age, sex and height. Regression coefficients (b) are presented with their t-values.

PARAMETERS	ALL N = 130		NON-SMOKERS N = 68		SMOKERS N = 62	
	b	t	b	t	b	t
Smoke*1	1.28	1.87	-	-	1.15	1.39
Age in yrs	1.37	0.70	-1.33	-0.44	4.88	1.90
Age squared	-0.01	-0.42	0.02	0.61	-0.04	-1.70
Sex*2	2.02	0.16	10.60	0.52	-10.89	-0.67
Height in cm	0.33	0.47	0.66	0.58	-0.07	-0.08
Constant	-6.44	-0.05	-16.50	-0.07	6.46	0.04
% explained variability	7		3		17	

*1 Mean number of cigarettes per day
*2 Men=1; Women=2.

5.2.4 Short term effects of exposure to traffic pollution on the peak expiratory flow

To test the short-term effect of air pollution on PEF, multiple linear regression analysis was performed separately on morning (minimum) and afternoon (maximum) PEF-value for each individual. The dependent variable was the difference between the measured PEF and the individual estimate of the cosinor function recalculated as per cent of the individual mean PEF. The independent variables in the model were mean estimated exposure to NO_2 since the last PEF-measurement (indicator for traffic pollution), and the mean outdoor relative humidity since the last PEF measurement. The number of hours preceeding the PEF measurement over which the mean exposure was calculated was restricted to a maximum of 16 hours. Table 12 gives an overview of number of PEF-readings taken at different times of day, and indicates also median length of the preceeding aggregating period.

Table 12: Number of PEF-readings registered at different hours of day, and median length of the aggregating period (max. length is 16 hours).

Hour of day	Readings regist.	Median length of agg.	Hour of day	Readings regist.	Median length of agg
0100	20	8	1300	489	5
0200	23	5	1400	212	5
0300	5	16	1500	114	5
0400	17	16	1600	1019	4
0500	12	9	1700	507	4
0600	64	9	1800	231	5
0700	143	10	1900	121	5
0800	970	11	2000	908	4
0900	415	12	2100	445	4
1000	172	13	2200	230	4
1100	112	5	2300	197	5
1200	998	4	2400	202	7

Air pollution did not significantly explain the variation of PEF in the population. Table 13 shows the number of men and women, smokers and non-smokers whose covariation between PEF and NO_2 exposure were significant for morning and afternoon PEFs.

The multiple regressions significantly explained variation in PEF for between 7% and 20% of the participants in the sub-groups. However, there are both individuals with positive and negative relationships between PEF and NO_2, and the group combined coefficients are not significantly different from zero for any group. As a combined coefficient we use the arithmetic mean. Even if more elaborate methods are possible, the standard error of the individual coefficients does not vary substantially and therefore this treatment is adequate. It should be borne in mind that the series of morning and afternoon PEF values for an individual are approximately 14 measurements long, with a natural variability that may not permit obtaining a significant result for such a relatively short series.

One may perhaps argue that to correct the data for individual cosinor function is to take away too much of the variability due to exposure. To elucidate this, the regressions were rerun for PEF values corrected for the common cosinor, with much the same result.

Another question that arises concerns the relationship between the individual amplitude of PEF readings and the cumulated pollution exposure (index from Level 1 study, mean and 90-percentile NO_2 exposure during the Level 2 study). No relationship with these exposure indicators was revealed.

Table 13: The results of individual regressions of PEF (corrected for individual cosinor) A) in the morning and B) in the afternoon.

A)

	Regression		Number with NO_2 coefficient			NO_2 coefficient	
	Total	Signif.	Negative	N.S.	Positive	Mean	Standard Deviation
Sex							
Men							
Smoker							
No	28	4	2	27		-.010	.085
Yes	25	4	1	27		-.054	.172
Female							
Smoker							
No	42	7	2	39	3	-.053	.345
Yes	39	5	2	40	2	-.017	.267

B)

	Regression		Number with NO_2 coefficient			NO_2-coefficient	
	Total	Signif.	Negative	N.S.	Positive	Mean	Standard Deviation
Sex							
Men							
Smoker							
No	27	2	2	27		-.006	.067
Yes	27	2		25	3	.020	.089
Female							
Smoker							
No	42	9	4	38	2	-.005	.128
Yes	40	4	1	42	1	-.016	.164

6 CONCLUSION

The goals of the investigation were to answer the following questions:

1) Does air pollution from vehicular traffic have an effect on human health?
2) Do the current air quality guidelines protect the population from adverse health effects?
3) Are some population subgroups more susceptible to the undesirable effects of air pollution than others?

The approach to answering these questions differed in the different design levels in this investigation.

Does air pollution from vehicular traffic have an adverse effect on human health? This question can actually be rephrased as two questions. 1) Does air pollution cause more people to be bothered by health symptoms? 2) Of those who are bothered by a health symptom, does air pollution aggravate the severity of being bothered?

Air pollution from vehicular traffic does have adverse effects on human health. This was evident in the cross-sectional study in Level 1. Air pollution was significantly related to having the disease chronic bronchitis. The influence of air pollution was as strong in non-smokers as smoking was for smokers.

Health is defined not only as the absence of disease, but reflects well being and thus the absence of unpleasant symptoms. It was therefore interesting to note that in Level 1, exposure to traffic pollution was significantly related to having such symptoms as headache, coughing, eye irritation, fatigue and being depressed.

In Level 2, lung function, CO in blood (COHb) and lead in blood were measured for each individual (ca. 150 individuals). There was a significant reduction of 10 to 16% in vital capacity, forced vital capacity and forced expiratory volume with increased concentrations of COHb in non-smokers. The correlation of lung function was not confirmed against CO exposure although a correlation between COHb and CO exposure was. The results should therefore be interpreted with caution.

Level 2 also correlated hourly fluctuations of health symptoms with hourly estimated exposure to NO_2. In Level 2 we are examining if air pollution influences the severity of the symptom since regression coefficients are calculated for those individuals who have reported having the symptom. Health symptom reporting was significantly related to air pollution exposure

for fatigue, sneezing, sore throat, tight chest and being annoyed by noise and smell.

These three studies confirm that using a broad definition of health, air pollution from vehicular traffic does have an adverse effect on human health.

Do the current air quality guidelines protect the population from health effects? Only Level 2 was designed to answer this question. As shown in Figure 36, the current hourly ambient air guidelines of 200 $\mu g/m^3$ do not seem to protect the population from health effects when, for example, the risk of sneezing or having a tight chest is 80% higher at exposures equal to the guideline level.

Are some population subgroups more susceptible than others to the effects of air pollution? There are two ways of answering the question. Some population subgroups are more susceptible through exposure, others due to biological susceptibility.

It was evident that population subgroups differed in their exposure through lifestyle factors. For example, children, being more outdoors, were the highest exposed during the daytime. The elderly, who sleep more often with windows open, have the highest nighttime exposure.

Biological susceptibility may operate in two ways. Air pollution may for example, cause a group of individuals that are biologically susceptible to have a health symptom. On the other hand, air pollution may cause a worsening of the symptom, among a biologically sensitive subset of those that have the symptom.

There is evidence in these data for both of these theories. Level 1 indicates that air pollution causes a biologically sensitive subset of the population to have the symptom. For example, younger women are more prone to headaches than men. Level 2 indicates that a percentage of those that report a health

symptom, are biologically sensitive to air pollution, such that their symptoms are worsened. (See Table 8).

What causes biological sensitivity is not yet clear, but it is not the same group of individuals that have each of the different health symptoms. Nor is it the same group among those reporting health symptoms that react to air pollution. Therefore, by summing all the health complaints, 40% of the population showed a relationship between the increased severity of at least one health symptom and air pollution.

The results presented in this report are those that were found in a random population living in the Vålerenga area in Oslo, Norway. This population includes both healthy and sick, old and young, smokers and non-smokers etc. Work remains to try to describe what factors contribute to biological sensitivity to air pollution. The next phase in data analysis will be an attempt to describe some of these factors.

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APPENDIX 1

Characteristics of the study population
as described by % of time
in different microenvironments

The subpopulation used in
the cohort study

The information is collected from
a daily diary

Table 1-1: Per cent of time spent in different locations, by population subgroups in the subpopulation of Vålerenga.

	n. of participants	% hours at home	% hours at work/ school/ day-care center	% hours other places	% whole hours travelling
Population group					
Children and teenagers	12	61.5	15.7	19.5	3.3
Adult women	77	65.0	18.7	13.7	2.7
Adult men	48	64.8	20.0	12.1	3.1
Elderly	20	85.3	2.5	8.7	3.5

Table 1-2: Per cent of time spent in different microenvironments, by population subgroups in the subpopulation of Vålerenga.

	n. of participants	% hours indoors	% hours indoors with open window	% hours outdoors
Population group				
Children and teenagers	12	84.5	6.8	5.0
Adult women	77	70.9	24.3	2.1
Adult men	48	72.2	19.0	5.7
Elderly	20	61.1	32.6	2.7

Table 1-3: Per cent of time spent at different activity levels, by population subgroups in the subpopulation of Vålerenga.

	n. of participants	% hours sleeping	% hours daily activities	% hours exercising
Population group				
Children and teenagers	12	41.9	55.4	2.7
Adult women	77	34.4	63.8	1.9
Adult men	48	33.3	61.1	5.7
Elderly	20	35.6	63.2	1.1

Table 1-4: Per cent of time spent smoking or exposed to passive smoking, by population subgroups in the subpopulation of Vålerenga.

	n. of participants	% hours with smoking	% hours exposed to passive smoking	Total number of cigarettes
Population group				
Children and teenagers	12	0.0	2.4	0.1
Adult women	77	16.4	4.3	75.5
Adult men	48	13.5	3.7	68.8
Elderly	20	9.4	0.3	36.5

Table 1-5: Mean duration of travelling or shopping trips, by population subgroups in the subpopulation of Vålerenga.

	n. of participants	Mean duration of trip (travelling)	Mean duration of trip (shopping)
Population group			
Children and teenagers	12	44.4	11.2
Adult women	77	30.7	19.1
Adult men	48	43.3	13.5
Elderly	20	45.7	16.8

Table 1-6: Mean number of hours travelling or shopping daily, by population subgroups in the subpopulation of Vålerenga.

	n. of participants	Mean number of hours travelling daily	Total number of hours travelled	Mean number of hours shopping daily	Total number of hours shopping
Population group					
Children and teenagers	12	1.5	22.2	0.2	2.5
Adult women	77	1.9	27.3	0.4	5.9
Adult men	48	1.9	27.2	0.3	4.7
Elderly	20	0.9	12.8	0.3	3.8

Table 1-7: Mean daily minutes spent travelling, by population subgroups in the subpopulation of Vålerenga.

	n. of partici-pants	Mean daily travelling in heavy traffic	Mean daily travelling in medium traffic	Mean daily travelling in light traffic	Mean daily travelling
Population group					
Children and teenagers	12	16.7	27.7	23.2	67.6
Adult women	77	17.3	23.6	21.2	62.2
Adult men	48	20.4	35.3	21.1	76.9
Elderly	20	12.4	28.8	15.3	56.4

Table 1-8: Mean daily minutes spent shopping, by population subgroups in the subpopulation of Vålerenga.

	n. of partici-pants	Mean daily shopping in heavy traffic	Mean daily shopping in medium traffic	Mean daily shopping in light traffic	Mean daily shopping
Population group					
Children and teenagers	12	4.5	6.0	1.3	11.8
Adult women	77	6.8	9.8	4.9	21.5
Adult men	48	3.5	7.8	3.1	14.4
Elderly	20	6.0	5.8	5.4	17.3

Table 1-9: Smoking habits (smokers only), by population subgroups in the subpopulation of Vålerenga.

	n. of partici-pants	Mean number of cigarettes daily	Mean number of hours with smoking daily
Population group			
Children and teenagers	1	0.1	0.1
Adult women	44	9.2	6.9
Adult men	22	10.1	7.1
Elderly	8	6.2	5.7

APPENDIX 2

Estimated exposure to carbon monoxide
and nitrogen dioxide as a function
of microenvironments, day and week

Exposure in different micro-environments.

	Home	Workplace	School/day -care centre	Other places	Travelling or shopping within hour
Carbon monoxide (mg/m^3)					
Mean	2.2	2.1	1.5	1.5	3.9
Median	1.8	1.4	1.1	0.6	3.1
Std. dev.	1.7	2.3	1.8	2.3	3.1
Max.	25.9	24.2	15.2	21.7	28.3
n of hours	34075	5667	1255	2670	5500
Nitrogen dioxide ($\mu g/m^3$)					
Mean	21.5	33.0	26.7	23.9	55.9
Median	15.0	28.0	21.0	20.0	48.0
Std. dev.	24.8	25.1	23.0	21.0	39.0
Max.	331.0	234.0	246.0	227.0	332.0
n of hours	34075	5667	1255	2670	5500

Exposure in different micro-environments.

	Indoors	Indoors with open window	Outdoors
Carbon monoxide (mg/m^3)			
Mean	2.1	2.5	2.8
Median	1.4	2.1	2.3
Std. dev.	2.1	1.5	2.7
Max.	24.2	23.4	25.9
n of hours	34809	11381	1403
Nitrogen dioxide (ug/m^3)			
Mean	23.8	27.5	51.5
Median	19.0	16.0	46.0
Std. dev.	24.0	32.1	35.3
Max.	257.0	331.0	292.0
n of hours	34809	11381	1403

Exposure by day and week

	Week			
	26.10 - 01.11	02.11 - 08.11	09.11 - 15.11	16.11 - 22.11
Weekday				
Monday				
Carbon monoxide (mg/m^3)				
Mean	2.3	2.2	2.0	1.5
Median	2.0	2.0	1.5	1.4
Std. dev.	1.8	1.5	1.4	.9
Max.	18.3	15.2	19.9	6.7
n of hours	392	1998	3280	1485
Tuesday				
Carbon monoxide (mg/m^3)				
Mean	1.5	2.6	2.4	1.9
Median	1.4	2.0	2.0	1.7
Std. dev.	.7	1.9	1.7	1.3
Max.	4.1	15.2	19.2	17.0
n of hours	801	1966	2924	1487
Wednesday				
Carbon monoxide (mg/m^3)				
Mean	1.5	2.3	1.7	4.0
Median	1.5	2.0	1.5	2.6
Std. dev.	.7	1.5	1.2	3.6
Max.	3.9	11.8	15.0	23.0
n of hours	1396	2165	2457	1416
Thursday				
Carbon monoxide (mg/m^3)				
Mean	1.9	2.3	1.2	1.8
Median	1.6	2.0	1.2	1.6
Std. dev.	1.1	1.6	.6	.9
Max.	7.6	18.9	3.2	8.3
n of hours	1864	3143	1880	593
Friday				
Carbon monoxide (mg/m^3)				
Mean	1.8	4.7	1.5	.
Median	1.6	3.1	1.4	.
Std. dev.	1.1	4.3	1.1	.
Max.	9.9	28.3	15.6	.
n of hours	2109	3394	1475	
Saturday				
Carbon monoxide (mg/m^3)				
Mean	1.7	3.7	1.6	.
Median	1.6	2.5	1.5	.
Std. dev.	.9	2.9	.8	.
Max.	6.1	22.0	8.5	.
n of hours	2020	3135	1370	
Sunday				
Carbon monoxide (mg/m^3)				
Mean	2.4	2.0	1.7	.
Median	2.0	1.7	1.6	.
Std. dev.	1.8	1.2	.9	.
Max.	25.9	11.7	9.1	.
n of hours	1952	3130	1335	

Exposure by day and week

	Week			
	26.10 - 01.11	02.11 - 08.11	09.11 - 15.11	16.11 - 22.11
Weekday				
Monday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	30.0	23.7	30.0	12.9
Median	19.0	18.0	28.0	8.0
Std. dev.	33.6	23.5	29.7	15.0
Max.	176.0	177.0	197.0	78.0
n of hours	392	1998	3280	1485
Tuesday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	14.1	24.1	43.4	18.1
Median	6.0	16.0	38.0	13.0
Std. dev.	18.0	27.9	36.6	19.4
Max.	81.0	246.0	329.0	237.0
n of hours	801	1966	2924	1487
Wednesday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	15.7	30.6	24.3	45.8
Median	9.0	26.0	21.0	32.0
Std. dev.	17.5	19.8	23.0	40.7
Max.	82.0	158.0	216.0	331.0
n of hours	1396	2165	2457	1416
Thursday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	19.5	23.0	6.1	23.6
Median	16.0	18.0	4.0	19.0
Std. dev.	20.7	21.7	7.3	24.9
Max.	101.0	156.0	44.0	233.0
n of hours	1864	3143	1880	593
Friday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	19.3	49.9	20.2	.
Median	16.0	39.0	15.0	.
Std. dev.	20.7	44.2	23.2	.
Max.	147.0	332.0	174.0	.
n of hours	2109	3394	1475	
Saturday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	19.1	42.3	17.4	.
Median	15.0	38.0	16.0	.
Std. dev.	16.4	31.9	14.9	.
Max.	126.0	232.0	86.0	.
n of hours	2020	3135	1370	
Sunday				
Nitrogen dioxide ($\mu g/m^3$)				
Mean	28.4	22.7	21.4	.
Median	22.0	18.0	19.0	.
Std. dev.	26.0	21.7	18.2	.
Max.	263.0	173.0	129.0	.
n of hours	1952	3130	1335	

Mean CO- and NO_2-exposure, by start day

	n. of participants	Mean CO exposure (mg/m^3)	Mean NO_2 exposure ($\mu g/m^3$)
Startday			
26.10.	17	2.2	26.4
27.10.	19	2.1	23.9
28.10.	26	2.2	24.9
29.10.	21	2.5	28.9
30.10.	10	2.4	28.6
01.11.	1	1.8	25.8
04.11.	9	2.2	26.5
05.11.	38	2.3	26.7
06.11.	16	2.5	30.4

Mean CO-exposure by population group

	n. of participants	Mean CO exposure (mg/m^3)	Std. dev. CO exposure (mg/m^3)	Maximum hourly CO exposure (mg/m^3)
Population group				
Children and teenagers	12	2.3	2.0	17.3
Adult women	77	2.3	1.9	15.6
Adult men	48	2.3	2.0	15.9
Elderly	20	2.4	1.8	14.9

Mean NO2-exposure by population group

	n. of participants ts	Mean NO_2 exposure ($\mu g/m^3$)	Std. dev. NO_2 exposure ($\mu g/m^3$)	Maximum hourly NO_2 exposure ($\mu g/m^3$)
Population group				
Children and teenagers	12	26.7	26.0	188.3
Adult women	77	26.3	26.8	174.1
Adult men	48	28.2	26.4	169.4
Elderly	20	25.4	26.2	163.0

Smoking habits (all participants), by population groups

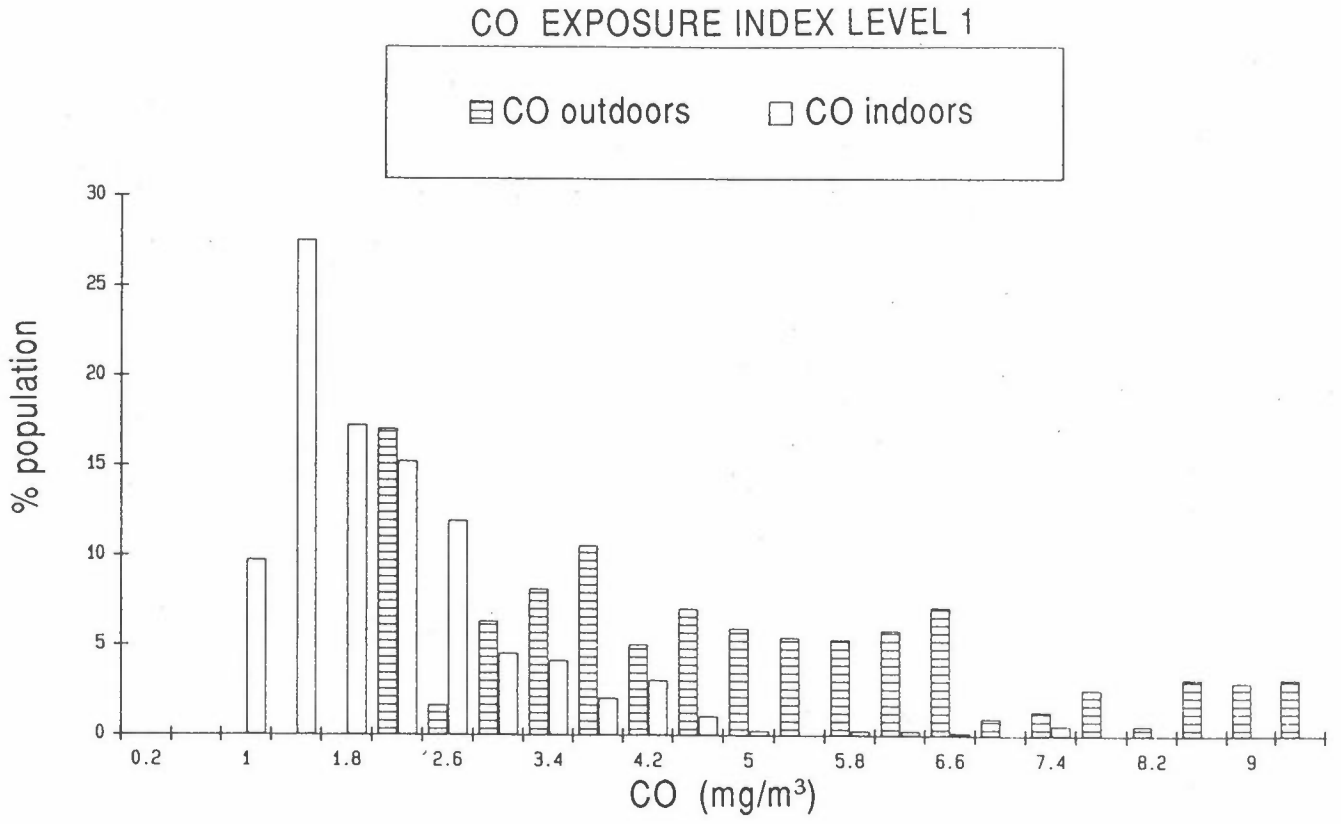
	n. of participants	Mean number of cigarettes daily	Mean number of hours with smoking daily
Population group			
Children and teenagers	12	.0	.0
Adult women	77	5.3	3.9
Adult men	48	4.6	3.3
Elderly	20	2.5	2.3

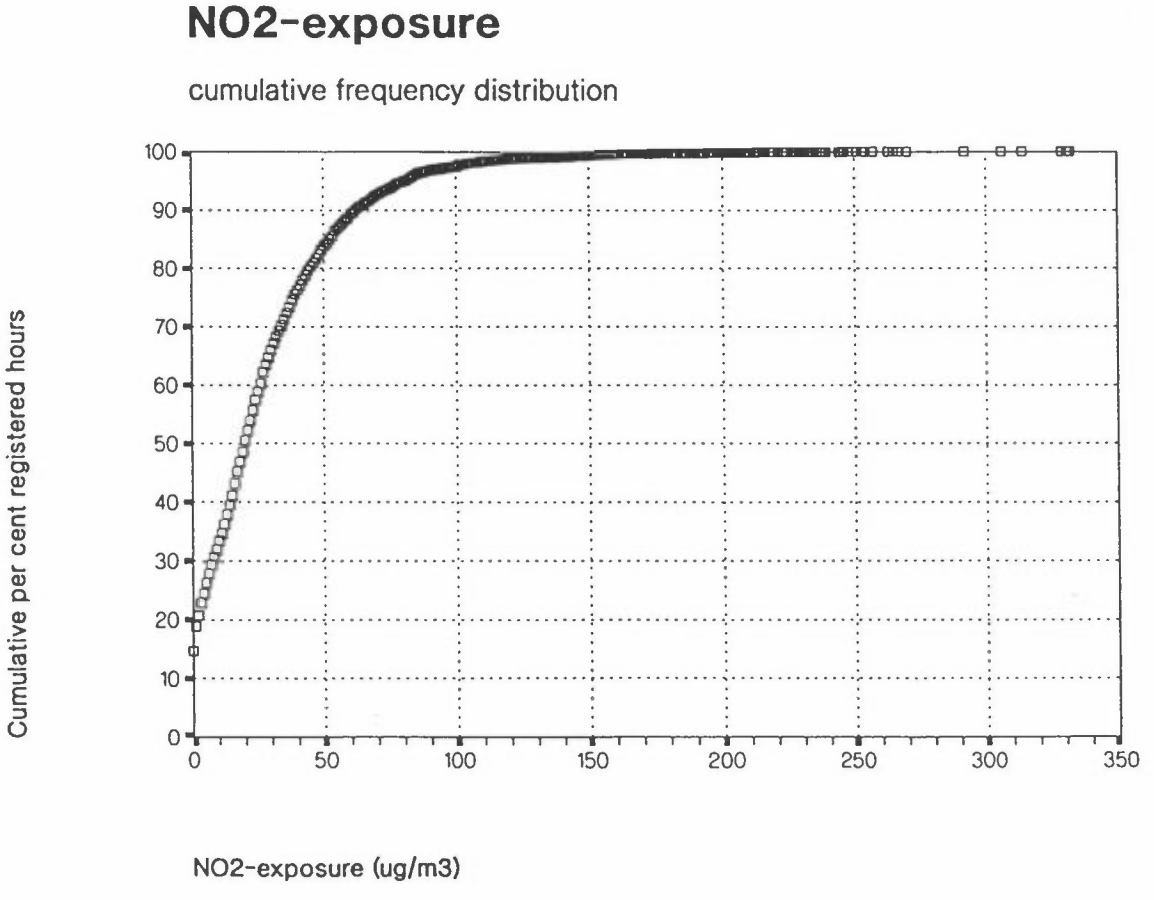
Smoking habits (smokers only), by population groups

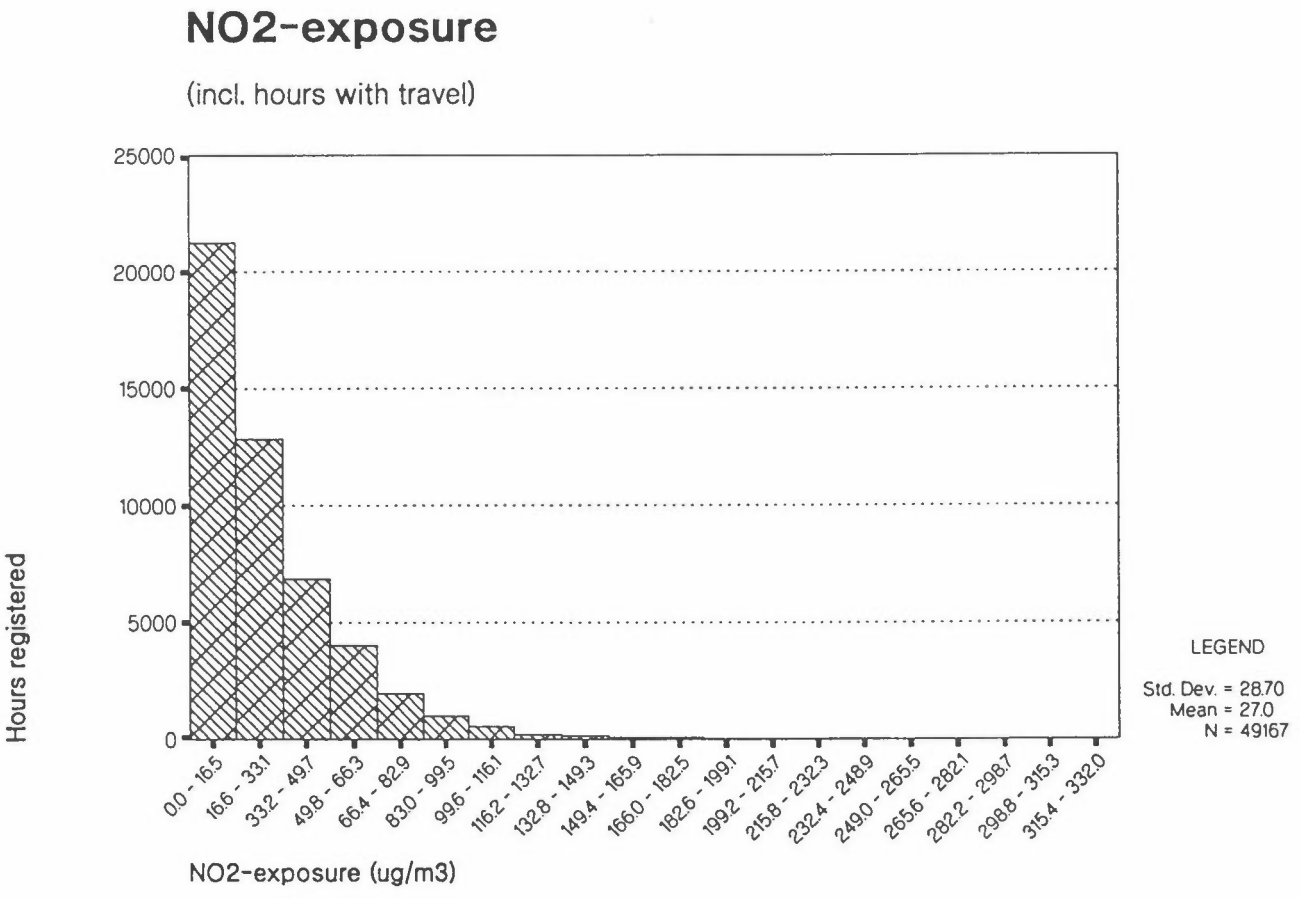
	n. of participants	Mean number of cigarettes daily	Mean number of hours with smoking daily
Population group			
Children and teenagers	1	.1	.1
Adult women	44	9.2	6.9
Adult men	22	10.1	7.1
Elderly	8	6.2	5.7

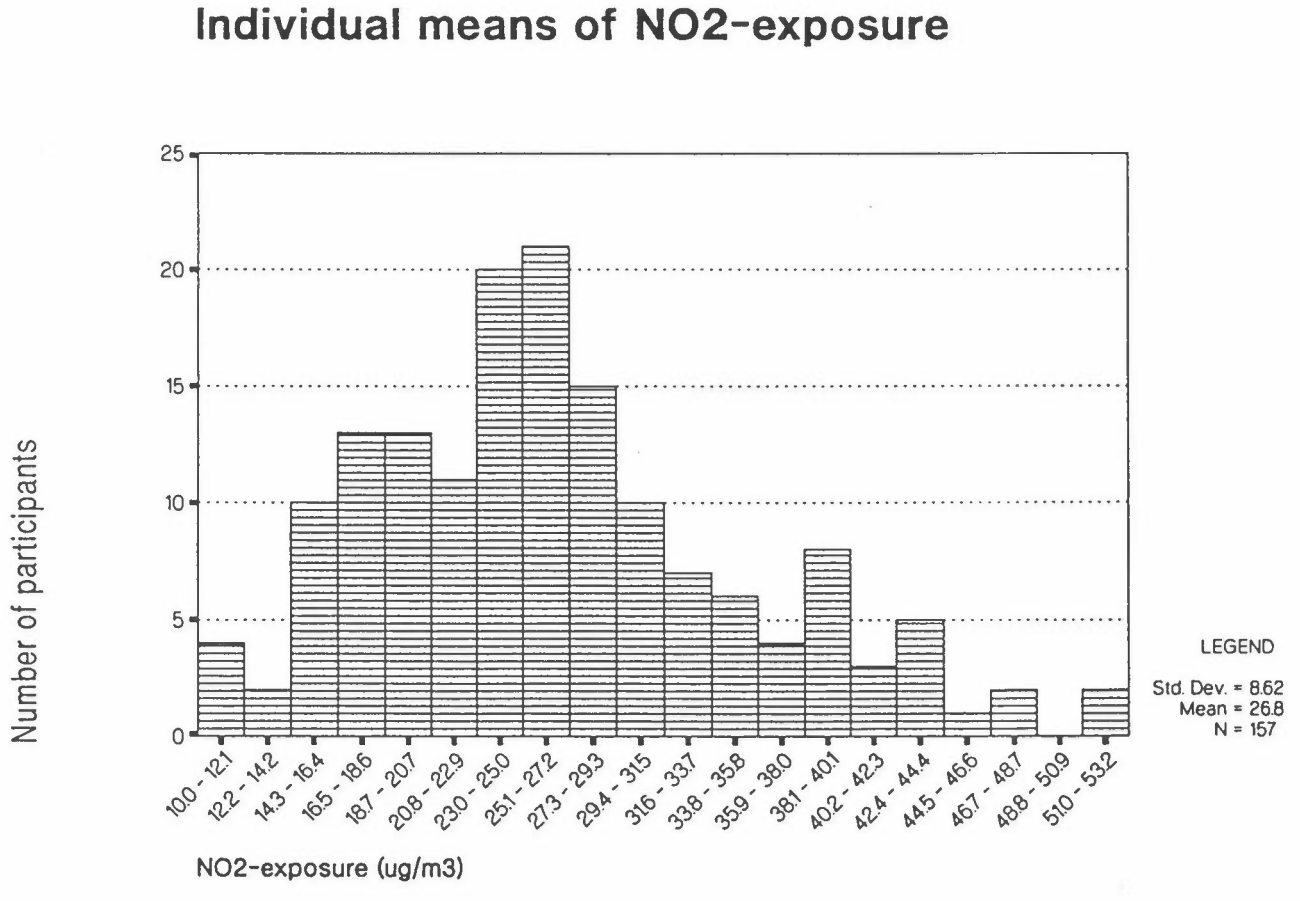
APPENDIX 3

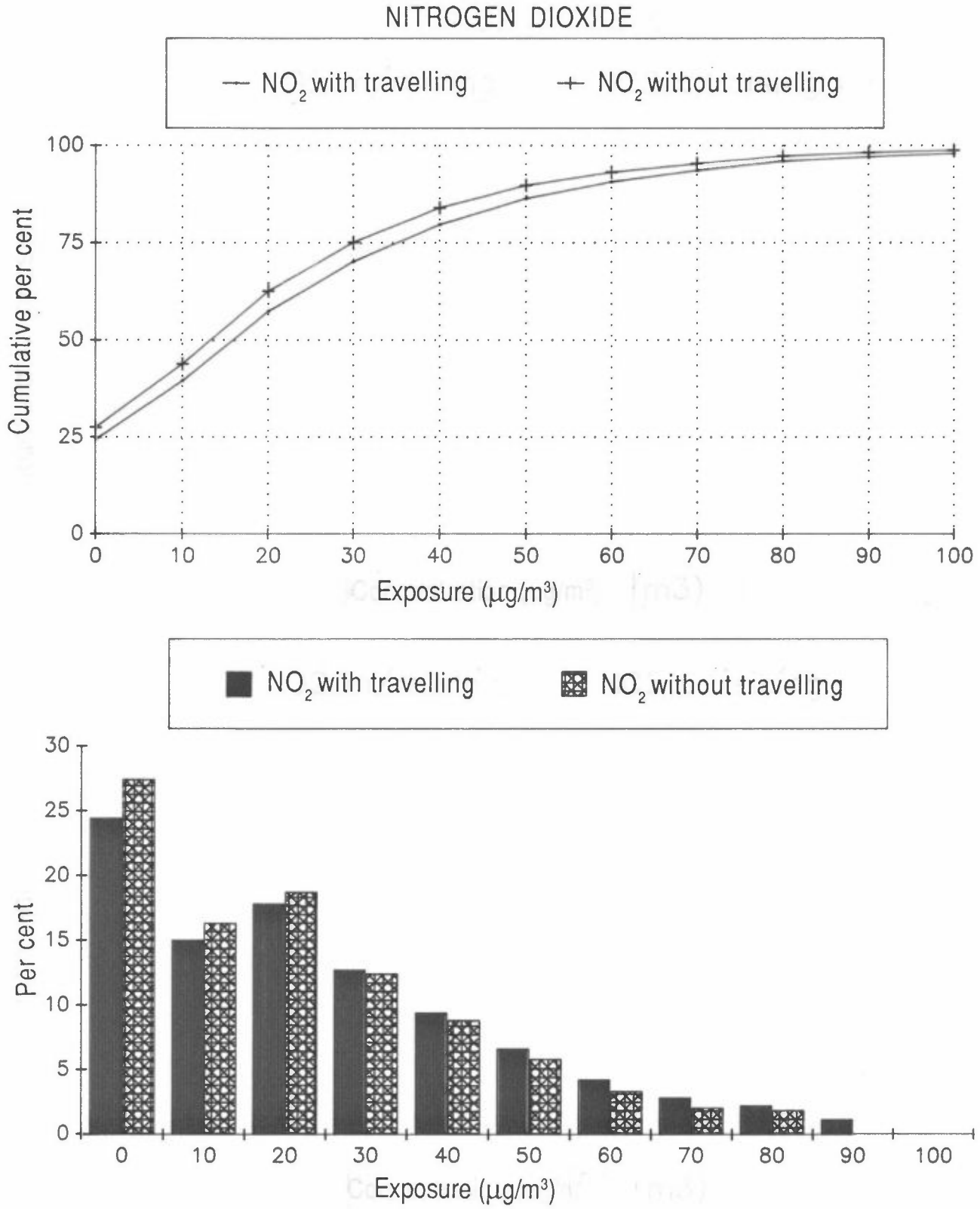
Frequency distributions of estimated exposure in Level 2 unless otherwise indicated in the title of the figure


CO EXPOSURE INDEX LEVEL 1
CO outdoors
CO indoors
% population
30
25
20
15
10
5
0
0.2
1
1.8
2.6
3.4
4.2
5
5.8
6.6
7.4
8.2
9
CO (mg/m³)


NO2-exposure
cumulative frequency distribution
Cumulative per cent registered hours
100
90
80
70
60
50
40
30
20
10
0
0
50
100
150
200
250
300
350
NO2-exposure (ug/m3)

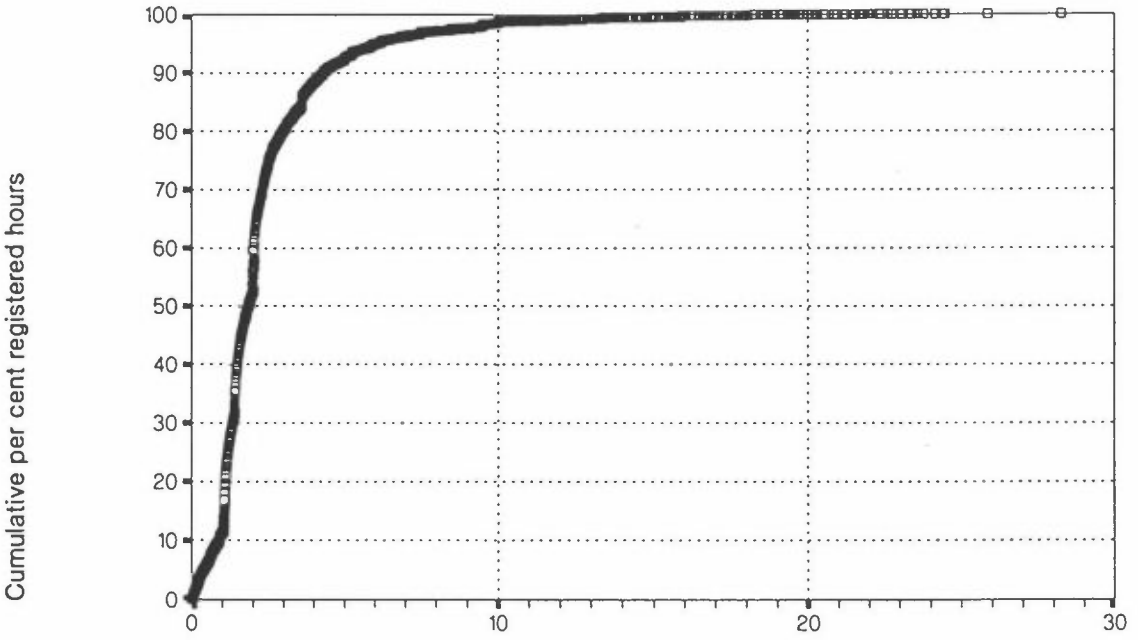

NO2-exposure
(incl. hours with travel)
Hours registered
25000
20000
15000
10000
5000
0
0.0 - 16.5
16.6 - 33.1
33.2 - 49.7
49.8 - 66.3
66.4 - 82.9
83.0 - 99.5
99.6 - 116.1
116.2 - 132.7
132.8 - 149.3
149.4 - 165.9
166.0 - 182.5
182.6 - 199.1
199.2 - 215.7
215.8 - 232.3
232.4 - 248.9
249.0 - 265.5
265.6 - 282.1
282.2 - 298.7
298.8 - 315.3
315.4 - 332.0
NO2-exposure (ug/m3)
LEGEND
Std. Dev. = 28.70
Mean = 27.0
N = 49167


Individual means of NO2-exposure
Number of participants
25
20
15
10
5
0
10.0 - 12.1
12.2 - 14.2
14.3 - 16.4
16.5 - 18.6
18.7 - 20.7
20.8 - 22.9
23.0 - 25.0
25.1 - 27.2
27.3 - 29.3
29.4 - 31.5
31.6 - 33.7
33.8 - 35.8
35.9 - 38.0
38.1 - 40.1
40.2 - 42.3
42.4 - 44.4
44.5 - 46.6
46.7 - 48.7
48.8 - 50.9
51.0 - 53.2
NO2-exposure (ug/m3)
LEGEND
Std. Dev. = 8.62
Mean = 26.8
N = 157


NITROGEN DIOXIDE
NO2 with travelling
NO2 without travelling
Cumulative per cent
100
75
50
25
0
0
10
20
30
40
50
60
70
80
90
100
Exposure (µg/m3)
NO2 with travelling
NO2 without travelling
Per cent
30
25
20
15
10
5
0
0
10
20
30
40
50
60
70
80
90
100
Exposure (µg/m3)

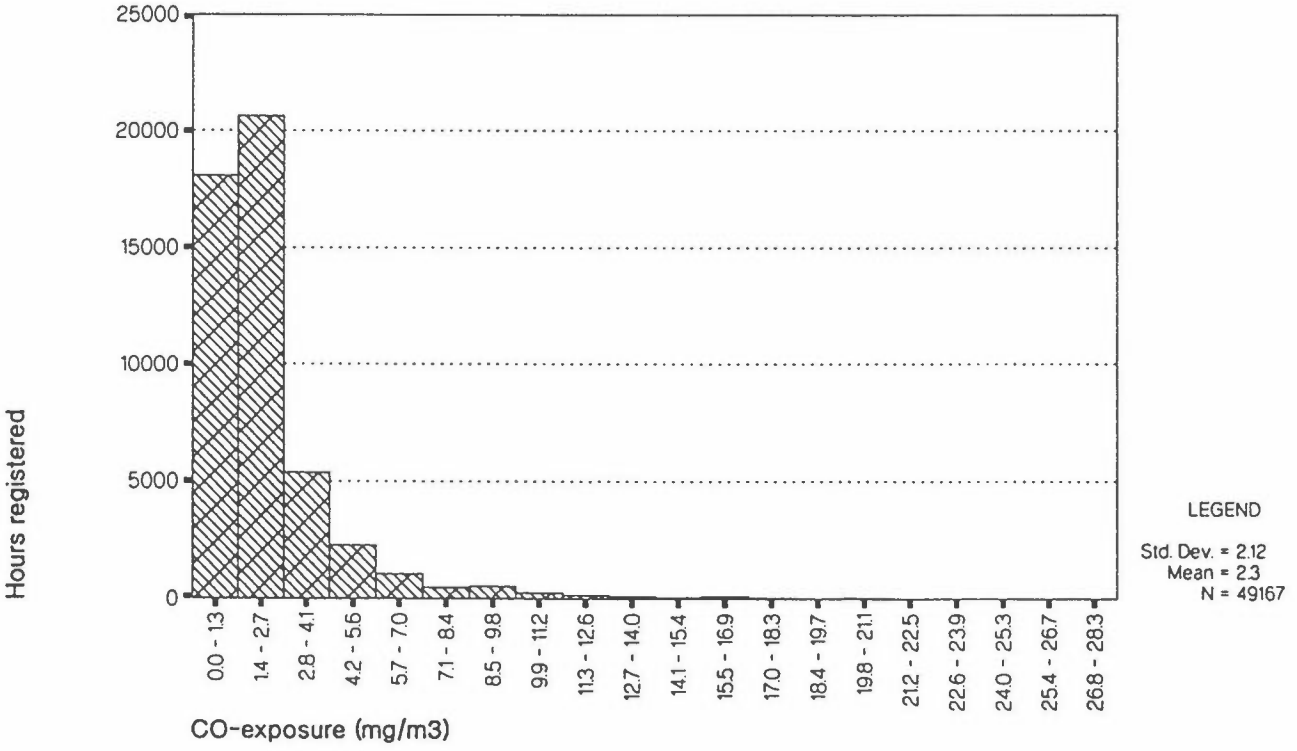
CO-exposure

cumulative frequency distribution

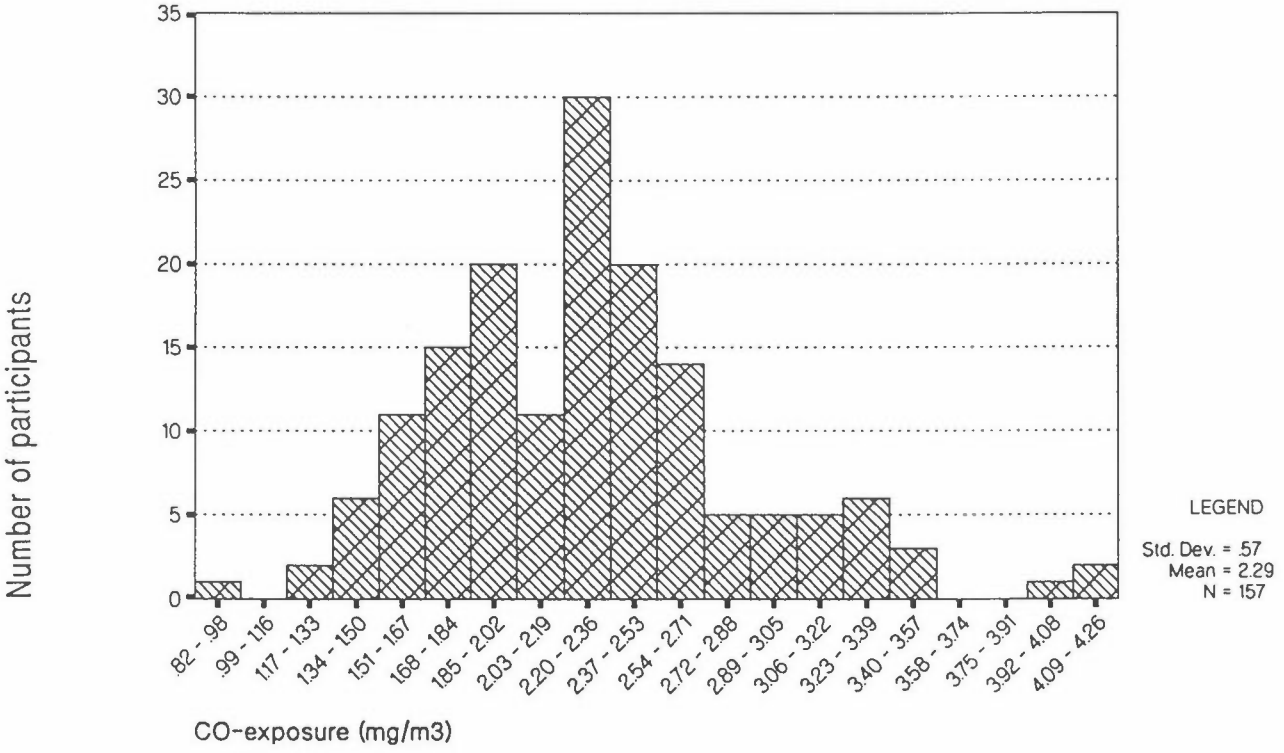


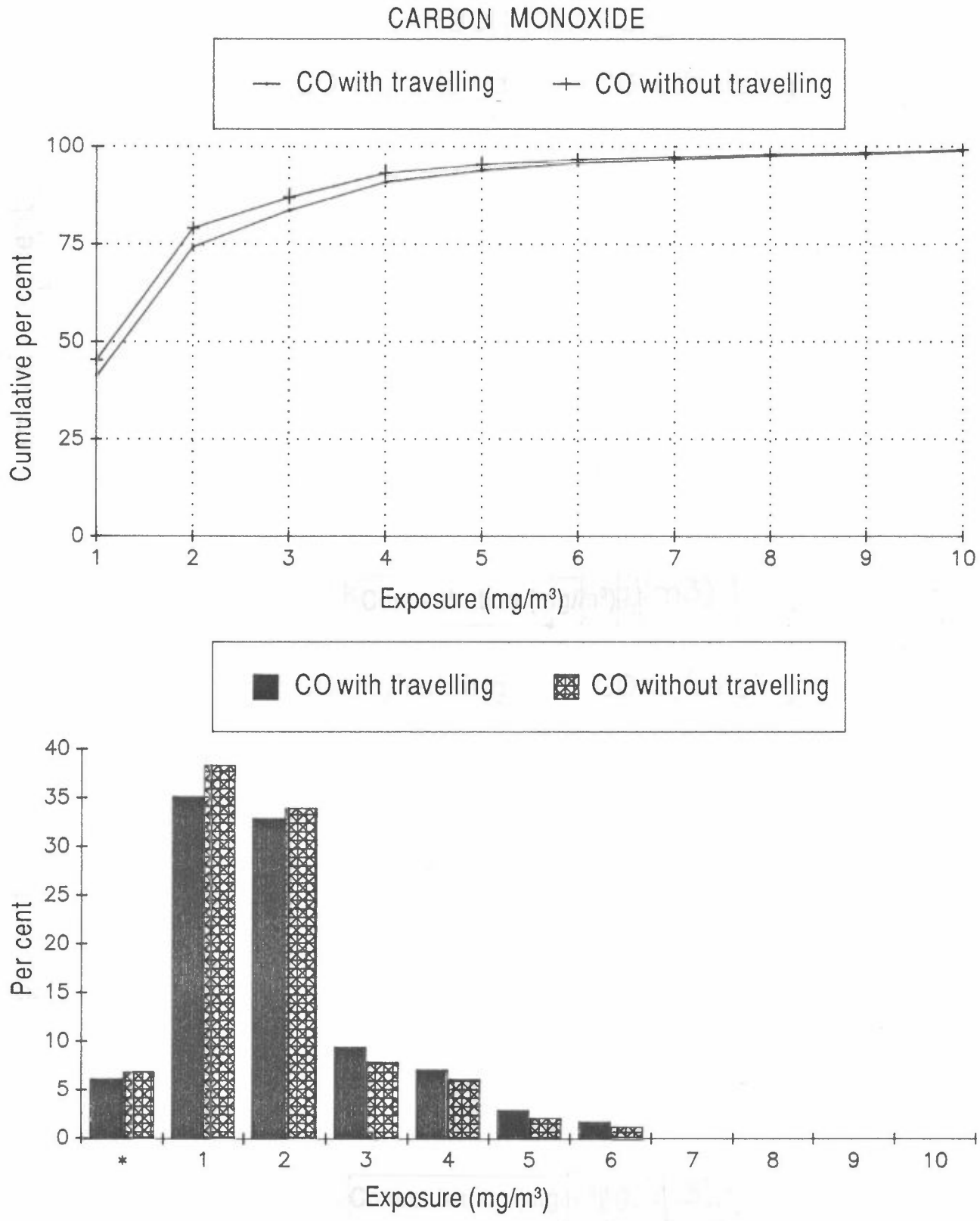
CO-exposure

(incl. hours with travel)



Individual means of CO-exposure




CARBON MONOXIDE
CO with travelling
CO without travelling
Cumulative per cent
100
75
50
25
0
1
2
3
4
5
6
7
8
9
10
Exposure (mg/m³)
CO with travelling
CO without travelling
Per cent
40
35
30
25
20
15
10
5
0
*
1
2
3
4
5
6
7
8
9
10
Exposure (mg/m³)

APPENDIX 4

Regression coefficients
and
standard deviations
for the logistic regressions
of the health symptoms
and
a set of explanatory variables,
separately for "often" and "sometimes"
bothered of each symptom

Regression coefficient (b) and standard deviation (s.d) for a set of independent variables in each of 16 health parameters, in those individuals that are sometimes bothered (S) and often bothered (O). Numbers under symptom name indicate size of the group, with those bothered in parentheses.

Dependent Parameter	INDEPENDENT VARIABLES						
Health Parameter	CO indoors	Age	Sex	Smoke	Educ.	Marital status	Intercept
Headache							
616(349)							
S b	.146	-.013	.402	.030	.017	-.012	5.312
s.d.	.107	.003	.091	.047	.067	.122	.265
410(155)							
O b	.292	-.011	.825	.069	-.255	-.076	5.008
s.d	.152	.003	.121	.060	.087	.145	.297
Nausea							
551(144)							
S b	.127	-.006	.162	.046	-.125	.141	4.710
s.d.	.122	.003	.104	.054	.076	.132	.297
473(45)							
O b	-.001	.003	.566	.097	-.177	-.133	3.586
s.d.	.205	.005	.191	.091	.129	.213	.477
Coughing							
584(327)							
S b	.194	-.000	.009	.147	.146	-.117	4.581
s.d.	.110	.003	.088	.047	.066	.117	.254
441(169)							
O b	.344	-.008	-.042	.213	-.084	.273	4.800
s.d.	.129	.003	.108	.055	.077	.138	.284
Neck pains							
529(296)							
S b	.268	-.003	.340	-.022	.053	-.019	4.820
s.d.	.117	.003	.092	.049	.067	.132	.258
497(266)							
O b	.292	.003	.397	.060	-.096	.204	4.564
s.d.	.123	.003	.099	.051	.072	.120	.273
Heart palpitation							
551(134)							
S b	.065	.001	.242	-.005	-.154	.161	4.476
s.d.	.128	.003	.107	.056	.079	.130	.292
473(62)							
O b	.142	.010	.052	-.053	-.308	.288	3.884
s.d.	.182	.004	.160	.081	.124	.165	.453
Indigestion							
532(115)							
S b	.152	.003	.171	-.017	.106	.059	3.823
s.d.	.134	.003	.111	.058	.081	.143	.312
493(71)							
O b	.141	.005	.113	.082	-.084	.221	3.682
s.d.	.161	.004	.141	.072	.103	.158	.402
Fatigue							
581(306)							
S b	.256	-.006	.157	-.073	.068	.214	4.964
s.d.	.110	.003	.087	.046	.065	.121	.247
444(164)							
O b	.296	-.004	.383	.086	-.021	.039	4.430
s.d.	.130	.003	.110	.055	.077	.131	.294
High blood pressure							
510(60)							
S b	.049	.020	-.026	-.063	.115	.131	2.734
s.d.	.198	.005	.157	.083	.115	.177	.460
512(72)							
O b	.042	.024	.151	.055	.012	.052	2.594
s.d.	.173	.004	.149	.075	.106	.154	.448

	CO indoors	Age	Sex	Smoke	Educ.	Mar.st.	Intercept
Dizziness							
558(146)							
S b	-.154	0.011	.337	.043	-.085	.097	3.912
s.d.	.134	.003	.109	.057	.080	.127	.309
466(75)							
0 b	.154	.013	.274	-.015	-.124	.256	3.385
s.d.	.167	.004	.149	.074	.107	.156	.421
Itching							
529(139)							
S b	-.056	-.004	.203	.006	.082	-.005	4.463
s.d.	.130	.003	.103	.054	.074	.140	.273
496(101)							
0 b	.188	-.007	.168	.154	.022	.196	4.126
s.d.	.138	.004	.121	.064	.089	.155	.354
Nervous							
560(212)							
S b	.007	-.002	.183	.118	-.109	.197	4.770
s.d.	.114	.003	.092	.048	.067	.123	.252
465(100)							
0 b	.285	.005	-.068	.165	-.321	.395	4.225
s.d.	.148	.004	.130	.068	.100	.139	.368
Depressed							
621(273)							
S b	.125	-.002	.329	.065	-.029	.205	4.644
s.d.	.106	.003	.087	.045	.063	.113	.240
404(66)							
0 b	.373	.003	.132	.200	-.358	.342	3.991
s.d.	.171	.004	.153	.082	.120	.165	.435
Problems sleeping							
543(158)							
S b	-.049	.003	.273	.067	-.184	.228	4.514
s.d.	.125	.003	.103	.054	.075	.123	.284
482(115)							
0 b	.296	.013	.176	-.013	-.209	.160	3.773
s.d.	.145	.003	.125	.063	.092	.140	.355
Eye irritation							
551(142)							
S b	.120	.002	.008	-.004	.087	.072	4.088
s.d.	.124	.003	.102	.054	.074	.131	.282
473(83)							
0 b	.391	.006	.056	-.021	-.010	.152	3.598
s.d.	.152	.004	.133	.067	.099	.157	.392
Cold/influenza							
680(375)							
S b	.081	-.009	.035	.047	.082	.054	5.194
s.d.	.101	.002	.081	.043	.060	.109	.227
345(63)							
0 b	.245	-.017	.045	-.003	-.127	.220	5.036
s.d.	.170	.005	.151	.077	.110	.197	.406
Problems breathing							
529(89)							
S b	.016	.009	.245	-.043	-.100	.068	3.835
s.d.	.157	.004	.128	.066	.092	.147	.346
493(47)							
0 b	.367	.005	-.359	-.027	-.307	.123	4.096
s.d.	.184	.005	.170	.086	.134	.192	.481

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TITTEL
The Health Effects of Traffic Pollution as measured in the Vålerenga area of Oslo.
A summary report.

PROSJEKTLEDER
J. Clench-Aas

NILU PROSJEKT NR.
O-8638

FORFATTER(E)
J. Clench-Aas, K. Myhre, F. Levy, S. Larssen, C.C. Christensen, A. Bartonova, I.L. Neslein, M.J. Aarnes, Y. Thomassen

TILGJENGELIGHET *
A

OPPDRAGSGIVERS REF.

OPPDRAGSGIVER (NAVN OG ADRESSE)
Forskningsprogram Trafikk og Miljø
Sekretariat. Transport økonomisk institutt

STIKKORD
Traffic pollution | Health effect | Lung function

REFERAT
Rapporten beskriver resultater funnet i en serie av undersøkelser om helse-effekter av trafikkforurensning. Målte parametre inkluderte CO og bly i blod, lungefunksjon og symptomer på helseeffekter. Det er antydning til en negativ effekt av luftforurensning på helse på nivåer lik eller mindre enn nåværende retningslinjer for luftkvalitet.

TITLE

ABSTRACT
This report describes the results of a series of investigations in 3 levels of the health effects of traffic pollution. The parameters measured are COHb, lead in blood, lung function and a set of symptoms of health effects. There seems to be an effect of air pollution on health, at or below suggested air quality guidelines.

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