United States
Environmental Protection
Agency

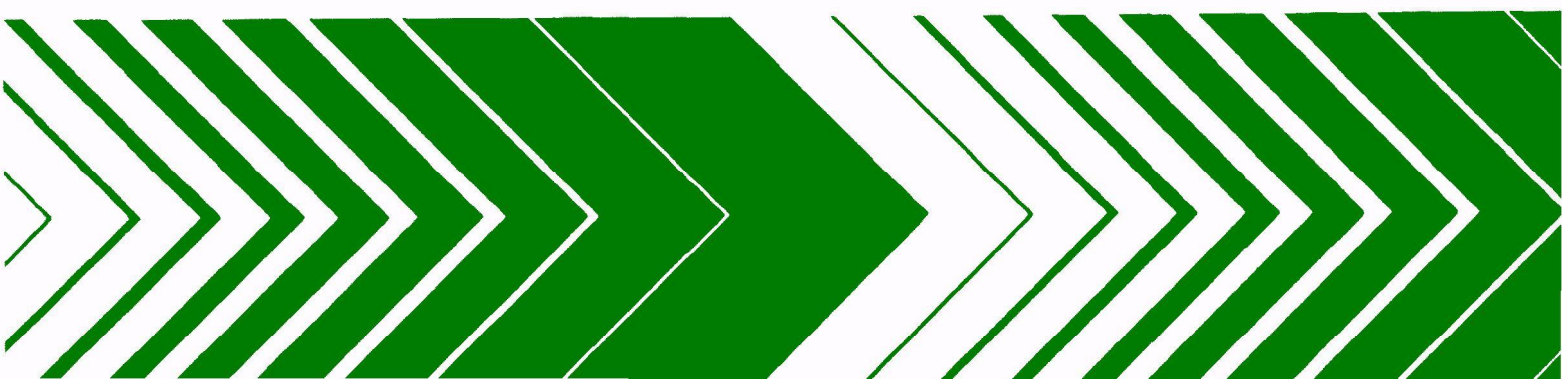
Health Effects Research
Laboratory
Cincinnati OH 45268

EPA-600/1-78-067
November 1978

Research and Development

EPA

Study of Children's Blood-Lead Levels Within Families



RESEARCH REPORTING SERIES

Research reports of the Office of Research and Development, U.S. Environmental Protection Agency, have been grouped into nine series. These nine broad categories were established to facilitate further development and application of environmental technology. Elimination of traditional grouping was consciously planned to foster technology transfer and a maximum interface in related fields. The nine series are:

1. Environmental Health Effects Research
2. Environmental Protection Technology
3. Ecological Research
4. Environmental Monitoring
5. Socioeconomic Environmental Studies
6. Scientific and Technical Assessment Reports (STAR)
7. Interagency Energy-Environment Research and Development
8. "Special" Reports
9. Miscellaneous Reports

This report has been assigned to the ENVIRONMENTAL HEALTH EFFECTS RESEARCH series. This series describes projects and studies relating to the tolerances of man for unhealthful substances or conditions. This work is generally assessed from a medical viewpoint, including physiological or psychological studies. In addition to toxicology and other medical specialities, study areas include biomedical instrumentation and health research techniques utilizing animals — but always with intended application to human health measures.

This document is available to the public through the National Technical Information Service, Springfield, Virginia 22161.

EPA-600/1-78-067
November 1978

STUDY OF CHILDREN'S BLOOD-LEAD LEVELS WITHIN FAMILIES

by

Danica Prpic-Majjic
Institute for Medical Research
and Occupational Health
Zagreb, Yugoslavia

Special Foreign Currency Program
JF-3-570-2

Project Officer

Robert J.M. Horton
Health Effects Research Laboratory
Research Triangle Park, N.C. 27711

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
HEALTH EFFECTS RESEARCH LABORATORY
RESEARCH TRIANGLE PARK, N.C. 27711

DISCLAIMER

This report has been reviewed by the Health Effects Research Laboratory, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the U.S. Environmental Protection Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory participates in the development and revision of air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is primarily responsible for providing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

Some information is available indicating that children respond differently to environmental lead exposure in terms of intake, absorption and blood lead level. However, few studies have been undertaken to compare different age groups in the same setting. In this investigation comparisons are made of lead exposure and its effects on parents and children in selected households in the presence of high and low atmospheric lead exposures.

F. G. Hueter, Ph. D.
Acting Director,
Health Effects Research Laboratory

ABSTRACT

The comparative studies of the biological indices of elevated exposure to lead in children and adults were conducted with the intention of reaching a better understanding of lead absorption in children. Three family groups were examined. Group 1 consisted of families who lived in the vicinity of a lead smelter and whose fathers were occupationally highly exposed to lead. Group 2 consisted of families settled in the same area, but whose fathers had no supplemental occupational exposure to lead. The third was the control group consisting of families who lived in an area with very low exposure and whose fathers were not occupationally exposed to lead. Families were selected with one child under 4 years and, if possible, another child of school age. In the environmental survey air, dustfall lead, household-dust lead, and drinking-water lead were analyzed.

It was found that the population living near a lead smelter, except for the fathers occupationally exposed to lead, had biological findings at the level of a "slightly elevated" exposure. The fathers occupationally exposed to lead could be classified as a group with "excessive" exposure.

Three biological parameters--erythrocyte δ-aminolevulinic dehydratase activity, erythrocyte protoporphyrin, and blood lead--are the most sensitive indices of increased lead absorption, regardless of age or sex. They are good parameters to establish the difference in lead absorption from the environment. On the basis of these parameters the following sequence of lead absorption was established in family members living in an area with elevated lead exposure: fathers > school-age children $\cong$ children up to 4 years > mothers. Children with fathers occupationally exposed to lead had a slight additional lead exposure in comparison with children whose fathers had no supplemental occupational exposure to lead.

The increased lead absorption from the environment in the area investigated near a lead smelter had no effect on hemoglobin decrease in the population.

There are some indications that children absorb and retain through inhalation about twice as much lead as adults.

This report was submitted in fulfillment of a Special Foreign Currency Program (JF-2-570-2) by the Institute for Medical Research and Occupational Health (Zagreb, Yugoslavia) under the sponsorship of the U.S. Environmental Protection Agency. The report covers a period of 2½ years (February 24, 1975 to August 23, 1977).

CONTENTS

FIGURES

FIGURES (continued)

FIGURES (continued)

FIGURES (continued)

FIGURES (continued)

FIGURES (continued)

TABLES

TABLES (continued)

TABLES (continued)

ACKNOWLEDGMENTS

The work presented in this report was performed by the following staff of the Institute for Medical Research and Occupational Health (Zagreb)

Research advisors: M. Fugaš (M.Sc., Chemical Engineering) and A. Markičević (M.D., Specialist in Occupational Health)

Research assistants: V. Karačić (Chemist), E. Keršanc (Biologist), R. Paukovič (M.Sc., Chemical Engineering), J. Pongračić (Chemist), S. Telišman (M.Sc., Chemist), A. Vuković (Chemist), B. Wilder (Physicist)

Technicians: M. Erceg, B. Matijević, M. Milas, A. Širec

Consultants: T. Beritić (M.D., Specialist in Internal Medicine and Occupational Health, professor), M. Šerić, (M.D., Ph.D., Epidemiologist, professor)

From other institutions the following coworkers also participated in the work:

B. Čretnik (M.D., Specialist in Pediatrics), Health Center, Ravne na Koroškem

J. Sušnik (M.D., Specialist in Occupational Health), Health Center, Ravne na Koroškem

Z. Skurić (Ph.D., Chemical Engineering, assistant professor), School of Public Health "Andrija Stampar", Zagreb

F. Valić (Ph.D., Chemical Engineering, Industrial Toxicologist, professor), School of Public Health "Andrija Stampar, Zagreb (as a consultant)

SECTION 1

INTRODUCTION

There is general agreement about the fact that data on lead absorption and toxicity in children are still quite scarce. The nature and extent of lead toxicity cannot be predicted on the basis of information obtained in adults. Comparative studies of the biological changes indicative of elevated exposure to lead in children and adults are therefore very useful. This aspect of lead toxicology is the basis of this report, the results of which have been obtained over a period of 2½ years by the team of workers listed in the acknowledgments.

The report is divided into sections. Sections 2 and 3 contain the main conclusions and recommendations for further studies. Section 4 reports on the results of research concerning biological indices of lead absorption, environmental survey, and residential distance from lead-emitting sources. The results obtained are presented in 38 tables and 73 diagrams. The Reference Section gives a list of publications pertinent to the subject of this report. The three tables in the appendix present the individual biological indices of lead absorption.

The situation in regard to lead exposure is somewhat unusual in Yugoslavia, where this study was done. In the smaller communities it has long been the custom that most household containers are made by local potters. These are usually lead glazed and fired at low temperatures. Acid foods and beverages can leach lead from such glazes, particularly during prolonged contact. The principal beverage is wine, which is somewhat acid. Persistent educational efforts by health authorities have reduced this problem considerably, but it is still present in rural areas in some parts of the country. This causes blood-lead levels in rural areas without industrial exposure to be higher than in such areas in other parts of Europe.

SECTION 2

CONCLUSIONS

The results of simultaneous studies of children and adults in three family groups comparable in their socioeconomic status, but differing in lead exposure, have shown that three biological parameters--erythrocyte δ-aminolevulinic dehydratase activity, erythrocyte protoporphyrin, and blood lead--were the most sensitive indices of increased lead absorption, regardless of age or sex. They are good parameters to establish the differences in lead absorption from the environment.

Families who lived in the vicinity of a lead smelter showed the following sequence of lead absoprtion: fathers > school-age children ≅ children up to 4 years > mothers.

Children from lead-exposed areas with fathers occupationally exposed to lead showed a slight additional lead exposure in comparison with children settled in the same area, but whose fathers had no supplemental occupational exposure to lead.

The increased lead absorption from the environment in the investigated area near a lead smelter had no effect on Hb decrease in the population.

When the four environmental media in the exposed and in the control area were compared, the lead content in the specimens examined from the exposed area had decreased according to the following sequence: air > dustfall > household dust > water.

The estimated air-lead absorption expressed on the basis of body weight was about twice as high in children as in adults. This finding is very important in the assessment of the permissible levels of lead exposure in children living near a lead smelter.

Erythrocyte protoporphyrin is the best biological index in the evaluation of body-response normalization with regard to increasing residential distance from the lead-emitting source.

Relationship analysis between biological parameters indicate that blood lead is not the best parameter to which other parameters should be related.

SECTION 3

RECOMMENDATIONS

In children with slightly elevated lead exposure from the environment, the most important recommendation is to control δ-aminolevulinic acid dehydratase activity, erythrocyte protoporphyrin, and blood-lead concentrations. The other biological indices of increased lead absorption are less significant Erythrocyte protoporphyrin concentration seems to be the most representative indicator of long-term lead absorption, while blood lead and to a lesser extent erythrocyte δ-aminolevulinic acid dehydratase activity reflect more actual exposure and day-to-day variability.

In the assessment of permissible levels of lead exposure from the environment, special care should be devoted to children because they absorb and retain a larger portion of inhaled and dietary lead than adults.

In the vicinity of a lead smelter where the combined exposure to lead and other metals accompanying lead ores takes place, effects of some other metals on hemoglobin synthesis could not be excluded. There were some indications that simultaneous exposure to other metals along with lead could have a moderating effect against increased lead absorption, but supporting data is needed. Therefore a further study which would take into account the simultaneous absorption of lead, zinc, iron, and copper is recommended.

SECTION 4

STUDY OF CHILDREN'S BLOOD-LEAD LEVELS WITHIN FAMILIES

The nature and extent of lead toxicity in children cannot be predicted on the basis of information on adults, because "the child is not just a little man"; the differences are anatomic, physiologic, pathologic, and immunologic (1).

The infant is more susceptible to lead than the adult. Balance studies showed that children absorb much more dietary lead than adults, approximately 50% in children (2) and 10% in adults (3). Comparative data on airborne lead absorption in children and adults are not available.

At present no evidence is known to exist to show whether neurochemical or neurophysiological changes precede changes in the hematopoietic system. Several biochemical tests demonstrate early effects of lead on the hematopoietic system. In contrast with these tests there are no comparable neurochemical tests for the measuring of early metabolic changes in the nervous system. The hematopoietic system is currently considered to be the site where the first measurable adverse effect ("critical effect") occurs (4-6). The quantitative determinations of characteristic indicators showing lead effect on the hemoglobin synthesis are the most common parameters in the assessment of elevated lead exposure.

A lead smelter area is a particularly good location in which to study lead absorption, both in adults and children. Chronic inhalation and ingestion of lead by contaminated air, food, and water are common characteristics of a population living in the vicinity of a lead smelter. It is in such an area, therefore, that comparative studies can be carried out most effectively.

The three aforementioned aspects--the deficiency of information on lead absorption and toxicity in children, the opportunity to measure the adverse effect of lead on hemoglobin synthesis by means of characteristic parameters in blood and urine, and an available location for conducting comparative studies in adults and children--constituted the basis of this project.

MATERIAL AND METHODS

Three family groups, comparable in their socioeconomic status but differing in lead exposure, have been investigated. Group 1 consisted of families who lived in the vicinity of a lead smelter and whose fathers were occupationally highly exposed to lead. Group 2 consisted of families resident in the same area with a high nonoccupational lead exposure, but whose fathers had no

supplemental occupational exposure to lead. The third was the control group, consisting of families who lived in an area with a low lead exposure and whose fathers were not occupationally exposed to lead. Families were selected with one child under 4 years and, if possible, another child of school age.

It had been planned to examine 20 families in each group. Although efforts were made to complete this number, in the first lead-exposed group and in the control group fewer families were examined. The explanation for this was the relatively small number of families with a small child and the negative attitude of parents to blood tests in small children. In the second lead-exposed group one additional family was included in the study. Table 1 presents the number of families in the groups examined and Table 2 the number of family members within the groups examined. The total number of subjects was 181.

The age of the fathers was 23 to 46 years, of the mothers 21 to 44 years, of the children of school age 5 to 16 years, and of the small children 11 months to 4 years. The age distribution for parents is presented in Table 3, and for both groups of children in Table 4.

The subjects in both lead-exposed groups lived in a river valley close to a lead smelter (Figure 1). Several settlements (A, B_1, B_2, C_1, C_2, C_3, D) are located at various distances from the smelter. Table 5 and Table 6 show the residential distance distribution from the lead smelter for both groups. The majority of families lived in settlement D, which was most distant from the emitting source.

The valley was about 500 m above sea level. Winds blew either from the southwest, bringing humid air and rain, or from the northeast, bringing dry, cold air and fair weather. The mean monthly temperatures varied from 3 to 17.5°C with a maximum of about 30°C and a minimum of about -16°C. Snow, which usually falls in the second half of November, remains on the ground for 100 to 210 days, approximately.

The subjects in the control group were matched to the exposed families with regard to the socioeconomic status and nutritional condition. They lived in several settlements (E, F, G, H, I) about 400 m above sea level in a climate very similar to that of the lead-exposed area.

In the subjects examined, the following biological indicators of elevated lead exposure were determined: hemoglobin (Hb), hematocrit (Htc), basophilic stipple cells (BpE) count, reticulocyte (Rtc) count, erythrocyte protoporphyrin (EP), δ-aminolevulinic acid in urine (ALA-U), and total coproporphyrin in urine (CP-U).

Hb was determined spectrophotometrically by the cyanmethemoglobin method (7). Hematocrit was determined by the standard method according to Wintrobe (8). BpE were fixed and stained according to the method of Hamel (9), using Löffler's methylene blue solution. In each blood film a total of 1000 red blood cells was counted. Rtc were vitally stained with 1% Brilliant-Cresyl-blue solution in ethanol, and a total of 1000 red blood cells was counted in the film.

The concentration of EP was determined according to the method of Rimington as modified by Cripps and Peters (10). In this assay 5 to 10 ml of blood with heparin as anticoagulant were used. The packed red cell volume (V) was calculated from the hematocrit. Red cells were separated from plasma and were treated with 50 ml of an ethyl acetate: acetic acid (4:1 ratio) mixture and allowed to stand overnight in the dark at 4°C. On the following day the erythrocyte extract was filtered and washed with the ethyl acetate and acetic acid mixture until the filtrate containing hematin and porphyrins (uroporphyrin, coproporphyrin, and protoporphyrin) became colorless. In this filtrate uroporphyrin was discarded with a saturated sodium acetate solution. Any remaining coproporphyrin and protoporphyrin in sodium acetate washings were reextracted with fresh ethyl acetate. Protoporphyrin and coproporphyrin were separated from hematin by extraction with 3 N NCl until the successive acid extracts no longer showed any fluorescence under the Hg-lamp (366 nm). The acid extract of porphyrin was neutralized with solid sodium acetate to pH 3.2 and then shaken with 50 ml portions of ether. From the ether extract coproporphyrin was extracted with successive 2- to 3-ml portions of 0.1 N HCl until all coproporphyrin was removed, which was confirmed by examination of the extracts for red fluorescence under the Hg-lamp (366 nm). Protoporphyrin was then extracted in the same way, using 1.5 N HCl, and the total volume (v) recorded. The absorption of the acid protoporphyrin solution was measured against water in a 1-cm cell at 380 nm (A_{380}), 430 nm (A_{430}) and at the maximum absorption in the Soret region (Amax) using a Beckman DB-G spectrophotometer. The EP concentration expressed as µg EP/100 mlE was calculated according to the formula suggested by Rimington (10). In this formula $[2A_{max} - (A_{430} \pm A_{380})] .C. \frac{v}{V}$, the symbol A is related to the corresponding absorptions, C is the correction constant with value of 1.226, v is the total volume of 1.5 N HCl protoporphyrin extract, and V is the volume of the packed red cells which was used in the analysis. The precision of the method expressed as a relative standard deviation was 4.0% ($\bar{X}$ = 207.9 µg EP/100 mlE; N = 8).

ALAD activity was determined according to the modified method of Bonsignore et al. (11). Modification of the original method was applied in the lower pH value of the ALA substrate (pH=6.8) by the use of a sodium phosphate buffer instead of a carbonate buffer and in the volume of reagents, which was increased to twice the original volume. First, 0.4 ml of whole blood was hemolyzed in 2.6 ml of distilled water, and then to this hemolyzate 2 ml of freshly prepared 0.01 M δ-aminolevulinic acid were added. From this solution 2-ml aliquots were distributed in two centrifuge tubes. In tube 1 ("blank") 2 ml of mercury (II) chloride-trichloroacetic acid solution were added. Both tubes were incubated in a 37± 0.2°C water bath for 1 hour, following which the porphobilinogen reaction in tube 2 was stopped by the addition of 2 ml of mercury (II) chloride-trichloroacetic acid solution. Both deproteinized samples were then centrifuged at 2500 rpm. The porphobilinogen formed was determined by the Ehrlich reaction: 2 ml of the supernatant was added to 2 ml of modified Ehrlich reagents, and after 10 minutes absorption was measured on the Beckman DB-G spectrophotometer against water at 555 nm in a 1-cm cell. The supernatant of the blank tube was treated in the same way. The enzyme activity was expressed in units, one unit being defined as the difference in absorption in matched 1-cm cells between the specimen in which reaction had taken place and the blank tube, corrected for the sample dilution and the percent of

hematocrit. The relative standard deviation for this method was 0.8% ($\bar{X}$ = 201.6 units/mlE; N=6).

Lead in blood was analysed by flameless atomic absorption spectrophotometry (12) using Perkin-Elmer HGA-72. The original method was slightly modified (13) with regard to the volume of the injected sample and the temperature program. To 2 ml of 0.1% Triton X - 100, 0.5 ml of whole blood and 0.1 ml of 0.5 % nitric acid were added. From this diluted sample 0.02 ml was injected directly into the graphite tube, and the absorption was measured at 283.3 nm. The follwoing temperature program was experimentally selected as optimum: drying at 100°C for 40 s, ashing at 100-450°C ("ramp" program), and atomization at 2045°C for 10 s. The calculation was made according to the addition method, using freshly prepared standard solutions of 1 µg Pb/ml and 5 µg Pb/ml in 0.5% nitric acid. The relative standard deviation for this method was 2.4% ($\bar{X}$ = 17.5 µg Pb/100 ml; N=11) and 4.9% ($\bar{X}$ = 48.8 µg/100 ml; N=11).

ALA-U was determined by the Davis-Andelman (14) modification of the Mauzerall-Grannick method. The separation and isolation of ALA from a urine sample (1 ml of a 24-hour specimen*) was carried out by disposable plastic chromatography columns (Bio-Rad Laboratories). From the cationic (hydrogen) column ALA was eluted with 7.0 ml acetate buffer (pH 4.6), and this eluate was collected in a 10-ml volumetric flask. After the addition of 0.2 ml of acetylacetone and acetate buffer to the mark, the sample was allowed to stand in a boiling water bath for 10 minutes. The porphobilinogen formed was determined by the modified Ehrlich's reagent: a 2-ml aliquot was mixed with 2 ml of modified Ehrlich reagent and after 15 minutes absorption was measured on a Beckman DB-G spectrophotometer against water at 555 nm in a 1-cm cell. A blank with water was prepared in the same way. The calculation was made according to the regression line obtained by known ALA concentrations (3 to 52 µg ALA/ml urine). The relative standard deviation for this method is 3.5% ($\bar{X}$ = 0.475 mg/100 ml; N=6).

CP-U concentration was measured fluorometrically by the method of Schwartz et al. (15). The urine sample (5 ml of 24-hour specimen*) was acidified by buffered acetic acid. Porphyrins were extracted with ethyl acetate and the extract washed with a sodium acetate solution to remove uroporphyrins. Coproporphyrin precursors were oxidized with iodine. Total coproporphyrins (I + III) were extracted with several portions of 1.5 N hydrochloric acid. The combined extract was made up to 25 ml with 1.5 N hydrochloric acid. The fluorescence was measured by means of the spectrofluorimeter Perkin-Elmer MPF-2A. A standard curve was made by the known coproporphyrin concentrations. The sensitivity of the method is 1 µg/100 ml urine.

Families from the smelter area were examined in May/June of 1976, and those in the control area in October of 1975. The results obtained were statistically evaluated and compared. Analysis of variance, Student's t-test, correlation, and frequency distribution analyses were carried out. In cases

---

* It was impossible to obtain a 24-hour sample from children up to 4 years of age.

where the F-test values showed that both standard deviations did not belong to the same population the significance of the difference between the two groups examined was determined by the method of Cochran and Cox (16), instead of using the standard t-test (results marked with an asterisk in the tables).

In the environmental survey air lead, dustfall lead, household-dust lead, and drinking-water lead were analysed.

Air-lead concentration in the exposed area was measured for 3 consecutive years (1974 to 1976) at five sites in settlements 4.0 and 0.5 km N. (Figure 1, sites 1 and 2), 1.5 and 2.0 km SSW. (Figure 1, sites 3 and 4), and 2.5 km SW. (Figure 1, site 5) from the lead smelter. Weekly samples from about 14 m^3 of air were collected on the membrane filters of 1-inch diameter filtration surface by means of diaphragm pumps. The exact volume of the air samples was recorded by a gas meter. The filters with the samples were dissolved in nitric acid solution, evaporated to dryness, and redissolved in EDTA solution (to prevent loss of lead due to absorption). The final solution, after being made up to a certain volume (2, 5, 10 ml or more) should have 1% EDTA and pH 8. The samples were analysed using a Unicam SP 90 atomic absorption spectrophotometer under the following conditions: current 6 mA, acetylene flow 1000 ml/min, airflow 5000 ml/ min, burner height 0.8 cm, and slit width 0.2 mm. A blank and a set of standards were run with each set of samples. The concentration of lead in the final solution (µg/ml) was read from a calibration curve and converted into the concentration of lead in air (µg/m^3). The sensitivity of the method was 2 µg Pb per 1 ml of the final solution.

In the control area over a 1-year period (November 1, 1974 to October 30, 1975) air-lead concentration was measured at one site. Daily samples from about 200 m^3 of air were collected on the membrane filters of a 4-inch diameter filtration surface by means of a high-volume sampler. The samples were analysed in the same way as the samples from the lead-contaminated area.

Lead content of dustfall was measured simultaneously at four sites in the exposed area (Figure 1, sites 1, 2, 3, and 5), and at one site in the control area, for a period of 1 year (November 1, 1975 to October 31, 1976). Monthly samples of deposited lead were collected in plastic containers of 1.5-liter volume with a 10-cm diameter opening, evaporated to dryness, dissolved in nitric acid, evaporated again, redissolved in 1% EDTA, and analysed by atomic absorption spectrophotometry.

Samples of household dust were collected in 26 homes, 14 from the exposed area and 12 from the control area. Household dust was sampled by a pump with a special adapter supplied with a screen at its opening to prevent coarse particles and small objects from being collected on the membrane filter, which served as a sampling surface. The total weight of dust was determined, and subsequently the sample was analysed for lead by the same procedure as the samples of airborne lead.

Samples of water were collected from five sites in both the exposed and the control area. In both areas only part of the homes were supplied from the public water supply. Many homes had a well of their own or an individual running water system. In some cases water was used from a fresh-water spring.

All kinds of water, therefore, were included in the lead analyses. Water samples were collected in 5-liter plastic containers with 0.2 ml HNO_3 to prevent absorption of Pb on the walls of the containers. One liter aliquot of the sample was neutralized by ammonia and separated by anion exchange from interfering ions. The eluate was evaporated to dryness, dissolved in 1% EDTA solution (pH=8), and analysed by atomic absorption spectrophotometry.

RESULTS AND DISCUSSION

Biological Indices of Lead Absorption

The individual laboratory findings of the lead-exposed groups, 1 and 2, and of the control group are presented in the Appendix (Tables A-1, A-2, and A-3). Figures 2 to 23 show the frequency distribution for each parameter in the groups examined. In the two lead-exposed groups the obtained results for the mothers and for both groups of children were pooled. As visible from the diagrams the distributions do not follow an ideal pattern of normal or skewed distribution. The small number of the subjects examined might be an explanation of this variation. Differences in nutritional habits, alcohol consumption, and environmental circumstances in the house or working place might be additional reasons.

The results obtained are summarized in subgroups--fathers, mothers, children of school age, and children up to 4 years--and presented separately as arithmetic means ($\bar{X}$) with standard deviations (SD) and standard errors (SE) for each group in Tables 7, 8, and 9. The fathers in group 1, occupationally exposed to lead, showed findings in accordance with their exposure which were significantly different from all other subjects examined. These findings can be classified as "excessive" exposure. The fathers in group 2 have findings at the level of a "slightly elevated" exposure. The mothers, school-age children, and small children in both exposed groups could be classified in the same category. Blood-lead concentrations obtained in the control group were a little higher than expected. Percentile distribution (Figure 24) differed from that proposed by Zielhuis (17), but was very close to the results of Secchi et al. (18). The environmental survey did not show any specific features for the group under investigation. It has been assumed, therefore, that the other factors, due to nutritional habits and lead-contaminated alcohol consumption, might be the source of individual "abnormal" blood-lead concentrations. This finding should not have any bearing on the final conclusion, because the control group was matched to the exposed groups with regard to the socioeconomic status and nutritional condition. Thus the expected differences reflect the actual values.

The significance of the differences in arithmetic means was tested among the fathers, mothers, school-age children, and small children within each group examined.

Within the lead-exposed group 1 (Table 10) there was a statistically significant difference in the majority of findings between fathers and mothers, and fathers and both groups of children. Mothers and children did not differ very much. Between the mothers and both groups of children the difference was

significant for ALAD activity (lower in children), Rtc number (higher in mothers), and ALA-U expressed in 24-hour diuresis (higher in mothers). In addition, mothers had significantly higher Hb, Hct, and CP-U expressed in 24-hour diuresis than small children. Between school-age children and small children the only significant difference was in ALA-U, expressed in 24-hour diuresis.

In the second lead-exposed group (Table 11) the fathers had significantly lower ALAD values than the mothers. Statistically significant differences in the same direction were found between fathers and school age children for Hb, Hct, Pb-B, and ALA-U, expressed in 24-hour diuresis, and between fathers and children up to 4 years for Hb, Hct, Pb-B, and ALA-U. Higher lead absorption in fathers could be attributed to the fact that the fathers possibly spent more time in areas with higher contamination levels. Alcohol consumption could be an additional factor. Between the mothers and both groups of children the difference was significant for Pb-B and EP concentration (both higher in children), and for ALA-U and CP-U, expressed in 24-hour diuresis (both higher in mothers). In addition, small children had significantly lower ALAD activity, Hb, and Hct values than the mothers. Between school-age children and small children the difference was significant for Hb, Hct, ALA-U, and CP-U, expressed in 24-hour diuresis (higher in school-age children).

In both lead-exposed groups the difference in Hb and Hct between the subjects may be attributed to the difference in sex and age. The higher Rtc figures in mothers than in both groups of children was probably caused by the regular menstrual bleeding, which stimulated the release of reticulicytes from the bone marrow to the peripheral blood.

Comparing three very important parameters, Pb-B, ALAD, and EP, first as a measure of the dynamic body-lead pool (19), second as the most sensitive index for an early response to the slightest lead exposure (20), and third as the best indicator of total lead body burden (21), one may conclude that the following was the sequence of lead absorption in family members of both groups: fathers > school-age children $\tilde{=}$ children up to 4 years > mothers. The population of mothers was the least-exposed group, which may be explained by the fact that mothers spend more time at home and in places which are less contaminated by lead.

In the control group (Table 12) there was a significant difference in Hb and Hct among family members too. With reference to the other findings the fathers showed significantly higher Pb-concentration and lower ALAD activity than all the other subjects. This could be associated with lead-contaminated alcohol consumption. The difference between adults and children for ALA-U and CP-U, both expressed in 24-hour diuresis, and between school-age and small children for CP-U, expressed in 24-hour diuresis, should be taken with caution, because urine samples for analysis in children were not always collected over a period of 24 hours.

The significance of the difference in arithmetic means between exposed groups 1 and 2, and between each exposed group and the control group, of fathers, mothers, and both groups of children has been presented separately in Tables 13, 14, and 15.

In the lead-exposed groups (Table 13) the fathers of group 1 had significantly higher EP concentration, ALA-U excretion, and lower ALAD activity than the fathers in group 2. A small difference was established in Hct values, while the other parameters did not differ significantly. The only difference in the mothers was a higher Pb-B concentration in group 1 than in group 2. Children too had one different parameter, ALAD activity, which was lower in group 1 than in group 2. The results call attention to the difference in the mothers and in both groups of children because the difference in the fathers was expected. It may be assumed that mothers and children in group 1, whose husbands and fathers were occupationally exposed to lead, had a slight additional lead exposure. There are two possible explanations: first, the residential distribution in regard to distance from the lead smelter may have been different in group 1 than in group 2, and second, fathers who were occupationally exposed to lead may have contaminated the environment of the house by bringing home dust in their clothes and hair. Tables 5 and 6 show that the residential distribution in regard to distance from the lead smelter was not the same in both groups. Another point, however, was that the number of families living close to the lead-emitting source in locations A, B_1, and B_2 were almost the same (33% in group 1 and 28% in group 2). The environmental survey showed that the average concentration of lead in household dust in group 1 was higher than in group 2 (see p. 14), but the difference was not statistically significant. It is not possible to verify which factor prevailed in the observed difference.

A comparative analysis of subjects in lead-exposed group 1 and in the control group (Table 14) demonstrated that all parameters except one (Hct) differed more or less significantly in fathers, which was compatible with their occupational exposure to lead in group 1. In the mothers and both groups of children the differences were highly significant in ALAD activity and in EP concentration in small children. Significant differences were found in Pb-B of mothers and small children, in EP of mothers and school children, and less significant differences in ALA-U and CP-U of mothers. Other parameters did not differ significantly, although in some of them (e.g. Pb-B and ALA-U/100 ml in school children) the difference was almost significant.

A comparative analysis of subjects in lead-exposed group 2 and in the control group (Table 15) showed that in each subgroup the difference between groups was very significant with regard to EP concentration and ALAD activity. The same level of significancy was found in Pb-B concentration except in mothers, whose difference was not significant ($P<0.10$). As mentioned previously, mothers of both lead-exposed groups were the least exposed to lead. Less significant differences were found in the BpE number in fathers, in ALA-U expressed per 100 ml of urine in school-age children, and in CP-U expressed in 24-hour diuresis in mothers. The other parameters did not differ significantly.

The statistical significance of the difference between the three examined groups was established by analysis of variance. Three parameters, ALAD, EP, and Pb-B, are the most sensitive indices of increased lead absorption, regardless of sex or age (Table 16). They are good parameters to establish the difference in lead absorption from the environment. ALA-U and CP-U were found to be good indicators in fathers and mothers but not in children. This may be

explained by the fact that urine samples in children were not always collected over a period of 24 hours. The mean diuresis in fathers was 822 ± 333.4 ml, in mothers 796 ± 294.9 ml, in school-age children 467 ± 275.5 ml, and in small children 146 ± 150.6 ml. A statistically significant difference in Rtc and BpE number in fathers should be attributed to their occupational exposure to lead. This was an indirect indication that both parameters were useful in the control of workers occupationally exposed to lead.

Hb concentration in four subgroups did not differ significantly between three examined groups (Table 16). On the other hand, a significant difference in Hb ($t=2.235$; $P<0.05$) between the fathers in lead-exposed group 1 and those in the control group (Table 14) was found by analysis of Student's t-test. This discrepancy may be explained by the fact that the level of the observed significance was at the upper limit ($P<0.05$) of significancy. The increased lead absorption from the environment in the investigated area close to the lead smelter had no effect on Hb decrease in the population. This is a very important conclusion of this report.

The fact that Hb was not significantly decreased, although lead absorption was significantly increased, can be attributed to the presence of zinc and other metals which accompany lead ores. In the animal studies zinc showed a moderating effect on ALAD inhibition (22-23). Lead-induced anemia in rats was completely prevented when optimal levels of copper and iron were supplemented in the diet (24). It is reasonable to assume that the findings in animals might be related to human beings, but this should be the subject of a separate study.

The ALAD activity, EP, ALA-U, and CP-U concentrations were compared with Pb-concentration in the total study population and in subgroups. Semilogarithmic relationship was found to be better than linear relationship. Figures 25-30 present such a relationship in a total study population. There is a highly significant ($P<0.001$) correlation between ALAD and Pb-B, EP and Pb-B, ALA-U and Pb-B, and significant ($P<0.01$) correlation between CP-U and Pb-B. In the subgroups of fathers (Figures 31-36) the results were almost the same. In mothers (Figures 37-42) correlation was significant for EP-Pb-B ($P<0.001$) and ALAD-Pb-B ($P<0.05$) but not for urine parameters with Pb-B. In children of school age (Figures 43-48) and in small children (Figures 49-54) highly significant relationship ($P<0.001$) was found between ALAD-Pb-B and EP-Pb-B. Furthermore, children of school age showed a significant relationship ($P<0.05$) between ALA-U expressed per 100 ml of urine and Pb-B. There is no plausible explanation for the negative correlation between CP-U expressed in 24-hour diuresis and Pb-B in small children.

The relationship between Hb and Pb-B was established in subgroups only. Both semilogarithmic and linear relationships were carried out. In fathers the correlation was negative but not significant (Figures 55 and 56). In mothers the correlation was unexpectedly positive, but significant ($P<0.05$) only for the relationship lin Hb/lin Pb-B (Figures 57 and 58). The findings in both groups of children were even more surprising. The correlation between Hb and Pb was positive and significant ($P<0.02$) for each group of children and for semilogarithmic and linear relationship (Figures 59-62). This is contradictory to the findings of Landrigan et al. (25), who found a significant

negative relationship between blood-lead level and hematocrit values in a study of 1047 children living near a lead smelter. In the present study a relatively small number of subjects were studied in each subgroup (less than 50). In a small group of subjects with slightly elevated lead exposure from the environment, there is more chance of a "false" direction of statistical assessment than in a large group with the same exposure, or in a small group with more marked excessive lead exposure, such as fathers occupationally exposed to lead. An additional explanation of the results obtained in mothers and children could be the simultaneous exposure to lead and other metals, such as zinc, copper, and iron, which have a "protective" effect against increased lead absorption (22-24). "Pure" and "mixed" lead exposure should not be identical, and we cannot exclude the possibility that lead alone may affect the Hb-concentration, but not in conjunction with other metals.

The results obtained are not unique. Wibowo et al. (26) found in males (N=57), not occupationally exposed to lead, a positive significant ($P<0.05$) correlation between mean corpuscular hemoglobin concentration (MCHC) and Pb-B. At the same time the correlation between Hb and Pb-B was positive too ($r=0.20$) but not significant. A positive significant correlation between MCHC and Pb-B was explained by the acknowledged fact that most of the lead in blood was in the erythrocytes and bound by hemoglobin (27-28).

Blood lead, owing to the dynamic interchange of the body-lead pool, is probably not the best parameter to which other parameters should be correlated. In order to check this assumption Hb was related with ALAD and EP for each subgroup. Lin Hb and log ALAD or Hb relationship was chosen for the following reasons: 1) in the present study lin Hb values correlated with lin Pb-B yielded better correlation coefficients than log Hb values for most relationships (Figures 55-62). The best fit for the regression line for ALAD or EP and Pb-B was found to be with log ALAD activity or log EP concentration and lin Pb-B level (21, 29-31). The results obtained for each subgroup have been presented graphically in Figures 63-66 for the relationship between lin Hb and log ALAD, and in Figures 67-70 for the relationship between lin Hb and log EP. Only in fathers was the correlation significant, e.g. positive ($P<0.001$) for the relationship Hb and ALAD, and negative ($P<0.01$) for the relationship Hb and EP (Figure 67). In the other subgroups no significant relationship was found. These findings have proved to be more convincing than those with Pb-B, which may be an indirect confirmation that Pb-B is not an adequate comparable parameter.

Environmental Survey

The concentration of airborne lead, measured at five sites, 1-5, in a lead-exposed area for a period of 3 consecutive years (December 1973 to October 1976) are presented in Table 17. The data are summarized and presented as arithmetic means ($\bar{X}$) with standard deviations (SD) and standard errors (SE) for 3 consecutive years together (Table 18) and for each year separately (Table 19). As shown in Table 19, the average concentrations of air lead in the exposed area did not vary too much over the 3 years of measurements. The highest average concentration was found at site 3, which was 1.5 km SSW. from the smelter. The concentration at site 1, which was the most distant from the emitting source, was consistently the lowest and differed significantly from that at sites 2, 3, 4, and 5 (Table 20). The differences

between sites 2, 3, 4, and 5 were not significant (Table 20). The actual exposure may have been less different than shown by fixed stations, since the population moved within the valley during the course of the day. Yearly cycles of mean monthly air-lead concentration (average of five sampling sites) have been presented in Figure 71. All yearly cycles showed a winter maximum, which was influenced by meteorological factors. The extremely high concentration in December 1975 was, however, partly caused by deficiency of filter operation. The mean of air-lead averages at five sampling sites for 3 consecutive years (16.73 $\mu g/m^3$) was about twice as high as the annual mean found by Landrigan et al. (32) near an ore smelter. The lowest value at site 1 (4.0 km N. from the smelter) was about twice as high as the level found by Landrigan et al. (25) at the same distance from the smelting plant.

The concentration of airborne lead measured at one side (community G) in the control area for a 1-year period (November 1974 to October 1975) is presented in Table 21. Summarized data are shown in Table 22. Comparing the results in the control area with those in the lead-exposed area, one may see that the lowest air-lead concentration in the lead-exposed area (Table 18, sampling site 1) was still about 100 times higher than that of the control area. The mean of five sampling sites for 3 consecutive years (16.73 $\mu g/m^3$) was as much as 178 times higher than air lead in the control area.

The lead amount in dustfall, measured simultaneously at four sites in the exposed area, over a period of 1 year (November 1975 to October 1976) is presented as monthly means in Table 23. The summarized results are shown in Table 24. Dustfall lead increased on approaching the lead smelter. The highest average amount of lead was found at site 3 (1.5 km SSW. from the smelter), and the lowest was found at site 1 (4.0 km N. from the smelter), identical to the case of air-lead concentration. The largest quantities of dustfall lead were collected in November and December, although there is no distinct yearly cycle. The results are comparable with those obtained by Nordman et al. (33).

The lead amount in dustfall measured at one site in the control area for a period of 1 year (November 1975 to October 1976) has been shown as monthly means in Table 25. The summarized results have been shown in Table 26. The average annual means at four sites in the lead-exposed area (173.75 mg/m^3) was 77 times higher than the average annual mean in the control area (2.252 mg/m^2).

The lead content of household dust in the lead smelter area collected in December 1976 simultaneously in seven homes where the father was occupationally exposed to lead (group 1), and in seven homes with the father not occupationally exposed (group 2), is shown in Table 27. The summarized data for each group are presented in Table 28. Household dust in group 1 contained more lead than the dust of group 2, although this difference was not significant ($t = 0.670$; $P>0.5$). In each group there was a negative trend in the lead content of household dust with regard to the residential distance from the smelter.

The lead content of household dust in the control area collected in October 1975 in 12 homes is presented in Table 29. The summarized data are shown in Table 30. Homes in the lead-exposed area (groups 1 and 2) contained about 21 times more lead in household dust than homes in the control area.

Concentrations of lead in water samples from the exposed area (December 1976) and the control area (October 1975) are presented in Tables 31 and 32. Though the lead content in the water was about six times higher in the exposed area than in the control area, drinking water did not represent a serious source of lead intake, as all levels were below 50 µg/1, the recommended safe upper limit for lead in drinking water (34).

When the four environmental media in the exposed and control areas are compared, the lead content in the specimens examined from the exposed area decreased according to the following sequence: air > dustfall > household dust > water (Table 33).

If one uses the data obtained on air lead (the average of 16.73 $\mu g/m^3$) and assumes that on the average 15 m^3 of air was inhaled by adults per day, that 95% of the particles were respirable, that 50% of what is inhaled was retained and completely absorbed (35), that men spent on the average 4 hours and women 2 hours per day outdoors, and that the indoor lead level was on the average 60% of the outdoor level (36), then the estimated air contribution to daily absorption was 79.47 µg for men and 75.49 µg for women. Comparative estimated values in the control area (air-lead average 0.09 $\mu g/m^3$) were 0.43 µg for men and 0.41 µg Pb/day for women. For children the actual percentage retention of inhaled lead is not known. Therefore the same percentage retention was used as that determined for adults. Adjustment was made for the smaller respiratory volume (12 m^3/day for school children and 6 m^3/day for small children). Assuming that school age children spent on the average 5 hours per day outdoors, the corresponding estimated values were 65.64 µg for the exposed area and 0.35 µg/day for the control area. On the assumption that small children spent on the average 2 hours per day outdoors, values of 30.20 µg for the exposed area and 0.18 µg/day for the control area were obtained. From the results obtained it became evident that with regard to absolute values the fathers had the highest and the small children the lowest lead absorption from the air. If, however, the daily lead absorption from the air has been expressed on the basis of body weight (the average for men 75, for women 70, for school-age children 29 and for all small children 13 kg), then the children's body burden from the air was twice that of the adults (Table 34). This finding is very important in the assessment of permissible levels of lead exposure in children living near a lead smelter. This explains why children from the lead-exposed area showed a higher response to lead than the mothers.

In the exposed area and the control area the air contribution to lead absorption was practically the same in fathers and mothers (Table 34). On the other hand the biological indices of lead absorption showed that in each examined group the fathers had higher lead exposures than the mothers. If the group of subjects occupationally highly exposed to lead were not taken into account, the logical assumption was that the fathers had an additional lead exposure, probably through consumption of lead-contaminated alcohol.

The dustfall polluted by lead in lead-exposed areas is another important source of increased lead absorption in children. It is a well known fact that children play with sand and earth and that they usually like to chew non-food substances (pica). Moreover they have higher metabolic rates, and their lead absorption from the gastrointestinal tract is significantly higher than in

adults. Alexander et al. (2) found an absorption of up to 53% in eight children aged from 3 months to 8 years. Recently Mahaffey (37) estimated lead exposure for normal children on the basis of 40% absorption. Generally one may assume that children absorb 3 to 10 times the amount of lead that adults absorb.

Household dust in lead-exposed group 1 had more lead than the household dust in lead-exposed group 2, although the difference was not significant. In agreement with this finding ALAD activity in children of both ages was significantly lower ($P<0.02$ in school-age children and $P<0.05$ in children up to 4 years) in group 1 than in group 2. Sayre et al. (38) found that if dirt or dust in the child's environment contained a high concentration of lead, more lead was present on the hands or on objects that were handled and was available for ingestion via normal mouthing activities. This could explain the results obtained.

Lead contribution from water in the present study has been estimated as the lowest. Estimates of water intake have ranged from 300 ml/day for children to as much as 2 liters/day for adults (39). Taking into account these data and the average lead concentration of 10.22 µg/l in the exposed area, the estimated daily intake for children was 3.07 µg Pb and for adults 20.44 µg Pb/day, which corresponded to absorption of 1.54 µg Pb/day in children (on the basis of 50% absorption) and 2.0 µg Pb/day in adults (on the basis of 10% absorption).

Biological Indices of Lead Absorption and Residential Distance from the Lead Smelter

Owing to the small number of families living in different settlements and/or asymmetric distribution, the comparison between biological indices of lead absorption and the residential distance from the lead smelter has been made by means of median values. In subgroup "Fathers" only nonoccupationally exposed subjects were included, while in other subgroups subjects from groups 1 and 2 were pooled. Tables 35-38 present, separately for each family subgroup, the studied biological indices in comparison with the residential distance to the lead smelter.

There was practically no alteration in Hb, Htc, BpE, and Rtc with proximity to the smelter in any of the subgroups. On the other hand there was a trend towards normal values with the distance from the lead smelter in EP concentration and ALAD activity. This was especially evident for EP concentration, which followed a curvilinear relationship (Figures 72 and 73). There was an exception in the case of small children living very close to the lead smelter in settlement A (median distance 500 m); these children had lower EP concentrations than children living in settlements B_1 and B_2 (median distance 1900 m). ALAD activity was also inverse to the expected findings; higher activity was found in children of settlement A than in children of settlement B_1 and B_2. One of the possible reasons could be that parents, aware of the environmental contamination by lead in settlement A, were more concerned about their children than parents in other places more distant from the lead smelter. Another reason could be the difference in nutritional habits and the source of food (lead-contaminated milk or milk commercially produced and packed elsewhere). Neither of these aspects has been studied, and it is not possible to draw any conclusions.

Although the concentration of Pb-B was not uniform, it did not show the expected trend towards normalization with residential distance from the emitting source. Actually the highest median value in each subgroup, apart from the mothers, was found in subjects living very close to the smelter (150-800 m). Thereafter the concentration did not decrease gradually with regard to residential distance. In the studies of Nordman et al. (33), who found a negative correlation between Pb-B and the residential distance from the emitting source, a total of 293 adult inhibitants were examined. Landrigan et al. (32) investigated 758 children from 1 to 19 years of age and found significant differences between Pb-B means and residential distance. In both studies a large group of subjects were analyzed. The small number of subjects in the present study did not allow us to compare the results obtained with those obtained in large groups. However, by dealing with our data exclusively, the conclusion may be drawn that Pb-B is a less valid parameter than ALAD, particularly if compared with EP in the assessment of body response normalization, with regard to increasing residential distance from the lead smelter.

Concentrations of ALA-U and CP-U showed a somewhat decreasing trend with regard to the distance in fathers only, while in other subgroups the proximity to the smelter had no effect. Our impression is that the scattering in results of normal range could be attributed more to natural variations and/or to inadequate urine sampling than to the residential distance from the smalter.

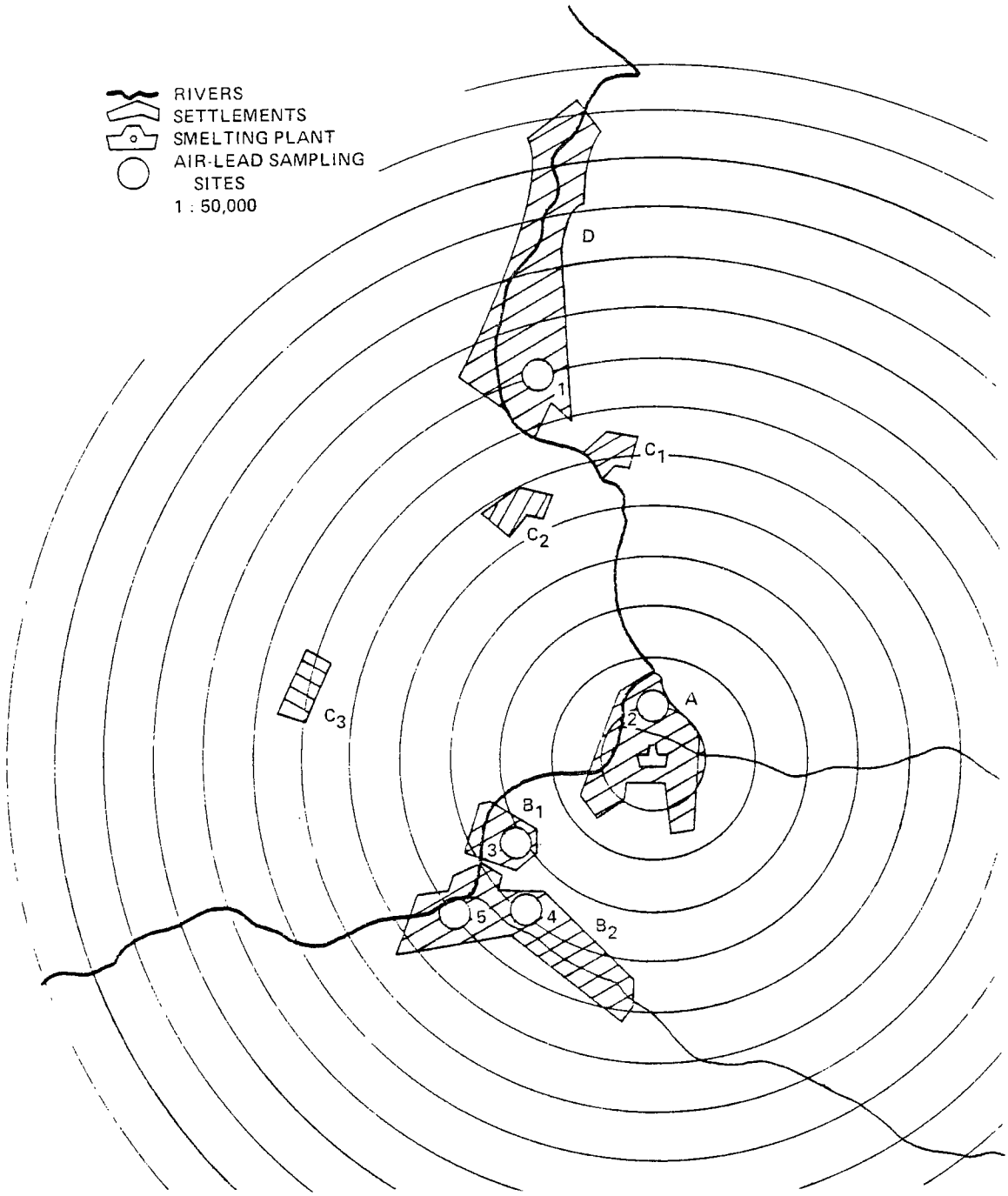


Figure 1. Scheme of the lead-contaminated area.

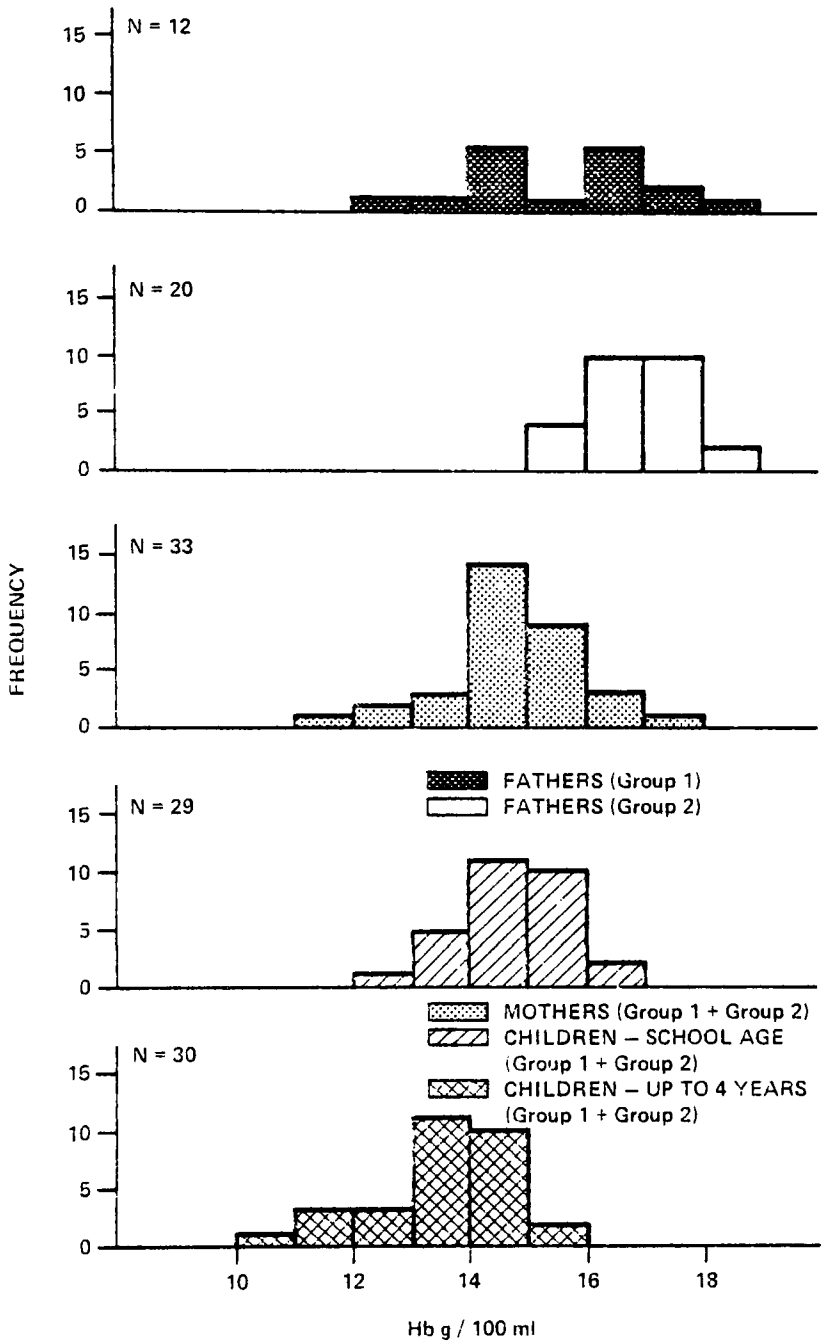


Figure 2. Frequency distribution of hemoglobin (Hb) in lead-exposed groups 1 and 2.

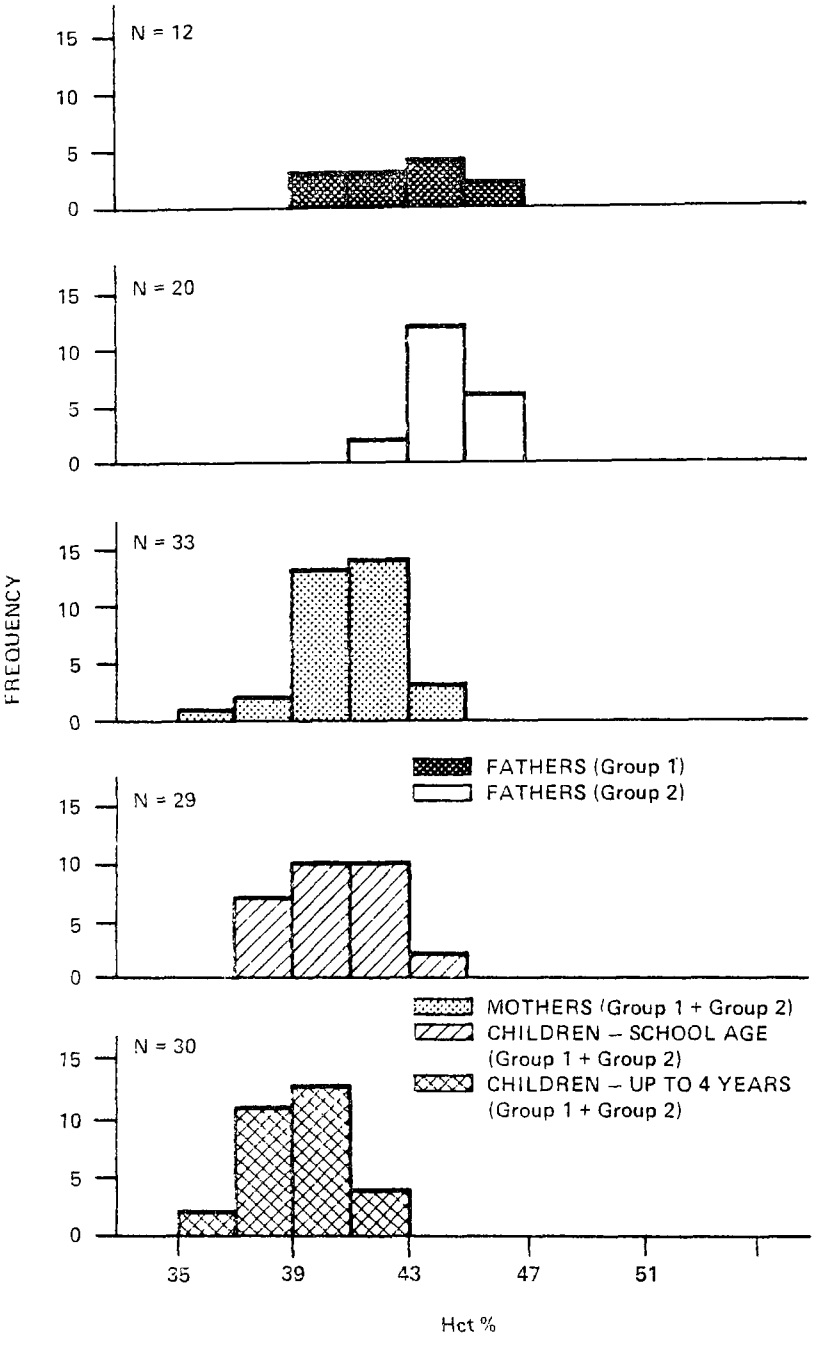


Figure 3. Frequency distribution of hematocrit (Hct) in lead-exposed groups 1 and 2.

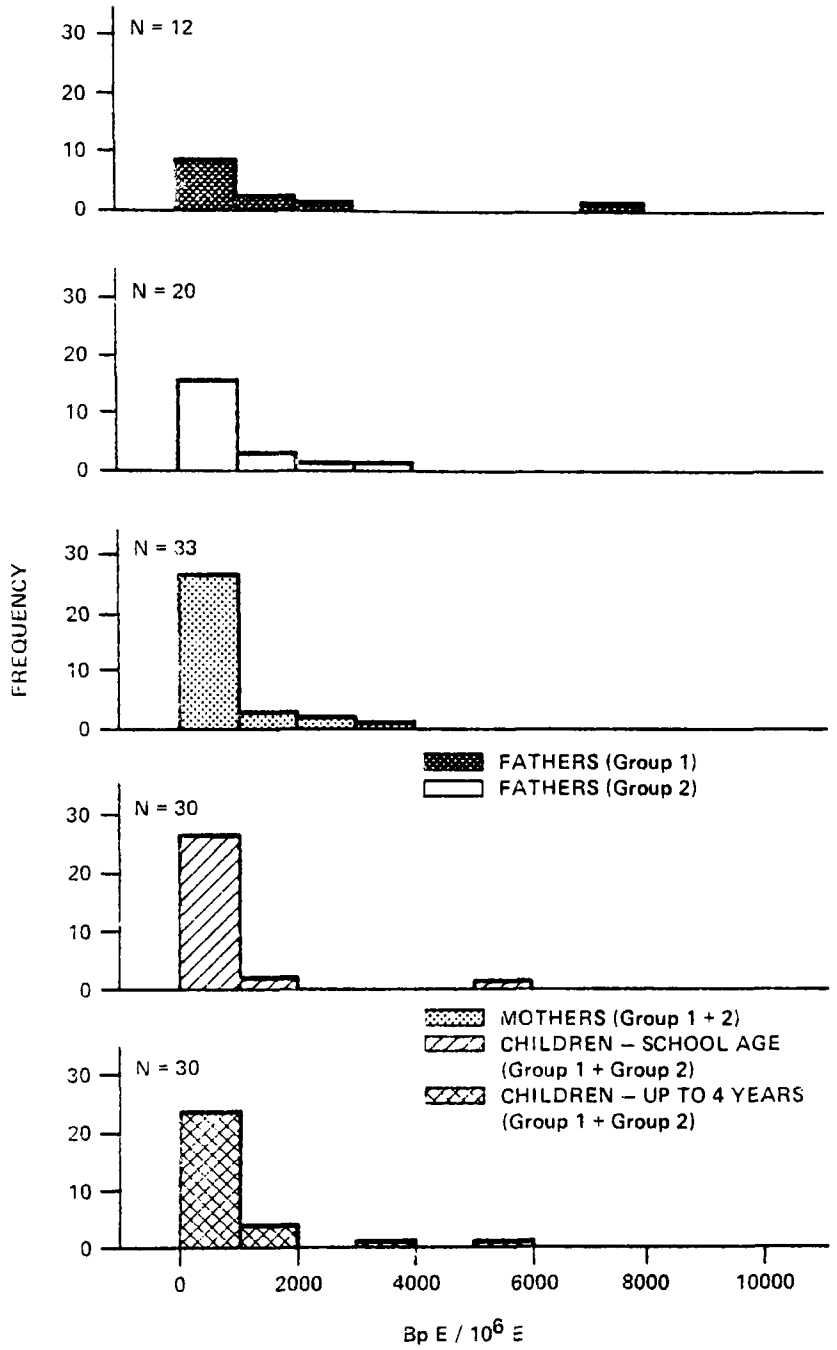


Figure 4. Frequency distribution of basophilic stippled cells (BpE) in lead-exposed groups 1 and 2.

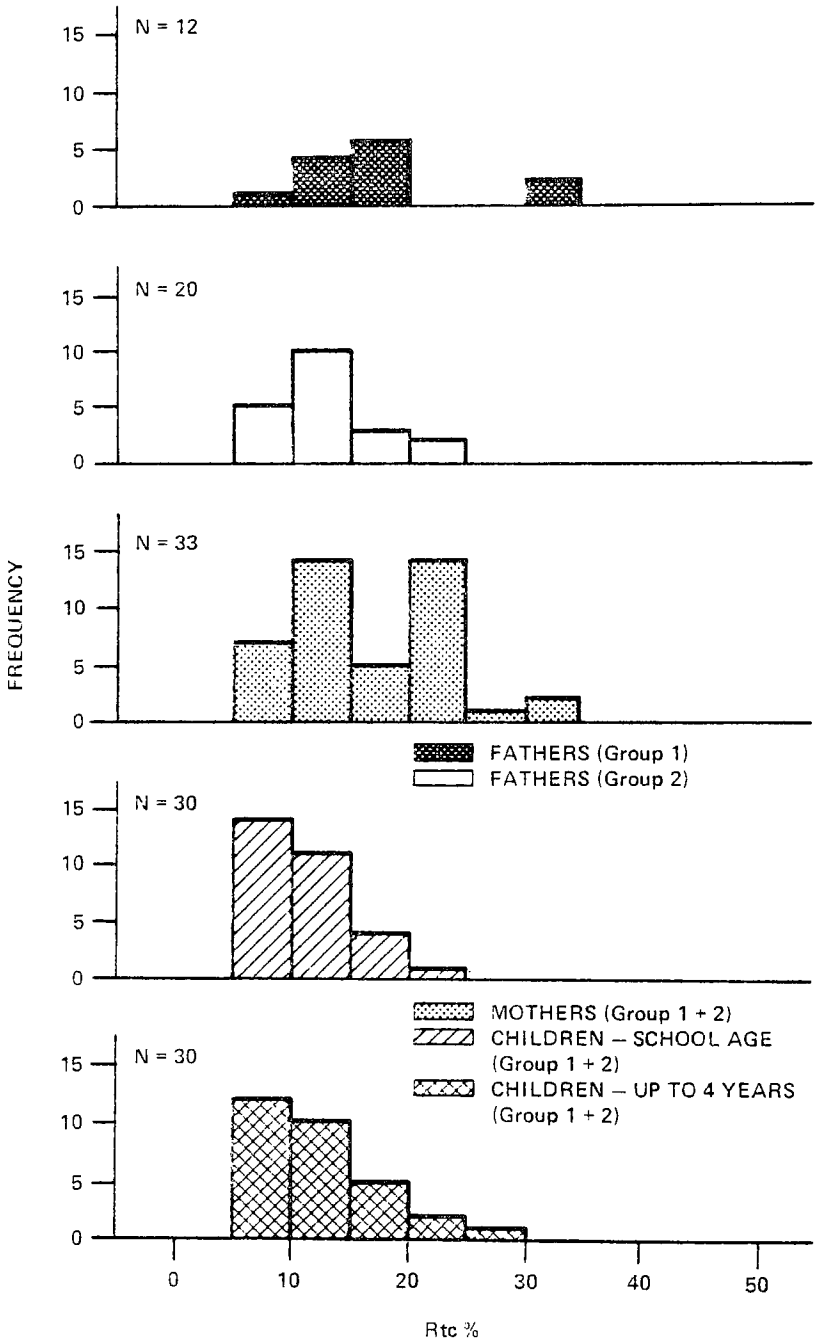


Figure 5. Frequency distribution of reticulocytes (Rtc) in lead-exposed groups 1 and 2.

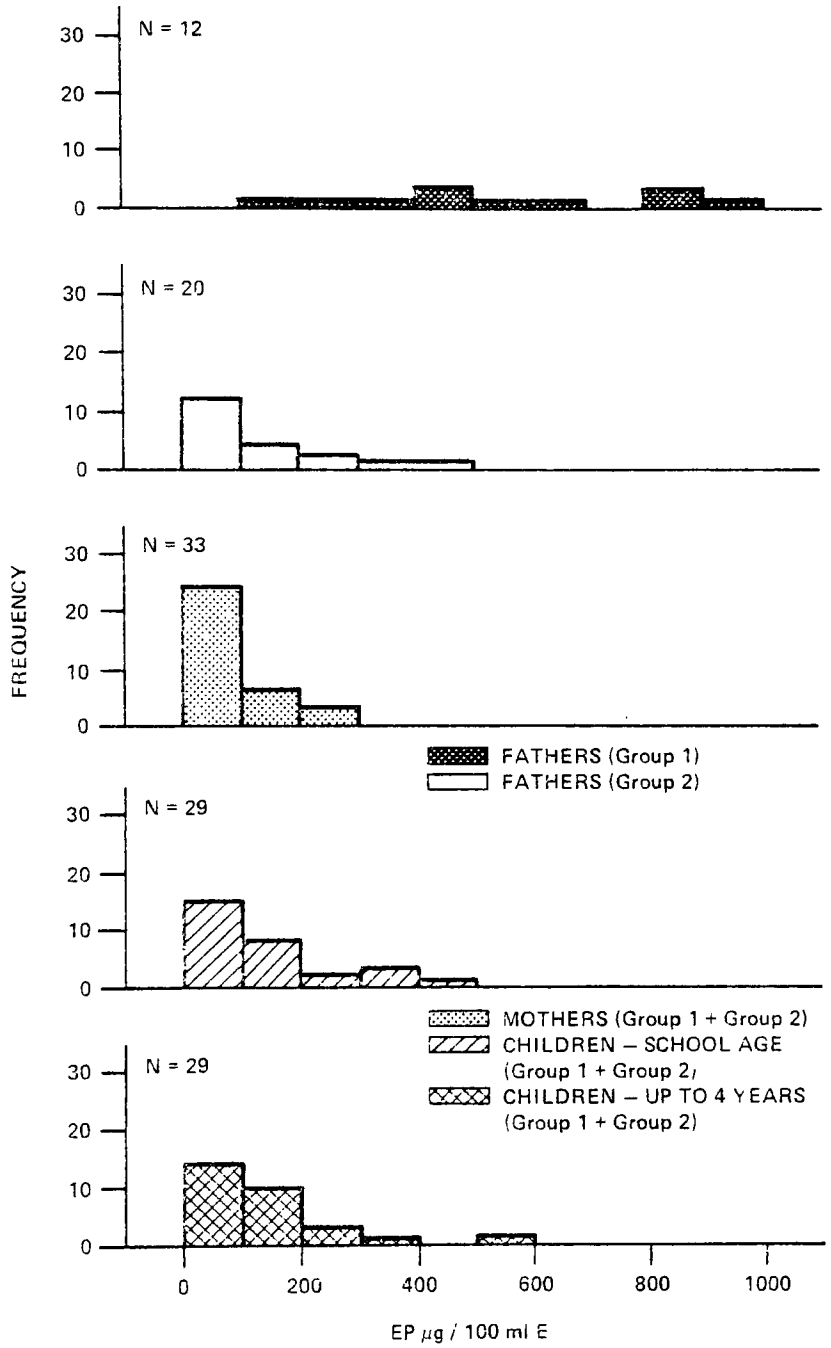


Figure 6. Frequency distribution of erythrocyte protoporphyrin (EP) in lead-exposed groups 1 and 2.

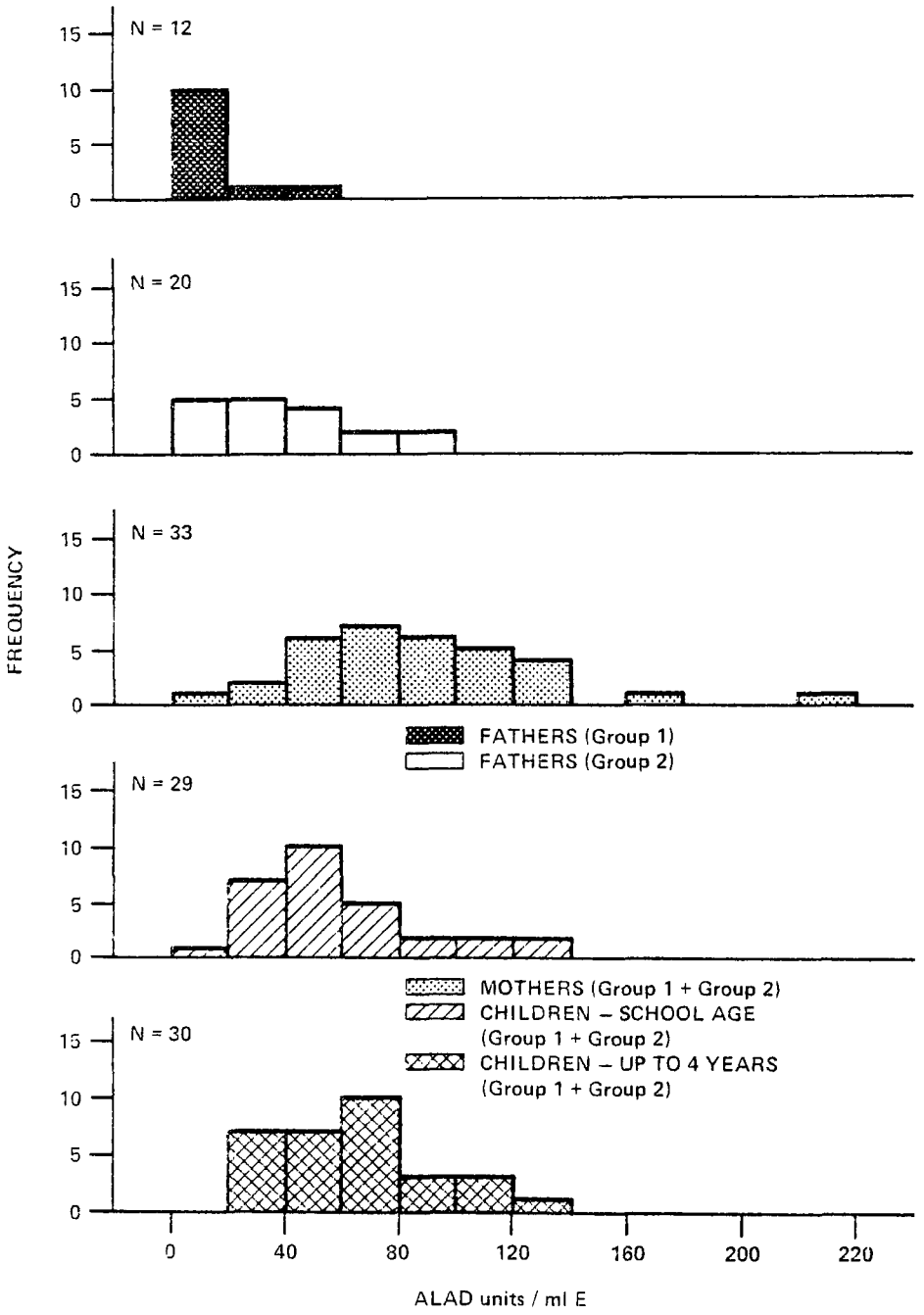


Figure 7. Frequency distribution of δ-aminolevulinic acid dehydratase activity (ALAD) in lead-exposed groups 1 and 2.

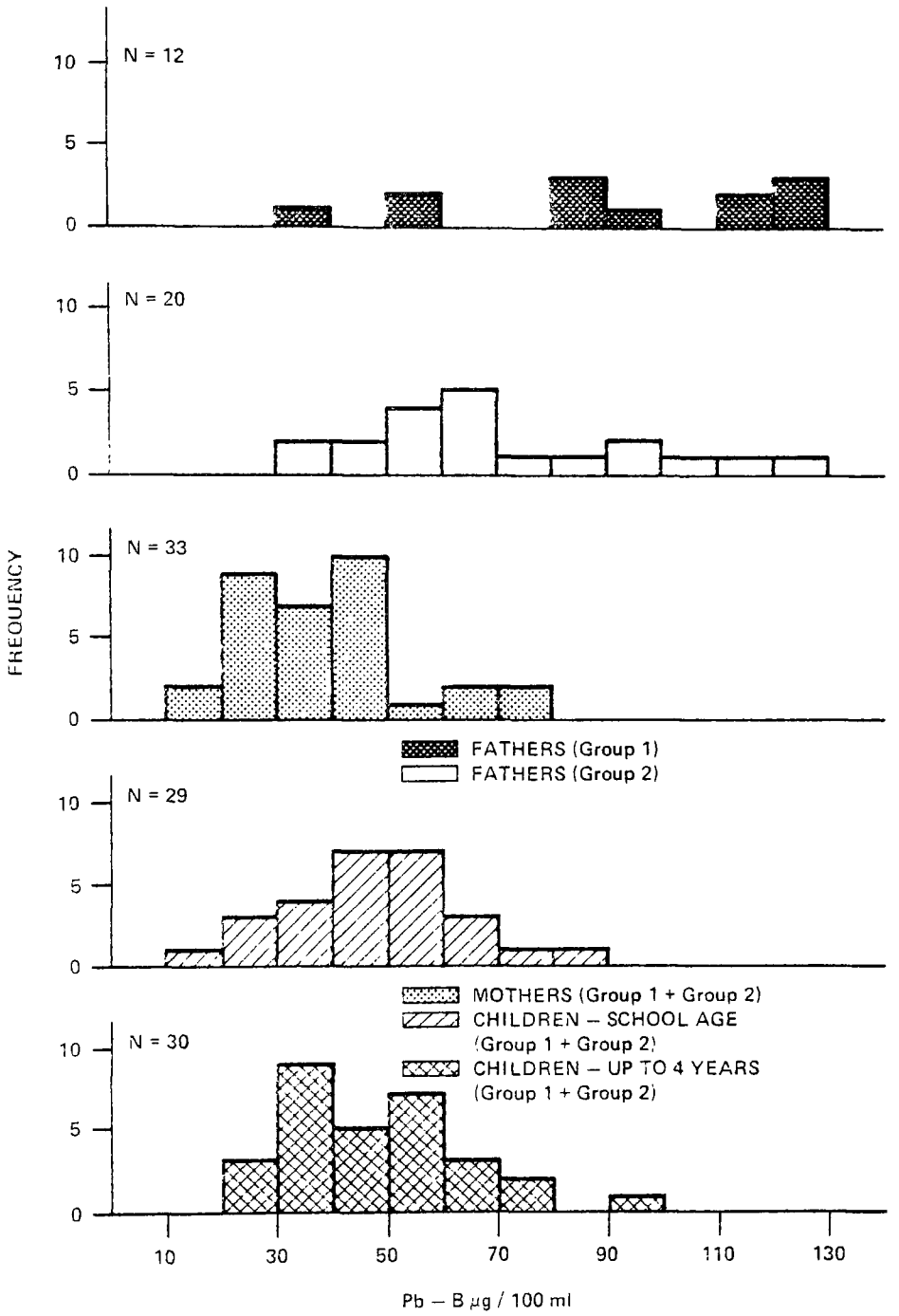


Figure 8. Frequency distribution of lead in blood (Pb–B) in lead-exposed groups 1 and 2.

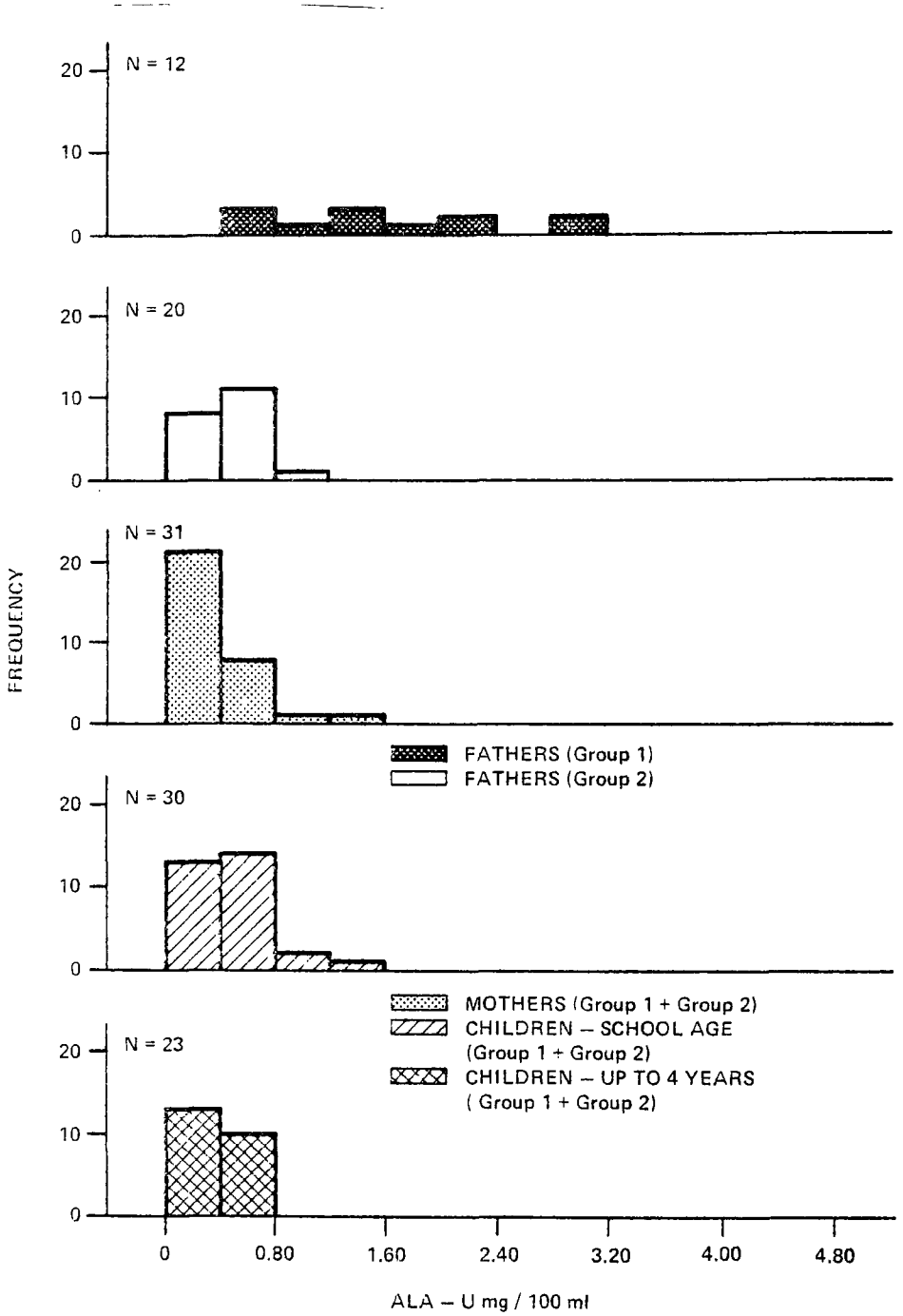


Figure 9. Frequency distribution of δ-aminolevulinic acid in urine (ALA–U mg/100 ml) in lead-exposed groups 1 and 2.

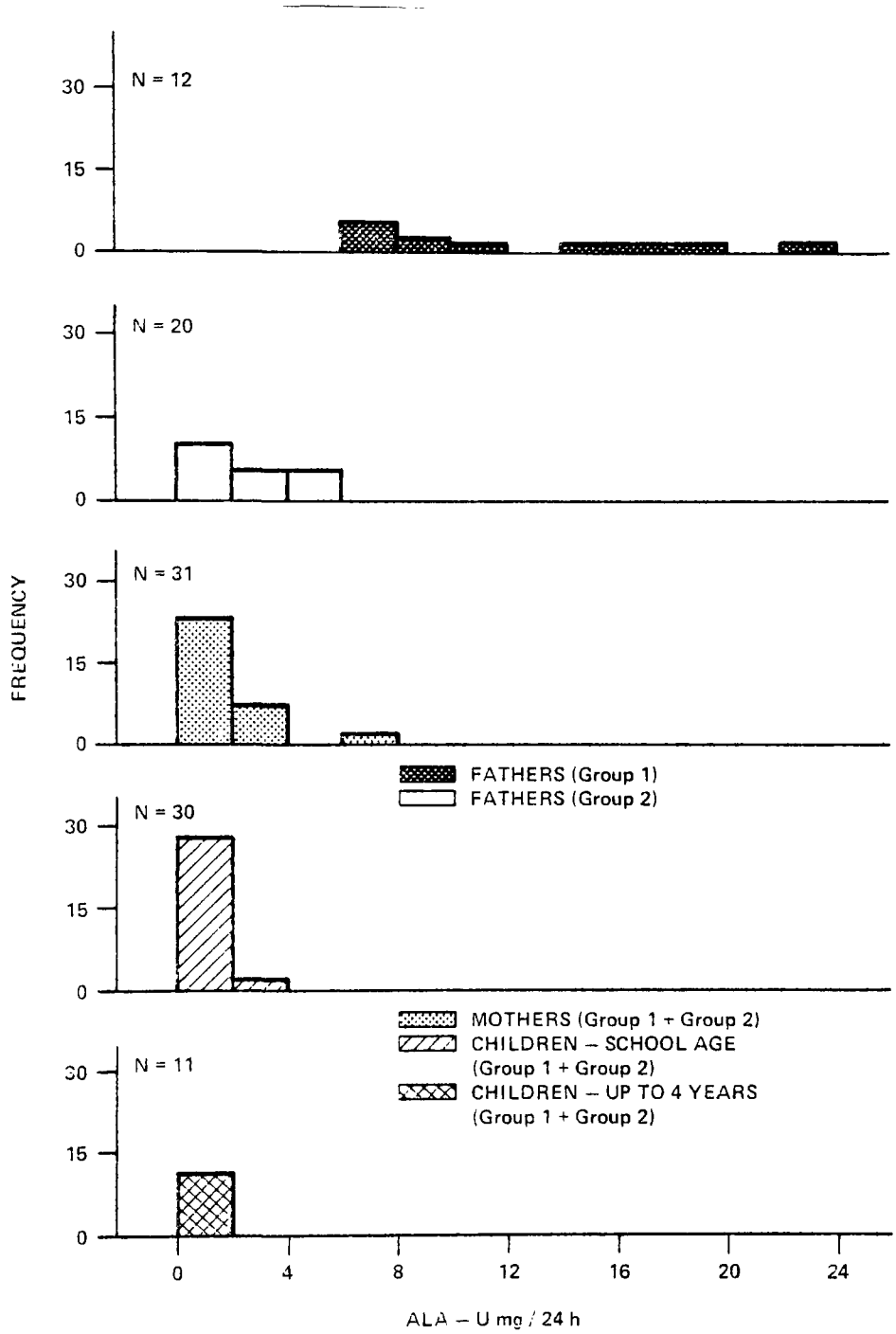


Figure 10. Frequency distribution of δ-aminolevulinic acid in urine (ALA–U mg/24 h) in lead-exposed groups 1 and 2.

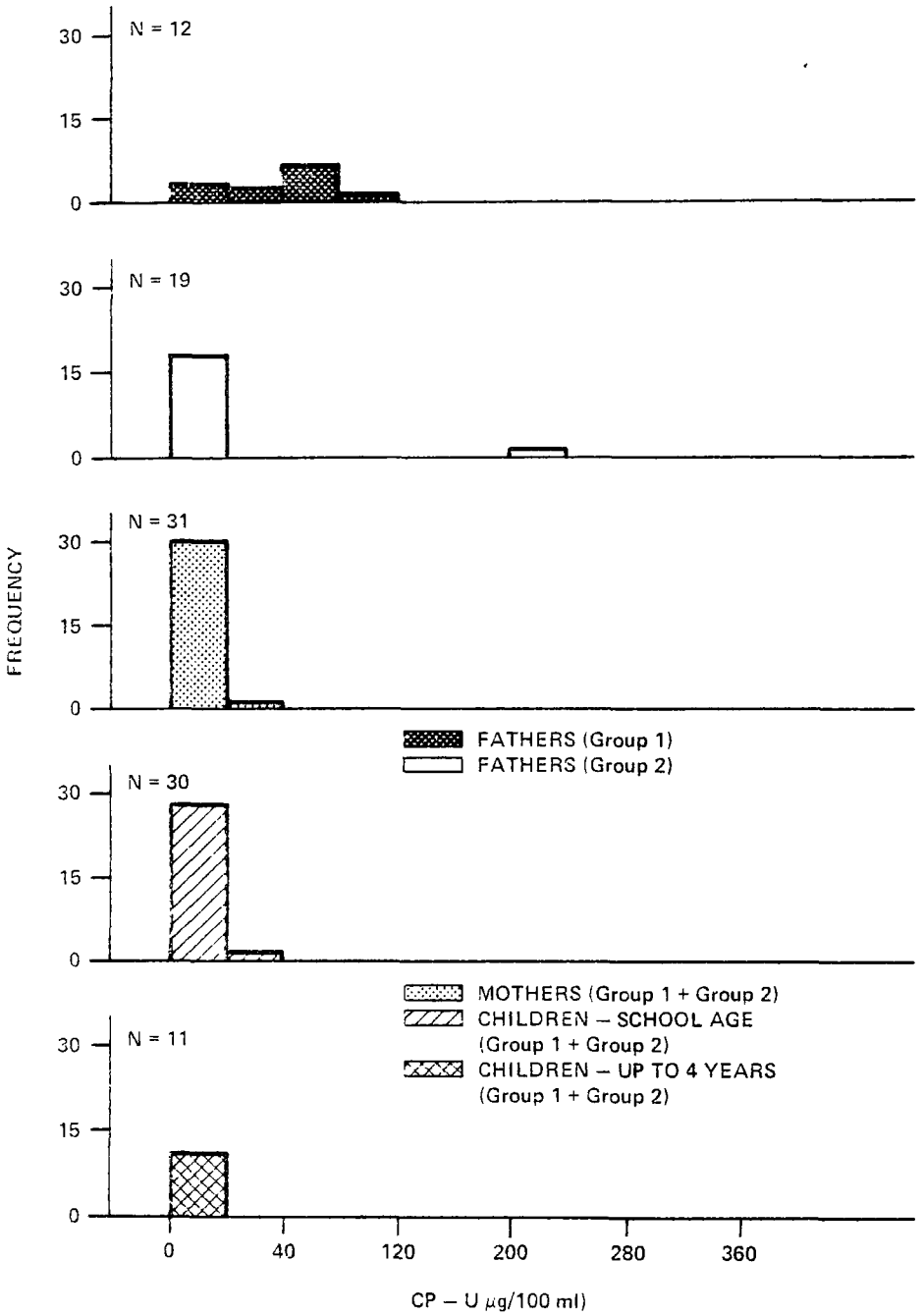


Figure 11. Frequency distribution of coproporphyrin in urine (CP–U µg/100 ml) in lead-exposed groups 1 and 2.

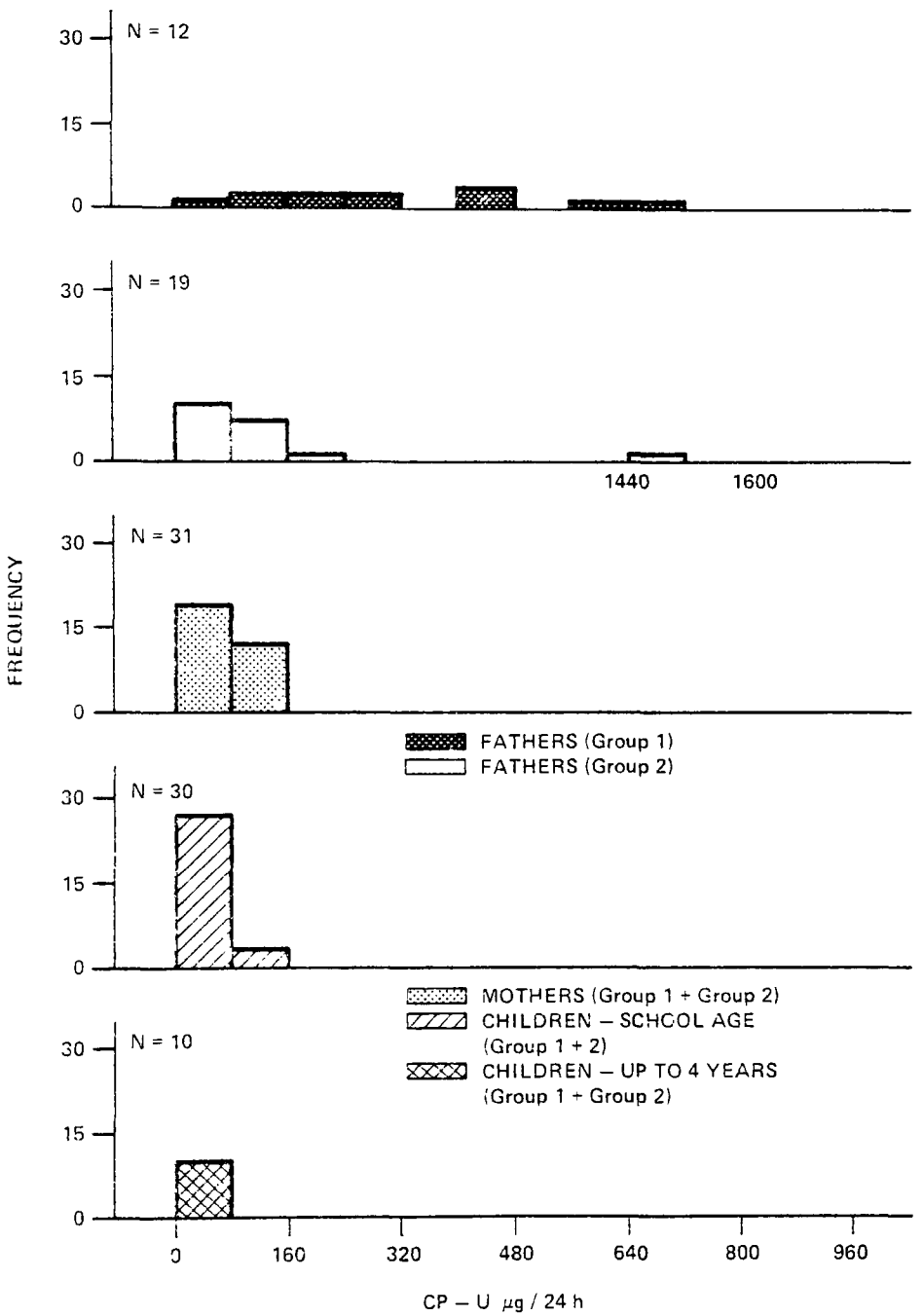


Figure 12. Frequency distribution of coproporphyrin in urine (CP–U µg/24 h) in lead-exposed groups 1 and 2.

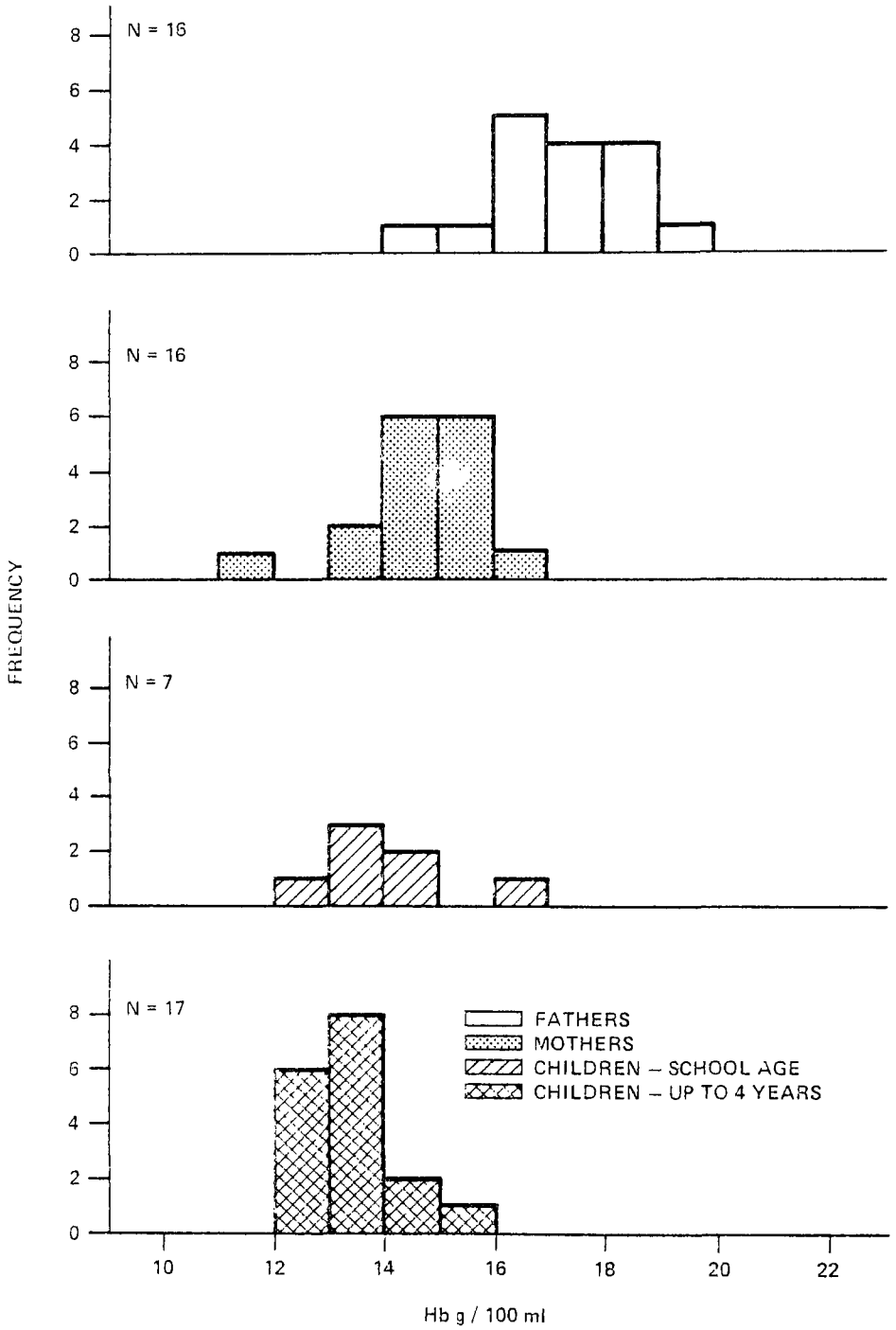


Figure 13. Frequency distribution of hemoglobin (Hb) in control group.

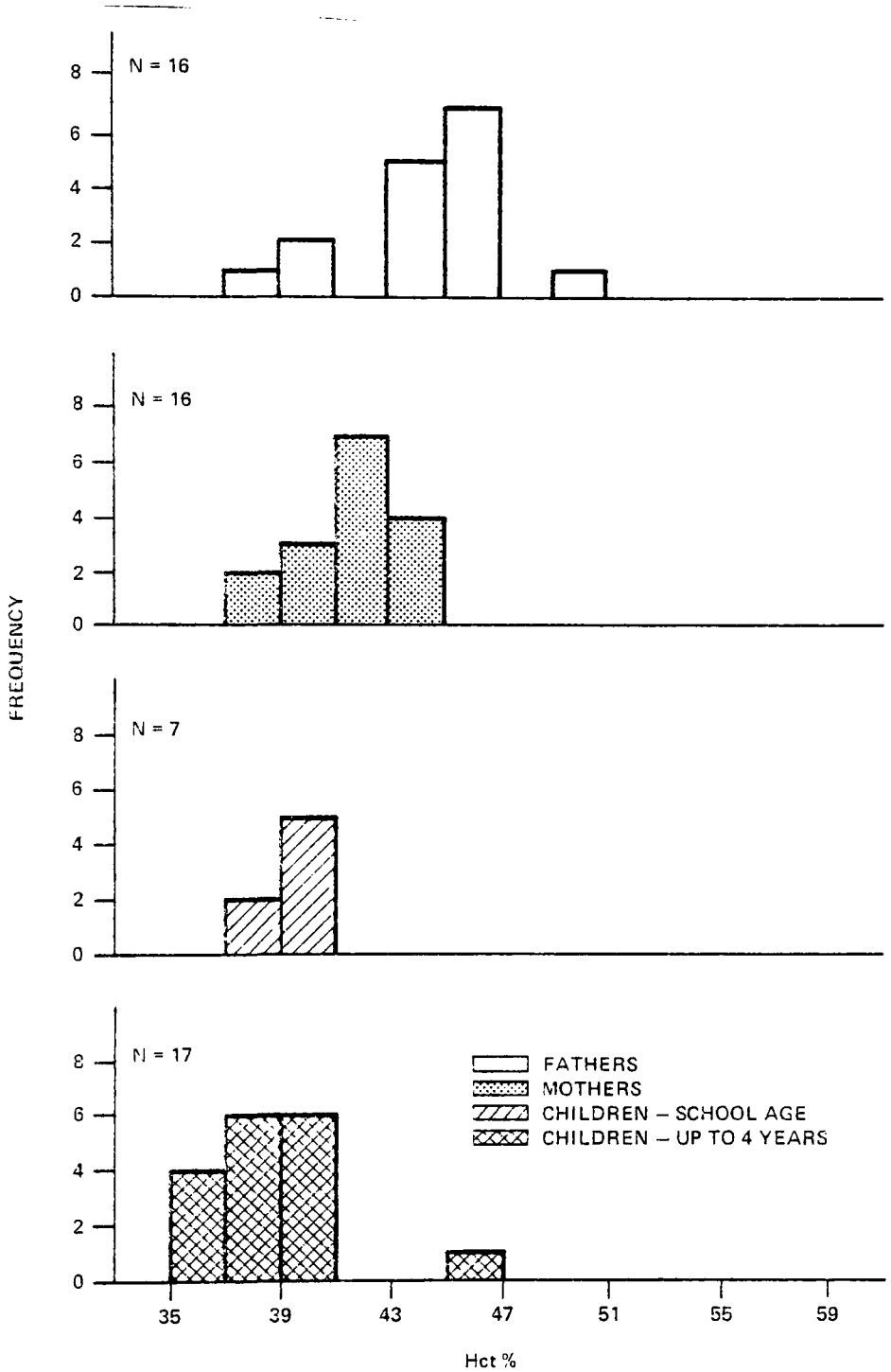


Figure 14. Frequency distribution of hematocrit (Hct) in control group.

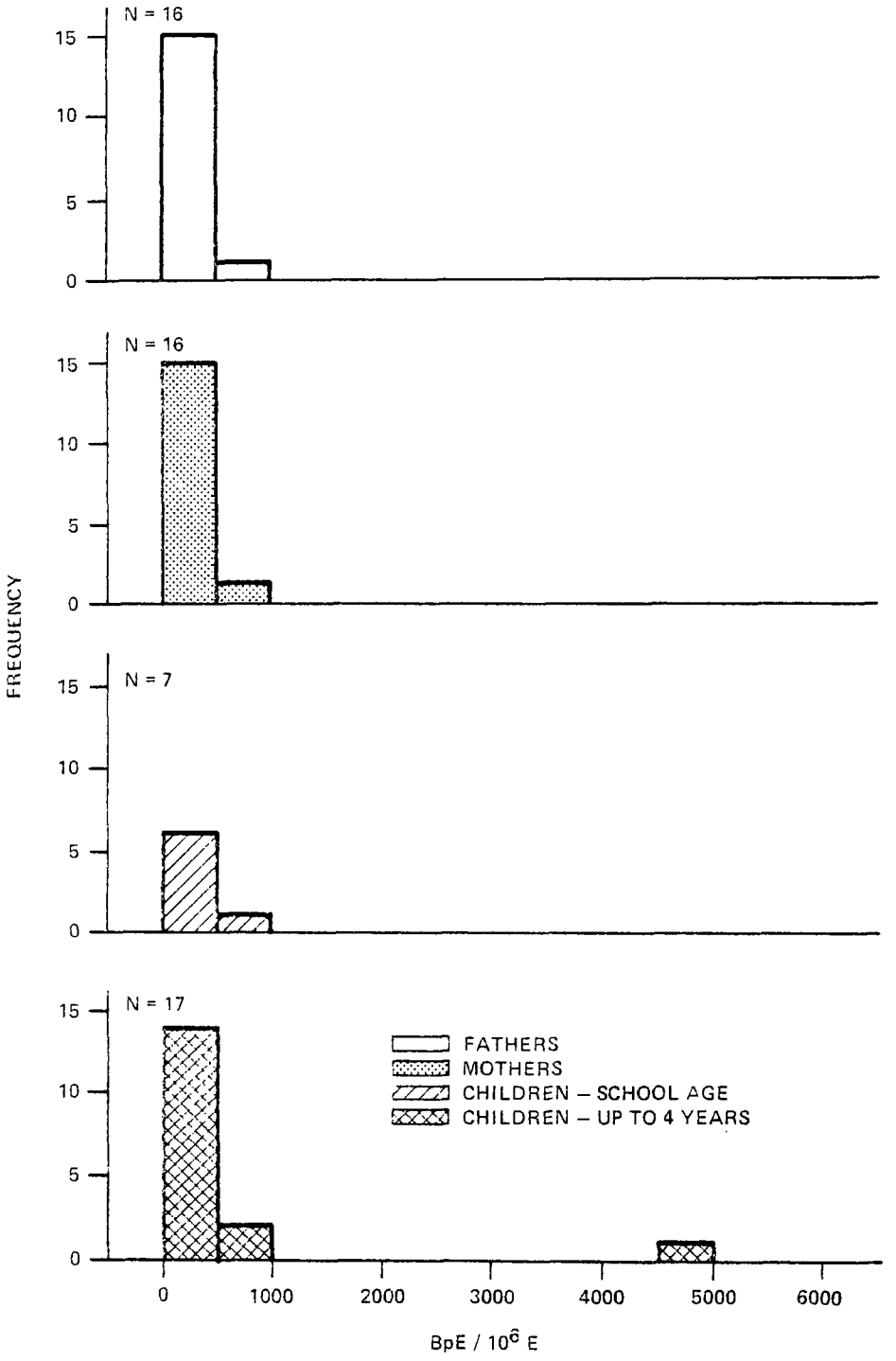


Figure 15. Frequency distribution of basophilic stippled cells (BpE) in control group.

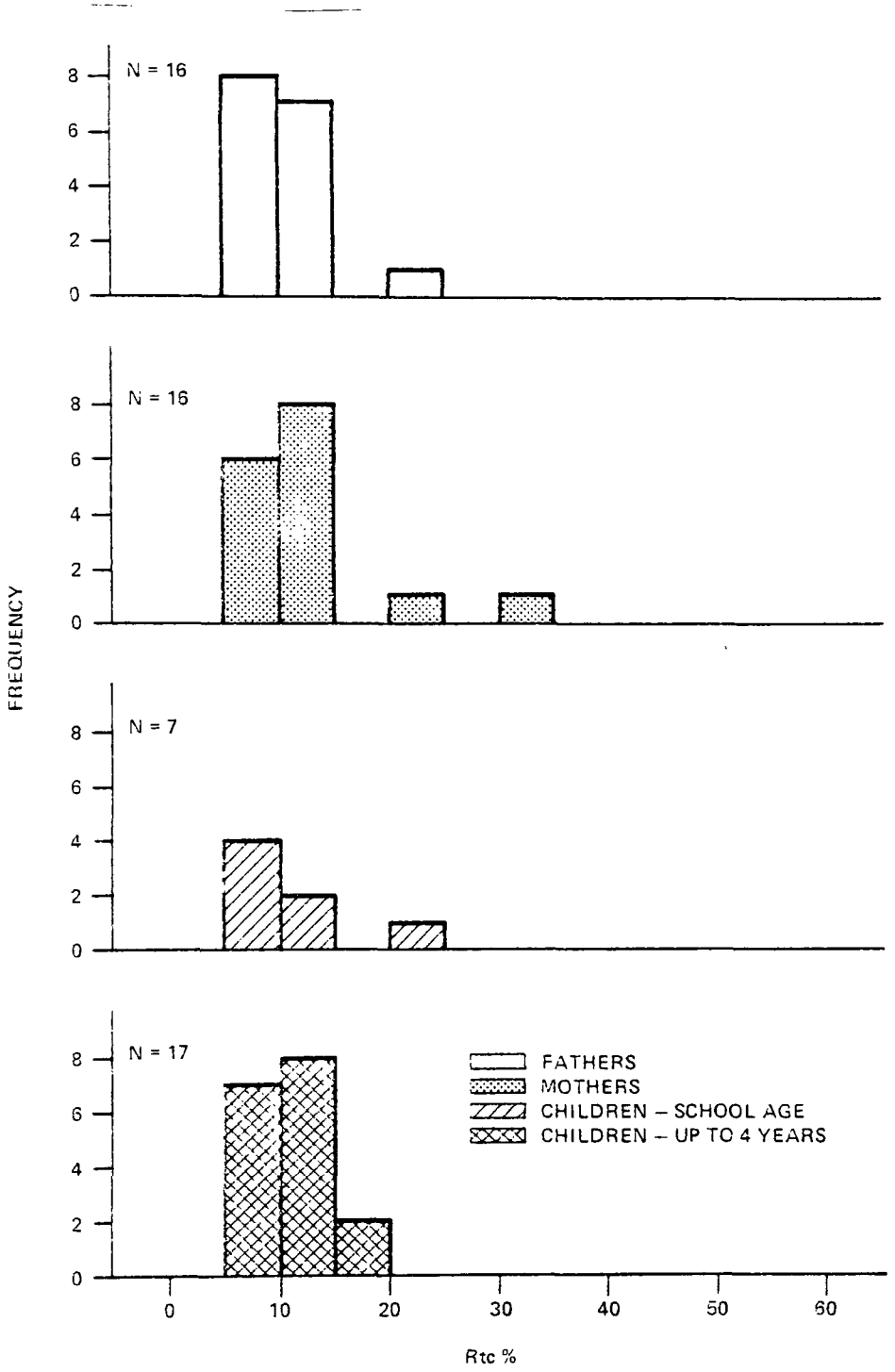


Figure 16. Frequency distribution of reticulocytes (Rtc) in control group.

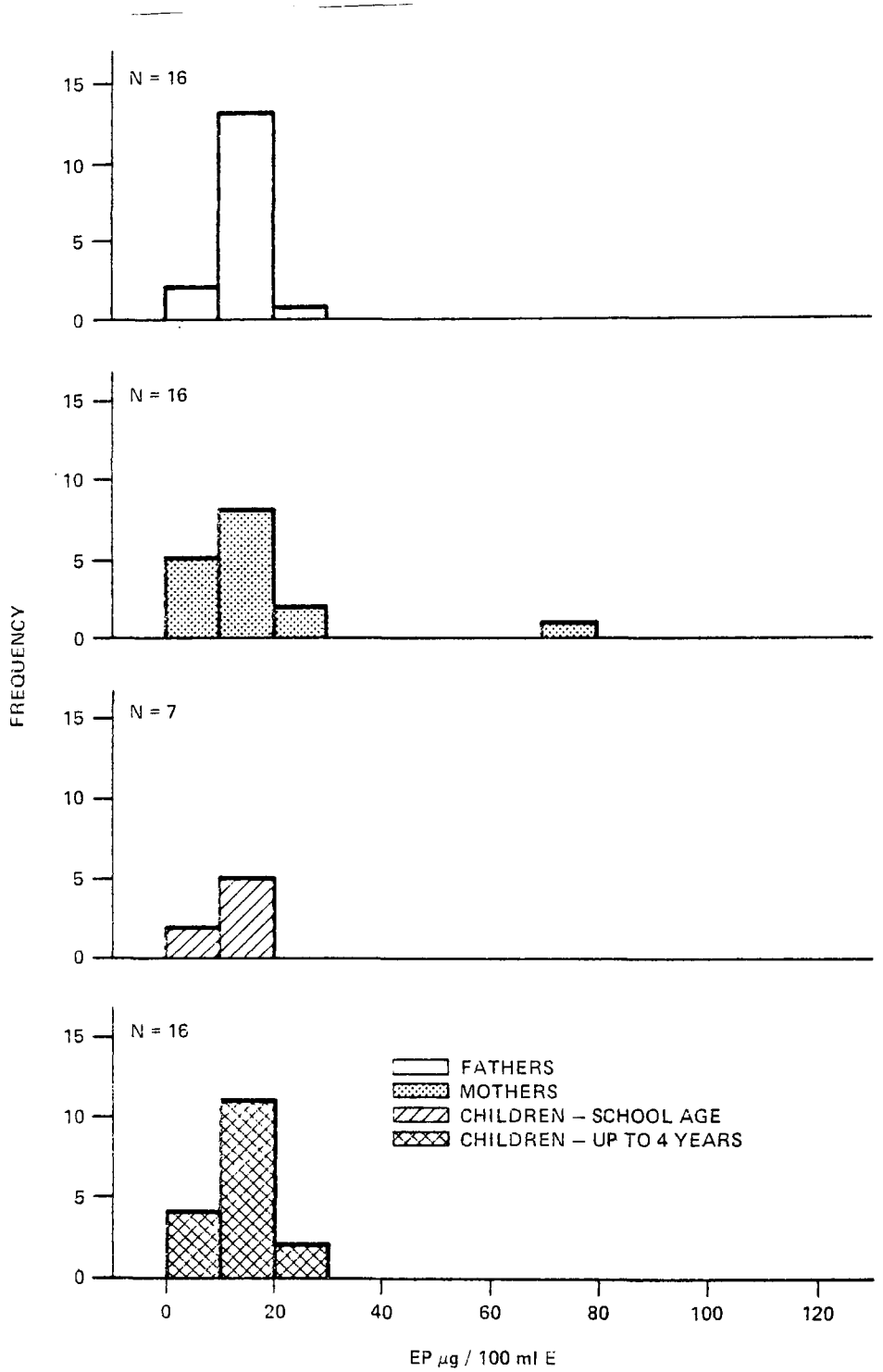


Figure 17. Frequency distribution of erythrocyte protoporphyrin (EP) in control group.

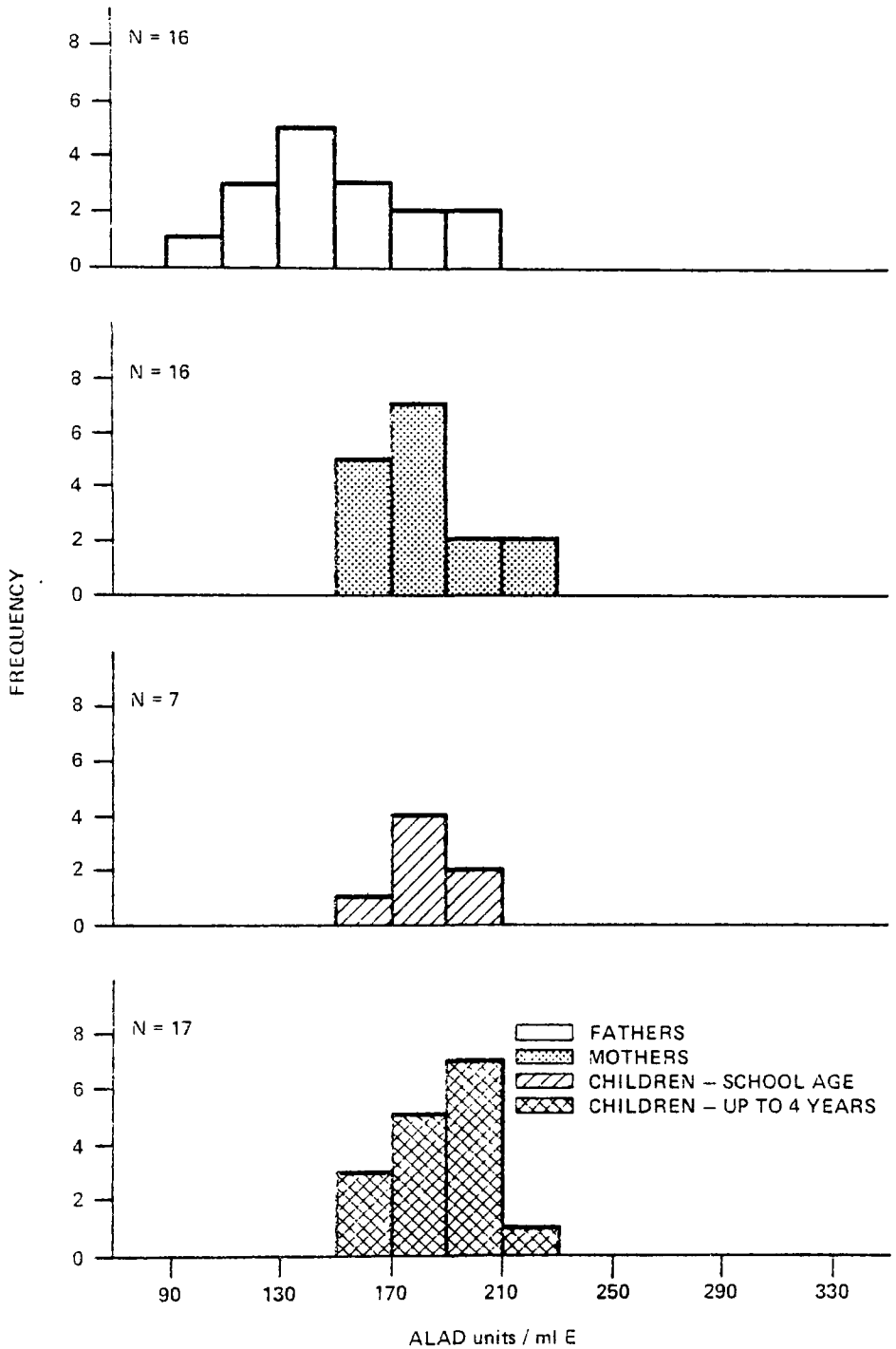


Figure 18. Frequency distribution of δ-aminolevulinic acid dehydratase activity (ALAD) in control group.

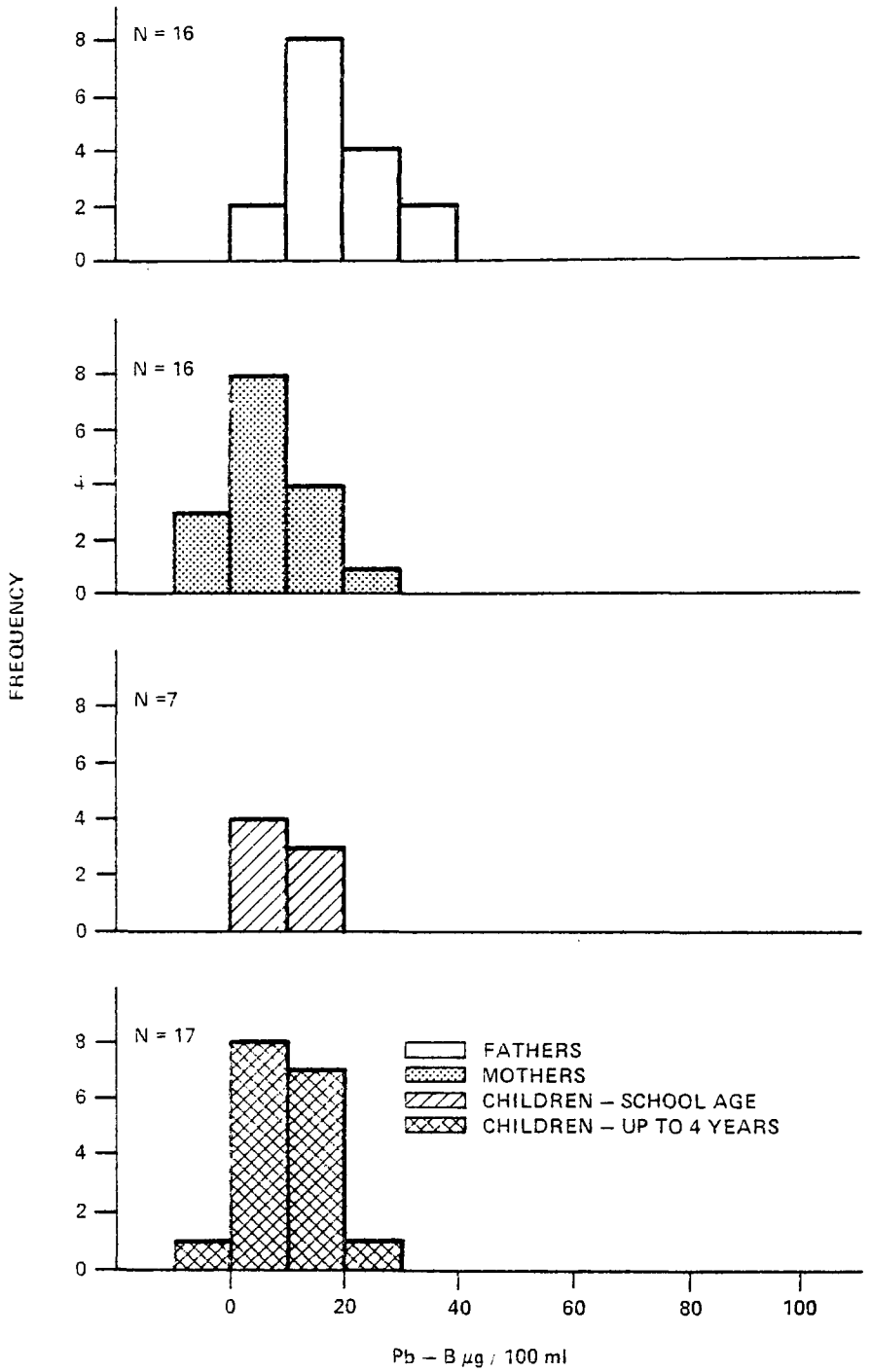


Figure 19. Frequency distribution of lead in blood (Pb–B) in control group.

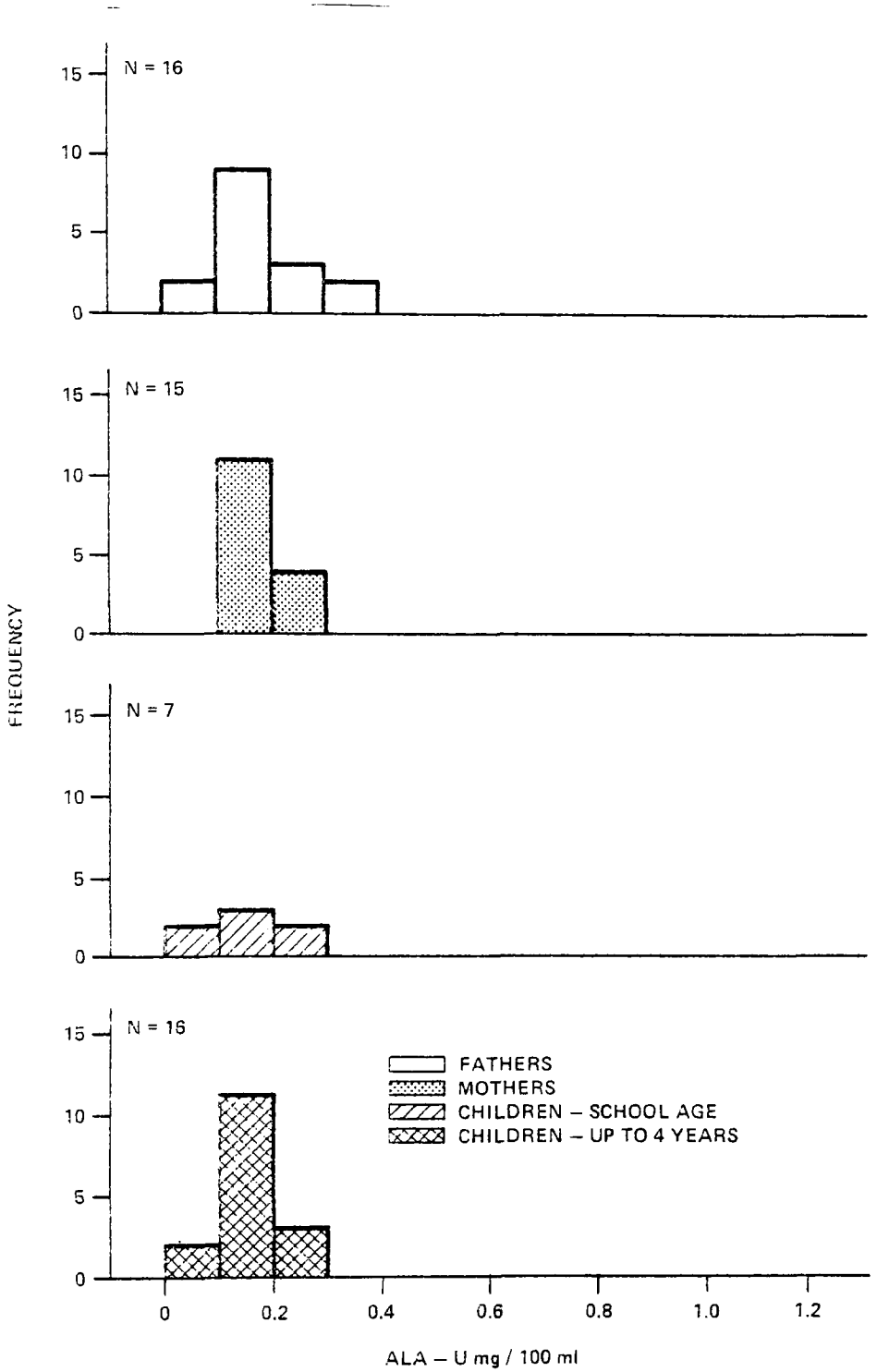


Figure 20. Frequency distribution of δ-aminolevulinic acid in urine (ALA–U mg/100 ml) in control group.

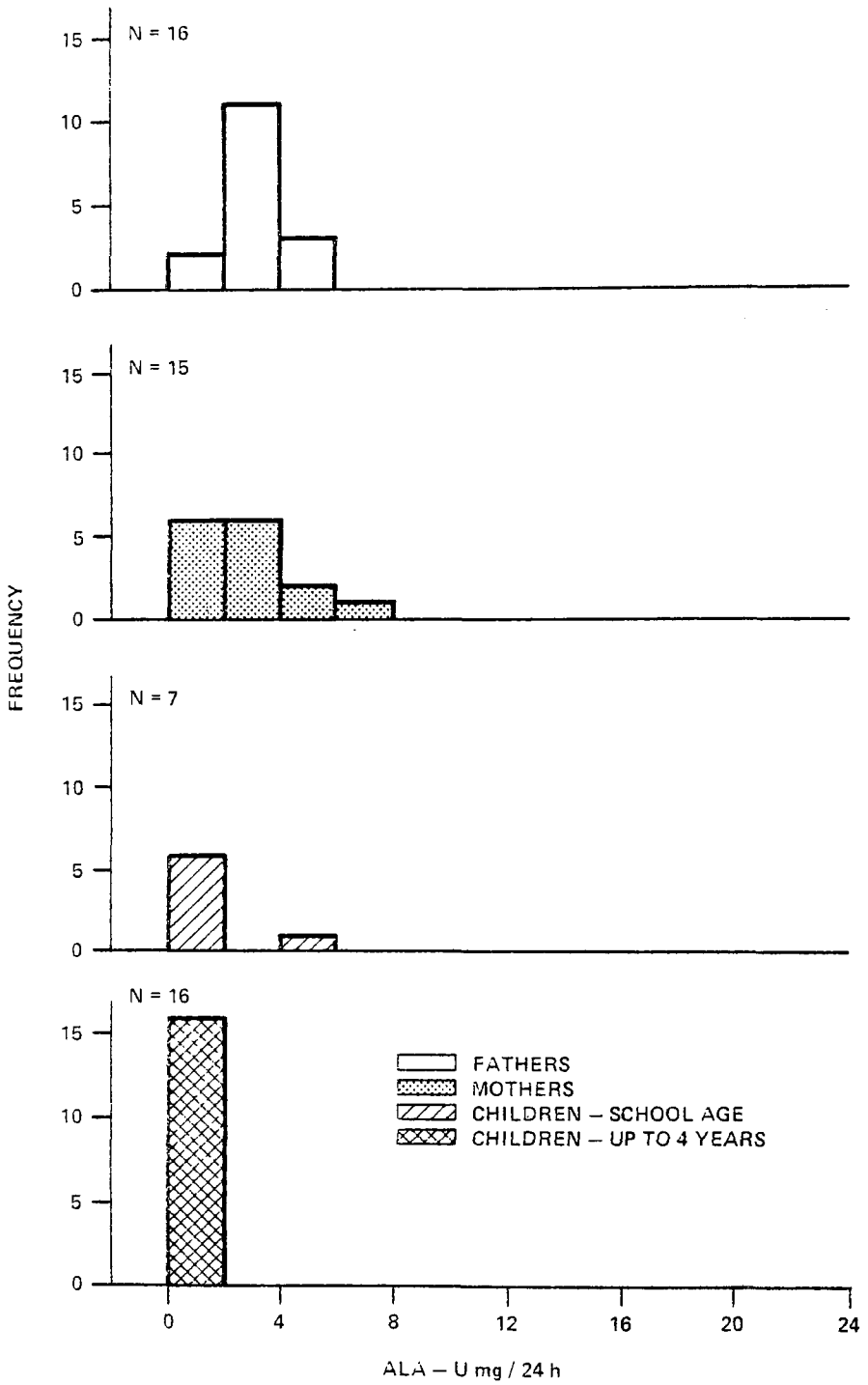


Figure 21. Frequency distribution of δ-aminolevulinic acid in urine (ALA–U mg/24 h) in control group.

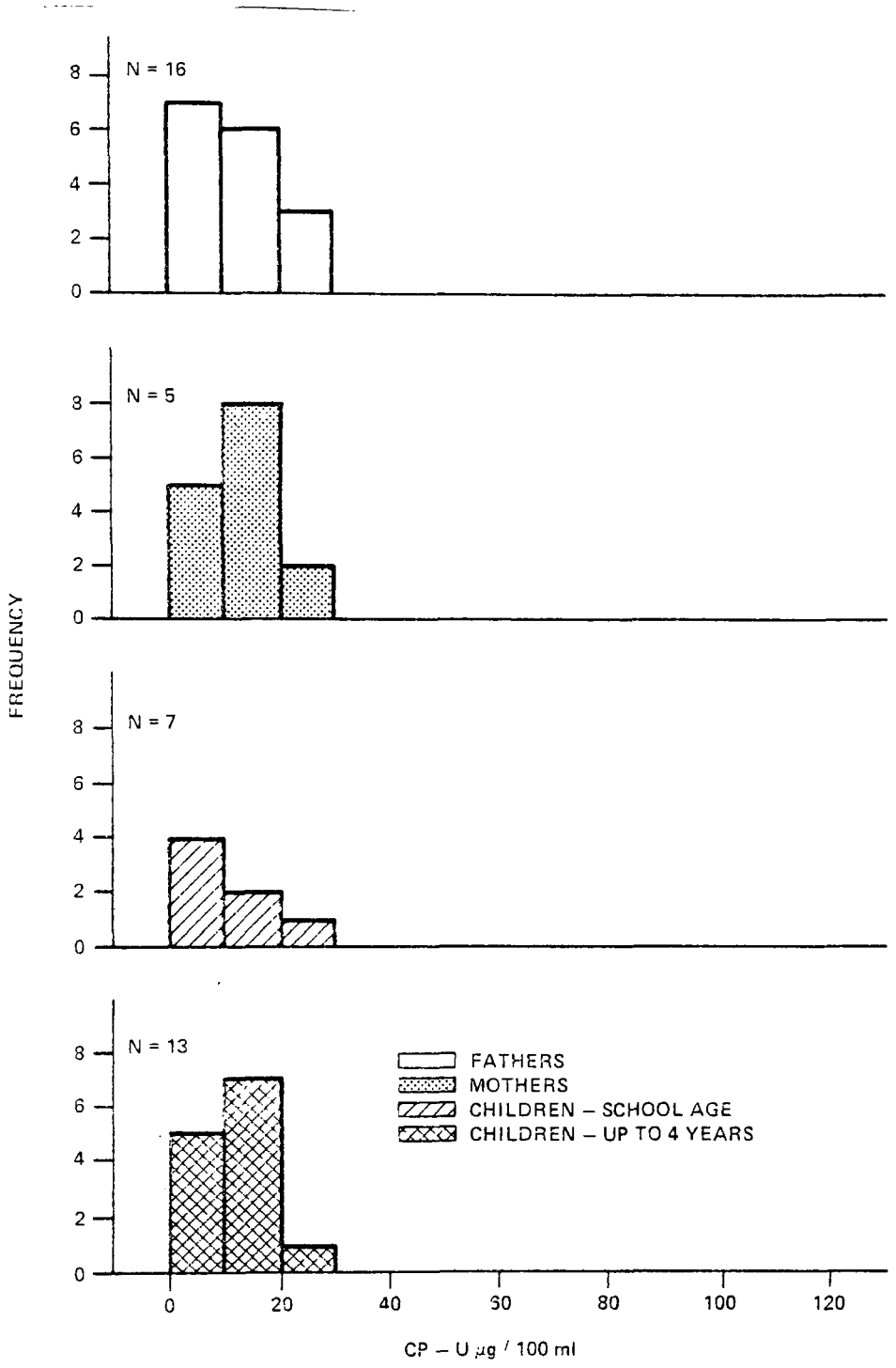


Figure 22. Frequency distribution of coproporphyrin in urine (CP–U µg/100 ml) in control group.

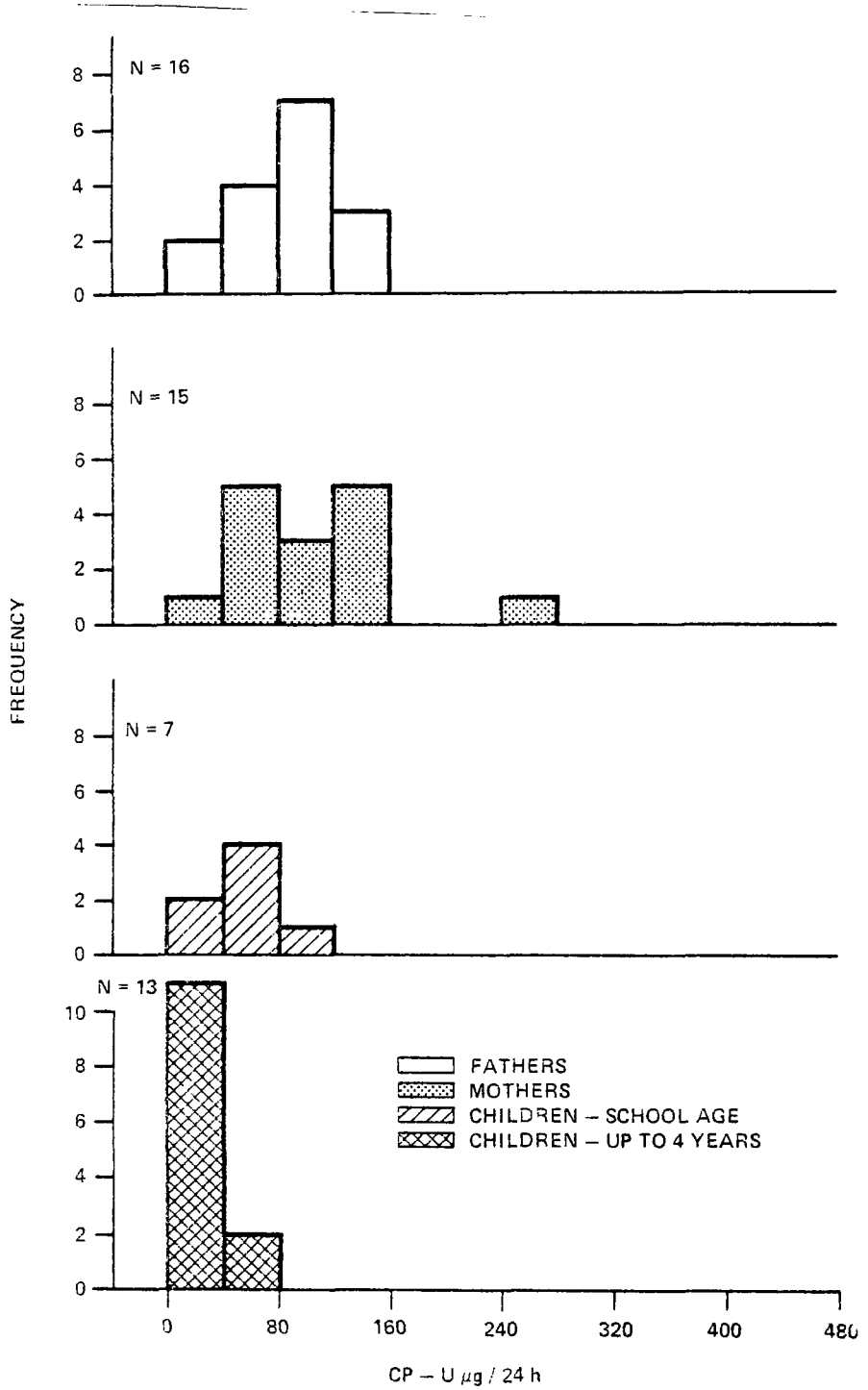


Figure 23. Frequency distribution of coproporphyrin in urine (CP–U µg/24 h) in control group.

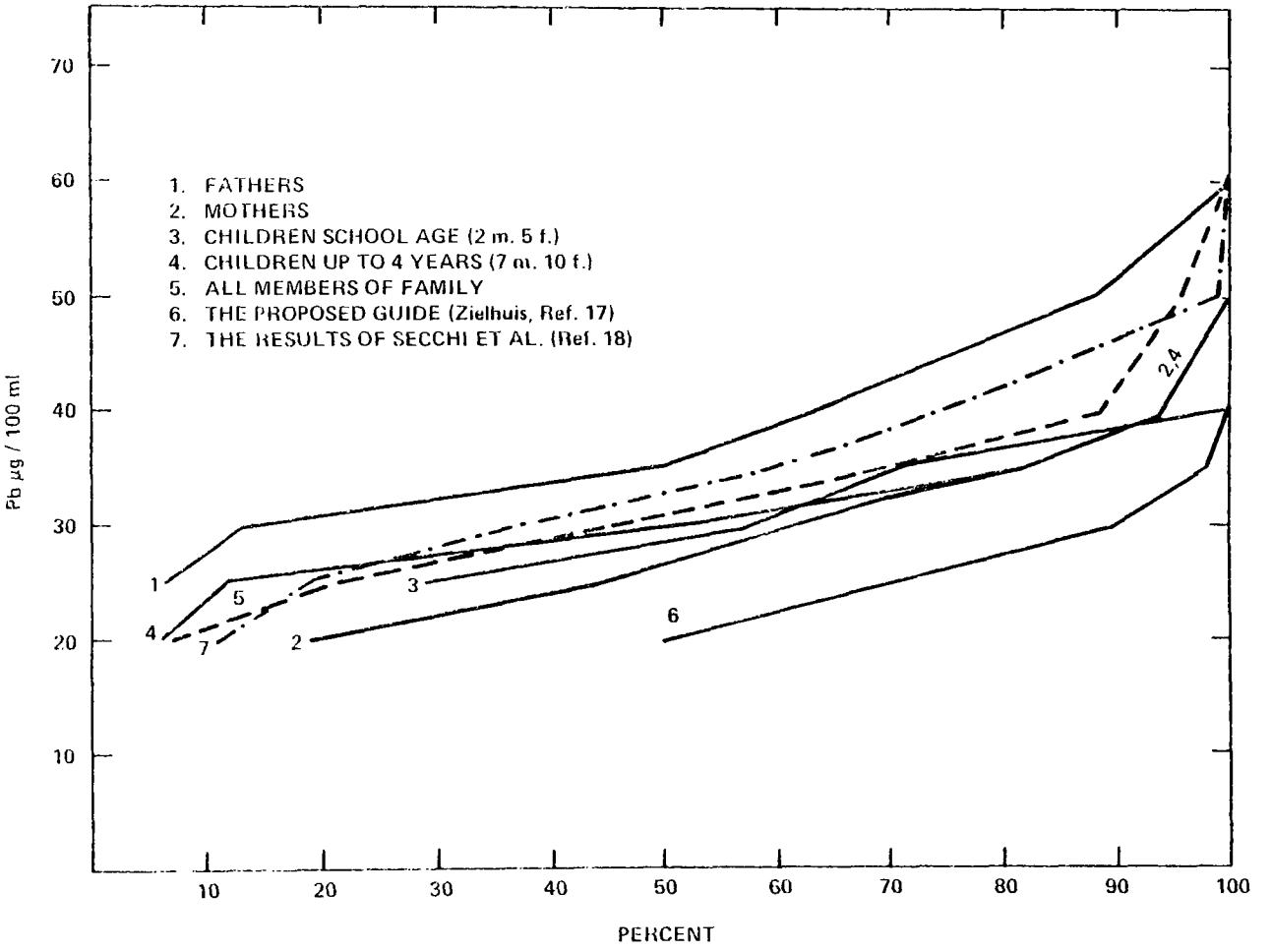


Figure 24. Percentile distribution of Pb–B (µg/100 ml) in control group.

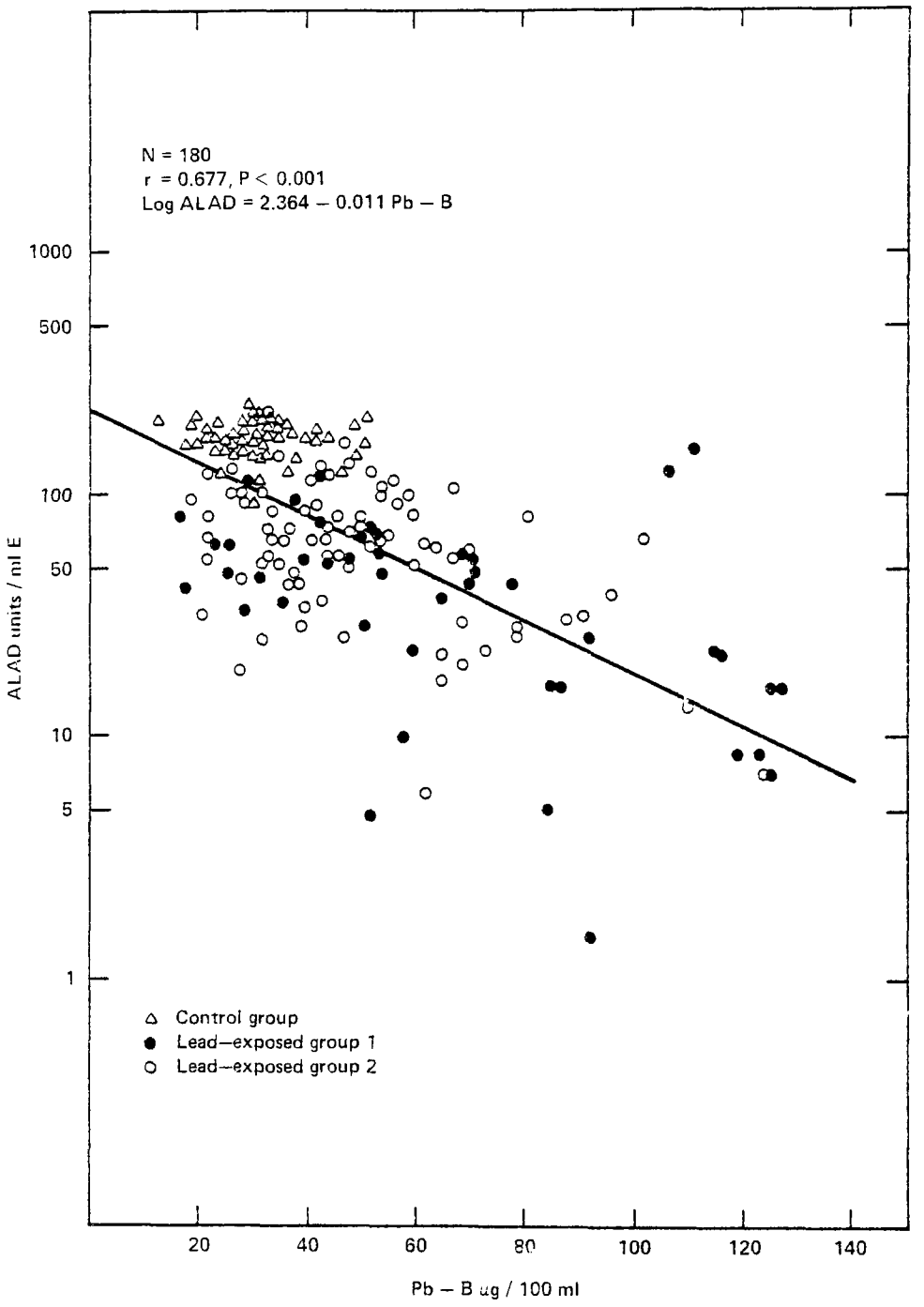


Figure 25. Semilogarithmic correlation in a total study population between δ-aminolevulinic acid dehydratase activity and lead in blood (log ALAD/lin Pb–B).

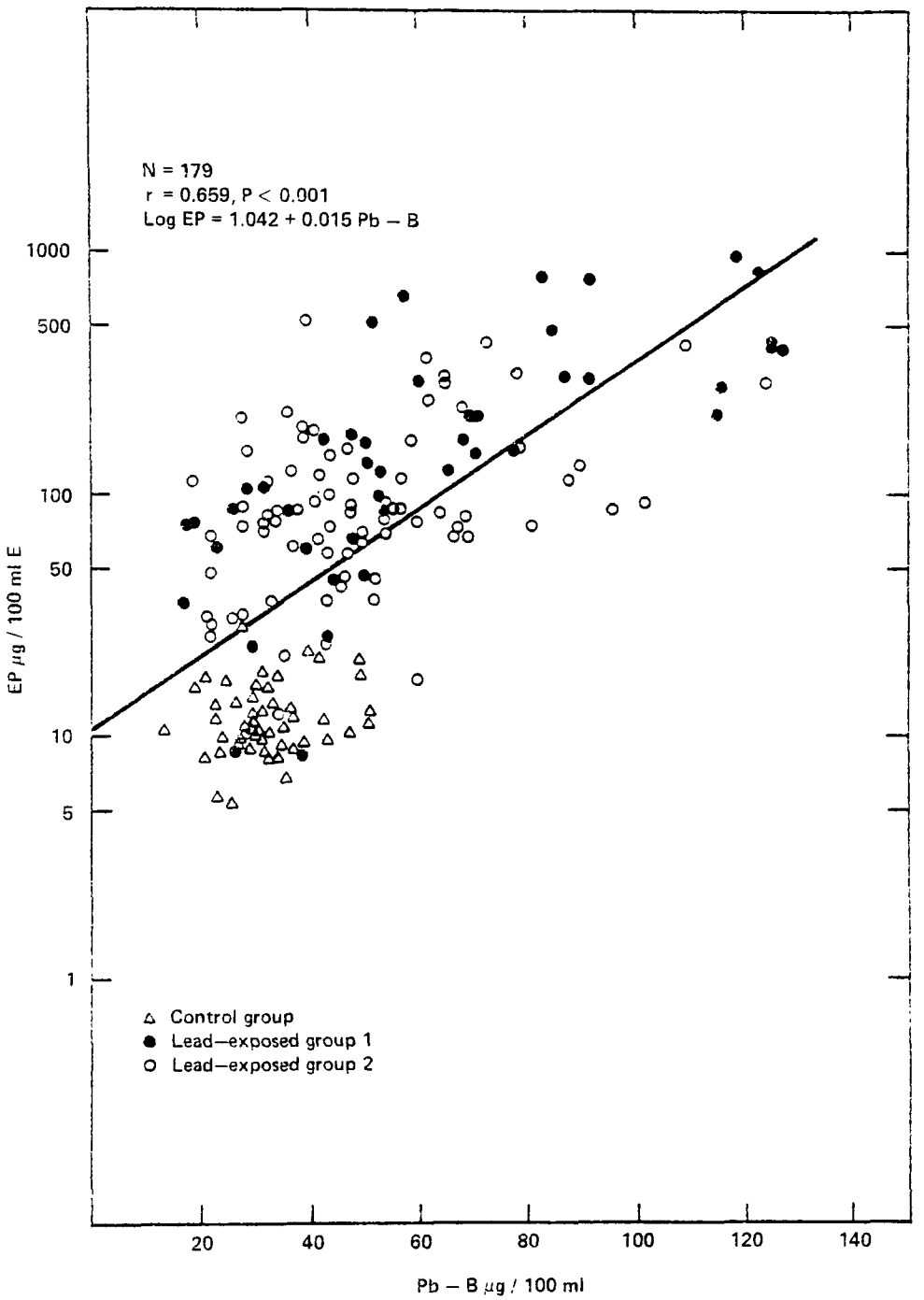


Figure 26. Semilogarithmic correlation in a total study population between erythrocyte protoporphyrin and lead in blood (log EP/lin Pb–B).

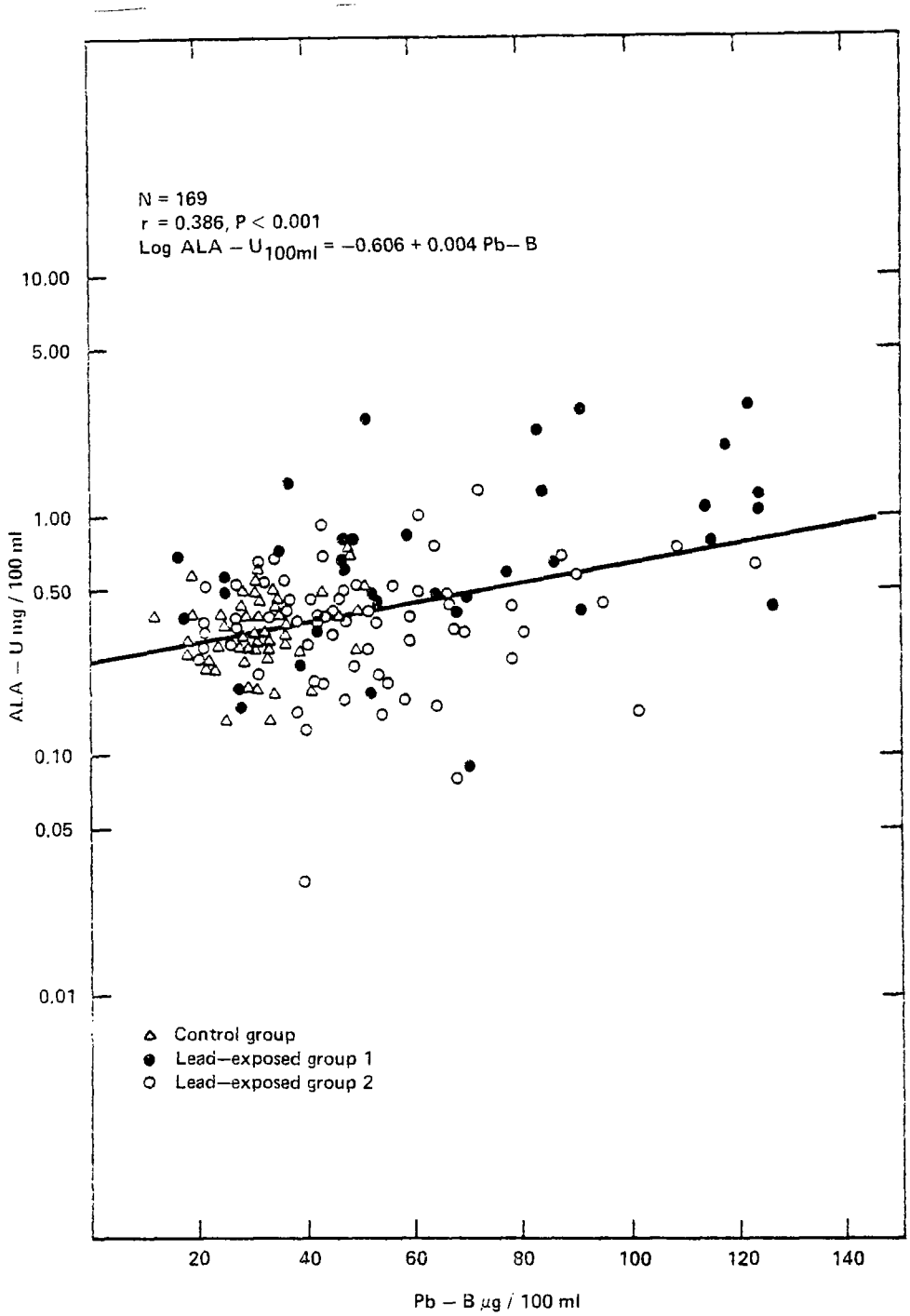


Figure 27. Semilogarithmic correlation in a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/100 ml/lin Pb–B).

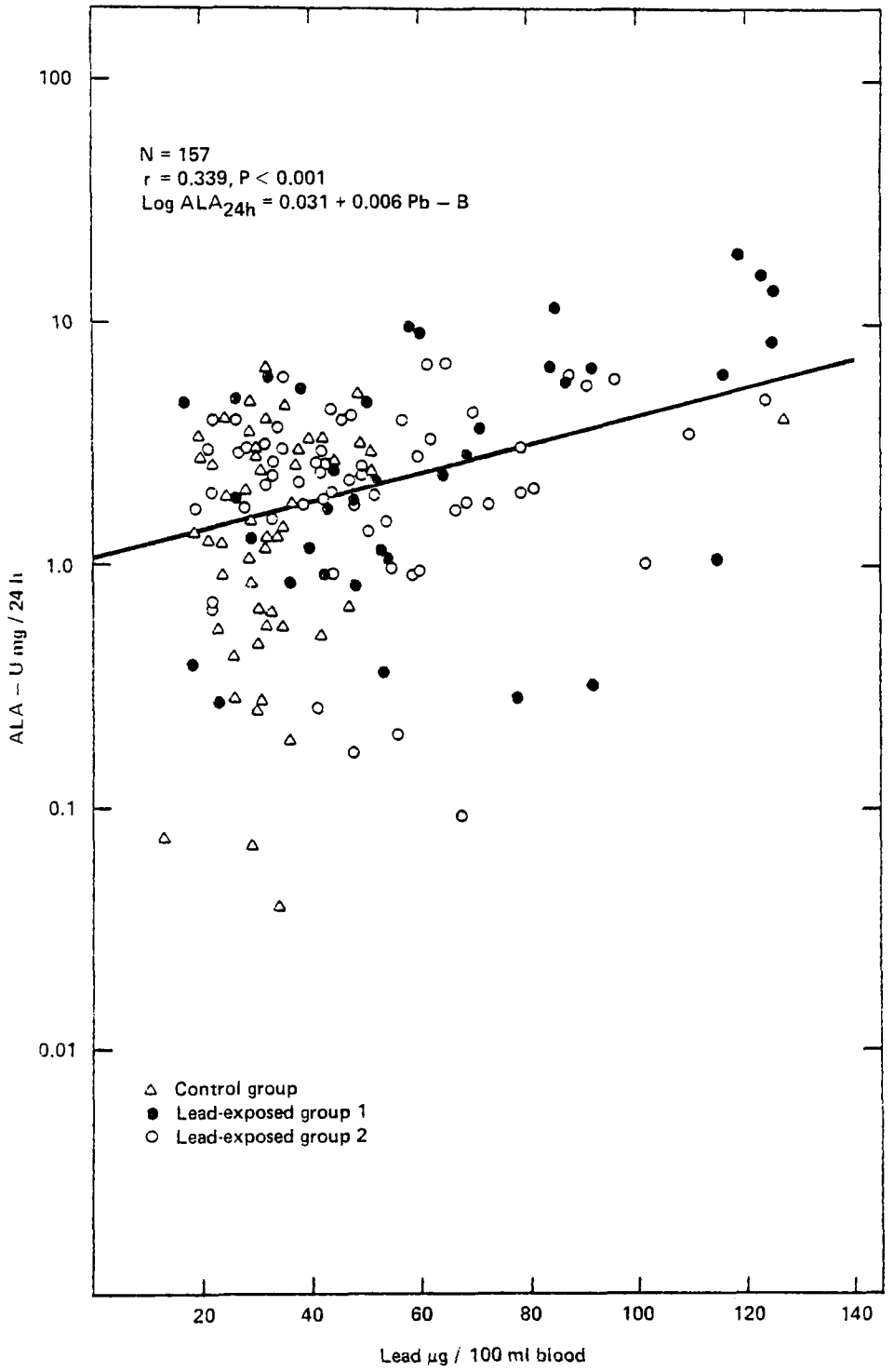


Figure 28. Semilogarithmic correlation in a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/24 h/lin Pb–B).

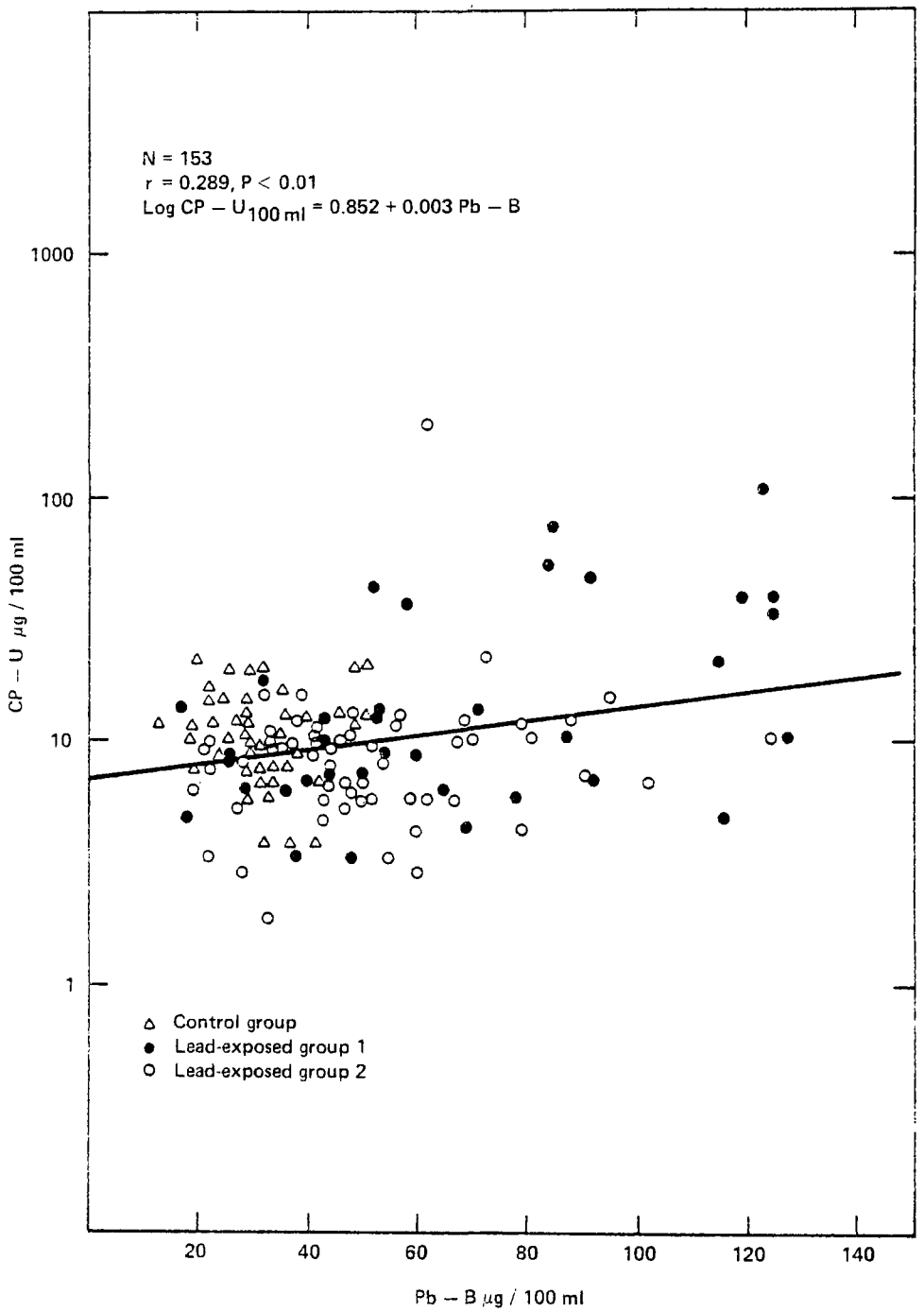


Figure 29. Semilogarithmic correlation in a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/100 ml/lin Pb–B).

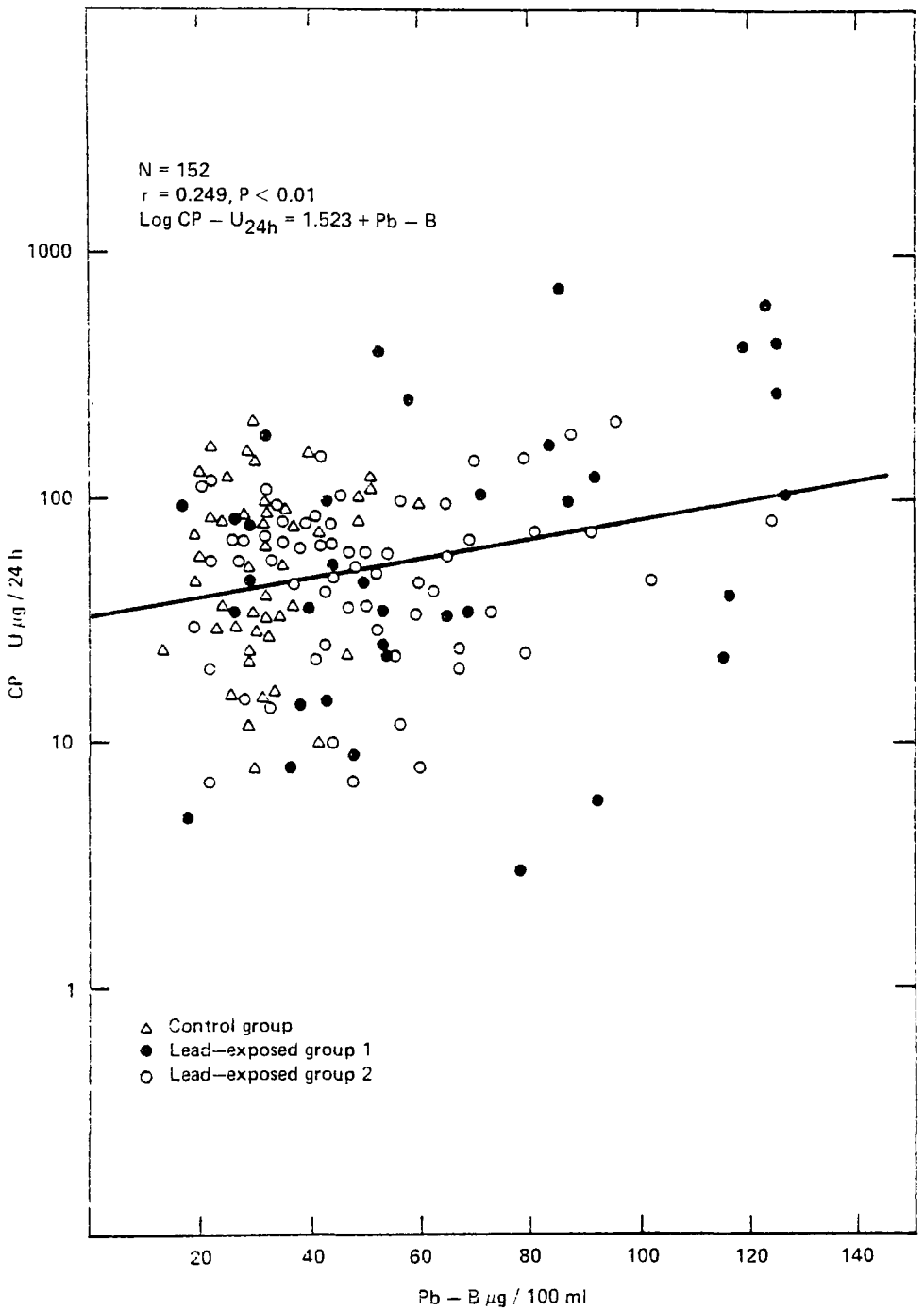


Figure 30. Semilogarithmic correlation in a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/24 h/lin Pb–B).

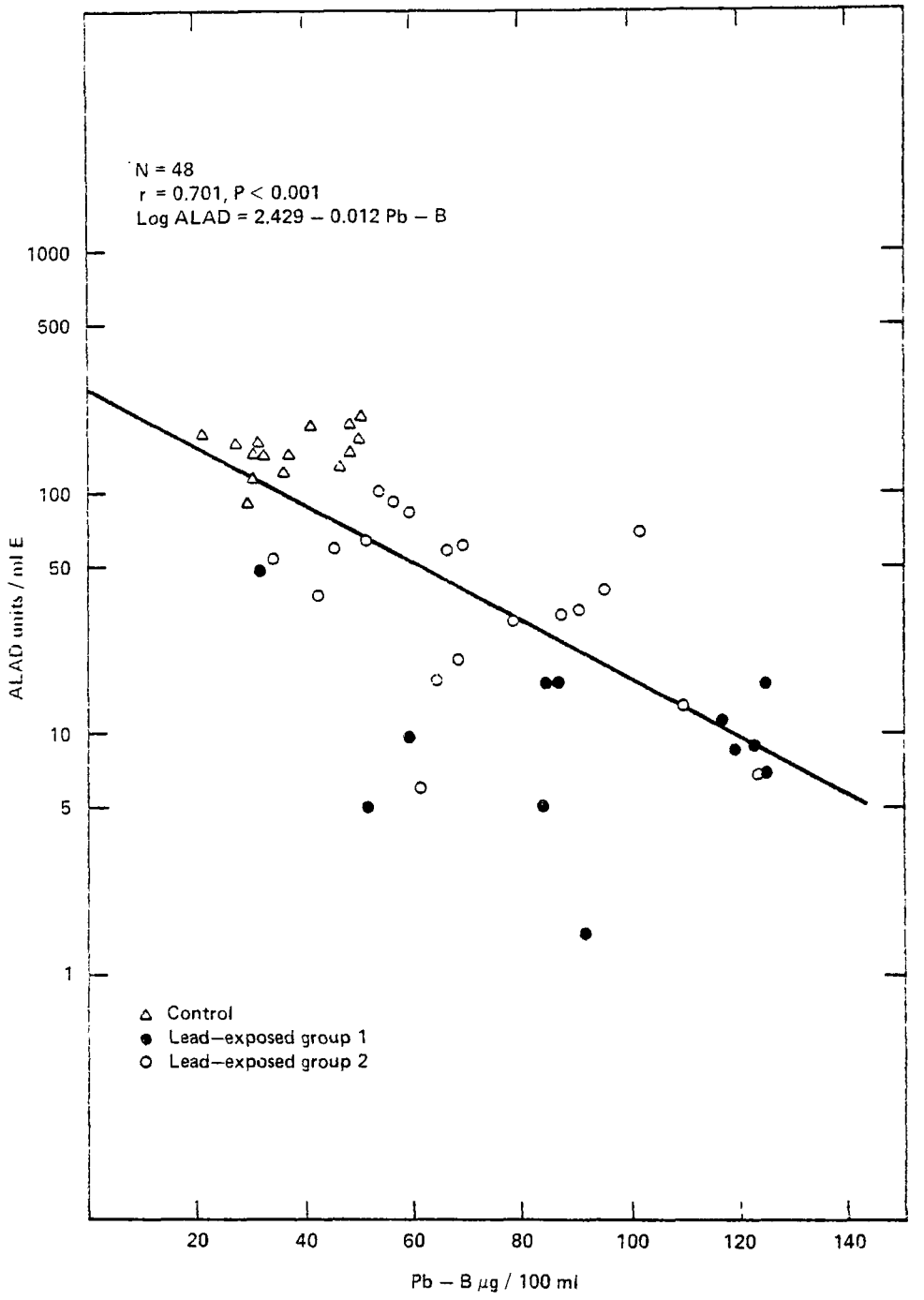


Figure 31. Semilogarithmic correlation in fathers of a total study population between δ-aminolevulinic acid dehydratase activity and lead in blood (log ALAD/lin Pb–B).

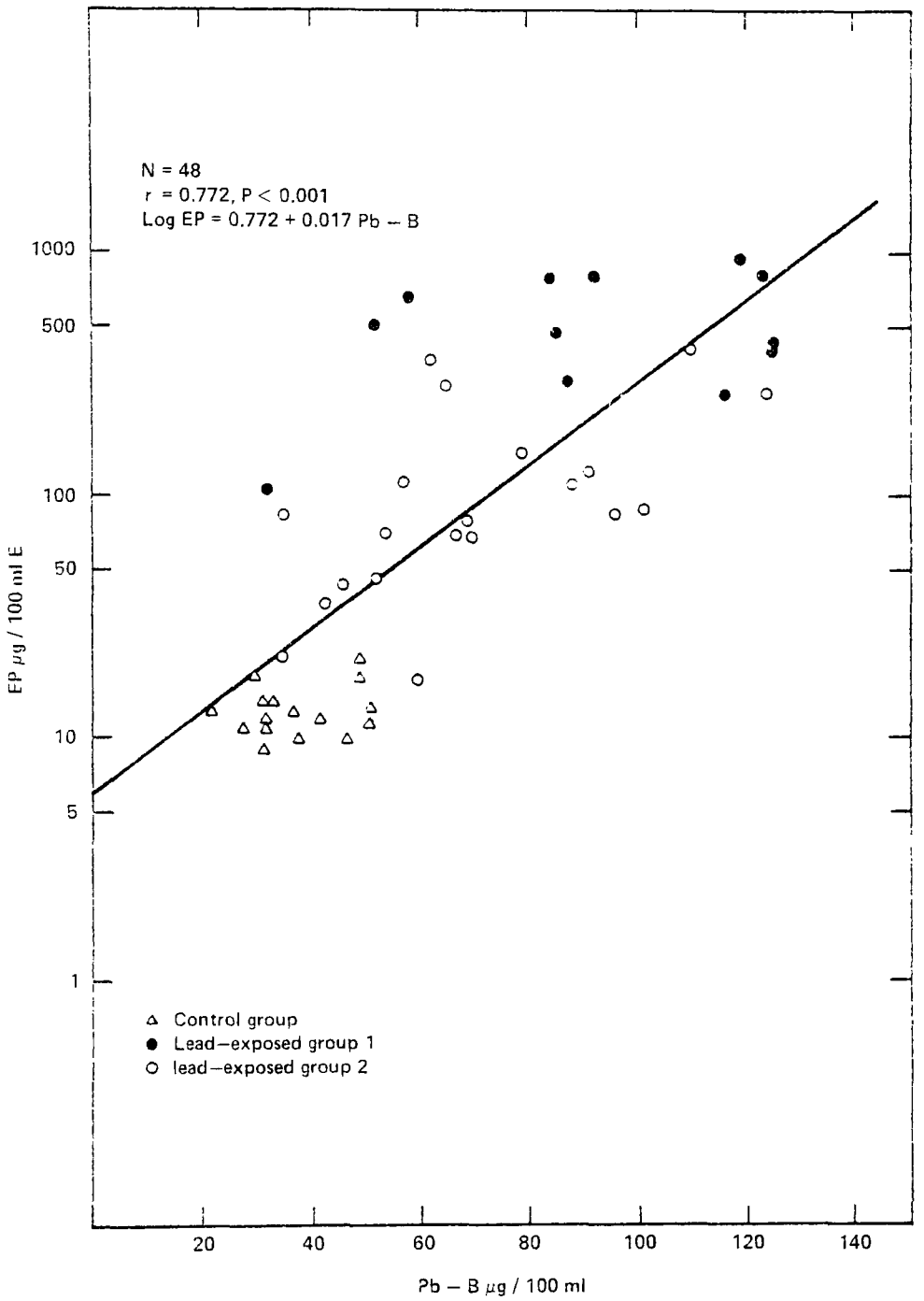


Figure 32. Semilogarithmic correlation in fathers of a total study population between erythrocyte protoporphyrin and lead in blood (log EP/lin Pb–B).

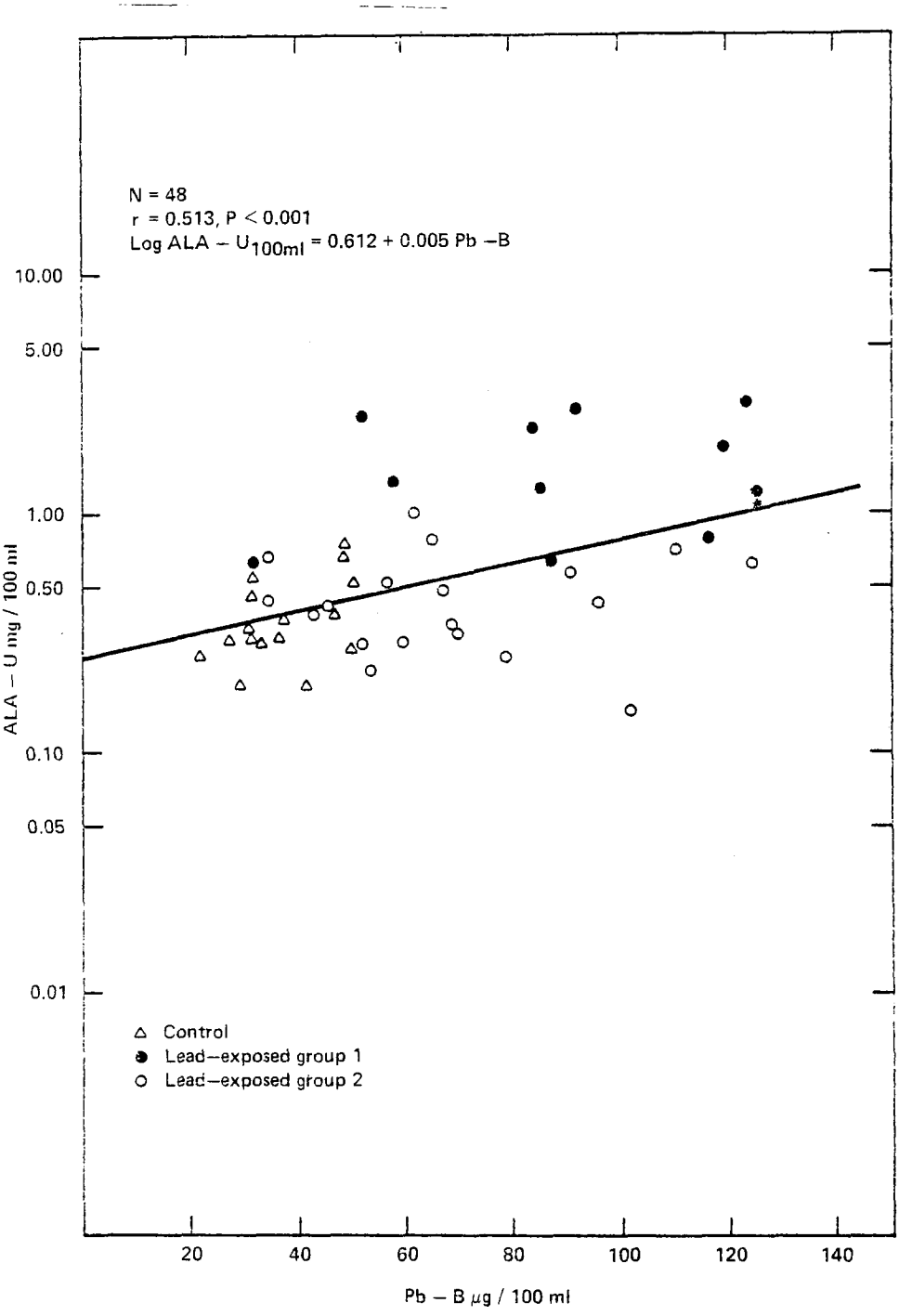


Figure 33. Semilogarithmic correlation in fathers of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/100 ml/lin Pb–B).

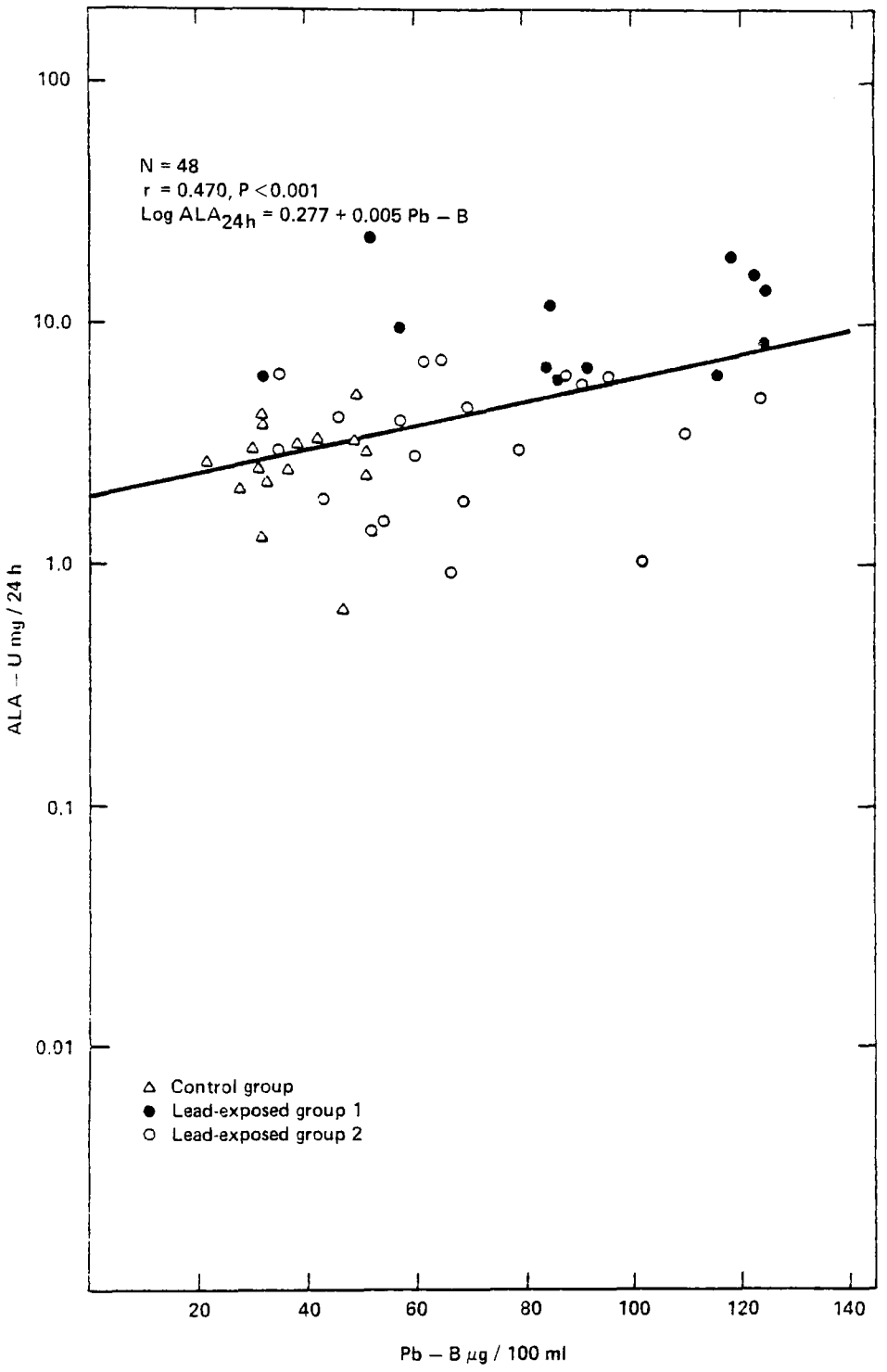


Figure 34. Semilogarithmic correlation in fathers of a total study population between δ-amino-levulinic acid in urine and lead in blood (log ALA–U mg/24 h/lin Pb–B).

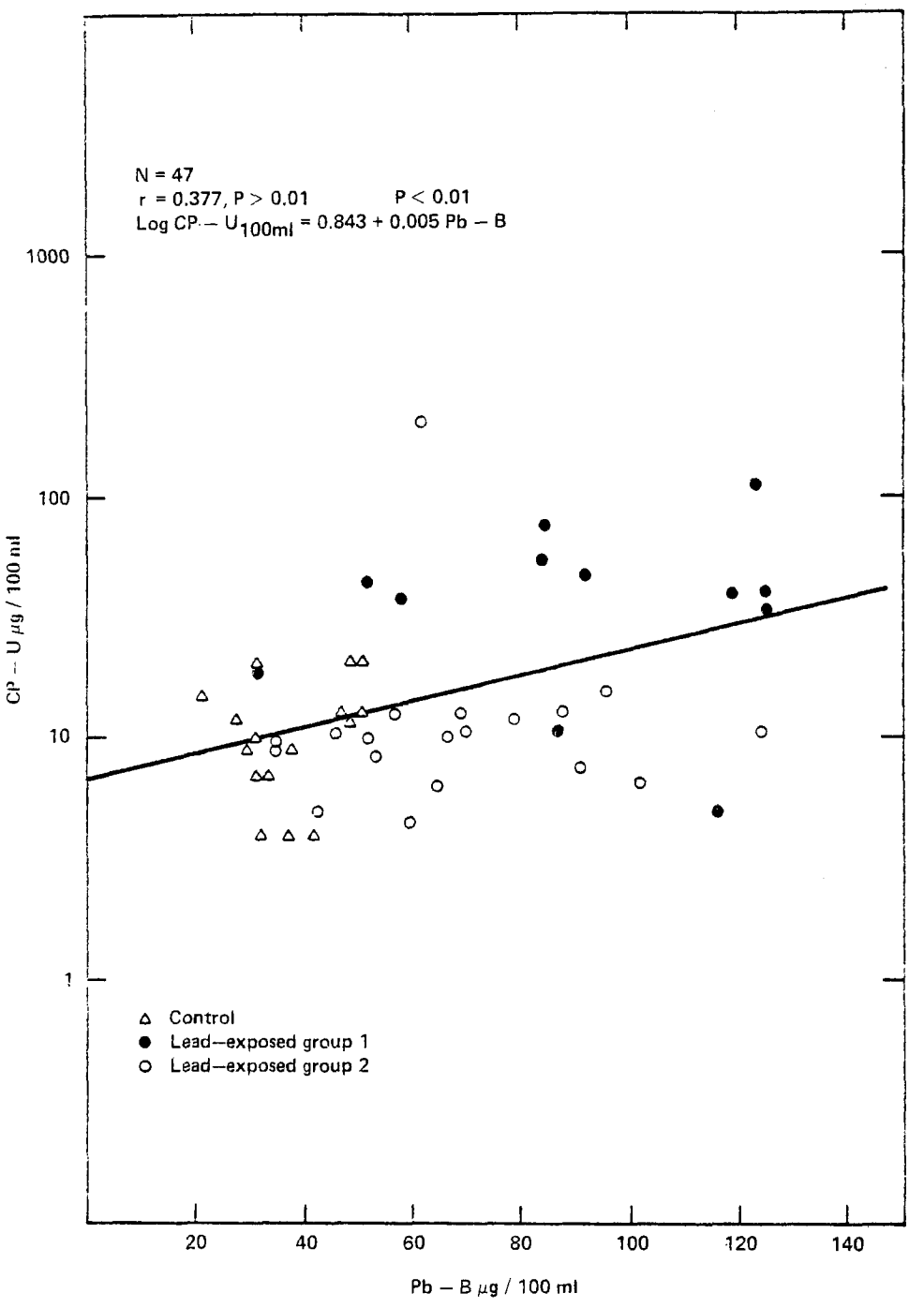


Figure 35. Semilogarithmic correlation in fathers of a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/100 ml/lin Pb–B).

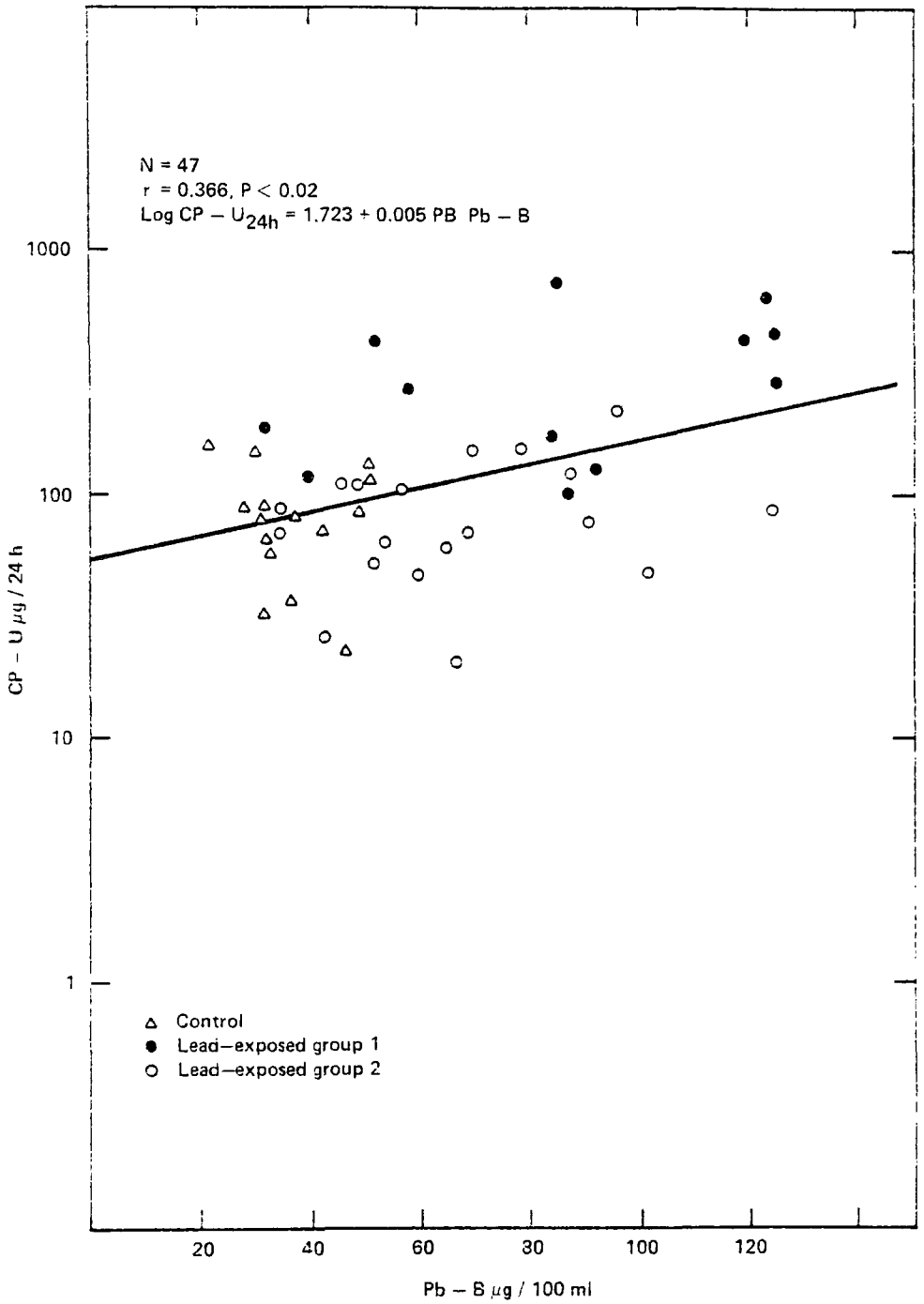


Figure 36. Semilogarithmic correlation in fathers of a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/24 h/lin Pb–B).

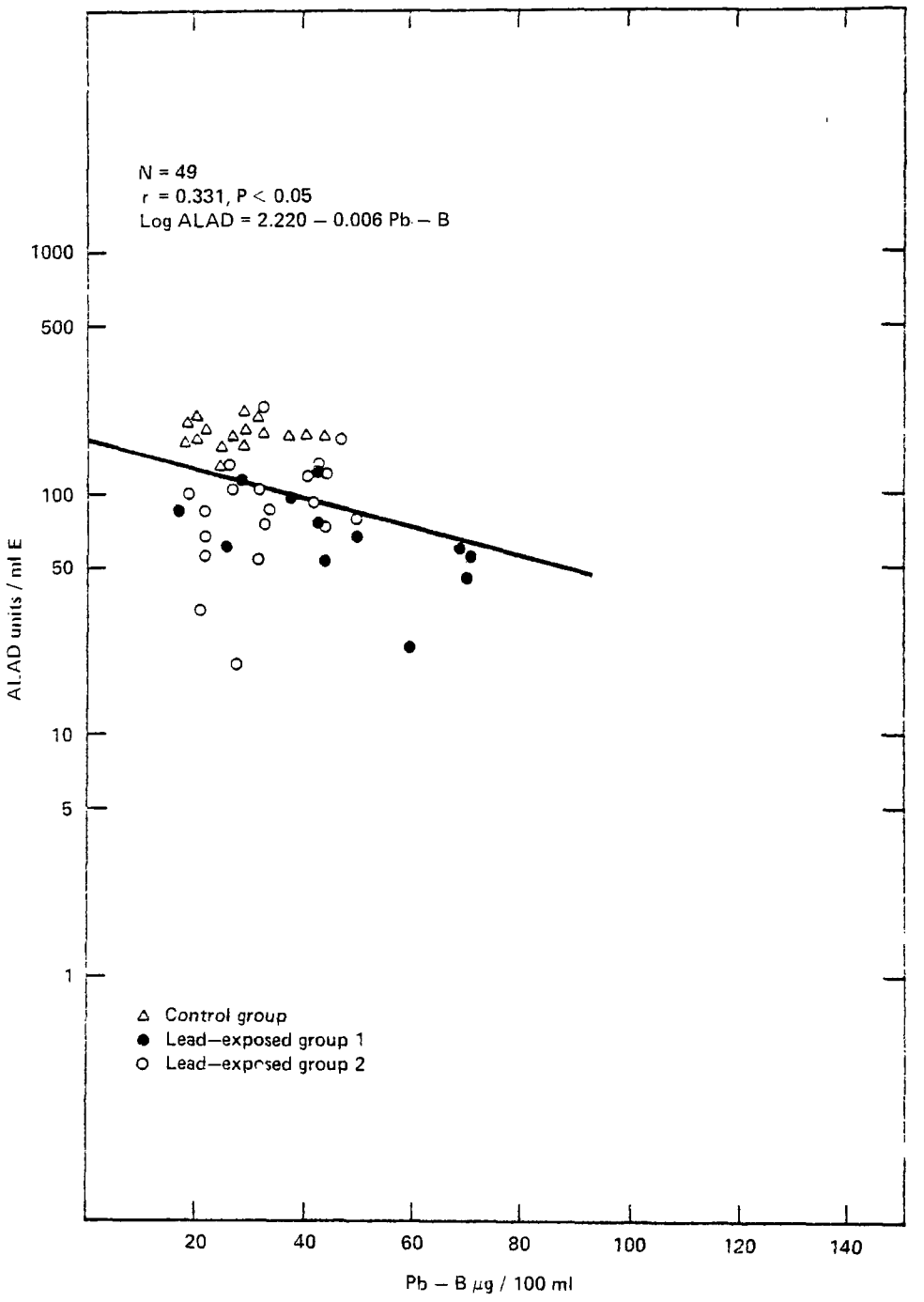


Figure 37. Semilogarithmic correlation in mothers of a total study population between δ-amino-levulinic acid dehydratase activity and lead in blood (log ALAD/lin Pb–B).

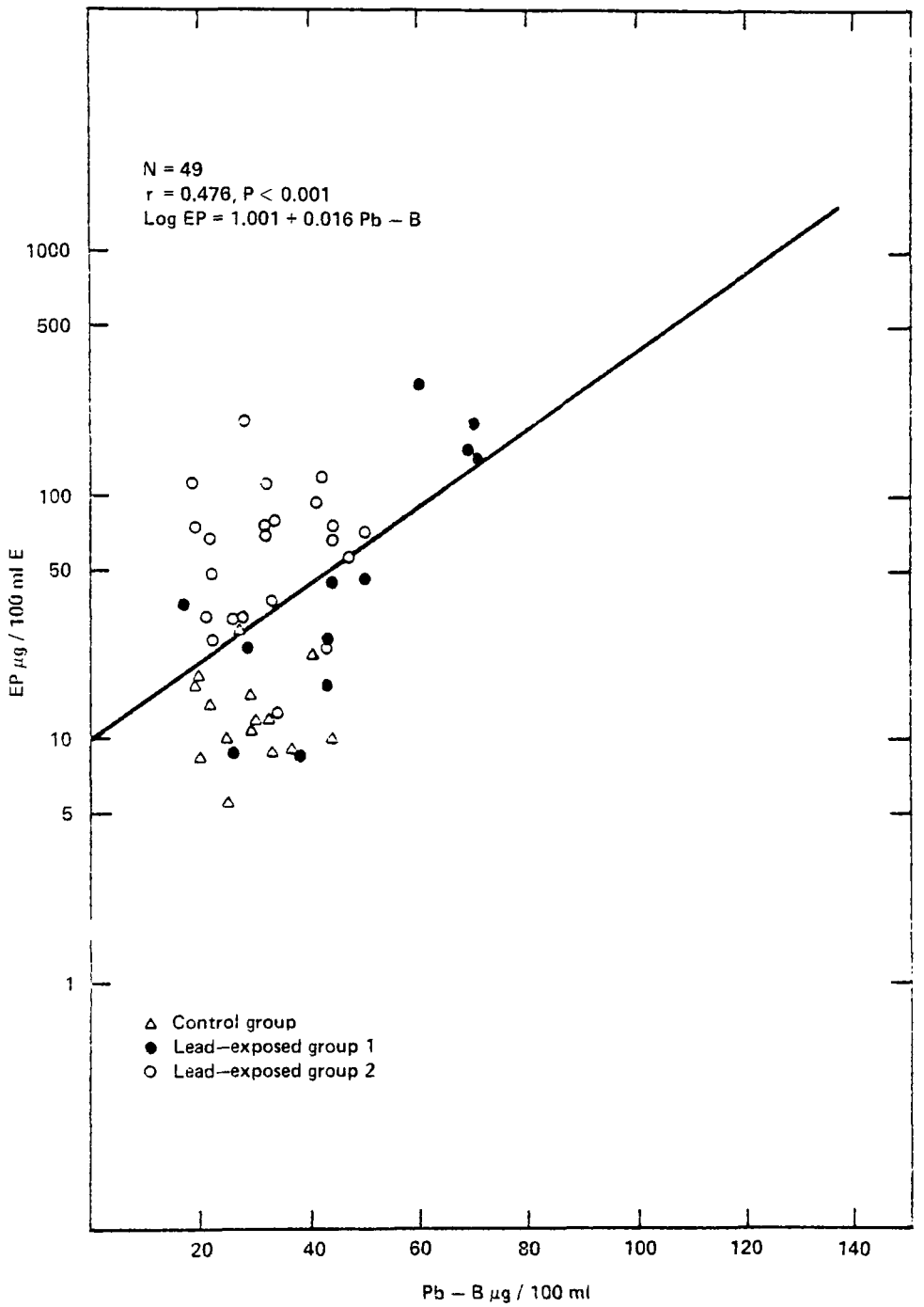


Figure 38. Semilogarithmic correlation in mothers of a total study population between erythrocyte protoporphyrin and lead in blood (log EP/lin Pb–B).

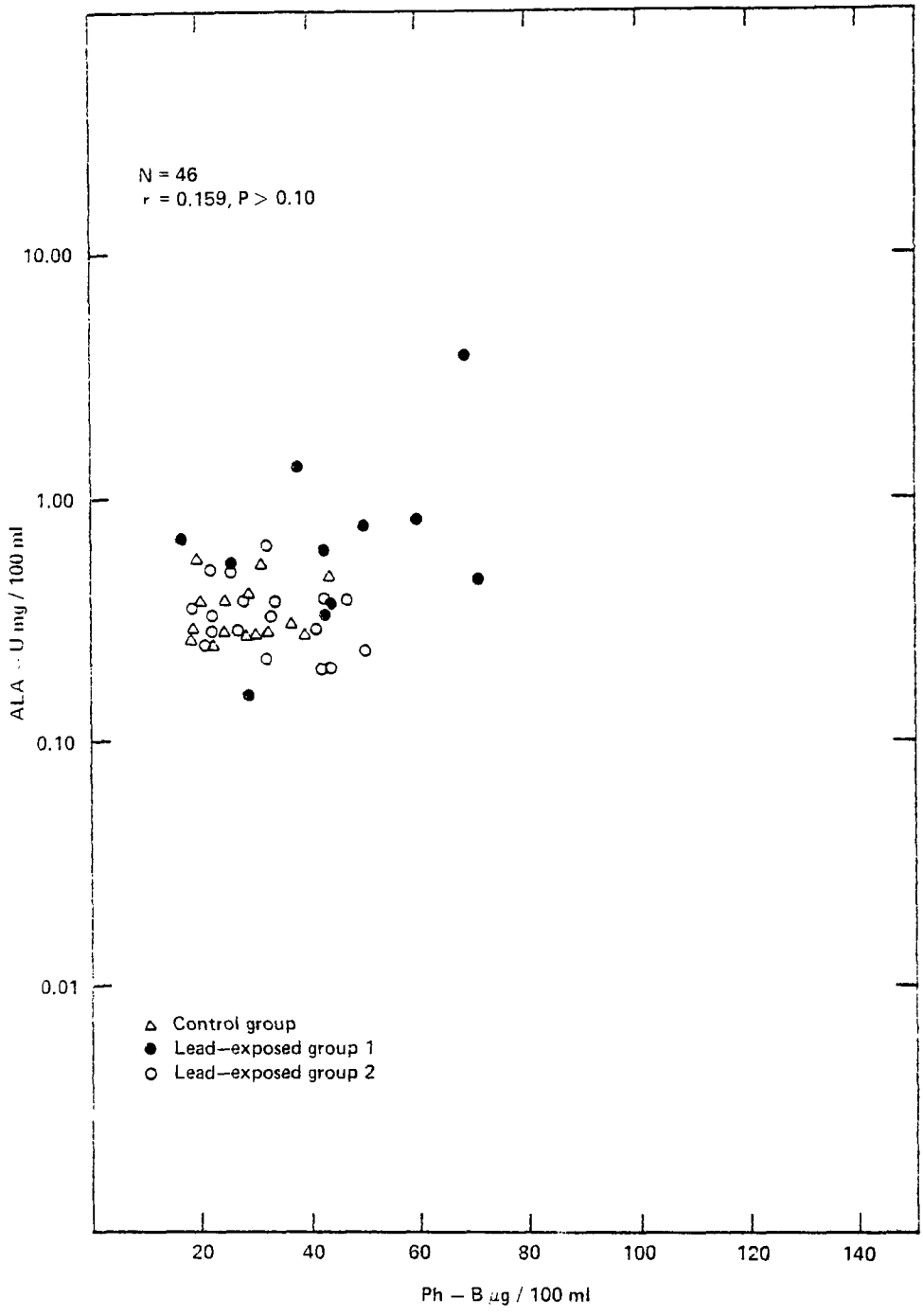


Figure 39. Semilogarithmic correlation in mothers of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/100 ml/lin Pb–B).

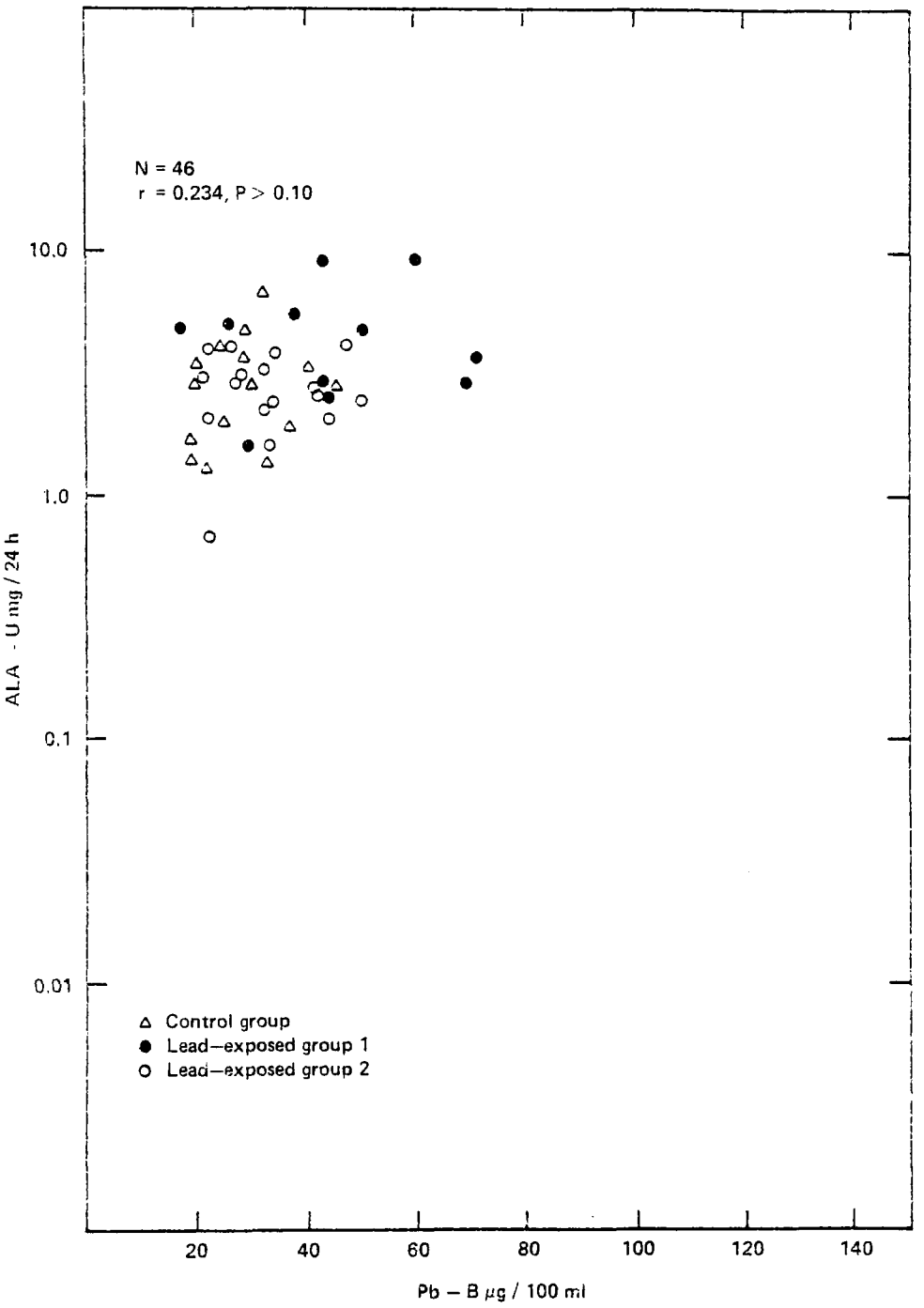


Figure 40. Semilogarithmic correlation in mothers of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/24 h/lin Pb–B).

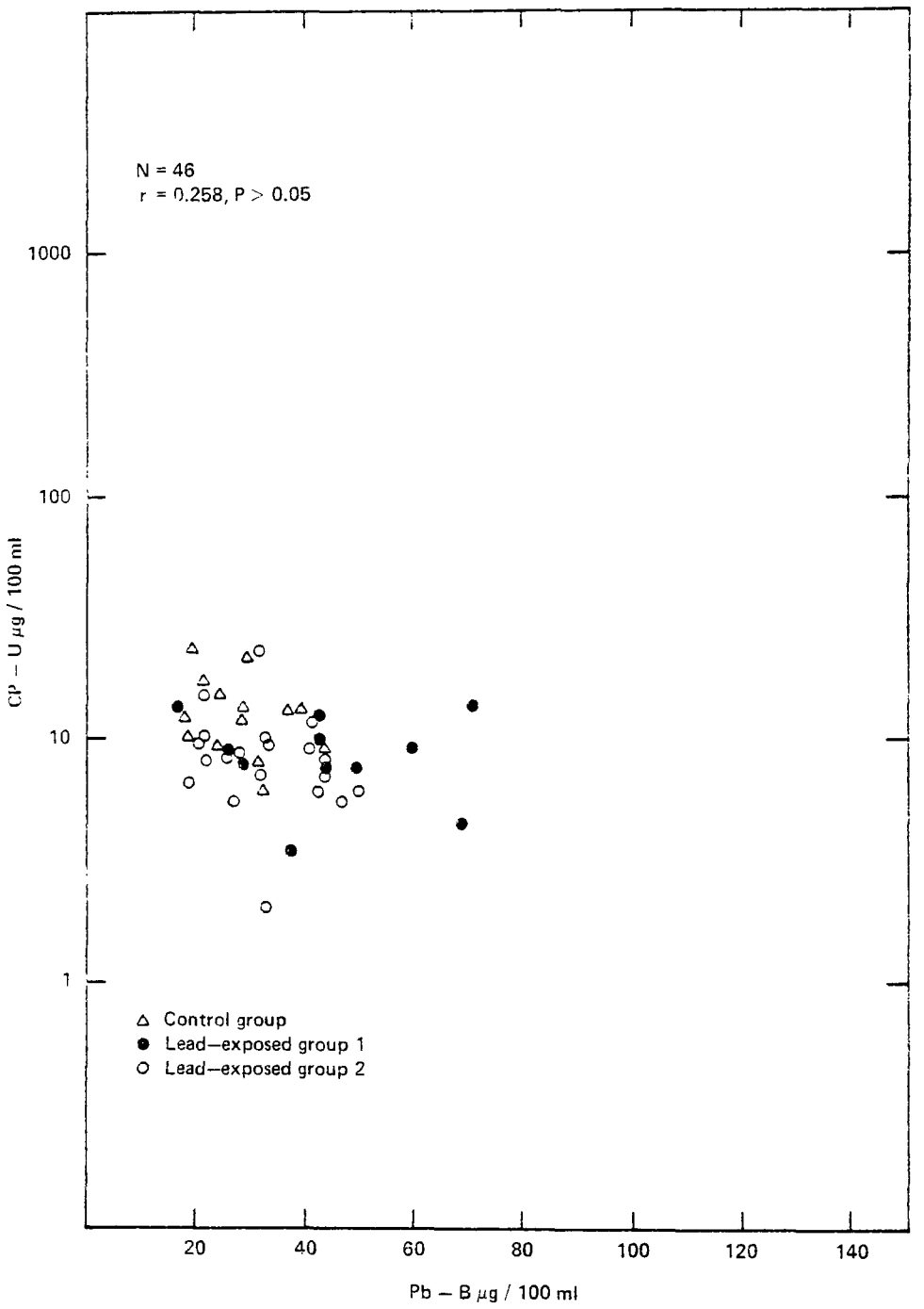


Figure 41. Semilogarithmic correlation in mothers of a total study population between coproporphyrin in urine and lead in blood (log CP – U µg/100 ml/lin Pb–B).

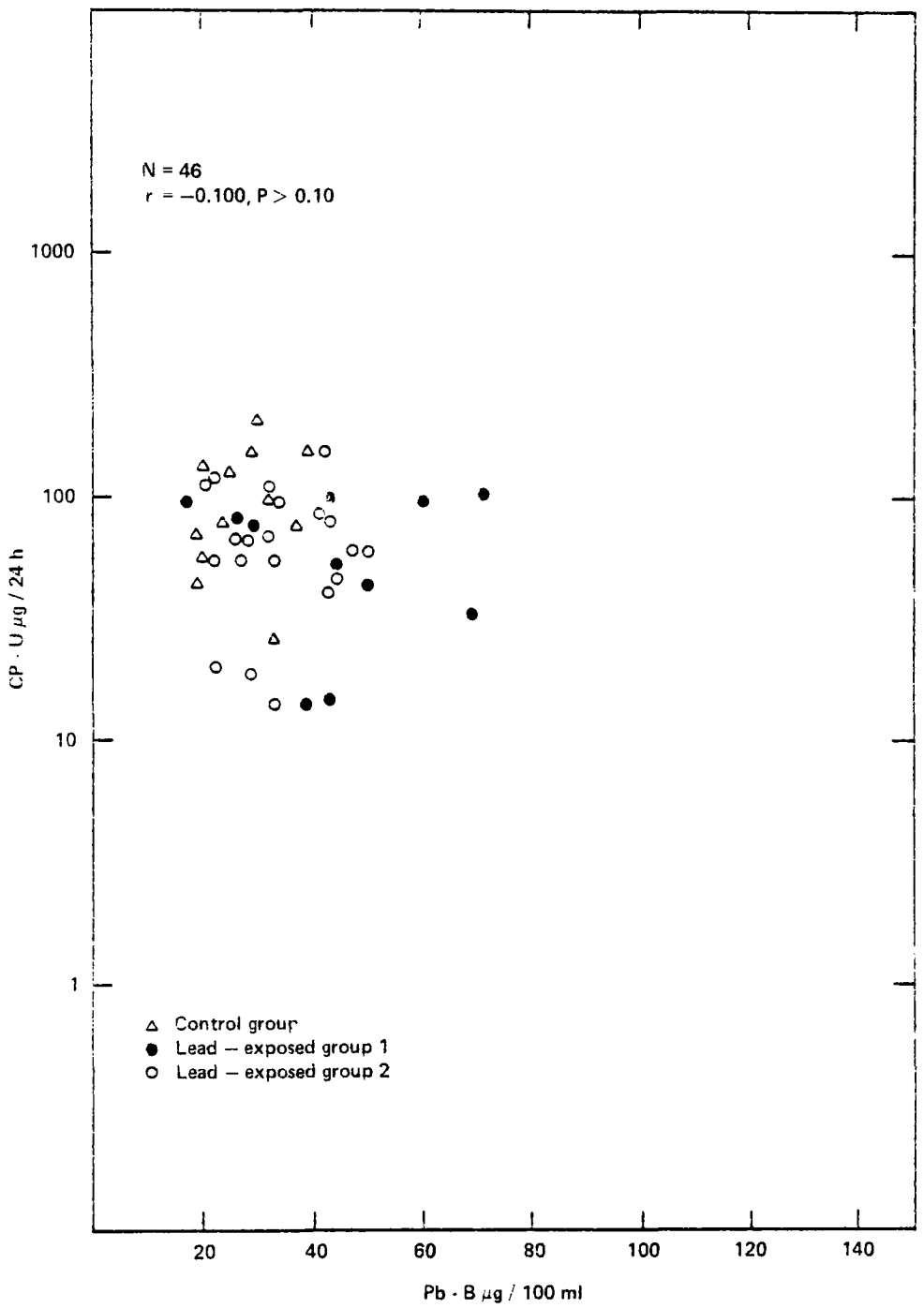


Figure 42. Semilogarithmic correlation in mothers of a total study population between coproporphyrin in urine and lead in blood (log CP—U µg/24 h/lin Pb—B).

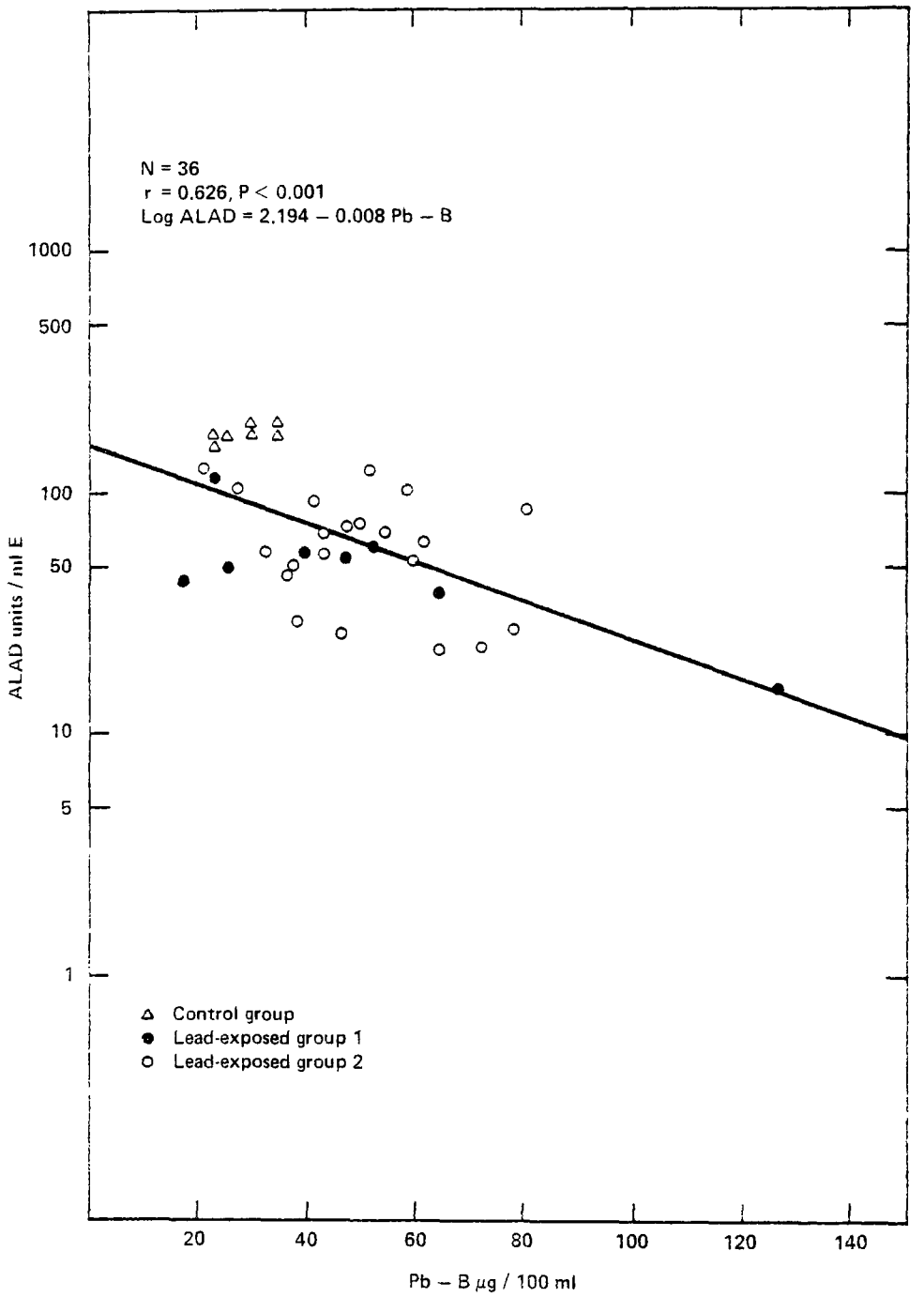


Figure 43. Semilogarithmic correlation in children of school age of a total study population between μ-aminolevulinic acid dehydratase activity and lead in blood (log ALAD/ lin Pb–B).

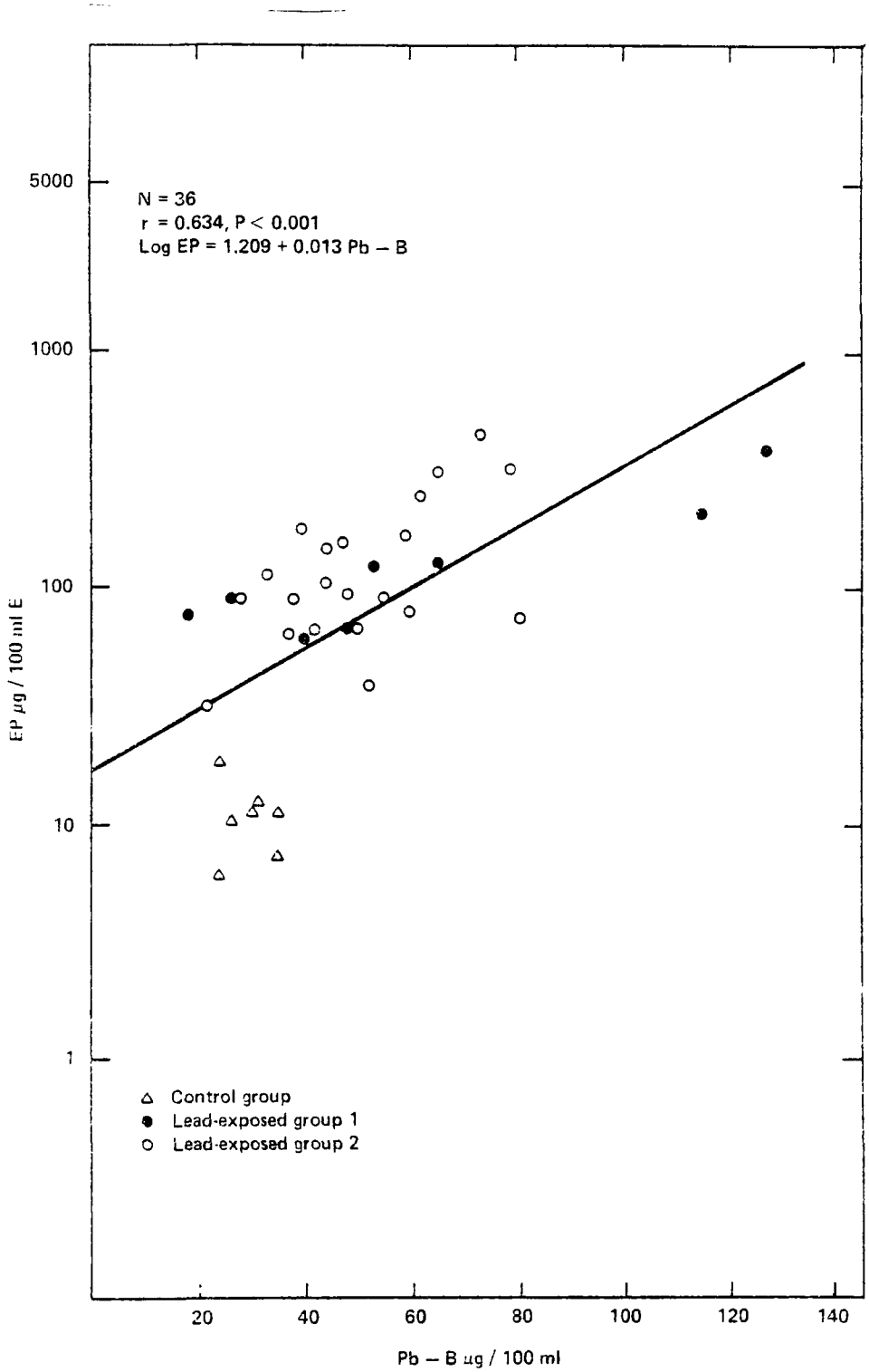


Figure 44. Semilogarithmic correlation in children of school age of a total study population between erythrocyte protoporphyrin and lead in blood (log DP/lin Pn–B).

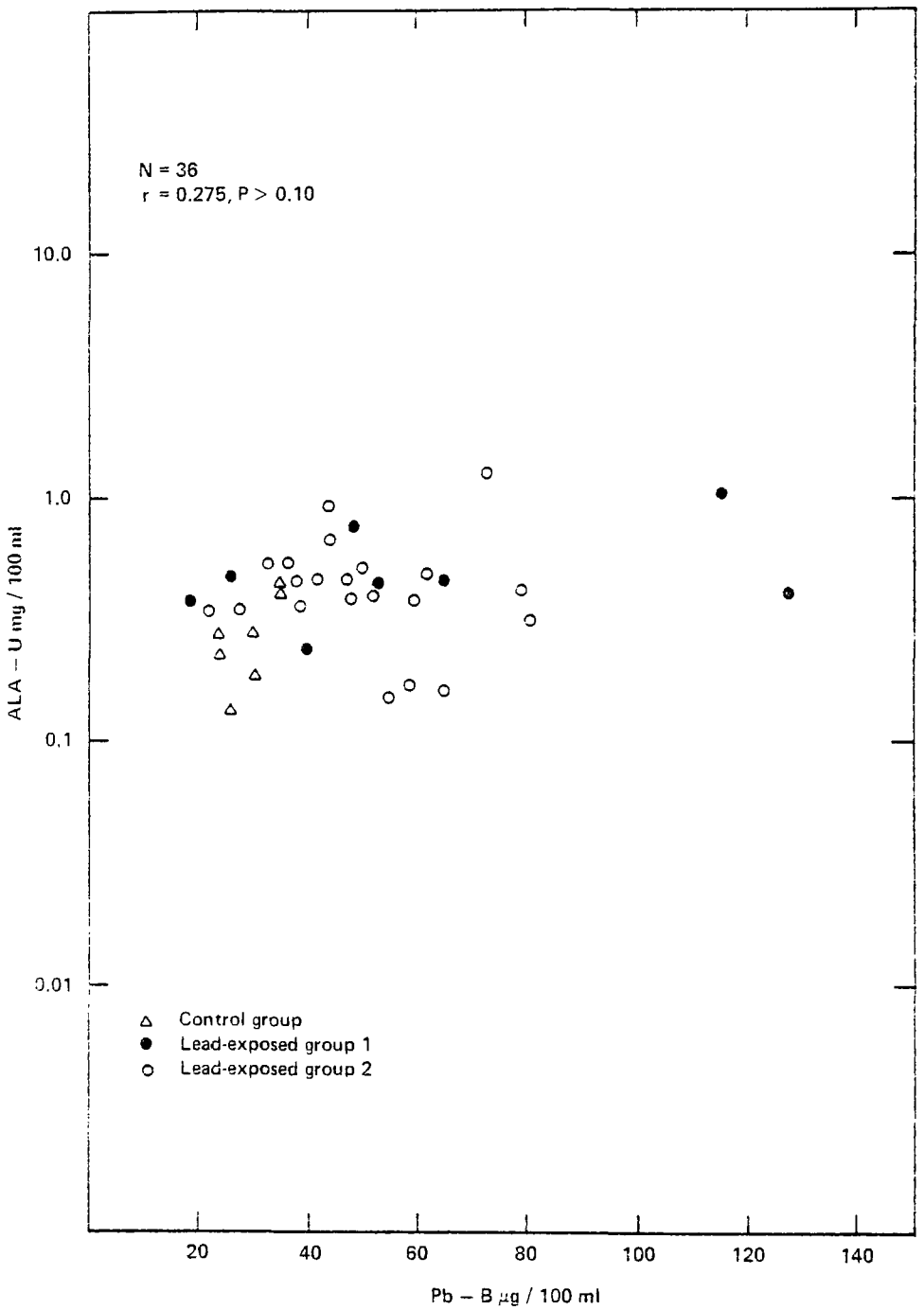


Figure 45. Semilogarithmic correlation in children of school age of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/100ml/ lin Pb–B).

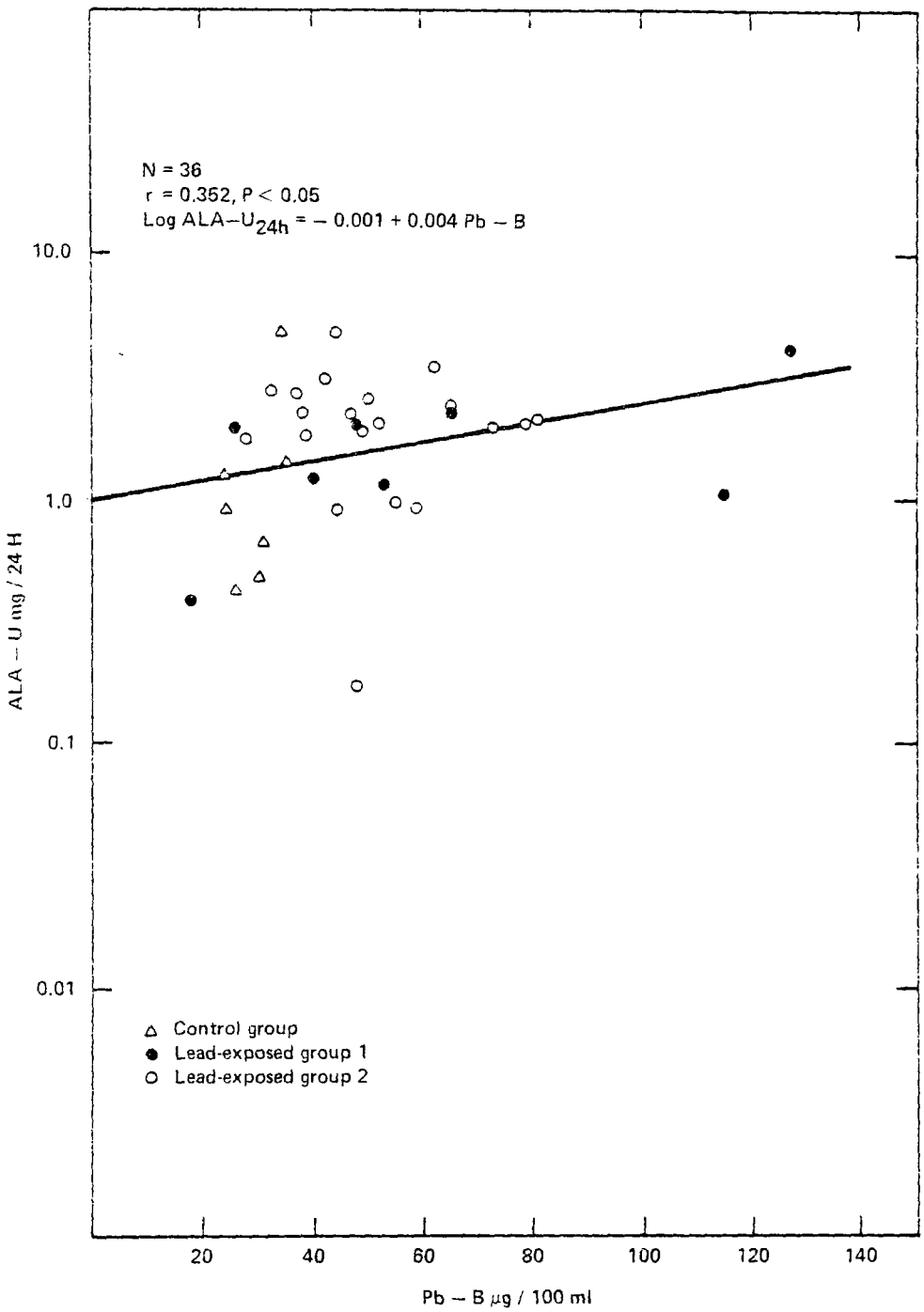


Figure 46. Semilogarithmic correlation in children of school age of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/24h/lin Pb–B).

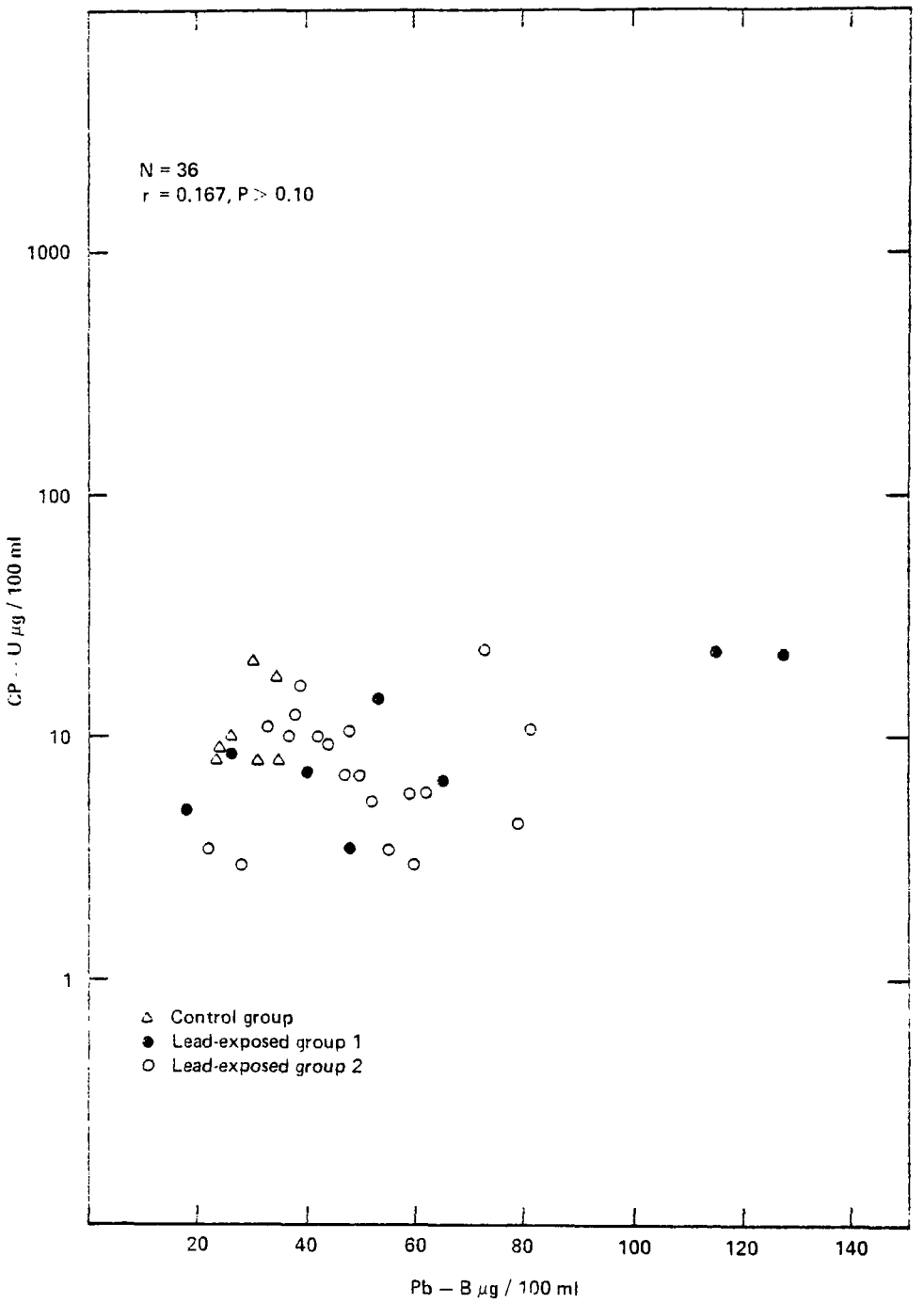


Figure 47. Semilogarithmic correlation in children of school age of a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/100 ml/lin Pb–B).

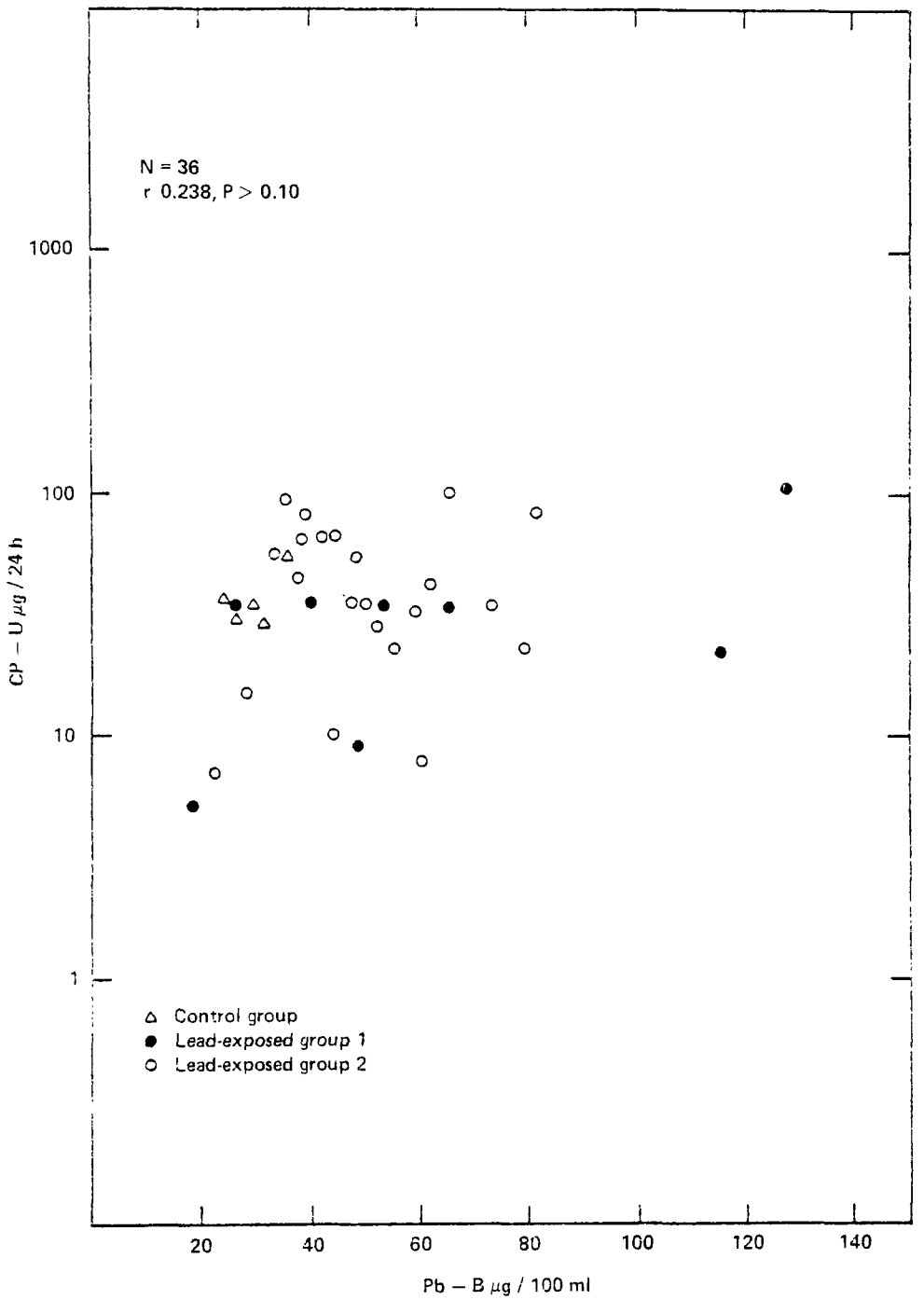


Figure 48. Semilogarithmic correlation in children of school age of a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/24 h/lin Pb–b).

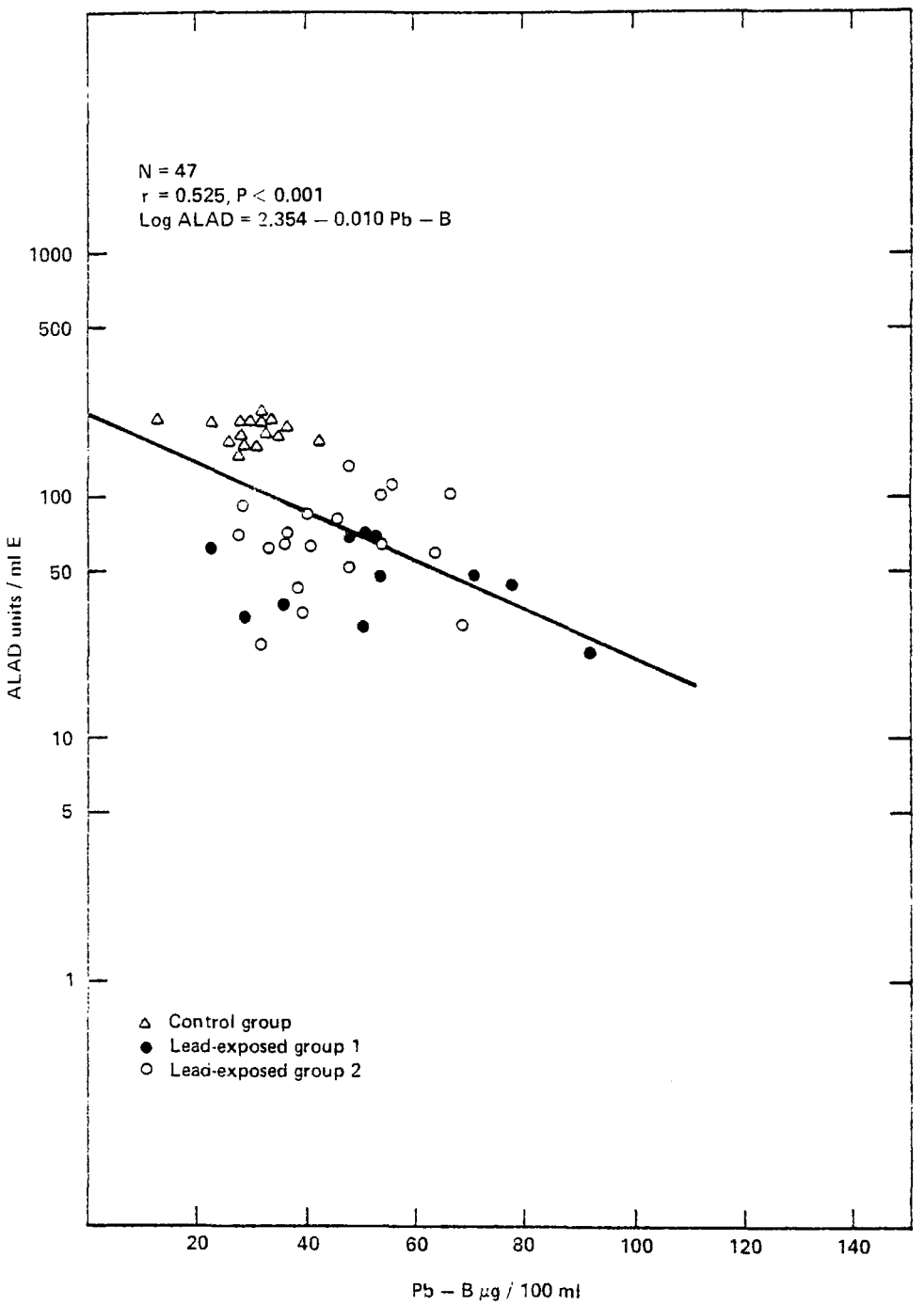


Figure 49. Semilogarithmic correlation in children up to 4 years of a total study population between δ-aminolevulinic acid dehydratase activity and lead in blood (log ALAD/ lin Pb–B).

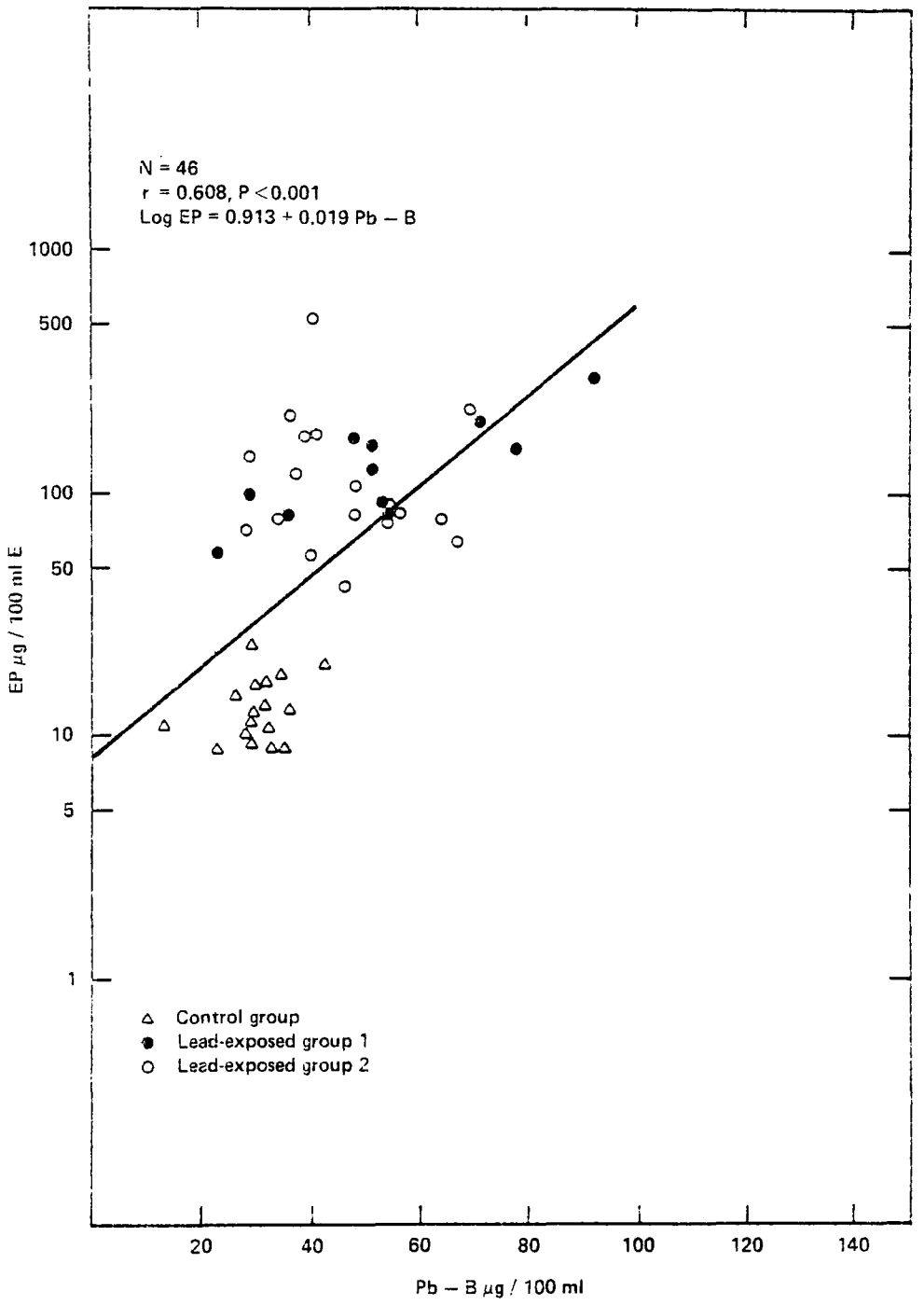


Figure 50. Semilogarithmic correlation in children up to 4 years of a total study population between erythrocyte protoporphyrin and lead in blood (log EP/lin Pb–B).

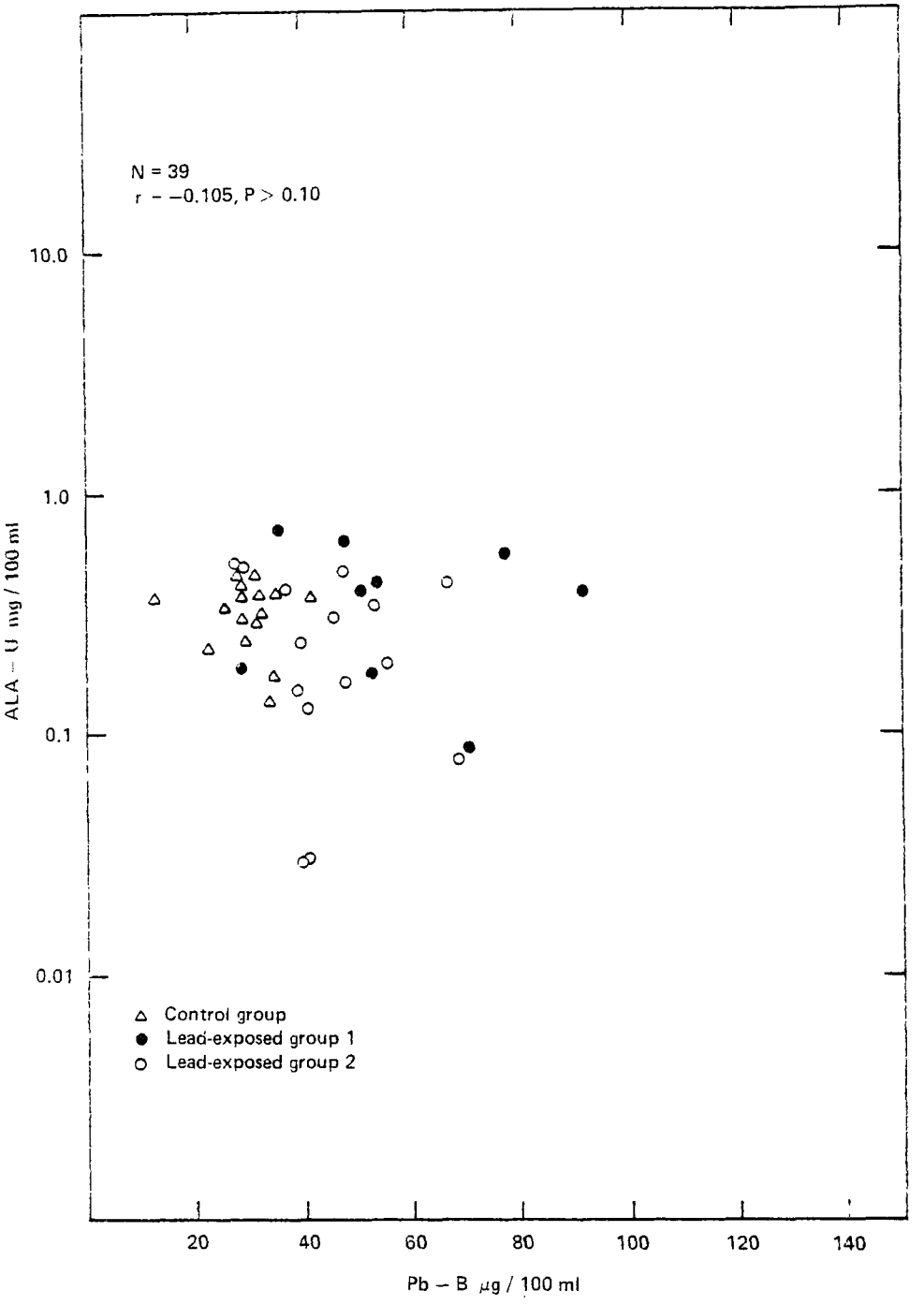


Figure 51. Semilogarithmic correlation in children up to 4 years of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/100 ml/lin Pb–B).

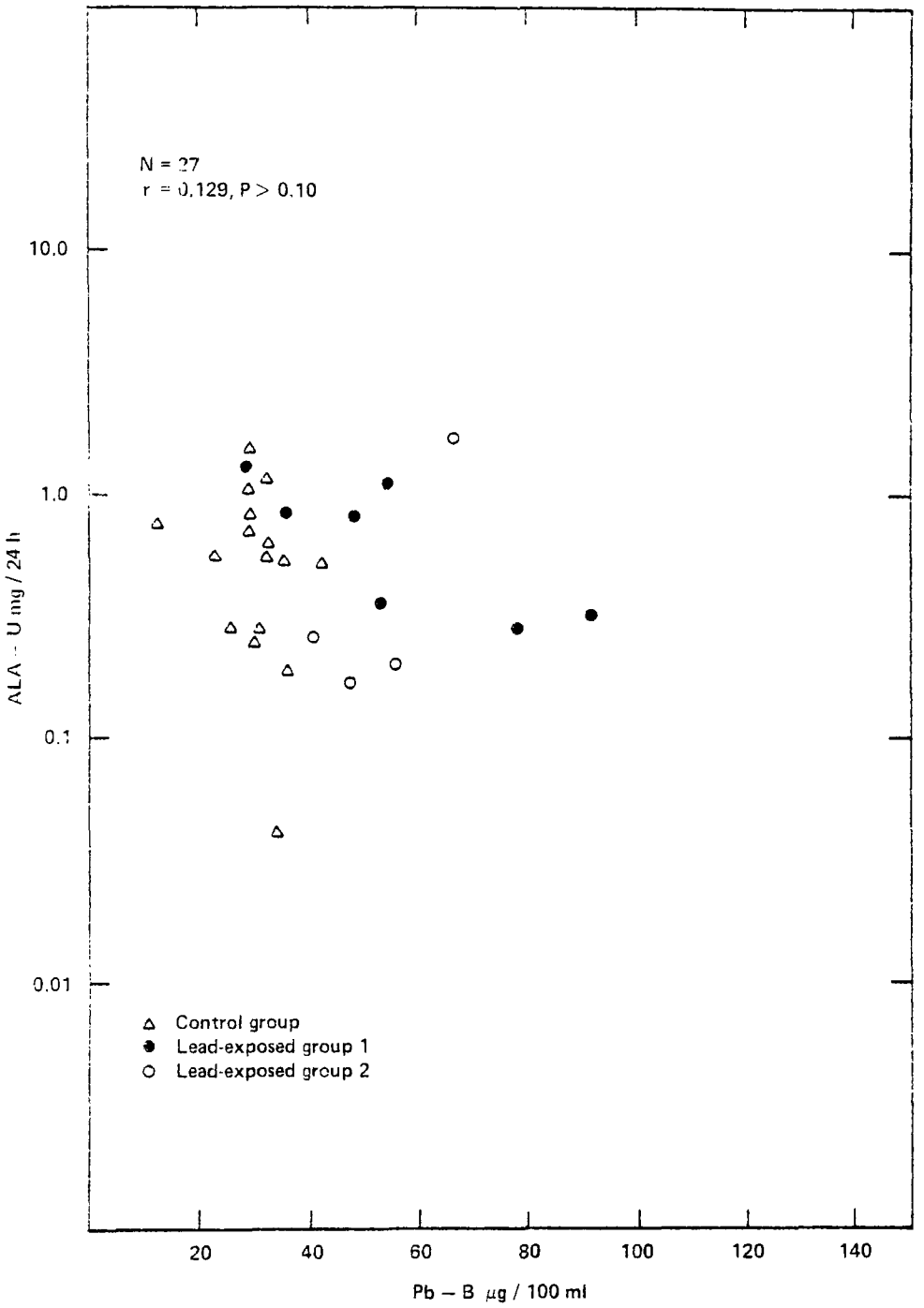


Figure 52. Semilogarithmic correlation in children up to 4 years of a total study population between δ-aminolevulinic acid in urine and lead in blood (log ALA–U mg/24 h/lin Pb–B).

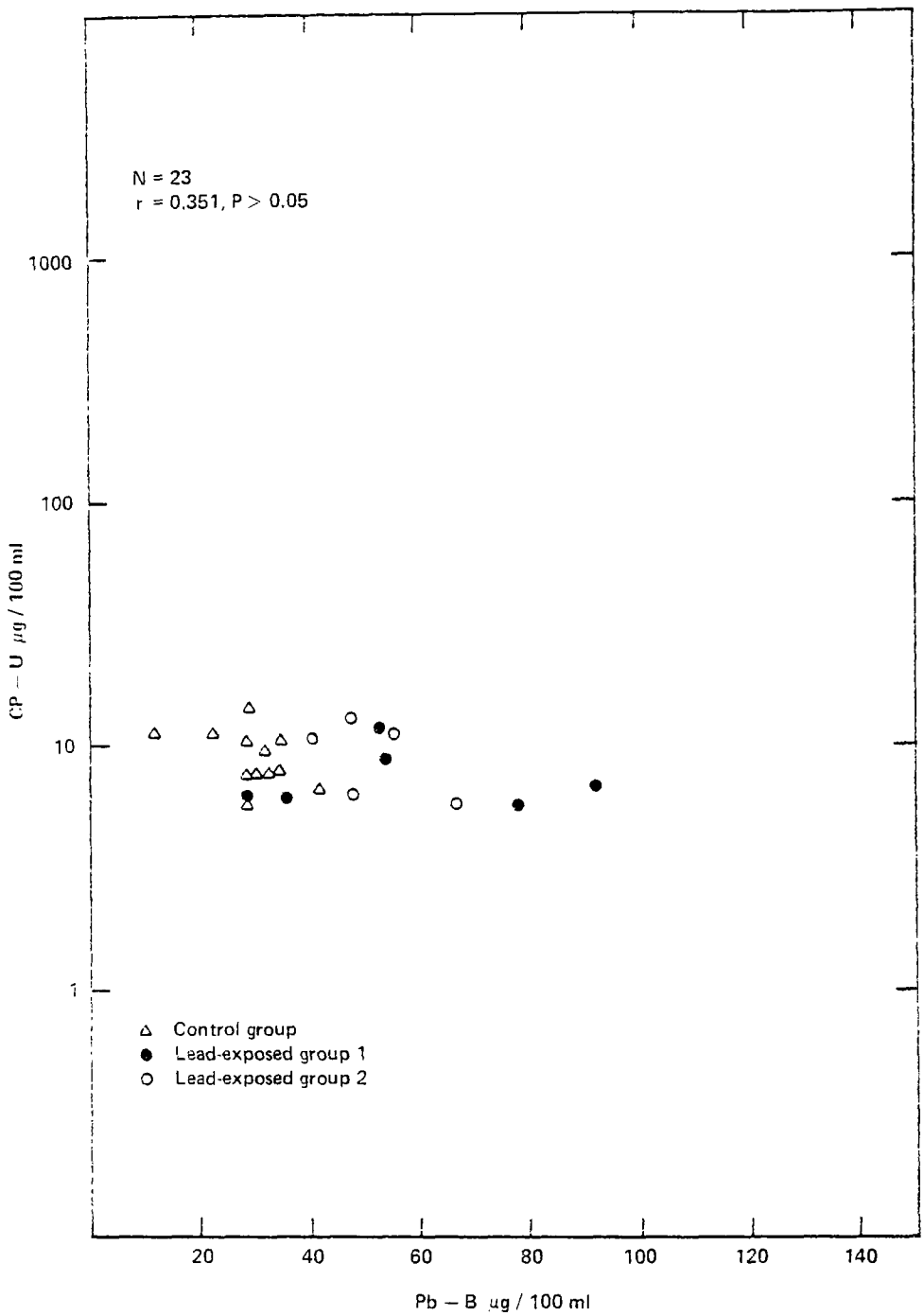


Figure 53. Semilogarithmic correlation in children up to 4 years of a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/100 ml/lin Pb–B).

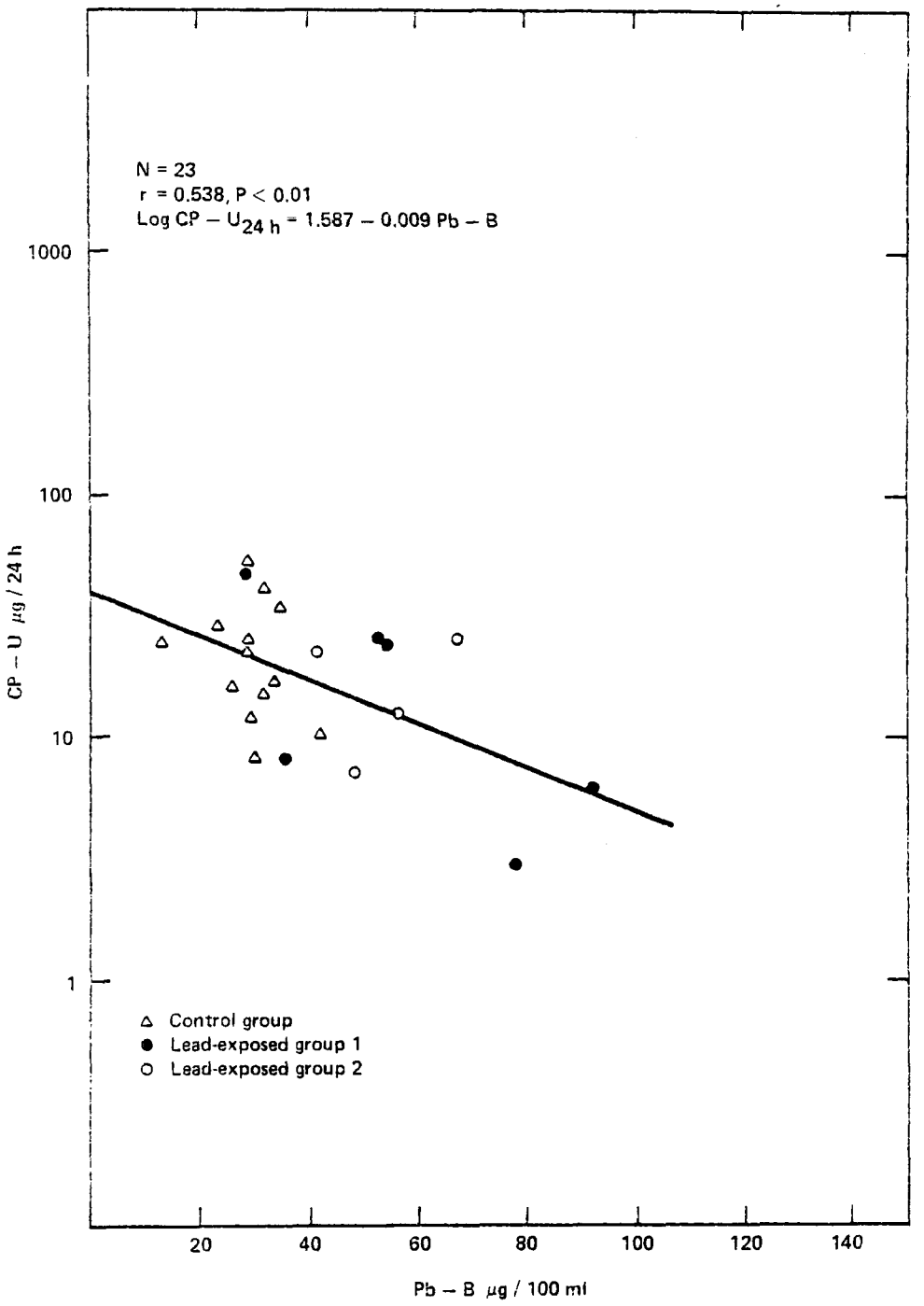


Figure 54. Semilogarithmic correlation in children up to 4 years of a total study population between coproporphyrin in urine and lead in blood (log CP–U µg/24 h/lin Pb–B).

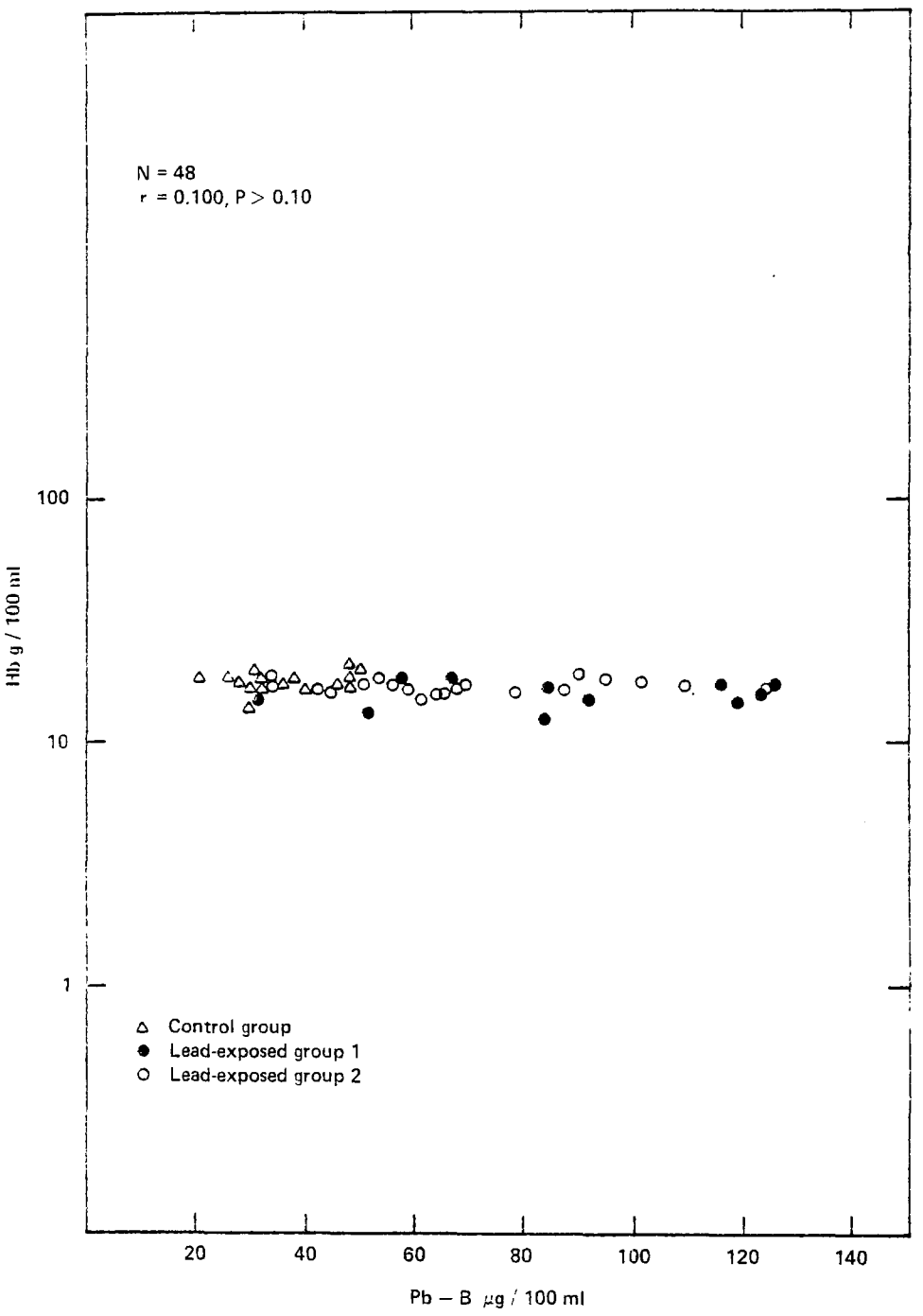


Figure 55. *Similogarithmic correlation in fathers of a total study population between hemoglobin and lead in blood (log Hb/lin Pb–B).*

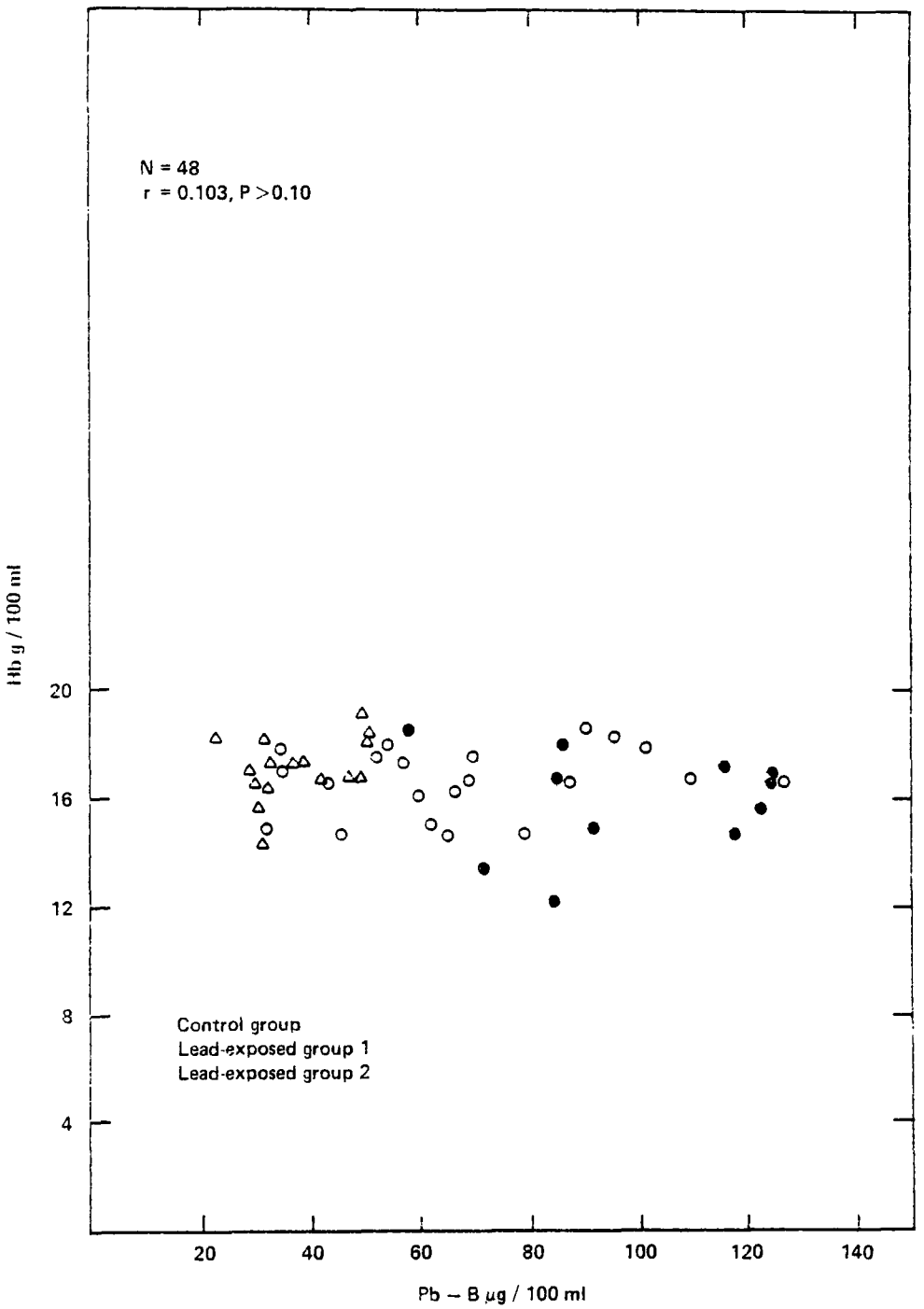


Figure 56. Correlation in fathers of a total study population between hemoglobin and lead in blood (lin Hb/lin Pb–B).

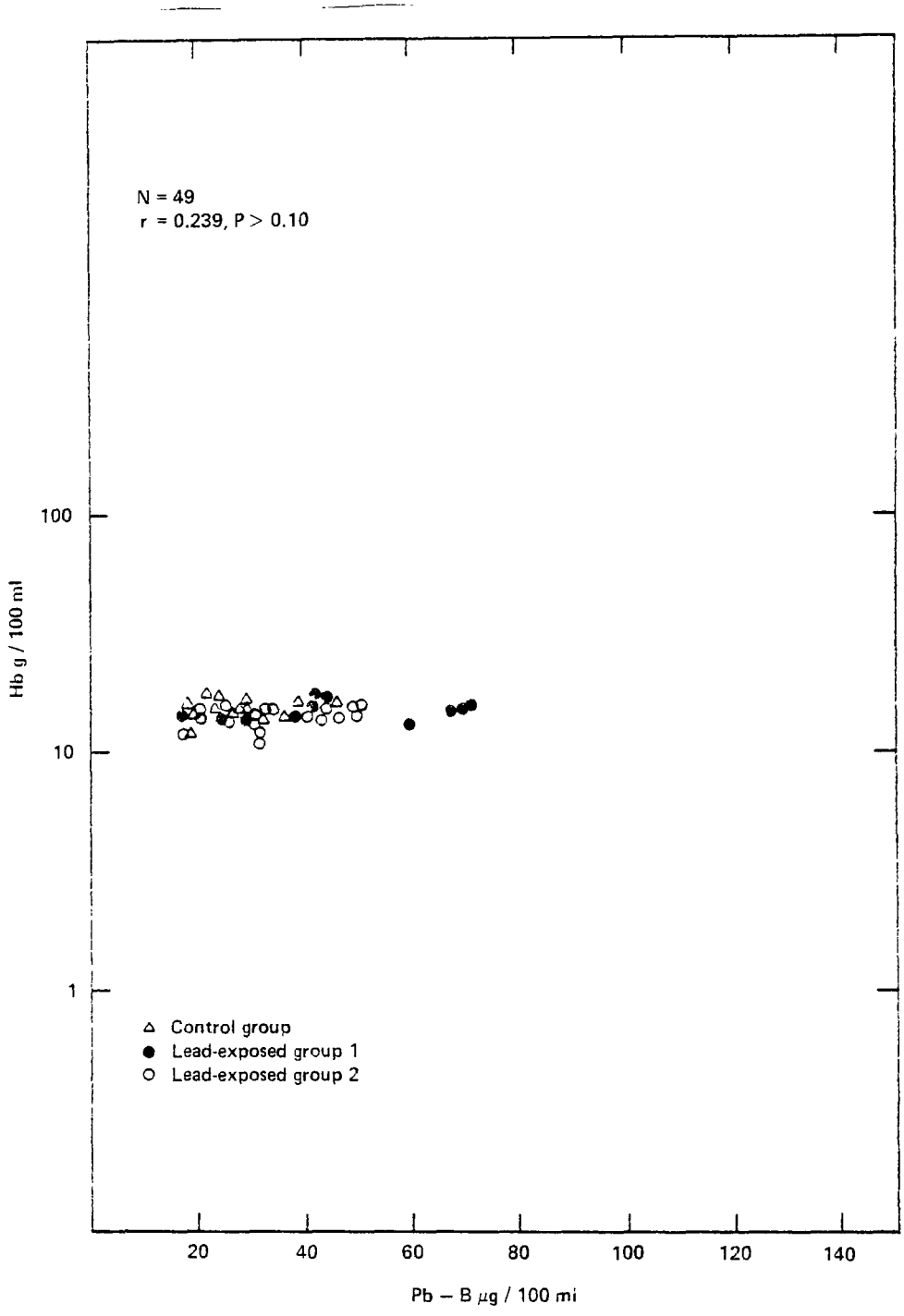


Figure 57. Semilogarithmic correlation in mothers of a total study population between hemoglobin and lead in blood (log Hb/lin Pb–B).

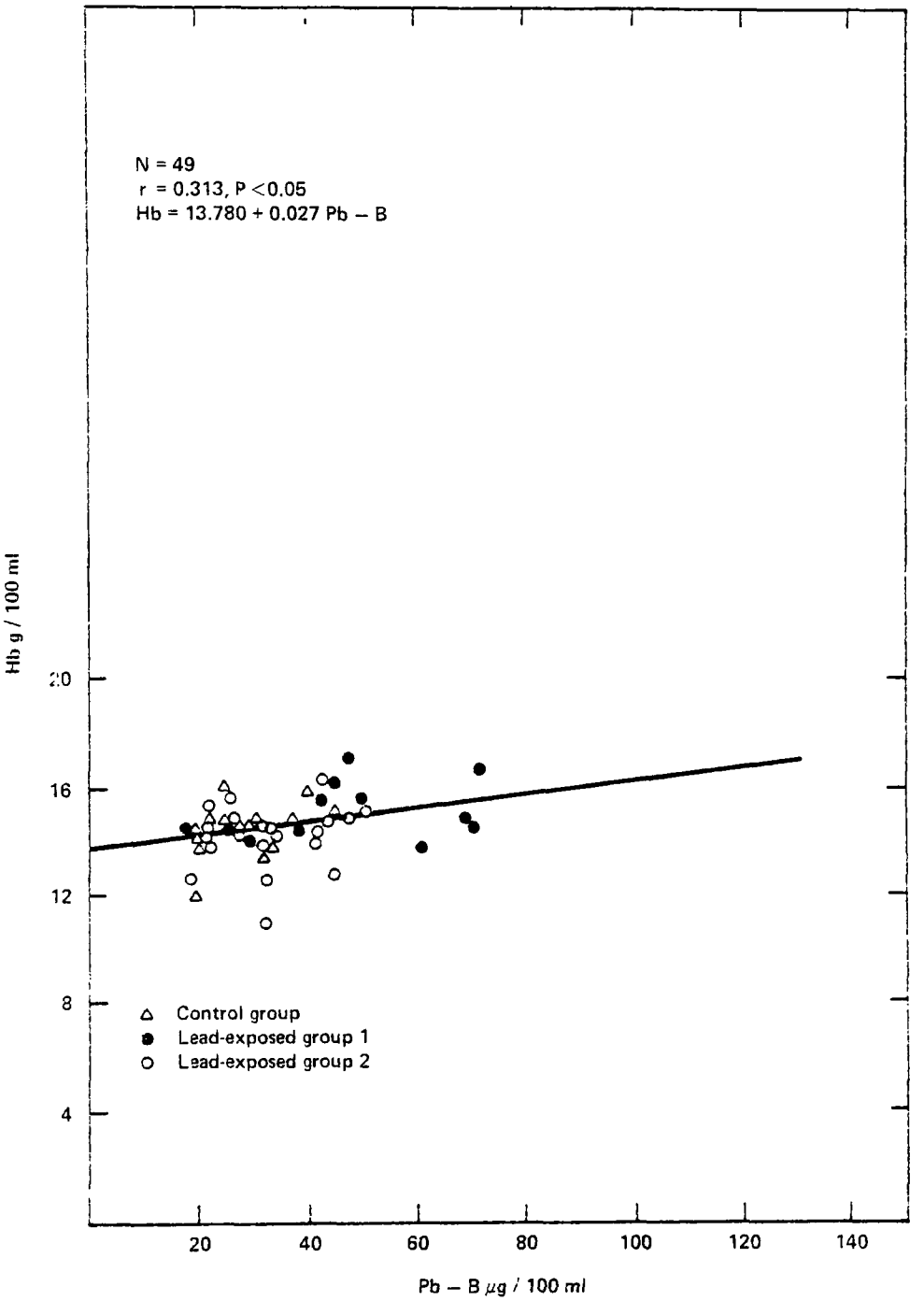


Figure 58. Correlation in mothers of a total study population between hemoglobin and lead in blood (lin Hb/lin Pb–B).

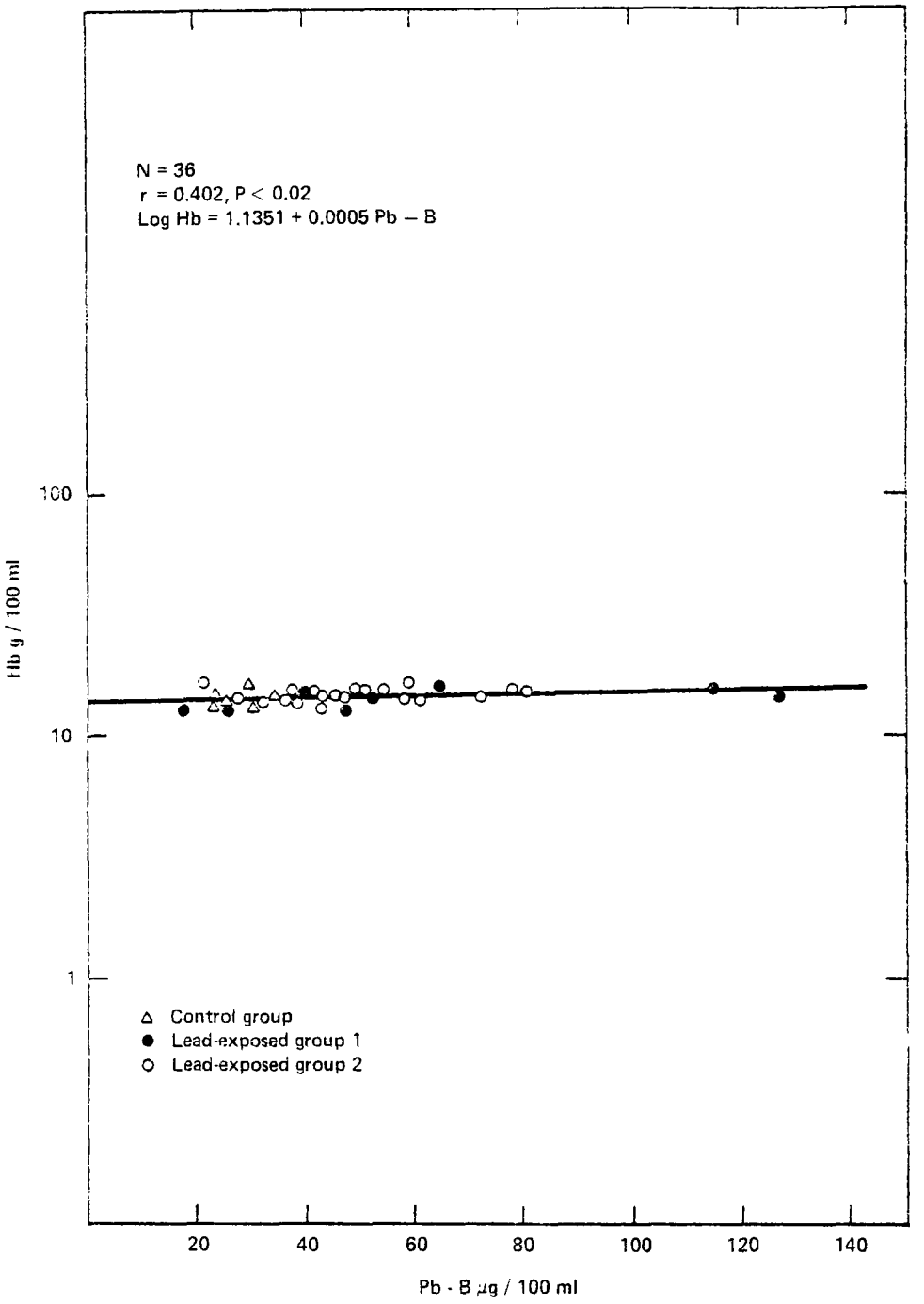


Figure 59. Semilogarithmic correlation in children of school age of a total study population between hemoglobin and lead in blood (lcg Hb/lin Pb–B).

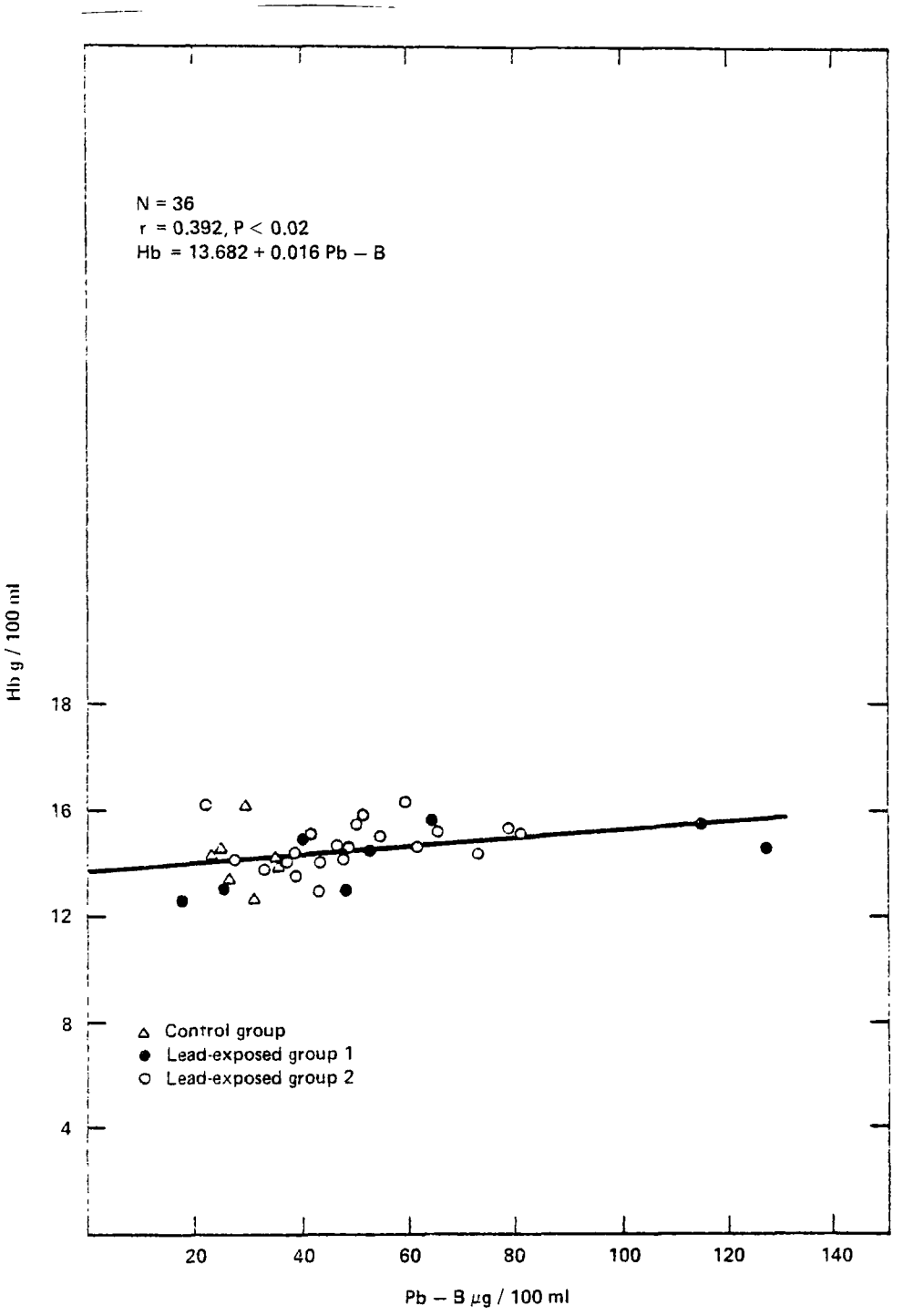


Figure 60. Correlation in children of school age of a total study population between hemoglobin and lead in blood (lin Hb/lin Pb–B).

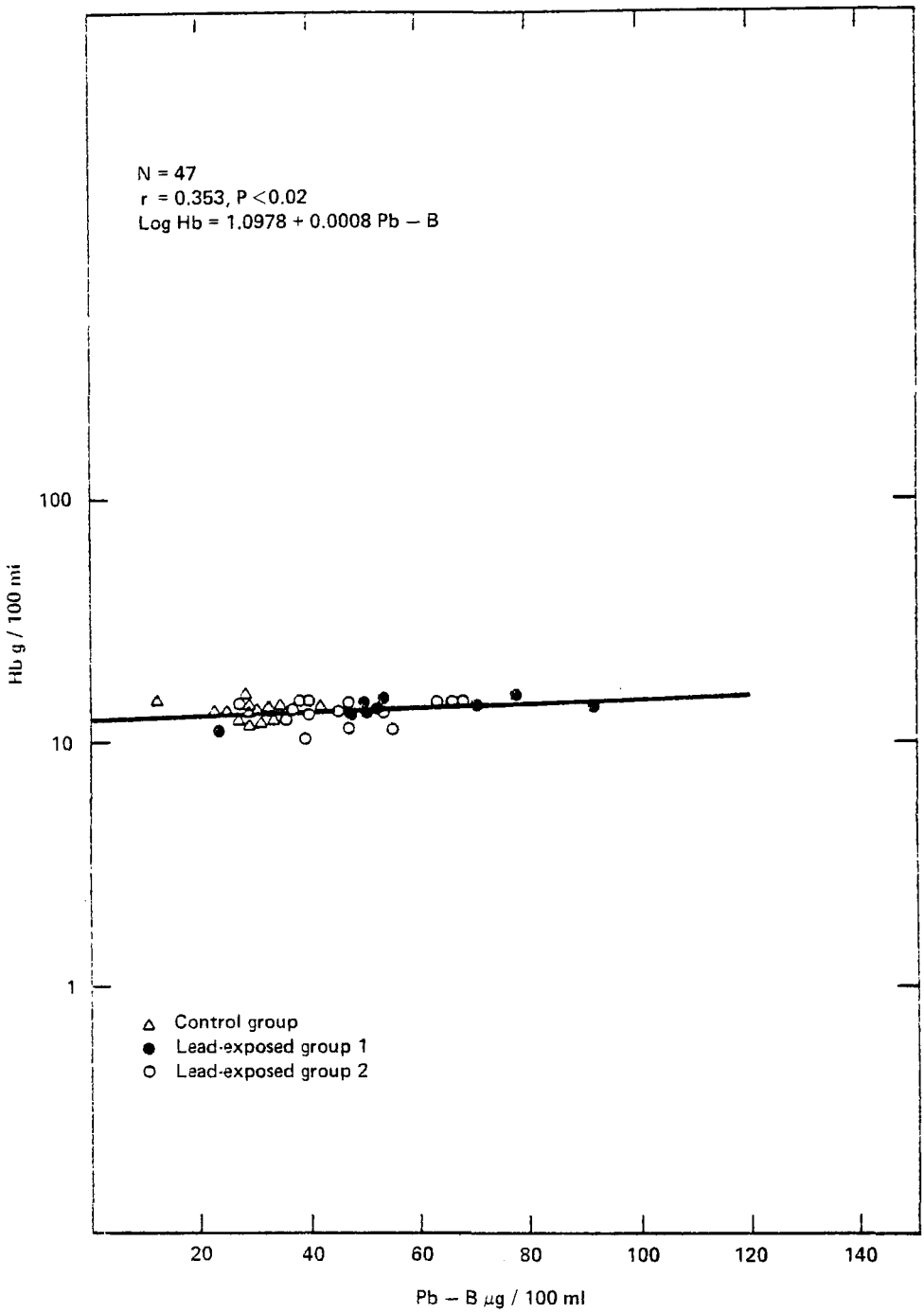


Figure 61. Semilogarithmic correlation in children up to 4 years of a total study population between hemoglobin and lead in blood (log Hb/lin Pb–B).

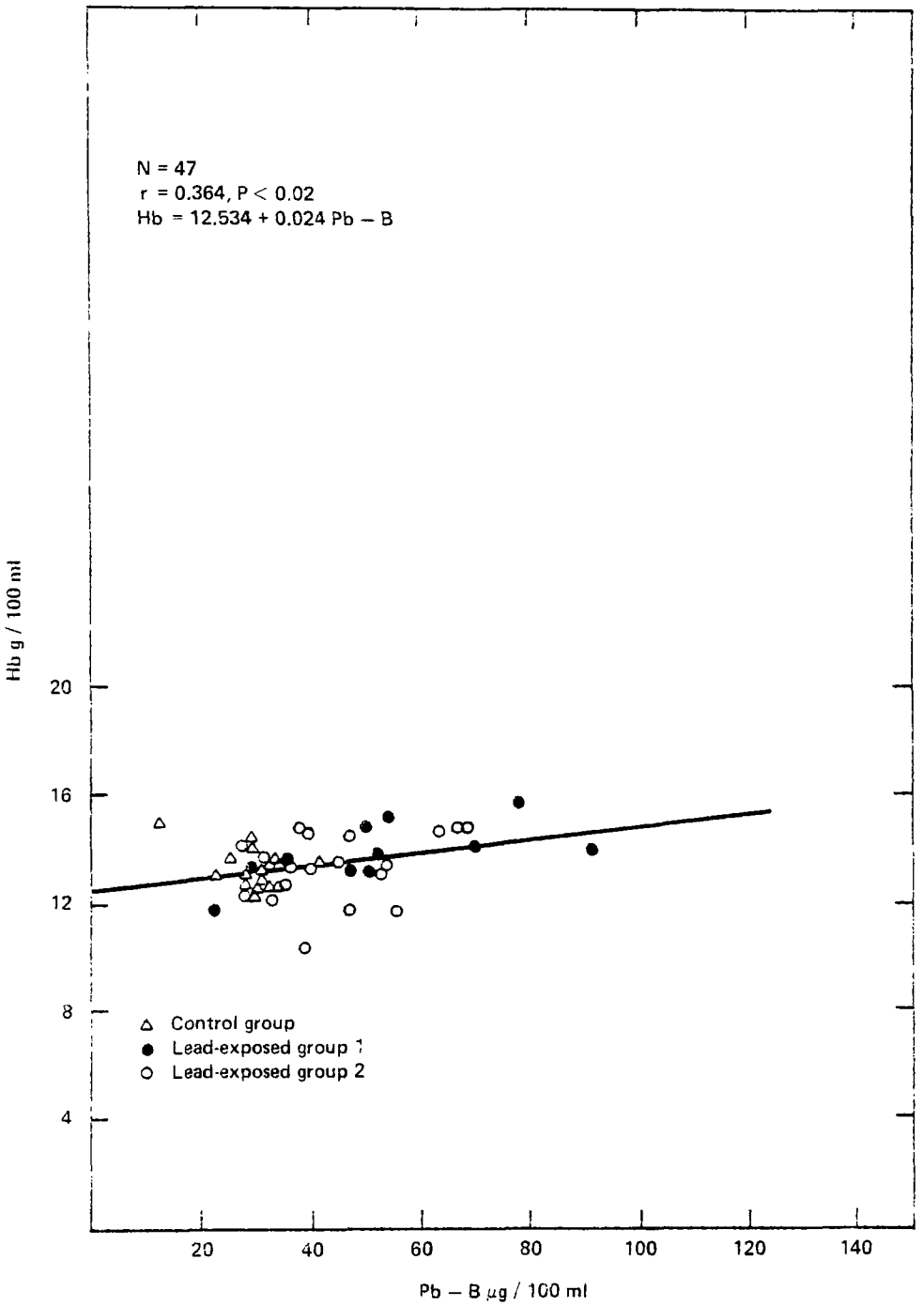


Figure 62. Correlation in children up to 4 years of a total study population between hemoglobin and lead in blood (lin Hb/lin Pb–B).

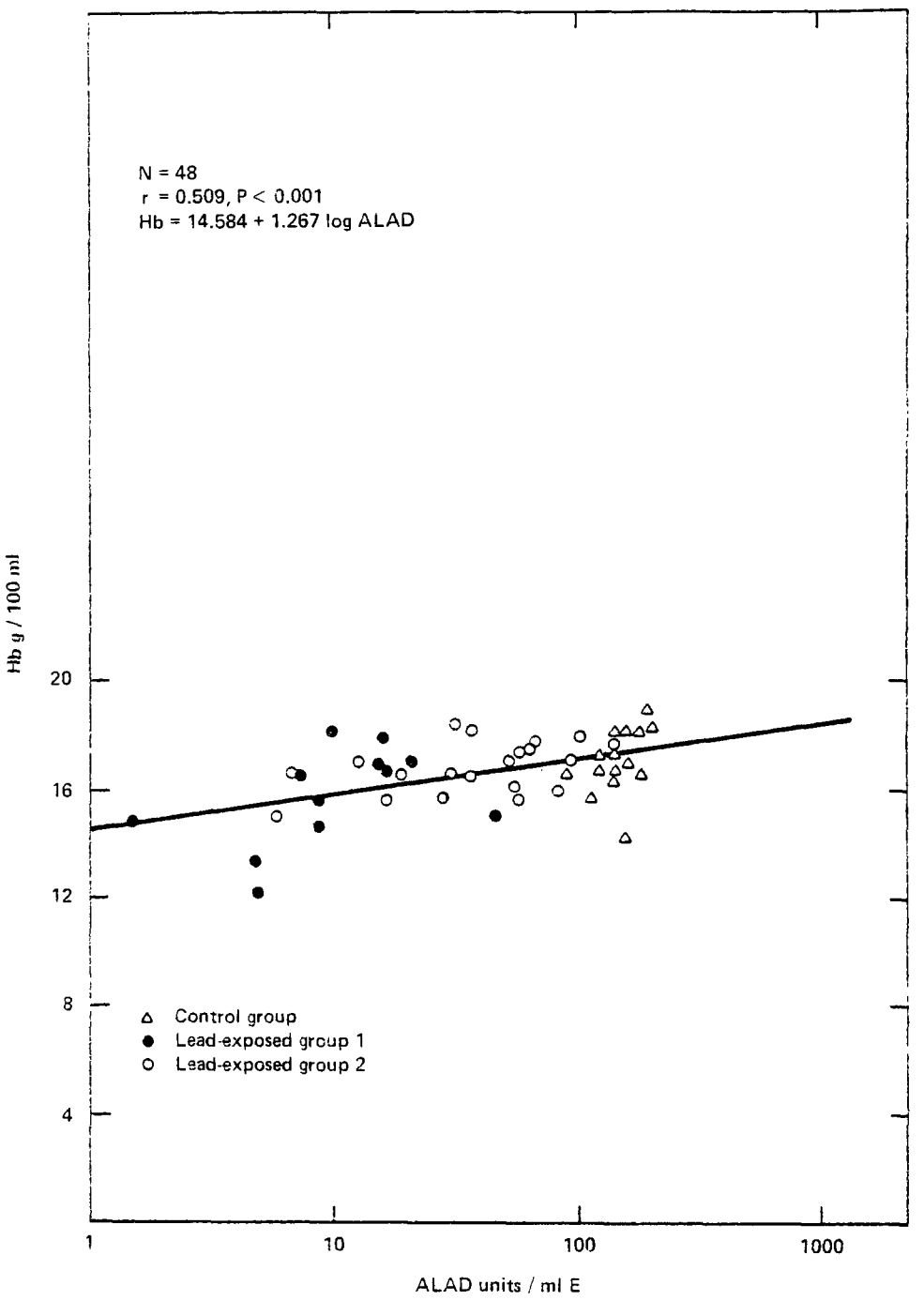


Figure 63. Semilogarithmic correlation in fathers of a total study population between hemoglobin and δ-aminolevulinic acid dehydratase activity (lin Hb/log ALAD).

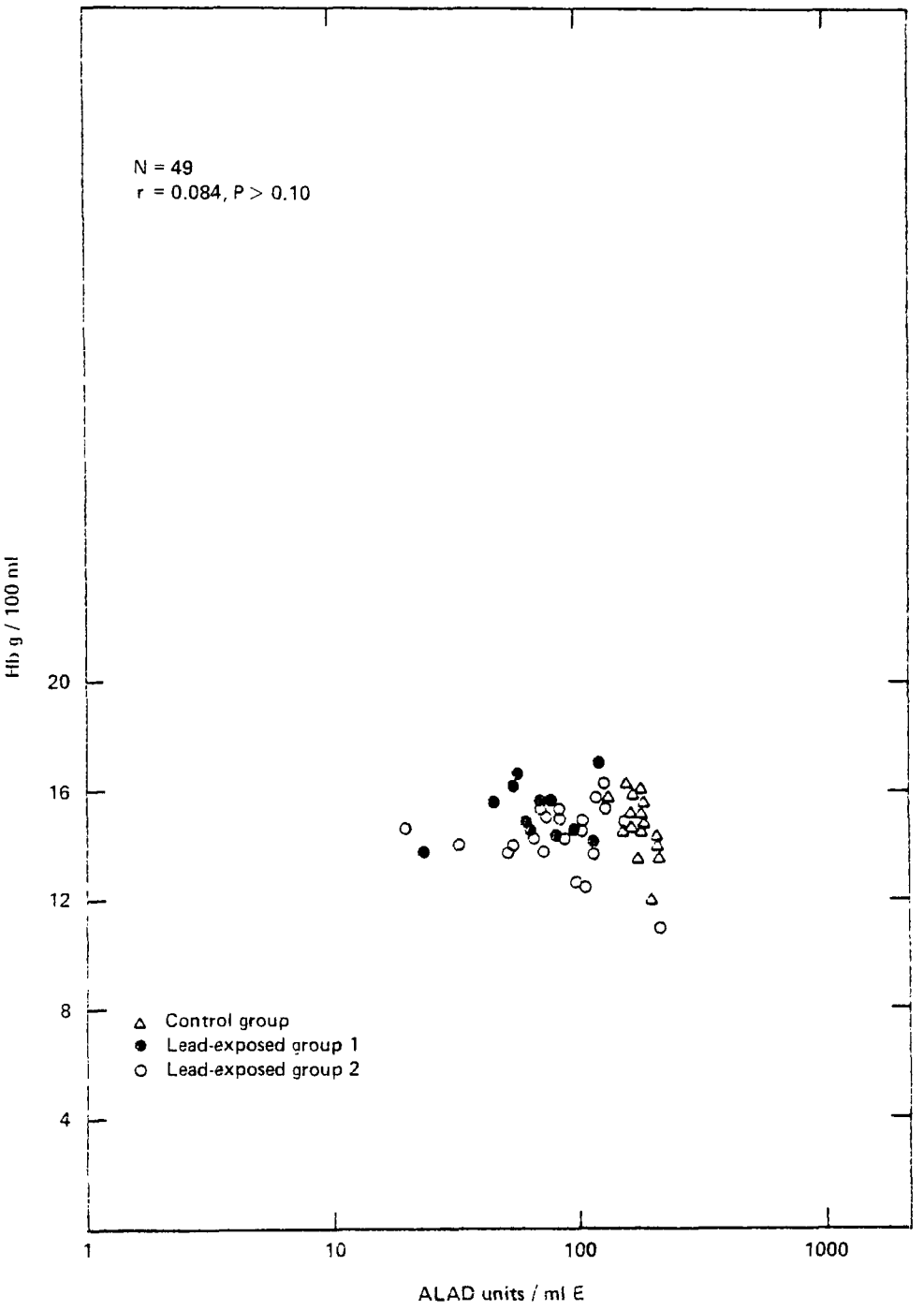


Figure 64. Semilogarithmic correlation in mothers of a total study population between hemoglobin and δ-aminolevulinic acid dehydratase activity (lin Hb/log ALAD).

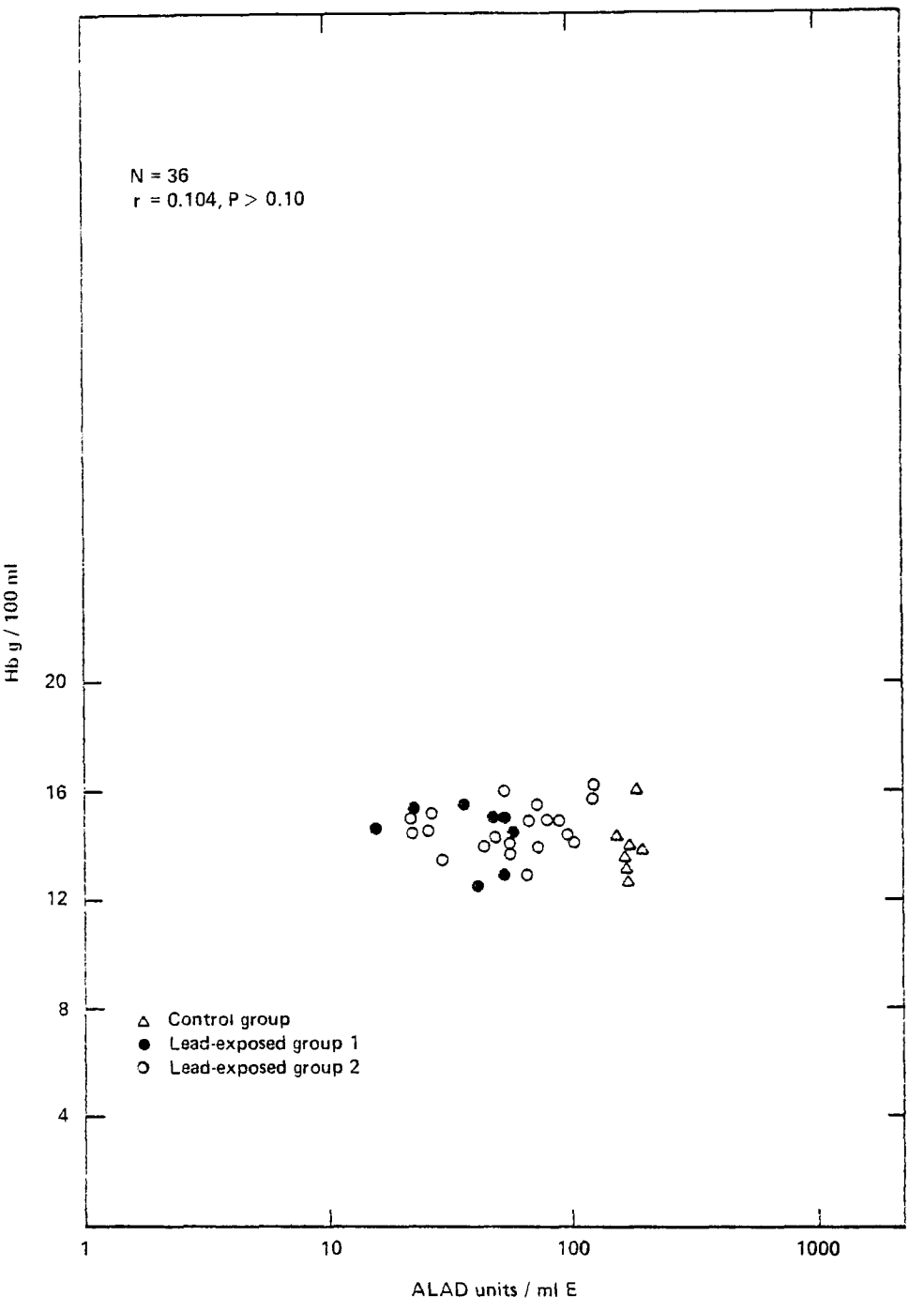


Figure 65. Semilogarithmic correlation in children of school age of a total study population between hemoglobin and δ-aminolevulinic acid dehydratase activity (lin Hb/log ALAD).

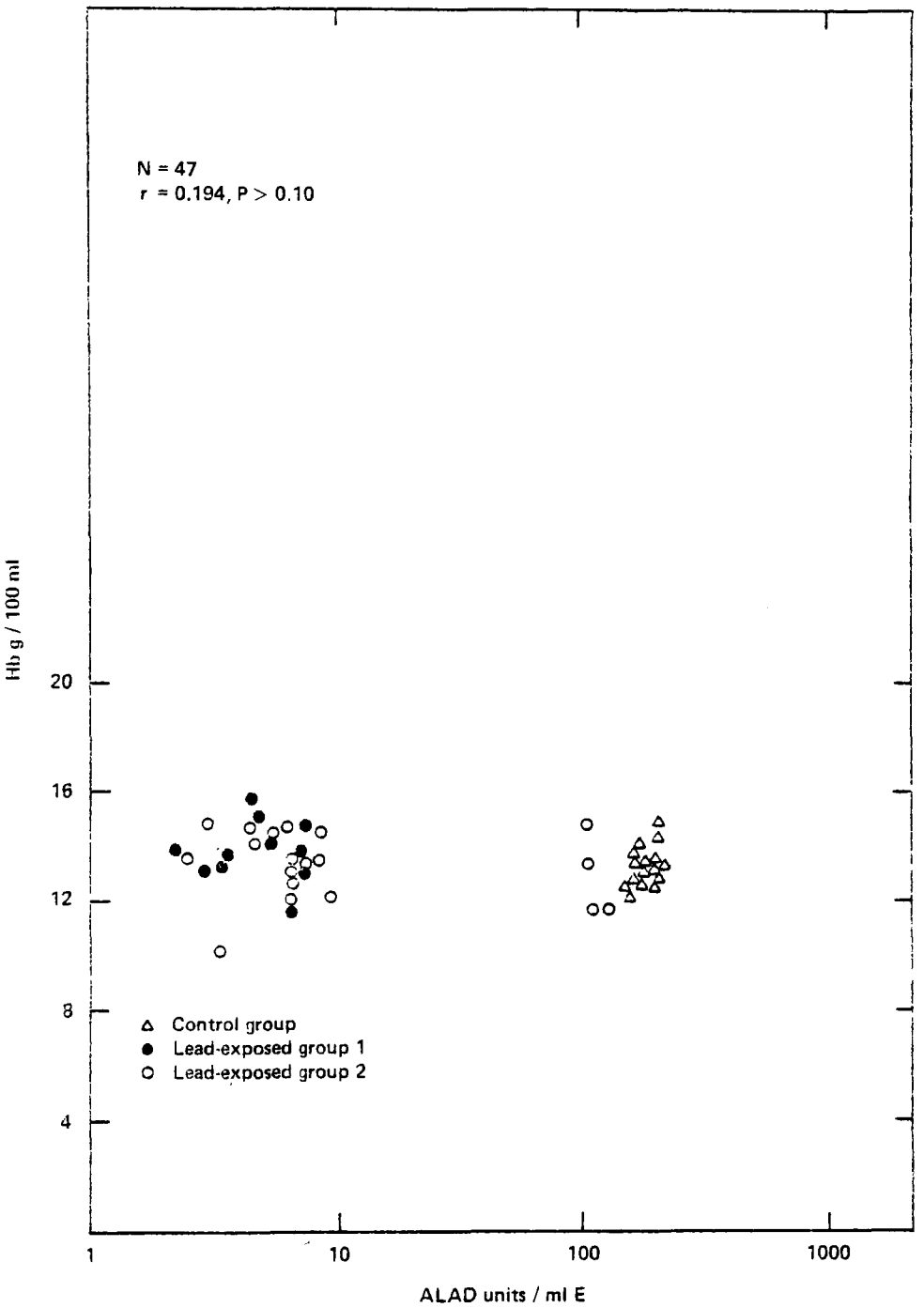


Figure 66. Semilogarithmic correlation in children up to 4 years of a total study population between hemoglobin and δ-aminolevulinic acid dehydratase activity (lin Hb/log ALAD).

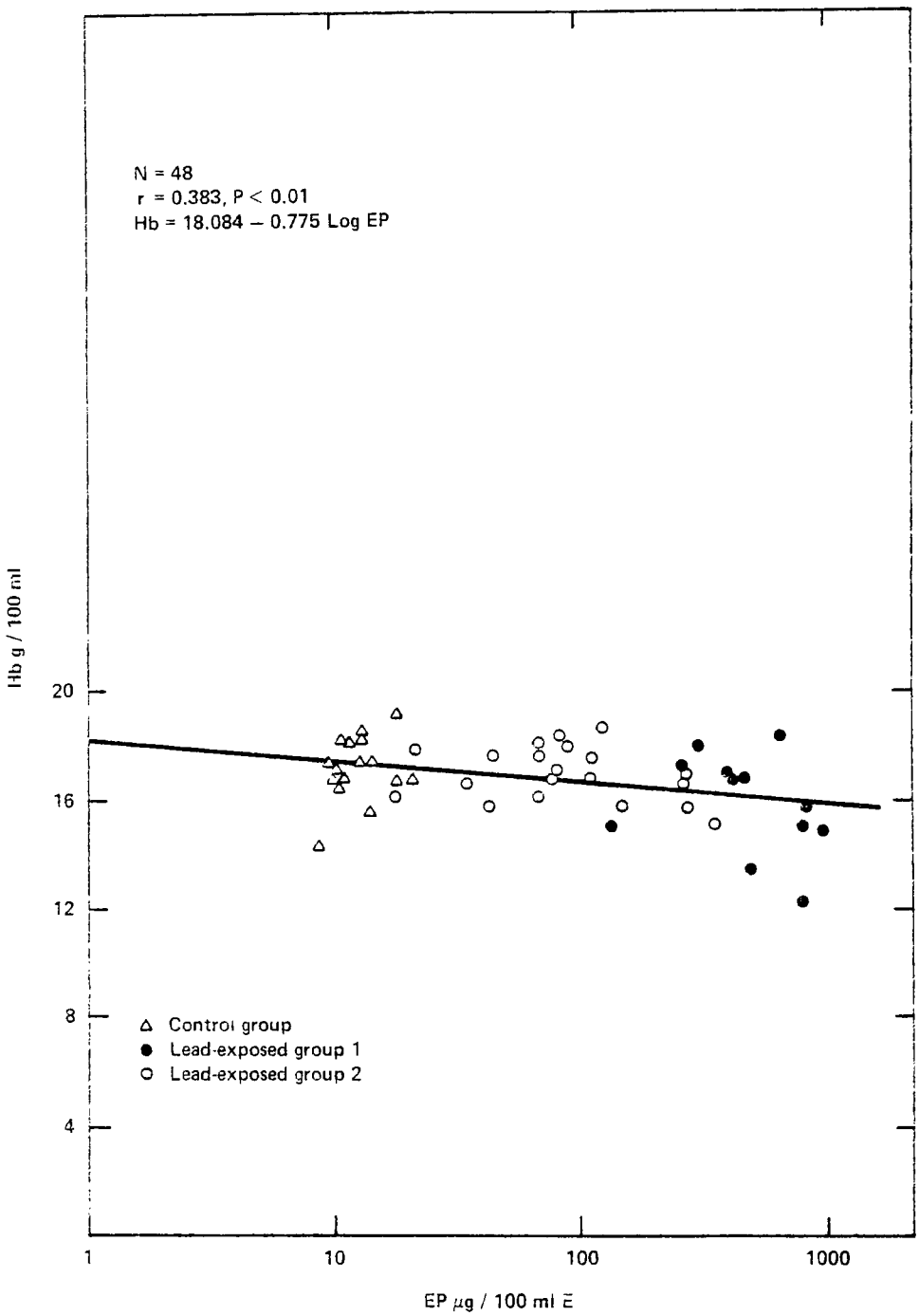


Figure 67. Semilogarithmic correlation in fathers of a total study population between hemoglobin and erythrocyte protoporphyrin (lin Hb/log EP).

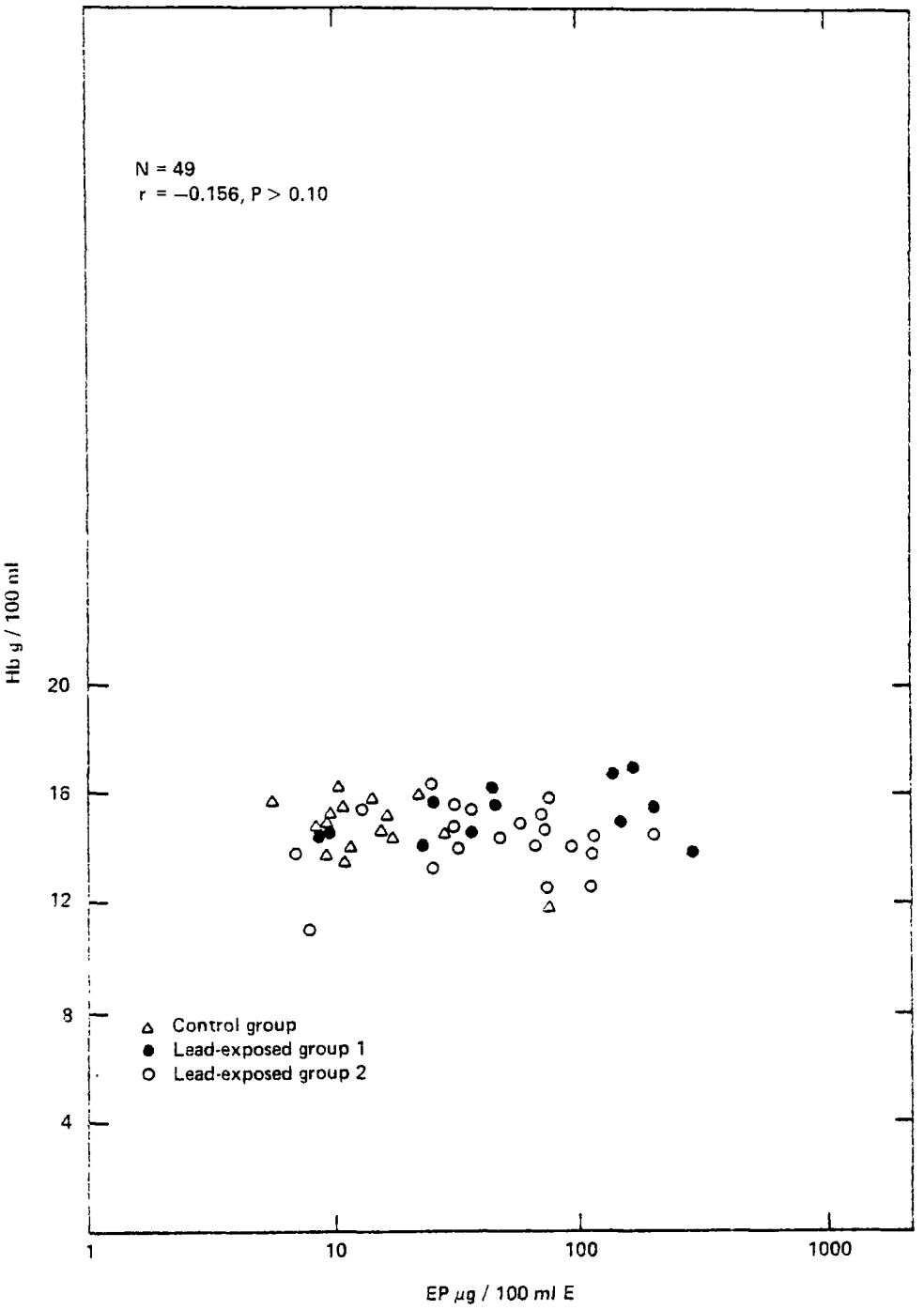


Figure 68. Semilogarithmic correlation in mothers of a total study population between hemoglobin and erythrocyte protoporphyrin (lin Hb/log EP).

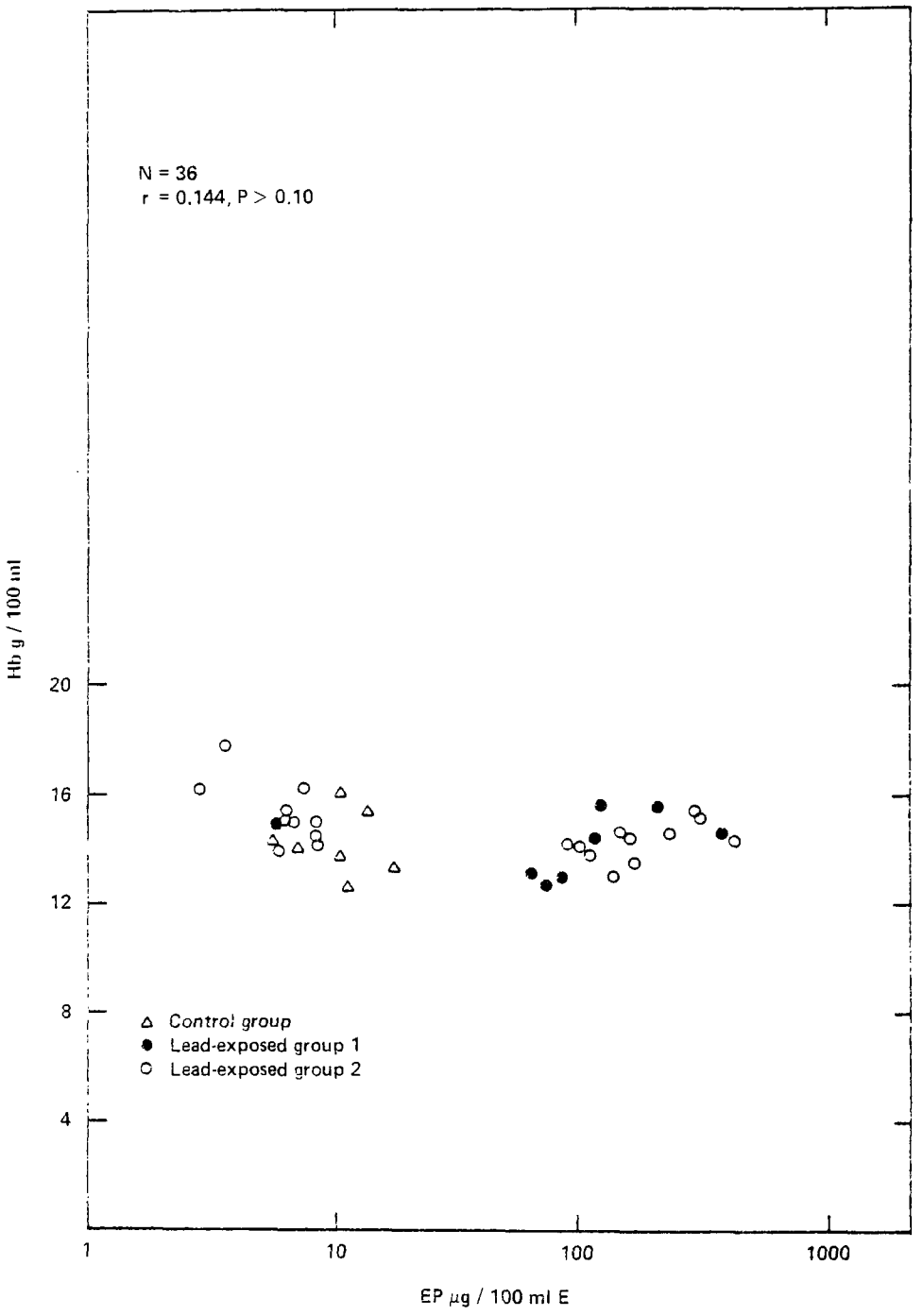


Figure 69. Semilogarithmic correlation in children of a school age of a total study population between hemoglobin and erythrocyte protoporphyrin (lin Hb/log EP).

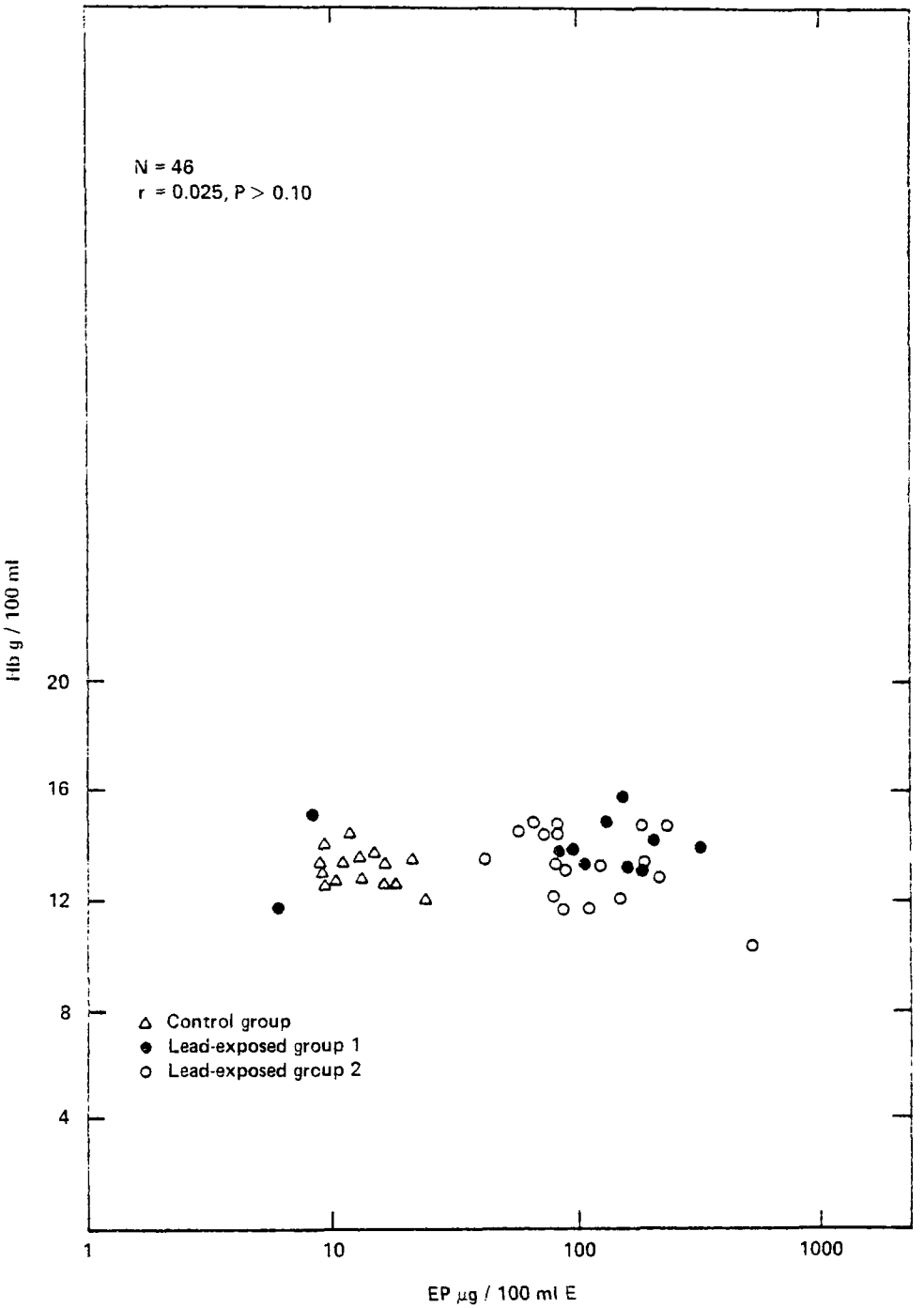


Figure 70. Semilogarithmic correlation in children up to 4 years of a total study population between hemoglobin and erythrocyte protoporphyrin (lin Hb/log EP).

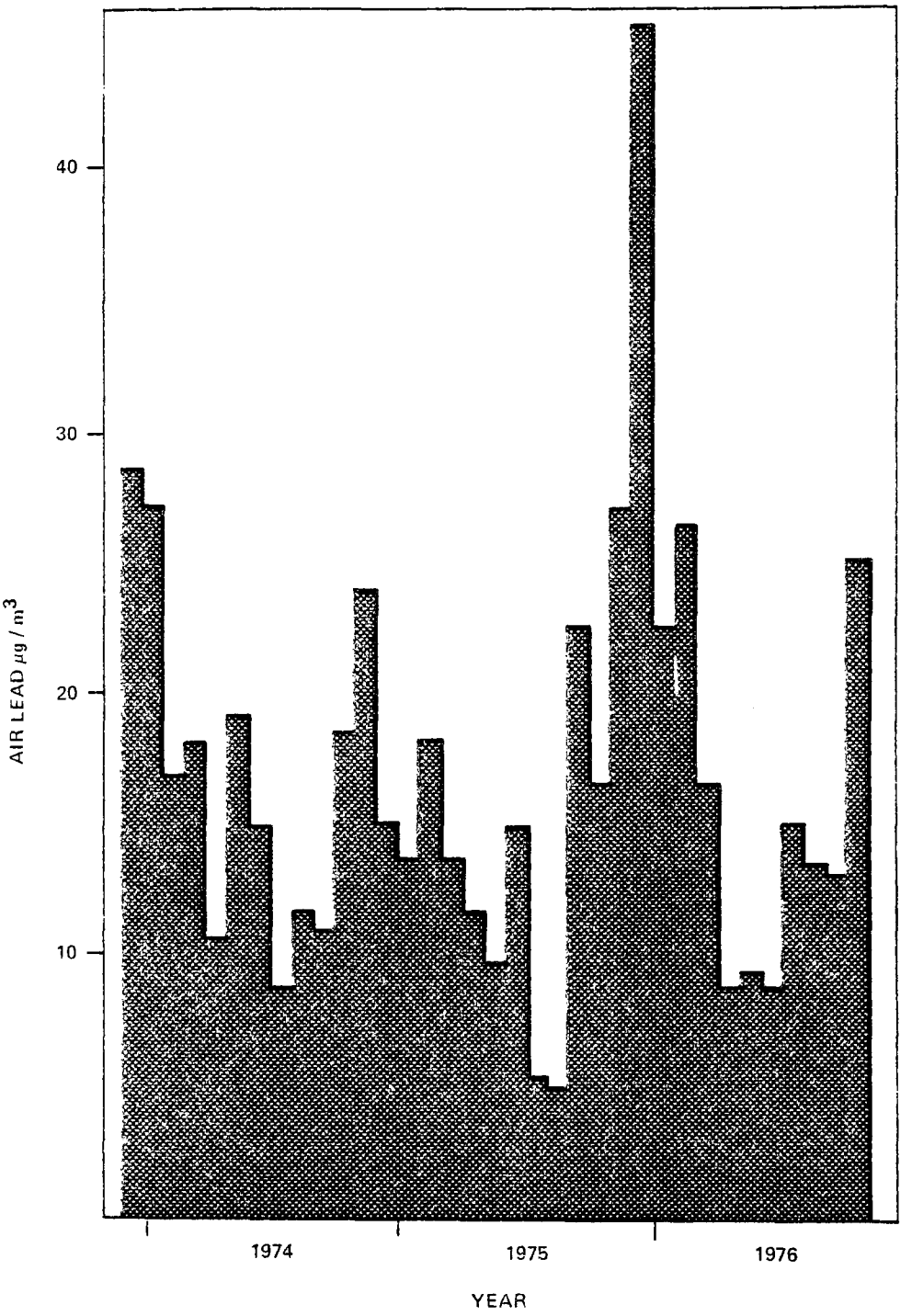


Figure 71. Yearly cycles of mean monthly air lead concentrations in lead smelter area (Averages of five sampling sites).

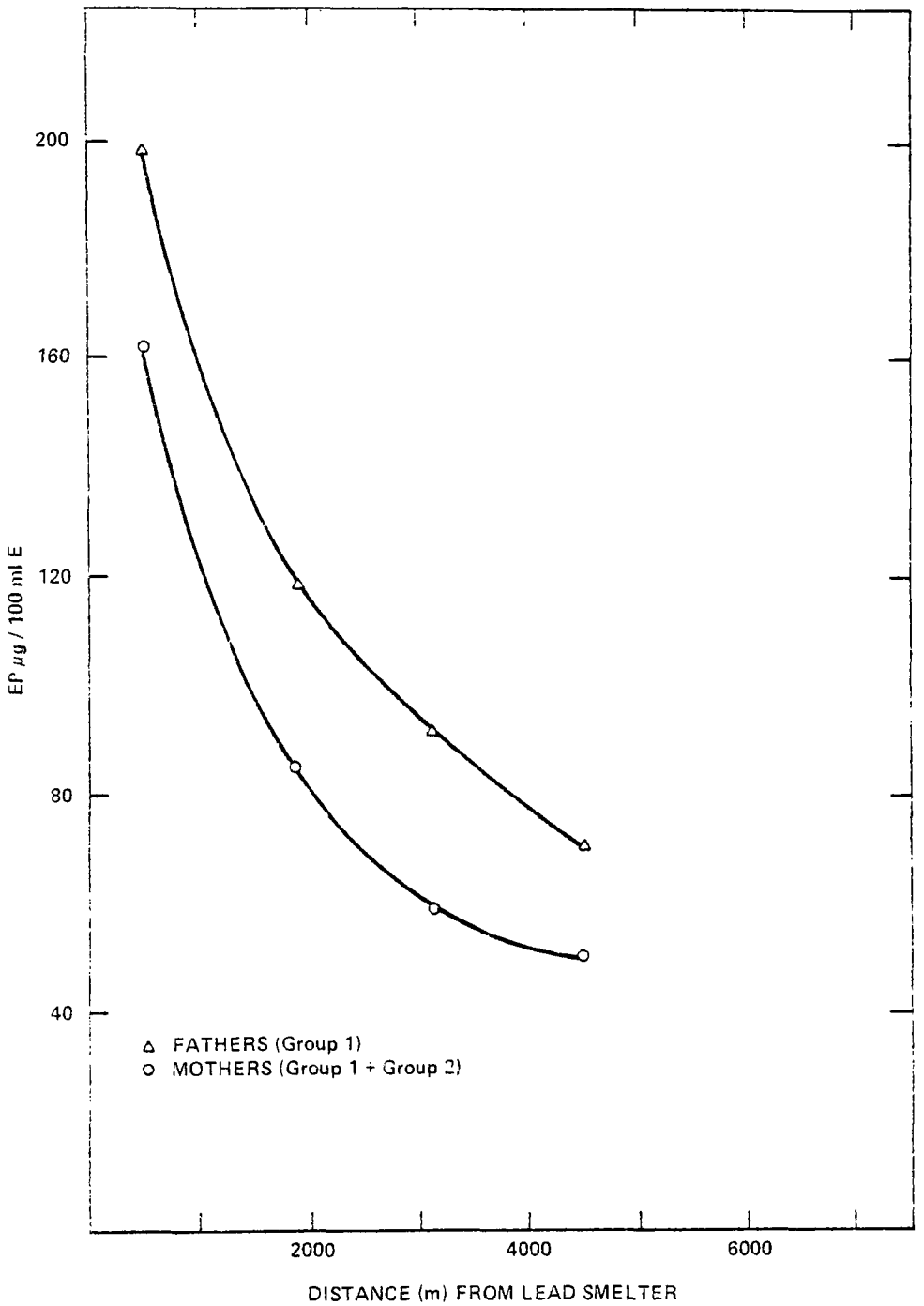


Figure 72. Median erythrocyte protoporphyrin (EP) in fathers and mothers according to median residential distance from lead smelter.

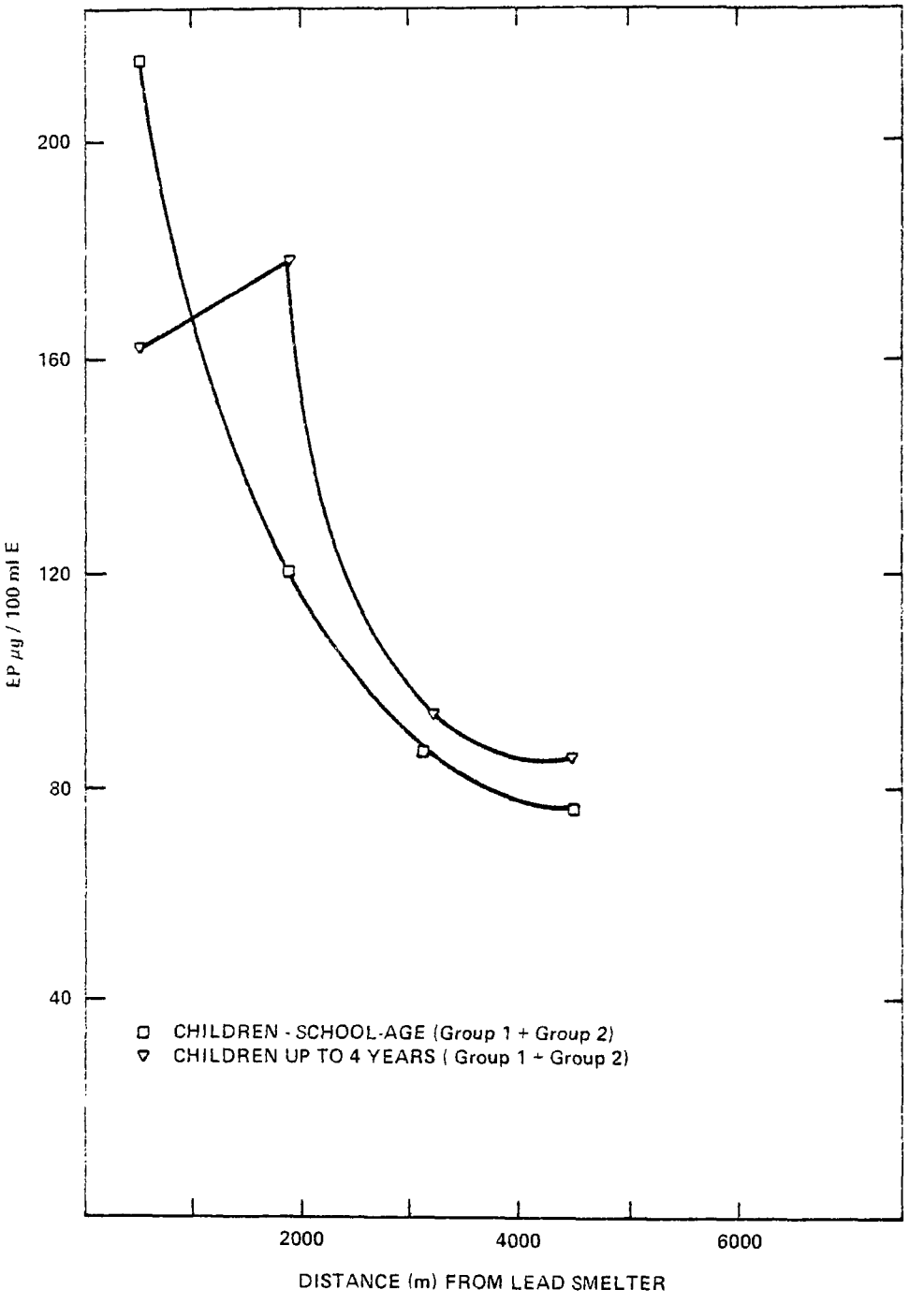


Figure 73. Median erythrocyte protoporphyrin (EP) in children of school age and in children up to 4 years according to median residential distance from lead smelter.

TABLE 1. THE NUMBER OF FAMILIES IN THE EXAMINED GROUPS

Group	N
Lead-exposed group 1	12
Lead-exposed group 2	21
Control group	16

TABLE 2. THE NUMBER OF FAMILY MEMBERS WITHIN EXAMINED GROUPS

Family relationship	Lead-exposed groups Group 1	Group 2	Control group	Total
Father	12	20	16	48
Mother	12	21	16	49
Child - school age	9 (6 m 3 f)	21 (12 m 9 f)	7 (2 m 5 f)	37
Child - up to 4 years	11 (6 m 5 f)	19 (8 m 11 f)	17 (7 m 10 f)*	47

m Male
f Female
* Two daughters were twins.

TABLE 3. AGE DISTRIBUTION IN PARENTS

Fathers' age	Lead-exposed groups				Control group	
	Group 1		Group 2			
	N	%	N	%	N	%
20-25	2	16.7	-	-	2	12.5
26-30	3	25.0	4	20.0	2	12.5
31-35	3	25.0	9	45.0	3	18.8
36-40	3	25.0	5	25.0	5	31.2
41-45	1	8.3	2	10.0	2	12.5
46-50	-	-	-	-	2	12.5

Mothers' age	Lead-exposed groups				Control group	
	Group 1		Group 2			
	N	%	N	%	N	%
20-25	5	41.7	2	9.5	3	18.8
26-30	3	25.0	7	33.3	2	12.5
31-35	3	25.0	8	38.1	4	25.0
36-40	1	8.3	3	14.3	4	25.0
41-45	-	-	1	4.8	3	18.8
46-50	-	-	-	-	-	-

TABLE 4. AGE DISTRIBUTION IN CHILDREN

School children's age	Lead-exposed groups				Control group	
	Group 1		Group 2			
	N	%	N	%	N	%
5-7	5	55.6	9	42.9	2	28.6
8-10	2	22.2	7	33.3	4	57.1
11-13	2	22.2	4	19.0	-	-
14-16	-	-	1	4.8	1	14.3

Small children's age	Lead-exposed groups				Control group	
	Group 1		Group 2			
	N	%	N	%	N	%
up to 11 mo	1	9.1	1	5.3	1	5.9
1-2	8	72.7	17	89.4	11	64.7
3-4	2	18.2	1	5.3	5	29.4

TABLE 5. HABITATION DISTRIBUTION BY DISTANCE FROM LEAD SMELTER IN LEAD-EXPOSED GROUP 1

Habitation	Distance (m) from lead smelter	Family number	Family members examined			
			Fathers	Mothers	Children school age	Children up to 4 years
A	150 - 800	3	3	2	3	2
B_1, B_2	1900	1	1	2	1	1
C_1, C_2, C_3	3000-3400	3	3	3	2	3
D	4200-6500	5	5	5	3	5

TABLE 6. HABITATION DISTRIBUTION BY DISTANCE FROM LEAD SMELTER IN LEAD-EXPOSED GROUP 2

Habitation	Distance (m) from lead smelter	Family number	Family members examined			
			Fathers	Mothers	Children school age	Children up to 4 years
A	150 - 800	2	2	2	2	2
B_1, B_2	1900	4	4	4	4	4
C_1, C_2, C_3	3000-3400	3	3	3	3	3
D	4200-6500	12	11	12	12	10

TABLE 7. STATISTICAL PARAMETERS OF BIOLOGICAL DATA IN LEAD-EXPOSED GROUP 1 (FATHERS OCCUPATIONALLY EXPOSED TO LEAD)

Statistical parameter	BLOOD							URINE			
								ALA		CP	
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	mg/100 ml	mg/24 h	µg/100 ml	µg/24 h
FATHERS											
N	12	12	12	12	12	12	12	12	12	12	12
$\bar{X}$	15.76	42.38	1341.7	17.1	552.42	13.48	91.42	1.613	11.238	44.1	315.8
SD	1.811	2.144	1940.22	7.49	266.409	11.694	31.577	0.8279	5.7235	29.46	211.93
SE	0.523	0.619	560.09	2.16	76.906	3.376	9.115	0.2390	1.6522	8.50	61.18
MOTHERS											
N	12	12	12	12	12	12	12	11	11	11	11
$\bar{X}$	15.24	41.17	608.3	15.6	96.80	71.49	46.66	0.596	3.958	9.0	65.6
SD	1.063	1.193	675.57	6.84	93.944	29.226	17.939	0.3274	2.2661	3.31	34.61
SE	0.307	0.345	281.62	1.98	27.119	8.437	5.179	0.0987	0.6833	1.00	10.43
CHILDREN (school age)											
N	8	8	9	9	8	8	8	9	9	9	9
$\bar{X}$	14.23	40.78	211.1	10.7	142.24	41.84	61.50	0.502	1.696	10.2	39.4
SD	1.194	1.778	437.16	3.43	111.328	15.606	39.902	0.2696	1.0780	5.76	32.25
SE	0.422	0.629	145.72	1.14	39.360	5.518	14.107	0.0899	0.3593	1.92	10.75
CHILDREN (up to 4 years)											
N	11	11	11	11	11	11	11	9	7	6	6
$\bar{X}$	13.97	39.18	436.4	10.7	144.32	49.09	53.22	0.403	0.723	7.9	18.5
SD	1.085	1.454	657.68	3.61	72.597	17.719	20.632	0.2173	0.4136	2.48	16.28
SE	0.327	0.438	198.30	1.09	21.889	5.342	6.221	0.0724	0.1563	1.01	6.65

TABLE 8. STATISTICAL PARAMETERS OF BIOLOGICAL DATA IN LEAD-EXPOSED GROUP 2 (FATHERS NOT OCCUPATIONALLY EXPOSED TO LEAD)

Statistical parameter	BLOOD							URINE			
								ALA		CP	
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	mg/100 ml	mg/24 h	µg/100 ml	µg/24 h
FATHERS											
N	20	20	20	20	20	20	20	20	20	19	19
$\bar{X}$	16.83	44.02	605.0	12.4	129.41	50.47	70.19	0.476	3.861	20.2	157.4
SD	0.962	1.235	900.57	4.64	115.858	35.591	24.806	0.2166	2.0415	46.05	321.47
SE	0.215	0.276	201.37	1.04	25.907	7.958	5.547	0.0484	0.4565	10.57	73.75
MOTHERS											
N	21	21	21	21	21	21	21	20	20	20	20
$\bar{X}$	14.41	40.45	200.0	13.7	69.76	95.38	32.93	0.344	2.690	8.8	70.0
SD	1.230	1.923	524.40	6.42	44.322	44.829	9.648	0.1128	0.9142	4.06	34.52
SE	0.268	0.420	114.43	1.40	9.672	9.782	2.105	0.0252	0.2044	9.08	7.72
CHILDREN (school age)											
N	21	21	21	21	21	21	21	21	21	21	21
$\bar{X}$	14.73	40.28	300.0	10.5	139.96	64.17	50.39	0.465	2.091	8.5	42.4
SD	0.850	1.724	1166.19	4.08	106.154	31.136	15.887	0.2569	0.9343	4.72	25.19
SE	0.186	0.376	254.48	0.89	23.165	6.794	3.467	0.0561	0.2039	1.03	5.50
CHILDREN (up to 4 years)											
N	19	19	19	19	18	19	19	14	4	5	4
$\bar{X}$	13.43	38.92	610.5	12.7	138.99	71.63	45.19	0.286	0.588	9.7	16.3
SD	1.271	1.564	1393.99	5.45	112.896	30.714	12.436	0.1643	0.7559	3.29	8.10
SE	0.292	0.359	319.80	1.25	26.610	7.046	2.853	0.0439	0.3780	1/47	4.05

TABLE 9. STATISTICAL PARAMETERS OF BIOLOGICAL DATA IN CONTROL GROUP

Statistical parameter	BLOOD							URINE			
	Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ ml E	Pb µg/ 100 ml	ALA mg/ 100 ml	ALA mg/ 24 h	CP µg/ 100 ml	CP µg/ 24 h
FATHERS											
N	16	16	16	16	16	16	16	16	16	16	16
$\bar{X}$	17.09	44.16	50.0	10.4	13.00	151.19	37.65	0.374	2.878	11.3	84.7
SD	1.134	2.700	200.0	3.71	3.335	29.461	9.278	0.1605	1.0801	5.74	38.76
SE	0.284	0.675	50.0	0.93	0.834	7.365	2.319	0.0401	0.2700	1.43	9.69
MOTHERS											
N	16	16	16	16	16	16	16	15	15	15	15
$\bar{X}$	14.74	41.47	50.0	11.9	17.36	177.74	28.23	0.355	2.922	12.5	102.2
SD	1.087	1.737	200.0	7.08	16.728	22.347	7.615	0.1043	1.5239	4.66	51.72
SE	0.272	0.434	50.0	1.77	4.182	5.587	1.904	0.0269	0.3935	1.20	13.35
CHILDREN (School age)											
N	7	7	7	7	7	7	7	7	7	7	7
$\bar{X}$	13.99	39.43	85.7	11.4	10.71	176.31	29.37	0.283	1.410	11.4	44.1
SD	1.075	1.134	226.78	6.45	3.836	12.422	4.936	0.1125	1.5062	4.96	22.60
SE	0.406	0.429	85.71	2.44	1.450	4.695	1.866	0.0425	0.5693	1.88	8.54
CHILDREN (up to 4 years)											
N	17	17	17	17	17	17	17	16	16	13	13
$\bar{X}$	13.36	38.38	400.0	10.6	13.79	188.62	30.05	0.334	0.626	10.6	23.2
SD	0.714	2.198	1104.54	3.10	4.538	20.494	6.056	0.0958	0.4025	3.73	12.92
SE	0.173	0.533	267.89	0.77	1.101	4.971	1.469	0.0239	0.1006	1.03	3.58

TABLE 10. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE WITHIN LEAD-EXPOSED GROUP 1 (FATHERS OCCUPATIONALLY EXPOSED TO LEAD)

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24h
					FATHERS & MOTHERS						
t	0.857	1.707	1.170	0.512	5.587*	6.384*	4.650	3.933*	4.072*	4.102*	4.031*
P	> 0.10	> 0.10	> 0.10	> 0.50	< 0.001	< 0.001	< 0.001	< 0.01	< 0.01	< 0.01	< 0.01
					FATHERS & CHILDREN (school age)						
t	2.277	1.813	1.954	2.620*	4.748*	4.384	1.781	4.351*	5.643*	3.890*	4.450*
P	< 0.05	> 0.05	> 0.05	< 0.05	< 0.001	< 0.001	> 0.05	< 0.01	< 0.001	< 0.01	< 0.01
					FATHERS & CHILDREN (up to 4 years)						
t	2.902	4.220	1.524	2.645*	5.104*	5.635	3.462	4.845*	6.336*	4.233*	4.831*
P	< 0.01	< 0.001	> 0.10	< 0.05	< 0.001	< 0.001	< 0.01	< 0.001	< 0.001	< 0.01	< 0.01
					MOTHERS & CHILDREN (school age)						
t	1.935	0.544	1.253	2.145	0.951	2.941	0.988	0.704	2.930*	0.559	1.749
P	> 0.05	> 0.50	> 0.10	< 0.05	> 0.10	< 0.01	> 0.10	> 0.10	< 0.02	> 0.50	> 0.05

(continued)

TABLE 10. (continued)

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24 h
	MOTHERS & CHILDREN (up to 4 years)										
t	2.831	3.569	0.499	2.168	1.364	2.243	0.810	1.577	4.615*	0.731	3.808
P	=0.01	<0.01	>0.50	<0.05	>0.10	<0.05	>0.10	>0.10	<0.01	>0.10	<0.01
	CHILDREN (school age) & CHILDREN (up to 4 years)										
t	0.487	2.087	0.916	0.000	0.046	0.944	0.537	0.858	2.483*	1.037	1.653
P	>0.50	>0.05	>0.10	>>0.50	>>0.50	>0.10	>0.50	>0.10	<0.05	>0.10	>0.10

* The significance of the difference between two groups examined was determined by method of Cochran and Cox (Ref. 16).

TABLE 11. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE WITHIN LEAD-EXPOSED GROUP 2 (FATHERS NOT OCCUPATIONALLY EXPOSED TO LEAD)

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc %0	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24 h
FATHERS & MOTHERS											
t	7.043	7.103	1.749	0.745	2.157*	3.561	6.280*	2.419*	2.341*	1.077	1.179
P	< 0.001	< 0.001	>0.05	> 0.10	<0.05	< 0.001	< 0.001	<0.05	< 0.05	> 0.10	> 0.10
FATHERS & CHILDREN (school age)											
t	7.387	8.018	0.940	1.388	0.293	1.309	3.027	0.149	3.540*	1.105	1.555
P	< 0.001	<0.001	>0.10	> 0.10	>0.50	> 0.10	< 0.01	>0.50	< 0.01	> 0.10	> 0.10
FATHERS & CHILDREN (up to 4 years)											
t	9.376	11.262	0.015	0.184	0.258	1.991	4.008*	2.908	5.522	0.982	1.910
P	< 0.001	<0.001	>>0.50	>>0.50	>0.50	> 0.05	< 0.001	<0.01	< 0.001	> 0.10	> 0.05
MOTHERS & CHILDREN (school age)											
t	0.981	0.302	0.358	1.929	2.796*	2.621	4.305*	1.968	2.075	0.226	2.912
P	> 0.10	>0.50	>0.50	>0.05	>0.01	< 0.02	< 0.001	>0.05	< 0.05	> 0.50	< 0.01

(continued)

TABLE 11. (continued)

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ ml E	Pb µg/ 100 ml	ALA mg/ 100 ml	ALA mg/ 24 h	CP µg/ 100 ml	CP µg/ 24 h
MOTHERS & CHILDREN (up to 4 years)											
t	2.473	2.769	1.209	0.533	2.445	1.970	3.458	1.146	4.892	0.544	6.160
P	<0.02	<0.01	<0.10	>0.50	<0.05	>0.05	<0.01	>0.10	<0.001	>0.50	<0.001
CHILDREN (school age) & CHILDREN (up to 4 years)											
t	3.755	2.616	0.760	1.434	0.027	0.762	1.158	2.513	3.500	0.696	3.821
P	<0.001	<0.02	>0.10	>0.10	>>0.50	>0.10	>0.10	<0.02	<0.01	>0.10	<0.001

* The significance of the difference between two groups examined was determined by method of Cochran and Cox (Ref. 16).

TABLE 12. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE WITHIN CONTROL GROUP

Statistical parameter	BLOOD							URINE			
								ALA		CP	
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	mg/100 ml	mg/24 h	µg/100 ml	µg/24 h
FATHERS & MOTHERS											
t	5.976	3.352	0.000	0.750	1.022	2.872	3.139	0.393	0.092	0.643	1.061
P	<0.01	<0.01	>0.50	>0.10	>0.10	<0.01	<0.01	>0.50	>0.50	>0.50	>0.50
FATHERS & CHILDREN (school age)											
t	6.257	5.914*	0.360	0.383	1.369	2.876*	2.782*	1.557	2.330	0.042	3.143
P	<0.01	<0.01	>0.50	>0.50	>0.10	<0.01	<0.02	>0.10	<0.05	>0.50	<0.01
FATHERS & CHILDREN (up to 4 years)											
t	11.217	6.720	1.284	0.166	0.572	4.212	2.769	0.857	7.816	0.397	5.953
P	<0.01	<0.01	>0.10	>0.50	>0.50	<0.01	<0.01	>0.10	<0.01	>0.50	<0.01
MOTHERS & CHILDREN (school age)											
t	1.535	3.343	0.360	0.166	1.502	0.196	0.428	1.431	2.185	0.493	3.666*
P	>0.10	<0.01	>0.50	>0.50	>0.10	>0.50	>0.50	>0.10	<0.05	>0.50	<0.01

(continued)

TABLE 12. (continued)

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24 h
	MOTHERS & CHILDREN (up to 4 years)										
t	4.281	4.496	1.284	0.673	0.826	1.455	0.757	0.584	5.653	1.201	5.716
P	<0.01	<0.01	>0.10	>0.50	>0.10	>0.10	>0.10	>0.50	<0.01	>0.10	<0.01
	CHILDREN (school age) & CHILDREN (up to 4 years)										
t	1.428	1.535	1.117	0.313	1.692	1.800	0.286	1.046	1.356	0.373	2.257
P	>0.10	>0.10	>0.10	>0.50	>0.10	>0.05	>0.50	>0.10	>0.10	>0.50	<0.05

x The significance of the difference between two groups examined by the method Cochran and Cox (Ref. 16).

TABLE 13. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE BETWEEN LEAD-EXPOSED GROUP 1 (FATHERS OCCUPATIONALLY EXPOSED TO LEAD) AND LEAD-EXPOSED GROUP 2 (FATHERS NOT OCCUPATIONALLY EXPOSED TO LEAD)

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24 h
FATHERS											
t	1.892	2.420	1.238	1.961	5.213*	4.279*	1.990	4.663*	4.304*	1.762	1.647
P	>0.05	<0.05	>0.10	>0.05	<0.001	<0.001	>0.05	<0.001	<0.01	>0.05	>0.10
MOTHERS											
t	2.037	1.325	1.343	0.784	0.937	1.849	2.456*	1.787	1.778	0.148	0.339
P	>0.05	>0.10	>0.10	>0.10	>0.10	>0.05	<0.05	>0.05	>0.05	>0.05	>0.05
CHILDREN (school age)											
t	1.084	0.682	0.303	0.138	0.050	2.551*	0.765	0.349	0.956	0.789	0.248
P	>0.10	>0.50	>0.50	>0.50	>0.50	<0.02	>0.10	>0.50	>0.10	>0.10	>0.50
CHILDREN (up to 4 years)											
t	1.232	0.459	0.463	1.206	0.155	2.549*	1.173	1.382	0.330	0.997	0.283
P	>0.10	>0.50	>0.50	>0.10	>0.50	<0.05	>0.10	>0.10	>0.50	>0.10	>0.50

* The significance of the difference between two groups examined was determined by method of Cochran and Cox (Ref. 16).

TABLE 14. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE BETWEEN LEAD-EXPOSED GROUP 1 (FATHERS OCCUPATIONALLY EXPOSED TO LEAD) AND CONTROL GROUP

Statistical parameter	BLOOD							URINE			
								ALA		CP	
	Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ ml E	Pb µg/ 100 ml	mg/ 100 ml	mg/ 24 h	µg/ 100 ml	µg/ 24 h
FATHERS											
t	2.235	1.943	2.294*	2.849*	7.014*	18.232*	5.717*	5.113*	4.994*	3.150*	4.595*
P	<0.05	>0.05	<0.05	<0.02	<0.001	< 0.001	<0.001	<0.001	<0.001	<0.01	<0.001
MOTHERS											
t	1.219	0.541	1.952	1.393	2.895*	10.500	3.340*	2.356*	1.314	2.241	2.160
P	>0.10	>0.50	>0.05	>0.10	<0.02	< 0.001	<0.01	<0.05	>0.10	<0.05	<0.05
CHILDREN (school age)											
t	0.410	1.773	0.742	0.260	3.339*	4.758	2.258*	2.202	0.425	0.447	0.342
P	>0.50	>0.05	>0.10	>0.50	<0.02	< 0.001	>0.05	>0.05	>0.50	>0.50	>0.50
CHILDREN (up to 4 years)											
t	1.649	1.160	0.109	0.075	5.956*	19.121	3.625*	0.905	0.522	1.872	0.622
P	>0.10	>0.10	>0.50	>0.50	<0.001	< 0.001	<0.01	>0.10	>0.50	>0.05	>0.50

* The significance of the difference between two groups examined was determined by method of Cochran and Cox (Ref. 16).

TABLE 15. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE BETWEEN LEAD-EXPOSED GROUP 2 (FATHERS NOT OCCUPATIONALLY EXPOSED TO LEAD) AND CONTROL GROUP

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24 h
FATHERS											
t	0.674	0.192	2.675*	1.433	4.491*	9.289	5.412*	1.623	1.853	0.834	0.977
P	>0.50	>0.50	<0.02	>0.10	<0.001	< 0.001	<0.001	>0.10	>0.05	>0.10	>0.10
MOTHERS											
t	0.864	1.689	1.201	0.798	4.973*	7.311*	1.656	0.298	0.523	0.404	2.094
P	>0.10	>0.05	>0.10	>0.10	>0.001	< 0.001	<0.10	>0.50	>0.50	>0.50	<0.05
CHILDREN (school age)											
t	1.657	1.490	0.798	0.346	5.569*	13.579*	5.339*	2.586*	1.126	1.353	0.167
P	>0.10	>0.10	>0.10	>0.50	<0.001	< 0.001	<0.001	<0.02	>0.10	>0.10	>0.50
CHILDREN (up to 4 years)											
t	0.206	0.840	0.505	1.430	4.701*	13.567	4.718*	0.960	0.097	0.501	1.276
P	>0.50	>0.10	>0.50	>0.10	<0.001	< 0.001	<0.001	>0.10	>0.50	>0.50	>0.10

* The significance of the difference between two groups was determined by method of Cochran and Cox (Ref. 16).

TABLE 16. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE BETWEEN LEAD-EXPOSED GROUP 1 (FATHERS OCCUPATIONALLY EXPOSED TO LEAD), LEAD-EXPOSED GROUP 2 (FATHERS NOT OCCUPATIONALLY EXPOSED TO LEAD), AND CONTROL GROUP

Statistical parameter	BLOOD							URINE			
	Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	ALA mg/100 ml	ALA mg/24 h	CP µg/100 ml	CP µg/24 h
FATHERS											
F	1.195	3.138	4.483	5.721	46.674	87.654	19.326	25.584	27.479	3.499	3.464
P	>0.20	>0.05	<0.02	<0.01	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	<0.05	<0.05
MOTHERS											
F	2.023	1.717	3.036	1.008	7.743	37.747	8.997	7.553	2.546	4.119	3.483
P	>0.05	<0.20	>0.05	>0.20	< 0.005	< 0.001	< 0.001	< 0.005	< 0.10	<0.02	<0.05
CHILDREN (school age)											
F	1.883	1.279	0.146	0.120	4.999	61.306	4.025	1.894	1.175	1.048	0.067
P	<0.20	>0.20	>0.20	>0.20	< 0.02	< 0.001	< 0.05	< 0.20	> 0.20	>0.20	>0.20
CHILDREN (up to 4 years)											
F	1.262	0.744	0.166	1.336	13.843	142.638	11.489	1.583	0.139	1.303	0.543
P	>0.20	>0.20	>0.20	>0.20	< 0.001	< 0.001	< 0.001	> 0.20	> 0.20	>0.20	>0.20

TABLE 17. AIR-LEAD CONCENTRATION ($\mu g/m^3$) AT FIVE SITES IN LEAD SMELTER AREA

Date		Sampling sites				
		1. (4.0 km N)*	2. (0.5 km N)*	3. (1.5 km SSW)*	4. (2.0 km SSW)*	5. (2.5 km SW)*
Month	Year	Pb $\mu g/m^3$				
XII	1973	24.21	15.53	39.69	13.39	45.11
I	1974	14.69	11.61	48.11	16.43	44.95
II		17.09	9.31	25.83	9.06	22.61
III		21.94	13.19	25.89	9.48	20.03
IV		12.69	8.54	8.71	16.92	6.79
V		22.80	13.97	9.50	18.26	31.19
VI		16.86	17.45	13.38	16.81	10.04
VII		12.82	8.93	7.14	8.87	5.35
VIII		6.76	10.91	12.55	16.46	11.19
IX		2.07	7.68	19.81	13.47	11.30
X		3.03	31.99	24.07	19.25	13.83
XI		5.39	30.64	34.05	26.36	22.80
XII		5.90	21.00	18.60	16.00	13.80
I	1975	5.00	19.20	16.20	15.60	12.00
II		4.10	22.20	26.90	19.50	17.60
III		2.90	21.30	17.80	11.60	14.50
IV		3.90	25.00	12.50	8.80	7.60
V		3.30	11.50	10.00	13.50	9.60
VI		3.80	22.00	17.30	18.50	12.90
VII		4.40	6.90	5.70	5.20	4.20
VIII		6.30	5.40	4.80	5.00	3.10
IX		3.30	32.30	33.30	22.40	21.10
X		4.70	22.90	22.70	23.00	9.40
XI		9.00	35.80	44.50	31.70	14.20
XII		23.30	35.10	59.50	54.20	55.10
I	1976	16.20	24.40	28.50	25.30	17.70
II		9.40	16.30	37.10	32.80	36.30
III		11.40	19.30	25.80	11.70	14.80
IV		11.00	13.80	9.10	4.80	5.00
V		6.60	14.30	6.70	7.30	11.50
VI		14.50	6.70	8.00	8.40	5.50
VII		13.90	6.60	18.30	22.80	14.00
VIII		13.30	17.30	15.60	11.10	10.90
IX		14.40	15.90	13.60	13.20	8.10
X		13.90	16.80	35.30	31.90	27.00

* Relative position of sampling sites (1-5) to the smelter.

TABLE 18. STATISTICAL PARAMETERS OF AIR-LEAD CONCENTRATION ($\mu g/m^3$) AT FIVE SITES IN LEAD SMELTER AREA (DECEMBER 1973-OCTOBER 1976)

Statistical parameter	Sampling sites				
	1. (4.0 km N)*	2. (0.5 km N)*	3. (1.5 km SSW)*	4. (2.0 km SSW)*	5. (2.5 km SW)*
	Pb $\mu g/m^3$				
N	35	35	35	35	35
$\bar{X}$	10.41	17.48	21.62	17.12	16.89
SD	6.551	8.480	13.306	9.915	12.383
SE	1.107	1.433	2.249	1.676	2.093

* Relative position of sampling sites (1-5) to the smelter.

TABLE 19. STATISTICAL PARAMETERS OF ANNUAL AIR-LEAD CONCENTRATION ($\mu g/m^3$) AT FIVE SITES IN LEAD AREA (DECEMBER 1973-OCTOBER 1976)

Statistical parameter	Sampling sites				
	1. (4.0 km N)*	2. (0.5 km N)*	3. (1.5 km SSW)*	4. (2.0 km SSW)*	5. (2.5 km SW)*
	Pb $\mu g/m^3$ DECEMBER 1973-NOVEMBER 1974				
N	12	12	12	12	12
$\bar{X}$	13.36	14.97	22.39	15.40	20.43
SD	7.689	8.188	13.123	5.002	13.719
SE	2.220	2.364	7.788	1.444	3.960
	Pb $\mu g/m^3$ DECEMBER 1974-NOVEMBER 1975				
N	12	12	12	12	12
$\bar{X}$	4.69	20.46	19.19	15.90	11.67
SD	1.721	9.050	11.459	7.805	5.222
SE	0.497	2.613	3.308	2.253	1.507
	Pb $\mu g/m^3$ DECEMBER 1975-OCTOBER 1976				
N	11	11	11	11	11
$\bar{X}$	13.44	16.95	23.41	20.32	18.72
SD	4.226	7.882	16.024	14.937	15.261
SE	1.274	2.370	4.831	4.504	4.601

* Relative position of sampling sites (1-5) to the smelter.

TABLE 20. STATISTICAL SIGNIFICANCE OF THE DIFFERENCE BETWEEN AIR-LEAD CONCENTRATION ($\mu g/m^3$) AT FIVE SITES IN LEAD SMELTER AREA (DECEMBER 1973-OCTOBER 1976)

Comparable sampling sites	Statistical parameter	
	t	P
1 - 2	3.904	<0.001
1 - 3	4.472	<0.001
1 - 4	3.341	<0.01
1 - 5	2.737	<0.01
2 - 3	1.552	>0.1
2 - 4	0.163	>0.5
2 - 5	0.233	>0.5
3 - 4	1.604	>0.1
3 - 5	1.540	>0.1
4 - 5	0.086	>0.5

TABLE 21. AIR-LEAD CONCENTRATION ($\mu g/m^3$) AT ONE SITE IN CONTROL AREA

Date		Sampling site in community G
Month	Year	Pb $\mu g/m^3$
XI	1974	0.09
XII		0.11
I	1975	0.10
II		0.19
III		0.05
IV		0.13
V		0.11
VI		0.08
VII		0.03
VIII		0.04
IX		0.08
X		0.12

TABLE 22. STATISTICAL PARAMETERS OF AIR-LEAD CONCENTRATION ($\mu g/m^3$) AT ONE SITE IN CONTROL AREA (NOVEMBER 1974-OCTOBER 1975)

Statistical parameter	Sampling site in community G Pb $\mu g/m^3$
N	12
$\bar{X}$	0.09
SD	0.044
SE	0.013

TABLE 23. LEAD AMOUNT IN DUSTFALL (mg/m^2/month) AT FOUR SITES IN LEAD SMELTER AREA

Date	Sampling sites			
	1. (4.0 km N)*	2. (0.5 km N)*	3. (1.5 km SSW)*	4. (2.5 km SW)*
Month Year	Pb mg/m^2/month			
XI 1975	126	419	612	401
XII	94	248	437	331
I 1976	104	333	312	203
II	-	225	287	253
III	110	58	268	119
IV	70	218	137	129
V	26	61	57	50
VI	5	15	7	30
VII	59	257	316	185
VIII	184	201	91	104
IX	58	108	120	46
X	21	147	486	135

* Relative position of sampling sites (1, 2, 3, 5) to the smelter.

TABLE 24. STATISTICAL PARAMETERS OF LEAD AMOUNT IN DUSTFALL ($mg/m^2/month$) AT FOUR SITES IN LEAD SMELTER AREA (NOVEMBER 1975-OCTOBER 1976)

Statistical parameter	Sampling sites			
	1. (4.0 km N)*	2. (0.5 km N)*	3. (1.5 km SSW)*	4. (2.5 km SW)*
	Pb $mg/m^2/month$			
N	11	12	12	12
$\bar{X}$	77.9	190.8	260.8	165.5
SD	52.54	119.14	186.22	115.40
SE	15.84	34.39	53.76	33.31

* Relative position of sampling sites (1, 2, 3, 5) to the smelter.

TABLE 25. LEAD AMOUNT IN DUSTFALL ($mg/m^2/month$) AT ONE SITE IN CONTROL AREA

Date		Sampling site in community G
Month	Year	Pb $mg/m^2/month$
XI	1975	0.30
XII		-
I	1976	0.30
II		1.10
III		1.80
IV		7.53
V		0.90
VI		2.73
VII		3.17
VIII		3.17
IX		2.40
X		1.37

TABLE 26. STATISTICAL PARAMETERS OF LEAD AMOUNT IN DUSTFALL (mg/m^2/month) AT ONE SITE IN CONTROL AREA (NOVEMBER 1975-OCTOBER 1976)

Statistical parameter	Sampling site in community G Pb mg/m^2/month
N	11
$\overline{X}$	2.25
SD	2.038
SE	0.615

TABLE 27. LEAD CONTENT OF HOUSEHOLD DUST (µg/g) IN LEAD SMELTER AREA (DECEMBER 1976)

Home (father's initials)	Community	Group 1	Group 2
		Pb µg/g	
K.N.	A	6300	
V.F.	A	5100	
J.K.	A	5100	
K.S.	B_1	4000	
Š.B.	C_1	3000	
H.A.	C_3	1400	
Š.L.	D	1300	
T.M.	A		6700
J.J.	B_1		6800
P.H.	B_2		2000
P.J.	B_2		1000
Č.F.	D		1700
B.D.	D		1200
P.D.	D		1000

TABLE 28. STATISTICAL PARAMETERS OF LEAD CONTENT IN HOUSEHOLD DUST (µg/g) IN LEAD SMELTER AREA (DECEMBER 1976)

Statistical parameter	Group 1	Group 2
	Pb µg/g	
N	7	7
$\bar{X}$	3742.8	2914.3
SD	1927.74	2646.02
SE	728.62	1000.10

TABLE 29. LEAD CONTENT OF HOUSEHOLD DUST (µg/g) IN CONTROL AREA (OCTOBER 1975)

Home (father's initials)	Community	Pb µg/g
V.S.	F	200
V.F.	F	160
V.I.	F	160
M.F.	G	250
J.F.	G	310
N.I.	G	200
J.A.	E	130
O.D.	E	80
A.I.	E	120
A.G.	H	110
P.J.	H	120
J.F.	H	50

TABLE 30. STATISTICAL PARAMETERS OF LEAD CONTENT IN HOUSEHOLD DUST (µg/g) IN CONTROL AREA (OCTOBER 1975)

Statistical parameter	Pb µg/g
N	12
$\bar{X}$	157.5
SD	73.13
SE	21.11

TABLE 31. WATER-LEAD CONCENTRATION (µg/l) IN LEAD SMELTER AREA (DECEMBER 1976)

Home (father's initials)	Community	Type of water supply	Pb µg/l
Ž.H.	B_2	Domestic running water system	12.9
G.E.	B_2	Domestic running water system	11.1
P.J.	B_2	Public water supply	10.0
O.S.	C_1	Well	7.4
P.J.	C_3	Well	9.7

TABLE 32. WATER-LEAD CONCENTRATION (µg/l) IN CONTROL AREA (OCTOBER 1975)

Home (father's initials)	Community	Type of water supply	Pb µg/l
J.A.	E	Well	2.4
O.D.	E	Well	2.6
V.I.	F	Well	2.5
V.S.	F	Well	1.5
M.F.	G	Public water supply	0.3

TABLE 33. RATIO CONTENT IN VARIOUS ENVIRONMENTAL MEDIA BETWEEN THE EXPOSED AND THE CONTROL AREA

Medium	Ratio in lead content
Air	178 : 1
Dustfall	77 : 1
Household dust	21 : 1
Water	6 : 1

TABLE 34. ESTIMATED LEAD ABSORPTION (µg Pb/day/kg) FROM THE AIR IN POPULATION FROM LEAD-EXPOSED AND CONTROL AREA*

Family relationship group	Lead-exposed area	Control area
	µg Pb/day/kg	
Fathers	1.060	0.006
Mothers	1.079	0.006
Children (school age)	2.264	0.012
Children (up to 4 years)	2.323	0.014

x Calculated on basis of the average air-lead concentration of 16.73 µg/m^3 in the exposed area and 0.09 µg/m^3 in the control area, and under the assumptions described on page 15.

TABLE 35. BIOLOGICAL INDICES OF LEAD ABSORPTION IN COMPARISON WITH MEDIAN RESIDENTIAL DISTANCE OF FATHERS (GROUP 2) FROM LEAD SMELTER

Biological index	Distance (m) of habitation from lead smelter			
	500 150 - 800	1900 1900 - 1900	3150 3000 - 3400	4500 4200 - 6500
Hb g/100 ml	16.6 (2) 16.6 - 16.6	16.1 (4) 15.0 - 17.0	17.5 (3) 16.7 - 17.8	17.3 (11) 15.6 - 18.5
Hct %	44.5 (2) 44.0 - 45.0	43.5 (4) 42.0 - 44.5	45.5 (3) 44.0 - 45.7	44.0 (11) 41.0 - 46.0
BpE/10^6E	100 (2) 0 - 200	750 (4) 0 - 3200	0 (3) 0 - 1600	500 (11) 0 - 2400
Rtc %o	9.5 (2) 9 - 10	15.0 (4) 6 - 22	12.0 (3) 5 - 17	12.0 (11) 8 - 20
EP µg/100 ml E	198.5 (2) 115.1 - 282.0	118.6 (4) 36.2 - 371.7	91.8 (3) 69.2 - 410.9	69.7 (11) 47.7 - 294.2
ALAD units/ml E	19.1 (2) 7.1 - 31.1	33.0 (4) 5.9 - 52.5	59.6 (3) 13.1 - 68.1	57.9 (11) 16.6 - 145.5
Pb-B µg/100 ml	105.8 (2) 87.9 - 123.7	52.5 (4) 35.0 - 79.4	101.6 (3) 69.8 - 110.1	59.7 (11) 35.0 - 95.8
ALA-U mg/100 ml	0.64 (2) 0.62 - 0.66	0.40 (4) 0.25 - 1.00	0.32 (3) 0.15 - 0.72	0.43 (11) 0.22 - 0.79
ALA-U mg/24 h	5.61 (2) 4.96 - 6.27	3.06 (4) 1.90 - 7.00	3.60 (3) 1.05 - 4.48	4.08 (11) 0.94 - 7.11
CP-U µg/100 ml	11.5 (2) 10.5 - 12.5	10.7 (4) 5.0 - 210.0	8.5 (2) 6.5 - 10.5	10.0 (11) 4.5 - 15.5
CP-U µg/24 h	101.0 (2) 84 - 119	108.4 (4) 25 - 1470	96.5 (2) 46 - 147	69.0 (11) 20 - 217

All values are expressed in median/range.
( ) Number of subjects.

TABLE 36. BIOLOGICAL INDICES OF LEAD ABSORPTION IN COMPARISON WITH MEDIAN RESIDENTIAL DISTANCE OF MOTHERS (GROUP 1 AND GROUP 2) FROM LEAD SMELTER

Biological index	Distance (m) of habitation from lead smelter			
	500 150 - 800	1900 1900	3150 3000 - 3400	4500 4200 - 6500
Hb g/100 ml	14.7 (4) 13.8 - 17.1	14.4 (6) 13.8 - 15.6	15.7 (6) 14.0 - 16.7	14.5 (17) 11.0 - 16.2
Hct %	40.7 (4) 40.0 - 41.0	40.2 (6) 39.5 - 42.5	40.2 (6) 41.0 - 43.0	40.0 (17) 35.5 - 44.5
BpE/10^6 E	0 (4) 0 - 900	200 (6) 0 - 2100	0 (6) 0 - 0	0 (17) 0 - 3200
Rtc %o	13.0 (4) 8 - 31	14.5 (6) 9 - 16	18.5 (6) 8 - 25	11.0 (17) 5 - 30
EP µg/100 ml E	161.5 (4) 117.3 - 205.1	84.4 (6) 31.9 - 297.6	59.0 (6) 23.6 - 142.7	45.0 (17) 8.7 - 113.8
ALAD units/ml E	55.5 (4) 19.4 - 124.1	55.1 (6) 23.4 - 91.0	96.9 (6) 55.0 - 131.4	95.8 (17) 52.7 - 221.8
Pb-B µg/100 ml	37.1 (4) 27.6 - 68.8	37.2 (6) 21.4 - 70.0	47.0 (6) 29.3 - 71.1	32.4 (17) 17.2 - 47.2
ALA-U mg/100 ml	0.36 (4) 0.22 - 0.38	0.33 (5) 0.20 - 0.83	0.31 (6) 0.16 - 0.79	0.38 (16) 0.29 - 1.37
ALA-U mg/24 h	2.78 (4) 2.20 - 3.04	3.00 (5) 2.31 - 9.13	2.53 (6) 1.56 - 4.74	2.82 (16) 0.66 - 5.48
CP-U µg/100 ml	7.7 (4) 4.5 - 12.5	9.5 (5) 2.0 - 15.0	7.7 (6) 6.0 - 13.5	8.7 (16) 3.5 - 21.6
CP-U µg/24 h	69.0 (4) 34 - 100	114.0 (5) 14 - 150	69.0 (6) 42 - 105	55.5 (16) 14 - 108

All values are exposed in median/range.

( ) Number of subjects.

TABLE 37. BIOLOGICAL INDICES OF LEAD ABSORPTION IN COMPARISON WITH MEDIAN RESIDENTIAL DISTANCE OF SCHOOL AGE CHILDREN (GROUP 1 AND GROUP 2) FROM LEAD SMELTER

Biological index	Distance (m) of habitation from lead smelter			
	500 150 - 800	1900 1900 - 1900	3150 3000 - 3400	4500 4200 - 6500
Hb g/100 ml	14.5 (5) 13.6 - 15.5	14.5 (4) 14.1 - 15.3	14.4 (5) 13.0 - 15.3	15.0 (15) 12.6 - 16.3
Hct %	40.0 (5) 38.5 - 42.0	40.0 (4) 40.0 - 40.5	39.5 (5) 38.7 - 42.5	41.7 (15) 37.0 - 43.2
BpE/10^6 E	0 (5) 0 - 1200	0 (5) 0 - 5300	0 (5) 0 - 700	0 (15) 0 - 1000
Rtc ‰	9.0 (5) 6 - 15	12.0 (5) 7 - 15	13.0 (5) 7 - 24	9.0 (15) 6 - 15
Ep µg/100 ml	214.4 (5) 121.0 - 442.0	120.6 (4) 61.3 - 305.0	87.1 (5) 65.9 - 320.3	76.0 (15) 29.8 - 246.5
ALAD units/ml E	22.7 (5) 16.2 - 59.2	35.1 (4) 21.7 - 48.7	54.4 (5) 27.2 - 100.3	66.9 (15) 37.7 - 123.6
Pb-B µg/100 ml	73.0 (5) 39.0 - 127.3	42.8 (4) 37.2 - 65.3	55.1 (5) 25.7 - 78.8	44.0 (15) 17.6 - 64.8
ALA-U mg/100 ml	0.45 (5) 0.36 - 1.29	0.45 (5) 0.16 - 0.54	0.41 (5) 0.15 - 0.78	0.40 (15) 0.24 - 0.93
ALA-U mg/24 h	1.80 (5) 1.08 - 4.10	2.25 (5) 1.15 - 2.63	1.92 (5) 0.93 - 2.05	2.00 (15) 0.38 - 4.62
CP-U µg/100 ml	16.0 (5) 10.5 - 22.5	10.0 (5) 6.5 - 15.5	4.5 (5) 3.5 - 8.5	7.0 (15) 3.0 - 13.5
CP-U µg/24 h	34.0 (5) 22 - 105	63.0 (5) 35 - 98	23.0 (5) 9 - 34	35.0 (15) 5 - 74

All values are expressed in median/range.

( ) Number of subjects.

TABLE 38. BIOLOGICAL INDICES OF LEAD ABSORPTION IN COMPARISON WITH MEDIAN RESIDENTIAL DISTANCE OF CHILDREN UP TO 4 YEARS (GROUP 1 AND GROUP 2) FROM LEAD SMELTER

Biological index	Distance (m) of habitation from lead smelter			
	500 150 - 800	1900 1900 - 1900	3150 3000 - 3400	4500 4200 - 6500
Hb g/100 ml	14.5 (4) 13.2 - 14.9	13.8 (5) 10.4 - 14.9	13.4 (6) 11.8 - 14.9	13.6 (15) 11.8 - 15.8
Hct %	39.7 (4) 38.0 - 41.0	40.0 (5) 36.5 - 41.0	39.2 (6) 36.0 - 40.0	38.0 (15) 37.0 - 42.5
BpE/10^6 E	0 (4) 0 - 800	0 - (5) 0 - 1700	0 (6) 0 - 1200	0 (15) 0 - 5100
Rtc %o	7.0 (4) 7 - 14	13.0 (5) 7 - 16	9.5 (6) 5 - 15	11.0 (15) 7 - 24
Ep µg/100 ml E	160.1 (4) 90.3 - 206.0	178.5 (4) 99.0 - 532.0	95.2 (6) 60.1 - 317.4	86.7 (15) 43.3 - 188.5
ALAD units/ml E	57.6 (4) 43.9 - 72.1	34.2 (5) 24.6 - 73.7	65.2 (6) 22.5 - 109.1	63.8 (15) 29.6 - 114.6
Pb-B µg/100 ml	52.1 (4) 38.6 - 70.8	39.7 (5) 32.2 - 68.5	44.9 (6) 22.9 - 91.8	47.6 (15) 28.4 - 78.3
ALA-U mg/100 ml	0.25 (4) 0.09 - 0.40	0.13 (4) 0.03 - 0.40	0.40 (3) 0.19 - 0.43	0.46 (12) 0.13 - 0.71
ALA-U mg/24 h	-	0.36 (1)	1.33 (3) 0.32 - 1.72	0.28 (7) 0.17 - 1.10
CP-U µg/100 ml	-	12.5 (1)	6.5 (3) 6.0 - 7.0	9.0 (7) 6.0 - 13.5
CP-U µg/24 h	-	25.0 (1)	24.0 (3) 6 - 46	10.0 (6) 3 - 23

All values are expressed in median/range.

( ) Number of subjects.

REFERENCES

1. Nelson, W.E., ed. Textbook of Pediatrics, 8th ed. W.B. Saunders Company, Philadelphia, Pa., 1964. 1636 pp.

2. Alexander, F.W., H.T. Delves, and B.E. Clayton. The Uptake and Excretion by Children of Lead and Other Contaminants. In: Proceedings of the International Symposium on Environmental Health Aspects of Lead, Commission of the European Communities, Luxembourg, 1973. pp. 319-331.

3. Kehoe, R. A. The Metabolism of Lead in Man and Health and Disease. I. The Normal Metabolism of Lead. J. Roy. Inst. Public Health Hyg., 24:81-97, 1961.

4. Chisolm, J.J., M.B. Barrett, and E.D. Mellits. Dose-Effect and Dose-Response Relationships for Lead in Children. J. Pediatr., 87:1152-1160, 1975.

5. Zielhuis, R.L. Dose-Response Relationships for Inorganic Lead. I. Biochemical and Haematological Responses. Int. Arch. Occup. Health, 35:1-18, 1975.

6. Nordberg, G.F., ed. Effects and Dose-Response Relationships of Toxic Metals. Elsevier Scientific Publishing Company, Amsterdam, Oxford, New York, 1976. 559 pp.

7. International Committee for Standardization at Haematology. Recommendations for Haemoglobinometry in Human Blood. Br. J. Haematol. (Suppl.), 13:71-73, 1967.

8. Wintrobe, M.M. Clinical Hematology, 5th ed. Lea and Febiger, Philadelphia, Pa., 1961. 1186 pp.

9. Hamel, C. On the Relationship Between Nuclear Changes of the Red Blood Cells and the Other Morphological Changes of the Blood with Special Regard to Lead Poisoning (in German). Dtsch. Arch. Klin Med., 67:357-376, 1900.

10. Cripps, D.J., and H.A. Peters. Fluorescing Erythrocytes and Porphyrin Screening Tests in Urine, Stool and Blood. Arch. Dermatol., 96:712-721, 1967.

11. Bonsignore, D., P. Calissana, and C. Cartasegna. A Simple Method for the Determination of δ-Aminolevulinic Acid Dehydratase in Blood. Behavior of the Enzyme in Lead Poisoning. (in Italian) Med. Lavoro, 56:199-209, 1965.

12. Fernandez, T.T. Micromethod for Lead Determination in Whole Blood by Atomic Absorption, With Use of the Graphite Furnace. Clin. Chem., 21:558-561, 1975.

13. Prpić-Majić, D., J. Firm, A. Keršanc, and B. Matijević. Lead Determination in Blood by Flameless Atomic Absorption Spectrophotometry (in Croatian). In: Processings of the Fourth Yugoslav Congress of Occupational Health, Documenta, Belgrade, 1975. pp. 434-436.

14. Davis, J.R., and S.L. Adelman. Urinary Delta-Aminolevulinic Acid (ALA) Levels in Lead Poisoning. 1. A Modified Method for the Rapid Determination of Urinary Delta-Aminolevulinic Acid Using Disposable Ion-Exchange Chromatography Columns. Arch. Environ. Health, 15:53-59, 1967.

15. Schwartz, L. Zieve, and C.J. Watson. An Improved Method for the Determination of Urinary Coproporphyrin and an Evaluation of Factors Influencing the Analysis. J. Lab. Clin. Med., 37:843-859, 1951.

16. Cochran, W.G., and G.M. Cox. Experimental Designs. John Wiley and Sons, New York, 1950. 455 pp.

17. Zielhuis, R.L. Biological Quality Guide for Inorganic Lead. Int. Arch. Arbeitsmed., 33:103-127, 1974.

18. Secchi, G.C., L. Alessio, and G. Cambiaghi. Studies on ALA-Dehydratase Activity of Erythrocytes in Subjects not Occupationally Exposed to Lead, Living in Urban and Rural Areas. Med. Lavoro, 62:435-450, 1971.

19. Baloh, R.W. Laboratory Diagnosis of Increased Lead Absorption. Arch. Environ. Health, 28:198-208, 1974.

20. Haeger-Aronsen, B., M. Abdulla, and B.I. Fristedt. Effect of Lead on δ-Aminolevulinic Acid Dehydratase Activity in Red Blood Cells. Arch. Environ. Health, 29:150-153, 1974.

21. Sassa, S., J.L. Granick, S. Granick, A. Kappas, and R.D. Levere. Studies in Lead Poisoning. 1. Microanalysis of Erythrocyte Protoporphyrin Levels by Spectrofluorometry in the Detection of Chronic Lead Intoxication in Subclinical Range. Biochem. Med., 8:135-148, 1973.

22. Finelli, V.N., D.S. Klauder, and M. Karaffa. Interaction of Zinc and Lead on δ-Aminolevulinate Dehydratase. Biochem. Biophys. Res. Commun., 65:303-311, 1975.

23. Haeger-Aronsen, B., A. Schütz, and M. Abdulla. Antagonistic Effect in Vivo of Zinc on Inhibition of δ-Aminolevulinic Acid Dehydratase by Lead. Arch. Environ. Health, 31:215-220, 1976.

24. Klauder, D.S., and H.G. Petering. Protective Value of Dietary Copper and Iron Against Some Toxic Effects of Lead in Rats. Environ. Health Perspect., 12:77-80, 1975.

25. Landrigan, P.J., E.L. Baker, R.G. Feldman, D.H. Cox, K.V. Eden, W.A. Orenstein, J.A. Mather, A.J. Yankel, and J.H. von Lindern. Increased Lead Absorption With Anemia and Slowed Nerve Conduction in Children Near a Lead Smelter. J. Pediatr., 89:904-910, 1976.

26. Wibowo, A.E.E., P. Del Castilho, R.F.M. Herber, and R.L. Zielhuis. Blood Lead and Serum Iron Levels in Non-Occupationally Exposed Males and Females. Int. Arch. Occup. Environ. Health, 39:113-120, 1977.

27. Barltrop, D., and A. Smith. Interaction of Lead with Erythrocytes. Experimentia, 27:92-93, 1971.

28. Bruenger, F.W., W. Stevens, and B.J. Stover. The Association of ^{210}Pb with Constituents of Erythrocytes. Health Phys., 25:37-42, 1973.

29. Hernberg, S., J. Nikkanen, G. Mellin and H. Lilius. δ-Aminolevulinic Acid Dehydrase as a Measure of Lead Exposure. Arch. Environ. Health, 21:140-145, 1970.

30. Haeger-Aronsen, B., M. Abdulla, and B.I. Fristedt. Effect of Lead on δ-Aminolevulinic Acid Dehydrase Activity in Red Blood Cells. Arch. Environ. Health, 23:440-445, 1971.

31. Piomelli, S. A Micromethod for Free Erythrocyte Porphyrins: The FEP Test. J. Lab. Clin. Med., 81:932-940, 1973.

32. Landrigan, P.J., S.H. Gelbach, B.F. Rosenblum, J.M. Shoults, R. Candelaria, W.F. Barthel, J.A. Liddle, A.L. Smrek, N.W. Staehling, and J.D.F. Sanders. Epidemic Lead Absorption Near an Ore Smelter. The Role of Particulate Lead. N. Eng. J. Med., 16:123-129, 1975.

33. Nordman, C.H., S. Hernberg, J. Nikkanen, and A. Ryhänen. Blood Lead Levels and Erythrocyte δ-Aminolevulinic Acid Dehydratase Activity in People Living Around a Secondary Lead Smelter. Work - Environ. - Health, 10:19-25, 1973.

34. Water Quality Criteria - 1972. National Academy of Sciences, National Academy of Engineering, Environmental Studies Board. Committee on Water Quality Criteria. Washington, D.C., 1972.

35. Zielhius, R.L. Lead Absorption and Public Health: An Appraisal of Hazards. In: Proceedings of the International Symposium on Environmental Health Aspects of Lead, Commission of the European Communities, Luxembourg, 1973. pp. 631-653.

36. Fugaš, M., B. Wilder, R. Paukovič, J. Hršak, and D. Steiner-Škreb. Concentration Levels of Particle Size Distribution of Lead in the Air of an Urban and Industrial Area as a Basis for the Calculation of Population Exposure. In: Proceedings of the International Symposium on Environmental Health Aspects of Lead, Commission of the European Communities, Luxembourg, 1973. pp. 961-968.

37. Mahaffey, K.R. Quantities of Lead Producing Health Effects in Humans: Sources and Bioavailability. Environ. Health Perspect., 19:285-295, 1977.

38. Sayre, J.W., E. Charney, J. Vostal, and I.B. Pless. House and Hand Dust as a Potential Source of Childhood Lead Exposure. Am. J. Dis., 127:167-170, 1974.

39. King, B.G. Maximum Daily Intake of Lead Without Excessive Body Lead-Burden in Children. Am. J. Dis. Child., 122:337-340, 1971.

TABLE A-1. LEAD-EXPOSED GROUP 1

Subject and community		Family relationship	Age	BLOOD Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	URINE ALA mg/100 ml	ALA mg/24 h	Coproporphyrin µg/100 ml	Coproporphyrin µg/24 h
V.F.	A	f.	37 yr	14.7	42.0	7100	33	966.9	8.6	118.7	1.90	19.95	40.5	425
V.V.	A	m.	38 yr	14.9	41.0	900	16	153.5	59.0	68.8	0.38	2.85	4.5	34
V.B.	A	s.	12 yr	15.5	42.0	1200	15	212.4	22.6	115.3	1.08	1.08	21.5	22
V.A.	A	s.	1 yr	14.2	41.0	0	7	206.0	49.4	70.8	0.09	-	-	-
Ž.M.	D	f.	28 yr	14.9	41.0	900	16	804.2	1.5	92.0	2.73	6.83	49.0	125
Ž.B.	D	m.	25 yr	14.5	42.0	3200	23	36.2	82.4	17.2	0.69	4.83	13.5	95
Ž.N.	D	d.	6 yr	12.6	43.0	0	10	76.0	42.1	17.6	0.38	0.38	5.0	5
Z.B.	D	s.	2 yr	13.8	40.5	300	9	85.6	37.3	36.1	0.71	0.85	6.5	8
O.P.	D	f.	23 yr	18.2	45.0	1200	17	670.1	10.0	57.9	1.39	9.73	38.0	266
O.M.	D	m.	22 yr	15.6	41.5	700	13	25.7	75.9	42.7	0.61	0.92	10.0	15
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
O.P.	D	d.	1 yr	15.8	40.0	0	9	151.2	43.8	78.3	0.57	0.28	6.0	3
J.K.	A	f.	42 yr	16.7	43.5	800	12	479.7	16.4	85.1	1.29	11.86	78.0	718
J.A.	A	m.	34 yr	17.1	40.5	0	11	169.5	124.1	42.7	0.34	2.72	12.5	100
J.A.	A	s.	6 yr	14.5	39.0	0	9	121.0	59.2	52.7	0.45	1.13	13.5	34
J.R.	A	s.	1 yr	14.9	38.5	0	8	134.4	72.1	50.7	0.40	-	-	-
Š.B.	C_1	f.	29 yr	16.9	43.0	900	14	406.9	15.6	125.1	1.08	14.04	34.5	449
Š.B.	C_1	m.	24 yr	16.7	42.0	0	8	142.7	55.0	71.1	0.47	3.66	13.5	105
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Š.K.	C_1	d.	2 yr	14.1	40.0	0	9	317.4	22.5	91.8	0.40	0.32	7.0	6

(continued)

TABLE A-1. (continued)

Subject and community	Family relationship	Age	BLOOD Hb g/100 ml	Hct %	BpE/10^6E	Rct ‰	EP µg/100 ml E	ALAD units/ml E	Pb µg/100 ml	URINE ALA mg/100 ml	ALA mg/24 h	Coproporphyrin µg/100 ml	Coproporphyrin µg/24 h
H.A. C_3	f.	35 yr	15.6	42.5	2400	14	851.2	8.8	122.7	2.93	16.11	113.0	622
H.M. C_3	m.	28 yr	15.6	43.0	0	24	46.2	68.8	49.8	0.79	4.74	7.5	45
H.A. C_3	d.	7 yr	13.0	39.0	700	13	65.9	54.4	48.3	0.78	1.95	3.5	9
H.S. C_3	s.	3 mo	11.8	36.0	1200	15	60.1	64.2	22.9	-	-	-	-
K.A. A	f.	29 yr	16.6	44.0	800	16	436.2	7.3	124.8	1.21	8.47	40.5	284
K.M. B_1	m.	22 yr	15.6	42.0	0	16	203.6	43.1	70.0	-	-	-	-
K.D. A	s.	9 yr	14.6	40.0	0	8	389.2	16.2	127.3	0.41	4.10	10.5	105
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
S.J. D	f.	36 yr	17.9	45.5	1500	17	309.7	16.4	86.8	0.64	6.08	10.5	100
S.A. D	m.	30 yr	16.2	40.5	0	30	45.0	52.7	44.3	0.37	2.59	7.5	53
S.A. D	d.	7 yr	15.6	42.0	0	13	127.2	37.7	64.8	0.47	2.35	6.5	33
S.J. D	d.	2 yr	15.2	40.0	0	7	86.7	47.8	54.3	0.44	1.10	9.0	23
G.F. C_2	f.	32 yr	14.9	40.5	0	7	106.6	45.6	31.8	0.61	6.10	18.5	185
G.M. C_2	m.	31 yr	14.0	41.0	0	11	23.6	115.2	29.3	0.16	1.56	8.0	78
G.R. C_2	s.	7 yr	13.0	38.7	0	7	87.1	47.8	25.7	0.48	1.92	8.5	34
G.J. C_2	d.	4 yr	13.4	39.0	0	10	107.1	33.3	29.3	0.19	1.33	6.5	46
K.S. B_1	f.	38 yr	12.2	39.0	500	13	807.8	5.1	84.4	2.29	6.87	56.5	170
K.A. B_1	m.	34 yr	13.8	40.0	1500	16	297.6	23.4	60.1	0.83	9.13	9.0	99
K.B. B_1	s.	11 yr	-	-	0	15	-	-	-	0.23	1.15	15.5	78
K.S. B_1	d.	3 yr	13.9	40.0	1700	16	99.0	68.9	52.9	0.18	0.36	12.5	25

(continued)

TABLE A-1. (continued)

Subject and community	Family relationship	Age	BLOOD Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rct ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/ 100 ml	URINE ALA mg/ 100 ml	ALA mg/ 24 h	Coproporphyrin µg/ 100 ml	Coproporphyrin µg/ 24 h
S.L. D	f.	32 yr	13.4	39.0	0	31	517.1	4.8	51.5	2.50	22.50	45.0	405
S.M. D	m.	30 yr	14.5	42.0	0	9	9.3	62.5	25.7	0.55	5.06	9.0	83
S.D. D	s.	8 yr	15.0	42.5	0	6	59.1	54.7	40.3	0.24	1.20	7.0	35
S.T. D	s.	2 yr	13.3	38.0	200	11	160.6	29.6	50.7	-	-	-	-
G.R. D	f.	24 yr	17.1	43.5	0	15	272.6	21.6	116.2	0.79	6.32	5.0	40
G.A. D	m.	22 yr	14.4	38.5	900	10	8.7	95.8	38.2	1.37	5.48	3.5	14
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
G.R. D	s.	2 yr	13.3	38.0	1400	17	179.4	71.1	47.6	0.65	0.82	-	-

f. - father
m. - mother
s. - son
d. - daughter

TABLE A-2. LEAD-EXPOSED GROUP 2

Subject and community	Family relationship	Age	BLOOD Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rct ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/ 100 ml	URINE ALA mg/ 100 ml	ALA mg/ 24 h	Coproporphyrin µg/ 100 ml	Coproporphyrin µg/ 24 h
T.M. A	f.	38 yr	16.6	45.0	200	10	115.1	31.1	87.9	0.66	6.27	12.5	119
T.S. A	m.	25 yr	14.5	40.0	0	31	205.1	19.4	27.8	0.38	3.04	8.5	68
T.B. A	s.	6 yr	14.4	38.5	0	15	442.0	22.7	73.0	1.29	1.94	22.5	34
T.Z. A	s.	1 yr	13.2	38.0	800	14	90.3	65.8	53.6	0.35	-	-	-
D.M. D	f.	36 yr	17.9	43.5	2400	20	69.7	102.0	54.0	0.22	1.54	8.5	60
D.J. D	m.	35 yr	12.6	37.5	0	12	113.8	98.3	18.6	0.37	1.66	6.5	29
D.M. D	d.	12 yr	16.2	37.0	1000	15	29.6	123.6	22.0	0.35	0.70	3.5	7
D.S. D	d.	2 yr	14.2	38.5	0	24	74.1	47.1	28.4	0.52	-	-	-
C.F. D	f.	29 yr	16.6	44.5	0	9	80.6	19.6	69.2	0.34	1.87	12.5	69
C.M. D	m.	27 yr	15.3	40.0	0	14	25.5	83.4	21.5	0.33	0.66	10.0	20
C.S. D	s.	7 yr	16.3	42.0	0	8	76.0	52.1	59.7	0.38	0.95	3.0	8
C.S. D	d.	2 yr	14.5	40.0	0	14	86.2	53.1	47.7	0.17	0.17	6.5	7
S.G. D	f.	30 yr	15.7	41.0	700	11	43.8	57.9	45.8	0.41	4.10	10.5	105
S.M. D	m.	29 yr	14.8	40.0	200	10	30.7	103.1	26.5	0.29	2.90	5.5	55
S.D. D	d.	9 yr	13.8	38.0	0	10	112.2	55.6	32.8	0.54	2.70	11.0	55
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
B.D. D	f.	33 yr	16.0	43.0	500	14	17.7	82.8	59.7	0.29	2.90	4.5	45
B.H. D	m.	29 yr	15.7	39.5	1300	23	31.3	129.7	25.8	0.52	4.16	8.5	68
B.K. D	s.	7 yr	15.5	41.5	0	10	66.6	71.9	50.0	0.51	2.55	7.0	35
B.H. D	d.	1 yr	14.7	39.0	3600	20	58.9	86.5	39.8	0.24	-	-	-

(continued)

TABLE A-2. (continued)

Subject and community		Family relationship	Age	BLOOD Hb g/100 ml	Hct %	BpE/10^6E	Rct ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/100 ml	URINE ALA mg/100 ml	ALA mg/24 h	Coproporphyrin µg/100 ml	Coproporphyrin µg/24 h
D.J.	D	f.	38 yr	18.5	43.0	0	12	130.0	31.9	90.5	0.57	5.70	7.5	75
D.M.	D	m.	33 yr	14.9	40.0	0	5	58.4	165.0	47.2	0.38	4.18	5.5	61
D.F.	D	s.	7 yr	14.1	38.5	0	7	101.4	56.4	44.0	0.93	0.93	9.5	10
D.H.	D	d.	1 yr	11.8	37.0	0	16	112.5	136.8	47.9	0.49	-	13.5	-
M.J.	C_3	f.	32 yr	17.5	44.0	0	5	69.2	59.6	69.8	0.32	4.48	10.5	147
M.J.	C_3	m.	27 yr	15.2	41.0	0	25	71.8	78.6	50.2	0.24	2.40	6.0	60
M.D.	C_3	d.	6 yr	15.3	39.5	0	13	320.3	27.2	78.8	0.41	2.05	4.5	23
M.N.	C_3	d.	8 mo	13.4	39.5	700	5	83.4	109.1	54.3	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
P.T.	D	m.	31 yr	14.0	39.5	0	13	95.5	118.0	41.1	0.29	2.75	9.0	86
P.LJ.	D	d.	11 yr	15.9	42.0	0	7	37.3	123.5	51.5	0.40	2.00	5.5	28
P.S.	D	s.	1 yr	14.8	42.5	5100	24	83.7	60.5	63.8	-	-	-	-
P.J.	C_3	f.	30 yr	17.8	45.5	0	12	91.8	68.1	101.6	0.15	1.05	6.5	46
P.Š.	C_3	m.	28 yr	15.8	43.0	0	20	75.7	120.6	44.3	0.20	2.00	8.0	80
P.A.	C_3	d.	6 yr	14.4	40.5	0	24	166.0	100.3	59.4	0.17	0.93	6.0	33
P.M.	C_3	d.	3 yr	14.9	40.0	0	13	69.3	107.8	67.2	0.43	1.72	6.0	24
J.J.	B_1	f.	33 yr	16.5	44.5	0	6	36.2	36.7	42.9	0.38	1.90	5.0	25
J.M.	B_1	m.	32 yr	15.5	42.5	0	9	36.9	73.8	32.9	0.33	2.31	2.0	14
J.M.	B_1	s.	7 yr	14.7	40.0	0	8	153.4	25.9	47.2	0.45	2.25	7.0	35
J.N.	B_1	d.	2 yr	10.4	36.5	0	13	532.0	34.2	39.7	0.03	-	-	-

(continued)

TABLE A-2 (continued)

Subject and community	Family relationship	Age	BLOOD							URINE			
			Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rct ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/ 100 ml	ALA mg/ 100 ml	ALA mg/ 24 h	Coproporphyrin µg/ 100 ml	Coproporphyrin µg/ 24 h
V.A. B_1	f.	29 yr	17.0	44.0	1000	22	84.1	52.5	35.0	0.43	3.01	9.5	66
V.A. B_1	m.	31 yr	14.1	39.5	2100	15	31.9	31.6	21.4	0.25	3.00	9.5	114
V.P. B_1	s.	7 yr	14.1	40.0	5300	12	61.3	44.3	37.2	0.54	2.63	10.0	45
V.A. B_1	s.	2 yr	13.8	40.0	0	13	-	24.6	32.3	-	-	-	-
V.F. A	f.	35 yr	16.6	44.0	0	9	282.0	7.1	123.7	0.62	4.96	10.5	84
V.M. A	m.	24 yr	13.8	41.0	0	8	117.3	52.1	31.6	0.22	2.20	7.0	70
V.E. A	s.	5 yr	13.6	40.0	0	6	179.3	29.3	39.0	0.36	1.80	16.0	80
V.L. A	s.	2 yr	14.8	41.0	0	7	185.9	43.9	38.6	0.15	-	-	-
P.H. B_2	f.	32 yr	15.7	43.0	500	11	153.2	29.3	79.4	0.25	3.12	12.0	150
P.M. B_2	m.	32 yr	14.4	40.5	400	12	120.7	91.0	41.5	0.20	2.50	12.0	150
P.T. B_2	s.	8 yr	15.3	42.0	0	13	305.0	21.7	65.3	0.16	2.40	6.5	98
P.M. B_2	d.	2 yr	14.9	41.0	0	7	231.1	30.4	68.5	0.08	-	-	-
T.J. D	f.	40 yr	18.2	46.0	0	10	85.0	38.5	95.8	0.43	6.02	15.5	217
T.A. D	m.	38 yr	13.8	42.0	0	11	69.3	73.8	44.3	0.38	2.68	7.0	49
T.M. D	d.	15 yr	15.1	42.0	0	9	72.9	81.8	81.0	0.31	2.17	10.5	74
T.J. D	d.	2 yr	11.8	37.8	0	10	88.0	114.6	55.7	0.20	0.20	11.5	12
K.J. D	f.	34 yr	17.3	45.0	300	12	116.1	93.6	57.2	0.51	4.08	12.5	100
K.E. D	m.	33 yr	11.0	35.5	0	8	80.3	221.8	32.7	0.29	1.59	10.0	55
K.M. D	s.	10 yr	14.2	39.5	0	7	90.9	71.8	48.3	0.38	1.90	10.5	53
K.M. D	s.	2 yr	13.6	38.0	1400	15	43.3	83.2	45.8	0.32	-	-	-

(continued)

TABLE A-2. (continued)

Subject and community	Family relationship	Age	BLOOD Hb g/100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ ml E	Pb µg/100 ml	URINE ALA mg/100 ml	ALA mg/24 h	Coproporphyrin µg/100 ml	Coproporphyrin µg/24 h
M.F. C_3	f.	41 yr	16.7	45.7	1600	17	410.9	13.1	110.1	0.72	3.60	-	-
M.M. C_3	m.	36 yr	16.3	42.5	0	17	25.0	131.4	43.3	0.38	2.66	6.0	42
M.D. C_3	d.	10 yr	15.1	42.5	0	12	87.0	68.8	55.1	0.15	0.98	3.5	23
M.R. C_3	s.	2 yr	12.8	39.0	0	9	217.6	66.3	35.5	-	-	-	-
P.J. B_2	f.	35 yr	15.0	42.0	3200	19	371.7	5.9	62.1	1.00	7.00	210.0	1470
P.M. B_2	m.	27 yr	14.4	40.0	0	14	48.1	67.1	21.5	0.51	4.08	15.0	120
P.M. B_2	s.	8 yr	14.4	40.5	0	7	87.8	48.7	38.4	0.45	2.25	12.5	63
P.T. B_2	s.	1 yr	13.5	39.0	0	7	125.9	73.7	36.5	0.40	-	-	-
P.O. D	f.	35 yr	17.5	44.0	300	12	45.4	61.9	52.3	0.28	1.40	10.0	50
P.A. D	m.	39 yr	12.6	39.0	0	10	75.6	100.9	32.4	-	-	-	-
P.M. D	d.	11 yr	14.2	38.0	0	10	87.9	101.3	27.7	0.35	1.75	3.0	15
P.N. D	d.	2 yr	13.4	38.0	0	8	188.5	64.8	40.5	0.13	0.26	11.0	22
L.Š. D	f.	35 yr	16.1	43.7	1400	18	69.7	55.7	66.7	0.47	0.94	10.0	20
L.R. D	m.	35 yr	14.1	41.0	200	14	67.8	55.1	21.5	0.29	2.03	8.0	56
L.M. D	s.	10 yr	13.0	39.0	0	9	144.7	66.9	43.6	0.68	4.62	9.5	65
L.A. D	d.	2 yr	12.3	37.7	0	11	150.1	94.1	29.3	0.49	-	-	-
D.D. D	f.	38 yr	17.7	45.0	0	10	21.8	145.5	35.0	0.67	6.16	9.0	83
D.A. D	m.	30 yr	15.3	44.5	0	9	12.8	86.2	33.8	0.38	3.80	9.5	95
D.B. D	s.	8 yr	15.1	43.2	0	8	64.6	90.8	42.2	0.46	2.99	10.0	65
D.J. D	s.	2 yr	12.3	37.2	0	11	81.0	64.5	33.6	-	-	-	-

(continued)

TABLE A-2. (continued)

Subject and community	Family relationship	Age	BLOOD Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units/ ml E	Pb µg/ 100 ml	URINE ALA mg/ 100 ml	ALA mg/ 24 h	Coproporphyrin µg/ 100 ml	Coproporphyrin µg/ 24 h
K.V. D	f.	42 yr	15.6	44.0	0	8	294.2	16.6	65.1	0.79	7.11	6.5	59
K.Š. D	m.	41 yr	14.5	41.0	0	8	71.4	102.1	31.6	0.64	3.20	21.6	108
K.D. D	d.	11 yr	14.7	41.7	0	10	246.5	62.9	61.9	0.49	3.43	6.0	42
- -	-	-	-	-	-	-	-	-	-	-	-	-	-

f. - father
m. - mother
s. - son
d. - daughter

TABLE A-3. CONTROL GROUP

Subject and community		Family relationship	Age	BLOOD Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/ 100 ml	URINE ALA mg/ 100 ml	ALA mg/ 24 h	Coproporphyrin µg/ 100 ml	Coproporphyrin µg/ 24 h
O.D.	E	f.	44 yr	18.1	46.0	0	7	11.6	160.1	50.6	0.50	3.00	21	126
O.A.	E	m.	44 yr	15.2	42.0	0	13	9.9	177.1	44.4	0.49	2.70	9	50
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
O.M.	E	d.	2 yr	14.1	45.0	0	10	9.4	170.6	29.3	0.42	0.84	11	22
H.A.	F	f.	36 yr	16.7	45.0	0	12	20.9	147.2	49.1	0.67	3.35	21	105
H.M.	F	m.	35 yr	15.9	42.0	0	10	22.3	173.2	39.7	0.28	3.36	13	156
H.A.	F	d.	9 yr	14.1	40.5	0	9	7.2	173.1	35.4	0.45	1.40	17	53
H.R.	F	s.	3 yr	12.3	38.0	400	12	24.6	161.8	29.1	0.31	1.09	15	53
V.F.	F	f.	35 yr	16.6	44.5	0	7	17.8	91.0	30.0	0.19	3.04	9	144
V.C.	F	m.	35 yr	13.5	38.5	0	11	11.6	212.7	31.9	0.54	6.75	8	100
V.A.	F	d.	7 yr	13.4	38.5	0	7	17.9	170.5	24.4	0.23	0.92	9	36
V.M.	F	d.	3 yr	13.2	36.0	0	8	12.6	205.6	28.8	0.39	1.56	6	24
P.F.	E	f.	38 yr	16.7	45.0	0	13	10.4	129.2	47.2	0.38	0.68	12	23
P.M.	E	m.	36 yr	13.7	40.0	0	10	9.2	178.4	33.0	0.29	1.31	6	27
P.J.	E	d.	10 yr	13.9	37.5	0	10	10.8	195.0	35.4	0.41	4.72	8	92
P.M.	E	d.	2 yr	13.8	38.0	0	16	15.1	172.0	26.4	0.35	0.28	20	16
P.I.	E	f.	46 yr	16.6	38.0	0	11	11.6	185.9	42.2	0.19	3.42	4	72
P.S.	E	m.	40 yr	16.1	44.0	0	10	10.3	156.5	24.6	0.29	4.06	9	126
P.G.	E	d.	14 yr	16.1	40.0	600	14	11.2	190.0	29.9	0.28	0.48	20	34
P.B.	E	d.	2 yr	13.2	39.5	200	10	9.3	199.1	22.8	0.23	0.55	12	29

(continued)

TABLE A-3. (continued)

Subject and community		Family relationship	Age	BLOOD Hb g/ 100 ml	Hct %	BpE/ 10^6E	Rtc ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/ 100 ml	URINE ALA mg/ 100 ml	ALA mg/ 24 h	Coproporphyrin µg/ 100 ml	Coproporphyrin µg/ 24 h
V.I.	F	f.	43 yr	19.0	40.0	800	22	18.2	195.6	49.3	0.73	5.11	12	84
V.A.	F	m.	41 yr	14.8	42.0	0	34	9.0	179.8	37.3	0.31	1.86	13	78
V.J.	F	d.	6 yr	12.7	40.0	0	25	11.6	176.5	31.0	0.19	0.67	8	28
V.E.	F	d.	3 yr	13.4	36.0	0	9	10.9	217.0	32.4	0.38	0.57	10	15
N.I.	G	f.	39 yr	18.3	40.0	0	8	12.9	202.9	50.8	0.27	2.43	13	117
N.H.	G	m.	37 yr	14.1	41.0	0	8	11.5	213.4	29.6	0.28	2.80	21	210
N.M.	G	s.	9 yr	13.4	40.5	0	8	10.4	170.4	26.0	0.14	0.42	10	30
N.A.	G	s.	1 yr	12.7	37.0	0	8	10.6	152.0	28.2	-	-	-	-
V.S.	F	f.	35 yr	18.1	46.0	0	9	12.6	174.7	22.4	0.25	2.65	15	158
V.A.	F	m.	25 yr	14.6	40.0	800	24	15.4	153.1	18.8	0.28	3.64	12	156
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
V.H.*	F	d.	2 yr	15.0	40.0	4600	10	11.4	217.5	13.2	0.38	0.76	12	24
V.S.*	F	d.	2 yr	14.5	40.0	600	12	12.0	206.9	29.4	0.47	0.71	8	12
K.F.	F	f.	38 yr	17.0	45.0	0	10	10.6	162.2	28.2	0.29	2.12	12	88
K.M.	F	m.	32 yr	15.7	42.0	0	8	5.6	129.8	24.8	0.39	1.95	15	80
K M.	F	s.	10 yr	14.3	39.0	0	7	5.9	158.7	23.5	0.28	1.26	8	36
K.F.	F	s.	3 yr	13.5	37.0	400	12	8.9	185.1	32.6	0.32	0.64	8	16
J.F.	G	f.	46 yr	18.1	46.0	0	7	11.9	145.7	31.5	0.45	4.05	7	63
J.M.	G	m.	43 yr	15.2	43.5	0	10	16.9	162.9	19.1	0.30	1.38	10	46
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
J.L.	G	d.	1 yr	12.7	36.0	0	7	17.0	204.9	29.5	0.25	0.25	8	8

* - twins

(continued)

TABLE A-3. (continued)

Subject and community	Family relationship	Age	BLOOD Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/100 ml	URINE ALA mg/100 ml	ALA mg/24 h	Coproporphyrin µg/100 ml	Coproporphyrin µg/24 h
P.J. H	f.	29 yr	17.3	45.0	0	10	14.1	144.2	32.5	0.28	2.24	7	56
P.V. H	m.	27 yr	14.6	41.0	0	9	29.5	178.4	27.3	-	-	-	-
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
P.M. H	s.	2 yr	13.3	39.0	0	18	16.9	200.6	31.8	0.30	1.20	10	40
D.F. H	f.	39 yr	17.3	44.5	0	12	9.5	143.3	38.4	0.35	3.15	9	81
D.E. H	m.	38 yr	14.8	42.0	0	11	8.6	162.8	20.2	0.57	3.42	22	132
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
D.E. H	d.	1 yr	12.7	36.5	0	7	18.6	206.8	33.6	0.14	0.04	-	-
A.G. H	f.	25 yr	14.3	49.0	0	9	9.0	157.1	31.5	0.53	4.24	4	32
A.J. H	m.	21 yr	15.9	44.0	0	10	13.8	184.4	22.4	0.25	1.25	17	85
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
A.T. H	s.	2 yr	12.7	38.0	0	9	9.3	180.9	35.0	0.18	0.54	11	33
J.F. H	f.	29 yr	17.3	44.5	0	12	12.6	121.6	36.6	0.30	2.70	4	36
J.A. H	m.	31 yr	15.5	43.0	0	8	10.6	178.8	29.3	0.41	4.92	13	156
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
J.T. H	d.	2 yr	13.7	39.5	600	12	13.1	190.1	35.9	0.39	0.19	-	-
K.F. E	f.	25 yr	16.4	44.0	0	7	10.5	142.3	31.5	0.29	1.31	20	90
K.E. E	m.	22 yr	11.9	38.5	0	9	76.0	198.1	19.4	0.27	1.62	12	72
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
K.A. E	s.	11 mo	12.8	38.0	0	8	13.5	165.5	30.8	0.47	0.28	-	-

(continued)

TABLE A-3. (continued)

Subject and community	Family relationship	Age	BLOOD Hb g/100 ml	Hct %	BpE/10^6E	Rtc ‰	EP µg/100 ml E	ALAD units ml E	Pb µg/100 ml	URINE ALA mg/100 ml	ALA mg/24 h	Coproporphyrin µg/100 ml	Coproporphyrin µg/24 h
P.J. I	f.	31 yr	15.7	44.0	0	11	13.8	116.5	30.6	0.32	2.56	10	80
P.P. I	m.	29 yr	14.4	40.0	0	6	17.5	204.4	19.9	0.38	2.81	8	59
- -	-	-	-	-	-	-	-	-	-	-	-	-	-
P.J. I	s.	3 yr	13.6	39.0	0	11	21.2	170.2	42.0	0.37	0.52	7	10

f. - father
m. - mother
s. - son
d. - daughter

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2.	3. RECIPIENT'S ACCESSION NO.
EPA-600/1-78-067		

4. TITLE AND SUBTITLE	5. REPORT DATE
STUDY OF CHILDREN'S BLOOD-LEAD LEVELS WITHIN FAMILIES	November 1978
	6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
Danica Prpic-Majjic	

9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
Institute for Medical Research and Occupational Health Zagreb, Yugoslavia	1AA601
	11. CONTRACT/GRANT NO. SFCP-JF-3-570-2

12. SPONSORING AGENCY NAME AND ADDRESS	13. TYPE OF REPORT AND PERIOD COVERED
Health Effects Research Laboratory Office of Research and Development U.S. Environmental Protection Agency Research Triangle Park, N.C. 27711	14. SPONSORING AGENCY CODE EPA 600/11

15. SUPPLEMENTARY NOTES

16. ABSTRACT

Comparative studies of the biological indices of elevated exposure to lead in children and adults were conducted with the intention of reaching a better understanding of lead absorption in children. Three family groups were examined. Group 1 consisted of families who lived in the vicinity of a lead smelter and whose fathers were occupationally highly exposed to lead. Group 2 consisted of families settled in the same area, but whose fathers had no supplemental occupational exposure to lead. The third was the control group consisting of families who lived in an area with very low exposure and whose fathers were not occupationally exposed to lead. Families were selected with one child under 4 years and, if possible, another child of school age. In the environmental survey lead in air, dustfall, household-dust, and drinking-water were analyzed. Three biological parameters, erythrocyte δ-aminolevulinic dehydratase activity, erythrocyte protoporphyrin, and blood lead were determined. On the basis of these parameters the following sequence of lead absorption was established in family members living in an area with elevated lead exposure: fathers > school-age children ≃ children up to 4 years > mothers. Children with fathers occupationally exposed to lead had a slight additional lead exposure in comparison with children whose fathers had no supplemental occupational exposure to lead. It was found that the population living near a lead smelter, except for the fathers occupationally exposed to lead, had biological findings at the level of a "moderately elevated" exposure, while those occupationally exposed had "excessive" exposure.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
lead (metal) children blood analysis occupational diseases environmental surveys		06 F, T

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)*	21. NO. OF PAGES
RELEASE TO PUBLIC	UNCLASSIFIED	153
	20. SECURITY CLASS *(This page)* UNCLASSIFIED	22. PRICE

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE