RESEARCH AND EVALUATION

Toxicity, Mutagenicity, and Teratogenicity of Lead

G.B. Gerber, A. Leonard, and P. Jacquet. MUTAT RES 1980 Sep;76(2):115-41.

Authors' summary: Environmental contamination by lead has greatly increased as more and more lead is used for gasoline and in various industrial products, although severe cases of lead intoxication, as often seen in former times, have become rare owing to better health care. These facts have drawn attention to effects of lead which may arise, possibly without a threshold, at low exposure levels. Among the classical manifestations of lead intoxication are anemia and gastrointestinal, renal, and nervous symptoms. Those in the nervous system and perhaps also in the kidney may have consequences even at subclinical levels of lead exposure. Thus, the possibility that such exposure may cause mental alterations in children, induce late renal damage, or affect nerve conduction is now much debated. The mechanisms of action of lead on the developing central nervous system are still uncertain. They may be related to a composition between lead and calcium on the synaptosomal level. Among the typical stochastic risks, tumors have been found in lead-intoxicated rodents, but do not seem to occur in man, and the situation with respect to genetic risks is not entirely clear. Certain effects of lead on genetic material have been observed in simple organisms, and it appears that lead exposure in vivo or in vitro can induce light chromosomal aberrations such as gaps and fragments. Under conditions of calcium deficiency, severe aberrations such as dicentrics may also arise; but the data on man are controversial because lead is almost never the only potentially mutagenic agent to which people are exposed in an industrial environment. Nevertheless, the efficiency of lead in causing such aberrations appears low compared with that of other mutagenic agents so that genetic effects of lead do not appear of primary concern for human health. Lead can prevent implantation of the embryo, delay its growth during later stages of pregnancy and, when injected during organogenesis particularly under conditions of calcium deficiency, cause malformations. The risks to the developing human organism are difficult to evaluate owing to the large differences in lead metabolism between man and rodent, so studies on species resembling man are desirable.

Prenatal and Neonatal Toxicology and Pathology of Heavy Metals

L.W. Chang, P.R. Wade, J.G. Pounds, and K.R. Reuhl. ADV PHARMACOL CHEMOTHER 1980;17:195-231.

The adverse effects of lead on the reproductive system have been well documented. The developing organism is especially sensitive to lead poisoning. The harmful effects of lead on the fertility and reproduction of humans as a result of occupational exposure have long been recognized. Lead readily crosses the placenta, producing an increase in blood lead levels in the fetus. Mammary transfer may pose a significant source of lead to newborns; in addition, the intestinal absorption of lead may be greatly enhanced by milk. The importance of lead as a human teratogen has not been established, although spontaneous abortion in human patients as a result of high lead exposure has been reported. Evidence for these effects has been borne out by studies in rats, mice, hamsters, and other animal models. Similar results and studies are discussed for mercury and cadmium. The phenomena of metal-metal interaction and metal-element interaction within maternal and fetal systems make the overall understanding of metal toxicology more complex. Metal-induced teratology represents one of the most important areas in toxicology.

Immunotoxicology of Heavy Metals

L.D. Koller. INT J IMMUNOPHARMACOL 1980;2(4):269-79.

From author's conclusions: From data which have been accumulated, lead appears to consistently suppress most of the segments of the immune system, while cadmium has produced mixed reactions. Mercury suppresses the primary humoral immune response, while selenium enhances humoral immunity. Zinc deficiency results in atrophy of the thymus with subsequent reduction in the humoral immune capacity. Nickel deters many segments of the immune response. Many of the other metals also compromise various parts of the immune system.

This review is certainly not complete, but [it] has compiled considerable data to document the adverse effects of metals on the immune response of experimental animals. Many of these immunosuppressive features are unaccompanied by clinical signs of disease. Therefore, it is apparent that many metals are detrimental to health by mechanisms other than direct toxicity. A chemical that impairs or destroys any portion of the immune system usually limits the defense mechanism of a host to infectious agents, thereby potentiating pathogenicity of that organism. A host that is rendered increasingly susceptible to an infectious agent is likely to develop complications or succumb to what normally would be a nonfatal condition.

Behavioral Effects of Moderate Lead Exposure in Children and Animal Models: Part 1, Clinical Studies

R. Bornschein, D. Pearson, and L. Reiter. CRC CRIT REV TOXICOL 1980;8(1):43-99.

From authors' conclusions and recommendations: A total of 22 studies were reviewed which addressed the question of the effects of moderate lead exposure on the behavioral development of children. There was no clear trend indicating that moderate lead exposure caused any abnormalities in behavioral development in children, nor was there any compelling evidence which suggested that this was not the case. A further complication with respect to these studies is that many of them had methodological deficiencies which undermined the credibility of their results. The findings from all 22 studies appear to be contradictory and do not offer any resolution to the question of whether moderate lead exposure is a significant health hazard. The data reviewed do not permit unequivocal determination of the level at which lead exposure begins to constitute a significant health hazard, as reflected in impaired neurobehavioral development. The ideal course that needs to be taken in this area of research is to prospectively monitor lead exposure and behavioral development in large samples of children. Careful and persistent monitoring of lead exposure should enable the investigators to more appropriately approach the controversial question of whether moderate lead exposure affects behavioral development in children. Careful attention needs to be paid to developing and using a number of tests to tap discrete behavioral functions that mediate intellectual and social behavior. A rigorous neuropsychological approach to assessing behavioral functions should be adopted in studies as the best way of ensuring measurement of areas of skill where deficits due to moderate lead exposure are likely to occur. A final recommendation would be for investigators to adopt multivariate research designs in approaching the study of this problem.

Behavioral Effects of Moderate Lead Exposure in Children and Animal Models: Part 2, Animal Studies

R. Bornschein, D. Pearson, and L. Reiter. CRC CRIT REV TOXICOL 1980;8(2):101-52.

From authors' critique: Clarification of the nature of the interaction between lead and behavior has been sought through the use of animal models. Unfortunately, much of the data generated by these animal studies are of little use due to inappropriate exposure levels or the lack of control over, and systematic variation of, critical independent variables known or suspected to interact with lead. However, one of the most damaging aspects of these animal studies has been a lack of a clinical perspective, both in the definition of the critical issues and in the design of studies to deal with these issues. Few animal studies adequately simulate the lead exposure conditions found in young children, either with respect to the levels of exposure or the timing of the exposure. There is often an inadequate or nonexistent history of lead exposure with respect to both the external and internal dose. Correlative measures of lead toxicity in other target systems, e.g., hematopoietic or renal indices, are generally absent. This makes it impossible to assess accurately the relative sensitivity of the major target organ systems in the lead-exposed animal. The present data do not permit us to distinguish clearly between the effects of lead exposure on cognitive function (learning/memory) and effects on sensory-motor function, arousal, or motivation, which in turn can produce performance differences. Relevance of these animal data to the clinical situation and ease of interpretation of data can be increased in future studies by consideration of the following: (1) Better documentation of internal dosage levels is needed. Blood and tissue lead levels during exposure and at the time of testing are necessary. (2) Use of exposure protocols

which do not produce growth retardation or the use of experimental designs which permit an evaluation of the nature of the interaction between level of lead exposure and degree of growth retardation should be encouraged. (3) Increased numbers of litters should be included in each treatment group in order to reduce the contribution made by "litter effects." A second alternative would be to use a genetically more homogeneous test population. (4) Standard reference compounds (positive controls) or environmental manipulations which permit an evaluation of test sensitivity should be incorporated into the experimental design. (5) Inclusion of correlative indices of lead toxicity, e.g., free erythrocyte protoporphyrin levels in blood or ALA in the urine, would aid in the extrapolation of animal data to humans.

Concentrations of Lead in the Tissues of Children

P.S. Barry. BR J IND MED 1981 Feb;38 (1):61-71.

Author's abstract: Twenty-four different tissues from 73 children and infants, including stillbirths, were analyzed for lead content. In the youngest group of 49 infants aged under 1 year, including 14 stillbirths, the mean concentrations of lead in their soft tissues were all less than 0.3 ppm and nearly all were less than the concentrations found in the soft tissues of older children or of adults. The mean concentrations of lead in the bones in the infant group were greater than in their soft tissues, but still less than 1 ppm, and were 10-40 times less than in bones of adults and about 3 times less than in the bones of older children. Lower concentrations of lead were observed in the tissues of stillbirths than in those of neonatal live births, at a 95% level of significance by analysis of variance. In 24 children aged 1-16 there was no clear evidence of increase of lead concentrations in the bones with increasing age; neither was there evidence of a difference in the concentrations of lead in types of bone. Although the mean concentrations in the bones were greater in the children aged 1-16 than in those of infants aged under 1 year, the data did not suggest that a progressive accumulation of lead occurred in the bones, [until] probably [at about] the end of the second decade of life, by which time the growing phase will be nearing completion. In 18 children aged 1-9 and in 6 children aged 11-16 the concentrations of lead in the soft tissues were similar, and comparable with those observed in women. The ratio differences between ash-weight and wet-weight measurement in the different types of bone in children did not differ proportionately from the adult ratios, suggesting a similarity in the patterns of deposition of lead in bone, irrespective of age. No differences in tissue lead concentrations by sex were observed in the infant group of children, or when the concentrations in the tissues were related to the years in which the samples were obtained. Individual tissues showed different concentrations and patterns of distribution of lead, which were skewed more towards low values in the infant group than in older children. The results of other studies, of which there have not been many, were found to be in general agreement with those reported here. The exposure of infants to lead appeared to be less than in older children or in adults, probably for reasons associated with lack of availability and parental care.

In Vivo Determination of Lead in the Skeleton after Occupational Exposure to Lead

L. Ahlgren, B. Haeger-Aronsen, S. Mattsson, and A. Schutz. BR J IND MED 1980 May;37(2): 109-13.

Authors' abstract: The concentrations of lead in the phalanges and in the blood were determined in 22 subjects who had formerly been exposed to lead in a storage battery plant, which had been closed for 7 years. The bone lead concentration was measured in vivo by using an X-ray fluorescence technique in which two ^{57}Co γ-ray sources were used for generating the characteristic X-rays of lead, which were measured with a Ge(Li) detector. In three subjects the variation of the lead concentration along the forefinger was measured together with the lead concentration in the tibia. The measured lead concentrations in the phalanges were between 20 μg/g (our detection limit) and 118 μg/g. The lead concentration in the phalanges was found to increase with the length of employment, but no simple relation was found between the lead concentrations in the blood and in the phalanges. The decrease in the blood lead concentration after the cessation of exposure was followed in four subjects. Seven years after exposure had ended, the blood lead concentration was found to be more dependent on the daily intake of lead than on the release of lead from the skeleton. These experimental results could be explained by a two-compartment model using exchange rates given in publications. This model has also been used to calculate the blood lead concentration that could be achieved after a sudden release of lead from the skeleton.

The Distribution of Lead in Human Teeth, Using Charged Particle Activation Analysis

T. Al-Naimi, M.I. Edmonds, and J.H. Fremlin. PHYS MED BIOL 1980 Jul;25(4):719-26.

Authors' abstract: A technique has been developed to measure lead content in teeth by using $^3He^{2+}$ activation analysis. The α-activity produced in the teeth is recorded on plastic film alpha-track detectors (LR-115) and compared with the activity produced by the lead contained in standard glass. The concentration in teeth from three areas of the United Kingdom has been determined as a function of age. The lead content is found to increase with age in dentine and pulpal dentine, but not in enamel.

A Nuclear Microprobe Investigation of Heavy Metal Distribution in Individual Osteons of Human Femur

U Lindh. INT J APPL RADIAT ISOT 1980 Dec; 31(12):737-46.

Author's abstract: The analytical capacity of charged-particle induced interactions for assessment of multi-element distributions in mineralized tissue was explored in autopsy sections of human femur. The PIXE [particle-induced X-ray emission spectroscopy] technique was employed in a nuclear microprobe with 2.5 MeV protons. Comparison of the standard method approach and the direct utilization of analytical formulae was performed. Detection limits of a few ppm were attained for some elements, and the positional resolution was 20 μm. The analytical errors did not exceed 15%. Concentration gradients of lead and zinc resulting from occupational exposure were clearly established within individual osteons of the femur.

Role of Altered Heme Synthesis in Lead Neurotoxicity

E.K. Silbergeld and J.M. Lamon. JOM 1980 Oct;22(10):680-4.

Authors' conclusions: The effects of lead on heme synthesis can be associated with the accumulation of possibly neuroactive precursors or depletion of heme for nonhemoglobin metabolism, or both. These consequences provide support for the following conclusions: (1) Increased aminolevulinic acid (ALA) associated with lead exposure may represent not only an accessible indicator of lead exposure, but also the presence of increased production of a potential neurotoxin; (2) The inhibition of heme synthesis produced by lead may have functional significance for heme-dependent processes (hemoproteins) other than red cell hemoglobin; (3) The relationship of lead absorption, or of tissue lead concentrations, to these phenomena is unclear and requires further research; (4) The possibility that altered heme metabolism may be responsible for the neurotoxic manifestations of lead poisoning may provide an explanation of the well-described neurological similarities between lead poisoning and the acute attack forms of porphyria.

High Affinity of Lead for Fetal Hemoglobin

C.N. Ong and W.R. Lee. BR J IND MED 1980 Aug;37(3):292-8.

Authors' abstract: In vitro experiments using ^{203}Pb were performed to identify lead-binding components in human hemoglobin. Sephadex A-50 ion-exchange chromatography of hemolysate showed that different types of hemoglobin had different affinities for lead. For the hemolysate from adults, lead was present in both Hb A ($\alpha_2 \beta_2$)and HB A_2 ($\alpha_2 \delta_2$). In the hemolysate from newborn infants, the hemoglobin of fetal origin, Hb F ($\alpha_2 \gamma_2$), showed a much greater affinity for ^{203}Pb than the adult hemoglobin, Hb A ($\alpha_2 \beta_2$), obtained from maternal blood. Analysis of the ^{203}Pb-labelled hemoglobin suggested that about 82% of ^{203}Pb was in the globin polypeptide. Further analysis with carboxylmethyl (CM) cellulose chromatography indicated that the γ globin of fetal origin had a higher affinity for ^{203}Pb than the β globin, whereas α globin appeared to be unimportant in lead binding. The results of the different affinities for lead of different Hb types are discussed with regard to the effect of lead upon hemoglobin synthesis.

Simultaneous Quantitation of Zinc Protoporphyrin and Free Protoporphyrin in Erythrocytes by Acetone Extraction

D. Hart and S. Piomelli. CLIN CHEM 1981 Feb;27(2):220-2.

Authors' abstract: Erythrocyte protoporphyrin content is increased in several human and veterinary disorders in which heme synthesis is disrupted. The nature of the defect determines whether zinc protoporphyrin, or unchelated (free) protoporphyrin, or both, are present in the erythrocytes. Extraction of the protoporphyrin into acetone/water (80/20 by vol) is a simple, rapid technique by which each protoporphyrin species is delivered into solution without zinc being removed from zinc protoporphyrin. The ratio of zinc protoporphyrin to total protoporphyrin concentration correlated significantly with the ratio of fluorescence emission intensities at the zinc protoporphyrin peak and at an isosbestic point. This concentration ratio may be used with a quantitative measure of total erythrocyte protoporphyrin to define the absolute zinc protoporphyrin and free protoporphyrin content of erythrocytes.

Quantitative Assay of Erythrocyte "Free" and Zinc-Protoporphyrin: Clinical and Genetic Studies

S. Schwartz, B. Stephenson, D. Sarkar, H. Freyholtz, and G. Ruth. INT J BIOCHEM 1980; 12(5-6):1053-7.

Authors' summary: An improved extraction procedure and two fluorimetric techniques have been developed for the quantitative assay of "free" and Zn-protoporphyrin in circulating erythrocytes. Sources of error (including nonspecific fluorescence and diminished fluorescence due to absorbance of exciting light) can be essentially eliminated, especially through analysis of second derivative fluorescence spectra. As an alternative, the content of the two forms of protoporphyrin can be determined by noting the magnitude and relative intensity of fluorescence before and after acidification to remove any bound zinc. The concentration of "free" protoporphyrin was elevated specifically in acute Pb poisoning, in bovine (heterozygous) carriers of the protoporphyria trait, and in groups of renal patients undergoing dialysis. Many of the latter also showed appreciable amounts of Zn-coproporphyrin. Though the "free" protoporphyrin comprised an average of only 7%-10% of the total in normal humans, it usually constituted 25% or more of the total in normal cows, rats, rabbits, and dogs.

New Method for Liquid-Chromatographic Measurement of Erythrocyte Protoporphyrin and Coproporphyrin

M. Salmi and R. Tenhunen. CLIN CHEM 1980 Dec;26(13):1832-5.

Authors' abstract: We describe a "high-pressure" liquid-chromatographic method for separating all the porphyrins in the heme biosynthetic pathway. The preliminary extraction and purification method is such that only erythrocyte porphyrins with fewer than five carboxyl groups, which include protoporphyrin and the much smaller amount of coproporphyrin present, are measured. The porphyrins extracted from erythrocytes are chromatographically separated by use of a simple eluent system and fluorometrically detected.

"Ultra-Clean" Isotope Dilution/Mass Spectrometric Analyses for Lead in Human Blood Plasma Indicate That Most Reported Values Are Artificially High

J. Everson and C.C. Patterson. CLIN CHEM 1980 Oct;26(11):1603-7.

Authors' abstract: We measured lead concentrations in venous blood plasma from two subjects, one having a typical exposure and the other a high exposure to lead. Our preliminary data, obtained by isotope dilution/mass spectrometric techniques in an ultra-clean laboratory, show lead concentrations of 0.02 μg/l and 2 μg/l, respectively, in their blood plasma, and 110 μg/l and 800 μg/l, respectively, in samples of whole blood. These results indicate that plasma lead concentrations previously reported have been overestimated by a large factor and that further improvements in analytical procedures are needed in most laboratories before data on lead concentrations in blood plasma can be properly interpreted. Our preliminary data indicate a positive correlation between lead intake and lead concentrations in blood plasma.

Lead in Bone. I. Direct Analysis for Lead in Milligram Quantities of Bone Ash by Graphite Furnace Atomic Absorption Spectroscopy

L.E. Wittmers, Jr., A. Alich, and A.C. Aufderheide. AM J CLIN PATHOL 1981 Jan;75(1):80-5.

Authors' abstract: A method for direct lead content analysis of milligram quantities of bone ash by flameless atomic absorption spectroscopy is described. Bone ash (25 mg) is dissolved with HNO_3 and diluted with H_2O and La_2O_3 (1,000 μg/ml) solution. Lanthanum ion is used to suppress matrix interferences possibly arising in part from sulfate components of the bone ash. Two bulk bone samples (about 14 and 60 μg Pb/g ash, respectively) were used to determine daily, within-day, and overall variability of the method. Values for "low lead" bone samples were 14.08 $\pm$ 1.74 (SD) μg Pb/g ash and for "high lead" bone samples were 60.85 $\pm$ 5.24 (SD) μg Pb/g ash. The overall value of 58 lead recovery determinations from bone ash analysis was 103.5% $\pm$ 12.9% (SD). These values compare favorably with results previously reported for gram amounts of sample.

Abnormal Distribution of Lead in Amyotrophic Lateral Sclerosis—Reestimation of Lead in the Cerebrospinal Fluid

S. Conradi, L.O. Ronnevi, G. Nise, and O. Vesterberg. J NEUROL SCI 1980 Dec;48(3):413-8.

Authors' abstract: The lead concentration in CSF [cerebrospinal fluid] was determined by flameless atomic absorption spectrophotometry in 16 ALS [amyotrophic lateral sclerosis] patients and 22 control cases. The mean values were 0.69

± 0.55 (ALS) and 0.41 ± 0.37 (controls), $p < 0.01$. This confirms our earlier findings of raised CSF lead levels in ALS, but the present values are lower than previously reported for both ALS patients and controls.

The Percutaneous Absorption of Lead-203 in Humans from Cosmetic Preparations Containing Lead Acetate, as Assessed by Whole-Body Counting and Other Techniques

M.R. Moore, P.A. Meredith, W.S. Watson, D.J. Sumner, M.K. Taylor, and A. Goldberg. FOOD COSMET TOXICOL 1980 Aug;18(4):399-405.

Authors' abstract: The percutaneous absorption of lead from two hair-darkening cosmetic preparations containing lead acetate has been measured by radioisotopic tracer techniques, using lead-203 acetate, in eight normal human male subjects. Spiked preparations were applied in fluid and dried forms to each subject's forehead (with periods of 1 month between each application). The quantity of lead absorbed was calculated from blood counts, whole-body counts, and urine radioactivity. Results were normalized for each subject by administration of an IV tracer dose of lead-203 chloride, from which absorption was calculated. It was found that absorption of lead through the skin was essentially zero, with results ranging between 0 and 0.3% of the dose applied to whole skin. Slight absorption was found when the skin was broken. The potential hazard of the use of such cosmetic preparations is therefore considered to be insignificant.

SELECTED BIBLIOGRAPHY

*Ahlgren L, Haeger-Aronsen B, Mattsson S, Schutz A: In vivo determination of lead in the skeleton after occupational exposure to lead. BR J IND MED 1980 May;37(2):109-13.

*Al-Naimi T, Edmonds MI, Fremlin JH: The distribution of lead in human teeth, using charged particle activation analysis. PHYS MED BIOL 1980 Jul; 25(4):719-26.

Arutjunov VD, Batsura JD, Gribova IA, Kruglikov GG: Scanning electron-microscopic and light-optics investigations of erythrocytes in toxic anemia. BR J IND MED 1981 Feb;38(1):72-5.

*Barry PS: Concentrations of lead in the tissues of children. BR J IND MED 1981 Feb;38(1):61-71.

Bernard A, Roels HA, Buchet JP, Lauwerys RR: Comparison, by sodium dodecyl sulfate-polyacrylamide gel electrophoresis, of urinary proteins excreted by workers exposed to cadmium, mercury or lead. TOXICOL LETT 1980 Mar;5(3-4):219-22.

*Bornschein R, Pearson D, Reiter L: Behavioral effects of moderate lead exposure in children and animal models: part 1, clinical studies. CRC CRIT REV TOXICOL 1980;8(1):43-99.

*Bornschein R, Pearson D, Reiter L: Behavioral effects of moderate lead exposure in children and animal models: part 2, animal studies. CRC CRIT REV TOXICOL 1980;8(2):101-52.

*Chang LW, Wade PR, Pounds JG, Reuhl KR: Prenatal and neonatal toxicology and pathology of heavy metals. ADV PHARMACOL CHEMOTHER 1980; 17:195-231.

Chiba M, Ogihara K, Kikuchi M: Effect of a lead on leucine aminopeptiadase activity in human blood. IND HEALTH 1980;18(1):55-7.

*Conradi S, Ronnevi LO, Nise G, Vesterberg O: Abnormal distribution of lead in amyotrophic lateral sclerosis—reestimation of lead in the cerebrospinal fluid. J NEUROL SCI 1980 Dec;48(3):413-8.

*Everson J, Patterson CC: "Ultra-clean" isotope dilution/mass spectrometric analyses for lead in human blood plasma indicate that most reported values are artificially high. CLIN CHEM 1980 Oct;26 (11):1603-7.

Fischer AB, Skreb Y: Cytotoxicity of manganese for mammalian cells in vitro—comparisons with lead, mercury and cadmium. ZENTRALBL BAKTERIOL (B) 1980;171(6)525-37.

Forni A: Chromosomal effects of lead: a critical review. REV ENVIRON HEALTH 1980;3(2):113-29.

*Gerber GB, Leonard A, Jacquet P: Toxicity, mutagenicity and teratogenicity of lead. MUTAT RES 1980 Sep;76(2):115-41.

*Hart D, Piomelli S: Simultaneous quantitation of zinc protoporphyrin and free protoporphyrin in erythrocytes by acetone extraction. CLIN CHEM 1981 Feb;27(2):220-2.

*Koller LD: Immunotoxicology of heavy metals. INT J IMMUNOPHARMACOL 1980;2(4):269-79.

Lalit BY, Ramachandran TV, Rajan S: Lead-210 content of food samples in India. RADIAT ENVIRON BIOPHYS 1980;18(1):13-7.

*Lindh U: A nuclear microprobe investigation of heavy metal distribution in individual osteons of human femur. INT J APPL RADIAT ISOT 1980 Dec; 31(12):737-46.

Moore MR, Graham DJ: Monopyrroles in porphyria, psychosis and lead exposure. INT J BIOCHEM 1980;12(5-6):827-32.

*Moore MR, Meredith PA, Watson WS, Sumner DJ, Taylor MK, Goldberg A: The percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate, as assessed by whole-body counting and other techniques. FOOD COSMET TOXICOL 1980 Aug;18(4):399-405.

Morava E, Gergely A: Recent problems with lead and cadmium. BIBL NUTR DIETA 1980;29:39-47.

*Abstracted

O'Flaherty EJ, Hammond PB, Lerner SI, Hanenson IB, Roda SM: The renal handling of delta-aminolevulinic acid in the rat and in the human. TOXICOL APPL PHARMACOL 1980 Sep 30;55(3):423-32.

*Ong CN, Lee WR: High affinity of lead for fetal hemoglobin. BR J IND MED 1980 Aug;37(3):292-8.

Pleban PA, Kerkay J, Pearson KH: Polarized Zeeman-effect flameless atomic absorption spectrometry of cadmium, copper, lead, and manganese in human kidney cortex. CLIN CHEM 1981 Jan;27(1):68-72.

Rockley MG, Davis DM, Richardson HH: Fourier-transformed infrared photoacoustic spectroscopy of biological materials. SCIENCE 1980 Nov 21; 210(4472):918-20.

*Salmi M, Tenhunen R: New method for liquid-chromatographic measurement of erythrocyte protoporphyrin and coproporphyrin. CLIN CHEM 1980 Dec;26(13):1832-5.

*Schwartz S, Stephenson B, Sarkar D, Freyholtz H, Ruth G: Quantitative assay of erythrocyte "free" and zinc-protoporphyrin: clinical and genetic studies. INT J BIOCHEM 1980;12(5-6):1053-7.

*Silbergeld EK, Lamon JM: Role of altered heme synthesis in lead neurotoxicity. JOM 1980 Oct;22(10):680-4.

Valentine WN, Paglia DE: Erythrocyte disorders of purine and pyrimidine metabolism. HEMOGLOBIN 1980;4(5-6):669-81.

*Wittmers LE Jr, Alich A, Aufderheide AC: Lead in bone. I. Direct analysis for lead in milligram quantities of bone ash by graphite furnace atomic absorption spectroscopy. AM J CLIN PATHOL 1981 Jan; 75(1):80-5.

Yunis AA, Salem Z: Drug-induced mitochondrial damage and sideroblastic change. CLIN HAEMATOL 1980 Oct;9(3):607-19.

*Abstracted