REPORT OF THE AD HOC COMMITTEE TO EVALUATE THE HAZARD OF LEAD IN PAINT

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1. INTRODUCTION

On December 31, 1972, the Bureau of Product Safety of the Food and Drug Administration (FDA) became responsible for administration of a federal requirement that the lead content of paints not exceed 0.5% by weight of the dried, hardened film.* More recently, the responsibilities of the FDA in this area have been transferred by Congress to a new Consumer Product Safety Commission. Among the first questions facing the Commission is whether the 0.5% limit should be reduced to 0.06% at a date that could be as early as December 31, 1973. The 0.06% limit originated as a recommendation in a report of the Committee on Environmental Hazards of the American Academy of Pediatrics (1972).

The Ad Hoc Committee to Evaluate the Hazard of Lead in Paint was constituted by the National Academy of Sciences-National Research Council at the request of the Bureau of Product Safety to ascertain whether there was sufficient scientific evidence to support a reduction of the limit from 0.5 to 0.06%. The Committee was requested, if it found that sufficient scientific evidence did not exist, to recommend research that should be undertaken to obtain the required information.

The basic question that the Committee must answer is whether there is a sufficient body of information to permit establishment of a limit on the lead content of paints applied to surfaces accessible to children.

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*Throughout this report, when the lead content of paint, drier, or other paint additive is given, the expressed percentage of lead is that in the solid dried film.

This question has been asked many times over the years, and many regulations have been promulgated. The first, in Germany in 1887, prohibited the use of any lead in paints applied to toys. At least 10 countries have regulated the lead content of paints up to the present. Except for the German law, which called for total elimination of lead from paints used for toys, the permissible lead concentrations have ranged from 0.5 to 2%.

2. THE USE OF LEAD COMPOUNDS IN PAINT

The essential components of paints are:

(a) Pigments: to provide opacity, color, and durability.

(b) Vehicles: oils, resins, and various polymers to bind the pigments together and to provide a continuous durable film.

(c) Volatiles: organic solvents or water (depending on type of paint) to control the consistency of the coating and to aid in its application.

(d) Additives: generally in low concentrations, to aid in drying, to control consistency, settling, viscosity, and stability, and to impart other special properties to the coating.

In making a white paint, or a white-paint base that can be tinted to light pastel colors, it is necessary to have an opaque white pigment. The most important early opaque white pigment was "white lead," the basic carbonate, whose composition approximates $2PbCO_3 \cdot Pb(OH)_2$. White lead was first produced in Holland during the sixteenth century and was later introduced in England and the United States in the late 1800's. For many years, white lead was the principal opaque white

forms reaction products with oil paint vehicles (vegetable oils, such as linseed oil) that impart toughness and elasticity to the paint, in addition to opacity. Basic sulfate white lead was also used as a paint pigment (approximate composition, $2PbSO_4 \cdot PbO$). Basic sulfate white lead has lower hiding power (opacity) and tinting strength than basic carbonate white lead and therefore was not as widely used. The sulfate was more commonly combined with other pigments, such as zinc oxide, and was generally used in exterior paints. White lead pigments, when used for interior paints, contained 40-60% lead. A representative figure for interior paints used before about 1940 would be 50%.

Zinc oxide was manufactured commercially in France during the early nineteenth century and in the United States shortly thereafter. It was followed by the manufacture and use of zinc sulfide. Lithopone, a pigment that contains approximately 30% zinc sulfide and 70% barium sulfate, was developed by coprecipitating barium sulfate and zinc sulfide from solution. Paint manufacturers now had a choice of opaque white pigments, and many paints were made with a combination of these materials. Lithopone replaced white lead in many interior paints, and became a very important pigment for paint in the last half of the nineteenth and early part of the twentieth centuries.

Titanium dioxide, the opaque white pigment used in modern paints, was being produced commercially by about 1920. It was introduced to the paint industry as a titanium-barium combination pigment. It was shortly made available also as a titanium-calcium pigment and as the anatase grade of pure titanium dioxide. Also widely used for a while was titanated lithopone, containing 15% titanium dioxide. Titanium pigments had gained major acceptance by the late 1930's and largely

replaced lithopone, zinc sulfide, etc., in most interior paints. By this time, there was little economic justification for the use of white lead in any type of interior paint. By 1941, the hiding power of titanium dioxide was rated at 115 ft^2/lb, compared with about 15 ft^2/lb for the lead pigments. It is estimated that more modern pigments containing titanium dioxide have a hiding power of about 135 ft^2/lb. Thus, 1 lb of titanium dioxide will provide the opacity or hiding power of about 9 lb of white lead, and for many years there has been no necessity to use white lead pigments in interior paints. The time when all white lead was removed from interior paints is not known, but it may be assumed that relatively little white lead pigment was incorporated into interior paints after 1942.

Lead chromate yellow pigments, first made in Europe in about 1800, have long been a valuable raw material for use in paints. These pigments range from light yellow to deep orange. Lead molybdate pigments range from medium orange to deep red, and "chrome greens," from light to deep green. All these contain lead, but in a much less soluble form than white lead pigments. These lead pigments provide excellent hiding power and color permanence, and they are economical. They are widely used in coatings for automobiles, trucks, farm machinery, signs, traffic markings, buildings, and hundreds of other articles. Red lead pigments and lead silicochromate pigments are valuable for use in anti-corrosion primers for steel and iron. Some lead pigments are still used in exterior primers and paints for wood surfaces that must withstand the effects of sunlight, rain, and weather conditions of all types. Lead compounds are, and will continue to be, extremely important to the paint manufacturer as essential ingredients of many types of primers, paints, and enamels for exterior uses.

3. HISTORY OF REGULATIONS LIMITING THE LEAD CONTENT OF PAINT IN THE UNITED STATES

On March 11, 1972, an order was published under Section 2(q) (1) (B) of the Federal Hazardous Substances Act declaring paint and other similar surface-coating material containing in excess of 0.5% lead (as metal) for use in or around the household to be a hazardous substance and to be banned from shipment in interstate commerce after December 31, 1972. Separate provisions of that order banned shipment in interstate commerce after December 31, 1973, of paint and other similar surface-coating material containing in excess of 0.06% lead (as metal) for use in or around the household. The order also specifically provided, as required by Section 2(q) (1) (A) of the Act, that any toy or other article bearing such paint and other similar surface-coating material and intended for use by children would be a banned hazardous substance after one of those dates, depending on the lead content.

On August 10, 1972, the portions of the regulation pertaining to the 0.5% lead content were confirmed as effective. However, the portions pertaining to the 0.06% content were stated to be the subject of a separate document to be published at a later date. It was found by reviewing the comments received with regard to the March 11, 1972, order that the two concentrations (0.5% and 0.06% lead) and effective dates presented separate issues that required separate consideration.

3.1 APPLICABILITY OF THE REGULATIONS AND DEFINITION OF TERMS

The provisions of the Federal Hazardous Substances Act are applicable to any hazardous substance in interstate commerce that is intended or packaged in a form suitable for use in the household or by children.

The term "banned hazardous substance" contained in Section 2(q) (1) (A) means

> any toy or other article intended for use by children, which is a hazardous substance, or which bears or contains a hazardous substance in such manner as to be susceptible of access by a child to whom such toy or other article is entrusted

and under Section 2(q) (1) (B) means

> any hazardous substance intended or packaged in a form suitable for use in the household which the Secretary, by regulation classifies as a "banned hazardous substance" on the basis of a finding that, notwithstanding such cautionary labeling as is or may be required under the Act for that substance, the degree or nature of the hazard involved in the presence or use of such substance in the household is such that the objective of the protection of the public health and safety can be adequately served only by keeping such substance, when so intended or packaged, out of the channels of interstate commerce.

The regulations implementing the Federal Hazardous Substances Act--specifically, Section 191.1(c)--define a "hazardous substance intended or packaged in a form suitable for use in the household" as any hazardous substance that, under customary or reasonably foreseeable conditions of purchase, storage, or use, may be brought into or around a house, apartment, or other place where people dwell or into or around any related building or shed, including but not limited to a garage, carport, barn, or storage shed.

The regulations expressly exclude industrial supplies that might be taken into a home by a serviceman and articles labeled and marketed solely for industrial use.

The paint regulations issued under the Federal Hazardous Substances Act are applicable to paint and similar surface-coating materials containing 0.5% lead that are sold for consumer use. They include paint and similar coatings that are used for exterior and interior surfaces of dwellings and related buildings, as well as surfaces of articles

that are maintained in or around households or related buildings, such as radiators, fences, furniture, and toys.

The regulations are not applicable to paint and similar coatings that are labeled and marketed solely for industrial use, such as paints applied at a factory to automobiles, trucks, lawn mowers, tractors, and appliances. However, paint applied to toys and other articles intended for use by children cannot contain more than 0.5% lead.

A petition to amend the lead-paint regulation has been received and published as a proposal. The proposed amendment would exempt some paints and coatings containing in excess of 0.5% lead from the banned-hazardous-substance classification. Included within the requested exemption are touchup coatings for automobiles, agricultural and industrial equipment, lawn and garden equipment, outboard motors, motorized recreational vehicles, and household appliances.

A proposal to exempt artists' paints from the lead-paint regulation has been published.

Comments on the two proposals are currently under consideration within the Consumer Product Safety Commission.

3.2 OTHER FEDERAL LAWS DEALING WITH LEAD PAINT

Congress, on January 13, 1971, enacted the Lead-based Paint Poison Prevention Act of 1971. Its purpose is to eliminate childhood poisoning by lead-based paint. Section 501(3) of the law prohibits the use of paint containing in excess of 1.0% lead in residential structures built or rehabilitated by the federal government or with federal assistance.

Several bills to amend this law have been submitted to Congress during 1973. The Senate, on May 5, 1973, passed S607, the Lead-based

Paint Poison Prevention Amendments of 1973, which would amend Section 501(3) as follows:

(a) Prohibit the use of paint containing in excess of 0.5% lead before December 31, 1973.

(b) Prohibit the use of paint containing in excess of 0.06% after December 31, 1973, except that, if the Secretary of the Department of Health, Education, and Welfare determines that, on the basis of studies conducted in accordance with Section 301(b), another lead content not to exceed 0.5% is safe, then such other content shall become effective after December 31, 1973.

Section 301(b) of S507 directs the Secretary of the Department of Health, Education, and Welfare to conduct appropriate studies on dried paint films to determine the safe lead content in residential paint products and to report to the Congress not later than October 1, 1973.

Several bills amending the Lead-based Paint Poison Prevention Act of 1971 have been under consideration within the House of Representatives. We understand that the full House Banking and Currency Committee has reviewed a number of the bills before the House and reported favorably on the "Barrett bill," HR8920. This bill, we understand, will be sent to the floor of the House to be voted on. Under the provisions of this bill, Section 501(3) of the Lead-based Paint Poison Prevention Act of 1971 would be amended to prohibit the use of paint containing more than 0.5% lead. Section 301(b) of the bill directs the Secretary of the Department of Health, Education, and Welfare to conduct appropriate research on multiple layers of dried paint films with respect to safe lead content and to report the results of such investigations to Congress not later than December 31, 1974.

4. HISTORY OF PROBLEM OF COMMUNITY LEAD POISONING

Lead poisoning* among young children living in deteriorating inner-city housing emerged as an urban problem in the United States as early as the second decade of this century (Williams et al., 1952). Thomas and Blackfan (1914) of the Johns Hopkins Hospital in Baltimore were the first to describe in the American literature the clinical and pathologic effects of lead-induced encephalopathy in children. Williams and his co-workers point out that multiple cases of lead poisoning had already been reported from 19 separate communities before 1951, and it was recognized that this problem was related to pica, the habit of chewing cribs, toys, furniture, and woodwork, such as window sills, and the eating of painted plaster and fallen paint flakes. Of the affected children, 60% were between 1 and 2 years old, and fewer than 3% of the children were over 5 years old. During the 20-year period 1931-1951 in Baltimore, 293 cases were found to have clinical lead poisoning defined by a blood lead content greater than 50 µg/100 ml associated with a clinical manifestation of lead toxicity (anemia or central nervous system symptoms); 85 of the patients died.

After the early Baltimore experience, public-health departments of other major American cities began efforts to determine the extent of lead poisoning. Generally, two steps were taken: lead poisoning

*Lead poisoning is defined as a case in which the clinical signs and symptoms to be discussed later are present. Some investigators would include the presence of biochemical changes such as a reduction in delta-aminolevulinic acid dehydrase or an increase in free erythrocytic protoporphyrin. For the purposes of this report such changes are considered to be compensatory and not pathognomonic of lead poisoning.

was made a reportable disease, and separate programs for control of lead poisoning were established (in 1950 in Philadelphia). A report of the results of these measures in Philadelphia from 1955 to 1960 (Ingalls et al., 1961) noted that about 50 cases of lead poisoning were reported each year, compared with the average of 41 per year reported by the Baltimore City Health Department in 1948-1951 (Williams et al., 1952). From 10 to 20% of diagnosed patients died in that period in Baltimore (Williams et al., 1952), and neurologic disorders, including mental retardation, persisted as sequelae in some of those who survived (Perlstein and Attala, 1966).

In more recent years, cases of severe lead poisoning seem to have been less frequent. In New York City, the health department reports that fatal cases have been reduced to one or two per year, compared with about 20 per year two decades ago. But there is little information on the frequency of occurrence of the various stages of lead poisoning short of death.

During the last 10 years, the public-health programs of many cities have been expanded to include large-scale screening of children at risk. The results of some of the most significant of these screening programs are summarized in Table 1. A more complete summary of screening-program results is found in a report from the National Bureau of Standards (Gilsinn, 1972). The children included in these surveys are usually between 1 and 6 years old and said to be at risk for undue absorption of lead because they reside in deteriorating, pre-World War II housing. The surveys, for the most part, involve areas at risk within very large metropolitan communities. However, the study from Portland, Maine

(Clark and Hallett, 1971), suggests that the "risk factor" is not necessarily the size of the city but, rather, the age of the housing. This notion is supported by the study of children at risk in 14 intermediate-sized Illinois communities (Fine et al., 1972).

There is a definite seasonal variation in blood lead content; the incidence of lead poisoning in young children peaks during the summer months (Chisolm and Harrison, 1956; Ingalls, 1961; Blanksma et al., 1969; Guinee, 1972). The larger screening programs have collected blood samples during all months of the year, but some smaller studies have been restricted to the summer months.

The methods of blood lead measurement are varied, but the dithizone procedure and atomic-absorption spectroscopy have been used most often in recent years. Anodic stripping voltammetry has been introduced recently (DHEW Publication, 73-10002, 1972), as has X-ray fluorescence (Kneip and Laurer, 1972).

The studies indicate that 9.1-45.5% of children surveyed have blood lead concentrations above 40 µg/100 ml and that up to 12.5% have concentrations above 60 µg/100 ml. The Bureau of Community Environmental Management and National Bureau of Standards (Gilsinn, 1972) report that about 23% of children at risk have high blood lead content (above 40 µg/ml) and that about 5% of these children have clinical symptoms.

4.1 CORRELATION OF BLOOD LEAD CONTENT WITH PATHOLOGIC EFFECTS

There seems to be general agreement that blood lead content is the most informative single index of exposure to lead, particularly when correlated with biochemical and clinical indices of lead intoxication.

Blood lead content has been used extensively for the control of industrial exposure of workmen to lead, as well as the recognition of lead poisoning in young children with environmental or accidental exposure to lead. Nevertheless, despite all this experience, it has been difficult to determine a blood lead content above which a person can be regarded as having lead poisoning and a blood lead content below which one can confidently predict no adverse health effect. It is recognized that increasing blood lead concentrations are associated with a continuum of probable effects, extending from the concentration at which no effect is seen to those at which manifestations of lead poisoning are highly probable. It is also recognized that there may be an intermediate range of blood lead concentrations at which biochemical changes of unknown pathologic or clinical significance are seen. Such considerations have resulted in division of the "lead effect" into three to five categories (Br. Med. J., 1968; Goyer and Chisolm, 1972).

A simple scheme for correlating blood lead content with clinical manifestations of lead toxicity is presented in Table 1 (Goyer and Rhyne, 1973). The diagnostic criteria are divided into three phases: no effect, an adaptive or subclinical phase, and overt or clinical toxicity. The boundaries of these categories reflect present knowledge and are subject to modification as new information becomes available. Furthermore, the table does not reflect individual differences in susceptibility to the effects of lead. A compensated lead-industry worker may have a blood lead content near 80 or even 100 ug/100 ml without overt symptoms of lead toxicity (American Academy of Pediatrics, 1961). However, children with blood lead concentrations greater than 80 ug/ml are usually regarded as experiencing lead intoxication because

of the frequency of associated adverse effects due to lead (Chisolm, 1971). Blood lead concentration, however, is not in itself a measure of pathologic effect of lead, but must be correlated with other biochemical, functional, or clinical manifestations of lead toxicity for the diagnosis of lead poisoning to be established in a particular person.

4.2 RECOGNITION OF CLINICAL LEAD POISONING

The Public Health Service (Steinfeld, 1971) has suggested that a child whose blood lead content is over 80 ug/100 ml should be considered unequivocally to have lead poisoning and should be handled as a medical emergency. A blood lead content over 40 ug/100 ml should be interpreted as evidence of undue lead absorption. Blood lead concentrations over 80 ug/100 ml are usually associated with fivefold or greater increases in urinary delta-aminolevulinic acid and coproporphyrin, as well as an increase in free erythrocyte protoporphyrin (Goyer and Chisolm, 1972). Each of these characteristics reflects impairment of heme synthesis; such changes are usually detectable at lower blood lead concentrations than those associated with anemia.

Nervous system and renal effects of lead are usually recognizable only during the phase of overt lead toxicity. In children with blood lead concentrations above 100 ug/100 ml of whole blood, the risk of acute encephalopathy is high and the onset unpredictable. Adults with blood lead concentrations over 120 ug/100 ml are regarded as having "dangerous" exposure with possible acute symptoms and long-term sequelae. Nerve conduction, as measured by nerve impulse amplitude, may be decreased in the adaptive phase of lead toxicity, as shown by Fullerton and Harrison (1969) in workers with excessive exposure to lead.

Clinical renal effects of lead toxicity--namely, renal tubular dysfunction of the Fanconi syndrome type--occur only during clinical lead toxicity. Industrial workers with excessive lead exposure and increased urinary excretion of lead but without signs of clinical lead toxicity have only minor increases in urinary amino acid excretion (Clarkson and Kench, 1956; Goyer et al., 1972). It is unlikely, therefore, that renal tubular function is impaired to a measurable degree during the adaptive phase. Nevertheless, intranuclear inclusion bodies (lead-protein complexes) occur in renal tubular lining cells of rats at a lower dosage of lead than any functional signs of lead toxicity. It is suggested, therefore, that inclusion body formation involving the binding of renal lead in a nondiffusible lead-protein complex probably occurs during the adaptive phase of increased exposure to lead.

Overt clinical toxicity may be subdivided into early, reversible, and irreversible phases. "Reversibility" is more useful than "acute" and "chronic," because the latter terms imply time as a factor. A child with acute lead intoxication may have more significant irreversible central nervous system sequelae than one with more prolonged but less severe exposure to lead. And a child with neurologic sequelae of lead toxicity may have a normal blood lead content.

4.3 RECOGNITION OF SUBCLINICAL LEAD POISONING

The closer the blood lead content gets to 80 ug/100 ml, the more likely are clinical manifestations. The "Statement on Diagnosis and Treatment of Lead Poisoning in Childhood" issued by the American Academy of Pediatrics in 1961 recommended that "two successive determinations of 60 ug/100 ml of whole blood or higher should be obtained for a definite

diagnosis of lead poisoning." Chisolm (1965) later suggested that the limit of "normal" blood lead concentration should be lowered to 40 µg/100 ml--a recommendation that has been endorsed by Steinfeld (1972).

Although blood lead concentrations of 40-60 µg/100 ml (in the early adaptive phase) are not usually associated with clinical or even biochemical evidence of toxicity, it is not necessarily true that there are no harmful effects. Two paths of investigation have been directed toward demonstrating that adverse health effects of lead may occur with a blood lead content as low as 40 µg/100 ml; one concerns the biochemical effects of lead on heme-synthesizing enzymes, and the second involves detection of subclinical central nervous system effects.

The most sensitive biochemical indicator of an increase in blood lead is *in vitro* assay of delta-aminolevulinic acid dehydrase (ALAD) in stroma of circulating red blood cells. Hernberg and co-workers (1970) have recently shown that a decrease in ALAD activity is correlated with increased blood lead content and is a particularly useful indicator when the blood lead content is 40-50 µg/100 ml (Hernberg and Nikkanen, 1972). When the blood lead content is higher, the activity of the enzyme is too low to be useful clinically. Whether the lead-associated decrease in ALAD represents any adverse health effect is uncertain. It must be kept in mind that this test measures the *in vitro* activity of ALAD in blood hemolysate and does not necessarily reflect any impairment of this enzyme in the intact red blood cell *in vivo*. Because of the extreme sensitivity of this test, its possible clinical significance deserves full evaluation.

Recent studies suggest that subtle effects on central nervous function, particularly intelligence and behavioral activity, are present in children with blood lead concentrations over 40 µg/100 ml. A comprehensive study of 58 asymptomatic children with blood lead concentrations over 50 µg/100 ml (or over 40 µg/100 ml with urine lead content exceeding 500 µg/24 hr after ethylenediaminetetraacetate provocation) was conducted by Fuesnel and co-workers in Boston (1972). A history of mild CNS symptoms, such as clumsiness and irritability, was obtained in about one-third of the 58 children. Minor neurologic dysfunction and various forms of motor impairment were detected by more elaborate testing in 22-27% of the children tested. Chelation therapy was administered, and measures were taken to improve their home environment. Eighteen months later, significant increase in some areas of intellectual performance was observed.

Similarly, David and co-workers (1972) found higher blood and urine lead concentrations after challenge with a single dose of a chelating agent in hyperactive children than in nonhyperactive children, suggesting a relation between increased lead content in these body fluids and hyperactivity. More than half the hyperactive children (28 of 54) had blood lead concentrations between 25 and 65 µg/100 ml. These workers argue that any blood lead concentration over 24.5 µg/100 ml is dangerous and may produce central nervous system effects. In addition, de la Burde and Choate (1972) showed that 70 asymptomatic 4-year-old children with increased blood lead concentrations (mean, 33 µg/100 ml; range, 40-100 µg/100 ml) or a lead content of at least 10 µg/100 ml with positive radiographic findings of lead lines in long bones or metallic densities in the intestines had deficits in fine motor function and behavior

In contrast with previous understanding, a recent English study suggests that anemia is common in children with blood lead concentrations between 37 and 60 ug/100 ml (Betts et al., 1973).

The common conclusion of each of these studies is that blood lead content over 40 ug/100 ml must be regarded as potentially hazardous to health.

4.4 ESTIMATE OF THE NUMBER OF U. S. CHILDREN WITH INCREASED BLOOD LEAD

The National Bureau of Standards has recently published the results of mathematical models with the assumptions and data used to formulate them (Gilsinn, 1972). Estimates are given of the number of children who have increased blood lead concentrations (> 40 ug/100 ml) in 241 Standard Metropolitan Statistical Areas in the United States. Present estimates based on these models suggest that approximately 600,000 children would show increased blood lead content if tested. The models and assumptions have been only partially validated.

By using the National Bureau of Standards model in conjunction with data from the major screening programs, it is possible to construct Table 3, which gives the estimates of the numbers of cases of childhood lead poisoning of various degrees that may occur annually in the United States (Anderson, D., J. Reed, and W. Heiden, personal communication). These estimates should be regarded as first approximations that can serve only to suggest the general magnitude of the problem. Better data must await the results of well-designed epidemiologic studies conducted on a national scale.

5. METHODS OF CONTROL OF LEAD POISONING

Lead poisoning is a preventable disease that can be controlled in three ways: by eliminating the sources of lead; by controlling the means by which lead enters the body; or by finding and treating cases early. Elimination of the sources of lead is certainly the most important approach over the long term and conforms with time-honored public-health practices directed at control of the vectors of disease.

The most significant identifiable source of lead poisoning today is lead-containing paint in houses to which young children have access. However, there are other sources of lead exposure whose epidemiologic significance is not known--for example, surface dirt and the lead that comes from the use of lead-containing gasoline. A host of other lead-containing substances around households have been identified as potential sources of lead exposure, such as the paint on pencils, evaporated-milk cans, toothpaste tubes, porcelain, and earthenware. Their contribution to the lead-poisoning problem is considered to be substantially less than that of lead paint on walls and woodwork of old houses, old toys, and beds. It is unlikely that sources other than lead paints can be responsible themselves for the cases of increased blood lead content under discussion, but it is of course possible that they could make a difference in borderline cases.

Thus, control can be achieved by removing children from delapidated housing in which peeling coats of leaded paint are available to them or by removing leaded paints from the interiors of such buildings. It may also be possible to reduce the tendency toward pica by dealing with the underlying social and environmental factors that have been

shown to have a role in this behavioral anomaly. However, this Committee has been appointed to assist in developing federal policies that will prevent future lead poisoning associated with paints yet to be applied to surfaces accessible to children with pica; hence, the Committee must focus on future sources of exposure, not present or past.

Recently, the Committee on Environmental Hazards of the American Academy of Pediatrics (1972) recommended that the lead content of paints be reduced to less than 0.06%. This recommendation was based on the assumption that the risk of lead intoxication in children is increased when the daily intake, from sources other than food, exceeds 150 μg/day. Estimates of daily intake from food, water, and air have also been made by Barltrop, who gives a total of 156 μg of lead per day. Barltrop goes on to reduce the daily permissible intake of 600 μg/day for an adult to 180 μg/day for a 2-year-old, and further corrects this number for calorie requirements of a child, thereby ending up with a final value of 133 μg/day, which he suggests as a maximal permissible safe intake (Barltrop, 1973). A maximal permissible lead content of paint (0.06% of dried film) was then derived from a number of assumptions, including: (a) that gastrointestinal absorption of lead is the same from paint as from food; (b) that a child eats 1 $in.^2$ of paint per day; and (c) that six layers of paint have accumulated. The validity of the recommended maximal lead content is sensitive to these and other assumptions in the calculation.

5.1 LIMITATIONS IN THE AVAILABLE KNOWLEDGE ON WHICH A STANDARD COULD BE BASED

If a limit is to be established on a rational basis, the following questions should be answerable, particularly for children in the susceptible age group.

(a) What body burden of lead is associated with symptoms of lead toxicity?

(b) How is blood lead related to body burden of lead and signs or symptoms of lead intoxication?

(c) What daily intake of lead in paint will result in the maximal permissible body burden of lead or the maximal permissible blood lead content?

(d) How much surface area is nibbled by children?

(e) What limit on the percentage of lead in paint would keep the blood lead content or the body burden of lead within acceptable limits?

The following brief summaries of the state of our knowledge with respect to each of these questions should serve to illustrate the serious limitations in the present state of our knowledge.

3.1 ASSOCIATION OF BODY BURDEN OF LEAD WITH SYMPTOMS OF LEAD TOXICITY

It should be emphasized that lead deposited in the skeleton is only potentially toxic, as opposed to lead in soft tissues, such as kidney, brain, and liver. Thus, the significance of a given body burden of lead will depend not only on the total amount of lead present in the body, but on its distribution between the skeleton and the soft tissues.

Barry and Mossman (1970) measured the lead concentrations in the tissues of 10 English children and 49 adults described as having been exposed to a normal urban environment. Table 4 summarizes their data. The total lead body burden of the children ranged from 0.5 to 3.1 mg, with a mean of 1.5 mg. Approximately 64% of the lead [illegible] in the skeletons

of the children, compared with 95% in the adults. The mean total body burden of the 29 adult males was about 160 mg, compared with about 110 mg for the 20 adult females. An interesting comment made by the authors is that "the relatively high proportion of lead in soft tissue, compared to the lead levels in bone of young children, may be accounted for by the lack of hard, dense bone at this stage of life which, when developed in later life, appears to take up and retain lead. It is suggested that this may account, in some measure, for the adverse response in young children to severe lead exposure."

The relation of either clinical signs of lead toxicity or changes in the early indicators of abnormal lead exposures, such as blood lead concentration, erythrocyte ALAD, and free erythrocyte protoporphyrin, to either the soft-tissue or the skeletal lead burden is poorly defined. Inasmuch as lead concentrations in tissues are not usually measured before autopsy, there is no basis for a correlation between signs and symptoms and the body burden, other than for fatal encephalopathy. Chisolm and Harrison (1956) and Kehoe et al. (1933) have measured the body burdens of lead in children who died of lead intoxication. Their results indicate that the associated soft-tissue body burdens ranged from 20 to 100 mg, or from about 35 to about 200 times the "normal" soft-tissue burden. The skeletal burden, as estimated from rib analysis, varied from 100 to 400 mg, or 100-400 times the "normal" skeletal burden. The "normal" values were determined in England and may not be applicable to a U. S. population.

In summary, only a few data are available to describe the normal lead content in children's tissues, and no data are available for cases of lead intoxication other than fatal encephalopathy.

5.3 RELATION OF BLOOD LEAD TO BODY BURDEN OF LEAD AND TO SYMPTOMS OF LEAD INTOXICATION

The blood lead concentration can reflect either recent exposure or an equilibrium relationship to lead in the skeleton and soft tissues.

To evaluate the former, it is necessary to understand the clearance rates of lead from blood. Using lead-210 as a tracer, it has been determined in the baboon that lead clears in three exponential components: approximately 73% of the blood lead 1 day after injection is removed with a biologic half-time of 1.2 days, 24% with a biologic half-time of 10 days, and 3% with a biologic half-time of 4 months (Cohen et al., 1970).

No information is available from which to estimate the reservoir of lead in the tissues of a person who has a blood lead concentration higher than normal, but who is removed from exposure.

The relation of blood lead concentration to symptoms of intoxication is not adequately defined. If the blood lead concentration does not exceed 40 ug/100 ml of whole blood, there are no clinically observable effects in adults or children, although erythrocyte ALAD activity may be less at these concentrations than at blood lead concentrations below 40 ug/100 ml (Hernberg et al., 1970).

The term "asymptomatic increased lead absorption" has been coined to designate the gray area of blood lead concentrations that are significantly higher than the normal range, or from 40 to 60 ug/100 ml of blood (NAS, 1972).

The available information concerning blood lead concentrations in relation to signs and symptoms, as summarized by the NAS Committee on Biologic Effects of Atmospheric Pollutants in 1972, is given in

Table 5. It should be noted, however, that these data have been obtained, for the most part, from studies on adults.

5.4 DAILY INTAKE OF LEAD IN PAINT THAT RESULTS IN MAXIMAL PERMISSIBLE BLOOD LEAD CONTENT

Concentrations of lead in the blood of unexposed children (12-36 months old) have been reported to range from 15 to 40 ug/100 ml of whole blood, with a median of 27 ug/100 ml (Robinson et al., 1958). On the basis of Tepper's observations on adult intake (1971), the total intake of lead for a child would be 136-206 ug/day. The contribution of inspired air to the estimated totals for average lead intake is approximately 3-6%. On the basis of a gastrointestinal-absorption factor of 10%, approximately 95-185 ug/day should be excreted in feces. In fact, Chisolm and Harrison (1956) and Barltrop and Killala (1967) observed a daily excretion of 120-175 ug in feces, with a mean of 132 ug/day. The percentage of ingested lead absorbed from the gastrointestinal tract of children is one of the major uncertainties. Alexander et al. (1973) have recently reported, based on metabolic balance studies, in 8 children over a 3-day period, that absorption in children may be as high as 50% with an average retention value of 18%.

Lead concentrations in the blood of children of 40 ug/100 ml or higher are considered evidence of undue absorption of lead. On the basis of data on excretion of lead in feces immediately after the cessation of pica and of the results of balance studies (Kehoe, 1961) involving the experimental intake of soluble forms of lead, King (1971a) has estimated that a daily intake of 300 ug of lead would not result in a significant increase in the blood lead concentration. Doubling of this intake over a period of 3-6 months would result in a blood lead concentration that could exceed the defined toxicity value of 60 ug/100 ml.

5.5 SURFACE AREA NIBBLED BY CHILDREN

The quantity of lead ingested with paint is related to paint-chip size, percentage of lead in the paint, and the number of layers of paint on a particular chip. On the basis of 6.5 mg of paint per square centimeter per layer of interior paint and 16.0 mg of paint per square centimeter per layer of exterior paint, Table 6 lists the amounts of paint necessary to obtain 200 µg of lead, or two-thirds the daily calculated permissible intake for different percentages of lead in paint and different numbers of layers (King, 1971b). For 0.06% and six layers of interior paint, this would correspond to a chip of about 8.5 cm^2 or 1.3 $in.^2$ It should be noted that the rate of absorption of lead from paint chips is not known and may in fact be highly variable. The American Academy of Pediatrics report assumed a chip intake of 1 in^2/day. This would be equivalent to about 2.5 ft^2/year. There is no basis for stating whether this is a reasonable assumption, and it may in practice be impossible to obtain realistic data.

5.6 LIMIT ON PERCENTAGE OF LEAD IN PAINT THAT WOULD KEEP LEAD INTAKE BELOW DAILY PERMISSIBLE INTAKE

Assuming a daily intake of 1 in^2 of six layers of paint per day, and 5-10% absorption of lead, 0.06% lead in paint would keep the daily lead intake below the recommended 300 µg. This was the line of reasoning adopted by the American Academy of Pediatrics. However, this conclusion could be grossly modified by varying the assumptions noted above.

6. DISCUSSION

It is apparent that there is not sufficient information on which to promulgate a standard based on knowledge of the essential quantitative relations that link the lead content of paint to symptoms of intoxication. However, this is not unusual [illegible]

standards have been established by informed people who make judgments based on whatever facts are available.

An alternative to a limit based on firm scientific evidence might be one founded on a more pragmatic approach. One might establish a limit on the basis of the lowest lead content of paints found in dwellings in which lead poisoning has been reported. The only statement in the literature that deals with this question comes from Chisolm and Harrison (1956), who stated, in relation to their own experience: "No case of lead intoxication has been found where the only source of lead contained less than 1% of lead in the dried paint surface." Although this conclusion was published 17 years ago, it has not been refuted by any subsequent publications. The paints used before about 1940 contained about 50% lead on the average, and it is not surprising that a fiftyfold reduction in the lead content would reduce the attack rate to the point where cases of lead intoxication would not be observed. This might serve as a basis for setting a limit. However, one cannot be assured that effects of lead intoxication so subtle as to escape attention do not occur at lead concentrations lower than 1%.

A third approach, and one that has been used in public-health practice, would be to require that the lead in interior paints be reduced to the lowest practicable concentration. By "lowest practicable" is meant the lowest concentration that can be achieved by existing technology. It is implied that the lowest practicable concentration will be interpreted with restraint and with due recognition given to the available scientific and medical information, as well as the administrative and economic factors. A decision could be made to prohibit completely the use of lead in paints, but lead can be expected to occur in trace quantities

as an industrial impurity in the ingredients that are used to formulate paints. This requires that the phrase "no lead" be defined. If lead is not used in paint manufacture, some other materials must take its place. A judgment must be made as to whether the substitute materials involve less risk to the public health. Finally, it is possible that each progressive reduction in the lead content of paint will involve greater cost, and commensurate benefit to the public health should be ensured. Thus, reducing lead to the lowest practicable content must inevitably involve a value judgment in which one balances the likelihood of health damage at a given lead concentration against the likelihood of health damage from substitute materials, the effect of the change on the performance of the paint, and the cost of making the change.

The various approaches are fundamentally different. The first two involve interpretation of scientific data and a decision as to the lead content of paint that will keep lead poisoning from developing in the future. The "lowest practicable" approach requires value judgments in which semiquantitative, almost intuitive reasoning is used to arrive at a conclusion. A standard based on the first two approaches is clearly one that can best be developed by scientists. However, scientists are not uniquely qualified to recommend a standard based on the "lowest practicable" concept.

As has been noted, an insufficient body of information exists to permit establishment of a standard for lead in paint based on scientific facts. Although lead has been known to be a toxic material for centuries and is a major industrial chemical, there has been inadequate research. Although lead in paint has been identified as an etiologic factor in lead poisoning among children with pica, there is a paucity of epidemiologic

data with adequate case histories by means of which one could describe the association of a given child's blood lead concentration to the condition of the painted surfaces to which the child had been exposed. There should be more information about the clinical significance of blood lead short of full-blown lead poisoning. Such information cannot be expected to come into being in less than a few years.

Any proposal to reduce the lead content of paints, in addition to being concerned with the health aspects, should also consider the effect of such a change on manufacturing practices and on the quality and safety of the modified products. The course of action ultimately selected should protect the public with a minimum of deleterious effects, either on the economics of the industry or on the quality of the products.

Lead compounds are used in paints principally for three reasons: as pigments, as driers, and as agents to prevent so-called loss of drying in storage. The use of lead in pigments requires such high concentrations in the paint formulation that current regulations setting a maximum of 0.5% lead have for all practical purposes already eliminated lead pigments in household paints.

Driers (metal salts or fatty acids) help to speed the oxidative hardening of the paint film when the vehicle contains unsaturated components, such as vegetable oils or alkyd resins (Stewart, 1969; Bikales, 1964, 1966, 1970). Lead driers (typically lead 2-ethylhexoate, lead naphthenate, or lead tallate used in lead concentrations of 0.2-0.4% based on weight of dried paint film) are particularly valued, because they effect relatively rapid drying throughout the mass of the film. Other driers--such as various fatty acid salts of calcium, cobalt,

manganese, zinc, and zirconium--are commonly used in conjunction with lead driers in empirical formulations designed to give the desired rate and thoroughness of drying at widely varying temperature and humidity (Stewart, 1969).

A further use of lead compounds is as additives to retard so-called loss of drying, i.e., the deterioration in drying properties of paints that can occur in storage. For this use, proprietary lead complexes are apparently preferred and are added in concentrations about equal to or somewhat lower than those of the lead driers (based on lead content). Relatively little information is available on the types of compounds used or on the mechanism of their action (Stewart, 1969).

A reduction in the permissible concentration of lead in paints to 0.06% would in effect prohibit any intentional addition of lead compounds to pigments, driers, or other paint additives. Paints based on oleoresinous substances would require reformulation to eliminate lead driers and all other lead compounds. Analytic procedures would be necessary to ensure that no accidental contamination by lead compounds had taken place during manufacture. Atomic-absorption spectrophotometry is particularly suitable for this purpose, and a simple colorimetric method has been devised to indicate whether the proposed maximal concentration of 0.06% has been exceeded (ASTM, 1973). It can be expected that reformulation to the 0.06% lead content and the associated testing would require at least a year. The burden would be heaviest on the many small paint manufacturers that lack the necessary technical staff and resources.

Household paints are generally classified as intended for interior or exterior use. Interior paints are likely to represent the greater potential hazard because of their greater accessibility and because children, at their most susceptible age, are more often left unattended when indoors. At present, most interior paint is based on aqueous emulsions; this type of paint does not usually depend on auto-oxidation of unsaturated compounds to harden and therefore does not require any metal salts as driers. Although emulsion-based paints normally produce surfaces with little gloss, new types that can give a semiglossy finish are now on the market. These are expected to make inroads into the last major use of oleoresinous paints in the interior of households--surfaces with glossy finishes. Because interior paints usually dry under more controlled conditions and are not subjected to as severe weathering conditions as exterior paints, adequate paints of both the emulsion and the oleoresinous types can be formulated for interior use without any intentional addition of lead compounds.

Paints designed for application to exterior surfaces require a useful rate of drying and adequate durability under wide variations of climatic conditions. The combination of low temperature and high humidity (e.g., 50 F and 70% RH) is particularly stringent. The use of emulsion-based exterior paints, which require little or no drier, is increasing rapidly, but oleoresinous paints still account for a large part of the market. Lead driers play an important part in the formulation of the latter type. Lead-containing primers are also useful on heavily chalked surfaces and to prevent staining by the water-soluble extractives present in some types of wood.

In the formulation of paints, the lead driers cannot usually be eliminated by mere substitution of an equal amount of another metal salt of a fatty acid. A combination of other metal driers must be fitted to each particular recipe, and there is some question as to whether paints so reformulated are equivalent in quality to those containing lead driers.

The substitution of other driers for the lead salts also raises the important question of the safety of the substitute materials. Under current regulations, the Food and Drug Administration permits the use of some driers for coatings that come into contact with food--namely, some fatty acid salts of aluminum, calcium, cerium, cobalt, iron, lithium, magnesium, manganese, zinc, and zirconium (Code of Federal Regulations, Federal Food, Drug, and Cosmetic Act). As already pointed out, several of these metal salts are normally added to paint in conjunction with the lead compounds. It is likely that paint manufacturers will use combinations of these same driers if the lead content must be reduced to a maximum of 0.06%. Zirconium-based driers appear to be the most useful single substitute. The cost of the substitute driers, being higher than that of the lead salts, would raise the raw-material cost of the reformulated paint by several cents a gallon.

The Committee has been apprised of the fact that Chicago, Illinois, already has a local ordinance calling for a maximal lead content in paint of 0.06% of dry-weight solids. This regulation was effective on the 1st of July, 1972, and is applicable only to interior surfaces of dwellings or dwelling units (domiciles). It specifically exempts varnishes, oil stains, floor paints, and coatings in aerosol cans. Conversations with Chicago city officials have indicated that compliance

with the ordinance has been successful in perhaps 90% of the "off-the-shelf" paints tested. Furthermore, the cost of paint produced in the Chicago area was reported to be no higher than the national average. In terms of paint already existing on wall surfaces, the Chicago ordinance calls for a limit of no more than 1 mg of lead per square centimeter of surface.

At the present time, there have not been enough epidemiological data collected to determine whether the 0.06% regulation has resulted in a change in the number of cases of increased blood lead in children screened within the City of Chicago. It is hoped that, in time, the experience of the City of Chicago in administering this ordinance will provide the CPSC with additional useful information relative to many of the points raised in this report.

7. CONCLUSIONS AND RECOMMENDATIONS

(a) Although it would be desirable to establish a limit on the lead content of paints sold to consumers by a quantitative rationale that takes into consideration the quantity of paint apt to be ingested by children with pica, the rate of absorption of lead from the gastro-intestinal tract, and the amount of absorbed lead that will produce intoxication, the Committee concludes that there is at present insufficient information to permit arriving at a recommendation on that basis. A broad program of research is required to understand better the mechanisms involved in lead intoxication and methods by which the disease can be controlled. National and local health education programs to alert parents to the health hazard to children are recommended.

(b) A more pragmatic approach would be to ascertain, through epidemiologic methods, the lowest concentration of lead in paint that has been associated with lead intoxication among children with pica. The only statement in the literature that deals with this [illegible]

was published 17 years ago and stated that no case of lead intoxication had been found where the only source of lead contained less than 1% of lead in the dried paint surface. Although this conclusion has not since been refuted, the Committee is unable to conclude that subtle but nevertheless significant forms of lead intoxication that may have escaped clinical detection have not occurred from exposure to paint films with lead concentrations lower than 1%.

(c) The Committee has learned from representatives of the paint manufacturing industry that the present requirement that the lead content of paints be limited to 0.5% already precludes the use of lead compounds as pigments. However, lead driers are widely used at concentrations of 0.2-0.4% (based on lead content of the dried paint film), to help speed the oxidative hardening of the paint film when the vehicle contains unsaturated components, such as vegetable oils or alkyd resins. In particular, lead driers (typically lead 2-ethylhexoate, lead naphthenate, or lead tallate) used in lead concentrations of 0.2-0.4% are especially valued because they effect relatively rapid drying throughout the mass of the film. Other lead compounds retard the so-called loss of drying on storage. A reduction in the permissible concentration of lead in paints to 0.06% would prohibit the intentional use of lead compounds as driers or as additives to retard the so-called loss of drying.

(d) If the lead content of paints is limited to 0.06%, it would be necessary to reformulate lead driers for paints based on oleoresinous substances. This would probably require substitution of metals other than lead. Although the Committee has no reason to believe that the substitute metals would introduce new toxicologic problems, this possibility should be considered before any steps are taken that will

(e) A program of reformulation of driers to meet a 0.06% lead requirement would take at least a year to implement.

(f) Evidence made available to the Committee makes it clear that the intentional incorporation of lead into pigments and driers can be eliminated for consumer paints applied to interiors, toys, and furniture and that it may be possible to eliminate the deliberate use of lead in paints for outdoor use. However, some lead may nevertheless be present in these products, owing to its adventitious presence in process materials.

(g) Until more scientific information can be accumulated from the research programs recommended in this report, the Committee suggests that it might be wise for the Consumer Product Safety Commission to institute an interim program of controls based on the concept that the lead in paint sold to consumers be reduced to the lowest practicable concentration. The administration of this concept should be implemented with due recognition of the need for balancing costs against benefits. It should also be recognized that the weighing of societal costs against societal benefits may not always involve easily quantifiable considerations; it may be necessary to define the lowest practicable lead content on the basis of the judgments of informed people rather than scientific analysis. Tests recently undertaken by the American Society for Testing Materials have shown that "lead-free" paints contained as much as 0.03% lead. This may be due to impurities in the materials used for paint manufacture and to the occasional carryover of lead from one industrial process to the other or to uncertainties in the analytic procedures. There is no assurance at present that the lead content of lead-free paints could not exceed 0.06% owing to these factors. Thus, any regulation that governs the maximal lead content of paint products must be based on

consideration of the practicability of controlling the lead that enters the process adventitiously and of the availability of reliable analytic procedures.

(h) The Consumer Product Safety Commission should develop a national paint surveillance system to ensure that established standards are being met. As part of the development of this surveillance network, it will be necessary to sponsor the development of better analytic methods for measurment of trace quantities of lead in off-the-shelf paint samples.

(i) Any standard issued to limit the lead content of paint should be reviewed biannually in the light of whatever additional information becomes available. The limits may be raised or lowered, depending on circumstances. However, ample time must be permitted for orderly reformulation of paints and driers, and no changes should be made without adequate consideration of the effects on the public health, of the cost, and of the effects on paint quality. The overriding considerations must of course be related to protection of the public health.

(j) The Consumer Product Safety Commission should sponsor research to identify the sources of adventitious lead in paints and driers.

The unusual opportunities for absorption of lead by children with pica has caused the Committee to recommend that use of lead in paints be reduced to the greatest practicable extent. However, the Committee emphasizes that there may be other uses of lead, now and in the future, that could bring significant benefits to the consumer. In accordance with good public-health practice, more must be learned about the mechanisms of lead intoxication, so that, as future applications for lead are developed, they may be incorporated into consumer products without undue

risk. The Committee has identified several research objectives as having particular importance.

8. RESEARCH REQUIREMENTS

(a) The "normal" body burden of lead in people of various ages should be better established for a variety of modes of habitation and socioeconomic classes.

(b) The absorption of lead from the gastrointestinal tract should be measured in experimental animals and children having a normal dietary intake of lead. The studies of children can be accomplished using the lead content of the normal diet.

(c) Comparative studies of the absorption of lead from the gastrointestinal tract of suitable experimental animals should be undertaken with regard to lead in the normal diet and various paints and driers.

(d) The clinical significance of increased blood lead content should be studied in children, with particular emphasis on the adaptive or subclinical phases.

(e) Epidemiologic studies should be extended to include the sequelae of overt lead intoxication in children who survive acute toxic manifestations.

(f) Better analytic methods should be developed to measure lead in off-the-shelf paint samples and body tissues, especially blood.

(g) Health departments and hospitals of major urban centers should be encouraged to adopt mutually compatible protocols for mass screening of children and for identification of the signs and symptoms of lead poisoning. Standard procedures should also be adopted for documenting the lead content of paint to which the children were exposed.

(h) The Consumer Product Safety Commission should serve as a national registry for data accumulated in the screening programs and should issue consolidated annual reports of the findings of such studies and the incidence of community lead poisoning in the United States.

TABLE 1

Results of Screening Programs of Blood Lead Concentrations of Children (1-6) Living in Deteriorated Housing in U. S. Cities

City	Year	Number of Children	Fraction of Children with Increased Blood Lead, %				Comment	Reference
			40 µg/100 ml	50 µg/100 ml	60 µg/100 ml	80 µg/100 ml		
Baltimore	1968	665	25.3		5.6	2.1		Lin-fu, 1972
	1969	746	28.9		7.2	2.8		
	1970	939	31.5		7.4	1.4		
Chicago	1967-70	120,000	20	4				
New Haven	1969-70	1,897	29.8		9.5			
Newark	1970	594	33.9		7.4			
Washington, D.C.	1970	1,158	22.0	12		2.2		
New York City	1969*	2,648	45.5	24.5	12.5			Guinee, 1972
	1970	84,493	28.7	12.6	5.9			
	1971	87,559	23.7	10.3	4.6			
HCEM Survey 27 cities	1971-72	2,309	9.1-41.6					DHEW Publ. No. 73-10002, 1972
Portland, Me.	1970	905			1.5		Initial screening for U-ALA 0.54 mg/100 ml	Clark and Bullett 1971
Boston	1972	705	16.3	5.8	5.4	1.0	Initial screening for hair lead 100 µg/g	Pueschel et al 1972

* Bureau of Lead Poisoning Control, New York City Department of Health

TABLE 2

Relations of Various Measurements of Lead Toxicity to Category of Lead Effect (Modified from Goyer and Rhyne, 1973)

I	I No effect,	II_a Adaptive,	II_b Subclinical,	III Clinical Toxicity,
Blood Lead µg/100 ml	<40 µg/100 ml	40-60 µg/100 ml	60-80 µg/100 ml	>80 µg/100 ml
FEP	normal	slight increase		large increase
Urinary ALA Urinary CP	normal	slight increase		> 5 fold
Anemia	none	reticulocytosis		usually
Nervous system effects	none	decreased nerve conduction?		ataxia, coma convulsions
Renal effects	none	none (inclusion bodies?)		Fanconi syndrome chronic nephropathy

ALA = Delta-Aminolevulinic Acid.
CP = coproporphyrin
FEP = free erythrocyte protoporphyrin

TABLE 3

National Data on Incidence and Medical Significance

OF COMMUNITY CHILDHOOD LEAD POISONING

Children in Susceptible Age Group (under 5 years old)	20,000,000
Children at Risk (under 5 years old, live in metropolitan areas, families in poverty classification)	2,500,000
Children with Increased Blood Lead Content (blood lead content above 40 ug/100 ml)	600,000
Children with Symptomatic Lead Poisoning per Year	30,000
Children with Asymptomatic Lead Poisoning per Year (all should be treated)	80,000
Children with Neurologic Handicaps, Including Mental Retardation	6,000
Children Requiring Lifetime Institutional Care	150
Deaths per Year (includes acute and long-term consequences of lead poisoning)	200

The children in susceptible age group and children at risk are based on Bureau of Census data which have been rounded off. Children at risk are under 6 years old living in dilapidated housing containing hazardous concentrations of lead.

Of those at risk, 24% are estimated to have blood lead concentrations of at least 40 ug/100 ml of whole blood. The percentage is the median values of surveys in Illinois and New York City. If other cities are used, the percentage is higher. There is, however, little information from the West and from small cities.

From current screening programs--such as those in New York, Philadelphia, and Chicago--it appears that about 5% of the children with blood lead concentrations over 40 ug/100 ml have clinical signs or symptoms--convulsions, anemia, cramps, etc.

The estimate of asymptomatic children with blood lead concentrations about 60 ug/100 ml of whole blood is 3.2% of the population at risk. This estimate is based on the results of Illinois and New York City screening projects.

Children with neurologic handicaps, including mental retardation, are estimated at 20% of the cases of symptomatic lead poisoning, on the basis of follow-up studies in Chicago.

"Human ...
and Soft Tissues of Adults ...

Subjects	No.	Age, Years	Skeletal Lead Burden, mg	Skeletal Lead, % of Total Body Burden	Soft-Tissue Lead Burden, mg	Total Body Lead Burden, mg
Children, both sexes	10	<6	1.0 (0.21-2.37)	64	0.55 (0.12-1.58)	1.53 (0.5-3.1)
Adult males	29	10-80+	153 (21-341)	94	9.5 (5.3-21.1)	162.19 (27-352)
Adult females	20	20-80+	107 (12-237)	95	5.7 (2.6-8.4)	112.49 (19-244)

Note: Numbers in parentheses are ranges.
All numbers have been rounded off.

[a]Data from Barry and Mossman (1970).

TABLE 5

Level and Types of Effects of Inorganic Lead Salts as Related to Estimates of Various Levels of Absorption—Recent and Remote*

Type of Effects	Level I: No Demonstrable in vivo Effect	Level II: Minimal Subclinical Metabolic Effect	Level III: Compensatory Biologic Mechanisms Invoked	Level IV: Acute Lead Poisoning		Level V: Late Effects of Chronic or Recurrent Acute Lead Poisoning
				Mild	Severe	
Metabolic (excessive urinary excretion of heme precursors)	Changing ALAU[a]	Slight increase in urinary ALA may be present	ALA, UCP, FEP progressively increased	ALA, UCP, FEP increased 5- to 100 fold		Increased if excessive exposure recent, but may not be increased if excessive exposure remote
Functional injury: Hematopoietic	None	None known	Shortened red cell life-span, reticulocytosis (±) (reversible)	Shortened red-cell life-span and reticulocytosis with or without anemia (reversible)		Anemia (±) (reversible)
Kidney (renal tubular function)	None	None known		Amino-aciduria, glycosuria (±) (reversible)	Fanconi syndrome (reversible)	Chronic nephropathy[b] (permanent)
Central nervous system	None	None known		Mild injury (?? reversible)	Severe injury (permanent)	Severe injury[b] (permanent)
Peripheral nerves	None	None known		Rare	Rare	Impaired conduction (wrist, foot drop usually improve slowly, but may be permanent)
Clinical effects	None	None known	Non-specific mild symptoms (may be due in part to coexisting diseases)	Colic, irritability, vomiting	Ataxia, stupor, coma, convulsions	Mental deficiency (may be profound), seizure disorder, renal insufficiency (gout) (permanent)
Index of level of recent or current absorption:						
Blood lead, μg/100 g of whole blood	<40	40-60	50-120+	>80 With anemia, intercurrent disease: 50-100+	>80	May be normal
Urine lead (μg/liter of urine), random	-	<80	<130	>130 [May be less in severe illness]	>130	Spontaneous excretion may be normal

[a] See p. 105 for discussion of clinical levels of ALAU.
[b] CaEDTA mobilization test in chronic nephropathy is positive; may or may not be positive in permanent central nervous system injury.

* National Academy of Sciences (1972).

TABLE 6

Lead Content of Paint*

Lead Content, %	Lead per Sq. Cm. Per Layer (Micrograms) ($\frac{1}{1000}$ Milligrams)	No. Sq. Cm. to Contain 200µg Non-Food Lead: 2/3 DPI				No. Layers Paint in Surface Covering Containing 30ug Pb/Sq. Cm. -- (0.03mg Pb/Sq. Cm.) Number
		Per Layer Sq. Cm.	Per Layer Sq. In.	Per 6 Layers Sq. Cm.	Per 6 Layers Sq. In.	
INTERIOR PAINT (6.5 mg/cm^2/layer)						
1.00*	65.0ug	3.1	0.48	0.51	0.08	Less than 1
0.50	32.50	6.2	0.96	1.0	0.15	1
0.20	13.0	15	2.32	2.5	0.39	2-3
0.10	6.50	30.1	4.7	5.0	0.77	4-5
0.06	3.90	51.3	8.9	8.5	1.3	7-8
0.05	3.25	61.5	9.3	10.3	1.6	9
0.01	1.95	102.6	15.9	17.0	2.6	14-16
EXTERIOR PAINT (16.0 mg/cm^2/layer)						
1.00	160ug	1.2	0.19.	0.2	0.03	Less than 1
0.50	80.0	2.5	0.39	0.4	0.06	1
0.20	32.0	6.3	.93	1.1	0.17	2
0.10	16.0	12.5	1.93	2.1	0.33	2
0.06	9.6	20.8	3.24	3.5	0.54	3
0.05	8.0	25.0	3.9	4.3	0.67	4
0.03	4.8	41.7	6.5	7.0	1.08	6

* Maximum indicated in industry standard Z-66 -- assumed to be the lead content of paint applied since the 1940's.

*King, 1971.

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