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Certain Aspects of the Problem of the Pollution of the External and Internal Environment by Lead

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Studies on the pollution of the biosphere by lead published in recent years are discussed. Numerous sources from which lead can be introduced into the atmosphere and the hydrosphere and, the problems of the contamination of the soil, plants, algae, and animal and human organisms as a consequence of the increased absorption and accumulation of lead are considered. The problem of the chemical forms of lead in the environment, including the internal environment, is described. The bibliography includes 153 references.

CONTENTS

I. Introduction 368
II. Lead in the atmosphere 368
III. Contamination of the soil and plants by lead 370
IV. Lead in the hydrosphere 372
V. The effect of lead on living organisms 374

I. INTRODUCTION

The manufacture and consumption of lead in various branches of industry increase continuously; at the same time its release into the biosphere has also increased. Thus 100 000 tonnes of lead were released into the atmosphere in the USA in 1964, which amounted to 10% of the total industrial consumption of lead; the corresponding amount in 1968 was 184 316 tonnes[1]. According to UNESCO data, more than 2.3 million tonnes of lead enter the seas with river water. The atmosphere is also a source of the contamination of the Pacific Ocean by lead. According to Kutyrin's calculations[2], 200 000 tonnes of lead fall on its surface every year.

The number of publications devoted to the problem of the pollution of the environment by lead is large, because lead contaminates various areas—the atmosphere, the hydrosphere, and soils. Many plants grow on soils contaminated with lead accumulate it. The concentration of lead in such lead-enriched plants exceeds the normal content by a factor of 2–100.[3,4] The accumulation of lead by plants merits attention, since not only wild but also cultivated plants, which serve as food products for people, are enriched in lead. Lead can be concentrated at all levels of the aquatic food chain—in both algae and molluscs and fish[5]. Studies in which large amounts of lead were detected in meat and internal organs of domestic animals used as food have been published recently[6,7]. Lead enters the human organism with water and food.

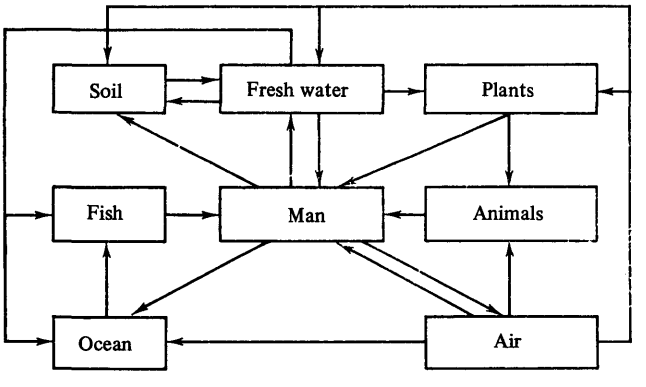
The food chain is not the only source of lead in the human organism. Contamination of air by lead also promotes its penetration into the organism. An increasing number of studies have shown that the main source of lead entering the organism is air contaminated by anti-knocking additives, which contain lead alkyls[8–10]. As a part of organic compounds[11] and also in ointments and cosmetic preparations, lead penetrates through the skin, but apparently only to an insignificant extent[12]. Thus there are two main pathways leading to the chronic action of lead on man—oral and respiratory. The Figure illustrates schematically the migraton of lead in the biosphere.

The global pollution of the environment by lead is indicated by its increased content in polar ice. Thus during the last 25 years the lead content in the snow cover of Greenland has increased by a factor of 400.[13] Lead has also been found in the soil of northern forests remote from industrial regions, where it has been deposited at a rate of 305 grammes per hectare annually[14]. This shows that lead migrates in the environment via the atmosphere in the form of aerosols of lead and its compounds, and the problem of the pollution of the environment by lead has become international, since there are no state boundaries to the movement of atmospheric pollution.

II. LEAD IN THE ATMOSPHERE

1. Sources from which Lead Enters the Atmosphere

The sources of lead in the atmosphere can be divided into natural and anthropogenic. The former include windborne dust, forest fires, volcanic eruptions, sea mist, and plant decomposition products. According to Nriagu[14], the annual amount of lead entering the atmosphere throughout the world from natural sources is 24.5×10^6 kg.



Scheme illustrating the migration of lead in the biosphere.

The anthropogenic sources of lead include lead mines, factories where lead is smelted and lead-containing articles are remelted, factories where various lead compounds are

manufactured, and ferrous and non-ferrous metallurgical plant. The atmosphere is extensively polluted by lead as a result of the combustion of coal, peat, wood, and petroleum fuels used in internal combustion engines, in the combustion of waste, and in the cleaning of tanks and cisterns for the storage of petrol.

The anthropogenic emission of lead amounts to 449×10^6 kg annually[14] and exceeds the natural emission by an order of magnitude. The amount of lead entering the atmosphere on combustion of petrol, which contains anti-knock compounds, amounts to approximately 60% of the total emission from anthropogenic sources.

The total amount of lead distributed in world ecosystems through the atmosphere during the period of civilisation is 20×10^9 kg.[14] A sharp increase of lead emission was observed at the beginning of the 20th century and then in 1923 when anti-knock alkyl-containing derivatives were introduced into petrol.

2. Lead in the Exhaust Gases of Automobiles

One of the principal sources of lead in the environment is the exhaust gases of automobiles and aeroplanes. The waste from automobiles constitutes 57–95% of the pollution of air in many cities of highly developed capitalist countries, more than 40% in Moscow, and 13.3% on average in the USSR.[15] During use, an automobile releases into the environment more than 100 different compounds, the majority having a harmful effect on the environment.

The results of the study of the pollution of air by lead from compounds used as anti-knock agents for internal combustion engines have been published[17]. The authors of this study determined with the aid of ^{203}Pb the amount of lead deposited on the leaves of plants, on the soil, and on grass. It was found that fine aerosol particles (0.01–0.03 μm) are deposited on the surface of plants more readily than large particles (0.2–0.25 μm). Only 10% of the total amount of lead in exhaust gases is deposited on plants 30 m from a motorway with a traffic density of 40000 automobiles per hour; the remaining lead-containing particles remain suspended and are deposited at a much greater distance from the highway.

The anti-knock additives includes mainly alkyl derivatives of lead: $Pb(CH_3)_4$, $Pb(CH_3)_3(C_2H_5)$, $Pb(CH_3)_2(C_2H_5)_2$, and $Pb(C_2H_5)_4$. In the combustion of fuel a significant proportion of the lead bound in organic compounds decomposes and is ejected into the atmosphere in the form of inorganic salts, but a certain amount is ejected unchanged. The results of a study of the levels of organic and inorganic lead compounds in air have been published[18]. By taking samples on filters with a pore diameter of 0.01 μm and using the gas-chromatographic method of analysis, it is possible to determine the concentrations of organic and inorganic compounds of the element. It has been established that most of the lead detected in the atmosphere is present in an inorganic form, while the concentrations of organic lead compounds in air are very low, amounting to 10^2–10^3 μg m^{-3}.

In a review[19] devoted to the problem of the pollution of the atmosphere by tetraethyl-lead (TEL) and tetramethyl-lead (TML) it is noted that their content in the atmosphere is less than 5% of the total amount of lead present. The authors[19] explained this finding by two causes: the fairly rapid photochemical decomposition of these compounds and the presence in the atmosphere of substances accelerating their decomposition. Among these, the most active are NO_x, O_3, atomic oxygen, and the radicals $OH^{\cdot}$, $CO^{\cdot}$, etc., the reactions of alkyl derivatives of lead with these substances being fairly rapid also in the dark. The kinetics and mechanisms of the main homogeneous reactions of TML and TEL in the atmosphere have been investigated[20]. It has been found that the lead alkyls are relatively stable in the dark and have half-lives of 320 and 100 h for TML and TEL respectively in purified dry air. Both compounds are unstable to the action of ultraviolet light and decompose in the light via first-order reactions with photolytic decomposition rate constants of 3.4×10^{-4} min^{-1} for TML and 1.3×10^{-3} min^{-1} for TEL (after subtracting the corresponding values for the dark reactions).

Thus the rate of photolysis of TEL is almost four times greater than that of TML. This is probably why, among lead alkyls, only TML can be detected in the air over our motor highways with a high traffic density, its content amounting to 1–20% of the total concentration of lead in the air[21]. As regards the mechanism of the homogeneous reactions of lead tetra-alkyls, the authors[20] concluded that it is analogous to the mechanism of the hydrocarbon decomposition reactions. The reactions of TML and TEL with O_3, $O^{\cdot}$, and $OH^{\cdot}$ are analogous to the reactions of alkenes, particularly ethylene and propene.

The following mechanism of the reaction of TML with ozone has been proposed[20]:

$$Pb(CH_3)_4 + O_3 \rightarrow (CH_3)_3PbOOH + CH_2O^{\cdot} ,$$

The rate constant for this reaction has been determined as 1.9×10^{-3} $(p.p.m.)^{-1}$ min^{-1}. The rate constant for the reaction of TEL with ozone is 1.6×10^{-2} $(p.p.m.)^{-1}$ min^{-1}. Lead alkyls can react with atomic oxygen either via reaction (1) or via reaction (2).

$$R_4Pb + O^{\cdot} \leftarrow R_3PbO^{\cdot} + R^{\cdot} ; \quad (1)$$

$$R_4Pb + O^{\cdot} \rightarrow R_3PbCH_2^{\cdot} + OH^{\cdot} ;$$

$$R = CH_3 \text{ or } C_2H_5 . \quad (2)$$

The rate constants for the above reactions are 7.0×10^2 and 18.9×10^2 $(p.p.m.)^{-1}$ min^{-1} for TML and TEL respectively[20].

The interaction of $OH^{\cdot}$ radicals with lead tetra-alkyls most probably proceeds via the mechanism

$$R_4Pb + OH^{\cdot} \rightarrow R_2PbCH_2^{\cdot} + H_2O .$$

The rate constants are 13.3×10^3 and 11.7×10^4 $(p.p.m)^{-1} min^{-1}$ for TML and TEL respectively.

According to the data of Harrison and Perry[22], the concentration of lead tetra-alkyls in city air near motorways is on average 0.1 μg m^{-3} and constitutes less than 5% of the total content of lead, which agrees with Baba's results[19].

Between 80 and 100% of lead compounds found in the exhaust gases of automobiles consist of the lead halides $PbBr_2$, $PbCl_2$, PbBrCl, and the α- and β-forms of the double salt $2PbBrCl.NH_4Cl$. The composition and size of the particles in the exhaust gases have been studied in detail[23] using a scanning microscope. It was found that the large particles (>200 μm) contain mainly PbBrCl, 2PbO.PbBrCl, and insignificant amounts of $PbSO_4$ and $Pb_3(PO_4)_2$; the main lead compound in 2–10 μm particles is PbBrCl. Particles with a diameter >5 μm contain 20–30% Pb, those with a diameter of 1–5 μm contain 50–70% Pb, and only 5% Pb is present in submicroparticles.

More than 90% of the particles in the exhaust gases have dimensions less than 2 μm. Thus, as a consequence of the high degree of dispersion of the particles in the exhaust gases from automobiles, there is a high probability of the penetration of lead into the organism via the respiratory pathway. We may note that the concentration of lead in city air has reached dangerous levels, bearing in mind that 1 litre of petrol contains 200–600 mg of lead, 50–70% of which is ejected into the air with the exhaust gases[24]. The possible ways of combating the pollution of the environment by lead

involve the creation of fundamentally new engines and new catalysts and the use of unleaded petrol. The results of experiments have demonstrated a decrease of the lead content in the parts of cities with a reduced automobile traffic[25].

3. The Lead Content in the Atmosphere and Its Biological Action

Although the views on the mechanism of the absorption of lead via the respiratory pathways are contradictory, it has been suggested that a considerable proportion of the lead breathed in enters the blood through the tracheobronchial system. According to the data in Ref. 21, 35% of the total amount of lead breathed in enters blood.

Results obtained in the inhalation by rats, over 40–60 min, of the aerosol of the exhaust gases from engines working on petrol with added TEL containing the ^{203}Pb isotope have been published[26]. The authors suggest that lead is absorbed by rats mainly through lungs and not through the gastrointestinal tract. It has been noted that lead assimilation processes during inhalation are fairly similar in rats and in men, but there are significant differences in the metabolism of the assimilated lead.

Cantrell et al.[27] caused baboons to inhale Pb_3O_4 dust (particle diameter 3.2 μm) at a concentration of 1.5 μg m^{-3} in the course of 4 h per day for 26 days. It was found that, whereas the initial lead content in the blood was 9.9 μg/100 ml, after 24 h of inhalation it rose to 19, and after two days to 41 μg/100 ml.

Data concerning the concentration of lead in the atmospheric dust of large cities in the industrial centres of Canada exceeding the natural concentrations by a factor of 18–300 have been published[28]. Following the inhalation of 0.1 g of dust with a lead concentration of 1500 μg g^{-1}, which corresponds approximately to its content in the dust of a medium-scale industrial city, man accumulates up to 20 μg of lead. In rural areas the absorption of lead with dust amounts to 5% of its total daily intake (with food, water, and air), while in a large industrial city it amounts to 60%.

People constantly exposed to air containing 4.5 μg m^{-3} of lead (the concentration over large industrial cities) absorb 91 μg of lead in 24 h. Depending on the particle size, the organism retains approximately 30–40% of the lead absorbed in this way, which amounts to 36 μg in 24 h.[29] In Philadelphia the level of lead in the blood of 11 women living in city areas amounted to 29 μg/100 g and more, while the concentration of lead in the city air was 1.7 μg m^{-3}.[25]

In many investigations it has been emphasised that the level of lead in the blood depends on a multiplicity of different factors and not only on the lead content in the atmosphere. Thus a detailed study of the biological action of lead aerosols on the inhabitants of Tokyo has been described[30]. The limiting concentration of lead in the atmosphere was 0.63 μg m^{-3}. The level of lead in the blood varied in the region of 16.8 to 18.7 μg/100 ml depending on the given person's place of residence and occupation. However, a positive correlation between the lead concentration in the blood and its content in the atmosphere has not been established. No correlation has been found either in a study[31] of the biological action of the lead content of the atmosphere on the inhabitants of 7 American cities. However, it has been shown[32] that the lead content in the blood of the inhabitants of Teheran is closely related to street traffic; the lead concentration in the blood increased with age and was higher in cancer patients than in healthy people. In Italy tests were made on the level in the blood of people living in industrial city regions and in the region devoted to the ceramic industry[33]. The average lead contents in the blood were respectively 26.02, 32.21, and 58.06 μg per 100 ml. The lead content in the blood was higher in men than in women and higher in male smokers than in non-smokers. Thus smoking can be regarded as one of the ways whereby lead penetrates into the human organism.

It has been shown[25] that the average level of lead in the blood increases slightly under the influence of air with a lead content of approximately 2 μg m^{-3}; in the presence of this amount of lead in air the lead concentration in the blood of most people is approximately 30 μg/100 ml. It has been calculated[34] that after exposure to an atmosphere containing lead at a concentration of 2 μg m^{-3}, the amount of lead absorbed from respiratory pathways is on average 14 μg per day. The average lead contents in the blood of city inhabitants in different countries are 12.1 μg/100 ml,[35] 25 μg/100 ml,[36] and 43 μg/100 ml.[37]

III. CONTAMINATION OF THE SOIL AND PLANTS BY LEAD

1. Lead in the Soil Near Smelting Plants, Mines, and Industrial Undertakings

The Clark level of lead in the Earth's core is 1.6×10^{-3}%.[38] In the region of abandoned lead amines, smelting plants, and industrial undertakings associated with the production of lead and its compounds, the content of this metal in the soil, particularly in the surface layers, is much higher. Extremely high concentrations of lead (>5000 μg g^{-1}) have been found in the upper layers of soils in direct proximity to industrial undertakings involved in the processing of scrap metal and used batteries and outside the storage areas of factories manufacturing batteries.

It has been shown[39] that the lead content diminishes exponentially with distance from the source of pollution. Analogous data have been quoted in other investigations. Dust and specimens of dirt collected on the surface of the Earth at a distance of 1 km from smelting plants contained 3541 to 5466 μg g^{-1} of lead, whilst at a distance of 2.5 km the content was 397 μg g^{-1}.[40] In many studies devoted to the determination of lead in soil near industrial undertakings its high content was also observed[41–43]. It is noteworthy that the lead concentrations in the soil of open spaces and playing fields remote from industrial undertakings and automobile highways were 800 μg g^{-1} (in 900 soil specimens[44]).

Lead accumulates in the soil not only as a consequence of its contamination by industrial waste, but, as mentioned above, also as a result of the pollution of air by the exhaust gases from motor transport. Near motorways, the concentration of lead in the soil and in the dust covering plants is enhanced. According to Strusinski's results[45], the highest amounts of lead have been detected at a distance of 10 m from the road; with increase of the distance from motorways, the concentration of lead diminishes. It has been shown[46] that lead accumulates on the surface of soils in layers 0–5 cm deep. Specimens taken from rice fields at a distance of 3 to 5 m from a motor highway contained 11–100 μg g^{-1} of lead; in specimens taken at a distance in excess of 3–5 m the Clark level of lead was not exceeded.

The ability of lead to be absorbed by various soils is extremely high. Thus, in a study of the distribution of metal ions between the soil and a solution, it was established[47] that in the pH range 3.5–4.5 the degree of adsorption diminishes in the following sequence: Pb > Cu > Cd ≅ Zn > Ca. The absolute amounts of adsorbed ions increase with the concentration in solution and the adsorption and desorption half-lives are in the range 5–15 s. Since soil is a complex multi-

component system incorporating fungi and enzymes in addition to organic and inorganic ingredients, it is to be expected that lead will influence them. In an experiment designed to investigate the influence of lead on the self-purifying capacity of soils it has been observed[48] that, among all the parameters of the biological activity of the soil, the activity of enzymes is the most sensitive to the action of lead. Lead at a concentration of 100 μg per 100 g of soil suppresses the dehydrogenase activity of the soil to a fairly high extent. In a study of the toxic effect of lead salicylate, acetate, and oxide on soil enzymes it was found that the salicylate causes the greatest inhibition of enzyme activity, while the oxide has the least effect[49], i.e. the most water-soluble compound exerts the greatest influence.

2. Contamination of Plants with Lead

The possibility of the stimulating effect of lead at low concentrations on plants has been pointed out in Soviet studies[50,51], but the problem of the relation between the lead concentrations in the soil and the biological effect has not been solved in the above investigations. In studies of recent years much attention has been devoted to the establishment of a correlation between the amount of lead introduced into the soil and its effect on the development of plants. Thus it has been demonstrated[52] that the introduction of lead acetate into the soil at a concentration of 100 mg kg^{-1} and of lead carbonate at concentrations of 500 and 5000 mg kg^{-1} has a positive effect on the growth and development of Khar'kovskaya-46 wheat. On the other hand, lead acetate at concentrations of 500 and 5000 mg kg^{-1} inhibited the growth of plants. The different effects of lead salts are attributed to the low assimilability of lead carbonate as a consequence of its low solubility and also to the change in the pH of the soil water after the introduction of lead salts. No data were published in the above study[52] concerning the lead content in the plant itself.

It has been stated[53] that the introduction of lead into the soil at concentrations up to 1000 μg g^{-1} does not have any effect on rye and oats sown in sandy loam. A further increase of the lead concentration leads to a sharp decrease of the rate of growth. The introduction of fertilisers does not decrease the harmful effects of lead at its concentrations in excess of 1000 μg g^{-1}.

According to Vinogradov's data[54], the Clark content of lead in plants is 1.0×10^{-4}%. In regions with soils contaminated by industrial waste and automobile transport, lead has been found to have been accumulated by plants. The accumulation of lead by plants grown on soils with its different contents has been investigated[3]. The results showed that potatoes, cabbages, tomatoes, cucumbers, onions, and carrots grown on soils with a lead content of 220–480 mg kg^{-1} contain 2–5 times more lead than the vegetables grown under the same climatic conditions but on soils with a lower lead content (18 mg kg^{-1}). The authors[3] conclude that the lead concentration in vegetables is on average smaller by two orders of magnitude than its concentration in the soil on which they have been grown and that the accumulation of lead by plants on lead-enriched soils compared with that on normal soils differs by a factor not greater than 10.

It has been established[4] that different plants are capable of absorbing different amounts of lead under identical conditions. The minimum amount of lead was found in the seeds of leguminous plants—peas and beans. The maximum amount was found in the leaves of the turnip and egg plant—16.2 and 22.4 mg kg^{-1} respectively. On average, the lead content in the majority of plants was 2–3 mg kg^{-1}. When lead was introduced into the soil in the form of an aqueous solution of its acetate in doses of 50 and 100 mg per kilogram of soil, the leaves of lettuce accumulated 17 and 32 times more lead respectively than in control plants. For the majority of plants, a proportionality has been observed between the lead content in the soil and its accumulation in the plants.

The ability of lettuce to accumulate lead in appreciable amounts has been reported in a study[55] where the species response of lettuce to lead was investigated. Nine species of lettuce were grown by the hydroponic method with addition of lead to the nutrient medium at concentrations of 0.1–50 mg kg^{-1}. It was shown that low concentrations of lead (0.1–0.5 mg kg^{-1}) stimulated the growth of the roots and leaves on the lettuce, particularly in the early stages, while concentrations in excess of 50 mg kg^{-1} significantly inhibited the growth of plants after 4–6 weeks. Differences in the responses of the lettuce species considered, characterised by similar morphological structures, were discovered, which indicates a genetic regulation of the physical mechanisms of the assimilation and transport of lead in plants. It has been suggested that species selection will make it possible to obtain plants with a reduced capacity for the accumulation of lead and hence to reduce to a minimum the risk of the introduction of lead into the organism with food. The important conclusion has been reached[56] that the lead content in pea plants grown in pots with a lead-contaminated soil is correlated with the level of the water-extractable lead and not with its total content in the soil.

As noted above, roadside plants in the vicinity of a highway with intense motor traffic or near industrial undertakings contain much more lead than plants in regions not subjected to the influence of automobile transport and industrial pollution. Thus the lead content in grain cereals grown on lead-contaminated soils is appreciably increased[57]. In the immediate vicinity of a motor highway grass was found to have a maximum lead concentration of 206 mg kg^{-1}.[58]

In the course of a number of years Ocker and coworkers[59–61] investigated the content of mercury, lead, and cadmium in specimens of wheat and rye and in their processing products. It was established that lead is present in the grain in amounts of 0.1–0.2 mg kg^{-1} and is concentrated in its outer layers. The lead content is associated with the region in which the plants are grown and is particularly high in fields located near industrial centres. In the course of the husking of the grain up to 70% of lead is removed; approximately 40% of the lead contained in the grain enters the flour in the milling of the Canadian "durum" wheat. It has been shown[60] that a daily intake of 3 mg of lead by the human organism is harmless to health. It has been pointed out that the permissible lead content in food products is $\leq$0.5 mg kg^{-1}. In a number of studies it has been emphasised that, provided that technical instructions as regards the growing and gathering of the crop are adhered to, there is at present no acute risk to the health of people due to the contamination of food products by lead in connection with automobile exhausts.

Studies over a period of many years[62–64] have demonstrated the ability of lichens to accumulate the radioactive isotope ^{210}Pb. Natural breakdown in the ^{226}Ra chain is the source of supply of ^{210}Pb into the biosphere. The long half-life of ^{210}Pb ensures a global distribution of the isotope in the atmosphere and its uniform deposition on the Earth's surface. The ability of lichens to accumulate lead is responsible for the increase of lead concentration in all parts of the chain lichen–deer–man. According to the data of Litver et al.[62], 1 kg of dry lichen contains 7.6×10^{-9} Ci of ^{210}Pb.

There are literature data concerning the ability of fungi to concentrate lead[65,66]. Lead at a concentration of 5.34–80.98 mg kg^{-1} has been detected in the majority of puff-balls collected at a distance of 23–24 m from an automobile highway. The metal concentration in the washed and unwashed fungi was virtually the same, its greatest amount being located in the fungal caps[65]. According to Schnellmann and Opitz[66], the greatest amount of lead is contained in the fungus *Psalliota campestris*—the common mushroom—1.0 mg per kg of dry weight.

3. Lead in Seaweed

Many types of seaweed as well as terrestial plants are able to accumulate heavy metals. It has been established[67] that, in the presence of sufficient illumination, thick growths of certain seaweed contain lead in an amount exceeding 70 g per kilogram of dry mass. Careful washing of seaweed in fresh water removed less than 10% of the bound heavy metals. Exposure of seaweed specimens to dissolved lead salts led to a rapid exchange of the Na^+, Ca^{2+}, and Mg^{2+} ions, which are intracellular cations and are bound to the outer surface of the walls and plasmatic membranes. When seaweed is exposed to lead acetate (lead concentration of 100–500 mg kg^{-1}), extremely high losses of organic material occur, which indicates cell damage[68].

Experiments with natural thickets of seaweed have shown that in the vicinity of industrial waste heavy metals such as lead can accumulate to considerable levels without any apparent physiological harm to the aqueous organisms. It may be that incorporation of heavy metals along the cell wall protects the normal state of the metabolic enzymes and the cell structure. However, no attempt has as yet been made to determine the relative abundance of the physiologically harmless lead within the cell or on its envelope. Plants collected at a site where industrial waste is released contain excess amounts of heavy metals. Thus in its habitat in the bays of the Sea of Japan, where the lead concentration in water is 0.01 mg $litre^{-1}$, the plant *Ulva fenestrata* contains 5.4 times more lead than in a control, uncontaminated bay[69].

The authors explained the increased lead content in seaweed by the affinity of lead for the sulphydryl groups of proteins. It has been noted that changes in the geochemical factors of the medium in a polluted bay led to the adaptation of the water plants to the change in its mineral composition. The slow influence of lead on cell growth as a result of its slow accumulation has also been noted in other investigations. Thus the influence of copper and lead on the growth of pigmentation of seaweed has been studied[70]. Even low lead concentrations tend to reduce the amount of chlorophyll and carotenoid. In addition, the generation of oxygen under illumination is suppressed. However, lead should not be regarded as extremely toxic to seaweed. It has been shown[71] that the accumulation of lead by seaweed and water plants is associated with the pollution of the underlying soil when snow collected from cities with intense motor traffic is disposed of in water basins.

The toxicity of TML in relation to the freshwater alga *Scenedesmus quadricauda* has been investigated[72]. When biologically generated TML from a container with a lead solution at a concentration of 5 mg $litre^{-1}$ is bubbled through a medium containing an alga, the cell growth of the latter is reduced by an amount ranging from 85 to 32%. Cells exposed to the action of lead tend to aggregate. Changes were observed in their fine structure.

IV. LEAD IN THE HYDROSPHERE

According to Chow's data[73], lead compounds are introduced into the oceans with continental water in an average amount of 1.7×10^{10} g annually. The main contribution of lead to the hydrosphere comes from industrial waste water, the precipitation of radioactive deposits, and the use in river transport of motors driven by petroleum fuels. It has been estimated[74] that the lead content in the surface layers of fresh water has increased from prehistoric times up to the present by an amount ranging from 0.5 to 5 μg $litre^{-1}$.

As mentioned in the previous sections, the transport and distribution of lead from various sources takes place mainly through the atmosphere. Large lead-containing particles are deposited along the roads and are responsible for the high level of lead in soils. Aerosols with smaller particles are transported by wind and are dispersed around the terrestial globe. On precipitation of snow and rain, these lead compounds are "washed out" of the layers of the atmosphere and enter the soil and the hydrosphere. Naturally the lead content in rain water is correlated with its local content in air, the intensity of motor traffic, and the degree of industrialisation of the region. According to the results of Drozdova and Machon'ko[75], the average concentration of lead in rain water at 16 points in the European territory of the USSR was 5.5 μg $litre^{-1}$ in 1968–1969. Andren and Lindberg[76] found that the average deposition of lead with rain is 20 g per hectare per month. The lead content in the river waters of South Western France has been determined[77] and it has been noted that it varies from 14.4 to 0.2 μg $litre^{-1}$ and that the precipitation of lead in atmospheric rain is higher than its transport in river water.

1. Lead in Polar Ice

The study of polar snow, frozen layers of which permit the determination of its chronological age, is a reliable means of estimating the anthropogenic contribution of lead to the environment as a function of time. The results of studies of the concentrations of 12 heavy metals, including lead, in the snows of Western Antarctic from 1914 to 1974 have been published[78]. It was found that snow became greatly enriched in lead, cadmium, copper, zinc, and silver as early as 60 years ago and that the enrichment factors found in 1914 were comparable to those determined at the present time. The concentrations of the heavy metals were higher than expected on the basis of their average values for the Earth's core and seawater, the values for lead being 0.018 and 0.04 μg kg^{-1}, depending on the kind of filter used. The authors[78] believe that the sources of the heavy metals in polar snows may be their emission from the surface microlayers of the oceans and from plants as well as human activity. However, they believe that the most probable source is volcanic activity, since the changes in the concentrations of metals with time agree with the historic records of volcanic aerosols. Evidence that volcanic aerosols can reach the Antarctic ice cover had been published previously[79].

The results of the above study[78] agree with those obtained by other workers. Thus, according to Murosumi et al.[80], the lead content in specimens of Antarctic snow after 1916 was 0.01 μg kg^{-1}. In northern Greenland the lead concentration proved to be higher than in the Antarctic. The lead content in the ice layers of Greenland extrapolated to 1753, which corresponds to the beginning of the European industrial revolution, is 0.011 μg kg^{-1}. This concentration exceeds by a factor greater than 25 its natural level. During the period from 1933 to 1965 the metal concentration increased

to 0.20 μg kg^{-1}, which exceeds the natural level by a factor of 500. The authors[80] explain this increase of the lead content in polar ice by the contribution of smelting operations, the combustion of coal, and the use of lead-containing antiknock additives, which began actively in 1940.

Sanak and Lambert[81] investigated the variation of the concentration of ^{210}Pb in the ice cover of the South Pole as a function of the depth from which the specimen was collected. 92 specimens collected from depths down to 18 m were analysed, which showed that between 1920 and 1954 the lead concentration increased. The authors explained this either by a change in the rate of the accumulation of snow or by an increase of the concentration of ^{210}Pb in the snow. Possibly both factors operated simultaneously.

2. Lead in Fresh Water

The concentration of lead in fresh waters depends on the site from which samples are removed, on the time of the year, and on the distance from industrial regions. A review[73] presents the results of the determination of lead in fresh waters. Lead was determined during the period 1956–1975 in 42 sources of water located in different parts of the Earth. It was found that the average lead concentrations in fresh waters vary from 1 to 24 μg $litre^{-1}$ and in the majority of rivers and lakes are in the range 1–10 μg $litre^{-1}$.

Higher amounts of heavy metals are located in the surface microlayer and in the foam generated by wind on the surfaces of rivers and lakes. The foam and the surface microlayers can also be enriched in aliphatic acids, esters, alcohols, chlorinated hydrocarbons, microelements, and organic and inorganic forms of phosphorus, nitrogen and carbon. The enrichment of the foam in heavy metals takes place as a result of the pollution of the surface of water by the metals, the retention of material particles in the surface film and in air bubbles, and the atmospheric contribution.

The foam collected in 1973–1974 at the southern and western coasts of Lake Mendota has been analysed[82]. The foam specimens were collected in plastic cylinders and, after destabilisation in the dark at 4 °C for 24 h by the voltamperometric method, the heavy metal content was determined. It was found that 27–28% of the metal from the destabilised foam is present in the dissolved phase. A considerable proportion of the dissolved metal is bound in complexes with organic substances or is associated with the colloid phase. The average lead concentration in the foam is 2.21 mg $litre^{-1}$. The average enrichment factor in the solid fractions was found to be a maximum for lead (1580), being 800, 235, and 80 for the remaining metals (Cd, Zn, and Cu respectively). It is noteworthy that the surface layers and foam have so far been little investigated from the standpoint of the accumulation of lead and other heavy metals.

At the sites of the disposal of city and industrial waste the lead concentration is appreciably increased and can reach 1000 μg $litre^{-1}$ and more. In connection with the struggle for the protection of the environment, industrial undertakings nowadays have purifying equipment to protect water resources from contamination by waste water. "Rules for the protection of surface water from contamination by waste water", defining the conditions for the disposal of waste water and specifying norms for the quality of water are operative in our country. The limiting permissible concentration (LPC) of lead in reservoirs for sanitary and domestic consumption is 0.1 mg $litre^{-1}$; tetraethyl lead must be absent (within the limits of the most sensitive research method)[83]. The following values of LPC have been established for radioactive isotopes[91]: 1×10^{-7} Ci $litre^{-1}$ for ^{203}Pb; 1×10^{-11} Ci $litre^{-1}$ for ^{210}Pb; 5×10^{-9} Ci $litre^{-1}$ for ^{211}Pb. When waste water is released within the boundaries of any inhabited site, the requirements established as regards the composition and properties of the water in a given body of water must apply to the waste water itself, since in practice one cannot hope for dilution and self-purification[84].

The limiting permissible concentration of lead in drinking water, amounting to 100 μg $litre^{-1}$, has been established by the World Health Organisation (WHO); the same quantity has been adopted as the LPC in the Soviet Union. A limiting level of 50 μg $litre^{-1}$ has been established in the USA and Canada, while in Sweden it is 20 μg $litre^{-1}$. Many investigators suggest that the LPC for lead established by WHO be reduced. Thus it has been proposed[85] that a limiting concentration of lead of 30 μg $litre^{-1}$ be established.

In certain countries where lead pipes are used in the water supply drinking water can contain increased quantities of lead. This applies to regions with soft water giving a weakly acid response in which the solubility of lead is enhanced. The average concentration of lead in such water usually varies in the range 50–100 μg $litre^{-1}$ and in certain cases it reaches ≳300 μg $litre^{-1}$. Cases of an increase of the lead concentration in water to 600 μg $litre^{-1}$ are known; when the lead pipes in the water supply were replaced, the concentration of lead in water fell to 3 μg $litre^{-1}$.[86]

3. Lead in Solid Deposits

The lead contents of solid deposits in rivers, lakes, and seas are various and depend on the geochemical conditions and the anthropogenic contribution. The concentration of lead in the solid deposits removed from various regions near Long Island (USA) varies in the range 200–350 μg $litre^{-1}$.[87] The concentration of lead on the surface of the deposits from the Lake Ontario, amounted to 120–137 mg kg^{-1}.[88]

The distribution of heavy metals in the solid deposits and waters of the Amur Bay has been investigated[89] and the degree of contamination of the deposits in the Golden Horn Bay, where there is a large anthropogenic contribution, has been established. The degree of contamination of the deposits in the latter bay decreases in the sequence Pb > Hg > Cd > Zn > Cu > Cr. The ratio of the average concentrations of the elements in the deposits of the Golden Horn and Amur Bays has been adopted as an index of the contamination of such deposits.

For lead, this quantity amounts to 11.1 (solid deposit) and 1.6 (water near the bottom). The above coefficients show that the lead content in the deposits of the Golden Horn Bay is much higher than the background concentration, which is due to the high sorption capacity of the deposits and the low mobility of the metal in the latter. The local character of the increase of the concentration of heavy elements, associated with the disposal of pollutants in the water of the bay, has been emphasised: the concentrations of the elements in the deposits removed from the mouth of the bay is reduced.

The deposits in Newport Bay (California) were analysed[90] by three independent methods. It was found that the concentrations of zinc and lead increase in the upper layers deposited since 1955, when industrial development began in the catchment area.

The compositions of the solid deposits from different bodies of water are different. In those places where argillaceous rocks predominate, the sorption of ions by clay particles takes place via ion-exchange processes and under the influence of adhesion forces. The overall sorption capacity of clays in relation to heavy metal ions depends on the mineral composition and on the pH of the medium[91]. With increase

of pH, the sorption capacity of clays as a rule increases as a consequence of the formation of stable hydroxo-complexes.

4. Chemical Forms of Lead in the Hydrosphere

The complexity of the behaviour of metals in the hydrosphere is due to the presence in water of a large amount of organic and mineral compounds of abiotic origin and those which are formed in the course of the vital activity of water organisms and as the result of their natural death or mass destruction due to unfavourable conditions. These are nitrogen-containing substances, organic acids, aminoacids, vitamins, hormones, and inorganic salts.

The determination of the overall concentration of trace metals in natural waters in order to establish the degree of their toxicity to water organisms and plants can yield erroneous results. Depending on the chemical form of the metal, water with a high lead concentration can be less toxic than another type of water with a lower lead concentration. The ionic form of lead is more toxic to organisms than lead bound in a complex and the greater the stability of the complex the lower the toxicity. The chemical behaviour of lead in the hydrosphere constitutes a problem which has been little investigated. Problems associated with the form of the chemical compounds of lead contained in the solid deposits have been inadequately studied; it is known that lead is present in the form of such compounds in fresh water and sea water.

The chemical simulation of the nature of the trace metals in water has been carried out[92]. A study was made of the behaviour of heavy metals, including lead, in the presence of inorganic ligands (CO_3^{2-}, SO_4^{2-}, Cl^-, Br^-, F^-, NH_3, PO_4^{3-}, and OH^-) and organic ligands [ethylenediaminetetra-acetic acid (EDTA), citrate ions (Cit), histidine, cysteine, and aspartic acid]. The adsorption of metals on the surface of SiO_2 has been studied. It has been established that at pH < 7.1 lead exists in the form of Pb^{2+} ions; above this pH, lead is present as $PbCO_3$ and hydroxo-species. The sum of lead carbonate and its hydroxo-complexes does not exceed 15%. The complexes with sulphate, chloride, and bromide ions exist in very small amounts. The study of the behaviour of lead in the presence of organic ligands has shown that complete binding in the complex takes place at [EDTA] = 30[Pb]. Appreciable binding of Pb^{2+} by citrate ions takes place when the ligand concentration is more than a factor of 10 greater than that of the metal; thus 30% of lead is bound in the form of complexes at [Cit] = 100[Pb]. The distribution of lead between water and the sorbing surface depends on the accessibility of the latter. With increase of the surface area, the degree of adsorption of metals increases. Copper(II) is most readily adsorbed on SiO_2 and is followed in this respect by lead(II) and mercury(II).

In a deposit–water system lead also exists in different forms. Part of the metal can remain in the intermediate water in the form of free or complex ions and can combine with soluble inorganic and organic substances, can be adsorbed on the surface of the deposits, or can be incorporated in the crystal structure of the latter.

The behaviour of different organic substances and heavy metals in the deposit–sea water system under various oxidation-reduction conditions has been investigated[93]. The authors studied three typical forms of natural solid deposits—clay, silt, sand, and the water above these deposits. The lead content in the specimens of the deposits was 177, 93.6, and 64.8 mg kg^{-1}, while the natural level is 20–50 mg kg^{-1}; the lead content in sea water corresponding to the deposits was 0.10, 0.04, and 0.08 μg m^{-3}. Oxidising conditions were established by reducing the pH from 8.2–8.4 to 7–7.5; reducing conditions were created by increasing the pH from 6.8 to 7–7.5. Under oxidising conditions, the amount of lead liberated from the deposits increased, while under reducing conditions it decreased compared with the natural level of lead in sea water. This can be explained by the formation of an insoluble sulphide. The solubilities of $PbCO_3$ and PbS (the compounds of which it is proposed to monitor), the formation constants of the principal species existing under specific conditions, and their percentage contents relative to the total concentration of soluble lead have been calculated[93]. Experimental data show that the type of deposit (clay, silt, or sand) does not control the direction of migration of the elements, which is regulated by the chemical composition of the water; the oxidising or reducing conditions are a fundamental factor.

V. THE EFFECT OF LEAD ON LIVING ORGANISMS

1. Lead in Animal Organisms

There is virtually no evidence in the literature concerning the biological necessity for lead to maintain the vital activity of an organism with the exception of a few publications. It has been reported[94] that the addition of 1.0–2.5 μg g^{-1} of lead (as the acetate) to a diet lacking this element increased the growth of rats by up to 20%; lead oxide and nitrate showed the same effect. Some data indicate that small amounts of lead have neither a favourable nor an undesirable effect. An experiment in which sheep were fed paper from newspapers and magazines (23% of their rations) in the course of 175 days has been described[95]. The lead contained in the paper accumulated in animal tissues, but histopathological studies with the aid of an electron microscope did not reveal any damage associated with the diet.

However, the majority of investigations on animals have shown that even a slight exposure to lead induces visible changes in the microstructure of the kidneys and the liver, affects the blood circulation, alters the central nervous system, and diminishes the reproductive function[96–98]. The biochemical effect includes the inhibition of the activity of erythrocyte aminolaevulinate dehydratese (ALD), an increased elimination of δ-aminolaevulinic acid (ALA) with urine, and an increased concentration of lead in the blood[99]. The reduction of the activity of ALD entails a disturbance of the synthesis of haem. In addition, lead inhibits the decarboxylases of coproporphyrinogen and uroporphyrinogen.

The inhibiting effect of $PbCl_2$ on the activity of adenosine triphosphatase has been demonstrated[100]. After the introduction of neutral lead acetate in the experimental lead intoxication of rats, an increase of the content of nucleic acid in the liver and in the kidneys was observed. The increase of the DNA and RNA concentration was proportional to the lead dose administered[101]. Lead can be regarded as a weak mutagen, since a slight increase of chromosomal aberrations was observed under the influence of the maximum dose tested (0.05 mg kg^{-1}).[102] It has also been established that lead disturbs the normal replication and genetic processes in the cell at doses much lower than those required for the appearance of signs of toxic activity[103,104].

Data concerning the lead content in the blood together with other factors determining the state of animals have been published in numerous investigations. Thus an increased content of lead in the blood, heart, and aorta of rabbits with atherosclerosis induced by a cholesterol-enriched diet[105] and

an increase of the concentration of lead in the blood of monkeys under the influence of psychological stress[106] have been reported.

It has been demonstrated on experimental animals that a deficiency of certain biogenic elements and vitamins as well as a high content of fats in food increase the toxicity of lead. Enhanced poisoning with lead occur in the presence of an excess or deficiency of protein in the diet. A deficiency of mineral components increases to the greatest extent the toxicity of lead, particularly in the presence of a deficiency of elements such as calcium, phosphorus, iron, zinc, and in certain cases copper[107,108]. A hypothetical mechanism of the enhancement of the toxic effect of lead in the presence of low contents of calcium in the food, based on the hypothesis that the observed retention of lead in the organism is due mainly to the decrease of its excretion and not an increase of its assimilation in the gastrointestinal tract, has been discussed in a review[109]. Other workers[110] arrived at the same conclusion. They did not observe significant differences in the assimilation of ^{203}Pb by rats receiving for two weeks a diet with low and high calcium contents, but in the presence of a deficiency of calcium in the diet a decrease of the excretion of lead and its accumulation in the organs and tissues were observed.

The iron deficiency in anaemia promotes an increased assimilation of lead in the gastrointestinal tract of rats[111], while in a lead-intoxicated embryo the incorporation of iron in liver haem was greatly depressed[112]. A vitamin *E* deficiency in poisoning by lead induces anaemia in rats[113,114]. Like other heavy metals, lead blocks the SH groups of enzymes[11,115].

Wild animals, fish, and birds are constantly subjected to the action of lead in the environment. Cases of acute poisoning of migratory birds by lead are known. Thus the concentration of lead in the kidneys and livers of geese, found in Canada, which were either dead or too weak to fly, proved to be 9.21–102.56 $\mu g\ g^{-1}$.[116]

An interesting method for the determination of lead in the environment has been proposed by Stone et al.[117]. In the course of two weeks, ten-day old Japanese quails received a diet containing 14.8–1223 $\mu g\ g^{-1}$ of lead. With increase in the concentration of lead in the diet, the latter accumulated in the tissues; at the end of the experiment the duodenum, the kidneys, the liver, and the shin-bone contained 16.76, 20.18, 3.8 and 1527.1 $\mu g\ g^{-1}$ respectively. The relation between the lead content in the tissues and in the diet was virtually linear. The results obtained can be used, according to the authors[117], to determine the lead content in the environment by a biological method.

It follows from the literature data that, among domestic animals, horses are most susceptible to lead intoxication[118–120]. The kidneys and liver of horses from North Western Saxony (Federal Republic of Germany) contained 0.4 and 0.9 $\mu g\ g^{-1}$ of lead respectively, which is 2–3 times more than its content in the organs of horses from Poland[120]. To prevent the risk to human health, the authors recommend that the liver and kidneys of horses be declared unsuitable for use in food and that horse meat should always be tested for lead content. Horses in regions polluted by smoke from industrial undertakings producing lead have shown symptoms of poisoning by lead and the organs and tissues of young animals were less contaminated than those of old ones. Analysis showed that hay and fodder grass were contaminated by lead in this region[118].

Attention should be drawn to the possibility of the lead poisoning of cattle grazing along highways with heavy automobile traffic. An increased concentration of lead in the internal organs of cattle, pigs, sheep, and horses has been noted in regions located near the sources of the pollution of the environment by industrial waste and under the conditions of a large consumption of green fodder. On the basis of data concerning the average annual consumption of meat, the average weekly intake of lead by the human organisms has been calculated as 0.006 mg.[119] The meat of 180 cows has been analysed[121] and the average lead content in the fresh meat proved to 0.31 mg kg^{-1}, which is higher than the maximum amounts found previously.

The considerable accumulation of ^{210}Pb in the muscles of deer in the extreme north of the USSR, exceeding its content in the muscle tissue of deer in Finland and Sweden by a factor of 10, has been noted[64]. The accumulation of ^{210}Pb in the bone of deer depends on the age of the animals. Five to six-fold seasonal variations in the content of radioisotopes have been noted—maximum amounts are observed at the beginning of spring and minimal amounts in the autumn; the average ^{210}Pb concentration in the bone of deer proved to be 8.3×10^{-9} Ci kg^{-1} according to the determinations in 1967 to 1968.[64]

It is believed[122] that the prolonged exposure of animals to lead at concentrations up to 25 mg kg^{-1} does not entail a significant intake of the element by the organisms of people utilising food products prepared from the meat or obtained from these animals. A large amount of the lead taken up by animal organisms is deposited in the bones, mainly in an inert form. In the determination of different elements in 20 specimens of the bones of domestic animals lead was found in the concentration range 1.5–8.5 $\mu g\ g^{-1}$.[123]

2. Lead in Aquatic Organisms

The toxic effect of lead on fish, molluscs, etc. depends on factors such as the form of lead, the duration of exposure, the type of water, and the specific features of the aquatic organisms. It has been established[5] that a lead concentration of 0.1–15 mg $litre^{-1}$ is acutely toxic to fish; lead arsenate at a concentration of 17.1 mg $litre^{-1}$ does not harm small fresh water fish in the course of exposure for 1 h, while at a concentration of 25 mg $litre^{-1}$ it leads to the death of the fish in the course of 24 h.

Lead can be biologically concentrated at all levels of the water food chain. The ability of sea organisms to concentrate elements at higher levels than those found in their environment has been known for many years. Lead concentration factors in the sea biosphere have been established[124]; they amount to 700 for seaweed, 40 000 for the phytoplankton, 3000 for the zooplankton, 4000 for molluscs, and 40 for the muscles of molluscs. It has been shown[125] that the lead content in the crude mass of the zooplankton varied from 0.3 to 1.5 $\mu g\ g^{-1}$ in 1972 and from 0.2 to 2.6 $\mu g\ g^{-1}$ in 1974. Approximate lead accumulation coefficients in the zooplankton amounted to 3.5×10^6 (for a lead content of 0.06 $\mu g\ litre^{-1}$ in the water of the Atlantic Ocean) and 8.8×10^4 (for a lead content in the water of 0.003 $\mu g\ litre^{-1}$).

Tissues of the oysters in the Missouri Basin contain 0.77 $\mu g\ g^{-1}$ of lead[126]. The tissues of the oysters and molluscs in the coastal waters of Tasmania contain lead within limits not exceeding the permissible sanitary norms[127]. As in the case of animals, the liver and kidneys of fish accumulate lead selectively[128]. Fish from different lakes in Poland and the Baltic Sea have been analysed[129] for heavy metals. The average level of lead was 0.199 $\mu g\ g^{-1}$ and the highest level (2.64 $\mu g\ g^{-1}$) was found in the beluga. In some investigations it has been noted that the accumulation of heavy metals by fish depends not only on the pollution of water by industrial waste but also on the species of the fish,

their age, and their size. The accumulation of metals is proportional to the length and weight of fish[130]. External tissues (shells, scale, and skin) contain higher concentrations of lead than internal organs, which is due to the partial adsorption of metals from sea water and especially to their release from the organisms in the course of detoxication. Like other heavy metals, lead probably forms complexes of mucous membranes, gills, or body surfaces.

3. Lead in the Human Organism

Food products are one of the main sources of the lead intake in the human organism under normal living conditions and the lead content must be monitored. The lead content in certain dietary products has been determined[131] by atomic-absorption spectrophotometry; the results in μg/100 g, are as follows: 90 in the tissues of the cod, 17 in the crab, 115 in the pig, 104 in the rabbit, 8 in apples, 23 in nuts, 160 in tea, and 30 in coffee. Apart from the pathways discussed previously, lead enters food products from the packing. Thus preserved orange and tomato juice absorbs lead from cans during storage—orange juice to a greater extent than tomato juice[132]. On the basis of the results of the investigation, it is suggested that the maximum permissible lead concentration in preserved fruit and vegetable juices is 0.4 mg litre^{-1}. As a result of contact with metallic caps, carbonated drinks are also contaminated with lead[133]. Lead can migrate from porcelain ware into its contents. It has been established[134] that it is extracted in greatest amounts into a 4% solution of acetic acid (0–3 mg litre^{-1} in 24 h at room temperature).

The lead content in food varies from 200 to 300 μg per 24 h.[29] Approximately 10% of the lead is extracted from food in adult humans in the gastrointestinal tract. Since the degree of absorption of lead by children is much greater than by adults (approximately 50% of the lead in food is assimilated), its daily consumption must not exceed 150 μg.[135] Analogous results have been presented in another communication[136].

Children, particularly embryos and newborn babies, constitute a group particularly susceptible to lead. This observation is associated not only with the unusual assimilation of lead by the child's organism but also with the fact that children can accidentally absorb much more lead than adults. Many instances of the poisoning of children who have eaten lead-based paint have been recorded. The lead content in four specimens of paint has been determined by atomic absorption methods[137] and has been found to be considerable; 5114, 2651, 1630, and 1350 mg kg^{-1}.

Lead enters the bodies of children also with dust and dirt. Day et al.[138] determined the potential risk to the child's body due to the lead contained in school yards, kindergartens, playing fields and city streets. The lead content in samples of dust, sand, and earth has been found by atomic absorption spectroscopy; it varies in the range 570–2130 μg g^{-1}. According to the authors[138], the increased lead content in the blood of city children up to the age of five is due to the intake of lead with street dust. The frequency of lead poisoning among children is directly proportional to the degree of industrialisation of the region and is directly related to the amount of food consumed by children. Protein deficiency in children enhances the toxic effect of lead on the organism and its deposition in tissues. Vitamins *E* and *C* and various preparations containing Ca^{2+}, Cu, Zn, Fe, and Se reduce the toxicity of lead, which can be used in the treatment of poisoning by heavy metals[139]. Numerous studies devoted to lead intoxication in children are also related to the non-specificity of the symptoms, which hinders the diagnosis of the intoxication of children, particularly at an early stage.

As in animal organisms, lead inhibits the activity of the enzyme ALD in the human organism. On the basis of the correlation observed between the level of lead in the blood and the ALD activity, it is suggested that the latter be determined to estimate the level of lead in the blood in mass investigations and in the cases where lead acts at low concentrations[140–142]. Other works[143,144] believe that the determination of Zn-protoporphyrin in the globin fraction which incorporates it in place of haem in the presence of lead is a particularly valuable method; this parameter is better correlated with the degree of damage to the nervous system and the generation of blood than the concentration of lead in the blood. Lead in the human organism interacts with nucleic acids, proteins, and cell membranes[145], accumulating in various organs and tissues. Organic lead compounds accumulate in tissues with a high lipid content, while inorganic compounds accumulate in bones[11]. According to a report[146], the levels of lead in the liver, blood, and bones of human beings not subject to lead intoxication are respectively 0.447, 0.026, and 4.002 mg kg^{-1}.

The role of urbanisation in the deposition of lead is reflected in the results of a study by Grandgean et al.[147]. It was established that the lead content in the bones of the inhabitants of Nubia is ten times lower than in the bones of the inhabitants of Denmark, while the lead content in the teeth of Nubians is thirty times lower than among Danes. Lead is one of the elements detected in dental enamel in different amounts. It proved to be the only element present in food products whose concentration is strongly and directly related to the incidence of caries.

In regions associated with the manufacture of lead, its increased content in hair has been noted[148]. It is suggested that hair be used as an indicator of the pollution of the environment by heavy metals, since these elements remain in hair for a long time after contact with them has ceased[149]. According to the data of the Pan-American Health Organisation, lead concentrations in hair less than 70 μg g^{-1} are nontoxic to man[149].

Complex forming agents such as EDTA, also employed in therapy, are used to diagnose lead intoxication. The concentration of lead is determined from the increase of the urinary excretion of the lead complex of EDTA after the injection of Na_2Ca.EDTA.[150] There have been reports of the effectiveness of the treatment of chronic intoxications with penicillamine, cysteine, and citric acid[151]. It is believed[152] to be useful to employ aqueous solutions of sodium pectate for the prophylaxis and treatment of lead intoxications. The protecting effect of tannic acid has been reported[153].

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It follows from the data presented that lead is a complex pollutant of the environment entering the organism via different pathways. Bearing in mind the ability of lead to accumulate and the large number of sources of lead in the environment, its danger is evident. On the basis of the latest studies, many investigators suggest that the limiting permissible amounts lead established previously be reduced. Thus it has been suggested that the PLC of lead in drinking water be set at 30 μg litre^{-1};[85] for children, the authors[28] propose a norm not exceeding 1 μg m^{-3} of lead in air or 50 μg g^{-1} in dust. A norm of 40 μg/100 ml of lead in the blood has been suggested[29] as more acceptable than the previous norm of 80 μg/100 ml adopted at the XVIth Combined Committee of FAO/WHO experts on food additives.

REFERENCES

1. Lead. Committee on Biological Effects of Atmospheric Pollutants. Washington: National Academy of Science, 1972, p.13. Quoted after A.E.Martell, Pure Appl.Chem., **41**, 91 (1975).
2. I.M.Kutyrin, "Okhrana Vozdukha i Poverkhnostnykh in Vod ot Zagryazneniya" (The Protection of Air and Surface Waters from Pollution), Izd.Nauka, Moscow, 1978, p.39.
3. T.N.Ruzhnikova, L.G.Zarubinskaya, and T.N.Kolesnikova, Symposium, "Svinets v Okruzhayushchei Srede" (Lead in the Environment), Moscow, 1978, p.15.
4. T.I.Grigor'eva and S.I.Khramova, Symposium, "Svinets v Okruzhayushchei Srede" (Lead in the Environment), Moscow, 1978, p.22.
5. "Lead Pesticides, Man and Environment", EPA, Washington, 1972, p.19.
6. W.Kreizer, K.Bunzl, and W.Kracke, Fleischwirtschatt, 1979, Vol.59, p.1529.
7. M.M.Varma and K.T.Doty, J.Environ.Health, 1979, Vol.42, p.68.
8. R.R.Kinnison, Environ.Sci.Technol., 1976, Vol.10, p.644.
9. A.Tomov, Priroda (Bulgaria), 1978, **27**, No.2, p.34.
10. R.L.Solomon and J.W.Hartford, Environ.Sci.Technol., 1976, Vol.10, p.773.
11. W.Kisser, Arch.Toxicol., 1977, Vol.37, p.173.
12. H.A.Waldron, Lancet, 1979, No.8151, p.1070.
13. A.E.Martell, Pure Appl.Chem., 1975, Vol.44, p.81.
14. J.O.Nriagu, Nature, 1979, Vol.279, p.409.
15. M.V.Sandler, Candidate's Thesis, in Medical Sciences, All-Union Institute of Medical and Medical-Technical Information, Moscow, 1980, p.3.
16. M.D.Artamonov, M.M.Morin, and A.M.Obel'nitskii, Symposium, "Snizhenie Otritsate'n Vozdeistvii Avtomobilya na Okruzhayushchuyu Sredu" (Reduction of the Negative Effects of the Automobile on the Environment), Moscow, 1977, p.3.
17. P.Little and R.D.Wiffen, Atmos.Environ., 1977, Vol.11, p.437.
18. J.W.Robinson and E.L.Kissel, J.Environ.Sci.Health., 1977, Vol.A12, p.411.
19. Yoshio Baba, Kagaky to Rogyo (Chem.and Chem. Ind), 1979, Vol.32, p.635; Ref.Zhur.Khim., 5I649 (1980).
20. R.M.Harrison and D.P.H.Laxen, Environ.Sci.Technol., 1978, Vol.12, p.1384.
21. "Lead in the Environment" U.S.Gov.Print.Off. Washington, 1976, p.18.
22. R.M.Harrison and R.Perry, Atmos.Environ., 1977, Vol.11, p.847.
23. K.Habibi, Environ.Sci.Technol., 1970, Vol.4, p.239.
24. O.V.Prudenko, T.V.Kolesnikova, and T.N.Efremova, Symposium, "Svinets v Okruzhayushchei Srede" (Lead in the Environment), Moscow, 1978, p.5.
25. J.H.Knelson, in "Comprehensive Analysis of the Environment" (Translated into Russian), Gidrometeoizdat, Leningrad, 1975, p.83.
26. A.Morgan and A.Holmes, Environ.Res., 1978, Vol.15, p.44.
27. A.C.Cantrell, T.A.Kolroe-Smith, and M.M.Simones, Brit.J.Ind.Med., 1977, Vol.34, p.110.
28. H.V.Warren, West Miner, 1979, Vol.52, No.3, p.26.
29. "Series of Technical WHO Reports", 1975, No.532, p.53.
30. K.Tsuchiya, Arch.Ind.Hyg.Toxicol., 1975, Vol.26, p.157.
31. L.B.Tepper, Arch.Ind.Hyg.Toxicol., 1975, Vol.26, p.139.
32. J.Bayat, Q.Bull, Z.Abedinzadeh, and H.Chabourian, Fac.Sci.Tehran.Univ., 1978, Vol.10, p.67.
33. P.Borella, A.Pini, R.Olivo, and G.Vivoli, Nuovi Ann.Ig.Mikrobiol., 1978, Vol.29, p.171.
34. M.B.Rabinovitz, G.W.Wetherill, and J.D.Kopple, J.Lab.Clin.Med., 1977, Vol.90, p.238.
35. T.Takagi and S.Matsuda, Japan.J.Hyg., 1977, Vol.32, p.366.
36. P.M.Kuhnert, P.Erhard, and B.R.Kuhnert, Environ. Res., 1977, Vol.14, p.73.
37. D.L.Moss, Am.J.Public Health, 1979, Vol.69, p.711.
38. "Khimicheskaya Entsiklopediya" (Chemical Encyclopedia), Izd.Sov.Entsiklopediya, Moscow, 1965, Vol.3, p.759.
39. S.N.Linson and B.L.Chai, J.Air Pollut.Control Assoc., 1976, Vol.26, p.650.
40. H.A.Roels and J.P.Buchet, Environ.Res., 1978, Vol.15, p.290.
41. L.C.Neri and H.L.Johansen, Trace Subst.Environ. Health, 1978, Vol.12, p.403.
42. J.N.Nwanko and C.G.Elinder, Bull.Environ.Contam Toxical, 1979, Vol.22, p.625.
43. B.G.Wixon, N.L.Gale, and K.Downey, Trace Subst. Environ.Health., 1977, Vol.11, p.455.
44. T.M.Spittler and W.M.Feder, Comm.Soil Sci., Plant Anal., 1979, Vol.10, p.1195.
45. A.Strusinski, Roczn.Panstwowego Zakladu Higleny (Pol.), 1978, Vol.29, p.411.
46. S.H.Park, Haguk Sikmul Poho Hanknol Chi., 1979, Vol.18, p.43, Chem.Abs., 1979, Vol.91, 173 969.
47. K.Bunzl, W.Schmidt, and B.Sansoni, J.Soil Sci., 1976, Vol.27, p.32.
48. O.V.Prudenko and G.M.Efremova, Symposium, "Gigiena Vneshnei Sredy" (Hygiene of the Environment), Izd.RGMI, Rostov-on-Don, 1977, Vol.29, p.38.
49. O.V.Prudenko, Sbornik Nauch Rabot Rost.-na-D. Med.Inst., 1977, Vol.86, No.3, p.87.
50. P.A.Vlasyuk and I.K.Onishchenko, in "Sbornik Rabot po Agropochvovedeniyu, Agrokhimii i Mikrobiologii Vsesoyuznoi Sakharnoi Promyshlennosti" (Collected Reports on Agricultural Soil Science, Agrochemistry, and Microbiology of the All-Union Sugar Industry), 1936; quoted after T.V.Kolesnikova, Symposium,"Svinets v Okruzhayushchei Srede" (Lead in the Environment), Moscow, 1978, p.18.
51. S.E.Sapatyi and R.N.Shkvaruk, Symposium, "Mikroelementy v Sel'skom Khozyastve i Meditsine" (Microelements in Agriculture and Medicine), Kiev, 1968, No.4, p.110.
52. T.V.Kolesnikova, Symposium, "Svinets v Okruzhayuschei Srede" (Lead in the Environment), Moscow, 1978, p.19.
53. R.W.Carlson and C.L.Rolfe, J.Environ.Qual., 1979, Vol.8, p.348.
54. A.P.Vinogradov, in "Geokhimicheskie Poiski Rudnykh Mestorozhdenii v SSSR" (Geochemical Search for Ore Deposits in the USSR), Gosgeoltekhizdat, Moscow, 1957, p.49.
55. J,K.Matt, Water, Air, Soil Pollut., 1977, Vol.8, p.133.
56. O.Horak, Bodenkultur, 1979, B.30, S.120.
57. R.Bulinski, A.Kot, K.Kutulas, and E.Szydlowska, Bromatol.i Chem.Toksykol., 1977, Vol.10, p.395.

58. H.Arfert and H.Harlos, Z.gesamte Hug., 1977, B.23, S.455.
59. H.Ocker, Getreide Mehl und Brot., 1974, B.28, S.204.
60. H.Ocker and W.Nierle, Getreide Mehl und Brot., 1974, B.28, S.285.
61. W.Siebel and H.D.Ocker, Dtsch.Iebensmittel-Rdsch., 1974, B.70, S.315.
62. B.Ya.Litver, A.I.Nizhnikov, P.V.Ramzaev, L.A.Teplykh, and M.N.Troitskaya, "Svinets-210, Polonii-210, Radii-226, Torii-228 v Biosfere Krainego Severa SSSR" (Lead-210, Polonium-210, Radium-226, and Thorium-228 in the Biosphere of the Extreme North of the USSR), Atomizdat, Moscow, 1976, p.3.
63. B.Ya.Litver, P.V.Ramzaev, A.A.Moiseev, M.N.Troitskaya, E.M.Krisyuk, E.B.Lisachenko, and A.I.Nizhnikov, "Svinets-210 i Polonii-210 v Priarkticheskikh Raionakh Vostochnogo Severa" (Lead-210 and Polonium-210 in the Arctic Regions of the North-West), Atomizdat, Moscow, 1969, p.9.
64. A.I.Nizhnikov, A.P.Ermolaeva-Makovskaya, M.S.Ibatullin, E.P.Lisachenko, B.Ya.Litver, P.V.Ramzaev, L.A.Teplykh, and M.M.Troitskaya, "Svinets-210, Polonii-210, Radii-226, Torii-228, Plutonii-239 v Tsepochke Lishainik-Olen'-Chelovek na Krainem Severe SSSR" (Lead-210, Polonium-210, Radium-226, Thorium-228, and Plutonium-239 in the Lichen–Deer–Man Chain in the Extreme North of the USSR), Atomizdat, Moscow, 1973, p.3
65. I.D.McCreight and D.B.Schroeder, Environ.Pollut., 1977, Vol.13, p.265.
66. B.Schnellmann and O.H.Opitz, Lebensmitteldem. Gerichte Chem., 1978, B.5, S.97.
67. N.L.Gale, B.G.Wixson, M.G.Hardie, and J.C.Jennet, Bull.Amer.Water Res.Assos., 1973, Vol.2, p.673.
68. B.G.Wixson and N.L.Gale, in "Vzaimodeistvie Mezhdu Vodoi i Zhivym Veshchestvom" (Interaction Between Water and Living Matter), Izd.Nauka, Moscow, 1979, Vol.3, p.84.
69. N.S.Khristoforova, N.A.Sin'kov, Yu.P.Badenkov, and O.L.Khristoforova, in "Vzaimodeistvie Mezhdu Vodoi i Zhivym Veshchestvom", (Interaction Between Water and Living Matter), Izd.Nauka, Moscow, 1979, Vol.1, p.97.
70. F.Pace, R.Ferrara, and G.D.Carrote, Bull.Environ. Contam.Toxicol., 1977, Vol.17, p.679.
71. J.B.Milne and M.Dickman, J.Environ.Sci.Health, 1977, Vol.A12, p.173.
72. B.A.Silberg, P.T.S.Wong, and Y.K.Chau, Arch. Environ.Contam.Toxicol., 1977, Vol.5, p.305.
73. T.J.Chow, Pure Appl.Chem., 1978, Vol.50, p.395.
74. C.C.Patterson, Arch.Environ.Health, 1965, Vol.11, p.344.
75. V.M.Drozdova and E.P.Machon'ko, J.Geophys.Res., 1970, Vol.75, p.3610.
76. A.W.Andren and S.E.Lindberg, Water, Air, Soil Pollut., 1977, Vol.8, p.199.
77. J.Servant and M.Delapart, Environ.Sci.Technol., 1979, Vol.13, p.105.
78. C.Bourton and C.Lorius, Nature 1979, Vol.277, p.551.
79. R.Delmas and C.Boutron, Atmos.Envir., 1978, Vol.12, p.723.
80. M.Murosumi, T.J.Chow, and C.C.Patterson, Geochim.Cosmochim.Acta, 1969, Vol.33, p.1247.
81. J.Sanak and G.Lambert, Geophys.Res.Letters, 1977, Vol.4, p.3579.
82. S.J.Eisenreich, A.W.Elzermann, and D.E.Armstrong, Environ.Sci.Technol., 1978, Vol.12, p.413.
83. "Pravila Okhrany Poverkhnostnykh Vod od Zagryazneniya Stochnymi Vodami" (Rules Governing the Protection of Surface Waters from Pollution by Waste Water), Izd.Min.Zdrav.SSSR, Moscow, 1961, p.18.
84. "Metodicheskie Ukazaniya dlya Organov Gosudarsvennogo Sanitarnogo Nadzora po Primeneniyu Pravil Okhrany Poverkhnostnykh Vod ot Zagryazneniya Stochnymi Vodami" (Methodological Instructions to Organs of the State Sanitary Supervision of the Application of the Rules Governing the Protection of Surface Waters from Contamination by Waste Water), Izd.Min.Zdrav.SSSR, Moscow, 1963, p.3.
85. G.N.Krasovskii, L.G.Vasukovich, and O.G.Chariev, Environ.Health.Perspect., 1979, Vol.30, p.47.
86. W.H.Parry, Lancet, 1967, No.7527, p.1207.
87. R.A.Greig, R.N.Reid, and D.R.Wenzloff, Mar. Pollut.Bull., 1977, Vol.8, p.183.
88. J.G.Farmer, Sci.Total.Environ.1978, Vol.10, p.117.
89. S.A.Mamontova and L.G.Bogashova, Vestnik, Moskov. Gos.Univ., Ser.Geol., 1979, Vol.4, p.93.
90. E.R.Christensen, J.Schherfig, and M.Koide, Environ.Sci.Technol., 1978, Vol.12, p.1168.
91. H.Tarrah and W.F.Pickering, Water, Air, Soil Pollut., 1977, Vol.8, p.187.
92. J.Vuceta and J.J.Morgan, Environ.Sci.Technol., 1978, Vol.12, p.1302.
93. J.S.Lu and K.Y.Chen, Environ.Sci.Technol., 1977, Vol.11, p.174.
94. K.Schwarz, "International Symposium on the Environment Lead Research, Dubrovnik, 1975", Arch.Ind.Hyg.Toxicol., **26**, (Suppl.), 13 (1975).
95. J.H.Heffron, G.S.Stoewsand, C.A.Bache, P.C.Wczolek, and D.J.Lisk, Cornell Vet., 1975, Vol.69, p.356.
96. A.A.Mylroie, L.Moore, B.Olyal, and M.Anderson, Environ.Res., 1978, Vol.14, p.57.
97. J.Monkiewicz, I.Dynarovicz, S.Jaczewski, and J.A.Madey, Med.Wet.1977, Vol.33, s471.
98. R.F.Willes, E.Lok, J.F.Truelove, and A.J.Sundaram, Toxicol Environ.Health, 1977, Vol.3, p.395.
99. M.-L.Johansson-Sjöbeck and A.Larsson, Arch. Environ.Contam.Toxicol., 1979, Vol.8, p.419.
100. B.R.Nechay and J.P.Sannders, J.Toxicol.Environ. Health, 1978, Vol.4, p.147.
101. Z.Zapryanov, and G.Antonov, Pharmakol.i Toksikol., 1977, Vol.40, p.617.
102. G.N.Krasovskii, O.G.Charyev, and S.P.Varshavskaya, Symposium, "Svinets v Okruzhayushchei Srede" (Lead in the Environment) Moscow, 1978, p.48.
103. Ch.Deknudt and M.Deminatti, Toxicology, 1978, Vol.10, p.67.
104. A.B.Fowler, Environ.Health.Perspect., 1978, Vol.22, p.37.
105. R.Angelieva and S.Novakova, Khig.Zdraveolaz., 1979, Vol.22, p.284.
106. J.P.Bushnell, S.E.Shelton, and R.E.Bowman, Bull.Environ.Contam.Toxicol., 1979, Vol.22, p.819.
107. C.Marcel and B.James, Castroenterology, 1978, Vol.74, p731.
108. R.A.Willoughby, D.G.Thawley, and G.Brown, "International Symposium on Environment Lead Research, Dubrovnik, 1975", Arch.Ind.Hyg.Toxicol., **26** (Suppl), **29** (1975).
109. R.A.Goyer, J.Lab.Clin.Med., 1978, Vol.91, p.363.
110. J.C.Barton, M.E.Courad, L.Harrison, and S.Nuby, J.Lab.Clin.Med., 1978, Vol.91, p.366.

111. A.R.Carol, M.S.McIntire, and G.Brunk, J.Toxicol. Environ.Health, 1977, Vol.3, p.557.
112. G.B.Gerber and J.Maes, Toxicology, 1978, Vol.9. p.173.
113. O.A.Levander, Environ.Health Perspect., 1979, Vol.29, p.115.
114. O.A.Levander, V.C.Morris, and R.J.Ferretti, J. Nutr., 1978, Vol.108, p.145.
115. A.I.Burkhanov, Symposium, "Gigiena Truda v Chernoi i Tsvetnoi Metallurgii" (Labour Hygiene in Ferrous and Non-Ferrous Metallurgy), Izd.Min. Zdrav.Kazakh.SSR, Karaganda, 1977, p.120.
116. D.R.Howard and L.Penumarthy, Vet.Human Toxicol., 1979, Vol.21, p.243, Chem.Abs., 1979, Vol.91, 152242.
117. C.L.Stone, K.R.Mahaffey, and M.R.Spivey, J. Environ.Pathol.Toxicol., 1979, Vol.2, p.767.
118. S.Ghergariu, L.Kadar, B.Bohm, I.Grindeanu, and M.Teucean, Recl.Med.Vet., 1978, Vol.154, p.633; Chem.Abs., 1979, Vol.90, 17250.
119. E.Forschner and H.O.Wolf, Fleischwirtschaft, 1979, B.59, S.872.
120. J.Holm, Fleischwirtschaft, 1979, Vol.59, p.737.
121. O.Ruttner and H.Jarc, Wien.Tieraerztl.Monatschr., 1979, Vol.66, p.259.
122. R.P.Sharma, J.C.Street, J.L.Shupe, and D.J.Wagstaff, Toxicol.Appl.Pharmacol., 1977, Vol.41, p.150.
123. S.G.Capar and J.H.Gould, J.Assoc.Anal.Chem., 1979, Vol.62, p.1054.
124. F.G.Lowman, Bull.Commercial Fisheries, 1970; quoted after "Lead Pesticides, Man and the Environment", EPA, Washington, 1972, p.25.
125. N.S.Risik and V.I.Romanov, in "Vzaimodeistvie Mezhdu Vodoi i Zhivym Veshchestvom" (Interaction between Water and Living Matter), Izd.Nauka, Moscow, 1979, Vol.1, p.222.
126. E.J.Harvey and L.A.Knight, Water, Air, Soil Pollut., 1978, Vol.9, p255.
127. J.D.Thomson, Austral.J.Mar.Fresh.Water.Res., 1979, Vol.30, p.421.
128. S.Ray, Bull.Environ.Contam.Toxicol., 1978, Vol.12, p.631.
129. R.Gajewska and M.Nabrzyski, Roczn.Państwowego Zakładu Higieny (Pol.), 1977, Vol.28, p.215.
130. K.S.Badsha and M.Sainsbury, Mar.Pollut.Bul., 1977, Vol.8, p.164.
131. N.Oudart, C.Guichart, and C.Gelage, Eur.J. Toxicol.Environ.Hyg., 1976, Vol.9, p.432.
132. H.J.Bielig, A.Askar, and H.Treptow, Fluess.Obst., 1978, Vol.45, p.410.
133. M.Nikonorow and H.Piekacz, Roczn.Państwowego Zakładu Higieny (Pol.), 1978, Vol.29, p.121.
134. T.Zawadska and B.Brzozowska, Roczn.Państwowego Zakładu Higieny (Pol.), 1979, Vol.30, p.115.
135. M.A.Wessel and A.Dominski, Amer.Sci., 1977, Vol.65, p.294.
136. E.E.Ziegler, B.B.Edwards, R.I.Jensen, K.R.Mahaffey, and S.T.Fomon, Pediat.Res., 1978, Vol.12, p.29.
137. N.Erenturk, Tech.J., 1979, Vol.6, p.13.
138. J.P.Day, J.E.Fergusson, and Tay Ming Chee, Bull.Environ.Contam.Toxicol., 1979, Vol.23, p.497.
139. Z.Szczepański and K.Król, Pediat.Polska, 1979, Vol.45, p.1077.
140. T.Koreeda, K.Kashiwabara, S.Shirouzu, and M.Yuguchi, Kyorin Igakkai Zasshi, 1979, Vol.10, p.15; Chem.Abs., 1979, Vol.91 188560.
141. R.F.Herbert and H.J.Salle, Int.Arch.Occup.Environ. Health, 1978, Vol.41, p.147.
142. C.E.Rolton and B.J.Horton, Austral.Vet.J., 1978, Vol.54, p.393.
143. J.Eisinger, Bull.Amer.Phys.Soc., 1978, Vol.23, p.191.
144. J.Eisinger, W.E.Blumberg, A.Fischbein, and J.J.Selikoff, Int.Arch.Occup.Environ.Health, 1978, Vol.41, p.217.
145. J.Eisinger, Quart.Rev.Biphys., 1979, Vol.11, p.43.
146. P.Wligert and H.Fischer, Wiss.Umwelt, 1979, B.2, S.99.
147. P.Grandgean, D.V.Nielson, and J.M.Shapiro, Amer. J.Phys.Anthropol., 1979, Vol.50, p.444.
148. E.L.Baker, C.G.Hayers, P.J.Landrian, J.L.Handke, R.T.Leger, W.J.Housworth, and M.J.Harrington, J. Amer.J.Epidemiol., 1977, Vol.106, p.261.
149. D.Jenkins, in "The Use of Biological Specimens for the Assessment of Human Exposure to Environmental Pollutants", Mattinus Nijhoff Publishers, The Hague–Boston–London, 1979, p.215.
150. J.Teisinger, Arch.Environ.Health, 1971, Vol.23, p.280.
151. W.Blumer, in "Diagnosis and Therapy of Porphyrias and Lead Intoxication", Springer–Verlag, Berlin, 1979, p.165.
152. M.M.Peaslee and F.A.Eigenllig, Experienta, 1977, Vol.33, p.1206.
153. A.J.Paskins-Hurlburt, Y.Tanaka, S.C.Skoryna, W.Moore, and J.F.Stara, Environ.Res., 1977, Vol.14, p.128.

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