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INTERNATIONAL SYMPOSIUM

Environmental health aspects of lead

Die gesundheitlichen Aspekte der Umweltverschmutzung durch Blei

Les problèmes sanitaires posés par le plomb présent dans l'environnement

LEAD ABSORPTION AND PUBLIC HEALTH:
AN APPRAISAL OF HAZARDS

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SUMMARY

Pb in blood (PbB) indirectly measures biologically active body burden. Usually PbB < 40 µg Pb/100 ml. This level prevents evident increase of ALA in urine and acute or chronic effects of lead. There is a large safety factor if compared to acceptable occupational load. ALA-dehydratase in erythrocytes decreases to ≥ 1/3; together with PbB it provides a sensitive and specific indirect indicator of biologically active body burden. Standards should apply to total daily load, and not to respiratory load. Acceptable total daily load corresponds to acceptable biological parameters for PbB, ALAU and ALAD; guidelines are suggested for groups and individuals. Undue attention to Pb in petrol misdirects allocation of funds and manpower from other much more important effects from traffic on public health. More attention should be paid to ecological effects of Pb.

Introduction

The question to be discussed is:
Does exposure to lead by non-occupationally exposed human subjects under normal living conditions produce any biological responses, and if so, do these responses present any risk to health and well-being? Specific conditions of slum areas, of living near Pb emitting plants, or of pica do not present "normal living conditions", and are not relevant for the main topic of discussion.

Due to limitations in space, imposed by the Organ. Cee of the Conference, this paper may sometimes appear to be apodictical; many literature sources could not be discussed as such.

Parameters of absorption

One should like to measure the internal chemical load to those receptor sites which react with a specific and sensitive response at the same time being relevant for health. A direct parameter is not available in epidemiology. In practice one has to rely on an indirect approach: determination of lead in blood (PbB), although valid objectivious exist against this parameter:

1. it is an indirect parameter of Pb burden in receptor sites.
2. various internal and external factors may influence Pb distribution through the body.
3. the body burden may be divided into: rapid exchange pool (blood, internal organs), intermediate exchange pool (muscles, skin), slow exchange pool (bone) (Pietrowsky 1970); PbB therefore does not indicate the total body burden. EDTA-induced Pb excretion (Teisinger et al 1966) might yield a more valid parameter for the rapid exchange pool than PbB.

Conclusion: in individuals PbB has its limitations; in groups variability will average out; up to the present distribution of Pb levels ($\bar{x} \pm \sigma$) offers the best practical tool to estimate the biologically active load burden in groups of population.

Table 1 and Figure 1 present a survey of PbB levels in various population groups from geographically widely distributed areas, published after 1960. We may draw the following conclusions from this table and from literature:

1. the normal range of PbB is 10-40 µg Pb/100 ml, with mean levels <30 µg Pb/100 ml.
2. in the majority of cases the upper limit does not exceed 40 µg Pb/100 ml, if high risk groups (a.o. slum areas) are excluded.
3. in last decades PbB has not evidently increased (Stoppe 1966).
4. urban PbB may be slightly higher than rural PbB.

The question to be discussed may now be rephrased as follows: Does an internal chemical load as indicated by PbB levels up to 40 µg Pb/100 ml present any risk to public health?

Parameters of response

The internal chemical load induces biological responses (effects). A no-effect level of PbB only indicates absence of specified effects to be determined with specified methods (analytical, statistical), and does not indicate absence of any effects.

A. Effects on porphyrin synthesis

If effects on porphyrin synthesis do not exceed specified limits, acute intoxication or chronic sequelae will not occur. Hb does not decrease below PbB < 50 µg Pb/100 ml. The workshop on inorganic lead Perm. Cee Int.Ass.Occ.Health agreed upon following limits for adult occupationally exposed workers: PbB 70 µg Pb/100 ml, PbU 130 µg/l, ALAU 10 µ/l, CPU 300 µg/l (Zielhuis 1969). ALAU (aminolaevulinic acid excretion in urin) is more specific and more sensitive than CPU (coproporphyrin in urin), so for public health ALAU is more relevant.

Table 2 and Figure 2 present the relationship between PbB and ALAU. The following conclusions may be drawn:

1. the no-effect level of PbB is about 40 µg Pb/100 ml; increase in figure 2 below 40 µg Pb/100 ml derives from mathematical calculation and is hardly evident in practice.
2. ALAU does not present a sensitive parameter for biologic monitoring in public health.

According to some authors (a.o. Chisolm 1971) ALAU does not appear to be sensitive in children.

More sensitive and more specific is the decrease in activity δ-ALA-dehydratase in erythrocytes (ALAD); it appears to be too sitive for use in monitoring of workers. Table 3 and Figure 3 pr sent the relationship with PbB. From these and other literature data the following conclusions may be drawn:

1. ALAD is highly specific for early responses to lead
2. in patients with neurological and haematological disorders no decrease of ALAD has been noted
3. haemsynthesis is probably not affected if ALAD > 1/3 normal l vel
4. in rats there exists a linear relationship between ALAD in bl and in brain
5. relationship PbB x ALAD is the same in adults and in children
6. there do exist indications that the no-effect level of PbB is about 5-15 µg Pb/100 ml (Schaller et al 1971).

The difference in sensitivity in regard to PbB between ALAU a ALAD is presented schematically in Figure 4: ALAD has little sig ficance for occupational health, ALAU has little significance fo public health.

As far as known decrease of ALAD as such has no or little sig ficance for health, if the activity remains > 1/3 normal level; body appears to possess a high reserve capacity. ALAD is a highl sensitive and specific parameter for early response, and indirec for biologically active internal load, but as such is not releva for health.

Protoporphyrin in erythrocytes (PP) increases from about PbB 40 µg Pb/100 ml (Haeger-Aronsen 1971a); it is less sensitive the ALAU (Albahary 1968), and therefore not relevant for public heal

B. Effects on erythrocytes

Lead affects erythrocytes: decreased: Na^+-K^+-ATPase activity membranes, life span, increased: K^+efflux, glucose consumption, osmotic resistance, pyruvate levels after glucose dosage, preval of Heinz bodies. These responses however are less specific and s sensitive than ALAU; they probably do not appear below PbB = 40 Pb/100 ml (Hernberg 1970, Qazi 1971, Koncrieff et al 1964, Ghel 1966a).

C. Other biochemical effects

Numerous effects have been reviewed by De Bruin (1971): decreased: immunobiological reactivity, alkaline phosphatase, cholinesterase, carbon anhydrase, amino acid levels, increased: serum aldolase and catalase, modifications in serum protein pattern, etcetera. Most or all of these effects have only been observed in workers with symptoms of poisoning or with relatively high exposure. They do not appear to be relevant for PbB < 40 µg Pb/100 ml. One animal experiment (Bingham 1969) may appear to be relevant: decrease in alveolar macrophage in young male rats, if exposed to Pb in air (PbA) = 10 µg Pb/m^3; this reversible effect still has to be studied in human subjects in order to evaluate its relevance for health.

Ghelberg et al (1966b) found increased urinary excretion of 5-hydroxyindolacetic acid in 11 year old children living in the vicinity of a lead emitting industry (PbA about 31 µg Pb/m^3); this parameter is less sensitive than ALAU (Urbanowicz et al 1969).

D. Effects on clinical health and life span

Possible effects on kidney function, tension, cerebrovascular incidents are only seen in subjects with longterm high exposure (PbA > 150 µg Pb/m^3) or with acute intoxications in their medical history (Malcolm 1970, 1971; Cramer et al 1966, Stopps 1966). Hickey et al (1967) studied specific mortality rates in the general population in relationship to air pollution with metals (PbA: 1.1396 µg Pb/m^3); there was no relationship with disease classification, in contrast to possible relationship with Cd and V. Szadkowsky et al (1969) did not observe any pathogenetic relationship between PbB and various internal diseases. In a soft water area with relative high Pb intake the increased cardiovascular mortality did not appear to be related to Pb intake (Crawford et al 1969). Effects on chromosomes have been reported, however only in subjects with high PbB (Schvanitz et al 1970); in drosophila there was no effect of inorganic lead (Ahlberg et al 1972).

The animal experiments of Schroeder et al (a.o. 1968) have drawn much attention: in mice and rats long term oral intake of 5 ppm Pb in drinking water gave rise to Pb levels in soft tissues in the same order of magnitude as in American adults; moreover there was a decreased life span. However, these data cannot be extrapolated to human beings, because apparently the kinetics of lead in rats and mice differ considerably from those in man; the daily oral intake of these animals was 400-500 µg/kg body weight, about 80-1 times that in human beings; moreover the rats received a Cr-deficient diet. Schroeder et al (1968) found increased Pb content of ribs in American adults up to the 5th decade; the lack of increase in older age groups might - according to Schroeder et al - be caused by shortened life span due to increasing Pb body burden. These data have been severily criticised by Kehoe (1969) and could not be confirmed by Barry et al (1970).

Conclusion: A lead body burden, represented by PbB < 40 µg Pb/100 ml probably is consistent with absence of effects on ALAU, PP, and other biochemical parameters, clinical health and life span. Conclusive evidence of such effects have not been established, and data with suggestive evidence hardly exist.

Relationship daily Pb uptake and PbB

There does not exist adequate information on the relationship between longterm daily exposure and PbB. The Nat. Research Council - NAS (EPA 1972) suggested a curvilinear relationship between Pb in air (PbA in µg Pb/m^3) and PbB: PbB = 69.2052 + 54.7605 log PbA. This aequation apparently is very approximate, more or less a "guestimate". The following assumptions have been made:

1. 24 hr continuous exposure → overestimation of daily exposure
2. daily respiratory volume 23 m^3 → overestimated; 15-20 m^3 appears to be more reasonable
3. 30% retention and 100% resorption; one may assume maximal 100% resorption after maximal 50% retention (Schlipköter 1972)
4. daily oral intake 300 µg Pb, which appears to be questionable.

The aequation probably gives maximal PbB in regard to current PbA levels and overestimates the realistic relationship.

Some data from other sources (Kehoe 1961, 1966; Williams 1968) (respiratory and/or oral intake) have been recalculated, and added to the NAS-aequation(Table 4); multiplication of PbA with 5 corresponds to multiplication of PbB with about 2.

The maximal allowable PbB for adult workers is 70 µg Pb/100 ml according to Working group on inorganic lead Perm. Cee Int.Ass.Occ. Hlth (Zielhuis 1969); this corresponds to a safety factor of 10-20 for continuous 24 hr exposure to Pb for the adult general population. This safety factor is large if compared to safety factors as applied in food additives (from animal → man: 10; from man → man:10).

The relationship between PbA and PbB is based upon calculations used in occupational health for uptake of particles with aerodynamic properties, and about 1 µ in diameter. Recently Lawther et al (1972) seriously critisized this procedure, because in sampling air near a heavy traffic road in London Pb proved to be present in aggregates and particles of much smaller diameter, e.g. 0.01µ; according to Lawther one may presume a maximal retention x absorption of 10-12%, much lower than assumed retention x absorption of 30-50%. This appeared particularly true for Pb emitted by motorcars. Therefore the safety factor in regard to respiratory Pb uptake from traffic exhaust may increase with a factor 2-4, and become 20-40.

In table 4 a daily oral uptake of 300 µg Pb has been assumed (review by Karthausen 1972). This intake may differ considerably: 100-500 µg Pb/day. In Europe Lehnert et al (1969) calculated a daily intake of 518 µg Pb for Germany (DBR), in U.K. Thompson (1971) measured average 274 µg Pb (70-750), Kloke et al (1968) estimated for Germany (DBR) an intake up to 1400 µg Pb/day, Viglianni et al (1969) estimated 400-500 µg Pb/day for Miland, Italy; Koriuchi measured 230-320 µg Pb/day for Japan. The Comm.Europ. Community undertook a comparison of total diets, excluding alcoholics, of adolescents in various countries within the Common Market (Smeets et al 1970). If we apply the data of Lehnert et al (1969) to the average diet, we arrive at a relative difference in oral daily Pb intake with a factor 2 (Table 5). If between groups the long term oral Pb uptake differs 200 µg Pb/day, then daily gastrointestinal absorption will differ about 20 µg Pb. This corresponds to a difference in 12 hr respiratory exposure of about 5 µg Pb/m^3. Such a large difference in daily respiratory exposure appears to be more unrealistic than a corresponding difference in oral exposure, at least for Europe.

These considerations emphasize an important point: the total Pb uptake mainly stems from oral uptake; it is therefore unrealistic to propose MIC values, one should determine an acceptable total daily load. However, it is very difficult to measure this. This may emphasize the feasibility of biological sampling, either through parameters of absorption (PbB) or parameters of response (ALAD), both indirectly measuring the biologically active body burden, and at least in groups of the population with a reasonable degree of reliability. In adults the biologically active body burden appears to a large extent to be independent from age (Barry et al 1970), in contrast to the slow or hardly exchangeable body burden in teeth and bones.

Children, especially in younger age groups, should be regarded as a special group, distinct from adults. They probably differ in:

1. increased hazard of oral uptake, particularly in toddlers
2. increased gastrointestinal resorption: in young rats up to 80% if milk with a low Ca/P ratio is being administered (Kostial et al, 1971a, 1971b)
3. the daily oral uptake if taken per kg body weight far exceeds that in adults (Chisolm et al 1956, Baltrop et al 1967: average 120-130 µg Pb, 95% < 183 µg)
4. the relative proportion of the body burden present in the slow exchange pool is smaller (Barry et al 1970)

Assessment of risks for adults in the general population

The data regarding adult population groups so far discussed may be summarized as follows:

1. PbB normally does not exceed 40 µg Pb/100 ml
2. the no effect level in regard to increased ALAU is about 40 µg Pb/100 ml and very probably higher for most signs and symptoms relevant for health
3. ALAD in erythrocytes decreases from PbB = about 10 µg Pb/100 ml to 1/3 of its activity at PbB = 40 µg Pb/100 ml; up to now there is no evidence that this decrease as such has any significance in regard to health or wellbeing, either at short or at long term

4. there exists a safety factor of at least 10-20 in respiratory 24 hr uptake for the adult general population if referred to acceptable respiratory uptake of workers, based upon the relationship between PbA and PbB
5. between population groups oral uptake probably differs to a larger extent than respiratory uptake; total daily uptake mainly stems from oral uptake; one should determine total acceptable daily Pb uptake
6. biological monitoring (PbB or ALAD) provides an easier, more realistic and more valid indirect yardstick of total daily uptake in population groups than a multitude of individual direct measurements of Pb in food, beverages and air.

There are gaps in knowledge; even when research is going on, gaps in knowledge will always remain. Nevertheless, at this moment there are sufficient data available in order to come to a justified policy for establishing acceptable total daily loads for adult population groups. However, we should bear in mind the gaps in knowledge as mentioned by for instance Hardy (1969) and Smith (1969):

1. not enough data exist regarding the extent of variability in total daily exposure
2. high risk groups (PbB 30-50 µg Pb/100 ml) especially should be studied more fully
3. there exists the possibility of increased susceptibility due to genetic defects, e.g. sickle cell trait, or due to pregnancy.

Assessment of risks for children

For children we must add some extra points for discussion, summarizing the data mentioned so far:

1. the total daily load per kg body weight is appreciably higher
2. the possibilities for oral uptake, particularly in toddlers, are higher, whereas intestinal resorption may appreciably go beyond the 10% as assumed for adults
3. the oxygen consumption in the brain is relatively high; there are indications that a body burden corresponding to PbB $\geq$ 40 µg Pb/100 ml may aggravate an already existing mental retardation

4. the rapidly exchangeable body burden is relatively higher
5. the acceptable limit of PbB = 70 µg Pb/100 ml as proposed for healthy adults, probably does not apply for children; the margin of safety is probably smaller.

Future research should especially be directed at gathering data from young children: measurement of daily total uptake, body burden (also with EDTA provocation tests), biological responses; particularly long term follow up studies are needed, taking fully into account data on mental development.

Acceptable biological limits

Environmental standards have to take into account total daily uptake; this can be measured indirectly by biological monitoring of PbB, ALAD and ALAU. Particularly for more or less homogeneous groups one may conclude to acceptable biological limits, characterised by group average and upper limit; for individuals one may present upper limits. These limits should not be regarded as fine lines discriminating innocent from noxuous environments; however, if these limits are exceeded the possibility of undue acute effects and/or chronic sequelae increases. The data as established in population groups should always be interpreted by experts, and not by the lay public as such. Single values moderately exceeding upper limits in individuals should cause awareness and caution; the measurement should be repeated. Group data have a stronger significance than individual data.

The following acceptable limits are suggested as guidelines:

For adults

	individual upper limit	group average	unit
PbB	$\leq$ 40	$\leq$ 25	µg Pb/100 ml
ALAU	$\leq$ 6	$\leq$ 3	mg/l urin
ALAD	$\geq$ 20	$\geq$ 30	procentual decrease from 100% (at PbB = 10 µg Pb/100 ml)

For children

	individual upper limit	group average	
PbB	$\leq$ 35	$\leq$ 20	as above
ALAU	$\leq$ 5	$\leq$ 3	"
ALAD	$\geq$ 30	$\geq$ 40	"

Pb in traffic exhaust

Within the Common Market recently much attention has been paid to the public health aspects of exposure to traffic exhaust. A few comments should be made:

1. There are insufficient data regarding the effect of Pb in petrol on total daily intake; as yet there are no indications that oral intake of Pb in adults has increased due to traffic exhaust. PbB levels in USA have not evidently increased in last decades (Stoppe 1966).
2. Regional variability in oral uptake probably exceeds variability in uptake due to traffic exhaust.
3. Traffic exhaust appears to yield a small contribution to Pb uptake in children if compared to many other sources of lead (Baltrop 1972).
4. Up to the present moment there are no indications that under conditions prevailing in the Common Market health and wellbeing of adults and children has been negatively affected by lead in traffic exhaust.
5. The overwhelming attention in the Common Market to Pb in traffic exhaust yields a greatly distorted picture of the total range of negative effects of traffic on public health: accidents, noise, traffic congestion, traffic exhaust. The most promising and rewarding approach to tackle the problem of traffic and public health is to decrease the distance between traffic and human subjects (living quarters, working quarters, playing grounds, etc.). If available money and manpower are solely or mainly directed to restriction of Pb in petrol, we may have a somewhat cleaner environment in respect to Pb, but the much greater other risks to health and wellbeing continue to exist and to increase.
6. Much attention is being paid to direct effects of Pb on human health. However, Pb ultimately pollutes the environment, particularly the oceans. If this affects primary production, indirect effects on human health and wellbeing may occur. This field of study deserves a higher priority in allocation of funds and manpower than research on direct effects on public health.

General conclusion

Pb is a non-essential element; therefore presence in the human biological system should be minimised as far as possible. In this respect restriction or elimination of Pb in petrol should be promoted. However, within the total significance of traffic for public health, other measures should receive a higher priority. If those measures - politically much more difficult to achieve - are taken, the potential risks of Pb in petrol to public health will indirectly decrease at the same time.

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TABLE 1. PbB levels (µg Pb/100 ml) in population groups; published after 1960

ADULTS

country	authors yr of publication	number of subjects	$\bar{x} \pm \sigma$	range	remarks
Germany (DBR)	Szadkowsky et al 1969a	176	$\bar{x}$: 10.3-19.2 σ: 3.0- 9.6	-	various internal diseases
Germany (DBR)	Szadkowsky et al 1969b	53	16.7 ± 6.8	-	-
Germany (DBR)	Lehnert 1968	400	14.9 ± 4.8	-	-
Germany (DBR)	Reichel et al 1970	44 10	15.1 ± 3.6 12.2 ± 5.9	- -	- policeman
USA	Stopps 1966	3637	about 22	-	some values outside USA; a few values up to 50-60
USA	Weissberg et al 1971	26	-	15-40	-
Finland	Hernberg et al 1970	16	-	5-16	-
Sweden	Haeger-Aronsen 1971a	100 35	8.5 ± 2.6 (♀) 12.3 ± 4.3 (♂)	- -	- -
Switzerland	Lob et al 1971	78	-	<40 19-71	traffic workers prisoners, alcoholics
Italy	Vigliani et al 1969	118	21.1 (♀) 30.0 (♂)	0-70 0-70	up to 70
Japan	Horiuchi 1970a	63	25	0-69	-
Un.Kingdom	Wilson 1966	8	8.7	0-20.4	pregnant women

Table 1 (continued)

country	authors yr of publication	number of subjects	$\bar{x} \pm \sigma$	range	remarks
S.America, Australia, Africa	Stopps 1969	204	-	12-23	primitive conditions
Various countries	Goldwater et al 1967	801	17 ± 11	-	98% < 50, in most countries <40
CHILDREN					
Germany (DBR)	Schaller et al 1971	196	11.5 ± 4.9	-	6-9 yr
Unit.Kingdom	Moncrieff et al 1964	80	-	14-42	4 mth - 14½ yr
Unit.Kingdom	Millar et al 1970	30 27	12.3 ± 2.7 14.7 ± 3.8	- -	IQ > 70 IQ < 70
Sweden	Haeger-Aronsen 1971a	15	10.9 ± 2.9	-	2 mth - 12 yr
USA	Blanksma et al 1969	746	23.5 ± 7.8	-	10 - 14 yr

Table 2: Relationship between PbB (x) and ALA-U (y)

author(s)	year of publication	number of subjects	relationship	remarks
Haeger-Aronsen	1971a	110	log y = 0.01294x - 0.8605	PbB 10-120 µg/100 ml y in mg/100 ml r=0.61
Lehnert et al	1970	158	no increase of y	PbB < 55 µg/100 ml
Lehnert et al	1969	127	y = 0.9x + 6.1	PbB 47.3 ± 24.8 µg/100 ml r=0.36
		138	no increase	PbB 26.0 ± 12.4 µg/100 ml
Hernberg et al	1970	141	x < 50 µg/100 ml no increase of y; x > 50 µg/100 ml increase of y	PbB 10-130 µg/100 ml
Haeger-Aronsen et al	1971b	260	x < 50/100 ml no increase of y; x > 50/100 ml increase of y	PbB 10-90 µg/100 ml
Selander et al	1970	150	log y = 0.0157x - 1.0985	y in mg/100 ml

Table 3: Relationship between ALAD-activity (y) in erythrocytes and PbB (x)

author(s)	year of publication	number of subjects	relationship	remarks
Weissberg et al	1971	234	linear	in total blood; children and adults; PbB < 80 µg/100 ml
Schaller et al	1971	196	no relationship	PbB 11.5 ± 4.9 µg/100 ml
		30	significant, r=-0.41	PbB 16-30 µg/100 ml
		15	significant, r=-0.63	PbB 20-30 µg/100 ml
		70	significant, r=-0.25	adults, average PbB 14.9 µg/100 ml
Hernberg et al	1970	158	log y = 2.274-0.018x r=-0.90	PbB 5-95 µg/100 ml
Haeger-Aronsen et al	1971b	260	log y = 2.291-0.020x r=-0.83	PbB 10-90 µg/100 ml children and adults
Millar et al	1970	57	log y = 6.7685-0.0436x r=-0.80	children. PbB 10-20 µg/100 ml
Secchio et al	1971	102	r=-0.41	PbB probably <50 µg/100 ml, not occupationally exposed
Baseeqz et al	1971	47	curvilinear r=-0.82	PbB 20-75 µg/100 ml (expressed as µg/100 ml ery)

Table 4. Relationship between lead in air (PbA in µg Pb/m³) and lead in blood (PbB in µg Pb/100 ml)

PbA NAS	PbA Kehoe	PbA Williams	PbB NAS	PbB Kehoe	PbB Williams
2.0	-	-	21.3	-	-
4.0	-	-	27.3	-	-
5.0	5.0	-	30.3	30	-
10.0	-	10.0	40.0	-	35
-	15.0	-	-	55	-
20.0	-	-	53.8	-	-
-	25.0	-	-	70	-
50.0	-	40	71.6	-	65
-	-	50	-	-	75
100.0	-	-	87.2	-	-
-	150	-	-	145	-

Table 5. Oral lead uptake in µg/day in various groups of adolescents within the Common Market, 1965-1967

foodstuff	milk	cereals	cheese	vegetables	potatoes	meat	
lead content	16.95 µg Pb/100 ml	16.20 µg Pb/100 g	61.00 µg Pb/100 g	83.86 µg Pb/100 g	9.15 µg Pb/100 g	36.04 µg Pb/100 g	total/day
Munster	85.43	83.59	18.30	209.65	33.12	62.71	492.80 µg Pb
München	33.56	103.19	13.42	245.71	33.67	63.79	493.34 µg Pb
Friberg	33.73	59.45	9.76	96.44	19.22	47.57	266.17 µg Pb
Zeist	112.38	61.07	13.42	93.92	16.01	37.48	334.28 µg Pb
Brussel	71.19	46.82	11.59	182.81	96.81	93.70	502.92 µg Pb
Rome	36.10	120.37	40.26	188.69	25.16	43.61	454.19 µg Pb
Catania	55.09	150.34	31.72	127.47	8.42	33.88	406.92 µg Pb
Milan	42.38	128.14	20.74	182.81	10.34	54.78	439.19 µg Pb
Sceaux	37.46	110.16	23.18	281.77	23.15	73.88	549.60 µg Pb

The calculated daily uptake is based upon:

1. Table 27: The principal diet constituents consumption and their variation coefficient, in Eur 3945, Comparison of the radioactive contamination of the total diet of adolescents in the community. Data for 1965-1967 (Smeets et al 1970)

2. G.Lehnert et al, Usuelle Bleibelastung durch Nahrungsmittel und Getränke. Arch. f. Hyg. und Bakteriologie 5(1969)403

Figure 1. PbB levels: $\bar{x} \pm 2\sigma$ (——) or range (——) in various countries, published after 1960

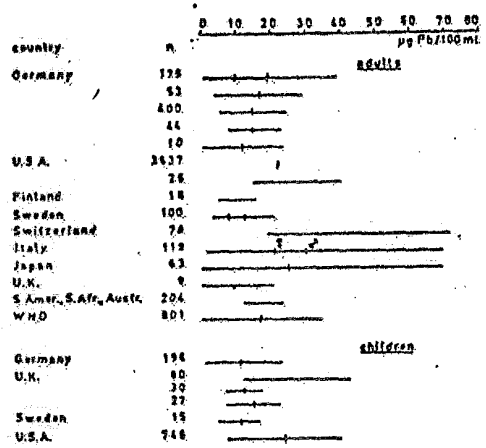

Figure 2. Increase of ALAU in relationship to PbB

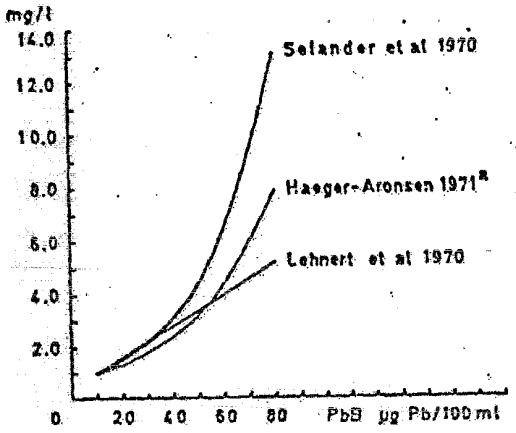

Figure 3. Percentual decrease in ALAD in relationship to PbB

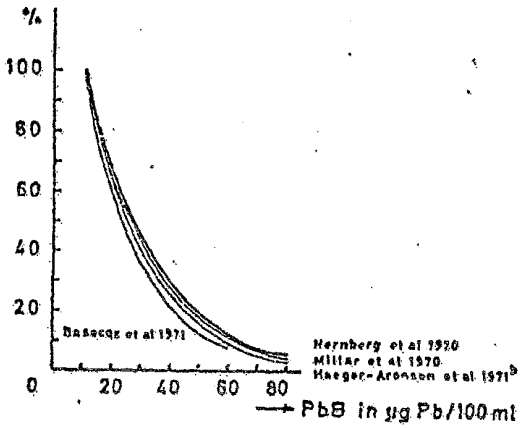

Figure 4. Percentual change in ALAD en ALAU in relationship to PbB