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The migration of lead from paint films in the rat gastro-intestinal tract

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Summary

The migration of lead from two paint films during their passage through the rat gastro-intestinal tract has been investigated. Both paints contained 2 per cent lead on the dried film, one as lead naphthenate and the other as lead chromate. Diets containing 1 per cent of the dried films were administered to groups of rats for a 12 week period; other groups received diets containing 100 ppm lead as lead naphthenate, and 20, 50 and 100 ppm lead as lead nitrate. All groups of rats remained in good health. Lead determinations were made at intervals on samples of bone, blood, kidney, liver, bile and urine. From a comparison of the analytical results it has been deduced that about one-half of the lead in the lead naphthenate paint, and about one-quarter in the lead chromate paint, migrates from the film under the experimental conditions. It is concluded that the present regulations controlling the lead content of paints intended for surfaces accessible to children are adequate to prevent future cases of lead poisoning from the ingestion of paint film.

Key words

Types and classes of coating
alkyd coating

Paint additives—driers
cobalt drier
lead naphthenate

Prime pigments
chrome orange

Properties primarily associated with materials in general
toxicity

La migration de plomb à partir des feuils de peinture dans le canal gastro-intestinal de rat

Résumé

On a étudié la migration de plomb à partir de deux feuils de peinture pendant leur passage à travers le canal gastro-intestinal de rat. Les deux peintures contiennent 2 pour cent de plomb dans le feuil sec, l'une sous forme de naphténate de plomb et l'autre comme chromate de plomb. Des régimes contenant 1 pour cent de feuils secs étaient donnés aux groupes de rats pendant une période de 12 semaines, d'autres groupes recevaient des régimes contenant 100 parties pour million de plomb comme naphténate de plomb, et de 20, 50, et 100 parties pour million de plomb comme nitrate de plomb. Tous les groupes de rats restaient en bonne santé. On a déterminé, par intervalles, la teneur en plomb aux échantillons d'os, sang, rein, foie, bile et urine. Par considération des résultats analytiques, on a déduit qu'un peu près de la moitié de plomb dans la peinture à naphténate de plomb, et un peu près du quart de plomb dans la peinture à chromate de plomb, doivent migrater sous les conditions expérimentales. On conclut que les règlements gouvernant la teneur en plomb des peintures destinées aux surfaces accessibles aux enfants sont suffisants pour éviter à l'avenir des cas d'intoxication par le plomb lors de l'ingestion des feuils de peinture.

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Das Wandern von Blei aus Anstrichfilmen in den Magen-Darmkanal von Ratten

Zusammenfassung

Die Migration von Blei aus zwei Anstrichfilmen während seines Durchganges durch den Magen-Darmkanal von Ratten wurde untersucht. Beide Farben enthielten 2 Prozent Blei (Trockengewicht), die eine als Bleinaphthenat und die andere als Bleichromat. Während zwölfwöchigen Perioden wurden Gruppen von Ratten mit 1 Prozent des Trockenfilms enthaltenden Diäten gefüttert; andere Gruppen erhielten Diäten mit 100 Teilen p.m. Blei als Naphthenat und 20, 50 und 100 Teilen p.m. Blei als Nitrat. Alle Rattengruppen blieben gesund. Bleibestimmungen wurden in Zwischenräumen an Proben von Knochen, Blut, Niere, Leber, Galle und Urin vorgenommen. Aus Vergleichen der analytischen Resultate wurde geschlossen, dass etwa die Hälfte des Bleies in der Bleinaphthenatfarbe und etwa ein Viertel in der Bleichromatfarbe unter den experimentellen Bedingungen aus dem Film wandert. Es wird gefolgert, dass die bestehenden Bestimmungen für die Kontrolle des Bleigehaltes von Farben, die für Kindern zugänglichen Oberflächen vorgesehen sind, genügen, um in Zukunft Fälle von Bleivergiftung durch Ingestion von Farbfilm zu verhüten.

Миграция свинца красочных пленок в желудочно-кишечном тракте крыс

Резюме

Исследовалась миграция свинца двух красочных пленок при их проходе через желудочно-кишечный тракт крыс. Обе краски содержали 2 процента свинца в сухой пленке, одна в виде нафтеносвинцовистой соли а другая в виде хромовокислого свинца. Диета содержащая 1 процент этих сухих пленок давалась группам крыс в течение 12-ти недель; другие группы крыс получали диету содержащую 100 частей на миллион свинца в виде нафтеносвинцовистой соли и 20, 50-и 100 частей на миллион свинца в виде азотносвинцовистой соли. Все группы крыс остались здоровыми. Определение свинца производилось через промежутки времени в образцах кости, крови, почек, печени, желчи и мочи. Сравнение аналитических результатов показало что около половины свинца в краске содержащей нафтеносвинцовистую соль и около четверти свинца в краске содержащей хромовокислый свинец, мигрирует из пленки при условиях опыта. Делается заключение что существующие требования контролирующие содержание свинца в красках предназначаемых для поверхностей доступных для детей, являются достаточными для предотвращения в будущем случаев отравления свинцом путем глотания красочной пленки.

Introduction

Since the turn of the century, there has been a progressive decrease in the incidence of occupational lead poisoning. In 1930 there were 32 fatal cases notified to the Factory Department, but none was reported over the period 1957-1966. Non-fatal cases fell from 233 in 1930 to 80 in 1966. These improvements have been in part due to the enforcement of regulations under the Factories Acts, and in part to advances in education and in the dissemination of information, and to a greater attention to occupational health.

Only a small proportion of the reported cases are attributable to the application of lead-containing paints. There have, however, been numerous cases of non-occupational lead poisoning, mainly in children, which are thought to derive from the ingestion of flakes of dried lead paints detached from treated woodwork. In Chicago alone, over the period 1959-1961, 429 such cases were recorded, of which 67 were fatal[2]. Other cases have been reported by Chisholm & Harrison[2] and by Moncrieff *et al*[3]. This high incidence of poisoning may be due to the high lead content of paints formerly used for interior decoration and to the friable nature of old paint films, which renders possible their disintegration into small particles with a large surface area when chewed and swallowed. It

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has also been suggested that young children are more sensitive to lead than are adults[4].

With the object of protecting children the paint industry gave a voluntary undertaking to the Ministry of Health in 1963 that paint containing more than 1 per cent lead on the dried film should bear a warning label indicating that the colour should not be applied to toys, furniture or interior surfaces which might be chewed by children. Subsequently the labelling of surface coatings intended for toys was controlled by the Toys (Safety) Regulations 1967 which limited the lead content of the film to 1.1 per cent, reducing to 0.5 per cent in 1968.

In a previous publication[4], the migration of lead from polymers in the gastro-intestinal tract has been studied by incorporating the ground polymer into the diet of rats and then measuring lead in tissues and excreta, and comparing the results with those of similar experiments in which a lead salt was incorporated in the diet. By comparing the amounts of lead absorbed in these two sets of experiments, it was possible to relate the diets containing the polymers to the diets containing lead salt, and hence to estimate the extent to which lead migrated from the polymer into the gut contents. It was concluded from these experiments that the difficulty in masticating these materials prevented any risk of lead poisoning from their ingestion. This limitation evidently does not apply to paint film, so the investigation has been extended to ascertain the extent to which lead can be mobilised from a paint film, and to confirm that the present regulations provide an adequate protection. Two paints have been included in the investigation; one containing lead as a drier and the other a lead pigment.

Experimental

Materials investigated

The paint used in this investigation was an off-white gloss finish based on an oil-modified alkyd resin. It contained cobalt drier without lead. To one portion was added Middle Orange Chrome pigment to produce approximately 2 per cent lead in the dried film, and to another portion was added lead naphthenate drier to produce approximately the same final lead content.

Preparation of diets

The paints were brushed on to pieces of tared rice paper, 75 × 50cm, and allowed to dry for three days. After reweighing to assess the weight of the paint film, the sheets were placed in an oven at 90°C for three hours to render them brittle. Unpainted rice paper was similarly dried for use in the control diet. The painted and control rice papers were broken up and homogenised in a small volume of water and the resulting slurry mixed with malt extract and corn oil and then incorporated into the standard rat diet[5] to give a final concentration of 1 per cent paint film in the pellets. The rice paper content of the diet was about 0.5 per cent, and the same amount was added to the control diet. The diets based on the lead chromate and lead naphthenate films were found by the analytical method described below to contain 192 and 190 ppm lead respectively. For the diets containing lead nitrate the salt was added in aqueous solution; lead naphthenate was dissolved in the corn oil. The control diet was found by the method described below to contain 1.3ppm lead.

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Animal experiments

The animal experiments were performed as previously described[4]. The diets were administered to groups of 30 young male albino rats for a 12 week period. At intervals animals were killed and samples of bone, blood, kidney and liver were taken for lead determinations. Analyses of bile and urine were made at 4 and 12 weeks; earlier experiments with rats receiving lead salt diets had shown that no appreciable amounts of lead were excreted in bile earlier than 4 weeks.

The weight of the rats was measured at weekly intervals throughout the experiment and the food intakes were recorded. At the end of the feeding period blood samples were examined for haemoglobin content, packed cell volume, white and differential cell counts, platelet count, and for the presence of stippled cells and reticulocytes. Bone marrow was examined for punctate basophilia and for immature red cells.

The determination of lead

The determination of lead in liver, kidney, bone, bile and urine was made by the methods previously described[4]. Increased sensitivity in the method for blood was achieved by carefully evaporating a 5ml sample to dryness with 2ml nitric acid and ashing the residue at 500°C. The ash was dissolved in 5ml water and 10ml 5N hydrochloric acid and the lead was determined by the colorimetric method. The experimental diets were ashed by the method used for bone and the residue was dissolved in 0.5ml hydrochloric acid and 0.25ml nitric acid and diluted to 10ml with water. The solution was polarographed with a Cambridge Pen-writing Polarograph and Univector over the range −0.2 to −0.8 v, and the peak heights obtained were compared with those of standard lead solutions.

Results

The concentrations of lead found in the tissues, bile and urine of the test and control rats throughout the experimental period are shown in Tables 1-6. In all experiments the rats remained in good condition and no significant difference was observed between the growth and food intake of test and control animals. No changes were observed in blood or bone marrow, indicative of early stages of lead poisoning.

The absorption of lead from lead salt diets

Lead nitrate: Table 4 shows little difference between the urinary excretion of lead by control animals, and by those receiving the lead nitrate diets, even at the highest concentration. These results confirm an earlier observation that urinary excretion in the rat cannot be used as an index of lead released into the gut[4].

The concentration of lead in bone, kidney and liver tissue increased with the dose. With kidneys and liver a rise is apparent within the first two weeks and the concentration remains fairly constant throughout the 12 week experiment. The accumulation in bone is slower and the concentration appears to reach an approximately constant value at about 8 weeks. The concentration of lead in the blood (Table 5) shows some correlation with the dietary concentra-

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Table 1
Lead content of bone (femur)

Diet	Weeks 0	2	4	8	12
Control	1.08 (10)	0.53 (4)	0.93 (4)	0.94 (4)	0.73 (4)
Lead nitrate					
as Pb 20ppm		0.90 (2)	1.36 (2)	3.31 (2)	1.67 (2)
as Pb 50ppm		2.14 (2)	3.00 (2)	5.11 (2)	3.74 (2)
as Pb 100ppm		4.41 (2)	6.90 (2)	5.72 (2)	7.34 (2)
Lead naphthenate					
as Pb 100ppm		3.11 (2)	6.03 (2)	6.17 (2)	6.20 (4)
Lead paints					
Chrome pigment		2.46 (2)	1.52 (2)	5.20 (2)	4.61 (2)
Naphthenate		4.34 (2)	2.15 (2)	9.23 (2)	7.57 (2)

Analytical results are expressed as μg/g wet weight, and are the means of determinations on the number of animals indicated in parentheses.

Table 2
Lead content of kidneys

Diet	Weeks 0	2	4	8	12
Control	0.45 (10)	0.24 (4)	0.40 (4)	0.27 (4)	0.22 (3)
Lead nitrate					
as Pb 20ppm		0.54 (2)	0.70 (2)	0.67 (2)	0.79 (2)
as Pb 50ppm		0.99 (2)	1.38 (2)	1.25 (2)	1.16 (2)
as Pb 100ppm		1.53 (2)	2.25 (2)	1.86 (2)	1.59 (2)
Lead naphthenate					
as Pb 100ppm		1.66 (2)	1.78 (2)	1.60 (2)	1.83 (2)
Lead paints					
Chrome pigment		1.00 (2)	0.99 (2)	1.05 (2)	0.98 (2)
Naphthenate		1.12 (2)	1.78 (2)	2.11 (2)	2.07 (2)

Analytical results are expressed as μg/g wet weight, and are the means of determinations on the number of animals indicated in parentheses.

Table 3
Lead content of liver

Diet	Weeks 0	2	4	8	12
Control	0.08 (5)	0.12 (4)	0.12 (4)	0.12 (4)	0.14 (2)
Lead nitrate					
as Pb 20ppm		0.21 (2)	0.19 (2)	0.22 (2)	0.28 (2)
as Pb 50ppm		0.23 (2)	0.28 (2)	0.30 (2)	0.29 (2)
as Pb 100ppm		0.35 (2)	0.30 (2)	0.37 (2)	0.31 (2)
Lead naphthenate					
as Pb 100ppm		0.28 (2)	0.28 (2)	0.24 (2)	0.31 (2)
Lead paints					
Chrome pigment		0.17 (2)	0.18 (2)	0.19 (2)	0.23 (2)
Naphthenate		0.23 (2)	0.33 (2)	0.30 (2)	0.32 (1)

Analytical results are expressed as μg/g wet weight, and are the means of determinations on the number of animals indicated in parentheses.

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Table 4
Lead content of urine

Week	Control	Lead salt diets (as Pb)				Paint diets	
		Lead nitrate			Lead naphthenate		
		20ppm	50ppm	100ppm	100ppm	Chrome pigment	Naphthenate
4	0.96 (26) 0.72 (17) 0.73 (20) 1.23 (19)	0.86 (22) 0.64 (8)	1.01 (18) 0.93 (17)	1.38 (18) 0.79 (13)	1.36 (30) 0.92 (14)	1.22 (30) 2.15 (23)	0.61 (23) 0.96 (10)
12	0.67 (12) 0.98 (12) 0.96 (32) 1.04 (24)	1.07 (32) 1.20 (30)	1.17 (16) 1.30 (21)	1.79 (24) 1.37 (25)	1.37 (31) 1.35 (26)	1.66 (19) 0.90 (24)	1.00 (16) 1.18 (27)

Results are expressed as μg/rat/24hr, and each is based on the pooled 24-hr urine from a group of five rats. Figures in parentheses, urine volume (ml/rat/24hr).

Table 5
Lead content of blood

Diet	Weeks				
	0	2	4	8	12
Control	0.12 (10)	0.07 (4)	0.04 (4)	0.07 (4)	0.11
Lead nitrate					
as Pb 20ppm		0.09 (2)	0.08 (2)	0.12 (2)	0.16 (2)
as Pb 50ppm		0.11 (2)	0.11 (2)	0.12 (2)	0.24 (2)
as Pb 100ppm		0.14 (2)	0.31 (2)	0.15 (2)	0.24 (2)
Lead naphthenate					
as Pb 100ppm		0.15 (2)	0.36 (2)	0.14 (2)	0.16 (2)
Lead paints					
Chrome pigment		0.10 (2)	0.06 (2)	0.06 (2)	0.13 (2)
Naphthenate		0.19 (2)	0.13 (2)	0.10 (2)	0.19 (2)

Results are expressed as μg·ml, and are the means of determinations on the number of animals indicated in parentheses.

Table 6
Lead content of bile

Week	Control	Lead salt diets (as Pb)				Paint diets	
		Lead nitrate			Lead naphthenate		
		20ppm	50ppm	100ppm	100ppm	Chrome pigment	Naphthenate
4	0.016 (16) 0.012 (15)	0.03 (6) 0.022 (15)	0.033 (15) 0.026 (10)	0.146 (19) 0.274 (12.5)	0.103 (20) 0.119 (20)	0.082 (12.5) 0.053 (20)	0.140 (17) 0.130 (16)
12	0.038 (12.5) 0.049 (19)	0.100 (11) 0.100 (18)	0.095 (11) 0.096 (12.5)	0.159 (13) 0.126 (14)	0.114 (17.5) 0.116 (20)	0.071 (17.5)	0.117 (10) 0.135 (13.5)

Results are expressed as μg/ml. Each determination was made on a separate rat. Figures in parentheses indicate volume of bile collected (ml/rat/24hr).

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tion, but this is not so marked as it is in other tissues. Bile provides a sensitive index of lead absorption, though the correlation with dietary concentration is somewhat erratic (Table 6).

Lead naphthenate: The concentration of lead in the tissues, urine and bile of rats receiving the lead naphthenate diet is similar to that observed after the administration of a diet containing an equivalent amount of lead nitrate. The acid radical does not, therefore, affect the availability of the lead.

The absorption of lead from paint films

Chrome pigment: The concentration of lead increases in kidneys and bone during the first two weeks: in the former tissue it thereafter remains fairly constant throughout the experiment, and with the latter the concentration continues to rise until the eighth week. There is a slight increase in the lead content of liver, but the blood concentration is not significantly different from the control value. There is an appreciable excretion of lead in the bile but the amount in urine is negligible.

The amount of lead in bone, kidney and bile of rats receiving the chrome pigment paint diet is similar to that observed in the rats on the 50ppm lead nitrate diet.

Lead naphthenate paint: The uptake of lead by kidney, bone and liver is much greater than that observed with the chrome pigment paint, and it parallels the findings in rats on the 100ppm lead nitrate diet. This conclusion is supported by the bile results and, to a smaller extent, by the blood analysis. There is again no significant increase in the lead content of urine in spite of the considerable absorption of lead demonstrated by the analytical results on tissue and bile.

Discussion and Conclusions

The work of Chisholm & Harrison[2] has shown that the daily average lead excretion in the faeces of children showing signs of lead poisoning after ingestion of paint flakes, is 44mg. As most of the lead ingested appears in faeces, it may be assumed that a daily intake of in the region of 50mg lead is very dangerous, unless the lead is in a form not available for absorption. On the other hand, Kehoe[6] has shown that a daily intake of 1mg soluble lead salt in the diet over a period of years is without risk. The present investigation has demonstrated that about one-half of the lead in the lead naphthenate paint, and one-quarter in the chrome pigment paint, can migrate from the film into the gut content, and Table 7 shows the amounts of the two paints which must be ingested daily

Table 7

Hazard from paint ingestion

Paint	Threshold (1mg dose)		Danger (50mg dose)	
	Weight, g	Area, cm²	Weight, g	Area, cm²
Chrome pigment	0.2	20	10	1,000
Lead naphthenate	0.1	10	5	500

Figures give weight and surface area (average single coat) of paint containing 2 per cent lead on dried film which, if ingested daily, would reach threshold of safety or be very dangerous.

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to produce the threshold and the dangerous lead intakes. The observed migration of lead from paint film is much greater than that recorded in earlier experiments with polymers[4]; this may be due to the much finer state of subdivision attained with paint film. It is possible that the mechanical means used to disintegrate the film was much more efficient than the processes of mastication and digestion, and the migration figures achieved in these experiments may be in excess of those encountered when paint flakes are ingested.

The undertaking by the paint industry to limit the lead content of finishes for surfaces likely to be chewed by children to not more than 1 per cent is in agreement with recommendations of the British Standards Specification and the American Standards Association. The amounts of such paints which must be ingested to achieve the threshold and toxic amounts would be about double the figures in Table 7. With coatings containing not more than 0.5 per cent lead in conformity with the latest Toys (Safety) Regulations, the amounts would be about four-fold. It is certain that these figures indicate no hazard so long as the film is adherent for it would be impossible to ingest sufficient either by detaching the film manually or by gnawing it off the support. A greater lead intake would be possible if detached films were available for ingestion but, even so, with paints of this type, there seems little justification for the fears expressed by Chisholm and Harrison, that poor maintenance of modern housing with resultant accumulation of many layers of paints on surfaces accessible to children, may in the future constitute a hazard to small children.

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Next month's issue

The Honorary Editor has accepted the following papers for publication and these are expected to appear in the April issue:

"Exterior durability of coatings," *by J. E. Fulford.*

"Epoxy resin floor finishes in South Africa," *by L. Muller.*

"Acrylic resins for industrial chemical coatings," *by H. L. Wampner.*

"The surface treatment of pigments," *by J. L. Mollier and D. A. Plant.*

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