one then obtains the rates depicted in the Chart. The correlation (0·71) between the number of infants who die of cancer and the prevalence of influenza in the year preceding their birth can now be seen to be statistically highly significant. The correlation for cases of neoplasms of the lymphatic and haematopoietic tissue (I.C.D. 200-209) is higher still (0·85), and if cases of leukaemia alone (I.C.D. 204) are considered the correlation is also statistically significant ($r = 0·78$, D.F. $= 7$, $P < 0·01$). The correlation with other neoplasms, although positive, is not statistically significant.

Discussion

Our analyses suggest that viral infection in utero could be a factor of importance in the aetiology of leukaemia. It is not possible to state categorically that the infection concerned was due to an influenza virus, although the epidemiological evidence makes this highly likely.

It has been shown experimentally (Seim, Ly, Imagawa, and Adams, 1960) that one strain (the neurotropic strain) of the influenza A virus does not cross the placental barrier in mice, but that another strain of the same virus does so, but only in the third trimester. Clearly, the factors responsible for transplacental viral passage are largely unknown.

It is possible that the effect of viral infection in utero is related to the consequences of the maternal infection rather than to the direct effect of the virus itself. For example, it is known that certain acute infections such as influenza are accompanied by a profound systemic toxaemia and, often, a reduction in the arterial oxygen saturation (Hardy, Ararowicz, Mannini, Medearis, and Cooke, 1961). Alternatively, the effect could be due to that of drugs taken by the mother at the time.

It should be stressed, however, that although the relative risk of childhood leukaemia appears to be raised after such an infection during pregnancy, the absolute risk, even after such a history, is still very low, between 3 and 4 per 1,000 children under 12 years.

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Lead Poisoning in Rural Scotland

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Summary

Nine people from four families living in rural parts of Scotland have been found to suffer from clinical or biochemical effects of lead poisoning. Five had symptoms and four had unequivocal evidence of excessive lead exposure. The source of lead has been traced to the domestic water supply which in all cases was grossly contaminated with lead acquired from lead plumbing systems, including lead storage tanks. Clinical improvement followed the replacement of lead piping in two families studied. Lead poisoning is a possible cause of chronic ill health in areas of plumbosolvent water.

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Introduction

Lead poisoning is a well-recognized industrial hazard, particularly among ship breakers, scrap-metal workers, and battery manufacturers. Domestic lead poisoning is less common apart from the well-known hazard in children (Moncrieff *et al.*, 1964; Gibson *et al.*, 1967). It was shown by Crawford and Morris (1967) that domestic water is sometimes contaminated by lead, but there are few reports of clinical lead poisoning as a consequence. Three severe cases from Yorkshire were described by Bacon *et al.* (1967). This paper describes four households with lead intoxication acquired from contamination of the domestic water supply. All four families lived in rural parts of the Scottish Highlands.

Patients and Methods

The patients were referred by their general practitioners for investigation to the Royal Northern Infirmary, Inverness, the Victoria Infirmary, Glasgow, and the Western Infirmary, Glasgow.

Blood lead and water lead levels were estimated by the dithizone method. Erythrocyte protoporphyrin was measured by the method of Rimington (1961). Urinary delta-aminolaevulic acid (ALA) and porphobilinogen were measured by the method of Mauzerall and Granick (1956).

TABLE I—*Clinical Features and Laboratory Findings in Affected Members of the Four Families*

		Clinical Features	Hb (g/100ml)	Basophilic Stippling	Serum Urea (mg/100 ml)	Serum Uric Acid (mg/100 ml)	Urinary Uric Acid (mg/24 hr)	Blood Lead (µg/100 g)	Eryth. Proto. (µg/100 ml)	Urinary ALA (mg/l.)	Urinary Copro. (µg/l.)
Family No. 1	Case 1	Abdominal pain, joint pain	10·0	Neg.	100	11·6	210	59·0	505	3·6	1,348 µg/day
	Case 2	Joint pain	15·2	Pos. in Marrow	64	8·5	140	75·1	401	7·0	158
	Case 3	Symptom-free	14·8	Neg.	40	7·8	370	46·0	91	3·2	136
Family No. 2	Case 4	Abdominal pain, torticollis, tremor	13·2	Neg.	22	7·0		47·1	354	11·0	342
	Case 5	Symptom-free	16·0	Neg.	34	5·4		39·5	135	4·0	250
	Case 6	Peripheral neuropathy	15·6	Neg.	29	6·7		23·9	56	2·6	35
	Case 7	Recurrent abdominal pain	12·3	Neg.	36	4·1		7·8	59	2·4	17
	Case 8	Symptom-free	16·2	Neg.	43	4·8		30·5	184	—	—
	Case 9	Symptom-free	13·8	Neg.	36	4·3		7·9	73	—	—
Family No. 3	Case 10	Gout, anaemia	11·6	Neg.	100	9·2		71	713	8·5	29
	Case 11	Tremor, nervousness, anaemia	12·2	Pos. in Blood	50	7·5		163	681	41	1,286
Family No. 4	Case 12	Symptom-free	—	—	—	—		69·3	508	9·8	354
	Case 13	Symptom-free	—	—	—	—		46·4	—	4·0	107
	Case 14	Symptom-free	—	—	—	—		67·5	—	17·0	1,220
Normal values			13-16	Neg.	<40	<6	<1,000	<40	<35	<5·5	<250

Eryth. Proto. = Erythrocyte protoporphyrin. Copro = Coproporphyrin.

Results

The clinical features and laboratory findings in the four families are summarized in Table I.

FAMILY 1

Three brothers (Cases 1–3) living on a farm in Sutherlandshire were investigated because of abdominal and joint symptoms. The first to present was a 23-year-old coalman who attended the Royal Northern Infirmary, Inverness, in 1968 with pain and stiffness in both shoulders. One month later he was admitted as a surgical emergency because of abdominal colic and vomiting. He was treated with alkalis and phenobarbitone 30 mg thrice daily, but this was followed by a deterioration in his condition during the next few days. He was noted to be anaemic (Hb 10 g/100 ml) and to have high serum urea and uric acid levels and a normal excretion of urinary uric acid (Table I). The urine contained coproporphyrin in excess. His condition improved rapidly after stopping all therapy and he was discharged after 21 days. The diagnosis of lead poisoning was suspected because of porphyrinuria with renal dysfunction and gout and was confirmed by the finding of a raised blood lead (59 µg/100 g) level.

His 36-year-old brother was a farmer in Sutherlandshire. His only complaints were recurrent attacks of pain in his left shoulder and slight chronic heartburn. He too had evidence of impaired renal function, hyperuricaemia, and lead poisoning. The third brother, aged 25, was also a coalman. He was symptom-free but had high blood lead level (46 µg/100 g) and an increased level of erythrocyte protoporphyrin in the absence of anaemia or sideropenia.

FAMILY 2

The second family (Cases 4–9) lived in Argyllshire and consisted of a husband, his wife, their six children, and a domestic staff of two. The wife (Case 4), aged 51 years, gave a history of spasmodic torticollis and lower abdominal pain of three years' duration which had not been explained by a barium-meal or barium-enema examination; gynaecological examination was also negative. In June 1969 she presented at a dermatology clinic with an acute photosensitive eruption. At this time the erythrocyte protoporphyrin was 350 µg/100 ml cells. In February 1970 she was seen again because of further abdominal pain, and the diagnosis of lead poisoning was made on the basis of raised blood lead and erythrocyte protoporphyrin levels and high urinary coproporphyrin and ALA. On examination she was found to have lower abdominal tenderness and a fine finger tremor. The pulse rate was normal and the blood pressure was 140/90 mm Hg.

Her 52-year-old husband (Case 5) was in apparently normal health and symptom-free, but his blood lead and urinary coproporphyrin were at the upper limit of normal and the erythrocyte protoporphyrin was clearly raised. The six children were clinically normal when seen. One son, aged 9 (Case 6), had been investigated because of an unexplained peripheral neuropathy four years previously, and when examined he had a fine finger tremor. Two children, aged 22 and 13, gave histories of recurrent abdominal colic in earlier childhood. All the children had been educated at boarding schools.

The cook and maid of the household (Cases 8 and 9), aged 56 and 19 respectively, were symptom-free.

FAMILY 3

The third family (Cases 10 and 11) lived in a different part of Argyllshire. The husband, a 57-year-old retired army officer (Case 10), gave a 15-year history of gout controlled by phenylbutazone. In addition he complained of effort dyspnoea and a chronic cough. On examination the pulse rate was normal and the blood pressure was 170/90 mm Hg. There was pallor of his mucous membranes consistent with the mild degree of anaemia noted (Hb 11·6 g/100 ml). There was biochemical evidence of impaired renal function and hyperuricaemia. A diagnosis of lead poisoning was established by the finding of a raised blood lead level (71 µg/100 g, uncorrected for packed cell volume), markedly raised erythrocyte protoporphyrin (713 µg/100 ml cells), and high urinary ALA.

His 64-year-old wife (Case 11) was more severely affected. Her presenting symptoms were pronounced tiredness and nervousness. The principal finding on examination was pallor of her mucous membranes but she was only mildly anaemic (Hb 12·2 g/100 ml). The reticulocyte count was 8% and punctate basophilic stippling was noted in the peripheral blood. The blood lead was markedly raised (163 µg/100 g) and there was a gross rise in the urinary excretion of ALA and coproporphyrin. The blood urea was raised (50 mg/100 ml) and hyperuricaemia was also noted.

FAMILY 4

This family (Cases 12–14) consisted of a woman of 81 years, her son of 45 years, and her daughter of 51 years. They lived in Sutherland, a few hundred yards from Family 1. All three were symptom-free but were tested because of the similarity of their water supply to that of the first family. Blood leads were raised in each case and the urinary excretion af ALA and coproporphyrin was high in Cases 12 and 14.

FURTHER STUDIES

A feature common to each family studied was that they lived in old houses with lead storage tanks and pipes carrying the domestic water supply. Analysis of the lead content of the domestic water is shown in Table II. The lead content of tap-water was markedly raised in each case, although the source water was normal.

The source water in each case was separate from the ordinary supply pipes of the local authority. The two Sutherland families lived in farm houses situated close to each other and used a common supply from a nearby stream. The two Argyllshire

TABLE II—*Lead Content of Domestic Water*

	Source Water (μg/l.)	Tap-water (μg/l.)
Family 1 (Sutherland)	32	570
Family 2 (Argyllshire)	11·4	3,136
Family 3 (Argyllshire)	10	1,440
Family 4 (Sutherland)	32	1,320
W.H.O. Limit	100	

families lived in large country houses with independent spring-water supplies.

All lead plumbing was removed in April 1970 from the house occupied by Family 1. Further tests on two members of this family were carried out one year afterwards, and the changes in blood lead and erythrocyte protoporphyrin are shown in Table III. Similar repeat tests on members of Family 2 three months after replacement of lead piping are reported in Table IV. The principal finding in each case was a fall in the level of erythrocyte protoporphyrin.

TABLE III—*Changes in Blood Lead and Erythrocyte Protoporphyrin in Family No. 1 One Year after Removal of Lead Pipes*

	Blood Lead (μg/100 g)		Erythrocyte Protoporphyrin (μg/100 ml cells)	
	Before	After	Before	After
Case 2	75·1	26·9	401	241
Case 3	46·0	41·9	91	80

TABLE IV—*Changes in Blood Lead and Erythrocyte Protoporphyrin in Family No. 2 Six Weeks after Removal of Lead Pipes*

	Blood Lead (μg/100 g)		Erythrocyte Protoporphyrin (μg/100 ml cells)	
	Before	After	Before	After
Case 4	47·1	46·8	354	91
Case 5	39·5	30·2	135	55
Case 6	23·9	9·8	56	16·8
Case 7	7·6	8·7	59	26·3

Discussion

Contamination of drinking water with lead has been recognized as a source of lead poisoning for centuries; Roman and Greek physicians are thought to have been acquainted with the problem (Major, 1965). In modern times, however, there have been few reports of clinical cases of lead poisoning from domestic water.

Three severe cases from the Yorkshire moors were described by Bacon *et al.* (1967). These authors stressed the importance of acid moorland waters in dissolving lead from domestic piping. It was suggested by Wilson (1966) that the incidence of stillbirth or abortion in a Scottish border county might have been influenced by soft domestic water containing excessive amounts of lead.

Out of 20 subjects tested in the four families nine were found to have clinical or biochemical evidence of lead intoxication. Five of these (Cases 1, 2, 4, 10, and 11) had symptoms of lead poisoning and the four others had unequivocal biochemical evidence of excessive lead exposure (Cases 3, 12, 13, and 14). Four of the patients had severe clinical symptoms in the form of acute abdominal pain, anaemia, arthropathy, or tremor. In five patients the blood urea was over 40 mg/100 ml. Three patients had haematological abnormalities manifested by anaemia, reticulocytosis, and basophilic stippling. It is of interest that only two of these had basophilic stippling and that this was found only in the bone marrow in one case. Basophilic stippling is now regarded as an unreliable index of lead absorption, but it is more common in the bone marrow than in the peripheral blood (Waldron, 1966).

Out of 11 patients tested seven had hyperuricaemia, including three with joint pain. This finding is likely to be related to impaired renal excretion of uric acid, since three patients tested had low normal daily urinary excretion of uric acid. The clinical syndrome of gout associated with lead poisoning (saturnine gout) has been long recognized (Bauer and Krane, 1964).

A diagnosis of lead poisoning should be based on clinical findings and supported by biochemical evidence of excessive lead absorption (Lane *et al.*, 1968). Nine subjects had raised blood lead levels between 46 μg and 163 μg/100 g. Although a blood lead level of 80 μg/100 g has been suggested as the "limit of acceptance" for industrial lead poisoning, it is clear that the blood lead level alone is not an accurate criterion of clinical lead poisoning. Levels below this are frequently associated with symptoms and signs, and levels above this are occasionally found in asymptomatic lead workers (Gibson *et al.*, 1968). The cause of this variability in blood lead is probably related to the dynamic state of blood lead, which reflects patterns of lead absorption from the gut and mobilization from bone. Further work is required to elucidate these factors. Only by an integration of the clinical evidence with the biochemical criteria (blood lead and erythrocyte protoporphyrin, urinary ALA, and coproporphyrin) can an accurate judgement be made.

In the present study the subjects were exposed to a domestic lead source and not an industrial one. The duration of exposure was of many years in all cases, and it is of interest that clinical evidence of lead toxicity was associated with blood lead levels of between 40 μg and 80 μg/100 g. A diagnosis of such toxicity was strengthened by the raised levels of urinary ALA and coproporphyrin and of erythrocyte protoporphyrin. Other causes of high erythrocyte protoporphyrin levels in these patients, such as iron-deficiency states or protoporphyria, were excluded. Increased erythrocyte protoporphyrin is a sensitive indicator of lead exposure and possibly more specific than the increased urinary excretion of coproporphyrin (de Bruin, 1971).

It appears from available records that all water supplied by local authorities in Britain has an acceptable lead content at source. The degree of plumbosolvency, however, seems to vary with factors such as hardness and content of certain organic acids. In a study of overnight water in inhabited houses of 17 county boroughs of England and Wales, Crawford and Morris (1967) found that 31 out of 95 samples contained more than 100 μg of lead per litre. The highest concentrations were found in soft waters, although some hard waters also contained excessive amounts. Acidity also appears to influence plumbosolvency although the relation is not a consistent one. Tests of plumbosolvency carried out by local authorities show that acid moorland waters appear to have the most aggressive action on lead piping (Bacon *et al.*, 1967). In the present study the houses were supplied with water containing an acceptable amount of lead, but in each case the tap-water was grossly contaminated. The lead content thus ranged from 5 times to 31 times the upper acceptable limit of normal suggested by the World Health Organization (1971) (Table II).

The legal position regarding the lead content of domestic water is rather loosely worded. The Water Act of 1945 demands that the authorities shall supply "wholesome" water. There is no reason to suspect that the source water entering the houses described was other than "wholesome," and it is clear that contamination occurred in lead piping and tanks within the houses. There is, however, no legislation requiring the removal of lead pipes from the older type of house which still commonly has them. It seems likely that lead poisoning may therefore be a commoner cause of insidious and chronic ill health than is at present suspected, particularly in soft-water areas.

This study in rural Scotland provoked a similar investigation in Glasgow, which also has soft water and many houses with lead storage water tanks. The results (Beattie *et al.*, 1972) showed that the tap-water in houses with lead tanks contained a high lead content and that some of the inhabitants of such households had biochemical evidence of excessive lead exposure. In no instance was there evidence of symptoms of lead poisoning.

The level of lead in the water of the rural population was in some instances of the same range as the urban group with lead-lined tanks. The cause of the presence of symptoms in the rural subjects and the absence of symptoms in the urban subjects requires consideration. The investigation of the rural subjects was prompted initially by their presentation with symptoms, while the urban study was entirely epidemiological. The durations of exposure in the five patients of the rural group who had symptoms ranged from 15 to 50 years, higher with one exception than in the urban group with lead tanks. This may be another factor to explain the difference in the incidence of symptoms.

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Environmental Lead Pollution in an Urban Soft-water Area

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Summary

An investigation has been reported on the clinical and metabolic effects of lead acquired by soft domestic water from lead plumbing systems in 23 Glasgow households. The lead content of water from cold taps was up to 18 times the upper acceptable limit and was proportional to the amount of lead in the plumbing system. The blood lead of 71 inhabitants of these houses showed a significant positive correlation with water lead content. Delta-aminolaevulic acid dehydrase activity, an extremely sensitive indicator of lead exposure, showed a significant negative correlation with water-lead content. Atmospheric lead was within acceptable limits in all but one house and no significant correlation could be found with biochemical measurements. A small number of clinical abnormalities were found but could not be directly attributed to lead toxicity. The results of the study underline the possible danger to health of lead plumbing systems in soft-water regions.

Introduction

A number of cases of lead poisoning from ingestion of domestic water have been described in recent years (Bacon *et al.*, 1967; Beattie *et al.*, 1972). In these cases lead was acquired by soft moorland water from lead tanks and lead pipes. There is evidence that soft water has a greater avidity for lead than hard water, which may form a shield of calcium salts over the lead surface of the pipes (Crawford and Morris, 1967). While the clinical effect of lead in severe cases can be shown little is known about the subclinical consequences of drinking lead-contaminated water over a long period of time. The present study of the clinical and metabolic effects of lead ingested in drinking water was made in Glasgow, which is one of the largest soft-water areas of the United Kingdom.

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Materials and Methods

The subjects studied were 71 volunteers from 23 families in whom the householder was an employee of either the Lower Clyde Water Board or the Health and Welfare Department of the Corporation of Glasgow. There was, in fact, a total of 73 members of these families but one was a 6-month old baby and another an adult female who was unwilling to take part in the study. Employees whose occupation involved contact with lead were excluded. A sample of tap-water taken first thing in the morning without previous running of the tap was obtained in lead-free plastic containers from each household. Water lead was measured in this sample by the dithizone method (Gonzales *et al.*, 1954). A sample of water was also obtained from the reservoir serving the area of the study.

A questionnaire was completed for each household asking the type of house, its age, and the duration of occupation by the family. An accurate assessment of the plumbing system was made with respect to the presence or absence of a tank for storage of cold water, the type of tank, and the absolute length of each type of pipe carrying the cold water from the mains supply to the kitchen tap. The questionnaire also asked about symptoms and signs which are known to occur in lead poisoning. The items are shown in the Fig. An effort was made to establish which member of the household was first to drink tap-water each morning.

Samples of blood and urine were obtained from each subject. Blood lead was measured by the dithizone method. Delta-aminolaevulic acid dehydrase (ALA dehydrase) was measured in erythrocytes by the method of Moore *et al.* (1971). Serum urea and uric acid were measured by standard techniques. A blood