

MERCURY- CONTAMINATED GRAIN IN IRAQ

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Contents

1. Introduction
 2. Epidemic of Mercury Poisoning in Iraq
 - 2.1. Outbreak of Methylmercury Poisoning by Agricultural Chemicals
 - 2.2. Poisoning-Causing Agents and Exposed Population
 3. Clinical Features of the Iraq Methylmercury Poisoning by Fungicide
 4. Prenatal Exposure in Iraq
 5. Treatment and Prognosis
 - 5.1. Mercury Eliminators in the Early Stage
 - 5.2. Treatments by Haemodialysis and Exchange Transfusion
 - 5.3. Prognosis
 6. Dose-Response Relations in Iraq Methylmercury Poisoning
 - 6.1. Onset of Symptoms and Blood Levels of Methylmercury
 - 6.2. Estimated Threshold Body Burden
 7. Solution to the Mercury Pollution by Mercurial Pesticides
 8. Conclusion
 9. A short guide for further study
- Acknowledgements
Glossary
Bibliography

1. Introduction

A variety of inorganic and organic mercury compounds have been used to treat seed potatoes, flower bulbs and especially grain seed (wheat, rice, barley, oats, rye, beans, cotton, peanuts etc.) to prevent fungal diseases during germination. The use of mercury in agriculture probably reached its height in the mid-1960s, and has generally declined because of the disquiet over the deaths amongst seed-eating and predatory birds with the consumption of mercurial seed treatments.

In 1956, an outbreak of human poisoning by mercury occurred in northern Iraq, which resulted in over 100 hospitalized patients and at least a dozen fatalities. In another outbreak in 1960 involving a thousand patients and many deaths, it was found that the affected people had been eating wheat dressed with an ethylmercury fungicide.

The most disastrous epidemic of mercury poisoning occurred in 1972: it also took place in Iraq, and involved the ingestion of wheat treated with methylmercury fungicide. The number of sufferers has been put at several thousand, and detailed investigations of this outbreak are being undertaken by the World Health Organization (WHO) at the time of

this writing (2002).

2. Epidemic of Mercury Poisoning in Iraq

2.1. Outbreak of Methylmercury Poisoning by Agricultural Chemicals

The significant outbreak in Iraq took place in the winter of 1971-1972, and the incidence of patients increased sharply early in January 1972. Seed grain treated with a methylmercury fungicide was used to prepare homemade bread in rural communities throughout the country. The primary cause of the poisoning was traced to the ingestion of seed wheat that was used for making flour. Victims appeared only in provinces where the inhabitants ate homemade bread, and not a single case of poisoning was reported in cities. The bread was supplied from government-inspected flour mills.

Ingestion probably began in October-November 1971, and the first patients with severe poisoning were admitted to the hospital at the end of December 1971. The incidence developed into the most catastrophic epidemic ever recorded, with 6,530 people hospitalized by March 27 of the following year, of whom 459 died in the hospital (Figure 1).

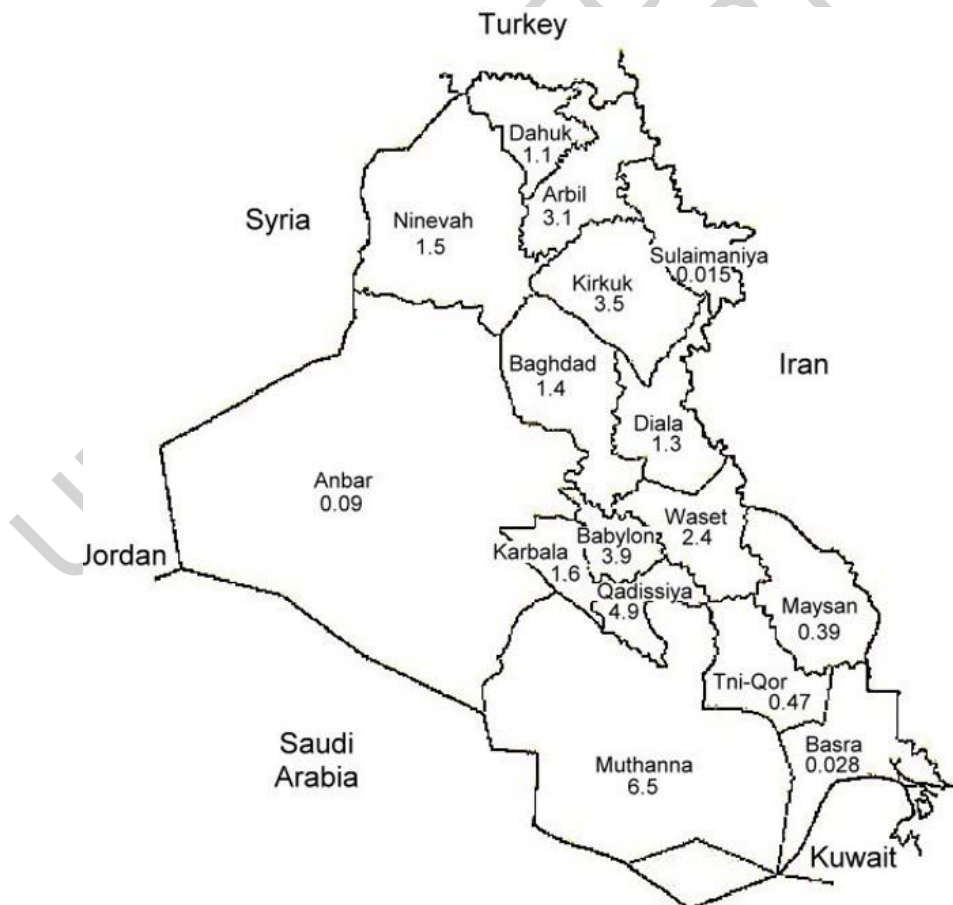


Figure 1. Incidence of hospital admission per 1,000 population according to province during the epidemic of methylmercury poisoning in 1972 in Iraq.

2.2. Poisoning-Causing Agents and Exposed Population

An area previously of great concern was the use of mercury compounds as seed treatments in agriculture. The amount of mercury in the seed treated formulation was small, but the effect was large, because it was applied to a large volume of seed that had been subsequently sowed over millions of acres.

A total of 73 201 tons of seed wheat was unloaded at the Basra port, Iraq, between September 16 and October 15 1971. In addition, 22 262 tons of barley treated with various mercury preparations were imported between October 22 and November 24, and delivered immediately to farmhouses across the country. A part of the wheat (no more than 5,000 tons) was reported to have been treated with phenylmercury and ethylmercury-p-toluenesulfonamide. However, these mercury compounds were not detected in the wheat or blood samples of the victims. Barley was reported to have been treated with phenylmercuric acetate. At least 13 000 tons of barley were treated with methylmercury dicyandiamide. Of 13 barley samples examined, 10 showed methylmercury and 3 phenylmercury.

Soon after the outbreak it was clear that the main source of poisoning was the bread made from the treated wheat. Thus, a predominant vehicle of exposure was investigated by analysis of samples of blood and the results of questionnaire surveys about the consumption of contaminated bread. The first cases of poisoning appeared around the end of December 1971. During the month of January 1972, there was a rapid and overwhelming admission to hospitals throughout the country. The high mortality of 459 cases out of 6,530 reported hospital cases reflected the severity of affection and exposure (Table 1). The morbidity varied greatly from one area to another, ranging from 7.6 per 1,000 of the population; the highest was in Muthana Province, to 0.015, the lowest, was in Sulaimaniya Province. Of the victims admitted to hospitals, women accounted for 52% and men for 48%. As for age groups, the 1-9 year groups accounted for 34% of the total. An incidence map of hospital admission according to province is given in Figure 1.

The exposure ranged from a low non-toxic intake to prolonged daily intake (1-2 months), which in some cases produced severe signs of poisoning. Ethylmercury was detected in the blood and hair samples of the victims and also in tissues of the deceased. Most of the mercury in blood was found in the red blood cells. The level of mercury in plasma and milk correlated closely with that in whole blood (Table 2). Mercury in whole blood was mostly organomercury, but the ratio of inorganic mercury was found in urine.

The total mercury and methylmercury levels were measured by cutting long hair at 1 cm intervals and examining each piece of cut hair in two victims. This method, used in the Niigata Minamata Disease incident, clearly indicates the changes with time in exposure to methylmercury. The total mercury level in hair samples from two patients in Iraq is mentioned as having been approximately $550 \mu\text{g g}^{-1}$ and $750 \mu\text{g g}^{-1}$, respectively (Figure 2). Although the biological half-life in hair closely follows those in blood, hair half-life tends to have a wider range. A bimodal distribution in 48 Iraq subjects had a half-life of 35-100 days, and the 10% a half-life of 10-120 days.

Age (years)	Number of cases admitted											
	Males			Nonpregnant females			Pregnant females			Total affected		
	Cases	Fatalities	Case ^a fatality	Cases	Fatalities	Case ^a fatality	Cases	Fatalities	Case ^a fatality	Cases	Fatalities	Case ^a fatality
<1	8	0	0.0	2	0	0.0	0	0	0.0	10	0	0.0
1-4	383	33	8.6	378	36	9.5	0	0	0.0	761	69	9.1
5-9	736	63	8.6	731	58	7.9	0	0	0.0	1470	121	8.2
10-19	606	45	7.4	650	55	8.5	0	0	0.0	1256	100	8.0
20-29	380	18	4.7	520	43	8.3	17	12	70.5	917	73	8.0
30-39	416	15	3.6	483	25	5.2	12	2	16.7	913	42	4.6
40-49	240	7	2.9	268	17	6.3	2	0	0.0	510	24	4.7
50-59	164	13	7.9	146	12	8.2	0	0	0.0	310	25	8.1
60+	211	3	1.4	172	2	1.2	0	0	0.0	383	5	1.3
Total	3144	197		3353	248		31	14		6530	459	
Average Mortality (%)			6.3			7.4			45.0			7.0

Notes:

a) (Fatalities/ cases) × 100

b) From: Bakir F. *et al.* (1973) Methylmercury poisoning in Iraq. *Science* **181**, 234.

Table 1. Distribution of cases of poisoning admitted to hospitals, and of deaths caused by poisoning, according to age and sex.

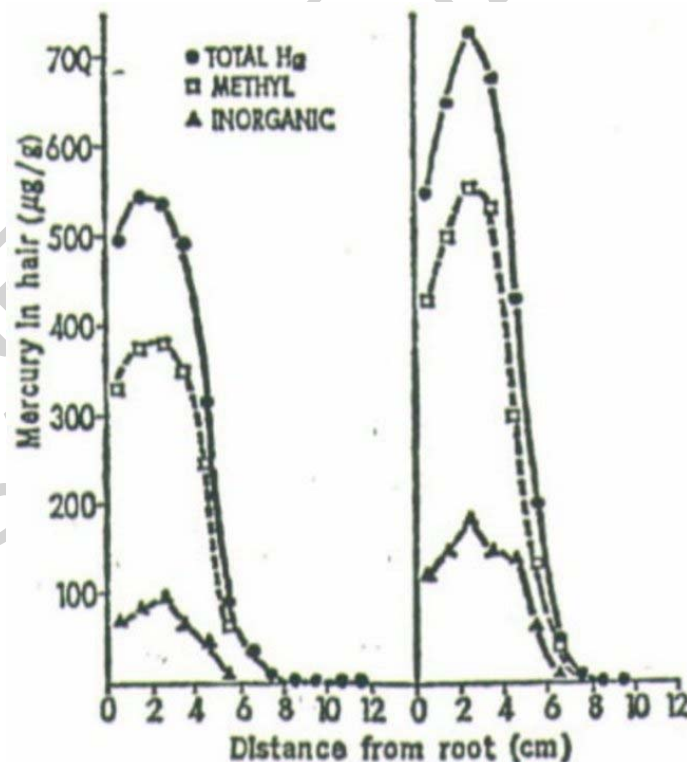


Figure 2. Concentrations of mercury (methyl, inorganic and total mercury) in hair samples from two patients in Iraq.

Biological fluid	N	Total Hg (% of whole blood)	Correlation with blood ^a	Inorganic Hg (% of total)
Blood	224	100	1.0	7
Plasma	14	18	0.8	22
Milk	44	5	0.9	39
Urine	21	6	0.1	73
Cerebrospinal	5	6		
Amniotic	1	2		

Notes:

a) Correlation coefficient with that of whole blood.

b) From: Bakir F. *et al.* (1973) Methylmercury poisoning in Iraq. *Science* **181**, 235.

Table 2. Total amounts of mercury in samples of biological fluids compared with heparinized whole blood obtained from patients with methylmercury poisoning.

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