Environmental Accidents: The Assessment of the Significance of an Environmental Accident Due to the Distribution of Lead-Contaminated Feedstuffs to Several Hundred Cattle Farms

WM Allen
Compton Paddock Laboratories, PO Box 91, Newbury, Berkshire RG20 7HB, UK

Introduction

In the late summer of 1989 several shipments of pelleted rice bran arrived at a small port in southwest England. The bran had originated from Burma and had been transported via a circuitous route through Europe and had then been relabelled as either 'Maize gluten replacer pellets' or 'High protein pellets'. During its sea passage, the bran had become contaminated with zinc ore concentrate material which contained 16.5% by weight of zinc, 34% by weight of lead, 0.75% of arsenic and a variety of other trace elements. The rice bran pellets were probably first fed to cattle in The Netherlands where lead toxicity was quickly recognised and the outbreak was described (1).

Within a few weeks there was a similar outbreak of lead poisoning in the UK, heralded by the death of six young calves. Once it was realised how widely the contaminated feed had been distributed, restrictions on the movement of animals, and on the sale of milk and animals for meat were introduced and remained in operation for several months after the feed had been removed from the food chain. However, this catastrophe was never the subject of either a public enquiry or any comprehensive publication. Two publications (2 and 3) have described parts of the incident. Lead toxicity was confirmed in only seven calves.

This paper briefly describes the background to the incident and the basis upon which the decisions to restrict the movements and sales of products were made. The insurance claims against the feed compounders alone cost more than £6 million (approx. $9 million U.S.); and in addition there were the costs of the executive operations and the costs due to the disruption to the agricultural industry. The milk processors claimed that they incurred more than £2.5 million (approx. $3.75 million U.S.) in extra charges for processing the contaminated milk. Table 1 lists the alleged bases of the claims for compensation from the feed compounders.

Table 1. Bases of claim against feed compounders valued at >£6 million

Deaths of 31 cattle
Retardation of growth
Abortions, infertility
Increased susceptibility to disease
Loss of milk yield and value
Costs of extra feed for imported cattle
Sundry costs

The History of the Event

The vessel 'Sagaing' loaded 4300 tonnes of rice bran in Rangoon in July 1989, as part of a purchase of 30,000 tonnes. It was loaded into three holds of the ship, but when it arrived in Rotterdam it was found that at least 150 tonnes from one of the holds had become admixed with the zinc ore in the adjacent hold. The contamination was identified immediately and the resulting mixture was certified as being fit only for destruction. There was a dispute as to its method of disposal but a trader in distressed goods purchased the condemned material which was then off-loaded into barges. The material was then transported into The Netherlands where 'opportunist' traders pelleted the contaminated product and described it as Maize Gluten Replacer Pellet (MGRP). Although the gross analysis of the rice bran pellet was not dissimilar to maize gluten, the metabolisable energy (ME) in the two products differed considerably (Table 2), the MGRP providing only 60% of the ME provided by maize gluten. The high content of lead and other contaminants in a sample of the rice bran when incorporated into final rations is also shown in Table 3.

Adapted from the Proceedings of the XX World Association for Buiatrics Congress, Sydney, Australia, July, 1998.

Table 2. Composition of maize gluten and rice bran (described as maize gluten replacer pellet)

	Maize gluten	Rice bran
Metabolisable energy (MJ/kg)	12	7.1
Crude protein (%)	21	13.5
Starch (%)	15.5	30.2
Sugars (%)	3	2.0
Crude fibre (%)	10	13.0
Ash (%)	5	5.3x
Pb (%)	<0.005	10
Zn (%)		5

Zinc ore contains 16.5% Zn,
35% Pb and 0.75% As.
[x]The ash % in rice bran is normally >10%

Table 3. Analyses of 2 samples of compound feeds incorporating MGRP (mg/kg dry matter or ppm)

	Sample A	Sample B	Statutory maximum
Lead	2196	34	10
Zinc	900	150	250
Cadmium	6.5	0.2	0.5
Copper	6	4	50
As	38	4	4

Data from MAFF Nov 1989(4)
*Maximum permitted under Feedingstuffs Regulations 1988.

The opportunist traders sold the MGRP onwards and there was an outbreak of lead poisoning in The Netherlands, beginning in October. That outbreak affected 330 farms and 15,500 animals (1). It was estimated that over 1000 tonnes of animal feed had been contaminated by approximately 1000 kg of lead. During the first week of November all the contaminated food was removed from the Dutch food chain.

Other batches of MGRP were purchased by a trader in the UK on or soon after 5th October 1989. These were sold to at least twelve feed compounders, mainly in the south and west of England. On the 25th of October, six calves died and lead toxicity was diagnosed (2). The source of the toxicity was quickly identified as the contaminated MGRP. It was also apparent that the MGRP had been widely distributed and had been incorporated into rations for calves, beef and dairy cattle. The extent of the contamination was established by the analysis of two samples of the contaminated feeds (Table 3) on the 7th and 8th November and by the measurement of the concentrations of lead in blood, milk and body tissues. The government imposed bans virtually immediately on the sale of milk for liquid consumption from cows which had eaten contaminated feed, and on the sale of beef animals. Routine monitoring of the lead concentrations in milk and body tissues was introduced and a series of ad hoc experiments was begun to investigate how quickly the concentration of lead in milk and body tissues returned to normal.

Despite poor record keeping, once the destinations of the contaminated food began to be traced, the magnitude of the enquiry mushroomed; more than 1800 farms and more than 50,000 dairy cows, were involved. The concentration of lead in the kidneys of calves which had received contaminated MGRP in their diet is shown in Fig.1.

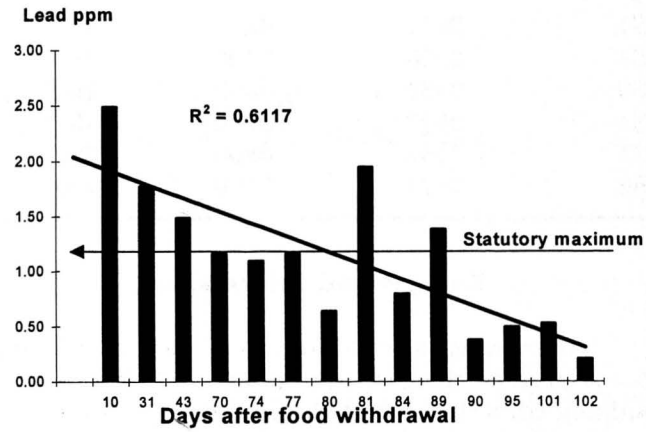

Figure 1. Lead concentrations in kidneys of calves after receiving contaminated feed (4).

By 1st December 1989, the mean value had decreased to below 2.0 ppm and by 1st January it was below 1 ppm. Over the same period the concentrations of lead in the liver were similar to those in the kidney.

Muscle tissue was examined only after the suspect food had been withdrawn from the diet for at least ten days; its lead concentration never exceeded 1.0 ppm. In contrast the concentration of lead in bone reached 16.0 ppm.

At least 800 of the 1800 farms which received supplies of contaminated cake were milk producers. The analysis of lead in milk samples suffered logistical problems and there were inadequate laboratory facilities. Table 4 shows some of the comparative analytical data for blood and kidney tissue and illustrates the wide variation between the results of analyses from different laboratories.

The concentration of lead in milk from many farms exceeded 50 µg/litre in early November but by the 10th November only eight of 28 milk samples contained between 50 and 164 µg/litre. Between the 6th and 12th of December, the maximum concentration in 63 samples of milk was 67 µg/litre.

Table 4. Observed analyses of cattle blood and kidney from three laboratories (4)

Sample	Blood PB µg/litre		
	Lab.1	Lab.2	Lab.3
S1	674	770	360
S2	552	470	360
S3	440	380	270
S4	555	490	390
S5	510	450	320
S6	646	690	340
Sample	Kidney Pb mg/kg		
	Lab.1	Lab.2	Lab.3
S1	2857	4400	4700
S2	2500	3500	4600
S3	2052	3400	4500
S4	2692	2900	4300
S5	2952	4800	4900
S6	1877	3100	4100

Background Information

After being ingested lead tends to accumulate in the excretory tissues, the kidney and liver, and in milking cows it is excreted into the milk. In the UK food legislation stipulates that the maximum permitted concentration of lead in kidney is 1.0 mg/kg and in the liver 2 mg/kg. It is uncertain why the values for the two tissues are so different particularly as it would be expected that people would eat more liver than kidney and that liver would therefore represent the greater risk of ingesting potentially hazardous amounts of lead.

There is no similar specified maximum permitted concentration of lead in milk. During this incident 50 µgPb/kg milk was chosen as an arbitrary cut-off point, presumably because it is the maximum permitted level in drinking water. Muscle does not retain high concentrations of lead because it is not a storage site. No maximum concentration for lead in muscle is specified in legislation (although 1.0 ppm could be conjectured as a probable maximum). By contrast bone is a storage organ and bone lead levels can increase substantially as a result of the long-term ingestion of lead.

Discussion

The initial decision to impose bans on the sale of milk, meat and offal was inevitable once it was realised how widely the lead-contaminated feedstuff had been distributed. However, once the restrictions had been imposed it was difficult to remove them and the final decision was delayed for two months. In part this may have been due to the inability to establish exactly where the contaminated material had been distributed, owing to the poor standard of record keeping and the wide variety of feeds into which the MGRP had been incorporated.

The basic knowledge of the metabolism of lead was, however, well documented and should have formed an important element in the analysis of risk. For example, lead is known to be accumulated in the excretory tissues, liver and kidney, and stored in bone. Muscle tissue accumulates little lead (5 & 6). Thus the risk to man could have been minimised effectively by a restriction on offals and bone alone. Meat could have continued to enter the food chain with minimum risk. Such a procedure could have been instituted immediately after the contaminated feed had been removed from the diet of the animals.

With milk too, pre-existing evidence was ignored. Although the majority of 'normal' milk samples contain less than 20 µg Pb/litre there is clear evidence that in areas of industrial activity, and especially in the old lead mining areas of the UK, milk may contain between 50 and 100 µg/litre. Thus, a ban on the sale of milk containing more than 50 µg/litre was probably unjustified. Once the dairy cattle ceased to receive contaminated feed, the concentration of lead in milk would have been expected to decrease rapidly from their high initial values, often more than 150 µg/litre. The restriction of the sale of milk for liquid consumption, often for several weeks after the withdrawal of the contaminated feed, could not therefore be justified.

Conclusion

The responses to this entire incident thus epitomise the problems faced by authorities responsible for public health in an era which has been described by Professor Duguid of Dundee as prone to health scares, some with little or no justification, and prone to media publicity scares which create alarm and pressure and provoke precipitate government action which may be directed, excessive or harmful.

The incident was triggered by the misrepresentation of a grossly contaminated feedstuff. Once the toxicity had been recognised, and the risks had been carefully evaluated (7), the health of animals could have been adequately protected by the immediate withdrawal of the contaminated feedstuff from the animal feed chain. The risks to man could have been minimised satisfactorily by a temporary ban on the sale of offals and milk for liquid consumption, rather than by a total ban on the sale of milk and beef animals for up to two months.

References

1. Baars AJ et al (1992) Lead intoxication in cattle: a case report *Food Addditives and Contaminants* 9:357-364.
2. Lund LJ and Brown JRH (1989) Lead Poisoning from contaminated feed *Vet Rec* 125;536.
3. Report of the Chief Veterinary Officer *Annual Report 1989 Lead Contamination Animal Health* ISBN 0.11.242893.2 HMSO.
4. MAFF News Releases November 1989 - February 1990 *MAFF London* .
5. Sharma RP, Street JC, Shupe JL and Bourcier DR (1982) Accumulation and Depletion of Cadmium and Lead in tissues and milk of lactating cows fed small amounts of these metals *J Dairy Sci* 65;972.
6. In Mineral Tolerance of Domestic Animals 1990 Lead *Nat Acad of Sciences Washington DC.*
7. Hathaway SC (1993) Risk Assessment procedures used by the Codex Alimentarius Commission and its subsidiary and advisory bodies *Food Control* 4;189-201.

CVM UPDATE

FDA, Center for Veterinary Medicine

"Helping man and animals by ensuring the availability of safe and effective animal health products."

November 13, 1998

Consent Decree Entered in Animal Drug GMP Case

On October 20, 1998, the U.S. District Court for the Central District of California incorporated into an order the entry of a Consent Decree for Permanent Injunction between the United States, Anthony Products Company, doing business as Anpro Pharmaceuticals, and its president, James Viscio. Anthony Products is an animal drug manufacturer that has a long history of violating current good manufacturing practice for finished pharmaceuticals (GMPs; 21 C.F.R 211) regulations.

Under the Consent Decree, the firm and its president are permanently restrained and enjoined from manufacturing, processing, packaging, labeling, testing, holding, and distributing any drugs at their establishment in El Monte, California until the methods, facilities, and controls used there are in compliance with current GMP regulations. Also, within 90 days of the entry of this decree, Anthony Products must select a person(s) qualified to make inspections of facilities where their drugs are manufactured, processed, packaged, labeled, tested, or held. That person(s) must inspect the defendant's facilities located in Irwindale and Arcadia, CA, and determine whether the facilities are in conformity with GMP requirements. All GMP deviations must be corrected within that 90-day period.

If the defendants fail to comply with these requirements within 90 days, they are required to immediately: 1) recall, at their expense, any drug found to violate the Federal Food, Drug, and Cosmetic Act or the requirements of the Consent Decree, and 2) halt all manufacturing, processing, packaging, labeling, testing, and distribution of all drugs or component parts, unless FDA provides written authorization to resume their operations. Failure to comply with the terms of the decree may also result in civil or criminal penalties.

FDA's Los Angeles District Office conducted the investigation which lead to this Consent Decree. CVM's Division of Compliance, FDA's Office of the Chief Counsel, and the U.S. Department of Justice's Office of Consumer Litigation were in charge of the case processing and approval.

Issued by: *FDA, Center for Veterinary Medicine, Office of Management and Communications, HFV-12*
7500 Standish Place, Rockville, MD 20855
Telephone: (301) 594-1755
FAX: (301) 594-1831
Internet Web Site: http://www.fda.gov / cvm