

Red kidney bean poisoning in the UK: an analysis of 50 suspected incidents between 1976 and 1989

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SUMMARY

Between July 1976 and February 1989, 50 incidents of suspected red kidney bean poisoning were reported in the UK. Nine incidents in which nausea, vomiting and diarrhoea developed within 1–7 h of ingestion, were confirmed by the detection of haemagglutinin in the beans. The diagnosis was made on a further 23 incidents on the basis of symptoms, incubation time and the description of preparation of beans prior to consumption. The haemagglutinin (lectin), which occurs naturally in the red kidney bean, is inactivated by thorough cooking of well soaked beans. In many of the outbreaks reported the implicated beans were consumed raw or following an inadequate heat process.

INTRODUCTION

Food poisoning following the consumption of castor beans was first reported in 1889 [1] and after an outbreak in Berlin in 1948 it was shown that the toxin in 'flaked' beans was destroyed by further cooking [2]. The first reported outbreak of food poisoning in the UK attributed to red kidney beans (*Phaseolus vulgaris*) occurred in 1976 and a detailed account of this and six other incidents between 1976 and 1979 was published by Noah and colleagues [3]. These and subsequent incidents in the UK have followed the consumption of raw or incompletely cooked red kidney beans. Symptoms develop after a 1–3 h incubation period and include nausea and vomiting, followed by diarrhoea and sometimes abdominal pain; recovery is usually rapid. Outbreaks have also been reported in Canada [4] and Australia [5], each incident involving two persons who had consumed 'chili con carne'.

This form of food poisoning is a chemical intoxication and is apparently due to the presence of high levels of phytohaemagglutinins in the beans. Haemagglutinins (lectins) are proteins or glycoproteins, capable of binding to specific carbohydrate residues [6], which have been detected in a wide variety of leguminous seeds including soybeans (*Glycine max*), lentils (*Lens esculenta*), lima beans (*Phaseolus*

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lunatus) and red kidney beans (*Phaseolus vulgaris*) [7]. Although the mechanism of toxicity is not known [8], the administration of lectins by mouth has been shown to reduce intestinal absorption and cause weight loss, growth retardation and diarrhoea in several animal species [6, 9]. Honavar and colleagues [10] showed that rats fed on raw red kidney beans died within 1 week, but when the beans were soaked and then autoclaved the toxic activity was destroyed. Purified kidney bean haemagglutinin when added to rat food at a concentration of 0.05% caused death within 2 weeks. Higher levels of haemagglutinin caused more rapid death but when the haemagglutinin preparation was boiled the toxic effect was eliminated.

Inactivation of the toxin in beans may be achieved by soaking overnight followed by boiling in fresh water for at least 10 min [1]. Heating beans at a lower temperature is reported to result in an increase in levels of lectin [1], hence the observation that beans cooked at 82 °C (which were still palatable) were toxic whereas those cooked at 91 °C were not [8]. Thompson and colleagues [11], however, did not observe an increase in phytohaemagglutinin activity when beans were heated at temperatures below boiling point.

In 1981, a television programme produced by the British Broadcasting Corporation high-lighted the problem of food poisoning due to the consumption of raw red kidney beans. An appeal for information on further possible cases resulted in 355 letters concerning incidents which had involved a total of 870 people. Only a tenth of this number of cases had previously been reported to the Public Health Laboratory Service, Communicable Disease Surveillance Centre (CDSC). A high proportion (93%) of incidents occurred in the home, and only 7% were associated with meals in restaurants and refectories [1]. This public awareness of the problem and the improvement in labelling of dried beans on retail sale may be responsible for the subsequent decrease in the number of reported incidents in the UK since.

An analysis of the 50 food poisoning incidents where red beans were implicated and which have been reported to either the Food Hygiene Laboratory (FHL) or the CDSC at Colindale since 1976 is presented. The series includes seven incidents the details of which have already been published [3].

MATERIALS AND METHODS

Samples

Between July 1976 and February 1989 50 incidents of food poisoning allegedly associated with red kidney beans, involving more than 200 cases were reported. Beans for analysis were received from 31 of the incidents, 28 by the Food Hygiene Laboratory, 2 were tested at Queen Elizabeth College, London and 1 at the Newcastle Public Health Laboratory. Beans were not available from the remaining 19 incidents. Haemagglutinin levels were measured on an extract of the beans by reaction with rabbit erythrocytes [12].

Extraction of the haemagglutinin

Beans (10–20 g) received at FHL were split into halves and frozen overnight at –20 °C. The sample was then dried for 48 h in a freeze drier (Edwards, Crawley, Sussex) and powdered using a pestle and mortar. One gram of dried beans was

suspended in 10 ml of 0.85% w/v saline, held at room temperature for 2 h and shaken occasionally by hand. The suspension was stored overnight at 4 °C to extract the lectin, centrifuged at 15000 g for 30 min and the supernatant fluid retained for analysis.

Preparation of blood

Fresh rabbit blood (approx. 7 ml) was added to 1 ml of citrate phosphate dextrose buffer [10] and diluted 1 in 40 in 0.85% (w/v) saline for use.

Haemagglutination assay

The test sample and a standard positive control of 500 µg/ml phytohaemagglutinin type V (Sigma L8754) were double diluted in 50 µl 0.85% (w/v) saline in a U-bottomed microtitre plate. A negative control of saline or a known negative extract was also included in each test.

To each well was added 50 µl of diluted blood and the plate rocked gently to mix the solutions: the plate was incubated at room temperature. Plates were read after 18–24 h.

Haemagglutinin activity is expressed as the reciprocal of the highest dilution causing agglutination (HAU/ml) multiplied by the initial dilution factor of the sample to give HAU/g dry weight bean.

A base level of 400 HAU/g was established in the FHL from a series of tests on well cooked beans (unpublished). Raw beans were shown to contain 80000–100000 HAU/g.

RESULTS

Of the 50 incidents studied, 32 were considered to be compatible with the diagnosis of red kidney bean poisoning on the basis of symptoms and high levels of haemagglutinin in the beans concerned (9 incidents) or on the basis of the history only, i.e. incubation period, symptoms and method of bean preparation (23 incidents) (Tables 1 and 2). Of the other 18 reported outbreaks, 14 were not considered clinically acceptable and in 11 of the 12 bean samples tested the haemagglutinin level was < 400. The remaining sample was a single bean with a level of 1600 HAU/g (slightly raised). In the other four insufficient information was available to support the diagnosis, though two bean samples had slightly raised haemagglutinin levels.

Details of the nine laboratory confirmed cases are given in Table 3. The concentrations of haemagglutinin in these incidents were > 3200 HAU/g. In episode 1, butter beans in a salad with the red kidney beans were also shown to be highly toxic. In seven of the incidents the beans were soaked prior to cooking, although in one instance for 3 h only (episode 3) and the subsequent cooking periods varied. In two instances the beans were soaked but not cooked prior to consumption (episodes 5 and 6). In only one incident was it stated that the beans were boiled (episode 2). There were no details as to the preparation of beans in episode 8.

The clinical and cooking details in 16 of the 23 incidents which were confirmed on the basis of clinical symptoms are given in Table 4. Details of the other seven

Table 1. *Fifty suspected incidents of red kidney beans poisoning, 1976-89*

No. incidents*	No. where beans tested	No. with HAU levels		No. where beans not tested	No. clinically compatible
		> 400	< 400		
9	9	9	0	0	9
23	6	0	6	17	23
4	4	2†	2	0	N.A.‡
14	12	1†	11	2	0
Total					
50	31	12	19	19	32

* Number of positive incidents = 32.

† Slightly raised.

‡ N.A., no information.

Table 2. *Incidents of food poisoning associated with red kidney beans, 1976-89*

Year	Number of suspected incidents	Number confirmed by haemagglutinin levels	Number confirmed by symptoms and history
1976-9*	7	0	7
1980	13	2	9
1981	8	1	5
1982	5	1	1
1983	1	0	0
1984	1	0	0
1985	6	0	0
1986	1	0	0
1988	7	5	1
up to Feb 1989	1	0	0
Total	50	9	23

* Noah and colleagues, 1980 [3].

Table 3. *Incidents of red kidney bean poisoning confirmed by haemagglutinin assay*

Incident	Year	Number of cases	Number at risk	Symptoms*	Incubation time (h)	HAU/g dry weight of beans
1	1980	9	20	V, D	2-3½	16000†
2	1980	4	4	V	2	'toxic'†
3	1981	1	1	V, D, Ap	6-7	3200
4	1982	4	20	V, D, Ap	1-3	13800
5	1988	1	?	V	3	6400
6	1988	'several'	?	V	3	102400
7	1988	11	31	V, D	3-7	51200
8	1988	8	?	V, D, Ap	2½-6	< 50%‡ inactivation
9	1988	5	?	V	3-6	102400

* V, vomiting; D, diarrhoea; Ap, abdominal pain.

† Assay at Queen Elizabeth College, London.

‡ Assay at PHL Newcastle.

Table 4. Incidents of red kidney bean poisoning confirmed by symptoms and history

Incident	Year	Number of cases	Number at risk	Symptoms*	Incubation time (h)	Preparation of beans
10	1980	1	1	V, D, Ap	2	Soaked raw
11	1980	1	1	N, V, D	?	Unsoaked, simmered 45 min
12	1980	4	5	N, V, D	2-3	Soaked, cooked in casserole
13	1980	3	3	N, V, D	4-5	Soaked, baked
14	1980	5	5	V, D	2	Soaked, cooked 20 min from cold
15	1980	4	6	N, V	3	Soaked
16	1980	4	4	V, D	2-4	Microwave †
17	1980	1	1	N, V	3½	Soaked, raw
18	1980	4	4	N, V, Ap	< 1	Soaked ½ h, pressure cooked & electric slow cooker
19	1981	2	2	V, D	6-12	Electric slow cooker
20	1981	9	9	V, D, N	2-5	Raw
21†	1981	7	35	V, Ap	1-4	Soaked, simmered 2 h in casserole
22†	1981	2	2	N, V, Ap	1½	Soaked, and baked
23	1981	1	1	V, D	1½	Raw
24†	1982	1	1	V	3	Soaked, boiled 4 min
25†	1988	1	?	N, A, D, Ap	1	Not known

* N, nausea; V, vomiting; D, diarrhoea; Ap, abdominal pain.

† Beans contained 400 HAU/g.

‡ Beans added during cooking.

incidents have been published previously [3]. In all cases, the cooking process described would have been insufficient to destroy the lectins. Beans were submitted for analysis in only six of these incidents, but the levels of lectin were < 400 HAU/g. A possible explanation is that uneven heat distribution and penetration during cooking resulted in some beans in the batch not being cooked thoroughly while in others the lectins were destroyed.

In ten of the incidents which were not confirmed by either HAU levels or clinical compatibility the beans were 'canned products'. Heat treatment during the canning process should be adequate to destroy haemagglutinin in the beans thus the diagnosis seems unlikely. None of the confirmed incidents was due to canned beans.

DISCUSSION

The rise in reported incidents between 1976 and 1980 has been linked to public interest in 'health foods' and 'healthy eating' [1]. Compared to other causes of food poisoning, relatively few incidents due to red kidney beans are reported. As red kidney beans are a fairly common dietary item and only a small number of incidents are reported it is assumed that the beans are generally well cooked before consumption. The low reporting may also be associated with such factors as improved labelling of raw beans, the common usage of canned beans which have been heat treated, the lack of suspicion of beans as a cause of food poisoning and to the under-reporting of food poisoning generally, particularly that of short duration. The sudden increase in incidents in 1988 is unexplained. The possibility that newer varieties of red kidney beans with naturally higher lectin content had become available was considered. However, in one instance in 1988 where the raw beans used for cooking were available for testing, the haemagglutinin in pre-soaked beans was destroyed within the recommended ten minutes of boiling.

While some workers have reported that the level of haemagglutinin in beans may be reduced by soaking the beans in cold water [1], others suggested that the volume of water used may be critical for the leaching out of the toxin, as they found no reduction in haemagglutinin levels [11].

The two critical factors in reducing the amount of lectin would appear to be the period of presoaking and the subsequent heat treatment. Without adequate soaking, leaching of toxins and heat penetration may be insufficient to destroy the toxin. Some reports suggest that lectins are resistant to inactivation by dry heat [11]. It is clear from the data reported here that in those incidents diagnosed as red kidney bean poisoning there had been either inadequate periods of soaking or of boiling or both. It has been reported that low temperatures may increase lectin activity [1], therefore simmering, oven baking at low temperature or slow cookers may be particularly unsuitable methods of preparation of beans.

In the nine laboratory confirmed incidents (see Table 3) the levels of haemagglutinin were shown to be between > 3200 and 102000 HAU/g. Levels between the baseline of 400 HAU/g and 3200 HAU/g were considered as 'possibly toxic' and episodes where the haemagglutinin levels fell within this range were confirmed in the light of other evidence. Small amounts of toxin may be sufficient to cause illness as in one incident a patient reported eating only one or two beans [3].

If an attack of vomiting, with or without diarrhoea or abdominal pain occurs within 1-3 h of ingesting red kidney beans it can be assumed that the beans are the cause of the symptoms. Other toxic causes such as *Staphylococcus aureus* and *Bacillus cereus* should be excluded. In the seven incidents reported by Noah and colleagues [3] faecal samples from the patients and samples of beans were examined microbiologically. No likely pathogens were isolated. This would suggest that the cause of the poisoning was a toxic component of the bean.

Haemagglutinins are present in most legume seeds in varying amounts [7]. In many seeds, the levels are not high enough to cause illness even when eaten raw. The identification of butter beans containing high levels of lectin is in accordance with the data of Grant and colleagues [13] who noted that butter beans were

'intermediate' with respect to lectin mediated toxicity. In one suspected outbreak, investigated at FHL, haricot beans made into vegetable burgers were associated with illness. While insufficient information was available to support the connection the raw beans contained similar levels of haemagglutinin to raw red kidney beans.

That food poisoning due to red kidney beans is easily prevented needs to be emphasized. The beans should be soaked in water for at least 5 h, the water poured away and the beans boiled in fresh water for at least 10 min.

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REFERENCES

1. Bender, AE, Reaidi GB. Toxicity of kidney beans (*Phaseolus vulgaris*) with particular reference to lectins. *J Plant Foods* 1982; **4**: 15-22.
2. Griebel C. Erkrankungen durch Bohnenflocken (*Phaseolus vulgaris* L.) and Platterbsen (*Lathyrus tingitanus* L.) *Z Lebensm Unters Forsch* 1950; **90**: 191.
3. Noah ND, Bender AE, Reaidi GB, Gilbert RJ. Food poisoning from raw red kidney beans. *Br Med J* 1980; **281**: 236-7.
4. Todd E, Pivnick H, Pivnick K. Illness from kidney beans. *Canada Diseases Weekly Report* 1980; **6**: No. 21: 101-3.
5. Tan A. Non-bacterial food poisoning. *Communicable Diseases Intelligence (Australia)* 1982; No. 6: 7.
6. Pusztai A. The role in food poisoning of toxins from plants. In: Robinson RK, ed. *Developments in food microbiology - 2*. London: Elsevier Applied Science Publishers 1986: 179-94.
7. Liener IE. Phytohaemagglutinins: Their nutritional significance. *J Agr Food Chem* 1974; **22**: 17-22.
8. Coffey DG, Uebersax MA, Hosfield GC, Brunner JR. Evaluation of the hemagglutinating activity of low-temperature cooked beans. *J Food Sci* 1985; **50**: 78-87.
9. Jaffe WG. Toxic proteins and peptides. In: *Toxicants occurring naturally in foods*, 2nd ed. Washington DC: National Academy of Sciences 1973; 106-29.
10. Perkins HA. Strategies for massive transfusion. In: Patz LD, Swisher SN, eds. *Clinical practice of blood transfusion*. New York: Churchill Livingstone 1981; 485-99.
11. Thompson LU, Rea RL, Jenkins DJA. Effect of heat processing on haemagglutinin activity in red kidney beans. *J Food Sci* 1983; **48**: 235-6.
12. Liener IE. The photometric determination of the haemagglutinating activity of soyin and crude soybean extracts. *Arch Biochem Biophys* 1953; **54**: 223-31.
13. Grant G, More LJ, McKenzie NH, Stewart JC, Pusztai AA. Survey of the nutritional properties of legume seeds available in the UK. *Br J Nutr* 1983; **50**: 207-11.