



Eva Crane Trust

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TITLE: Honey in relation to infant botulism

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Bee Products

Honey in relation to infant botulism

Botulism is a type of food poisoning that occurs very rarely; an outbreak is likely to affect a number of people who have all eaten the same item of contaminated food, and over half of them may die. In the USA between 1900 and 1950 the mortality rate was 60%-70%, but improved treatment methods have now reduced it to 13%; there were 48 cases in 1978¹⁴.

The poisoning is caused by ingestion of a neural toxin which is produced as a by-product of the metabolism of *Clostridium botulinum*. This saprophytic bacterium occurs very commonly in soils, but germination and growth can occur only in a neutral environment (pH very near 7) and in the absence of air or oxygen, i.e. in anaerobic conditions. The toxin inhibits the release of acetyl choline in the nerve synapses, preventing the transmission of nerve impulses which control breathing and many other essential functions. It is the most poisonous substance known, the lethal dose for man being 10^{-9} mg/kg body weight⁷. The toxin is destroyed by heat, and foods containing it can be made safe by boiling.

The bacterial spores, on the other hand, are very resistant to heat. They are ubiquitous, occurring in plants and animals as well as in the soil. For instance the raw vegetables we eat are often contaminated with them. The spores can also occur in hive products, including honey, and (like spores of *Bacillus larvae* that cause AFB) they can survive in honey for very long periods. The spores have been regarded as harmless to man, because the adult digestive tract is too acid for their germination, and the bacterial flora already present provides too much competition for a population of *C. botulinum* to build up and produce toxin.

In California, USA, botulism due to *in vivo* production of toxin* was for the first time suspected, and confirmed, in 1976; this was not in adults but in infants under 6 months old. In the next three years 50 cases were confirmed in California, and 48 in

*known as *toxicoinfection* to distinguish it from poisoning due to ingestion of the *preformed* toxin.

the rest of the USA (up to the end of 1978¹⁴; single cases have been reported from England⁹ and Australia¹⁴).

The human intestinal flora develops after birth; also until 6 months the digestive system is probably less acid than in adults. So if spores are ingested by a baby they have a greater chance of germinating and producing a population of *C. botulinum* which can form toxin within the digestive system. However, it is a slow process, and when this form of botulism is recognized in an infant, supportive treatment can be given. As a result, 96% of the babies known to have been infected with *C. botulinum* have recovered¹⁶, although the mechanism of recovery was obscure.

Babies are given a much more limited range of foods than adults, and intensive enquiries were made as to the source of the spores that infected the infants in California. Honey was one of the foods that some of them had eaten, and this caused a great stir in the honey industry of the United States. Investigations include the following statements.

- (a) Of 41 cases in California, 12 had recently been fed honey¹⁶.
- (b) Spores were found in 10%-15% of honeys tested in California⁸.
- (c) No spores were found in 17 samples from previously unopened jars; the pH of the honey was between 3.5 and 5.0⁴.
- (d) Spores were found not only in honey in opened jars (which might have become contaminated in many ways) but also in honey in closed jars offered for retail sale, and in bulk honey before it was processed¹⁶.
- (e) Among 241 samples of commercial honey from 32 states of the USA, 18 (from 9 states) contained *C. botulinum* spores; the maximum probable number was 7 spores per 25 g honey¹³.

It is clear that honey has not been a major cause of infant botulism. There is little evidence on the presence or absence of spores in the honey eaten by the 12 Californian babies that became infected¹⁶, but their presence has been established in 3 cases¹⁶. No infants affected were older than 8 months¹⁴.

The whole affair raises many problems, and although most of these are unrelated to honey, some beekeepers have become alarmed since honey has become entangled with it, with statements in the press that honey is a possible source of infant botulism. The following are a few of the questions that may be asked.

- (a) Why should the majority of cases be reported in California? Do they occur as widely elsewhere, unrecognized?
- (b) Is infant botulism really a new occurrence, or has it existed for many years?
- (c) If it has occurred, unrecognized and untreated, does it have any connection with what are known as cot deaths or crib deaths in babies? Some recent studies on 280 such deaths in California showed *C. botulinum* in 10 of the babies, but none of these 10 had been fed honey⁸.
- (d) Is honey implicated simply because it is a more stable substance than most baby foods, and therefore still available for analysis when other foods have been fully consumed or thrown away¹¹?

From the beginning of recorded history, and probably earlier, honey has been given to newborn children as part of the birth rite, in many regions of the world. Three independent studies in different parts of India were published in 1973, and they showed that the majority of newborn children were still fed honey before breast feeding was commenced¹⁷⁻¹⁹. It is notable that the reports of infant botulism have come from the USA, a country with very high standards of hygiene.

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Selected references (all in the IBRA Library)

Papers are listed here in chronological order. Most are primary reports in scientific and medical journals. The many newspaper articles provide information on mental reactions to the subject, rather than on the subject itself.

1976

1. MIDURA, T. F.; ARNON, S. S. Identification of *Clostridium botulinum* and its toxins in faeces. *Lancet* 30 October : 934-936
2. EDITORIAL. Botulism in infants—California. *Morbidity and Mortality Weekly Report* 25(34) : 269

1977

3. EDITORIAL. Follow-up on infant botulism—California, Pennsylvania, Tennessee, Texas. *Morbidity and Mortality Weekly Report* 26(5) : 30-31
4. ARNON, S. S.; MIDURA, T. F.; CLAY, S. A.; WOOD, R. M.; CHIN, J. Infant botulism: epidemiological, clinical and laboratory aspects. *J. Am. med. Ass.* 237(18) : 1946-1951
5. ARNON, S.; DAMUS, K.; MIDURA, T.; TAYLOR, P.; WOOD, R.; CHIN, J. Infant botulism in California: preliminary report on environmental and host risk factors. *17th Interscience Conference on Antimicrobial Agents and Chemotherapy* : 4 pages

1978

6. EDITORIAL. Follow-up on infant botulism—United States. *Morbidity and Mortality Weekly Report* 27(3) : 17-18, 23
7. ARNON, S. S.; MIDURA, T. F.; DAMUS, K.; WOOD, R. M.; CHIN, J. Intestinal infection and toxin production by *Clostridium botulinum* as one cause of sudden infant death syndrome. *Lancet* 17 June : 1274-1277
8. MARX, J. L. Botulism in infants: a cause of sudden death? *Science, N.Y.* 201 : 799-801
9. TURNER, H. D.; BRETT, E. M.; GILBERT, R. J.; GHOSH, A. C.; LIEBESCHUETZ, H. J. Infant botulism in England. *Lancet* 17 June : 1277
10. EDITORIAL. A new role for botulism? *Lancet* 17 June : 1295-1296
11. MORSE, R. A. The botulism story. *Glean. Bee Cult.* 106 (9) : 427, 437
12. HATHEWAY, C. L.; MCCROSKEY, L. M.; DOWELL, V. R. Activities of the Center for Disease Control, Botulism Laboratory. *Interagency Botulism Research Coordinating Committee Meeting* : 14 pages
13. SUGIYAMA, H.; MILLS, D. C.; KUO, L.-J. C. Number of *Clostridium botulinum* spores in honey. *J. Fd Prot.* 41(11) : 848-850

1979

14. EDITORIAL. Botulism—United States, 1978. *Morbidity and Mortality Weekly Report* 28(7) : 73-75
15. ROGERS, E. F. Sudden infant death. *Science, N.Y.* 203 : 1197
16. ARNON, S. S.; MIDURA, T. F.; DAMUS, K.; THOMPSON, B.; WOOD, R. M.; CHIN, J. Honey and other environmental risk factors for infant botulism. *J. Pediatr.* 94 : 331-336

Studies in India, 1973

17. BANSAL, R. D.; GHOSH, B. N.; BHARDWAJ, U. D.; JOSHI, S. C. Infant feeding and weaning practices at Simla-Hills, Himachal Pradesh. *Indian J. med. Res.* 61(12) : 1869-1875
18. BHANDARI, N. R.; PATEL, G. P. Dietary and feeding habits of infants in various socio economic groups. *Indian Pediatrics* 10(4) : 233-238
19. ARORA, D. D.; KUAL, K. K. Feeding practices during the first five years among central Indian communities. *Indian J. Pediatr.* 40(305) : 203-216