Advisory Committee on the
Microbiological Safety of Food

Ad Hoc Group on Infant Botulism

Report on Minimally Processed Infant Weaning Foods and the Risk of Infant Botulism

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Contents

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The ACMSF accepts full responsibility for the final content of the report.

Summary

1. In this report we have attempted to establish the potential risk to human health associated with the consumption of chilled or frozen baby foods, particularly in relation to *Clostridium botulinum* and infant botulism.

2. This report comes against the background of an apparent increasing move towards development and sale of chilled and frozen baby foods as a wholesome and more nutritious alternative to traditional pre-cooked puréed meals packaged in jars, cans or pots, and dried foods which need rehydrating. These chilled and frozen products only receive a moderate cooking process and may therefore contain *C. botulinum* spores.

3. In September 2003 we decided to set up an *ad hoc* group to assemble information and review evidence about the products and processes concerned in the production of chilled and frozen foods for babies aged 4-12 months, and to inform the development of Advisory Committee on the Microbiological Safety of Food advice to the Food Standards Agency. The Group met on several occasions over a period of 15 months.

4. At the beginning of our investigations we considered whether there was a possible association between *C. botulinum* and Sudden Infant Death Syndrome. We concluded that there were mixed views within the medical and research communities as to whether some cases of Sudden Infant Death Syndrome could be misdiagnosis of extreme forms of infant botulism, noting that there were no UK data to contribute. We recommended that there is merit in assessing the link between *C. botulinum* and Sudden Infant Death Syndrome and research should be undertaken in the UK.

5. Subsequently we examined how minimally processed baby foods were manufactured, and reviewed current food safety controls in place and any similarities with processes and controls in place for other foods that are currently consumed by infants and babies. The *Ad Hoc* Group received evidence from a range of companies and individuals on their understanding of the risk regarding *C. botulinum*, current processing conditions and controls. The Group also considered infant food production in the home and guidance available to manufacturers, enforcers and consumers. We concluded that, based on relative risk, chilled and frozen minimally processed baby foods do not seem to be at any greater risk of containing proteolytic *C. botulinum* than current commercially available and home produced foods consumed by infants. We recognised that minimally processed baby foods can support growth of non-proteolytic *C. botulinum* when stored under chilled conditions, and any growth hazard needs to be controlled. The Group also received evidence which indicated that the nature of the production of minimally processed baby foods by some businesses offered opportunities for contamination with general foodborne pathogens, if

conditions were not properly controlled. We therefore recommended and outlined specific controls that should be put in place for minimally processed foods to destroy non-proteolytic *C. botulinum* spores, or prevent growth during the shelf life of the product, or after defrosting (for frozen products). We also recommended controls and procedures to prevent recontamination of minimally processed foods after heat processing, whether by *C. botulinum* or other organisms, including *Listeria monocytogenes*. In addition we recognised that good practice guidelines for re-cooking any ready to eat foods that can support growth of microbial pathogens should also be applied to minimally processed baby foods of a similar composition.

6. We considered how product safety controls were enforced, and the Group heard evidence from representatives of three Local Authorities who had been involved in the examination and/or approval of minimally processed baby food manufacturing within their areas. We concluded that, based on the evidence presented Local Authorities were not generally aware of the microbiological risks involved with the production of minimally processed infant foods. There were also inconsistencies in the level of awareness of the risks and hazards associated with minimally processed foods, and the approaches used within individual environmental health departments across the UK. Evidence presented to the Group also indicated that the microbiological and process control knowledge of minimally processed infant food companies was variable. We therefore made several recommendations to ensure Local Authorities are aware of the risks of and hazards associated with infant botulism, and controls required to control the hazards. We also highlighted the need for consistent guidance for EHOs to inform baby food manufacturers and identified that such guidance should also be applicable to industry and parents in the home. The need for key controls and observation of good manufacturing practice is highlighted, as is the need for reiteration of existing advice on food safety management based on HACCP principles.

7. Finally, a Sub-Group was convened which carried out a risk assessment for *C. botulinum* in infant foods. This risk assessment work was subsequently peer-reviewed by independent scientific experts. Based on the results of the risk assessment, we concluded that minimally processed infant foods are not a major source of exposure with regard to infant botulism. Further, the risk of infant botulism from packs of food other than honey is low, as it is rare for packs to be contaminated with 10 or more spores. A lack of information on the dose-response relationship precludes estimation of the risk presented by packs of infant food containing less than 10 spores. As further information becomes available, we have recommended that there would be further merit in conducting an extended risk assessment.

8. The Sub-Group also considered the risk of cases of infant botulism associated with the consumption of honey, recognising current advice from the Food Standards Agency and Department of Health that recommends that honey should not be given to infants less than 12 months of age. We recommended based on a review of the evidence, honey should not be added to foods specifically targeted at infants under 12 months of age (unless these foods receive a full botulinum cook or an equivalent process control).

9. The assessments made and the conclusions the Group have reached reflect, in large measure, the evidence, oral and written, drawn from the scientific community, enforcers, manufacturers, government departments, and from the scientific literature. Our conclusions and recommendations are presented at the end of each chapter and are also drawn together at the end of the report.

Chapter 1

Introduction

Introduction

1.1 This report from the Advisory Committee on the Microbiological Safety of Food (ACMSF) reviews the potential risk to human health associated with the consumption of chilled or frozen baby foods, particularly in relation to *Clostridium botulinum* and infant botulism. The report also describes measures that may be taken to reduce the risk of cases of infant botulism from the consumption of these baby foods.

1.2 There appears to be an increasing trend towards development and sale of chilled and frozen baby foods since they are perceived as being a wholesome and more nutritious alternative to traditional ambient-stable pre-cooked puréed meals packaged in jars, cans or pots, and dried foods which need rehydrating. These chilled and frozen products only receive a moderate cooking process and may therefore contain *C. botulinum* spores.

1.3 Infant botulism was first recognised in 1976, since when over 1,500 cases have been reported in more than 15 countries. The condition is caused by ingestion of *C. botulinum* spores which leads to subsequent colonisation and toxin production in the gastrointestinal tract. Symptoms include constipation, weak cry, feeding difficulty and muscle weakness. Treatment involves supportive and respiratory care, and recovery generally occurs in weeks or months. Mortality rates are low (around 5%), and there are usually no long-term effects. In the USA, prompt treatment of infant botulism type A or type B with BabyBIG® (Botulism Immune Globulin Intravenous (Human) BIG-IV) has been shown to be safe and effective in shortening the severity of illness and the length and cost of the hospital stay[†].

1.4 Exposure via food may arise through consumption of commercial baby foods which do not receive sufficient heat treatment to destroy all *C. botulinum* spores. It may also occur via consumption of home made baby foods and other foods not aimed at children, which may contain spores. Opened foods may also become contaminated with spores from the environment, particularly where there is prolonged use.

[†] Arnon, SS, Schechter, R Maslanka, SE, Jewell, NP and Hatheway CL (2006) Human botulism immune globulin for the treatment of infant botulism. New England Journal of Medicine 354 (5): 462-471

The ACMSF's approach to its work

1.5 In 2003 the Committee established an *ad hoc* group of members and co-opted experts which sought to establish the size and nature of the risk of infant botulism associated with chilled and frozen weaning foods. The Terms of Reference for the Group are outlined at Annex 1. We met on seven occasions and considered documentary and verbal evidence relating to the clinical epidemiology of infant botulism, and Sudden Infant Death Syndrome and *C. botulinum.*

1.6 We also considered a wide-range of information and heard evidence relating to minimally processed baby foods manufacturing and process safety controls, infant food production in the home and guidance available to manufacturers, enforcers and consumers.

1.7 We reviewed general good manufacturing principles, heat processing conditions and product shelf life and critical points for enforcers.

1.8 Finally, a Sub-Group was convened which carried out a risk assessment for *C. botulinum* in infant foods. This risk assessment was subsequently peer-reviewed by independent scientific experts.

Chapter 2

Infant (Intestinal Colonisation) Botulism

Microbiology

2.1 *Clostridium botulinum* is an anaerobic organism that produces heat resistant spores and one or more potent neurotoxins. Three types of disease are recognised in humans: foodborne botulism, infant botulism and wound botulism. In all three, symptoms are due to the neurotoxin. Occasionally, other clostridial species, notably *Clostridium butyricum* and *Clostridium baratii*, produce botulinum toxins[1].

2.2 *Clostridium botulinum* is sub-typed on the basis of the toxin(s) it produces, of which there are seven types (A-G). Toxin types A, B and E are the most common causes of human foodborne botulism, but type F may be involved in rare cases. To date, almost all cases of infant botulism have been due to types A and B. There are a few reports of infant botulism associated with *C. butyricum* and *C. baratii*, with production of type E and F toxins[2,3,4], and there is also a single case reported as being due to a type C producing *C. botulinum*[5]. The evidence for the latter is much less strong.

2.3 The seven types of *C. botulinum* are also classified into four sub-groups, based on physiological characteristics. Human illness is associated with organisms belonging to Groups I and II. Group III contains types C + D, which are associated with avian and non-human cases of botulism. The main distinctions between Groups I and II are that Group I is proteolytic and grows at temperatures between 10°C and 48°C, whereas Group II is non-proteolytic and grows at 3°C to 45°C. Because of their ability to grow at temperatures below 5°C, the latter are also termed psychrotrophic. This group comprises strains forming toxins of types B, E and F. Group I, which accounts for almost all reported cases of infant botulism, includes strains forming toxins of types A, B and F.

2.4 Proteolytic *C. botulinum,* and *C. butyricum* and *C. baratii,* grow optimally at or close to body temperature whereas non-proteolytic *C. botulinum* grows optimally at temperatures below this. Hence non-proteolytic *C. botulinum* may be at a competitive disadvantage in the human gut.

2.5 Whilst spores of all types of *C. botulinum* are heat resistant, the spores of proteolytic *C. botulinum* are more heat resistant than the spores of non-proteolytic *C. botulinum*. At 100°C, 90% of spores of non-proteolytic *C. botulinum* are killed in less than 0.1 minute, whereas, in the case of the

proteolytic *C. botulinum*, this takes 25 minutes. In order to assure destruction of spores of proteolytic *C. botulinum* in low acid foods, a treatment sufficient to give a 12 log reduction is required, namely moist heat at 121°C for 3 minutes. These conditions are generally delivered during canning and result in products that are stable at ambient temperatures for very long periods of time. A less stringent treatment, sufficient to produce a 6 log reduction, is designed to destroy spores of non-proteolytic *C. botulinum* in foods that will then be stored for a limited period under chilled conditions. A typical time/temperature combination to effect this level of kill is 90°C for 10 minutes.

2.6 Foodborne botulism is due to ingestion of pre-formed toxin, produced in foods where anaerobic conditions and high pH allow growth from spores that have survived earlier heat treatments. The toxin is potentially lethal at a very low dose. In contrast, infant botulism is due to the ingestion of spores that survive in the GI tract, germinate and multiply, producing toxin in situ. It occurs almost exclusively in infants below the age of 12 months although occasionally, adults may experience a similar picture of colonisation with toxin-formation *in situ*. Wound botulism tends nowadays to be associated with injecting drug use[6].

2.7 Botulinum toxin interrupts the transmission of nerve impulses to muscles. It produces constipation, double vision, dry mouth, difficulty in swallowing, weakness of limbs, descending paralysis and respiratory failure. The mortality in foodborne botulism is now around 10%, depending upon the type of toxin, dosage, speed of diagnosis and treatment, and the age of the patient. Mortality in cases diagnosed as infant botulism is lower. However, there is a possibility that some cases of Sudden Infant Death Syndrome may be due to rapid and overwhelming infant botulism. This is discussed elsewhere in this report (Chapter 3).

2.8 Since infant botulism is associated with the ingestion of spores, it is the likelihood of spores being present in raw materials used to make baby foods, and their ability to survive the production process, that are key considerations. The bacterium is widespread in the environment and can be found in dust (both indoors and outdoors), soil, marine sediments, the intestinal tracts of animals and fish, vegetables, fruits, leaves, mouldy hay, silage and animal manure. Thus there is the potential for spores to be present in a wide range of raw materials, as well as the factory environment.

Epidemiology

2.9 Infant botulism was first recognised and described in 1976[7]. It is caused by enteric infection, usually as a result of toxin types A or B produced by *Clostridium botulinum* Group I (proteolytic) organisms[8,9].

2.10 Approximately 90% of the cases which occur worldwide are recognised in the United States of America. The average annual incidence in the US is less than 3 per 1,000,000 live births although higher than average rates have been reported from Delaware, Hawaii, Utah and California. Within the US approximately 47% of cases occur in California[10,11].

2.11 In the United Kingdom there have been 6 confirmed cases of infant botulism between its first recognition in 1978 and April 2005. The features of these cases are summarised in Table 2.1.

2.12 Ninety nine percent of cases occur in children less than one year old although 94% of cases occur in children less than 6 months old. Children aged between 2 to 32 weeks of age seem to be the most vulnerable. In formula fed children the greatest risk is in the first few weeks of life whilst for those who are breast fed the period of greatest risk appears to be at weaning. Infant susceptibility to gut colonisation is thought to be due to age-associated perturbations in the normal gut flora caused by immaturity or dietary changes[1]. Since the syndrome has been associated with weaning from breast milk, changes in gut flora with the introduction of new foods have been implicated in allowing ingested clostridial spores to colonise the intestine. The infectious dose may be as low as 10-100 spores[12].

Symptoms and Signs of Infant Botulism

2.13 A baby with infant botulism may present with constipation, irritability, lethargy or decreased activity. The mother may notice a weak cry with decreased suckle and the baby may fail to thrive. There may be loss of facial expression and loss of head control. Generalised muscle weakness can occur with respiratory paralysis and dysfunction of the autonomic nervous system – hypotension and neurogenic bladder. The combination of symptoms and signs comprising infant botulism is sometimes described as “floppy baby syndrome”. At the mild end of the spectrum the child may present with failure to thrive whilst moderate disease may leave a child paralysed and needing to be ventilated for several months[13]. For the six cases of infant botulism that have presented in the UK since 1978 all have needed to be ventilated.

2.14 The differential diagnosis of infant botulism is not straightforward and, on admission, none of the children was originally suspected of having infant botulism. The admission diagnoses included:

- Failure to thrive, sepsis, dehydration
- Viral syndrome, encephalitis, meningitis
- Pneumonia
- Idiopathic hypotonia

The differential diagnoses also include:

- Hypothyroidism, metabolic disorders
- Infantile spinal muscular atrophy, poliomyelitis
- Congenital myasthenia gravis
- Hirschprung disease
- Poisoning with drugs, toxins or heavy metals[13].

Table 2.1: Confirmed Cases of Infant Botulism in the United Kingdom

Year	Sex	Age	Toxin	Faeces	Serum
1978	Female	5.5 month	A	Toxin + organism	ND
1987	Male	4 months	B + F	Toxins + organism	ND
1989	Female	2 months	B	Toxin	ND
1993	Female	4 month	B	Toxin + organism	Toxin
1994	Male	4.5 months	B	Toxin + organism	Toxin
2001	Female	5 months	B	Toxin + organism	ND

Source: Dr MM Brett, Health Protection Agency, Centre for Infections
ND = Not Detected

2.15 Diagnosis of infant botulism is made by the identification of *C. botulinum* toxin in the serum, or toxin and/or organisms in the faeces from infants exhibiting appropriate clinical symptoms. This can be a lengthy procedure. It may be necessary to culture the sample in cooked meat medium with added glucose and starch, incubate for 4-14 days at 30°C and then test cell-free culture supernatants for toxin using a mouse bioassay. *C. botulinum* isolates are purified from toxin-positive enrichment cultures by incubation of plate sub-cultures under anaerobic conditions for 1-3 days at 30°C. These pure cultures are further incubated in cooked meat medium and the toxin type is then determined by testing and neutralisation of the cell-free supernatant in the mouse bioassay.

Sources of the Organism

2.16 As mentioned in paragraph 2.8, organisms are widespread in the environment and can be found in dust inside or outside the house and in soil. Disturbing the soil, for example on a building site, may increase an infant's exposure to *C. botulinum* from the soil.

2.17 Honey has been linked to cases occurring in the USA, Japan, Argentina, Italy and Denmark by isolation of the same toxin type from cases and jars of honey consumed by the cases[14]. *Clostridium botulinum* has been isolated from up to 20% of honey samples originating worldwide at between 2.5 and 80,000 most probable number (MPN) per kilogram. It has also been isolated from 5 of 961 samples of corn syrups. Proteolytic *C. botulinum* type B organisms have also been isolated from one sample of baby rice out of 40 samples of dry cereals tested[15,16].

2.18 Of the six confirmed cases of infant botulism in the UK since 1978 three (case 2, 1987; case 4, 1993; case 6, 2001) have definitely not consumed honey. The first case in 1978 was reported to have consumed honey but this was not available for testing. Case three (1989) was reported to have eaten honey in The Yemen. *Clostridium botulinum* spores were not detected. Case five (1994) had also eaten honey but this was not available for testing. However *Clostridium botulinum* spores were not detected in a jar that had been processed within 30 minutes of the one that had been eaten. Case six (2001) had consumed infant formula milk powder, and *C. botulinum* toxin type B organisms were isolated from one of five unopened packets from the same batch. However, using more detailed molecular typing techniques (amplified fragment length polymorphism and pulsed field gel electrophoresis) the *C. botulinum* organisms recovered from the infant's faeces could be distinguished from the isolates obtained from the infant formula[17].

Conclusions

2.19 Infant botulism is rare and there is no evidence of an association between development of illness and consumption of chilled and frozen baby foods anywhere in the world literature.

Recommendations

2.20 We recommend that clinicians are reminded of the possibility of infant botulism so that they may consider it earlier in the differential diagnosis of a child with compatible symptoms.

Chapter 3

Sudden Infant Death Syndrome (SIDS) and *C. botulinum*

Possible association between *C. botulinum* and Sudden Infant Death Syndrome (SIDS)

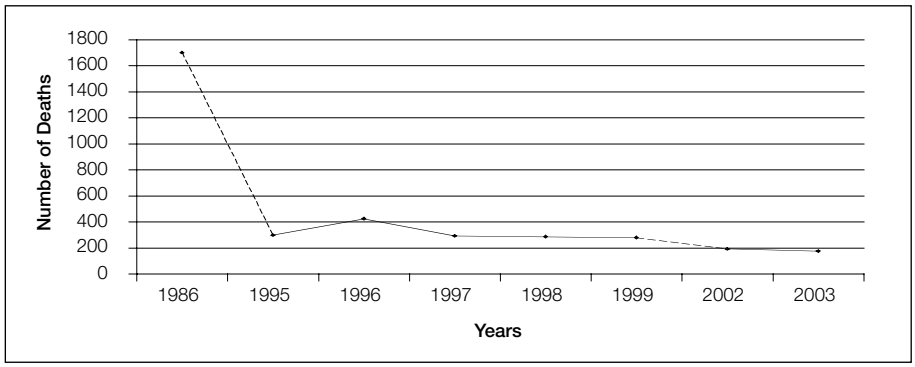
3.1 Most infant diseases including infant botulism, have a spectrum of severity. Consequently, it has been suggested that rapid *in vivo* production of botulinum toxin in the infant's intestinal tract might lead to sudden death without previous signs of illness. This could lead to misdiagnosis of the disease as SIDS and not infant botulism (IB)[18]. Many studies have been carried out to examine this hypothesis.

3.2 SIDS is defined as the sudden death of any infant or young child which is unexpected by history, occurring in association with sleep and lacking explanation after post-mortem investigation. The characteristic age distribution of SIDS is virtually identical to that of infant botulism. The number of SIDS cases in the UK is decreasing as can be seen from the Table 3.1 and Figure 3.1 below[19,20]:

Table 3.1: Sudden Infant Deaths by Year of Occurrence (figures from The Office of National Statistics (ONS)).

Year	Number of SIDS Deaths
1986	1,700
1995	398
1996	424
1997	393
1998	286
1999	279
2002	192
2003	175

Figure 3.1: Sudden Infant Deaths by Year of Occurrence



3.3 This dramatic reduction (75%) in SIDS cases has been attributed to the 'Back to Sleep' campaign which was launched in 1991. Some of the fall in SIDS between 1997 and 1998 may have been due to a diagnostic transfer from SIDS to other causes, particularly for post neonatal deaths. In 2000, death rates from SIDS in England and Wales were 0.47 per 1,000 live births for boys and 0.33 for girls compared to 1996 when rates were 0.75 per 1,000 live births for boys and 0.55 per 1,000 for girls. The rate of SIDS cases reported in various countries can differ markedly (Table 3.2[21]).

Table 3.2: Rates of Sudden Infant Death Syndrome around the World

Country	SIDS (Cases per 1,000 live births)
US	1992 – 1.2 1997 – 0.77
Asia	0.04
Hong Kong	0.2
Scandinavia	0.2 – 0.6
Italy	0.7
UK	1980's – 3.5 2000 – 0.4
New Zealand	1987 – 4.3†† 2000 – 1.1

Risk Factors for SIDS

3.4 More than seventy different risk factors have been implicated in Sudden Infant Death Syndrome including[22,23]:

- Infant sleeping in the prone position.
- Smoking while pregnant (if this risk factor was eliminated it is proposed that the number of SIDS cases would be reduced by 46.7%).

†† New Zealand Ministry of Health 2004

- Caffeine intake during pregnancy.
- Relatively low weight-gain during pregnancy and low pre-pregnancy weight.
- The mother's age at birth of the child.

Link between Infant Botulism and SIDS

3.5 Table 3.3 below summarises the findings from studies which reported cases of SIDS or infant botulism or reviewed evidence for infant botulism as a cause of SIDS[24,18,25,26,10,27,28,29].

Table 3.3: Summary of the findings from studies reporting cases of SIDS or infant botulism or reviewed evidence for infant botulism as a cause of SIDS

Study	SIDS Cases		IB Cases		Controls	
	Positive Isolate	**Positive Toxin**	**Positive Isolate**	**Positive Toxin**	**Positive Isolate**	**Positive Toxin**
US 1977[24]	10/212	2/212			0/68	0/68
Utah 1977-79[18]			12/12		20/87	2/87
US 1981[25]			All cases positive	All cases positive	1/205	0/205
			Possible cases 0/141	Possible cases 0/141		
Canada[26]	1/1					
Switzerland[10]	9/59					
Australia[27]	0/248					
Germany[28]		11/57				4/18
Finland[29]	1/1	1/1				

Table to be read in conjunction with paragraphs 3.6 – 3.13

3.6 Arnon *et al.* (1977) examined autopsy specimens from 280 Californian infants in 1978; 68 who died of known causes and 212 cases of SIDS. *C. botulinum* organisms were found in 10 of 212 SIDS cases (4.7%) and botulinum toxin was detected in 2 specimens of the 10 culture positive cases. The case histories and autopsy findings in these ten cases were indistinguishable from those of typical cases of SIDS. Neither *C. botulinum* organisms nor toxin were found in 68 infants who died of known causes[24].

3.7 Between 1977 and 1979, 12 cases of infant botulism were diagnosed in Utah, USA[18]. In the case control study, the 87 control patients consisted of 32 randomly selected normal babies, 42 infants with nonbotulism neurological disease and 13 infants with systemic illness other than botulism.

C. botulinum was isolated from the stools of 20 infants in the control groups. Toxin and organism were isolated from the stools of two of these infants. Only the organism was isolated from the remaining 18 patients. These 18 children fell into three distinguishable groups including (a) 3 normal children, (b) 9 with nonbotulism neurologic disease and (c) 6 with histories, physical examinations and clinical courses which were mildly suggestive of infant botulism but atypical enough to warrant exclusion from the infant botulism group[18].

3.8 Arnon reported in 1981 that of the 205 healthy infants that had been studied in California since 1976, 72 were selected as residence controls i.e. lived within 1.6 km radius of an infant botulism case to test similar environmental exposures. All had culture negative faecal specimens. Only one of the remaining 133 controls faecal samples was positive for *C. botulinum* but no toxin was found. One hundred and forty one additional infants with diseases that prompted their doctors to request infant botulism testing were negative for both *C. botulinum* and its toxin[25].

3.9 In 1983, *C. botulinum* type A was detected in a case of SIDS that occurred in Canada[26].

3.10 In a study of necropsy specimens in Switzerland, *C. botulinum* of various toxin types were reported to have been isolated from 9 of 59 SIDS cases[10].

3.11 In a study conducted in Australia over a 10-year period from 1981-1990, both small and large intestine specimens from 248 SIDS cases were cultured specifically for *C. botulinum.* However, because no specimens were positive, the investigators concluded that botulism was not a significant factor in the cause of death[27].

3.12 In Central Germany over a 5-year period, 75 infant deaths including 57 SIDS cases were examined. Standardised specimens of blood, liver and intestine were taken at autopsy and tested for botulinum toxin only. In 15 cases, the presence of toxin was proven – 9 by direct toxin tests and 6 with preceding bacteriological enrichment. Eleven of these were from the SIDS group. Eight positives were found in the intestines and the three remaining were found in the liver[28]. In a previous report from Germany no *C. botulinum* organisms were found on bacteriological examinations from 148 SIDS cases[28a].

3.13 Nevas *et al.* (2005) described the first case of infant botulism in Finland[29]. The 11 week-old infant died unexpectedly and the death was classified by the pathologist as SIDS on the basis of the autopsy findings. *C. botulinum* type B toxin gene was identified by PCR in two samples of the infant's intestinal contents and *C. botulinum* was successfully isolated from one intestinal sample. Household samples were tested. *C. botulinum* type B was detected by PCR in, and also isolated from the vacuum cleaner dust. The quantity of spores in the dust was estimated to be 10/kg of examined material. Molecular comparison between the dust isolate and the patient's intestinal isolate demonstrated using PFGE and RAPD that the strains were indistinguishable from each other.

Conclusions

3.14 There are mixed views within the medical and research communities as to whether some cases of SIDS could be misdiagnosis of extreme forms of infant botulism. There are no UK data to contribute.

Recommendations

3.15 We are of the opinion that there is merit in assessing the link between infant botulism and SIDS further and recommend that research should be undertaken in the UK.

Chapter 4

Minimally processed baby foods

Introduction

4.1 Chilled and frozen minimally processed baby foods currently form a minor part of the baby food market (circa. 1%). In assessing the potential risk associated with these foods, it is important to understand how they are processed, the current food safety controls and any similarities with processes and controls in place for other foods that are currently consumed by infants and babies. The *Ad Hoc* Group received evidence from a number of companies and individuals (Annex 2) on their understanding of the risk regarding *C. botulinum*, current processing conditions and controls.

Infant food product market, volumes and sales

4.2 The baby food market sector had an estimated value in 2002 of approximately £381M[30]. The sector is divided into the following categories; milk (infant formulae, follow on, ready-to-feed and soya milk), meals (jars, canned, chilled and pots/trays), finger foods (rusks, breadsticks, rice cakes and others) and drinks (ready-to-serve, concentrates and granulated) (Table 4.1). The data in Table 4.1, although including food consumed by young children over the age of 1 year, are likely to reflect predominantly foods consumed by infants of <1 year of age.

4.3 Due to the extremely small volume of minimally processed baby food on sale prior to 2002, data on the sales of this sector were not separately specified in the Mintel report (2002)[30]. Estimates of the current annual sales value of minimally processed baby foods are approximately £1.5M for frozen and <£1M for fresh with a 3-4 fold increase expected over the next 1-2 years (2005/6), especially in the frozen sector.

4.4 In general, baby milks are sold for consumption from birth to <12 months, whereas meals, finger foods and drinks are usually introduced at weaning from 4-6 months upwards.

Table 4.1: Baby food market (2002)*

Type	Sales (£M)	Market share (based on sales value, £M)	Market share (based on sales volume)x
Milk	175.8	46.1%	14.3%
Meals	170.1	44.6%	75.2%
Finger Foods	14.0	3.7%	3.9%
Drinks	21.2	5.6%	6.6%

* frozen and chilled minimally processed baby foods are not separately specified in this table due to their small sales volumes in 2002

x based on stock keeping units

Manufacturing and process safety controls for commercially produced baby foods

4.5 Baby foods are made from virtually any ingredient or combination of ingredients encompassing all of the main food groups e.g. meat, fish, poultry, cereal, dairy, fruit and vegetables. In most cases the ingredients for baby foods do not differ from those for adult food and are therefore subject to the same types and levels of microorganisms that would occur on these ingredients naturally. Spore forming bacteria including *C. botulinum* will occur in all of these ingredients from time to time.

4.6 Infant and baby foods must be manufactured in accordance with the legislative requirements as defined in Statutory Instruments 2003 and 1995[31,32]. Neither of these stipulates specific microbiological controls required for *C. botulinum* or other foodborne pathogens, outside of the generic requirements to ensure they are safe for consumption.

4.7 The safety of baby foods needs to be considered in the context of the processes applied to them and their likely effect on levels of C. *botulinum* spores in the foods. There are two main heat processes designed to achieve a 'safe' reduction in spores of C. *botulinum*. Proteolytic C. *botulinum* spores are 'destroyed' by a process of 121°C for 3 minutes, the $F_0$3 process or 'botulinum' cook. This 'destruction' process is designed to reduce the level of the spores by 12 log units and foods where this process has been applied are considered to be safe with regard to *C. botulinum* as the risk of the organism being present is reduced to an acceptable level. Non-proteolytic *C. botulinum* spores are less heat resistant and are 'destroyed' by a process of 90°C for 10 minutes. This process is designed to achieve only a 6 log reduction but, in the context of non-proteolytic *C. botulinum* and the foods in which it is a hazard, this is considered to reduce the risk of it being present to an acceptable level and foods processed in such a way are considered 'safe' (Table 4.2). The principle of a 6 log reduction being considered safe is consistent with the standards applied to foods for the safe reduction of a variety of other foodborne pathogens[33].

4.8 The majority of baby foods produced commercially are not processed under conditions designed to achieve the destruction of proteolytic *C. botulinum* spores. Processing usually incorporates a heating stage ranging from pasteurisation (70-80°C for seconds to minutes) to boiling for several minutes. Typical examples of low heat processed foods fed to infants include pasteurised milk (71.7°C for 15-25 seconds), yogurt/fromage frais (80-90°C for <1-5 minutes) and jarred/canned fruit purées (70-100°C for <1-20 minutes). None of these processes would achieve more than a 1 log reduction in levels of proteolytic *C. botulinum* spores and most would be similarly ineffective on the less heat resistant spores of non-proteolytic *C*. *botulinum*. Temperatures of 90°C for 10 minutes or equivalent processes would be necessary to destroy (6 log reduction) non-proteolytic C. *botulinum* spores.

4.9 Higher temperature processes include those used for infant formulae (pasteurised milk, followed by heat processing at 90-110°C for 1-2 minutes and spray drying at 90°C for 10-30 seconds), dried savoury infant meals (80-100°C for <10 minutes followed by drying at circa. 90°C for <1 minute). These processes may reduce (<1 log to >6 log) non-proteolytic *C. botulinum* spores but even the highest process (110°C for 1-2 minutes) would only reduce proteolytic *C. botulinum* spores by c.1 to 2 log units.

4.10 The reason why most foods i.e. those described in 4.8 and 4.9 do not receive a process sufficient to destroy (12 log reduction or more) proteolytic *C*. *botulinum* spores is because the foods do not support their growth i.e. they are dry, acidic or chilled. Historically, it has been recognised that whilst proteolytic *C. botulinum* may be present in a food, it is the potential for it to grow and produce toxin in the food that presents the risk of foodborne botulism to consumers. Therefore, only where a food may support the growth of proteolytic *C. botulinum* is a process applied to destroy (12 log reduction or more) the spores. The process is used for low acid, high moisture ambient meals (savoury canned and jarred wet meals). Alternatively, an ultra high temperature (UHT) process (>130°C for several seconds) may be applied as used in the manufacture of ambient stable, ready-to-feed infant milks. If it is a chilled food, where proteolytic *C. botulinum* cannot grow (they do not grow below temperatures of 10°C) but where non-proteolytic *C. botulinum* may be able to multiply, the controls as defined by the ACMSF in its report on vacuum packed and modified atmosphere packaged foods, are employed[33]. This can include a heat process of 90°C for 10 minutes, although it is important to note that this guidance was not specifically designed for minimally processed baby foods.

4.11 It is also important to recognise that the majority of commercially prepared foods are not produced under sterile packing/filling conditions and that processing is usually designed to minimise recontamination with microorganisms but not necessarily eliminate it, especially regarding spores (Table 4.2). Thus, some products such as milk, yogurt and fromage frais will be filled into containers in non sterile but clean conditions where the product flows in enclosed pipework to the filler head and the product is only exposed momentarily during the fill into the container and prior to sealing/lidding. The filling areas do not receive microbiologically filtered air and the areas/personnel are not subject to controls in place for high risk foods such as infant formulae (see below). Baked baby foods such as cereals, breadsticks, etc are usually cooled in non sterile, albeit clean environments applying good manufacturing practices (GMP) prior to filling and packing.

4.12 The highest standards of environmental hygiene are employed for the production of spray dried infant formulae. Infant formulae are spray dried and filled in high risk environments which are physically segregated from low risk areas of the factory. Personnel change into dedicated clothing before entry into these areas and the air is filtered to reduce inflow of high levels of microorganisms. These controls are principally designed to prevent the entry and recontamination with vegetative microorganisms, especially enteric pathogens such as *Salmonella* and *E. coli* and not necessarily spore formers such as *C. botulinum*. Roller dried infant powders such as savoury dried/powdered meals are manufactured under similar conditions although air filtration may not always be in place and the high risk area is subject to less control than for spray dried products.

4.13 Low acid, high moisture ambient meals (savoury canned and jarred wet meals) that receive an $F_0 3$ process are processed in pack and recognised controls exist to prevent re-entry of microorganisms during cooling. UHT processed products such as ready-to-feed infant milks are filled under aseptic conditions where packaging is sterilised prior to filling in enclosed sterile cabinets/rooms receiving microbiologically filtered air.

4.14 None of the processes currently employed for the production of infant foods, with the exception of those used for low acid, high moisture canned/jarred foods and UHT milks are designed to destroy (12 log reduction or more) proteolytic *C. botulinum* spores.

Infant food production in the home

4.15 A significant proportion of food consumed by infants under the age of 12 months is prepared in the home. A variety of advice exists on what to feed infants at different ages and how to prepare the food. This advice comes from official government websites (Food Standards Agency, Department of Health) and a plethora of infant and baby books. The Department of Health and the Food Standards Agency advise mothers to feed infants under 6 months of age breast milk or formula milk. From 6 months, mothers are

encouraged to begin weaning by introducing a more varied diet including particulate food such as fruit, vegetable and meat purées. Historically, mothers were advised to begin weaning from 4-6 months and many of the current recipe books for babies still recommend this practice. Finger foods such as bread, rusks, breadsticks, etc are also encouraged and by the time the infant is 8-9 months it is expected that most babies will have been introduced to solid foods e.g. pieces of fruit and bread.

4.16 Advice on cooking foods for infants does not vary significantly from that for adults. Recipe books for 4-6 month infant food suggest steaming or lightly boiling foods prior to making purées although in some cases no cooking is advised such as for soft fruit like bananas and peaches. Solid foods introduced from 6 months upwards include bread, raw fruit such as apples, pears and cooked meat, fish.

4.17 Food prepared in the home is unlikely to be sterilised and is not subjected to processes expected to destroy (12 log reduction or more) spores of proteolytic *C. botulinum*, although light boiling/steaming of foods for 4-6 month old infants may achieve a slight reduction (<1 log) in spores and would significantly reduce (>1 log) and possibly destroy (6 log reduction or more) non-proteolytic *C. botulinum* spores.

Manufacturing and process safety controls for minimally processed baby foods

4.18 The *Ad Hoc* Group received evidence from companies producing and selling minimally processed baby foods, most of whom would be considered small businesses. Products included savoury and fruit based meals that were sold either chilled or frozen to retailers (large and small), nurseries or local friends. Knowledge of the potential hazard of *C. botulinum* varied as did the associated process controls with some relying heavily on end product testing and others applying cooking processes varying from mild pasteurisation (70-80°C for minutes) to higher heat processes (boiling for several minutes). Finished products were in some cases designed to be eaten without any further heat processing (cooking) by the customer i.e. ready-to-eat whilst others had full cooking and cooling instructions designed to ensure the customer applied a re-cook sufficient to destroy vegetative microorganisms (but not spores of *C. botulinum*) prior to consumption. The manufacturing premises used to produce these foods again varied but ranged from kitchen scale to large food manufacturing facilities. Controls in place to prevent re-contamination of the cooked product included cooking in pack, hot filling (above 70°C), full high/low risk segregation or, in some cases, very limited separation of raw and cooked materials. The post cooking controls described in some operations gave cause for concern in relation to the potential for cross contamination with vegetative organisms such as enteric pathogens e.g. *Salmonella* and *E. coli*. This hazard particularly arises in premises handling raw meat as an ingredient where significant hazards are introduced into the production facility. Shelf lives were allocated to products

based predominantly on quality considerations. Products were targeted at a range of age groups starting as low as 4 months.

4.19 Processing conditions and associated shelf lives of chilled minimally processed baby foods did not, in general, take account of the potential risk associated with the presence and growth of non-proteolytic *C. botulinum* and the controls recommended by the ACMSF (1992)[33]. It is recognised that these may not be considered vacuum packaged or modified atmosphere packaged foods, but, if present in the finished product, non-proteolytic *C. botulinum* growth could present a risk in some of these foods.

4.20 Where manufacturers of these products had recognised the potential risk of *C. botulinum,* some had approached local authorities, private consultants and/or research establishments. In some cases they were advised that the potential risk could only be avoided by applying a full 'botulinum' cook (121°C, 3 minutes) whereas in other cases the controls in place i.e. pasteurisation were considered acceptable.

Infant feeding

4.21 In 2003, the number of live births recorded in the UK was 695,549. Studies have been conducted in the UK assessing infant feeding patterns over the first 9 months and these data provide a useful means of estimating previous and existing exposure of infants to different types of food.

4.22 In the infant feeding study of 2000[34] which included nearly 9500 mothers of babies born in that year, 70% breastfed their babies, indicating that 30% were fed infant formulae from birth. Forty-two percent of mothers were breastfeeding after six weeks and this number decreased to 21% at six months. Twenty four percent of mothers introduced solid food into the diet by three months, 85% by four months and virtually all had introduced solids by six months. In babies aged eight to nine months, cows' milk was the main milk drink for 8%, the secondary drink for 28% and was used to mix food for 48% of the babies. Of those mothers feeding solid foods, 38% were preparing the food at home when the baby was four to five months, 62% were using commercial baby food and 66% were using cereals including baby or adult types (Note: These figures do not add up to 100% as mothers use several types of food on each meal occasion). At eight to nine months of age, home produced food was being used by 70% although 52% were still using commercial baby foods and 78% cereals.

4.23 The significance of these findings relates to the fact that a high proportion of foods historically and presently consumed by infants and young children are done so without a process sufficient to destroy (>12 log reduction) or even significantly reduce (>1 log reduction) spores of proteolytic *C. botulinum*. It is therefore likely that spores of the organism will have been consumed in both commercially produced and home-made foods for many years.

Table 4.2: Typical baby food processing*

Product type	**Process times and temperatures**	**Effect on the reduction of proteolytic *C. botulinum* spores**	**Post process conditions (see text)**
Commercial products			
Infant formulae	Pasteurisation of milk 72°C, 15 seconds Further heat treatment 90 – 110 °C for 1 – 2 minutes , or an equivalent UHT process Spray drying (inlet air temperatures 180-250°C, exit air temperature 70-90°C, milk droplet temperature 80-90°C for 10-30 seconds)	Minimal (<1log)	High risk controls
Infant dried savoury powders	Pre-processing of bulk ingredients (80-100°C, <10minutes) Roller drying (drum temperature 140-160°C, product temperature c.90°C, <1 minute)	Minimal (<1log)	High care
UHT milk	135-150°C, 1-10 seconds	Destroyed (>12 log reduction)	Aseptic filling
Pasteurised milk	71.1°C, 15-25 seconds	None	Clean fill
Yogurt/Fromage Frais	Pasteurisation of milk/cream 80°C-90°C, 1 minute – 5 minutes Fruit purée (processed as ambient stable, acidified purées, (see below)	None	Clean fill
Baked finger foods (breadsticks, etc)	95-100°C, 10-20 minutes	Minimal (<1 log)	Good manufacturing practice
Ambient stable fruit purées	Peeling/washing. Heat process as follows; pH <3.7, <80°C-90°C for <1 minutes-5 minutes pH 3.7 – 4.2, 85°C-95°C for <1 minute-5 minutes pH 4.2 – <4.5, 95°C-100°C for 10-20 minutes	None to minimal (<1 log)	Cooked in pack
Ambient, low acid cans and jars	Peeling/washing where appropriate, Pre-cooking 70-100°C, 5-10 minutes; Retorting to 121°C for >3 minutes	Destroyed (>12 log reduction)	Cooked in pack
Home prepared food			
Puréed fruit, vegetables, meats	Peeling/washing Boiled or steamed (95-100°C, 1-10 minutes)	Minimal (<1log)	General home hygiene
Finger foods like sandwiches, apple slices, carrot batons	No heat processing, some peeling/washing	None to minimal (<1 log)	General home hygiene
Minimally processed bat			
Puréed fruit and vegetables	Peeling/washing where appropriate Cooking 70-95°C for minutes (5-15)	None to minimal (<1 log)	Some cook in pack; some hot fill (>70°C) and some fill <70°C
Savoury meat meals	Cooking 70-95°C for minutes (5-15)	None to minimal (<1 log)	Some cook in pack; some hot fill (>70°C) and some fill <70°C

* relevant generic processing conditions were verified as accurate by industry trade bodies

Estimation of numbers of foods consumed by infants that may have spores of *C. botulinum* present

4.24 Based on knowledge of the processing of commercial foods in Table 4.2, the size of the baby food market (Table 4.1) and data on infant feeding, it is possible to determine an approximate estimate of the number of foods that may have been consumed by infants over the years that were not subject to processes that would have destroyed (>12 log reduction) or significantly reduced (>1 log reduction) proteolytic *C. botulinum* spores. These foods may therefore have contained proteolytic *C. botulinum* and, in some cases, non-proteolytic *C. botulinum* spores (see Table 4.3). This estimate indicates that commercially prepared foods are used for approximately 665 million meals consumed by babies in the UK on an annual basis. Of this, 540 million meals are likely to have received a process not designed to destroy or significantly reduce the loading of proteolytic *C. botulinum* spores. Using a conservative estimate that 75% of the baby foods detailed in Table 4.1 are consumed by infants <12 months of age, this results in 405 million meals not processed to destroy or significantly reduce proteolytic *C. botulinum* spores. Finally, using an assumption that an equivalent quantity of home-prepared food is consumed by infants as commercially prepared food, it is likely that approximately 810 million meals are consumed annually by infants <12 months of age where the food has not received a process capable of destroying or significantly reducing proteolytic *C. botulinum* spores.

Available guidance to manufacturers, enforcement officers and consumers

4.25 Advice is generally available regarding infant feeding from government and agency websites including the Department of Health and the Food Standards Agency. However, with the exception of specific advice on the risk of infant botulism through honey consumption, there is little current advice regarding the potential risk presented from *C. botulinum*. Most advice regarding home cooking of foods for infants under the age of 12 months does not significantly differ from the normal food safety advice given to most members of the public i.e. avoiding cross contamination, cooking foods appropriately.

4.26 In the course of our investigations it is interesting to note that local authorities, when approached for advice on the risk of *C. botulinum* in minimally processed infant foods had come to different conclusions. In one instance, the risk was considered sufficiently high such that the producer was advised to avoid making such food for infants <12 months unless they were to apply a process of 121°C for 3 minutes (the 'botulinum' cook). A different local authority judged that given the fact that the processing of this type of food did not differ significantly from other foods already available or indeed made in the home for infants then these products could continue to be made.

Relative risk of infant botulism from baby foods and from minimally processed baby foods

4.27 Ingredients used for the production of minimally processed baby foods are not likely to differ from those used for all other baby foods and the incidence/levels of *C. botulinum* spores is likely to be the same. Processing methods prior to cooking i.e. washing/peeling do not differ and therefore the spore loading of the mixture prior to processing is likely to be the same. Heat processing conditions vary significantly between different products and manufacturers. It is evident that commercial food and home produced food in the most part are not processed in a way that will destroy or significantly reduce levels of proteolytic *C. botulinum* spores.

4.28 In general, commercial processes for fruit, meat and vegetable based baby foods and many home produced foods e.g. fruit, vegetable and meat purées intended for infants involve heat processing that will achieve either a reduction (>1 log) or complete destruction (6 log reduction) of non-proteolytic *C. botulinum*. The main exception to this are commercial dairy based products e.g. yogurts and pasteurised milk and some home prepared raw fruits e.g. pears, apples, fed during or after weaning. Non-proteolytic *C. botulinum* are likely to be less prevalent in dairy ingredients as they are more commonly associated with fish and meat.

4.29 Formulations and shelf lives of commercially produced baby foods are designed to prevent the growth of *C. botulinum* during their shelf life i.e. dry, low pH or short life. Home produced baby foods are usually consumed very quickly or they may be frozen and consumed at a later date. Minimally processed baby foods may be sold chilled or frozen (prior to defrosting/reheating). Minimally processed baby foods not receiving a process sufficient to destroy non-proteolytic *C. botulinum* spores offer the potential for any such spores present to grow thereby potentially exposing infants to higher levels depending on the shelf life. This is particularly relevant to chilled products but may also be relevant to frozen products if instructions allow extended chilled storage after defrost.

Conclusions

4.30 Based on a relative risk, chilled and frozen minimally processed baby foods do not seem to be at any greater risk of containing proteolytic *C. botulinum* than current commercially available and home produced food consumed by infants and an $F_0$3 process does not therefore seem warranted for the production of these products. An $F_0$3 process is applied when a food is formulated and stored under conditions that can support the growth of and toxin production by proteolytic *C. botulinum* and achieves the virtual elimination (a 12 log reduction) of spores in the finished product. As this process is not applied to other foods that are and have been consumed by infants safely over many years, based on a relative risk, it does not seem warranted to apply this process to minimally processed baby foods.

4.31 A high proportion of, but not all, foods for infants produced commercially and in the home have stages that would reduce (>1 log reduction) or destroy (>6 log reduction) non-proteolytic *C. botulinum* spores. A key difference between existing baby foods consumed by infants and minimally processed foods is that current foods do not in general provide opportunities for growth of non-proteolytic *C. botulinum*. Many minimally processed baby foods can support their growth when stored under chilled conditions. Notwithstanding the fact that current information indicates that infant botulism is predominantly associated with proteolytic *C*. *botulinum* and occasionally *C*. *baratii* and *C*. *butyricum*, the foodborne hazard presented by non-proteolytic *C. botulinum* needs to be effectively controlled.

4.32 Although this report has considered the risks associated with minimally processed baby foods and *C. botulinum*, the *Ad Hoc* Group received evidence that leads to believe that the nature of the production of these foods by some businesses, especially small companies with limited technical resources, offered opportunities for end product contamination with general foodborne pathogens such as *E. coli*, *Salmonella* and *Listeria monocytogenes* if not properly controlled.

Recommendations

4.33 The *Ad Hoc* Group recommends that any minimally processed chilled or frozen baby food intended for infants should have suitable controls in place to destroy non-proteolytic *C. botulinum* spores (6 log reduction) or prevent ANY growth during the shelf life of the product or, in the case of frozen products, after defrosting. For non pH controlled minimally processed baby foods, the best means to achieve safety with regard to non-proteolytic *C. botulinum* will be to apply a heat process equivalent to 90°C for 10 minutes. It is important to note that the *Ad Hoc* Group has not considered other foodborne pathogens in detail in this report and it is essential that any minimally processed food intended for infants has sufficient controls to eliminate other hazards or reduce them to an acceptable level. For example, a minimum cook of 70°C for 2 minutes will deliver at least a 6 log reduction in vegetative pathogens such as *E. coli*, *Salmonella* and *Listeria monocytogenes* whilst cooling to <5°C within 4-6 hours of cooking and then appropriate storage will prevent growth from surviving spores.

4.34 Procedures must be in place to prevent recontamination of minimally processed baby foods after heat processing, whether by *C. botulinum* or by other contaminants such as enteric organisms or *Listeria monocytogenes*. This is best achieved by cooking in pack or, in the case of vegetative organisms, by hot filling at temperatures above 70°C. Use of aseptic filling equipment or effective operation of high and low risk segregation in a production facility may also be means of achieving this goal.

4.35 It is general good practice for any prepared, ready to eat food that can support growth of microbial pathogens i.e. ready meals to receive a full re-cook by the consumer to achieve a minimum of 70°C for 2 minutes and the *Ad Hoc* Group sees no reason why this should not be equally applied to minimally processed baby foods of a similar nature. It is recognised however, that such cooking instructions on pack need to be carefully constructed to ensure the food is then properly cooled to avoid scalding.

4.36 A code of practice for the safe production of minimally processed baby foods should be developed to include recommended cooking and cooling processes, measures to prevent post process contamination such as in pack processing or aseptic filling, shelf life allocation and customer/storage instructions.

Table 4.3 Estimated UK consumption of unsterilised commercial foods by infants and babies

	Sales (Million units/year) (see 1)	Sales (£M/year)	Sales (% based on £ sales)	Estimated Sales (million units based on £ sales)	Sterilised (estimated %)	Unsterilised (estimated %)	Sterilised (estimated million units/year)	Unsterilised (estimated million units/year)	Sterilised (estimated million meals/year) (see 2)	Unsterilised (estimated million meals/year) (see 2)
Meals (see 2)	231	170.1								
Jars		92.6	54.43	125.75	70	30	88.02	37.72	88.02	37.72
Cereals (Dry)		41.2	24.22	55.95	0	100	0	55.95	0	55.95
Canned		23.8	13.99	32.32	100	0	32.32	0	32.32	0
Chilled		10	5.87	13.58	0	100	0	13.58	0	13.58
Other		2.5	1.46	3.39	100	0	3.39	0	3.39	0
Milk (see 3)	44	175.8								
Formulae		124.5	70.81	31.16	0	100	0	31.16	0	311.60
Follow-on		38.6	21.95	9.66	0	100	0	9.66	0	96.60
Ready to feed		7.5	4.26	1.87	100	0	1.87	0	1.87	0
Soya		5.2	2.95	1.30	0	100	0	1.30	0	13.01
Finger foods (see 4)	12	14								
Rusks		8.8	62.85	7.54	0	100	0	7.54	0	7.54
Breadsticks		2.2	15.71	1.88	0	100	0	1.88	0	1.88
Rice cakes		0.3	2.14	0.25	0	100	0	0.25	0	0.25
Other		2.7	19.28	2.31	0	100	0	2.31	0	2.31
							Total sterilised meals consumed (million units/year)		125.62	
							Total unsterilised meals consumed per year (million units/year)			540.48

1: Stock keeping units – this underestimates the actual number of units as a SKU may comprise a multi-pack
2: All units are one meal
3: All units (excluding RTF) are 10 meals
4: All units are one meal

Chapter 5

Enforcement

Enforcement of product safety controls

5.1 Local authorities (LAs) have a duty to enforce food hygiene legislation with the view to ensuring that food businesses comply with their legal responsibilities. They also have a duty to provide assistance and help to businesses to enable them to comply. This is partly to encourage a thriving business sector but mainly to ensure compliance with legislation and good practice amongst a sector of the food trade largely without technical expertise of its own.

5.2 The responsibility for compliance with food safety legislation rests with the business concerned. The current complexity of legislation relating to the manufacture and sale of food products means that interpretation is needed in relation to each product according to its ingredients and method of sale. Generally, if meat, fish, milk, shellfish and some other specific ingredients are used, the manufacturing process may require approval by the local authority. The method of marketing i.e. direct to the consumer or through a wholesaler, will also determine what legislation is applicable. In January 2006 new legislation will come into force that is likely to simplify this situation through the full implementation of Regulation (EC) No. 852/2004. The new legislation will require food business operators to put in place, implement and maintain a permanent procedure, or procedures, based on the HACCP principles.

5.3 In some instances, businesses will contact local authorities to ask for guidance, advice and approval of their processes and products. There is no way of knowing how many businesses are active that have not sought advice or assistance or registered with their local authority as required.

5.4 LAs would need a high level of confidence in the evidence available to enable them to institute legal proceedings in the event of a failure to comply with legislation. Larger authorities are likely to have specialist officers while in smaller or rural LAs the officers often have a wide range of duties with no particular specialism. Having little microbiological expertise “in-house” they would have to rely on external agencies, including Food Research establishments, food scientists or microbiologists or the Food Standards Agency, to provide support. There is currently a lack of clear and consistent advice upon which such decisions could be made.

Evidence from local authorities approached directly by processors

5.5 Three local authorities were involved in the examination and/or approval of manufacturing processes for these types of produce within their areas. Representatives of the three local authorities were invited to give evidence. Beforehand, they were provided with questions to assist their presentations. The purpose of the questions was to establish whether the current food control system was sufficient to control the risks posed by the small scale production and marketing of minimally processed infant foods.

5.6 All authorities that gave evidence realised that the effect of *C. botulinum* could be serious and recognised and accepted at the outset that their level of competence was insufficient to be able to give assistance or recommend approval. Advice was therefore sought from the Food Standards Agency and LACORS. The FSA responded suggesting that a case-by-case assessment needed to be undertaken but specific advice was not offered. The EHO concerned would need to decide whether to approve a non-proteolytic *C. botulinum* cook at 90°C for 10 minutes or to insist on a full botulinum cook. The advice suggested that, in view of the very low incidence of disease and despite the widespread existence of *C. botulinum* in the environment, commercially produced food need not be highly processed. The issue was ultimately one for the businesses to decide, as they are ultimately responsible for compliance.

5.7 All three LAs confirmed that they felt they lacked expertise in this issue when first approached by the food businesses. Neither the LAs nor the businesses were aware of the risk of infant botulism in connection with their products at the outset. EHOs would have been aware, through their training, about the hazard associated with *Clostridium botulinum*, its severity and the means of controlling food-borne intoxications by utilising a full botulinum cook. However, the hazard to infants through the consumption of a range of foods, following a less effective heat treatment process with the subsequent possible production of the toxin in the intestines, was largely unknown.

5.8 There had been no evidence that businesses had expected local authorities to take responsibility for compliance with legislation and the businesses concerned all understood that they had a legal and moral duty to ensure food safety.

5.9 Both LAs and businesses also approached Food Research establishments in an attempt to obtain authoritative guidance. LAs involved formulated differing advice to businesses on the basis of guidance received from the various sources. The businesses themselves also obtained advice from other sources, some of which could hardly be described as “expert”. Businesses were therefore in a difficult position, not knowing whether the food they sold would meet food safety requirements.

Involvement of other local authorities

5.10 Efforts were made to obtain a clearer picture of the size of the commercial market by asking for information from local authorities. A request about known businesses was made through the private food safety email system (Ehc-Net) to the convenors of all food safety enforcement liaison groups. A direct email was also sent to all local authorities through the private email system. An item requesting information was then published in the weekly professional journal sent by the Chartered Institute for Environmental Health direct to all its members. The volume of responses received was minimal (personal communication) but those that were received reinforced certain comments given in evidence to the Committee[35].

Conclusions

5.11 We concluded that LAs are not generally aware of the risks of infant botulism involved in the production of minimally processed infant foods. They did not have the expertise at hand to offer immediate advice but the LAs giving evidence were able to identify a potentially hazardous situation and sought guidance from those more qualified to provide it. Most advice and opinion was obtained through informal contacts between individual EHOs within different authorities. At best, however, there are inconsistencies in the level of awareness and approach within individual environmental health departments.

5.12 It is possible that there are small businesses producing minimally processed infant foods and that some of these are unknown to LAs. The existence of such operations is also unknown, in other words the food control system cannot currently be relied upon to be effective despite the best endeavours of individuals within LAs and businesses.

5.13 Evidence presented to the Group has suggested that the microbiological knowledge of some companies involved in the manufacture of infant weaning foods is patchy. Also, advice to support new businesses is lacking (for example poor HACCP use) and patchy knowledge of process controls exists.

Recommendations

5.14 We recommend that those providing LA training are made aware of the risks of infant botulism, can identify hazards associated with infant foods, and are aware of how to control the hazards.

5.15 We recommend that those seeking advice on risks posed by infant botulism need to assure themselves that those they consult are suitably qualified to provide that advice.

5.16 We recommend that existing advice on food safety management based on HACCP principles should be reiterated.

5.17 We recommend that there is a need for consistent guidance for EHOs to inform baby food manufacturers. Advice should also be applicable to industry and parents in the home.

5.18 We recommend that there is a need for key controls and good manufacturing practice to be observed during the manufacturing process.

Chapter 6

A risk assessment of infant botulism

Estimates from the literature of the minimum infectious dose

6.1 Proteolytic *Clostridium botulinum* has been identified as the agent responsible for most cases of infant botulism. Neurotoxigenic strains of *C. baratii* and *C. butyricum* have occasionally also been responsible for cases of infant botulism.

6.2 Information from the literature on the minimum infectious dose is extremely sparse. A commonly cited estimate is that the minimum infectious dose is 10-100 spores[12]. This estimate is based on reports that honey samples that have been associated with infant botulism contain 5-25 spores per g[36], and 5-70 spores per g[37]. Sugiyama *et al* estimated that the highest concentration of spores in honey not associated with infant botulism cases was 7 spores per 25g[38]. In experiments with infant mice, 10 spores were sufficient to lead to colonisation[10].

6.3 There is likely to be variability in the minimum infectious dose for different infants. For example, infant age, and whether breast or formulae-fed are likely to be important factors. Infants less than 16 weeks of age appear most susceptible to infant botulism (Table 6.1, Figure 6.1), and the susceptibility of infant mice peaked at 8-11 days[12]. In USA, the mean age for onset for formula-fed infants is 7.6 weeks, compared to 13.7 weeks for breast-fed babies[12].

6.4 Since infants less than 16 weeks of age do not consume substantial quantities of solid infant foods (section 4.22), it is reasonable to assume that such foods are not a major source of exposure in the case of infant botulism.

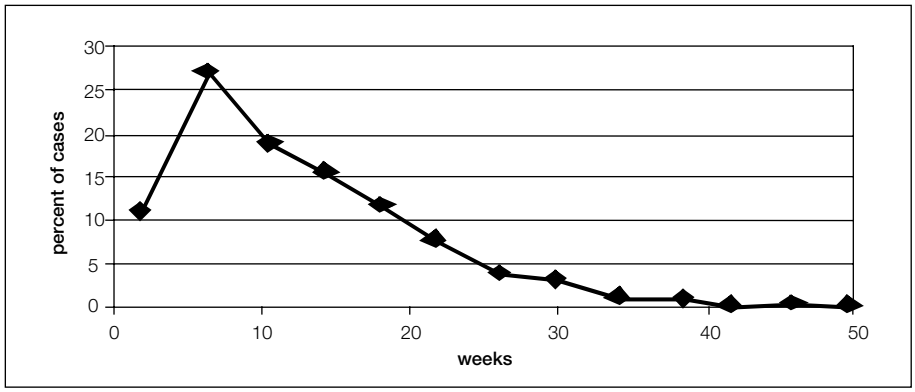
6.5 In view of the belief concerning the infectious dose, two different scenarios are considered, i.e. for packs containing ten or more spores, and for packs containing less than ten spores.

Table 6.1: Effect of age on distribution of infant botulism cases in Europe and USA[12,14]

Age of infants	Percent of cases in Europe	Percent of cases in USA
<16 weeks	77%	72%
16-32 weeks	21%	25%
>32 weeks	2%	3%

6.6 The data in Table 6.1 and Figure 6.1 are from 49 reported infant botulism cases in Europe (1978-2002) and 929 hospitalised infant botulism cases in USA (1976-1990).

Figure 6.1: The age distribution of infant botulism in the USA (Arnon, 1992)



Estimates from the literature of the number of spores of proteolytic *C. botulinum* per pack

6.7 Based on a review of the literature, the distribution of spores of proteolytic *C. botulinum* in packs of a typical "highly contaminated" infant food (prior to any processing) was estimated by a group of experts. It was agreed that a log-normal distribution should be used to reflect beliefs concerning the variable concentration of spores in infant food material[39]. The log-normal distribution has two parameters which are chosen as the mean concentration of spores in infant food materials and the coefficient of variation of the spore concentration. A BetaPERT distribution has been used to represent uncertainty concerning the mean of the spore concentration, and two extreme (point) values to represent uncertainty concerning the coefficient of variation. In turn, and based on a review of the literature, parameters of the BetaPERT distribution and of the two extreme values of the coefficient of variation were obtained from a set of experts.

6.8 In order to capture uncertainty for the mean value of the contamination of spores, the experts agreed that the modal value of the mean (most likely mean) was 0.3 spores per kg, that the minimum mean value was 0.001 spores per kg, and that the maximum mean value was 10 spores per kg. In order to consider uncertainty in the coefficient of variation, a low value (102%) and a high value (2706%) were adopted. These limits were derived from expert view of the shape of the variability of the spore concentration. From these considerations the distribution of the number of spores in 1kg was determined using simulation in Winbugs (http://www.mrc-bsu.cam.ac.uk/bugs/winbugs/contents.shtml). For the pack size of 121g that was considered, the probability that a pack will exceed the specified numbers of spores was determined by calculation based on a Poisson distribution (Table 6.2).

Table 6.2: Calculation of the probability that the number of spores of proteolytic C. *botulinum* in a pack (121g) will exceed the specified number

Number of spores per pack	Coefficient of variation	Probability that a pack will exceed specified number of spores	
		Most likely estimate	Upper 97.5% limit
0	Low (102%)	1×10^{-1}	3×10^{-1}
1		9×10^{-3}	4×10^{-2}
2		4×10^{-4}	4×10^{-3}
3		1×10^{-5}	3×10^{-4}
4		4×10^{-7}	2×10^{-5}
5		1×10^{-8}	9×10^{-7}
6		2×10^{-10}	4×10^{-8}
7		4×10^{-12}	1×10^{-9}
8		6×10^{-14}	5×10^{-11}
9		8×10^{-16}	1×10^{-12}
0	High (2706%)	2×10^{-1}	4×10^{-1}
1		1×10^{-2}	1×10^{-1}
2		8×10^{-4}	2×10^{-2}
3		3×10^{-5}	3×10^{-3}
4		1×10^{-6}	3×10^{-4}
5		3×10^{-8}	3×10^{-5}
6		8×10^{-10}	2×10^{-6}
7		2×10^{-11}	2×10^{-7}
8		3×10^{-13}	1×10^{-8}
9		6×10^{-15}	5×10^{-10}

Estimation of the risk presented by packs containing ten or more spores

6.9 From Table 6.2, for the more conservative (higher) coefficient of variation, the most likely estimate is a probability of 6×10^{-15} that a pack will contain ten or more spores, and if an upper 97.5% estimate of the contamination level is used, the probability that a pack contains ten or more spores is 5×10^{-10}. Furthermore, if it is assumed that (i) consuming ten or more spores in one meal will lead to infant botulism, and (ii) 10^8 packs are consumed annually (see Table 4.3), then this equates to a most likely estimate of there being 6×10^{-7} hazardous packs per year (or one pack every 2 million years), and a 2.5% chance that the rate at which hazardous packs appear is 5×10^{-2} hazardous packs per year (or one pack every 20 years). This is a low risk. Possible limitations of these estimates were identified, these include: (i) it is assumed that ten or more spores will always present a risk (a sparseness of data on the dose response precludes a more detailed estimate); (ii) the most likely estimate is of a probability of 6×10^{-15} that a

pack will contain ten or more spores while a literature review identified six studies (five in fish and one in mushrooms) reporting ten or more spores per 100g[40,41,42,43,44,45,46].

Estimation of the risk presented by packs containing less than ten spores

6.10 The literature data are too sparse to enable a meaningful estimate of whether less than ten spores might (or might not) be infectious for some infants, making a calculation of the risk difficult. However, the possible effect of these low concentrations of spores can still be considered. For example, if a concentration exceeding five spores per pack is considered an infectious dose for all infants (and the more conservative coefficient of variation is used), then the probability of a hazardous pack has a most likely estimate of 3×10^{-8}, and if an upper 97.5% estimate of the contamination level is used the probability that a pack contains more than five spores is 3×10^{-5} (Table 6.2). While if only 1% of infants are susceptible to a concentration of more than five spores per pack, then the probability of a hazardous pack has a most likely estimate of 3×10^{-10} and if an upper 97.5% estimate of the contamination level is used the probability is 3×10^{-7}. If only 0.01% of infants are susceptible then the probability of a hazardous pack has a most likely estimate of 3×10^{-12} and if an upper 97.5% estimate of the contamination level is used the probability is 3×10^{-9}. A lack of information on the dose-response relationship precludes identifying which of these scenarios is most likely.

Consideration of the risk based on sales of unsterilised baby foods since 1978

6.11 It is possible to estimate the number of foods that may have been consumed by infants over the years that were not subject to sterilisation and therefore may have contained spores of *C. botulinum*, based on knowledge of the processing of commercial foods, the size of the baby food market and data on infant feeding (see chapter 4). This estimate indicates that commercially prepared foods are used for approximately 6.6×10^8 meals consumed by infants in the UK on an annual basis. Of these, 5.4×10^8 meals are likely not to have received a 12D process for spores of *C. botulinum* (i.e. a botulinum cook for $F_0 3$). Using a conservative estimate that 75% of the baby foods detailed are consumed by infants <12 months of age, this results in 4.0×10^8 meals not receiving a 12D process for spores of *C. botulinum*. Finally, using an assumption that an equivalent quantity of home-prepared food is consumed by infants as commercially prepared food, it is likely that approximately 8.0×10^8 meals are consumed annually by infants <12 months of age where the food has not received a 12D process for *C. botulinum*. This equates to a total of approximately 2.1×10^{10} packs (meals) between 1978 and 2004.

6.12 The first case of infant botulism was reported in the UK in 1978, and a total of six confirmed cases were reported between 1978 and 2004. While only one of these cases has been weakly linked to a specific food, it can be calculated that if all had been associated with infant food that had not received a 12D process for C. botulinum, this would be one case per 3.5 x 10^9 packs. Although it is possible that some of the cases of SIDS may have been mis-diagnosed cases of infant botulism, it is also unlikely that all of the six cases could have been associated with food, based upon the evidence presented for those cases. In summary, the lack of cases associated with infant food not receiving a 12D process for C. botulinum, considered against the number of meals ingested over this period, suggests that the risk of infant botulism presented by consumption of these foods is low.

Consideration of the risk presented by addition of honey to infant foods

6.13 Honey has been identified as a source of *C. botulinum* spores, and has led to cases of infant botulism in Europe and the USA[12,40,14]. It has been reported that 29 out of 49 cases of infant botulism described in Europe were associated with a history of honey consumption[14]. Currently it is recommended that honey is not given to infants under 12 months of age[47,48].

6.14 Honey can be heavily contaminated with spores of *C. botulinum,* with some surveys reporting samples containing more than 10 spores/g[40]. Furthermore, in the USA, six samples of honey containing 5-25 spores/g have been associated with cases of infant botulism[36], and in Canada a sample of honey containing 1 spore/g was associated with infant botulism[16]. Since cases of infant botulism have been associated with consumption of very small amounts of honey (possibly a few grams), the *Ad Hoc* Group recommends that honey should not be added to foods specifically targeted at infants under 12 months of age (unless these foods receive a full botulinum cook or an equivalent process control). It is hoped that, in the future, as further information becomes available, a full assessment of the risk presented by addition of honey to infant foods can be carried out.

Conclusions

6.15 Approximately 75% cases of infant botulism occur in infants less than 16 weeks of age. Since infants of this age do not consume substantial quantities of solid infant foods, it is reasonable to assume that such foods are not a major source of exposure in the case of infant botulism.

6.16 The literature indicates that the minimum infectious dose for infant botulism is 10-100 spores of proteolytic *C. botulinum*. Based on a risk assessment, it is concluded that the risk of infant botulism from packs of food other than honey is low, as it is rare for packs to be contaminated with 10 or more spores. A lack of information on the dose-response relationship precludes estimation of the risk presented by packs containing less than 10 spores.

6.17 Cases of infant botulism have been associated with consumption of honey, and current advice from the Food Standards Agency and Department of Health is that honey should not be given to infants less than 12 months of age. Based on a consideration of the risk, the *Ad Hoc* Group concluded that honey should not be added to foods specifically targeted at infants under 12 months of age (unless these foods receive a full botulinum cook or an equivalent process control).

Recommendation

6.18 As further information becomes available, there would be merit in conducting an extended risk assessment. Further information that would enable such a risk assessment to be carried out in a more informed manner includes:

1) Better information on the contamination level of raw materials (including honey) with spores of proteolytic *C. botulinum.*

2) Better information on the dose response relationship. There should be a thorough follow up of any further cases of infant botulism that arise in the UK, including a consideration of all sources of exposure, including food.

6.19 Based on a consideration of the risk, the *Ad Hoc* Group recommends that honey should not be added to foods specifically targeted at infants under 12 months of age (unless these foods receive a full botulinum cook or an equivalent process control).

Chapter 7

Conclusions and Recommendations

Infant (Intestinal Colonisation) Botulism

7.1 Infant botulism is rare and there is no evidence of an association between development of illness and consumption of chilled and frozen baby foods anywhere in the world literature, at least not yet.

7.2 We recommend that clinicians are reminded of the possibility of infant botulism so that they may consider it earlier in the differential diagnosis of a child with compatible symptoms.

Sudden Infant Death Syndrome (SIDS) and *C. botulinum*

7.3 There are mixed views within the medical and research communities as to whether some cases of SIDS could be misdiagnosis of extreme forms of infant botulism. There are no UK data to contribute.

7.4 We are of the opinion that there is merit in assessing the link between infant botulism and SIDS further and recommend that research should be undertaken in the UK.

Minimally Processed Baby Foods

7.5 Based on a relative risk, chilled and frozen minimally processed baby foods do not seem to be at any greater risk of containing proteolytic *C*. *botulinum* than current commercially available and home produced food consumed by infants and an $F_0$3 process does not therefore seem warranted for the production of these products. An $F_0$3 process is applied when a food is formulated and stored under conditions that can support the growth of, and toxin production by, proteolytic *C. botulinum* and achieves the virtual elimination (a 12 log reduction) of spores in the finished product. As proteolytic *C. botulinum* spores are not reduced to this level in other foods that are and have been consumed by infants safely over many years, based on a relative risk, it does not seem warranted to apply this process to minimally processed baby foods.

7.6 A high proportion of, but not all, foods for infants produced commercially and in the home have stages that would reduce (>1 log reduction) or destroy (>6 log reduction) non-proteolytic *C. botulinum* spores. A key difference between existing baby foods consumed by infants and minimally processed foods is that current foods do not in general provide opportunities for growth of non-proteolytic *C. botulinum*. Many minimally processed baby foods can support their growth when stored under chilled

conditions. Notwithstanding the fact that current information indicates that infant botulism is predominantly associated with proteolytic *C. botulinum* and occasionally *C. baratii* and *C. butyricum*, the foodborne hazard presented by non-proteolytic *C. botulinum* needs to be effectively controlled.

7.7 Although this report has considered the risks associated with minimally processed baby foods and *C. botulinum*, the *Ad Hoc* Group received evidence that lead it to believe that the nature of the production of these foods by some businesses, especially small companies with limited technical resources, offered opportunities for end product contamination with general foodborne pathogens such as *E. coli*, *Salmonella*, and *Listeria monocytogenes* if not properly controlled.

7.8 The *Ad Hoc* Group recommends that any minimally processed chilled or frozen baby food intended for infants should have suitable controls in place to destroy non-proteolytic *C. botulinum* spores (6 log reduction) or prevent ANY growth during the shelf life of the product or, in the case of frozen products, after defrosting. For non pH controlled minimally processed baby foods, the best means to achieve safety with regard to non-proteolytic *C. botulinum* will be to apply a heat process equivalent to 90°C for 10 minutes. It is important to note that the *Ad Hoc* Group has not considered other foodborne pathogens in detail in this report and it is essential that any minimally processed food intended for infants has sufficient controls to eliminate other hazards or reduce them to an acceptable level. For example, a minimum cook of 70°C for 2 minutes will deliver at least a 6 log reduction in vegetative pathogens such as *E. coli*, *Salmonella* and *Listeria monocytogenes* whilst cooling to <5°C within 4-6 hours of cooking will prevent growth of surviving spores.

7.9 Procedures must be in place to prevent recontamination of minimally processed baby foods after heat processing, whether by *C. botulinum* or by other contaminants such as enteric organisms or *Listeria monocytogenes*. This is best achieved by cooking in pack or, in the case of vegetative organisms, by hot filling at temperatures above 70°C. Use of aseptic filling equipment or effective operation of high and low risk segregation in a production facility may also be a means of achieving this goal.

7.10 It is general good practice for any prepared, ready to eat food that can support growth of microbial pathogens i.e. ready meals to receive a full re-cook by the consumer to achieve a minimum of 70°C for 2 minutes and the *Ad Hoc* Group sees no reason why this should not be equally applied to minimally processed baby foods of a similar nature. It is recognised however, that such cooking instructions on pack need to be carefully constructed to ensure the food is then properly cooled to avoid scalding.

7.11 A code of practice for the safe production of minimally processed baby foods should be developed to include recommended cooking and cooling processes, measures to prevent post process contamination such as in pack processing or aseptic filling, shelf life allocation and customer cooking/storage instructions.

Enforcement

7.12 We inferred that LAs are not generally aware of the risks of infant botulism involved in the production of minimally processed infant foods. They did not have the expertise at hand to offer immediate advice but the LAs giving evidence were able to identify a potentially hazardous situation and sought guidance from those more qualified to provide it. Most advice and opinion was obtained through informal contacts between individual EHOs within different authorities. At best, however, there are inconsistencies in the level of awareness and approach within individual environmental health departments.

7.13 It is possible that there are small businesses producing minimally processed infant foods and that some of these are unknown to LAs. The existence of such operations is also unknown, in other words, the food control system cannot currently be relied upon to be effective despite the best endeavours of individuals within LAs and businesses.

7.14 Evidence presented to the Group has suggested that the microbiological knowledge of some companies involved in the manufacturer is patchy. Also, advice to support new businesses is lacking (for example poor HACCP use) and patchy knowledge of process controls exists.

7.15 We recommend that those providing LA training are made aware of the risks of infant botulism, can identify hazards associated with infant foods, and are aware of how to control the hazards.

7.16 We recommend that those seeking advice on risks posed by infant botulism need to assure themselves that those they consult are suitably qualified to provide that advice.

7.17 We recommend that existing advice on food safety management based on HACCP principles should be reiterated.

7.18 We recommend that there is a need for consistent guidance for EHOs to inform baby food manufacturers. Advice should also be applicable to industry and parents in the home.

7.19 We recommend that there is a need for key controls and good manufacturing practice to be observed during the manufacturing process.

A Risk Assessment of infant botulism

7.20 Approximately 75% cases of infant botulism occur in infants less than 16 weeks of age. Since infants of this age do not consume substantial quantities of solid infant foods, it is reasonable to assume that such foods are not a major source of exposure in the case of infant botulism.

7.21 The literature indicates that the minimum infectious dose for infant botulism is 10-100 spores of proteolytic *C. botulinum*. Based on a risk assessment, it is concluded that the risk of infant botulism from packs of food other than honey is low, as it is rare for packs to be contaminated with 10 or more spores. A lack of information on the dose-response relationship precludes estimation of the risk presented by packs containing less than 10 spores.

7.22 Cases of infant botulism have been associated with consumption of honey, and current advice from the Food Standards Agency and Department of Health is that honey should not be given to infants less than 12 months of age. Based on a consideration of the risk, the *Ad-Hoc* Group concluded that honey should not be added to foods specifically targeted at infants under 12 months of age (unless these foods receive a full botulinum cook or an equivalent process control).

7.23 As further information becomes available, there would be merit in conducting an extended risk assessment. Further information that would enable such a risk assessment to be carried out in a more informed manner includes:

1) **Better information on the contamination level of raw materials (including honey) with spores of proteolytic *C. botulinum.***

2) **Better information on the dose response relationship. There should be a thorough follow up of any further cases of infant botulism that arise in the UK, including a consideration of all sources of exposure, including food.**

7.24 Based on a consideration of the risk, the *Ad Hoc* Group recommends that honey should not be added to foods specifically targeted at infants under 12 months of age (unless these foods receive a full botulinum cook or an equivalent process control).

Annex 1

AD HOC GROUP ON INFANT BOTULISM

Terms of reference

To consider the potential human health risk associated with the consumption of chilled or frozen, puréed baby foods, particularly in relation to *Clostridium botulinum* and infant botulism, to inform the development of ACMSF advice to the Food Standards Agency

Chair

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Annex 2

List of companies/organisations who assisted the *Ad Hoc* Group

Presentations

Dr Peter McClure – Unilever plc

Little Gourmets Ltd

Fresh Daisy Babyfoods

Pots for Tots Ltd

Dr Moira Brett – Consultant

Dr Roy Betts – CCFRA

London Borough of Wandsworth

South Hams District Council

Suffolk Coastal District Council

Health Protection Agency – Dr Jim McLauchlin and Dr Nick Andrews

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Written evidence

Cavaghan & Gray

Infant and Dietetic Foods Association

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Risk Assessment

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Glossary of terms

D value	The time required, at a given temperature, to reduce the number of viable cells or spores of a given microorganism to 10% of the initial number.
12D process	Heating, at a given temperature, for a period equal to twelve times the D value at that temperature.
Proteolytic *C. botulinum*	*Clostridium botulinum* Group I. Drives energy by degrading proteins. A mesophilic spore-forming anaerobic bacterium. Associated with infant and foodborne botulism.
Non-proteolytic *C. botulinum*	*Clostridium botulinum* Group II. Derives energy by degrading sugars. A psychrotrophic spore-forming anaerobic bacterium. Associated with foodborne botulism.
C. baratii	*Clostridium baratii*. A mesophilic spore-forming anaerobic bacterium. Occasionally associated with infant and foodborne botulism.
C. butyricum	*Clostridium butyricum*. A mesophilic spore-forming anaerobic bacterium. Occasionally associated with infant and foodborne botulism.
Infant botulism	A form of botulism affecting infants less than 12 months of age (typically 4 to 26 weeks of age). It results from botulinum toxins produced in the gut following the germination and subsequent growth from ingested spores of proteolytic *C. botulinum, C. butyricum or C. baratii.*
Proteolytic	Splitting of proteins or peptides by the action of enzymes for example during the process of digestion.
Risk	A function of the probability of an adverse health effect and the severity of that effect, consequential to a hazard(s) in food.

Risk assessment	A scientifically based process consisting of the following steps: (i) hazard identification – The identification of biological, chemical, and physical agents capable of causing adverse health effects and which may be present in a particular food or group of foods. (ii) hazard characterisation – The qualitative and/or quantitative evaluation of the nature of the adverse health effects associated with the hazard. (iii) exposure assessment – The qualitative and/or quantitative evaluation of the likely intake of biological, chemical, and physical agents. (iv) risk characterisation – The process of determining the qualitative and/or quantitative estimation, including attendant uncertainties, of the probability of occurrence and severity of known or potential adverse health effects in a given population.
Sudden Infant Death Syndrome	The sudden death of any infant or young child which is unexpected by history, occurring in association with sleep and lacking explanation after post-mortem investigation.

Glossary of abbreviations

HACCP	Hazard Analysis Critical Control Point
PCR	Polymerase Chain Reaction
PFGE	Pulsed Field Gel Electrophoresis
RAPD	Randomly Amplified Polymorphic Deoxyribonucleic acid (DNA)

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