Botulism Risk from Post-Processing Contamination of Commercially Canned Foods in Metal Containers

NFPA/CMI CONTAINER INTEGRITY TASK FORCE, MICROBIOLOGICAL ASSESSMENT GROUP REPORT¹²³

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ABSTRACT

This report focuses on the potential public health risks of Clostridium botulinum from post-process contamination of commercially produced foods in metal containers. This review examines the environmental sources of C. botulinum, the effect of sanitizers in cannery cooling water and the botulism incidents involving U.S. canned foods. There is no evidence that leakage spoilage due to container defects is increasing. The post-processing contamination of commercially produced foods in metal containers by C. botulinum is a rare event which occurs randomly. Based on historical information, its probability of occurring is very small. This is a probability which compares well with the risk associated with the minimum acceptable thermal process of low-acid canned foods.

Largely as a result of the isolated botulism incidents involving canned salmon in 1978 and 1982, the Food and Drug Administration became increasingly concerned over the potential health risk of low-acid canned foods related to defective containers. At a meeting on July 19, 1982 with invited industry executives and technical management, FDA proposed a cooperative effort to deal with this issue. Specifically, Deputy Commissioner Mark


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³Dr. Paul Thompson suddenly and unexpectedly died on March 22, 1984. Paul will be remembered for his valued participation in the preparation of this report as well as his other activities on behalf of the industry. He will be missed by those who worked with him on this report as well as those who worked with him in his other activities.

Novitch posed three questions and action points: (a) Has there been an increase in the number of container defects over the past several years? (b) If the answer to the first question is yes, then we need to find the origin and cause. (c) Then we need to establish guidelines and tolerances to minimize the occurrence.

In response to the container integrity issue, the National Food Processors Association and the Can Manufacturers Institute formed a joint Task Force consisting of four working groups: (a) container defects, (b) quality assurance, (c) leak detection methodology, and (d) microbiological assessment. The Microbiological Assessment Committee was assigned to: (a) determine if there has been an increase in leakage spoilage in commercially processed foods in metal containers, (b) assess the public health risk of post-processing leakage, (c) recommend measures to minimize leaker spoilage. This report is focused on the public health risk of Clostridium botulinum in foods in metal containers. Conclusions not supported by validly published documents are based on knowledge, experience and best opinion of committee members.

ENVIRONMENTAL SOURCES OF CLOSTRIDIA, PARTICULARLY C. BOTULINUM

Distribution of C. botulinum in nature

Van Ermengem (118) was the first to isolate Bacillus botulinus, now called C. botulinum, from ham in 1897. He also tried repeatedly to isolate the organism from nature, but his efforts were unsuccessful. It was not until 1922 that Meyer and Dubovsky showed conclusively that the natural habitat of spores of C. botulinum is soil.

In California, Meyer and Dubovsky (75) obtained toxic cultures in nearly 30% of over 600 soil and plant specimens examined. Almost one-third of the toxic cultures could not be typed, but of those that were typable, 75% were type A, 22% type B, and 3% a combination of both type A and type B.

Meyer and Dubovsky (76) also examined more than 1,500 soil specimens collected from across the continental United States. Their classical study showed an apprec-
ibly higher incidence of \( C. \) botulinum in the western half of the United States as compared to the eastern half (23% versus 14.5%). Further, they demonstrated a predominance of type A in the West, and a predominance of type B in the East. Of soil samples from the West, 84% of the typable cultures were type A, while in the samples from the East, only 19% were type A.

The prevalence of \( C. \) botulinum type B in the central part of the United States was also demonstrated by Tanner and Dack (110). From 55 soil samples from Illinois and 17 samples from elsewhere in the Midwest, 7 cultures showed \( C. \) botulinum; all were confirmed as type B. In another survey of Illinois, Jones and Tanner (63) found \( C. \) botulinum in only 10 out of 604 soil specimens, but, again, all were type B.

Smith (101) investigated the relative numbers of facultative and obligately anaerobic bacteria in 21 soil samples. The samples were collected in Virginia, Washington, Idaho, South Dakota, Wyoming, Iowa and Costa Rica. The obligate anaerobes were all clostridia. Their numbers ranged from \( 2.7 \times 10^2 \) to \( 3.3 \times 10^6 / g \) as determined by the most probable number (MPN) method. \( C. \) botulinum type A was demonstrated in six soil samples and type B in one. In addition, \( C. \) botulinum types E and F were both recovered from a sample taken in the rain forest of the Washington Olympic peninsula. It is noteworthy that the type A and B isolates were associated with neutral to alkaline soils and soils with relatively low organic matter content (1.4%). In contrast, the type E and F isolates were associated with a soil sample showing pH 4.5 and having a comparatively high organic matter content (13.7%).

Gibbs and Freame (46), using an MPN method, found clostridial spore counts in six soil samples to range from \( 3.5 \times 10^3 \) to \( 1.3 \times 10^6 / g \). The clostridia represented 2-37% of the total spores counted. Thayer (111), investigating microbial population of the Texas prairie, found about \( 10^5 \) anaerobes/g of wet soil with practically no drop in population to a depth of 40 cm.

Fishborne botulism had long been recognized as a clinical malady in Russia, but the causative organism was not isolated until 1936 by Hazen (53). This new toxin type was called \( B. \) botulinus type E, in keeping with the taxonomic nomenclature in use at that time. In contrast to \( C. \) botulinum type A and proteolytic type B, type E was recognized as being nonproteolytic and having very heat-sensitive spores.

There was little interest in \( C. \) botulinum type E in the United States until the beginning of 1960. In that year, a botulism outbreak occurred involving commercially vacuum packed smoked ciscos that had been caught in Lake Michigan. In 1963, two more outbreaks occurred involving commercially handled products. These involved California canned tuna fish and vacuum packed smoked whitefish chubs that had been caught in Lake Michigan. As a result of these outbreaks, interest was greatly stimulated in the ecological distribution of type E in the United States, and particularly in the Great Lakes.

One of the early surveys of the Great Lakes showed that about 9% of the fish taken from the main body of Lake Michigan harbored type E in their intestinal tracts (20). Fifty-seven percent of the fish taken from Green Bay carried spores of type E (later studies showed as high as 90% of the fish caught in some areas in Green Bay were positive for \( C. \) botulinum type E). In contrast to the overall incidence of type E in Lake Michigan, only 1% of the fish sampled from Lake Superior and Lake Erie and 4% of those samples from Lake Huron harbored type E.

Two theories were proposed to explain the high incidence of spores of \( C. \) botulinum type E in Green Bay (21). One was the catchment basin concept, or the possibility that type E was being concentrated from runoff from the surrounding land mass. The other theory suggested active multiplication of type E in the areas of high incidence rather than passive accumulation. Type E was shown to be present in over 90% of all underwater soil specimens from the main body of Lake Michigan, but the organism was found less often in shoreline sediments. Furthermore, the incidence of type E from the upper reaches of rivers emptying into Green Bay and from the land mass surrounding these areas was comparatively low. From this, it was concluded that if the catchment basin concept was operative, a more even distribution of type E along the entire lengths of the river and land mass should have been found. Since this was not true, areas of high incidence of type E in Green Bay (from 100 to over 1,000 organisms/g) were believed to be due to active multiplication in the Bay itself.

Early studies of the ocean bottom sediment taken some distance from the coast (32) and soil samples (33) from Alaska did not reveal the presence of \( C. \) botulinum type E. It was, however, found in shore line samples (32). Recent studies have shown it to be present in 17/23 samples of soil from beaches in Northwest Alaska by the Arctic Circle (78). Subsequent studies have shown the organism to be widely distributed in Alaska (79) having been found in 5/12 beaches and the sediment samples from 6/8 locations. Interestingly, only 7/115 specimens from non-coastal areas produced the organism. \( C. \) botulinum type F has been isolated from a culture of gills and viscera of a sockeye salmon taken from the Columbia River (28). The salmon was taken about 20 miles upstream from the river's mouth. \( C. \) botulinum type E has been isolated from salmon (29,54,79) crabs (29) oysters (29), clams (29) other fish species and marine mammals (79) in Alaska and along the coast of Oregon and Washington. Of particular interest is the observation that the proportion of fish which yielded toxic cultures was higher in fish caught in the river, than in fish caught in the ocean.

The fact that \( C. \) botulinum type E has a definite predilection for a marine or an aquatic environment has been thoroughly documented. The organism is distributed in marine life and sediments along the Atlantic, Pacific and Gulf Coasts, as well as in ponds and fresh water lakes, besides the Great Lakes. Suffice it to say that \( C. \)
The intestinal contents of fish in most Northern areas of the world. It is not within the scope of this review to cite the voluminous literature concerning the worldwide distribution of type E. However, it seems appropriate here to mention the extensive survey by Kravchenko and Shishulina (66), who examined 4,242 soil samples and 103 water samples from five geographical regions in the U.S.S.R. About 10% of the soil specimens showed the presence of *C. botulinum*; only four water samples were positive for *C. botulinum*. Of the toxic cultures from soil, about 60% showed *C. botulinum* type E, 28% type B, 8% type A, and 2% type C. Type D was detected only once in a soil sample mixed with fishery scraps.

There seems to be little question that in the early *C. botulinum* surveys type E would not have been detected, as a heating step was an integral part of the isolation procedure. Of course, the reason that type E would not have been found is the very low order of heat resistance of its spores as compared to those of type A and proteolytic type B, as already mentioned. As Kravchenko and Shishulina showed, heating increased the rate of detection of *C. botulinum* type A almost 30% and of type B more than 14% over corresponding unheated samples. However, type E was never isolated from heated samples.

The Russian work also pointed out that type E was present only in soils along the sides of ponds. Soils rich in organic matter and with high moisture content were the most likely to exhibit spores of type E.

Besides adequate moisture and soils with high organic matter, another factor likely involved in the high incidence of type E in marine environments is its ability to grow at relatively low temperatures. The fact that nonproteolytic strains of *C. botulinum* type B and type F can also grow at low temperatures (35,36) might explain their existence in a marine environment as well.

Thus, while *C. botulinum* type E and nonproteolytic types B and F exist primarily in marine environments, most clostridia, in general, are soil organisms in origin. Their preference for soil is not specific and their numbers are not predictable in any given soil. However, the literature indicates that *C. botulinum* usually makes up a relatively small portion of the total anaerobe population of terrestrial soils and marine environments.

**Distribution of clostridia, particularly *C. botulinum*, in food**

Clostridia are ubiquitous in the soil (103), so it is not surprising that these organisms sometimes appear on or in fresh fruits and vegetables and meat, fish, and poultry. Gibbs and Freame (46) surveyed 20 food items ranging from fresh to frozen to prepared products. They found a range of 0 to 3,500 clostridial spores/g of food. Greenberg et al. (52) investigated the putrefactive anaerobe level in raw beef, pork and chicken at packing plants in the U.S. and Canada. They found a mean spore level of 2.5/g for chicken, 3.63/g for beef, and 3.03/g for pork. Steinkraus and Ayres (105) examined fresh, cured and processed pork trimmings for presence of putrefactive anaerobes. For the fresh pork trimmings, they found about 70% of the samples had putrefactive anaerobe spore counts of 1 or less/g. The maximum number recorded was 51/g. A similar result was obtained for cured pork trimmings with a maximum count of 43/g. About 75% of the processed pork trimmings had spore counts of 1 or less/g and the maximum number obtained was 4. They also did a limited study on fresh beef trimmings, which indicated an average spore count of 6.5/g of meat. This is only slightly higher than the values obtained by Greenberg et al. The difference might be accounted for by the different sampling locations which, for Greenberg et al., was in the packing plant, whereas Steinkraus and Ayres sampled at a retail outlet.

The incidence of *C. botulinum* in various commercially handled foods, according to the literature, is given in Table 1. It is apparent that the incidence can vary widely. Greenberg et al. (52) examined 624 beef, 656 pork and 1,078 chicken samples and found only one *C. botulinum* type C isolate. In contrast, Roberts and Smart (95) in England found 19 (73%) out of 26 samples from one lot of collar bacon contained *C. botulinum* type A. This study by Greenberg et al. (52) used direct colony isolation, whereas the study by Roberts and Smart (95) used enrichment procedures. It is possible that these differences in methodology would account for the higher incidence of *C. botulinum* in the English study. However, this methodology difference may not be the only factor responsible for such variations in the incidence figures.

Since clostridia are essentially soil organisms, their presence in foods cannot always be completely eliminated. Fortunately, the presence of *C. botulinum* in the soil is low so that its probability of being on or in products in large numbers is very low.

**Competitive growth of *C. botulinum***

In Georgia, Morse et al. (81) found *C. botulinum* in only 1 of 152 soil samples cultured. They were unable to recover toxic cultures even when they inoculated the soil with spores of *C. botulinum*. Sugiyama et al. (107) observed that samples of a river sediment were so inhibitory that up to one million spores of *C. botulinum* type E were needed to produce toxic cultures. Wentz et al. (122) also demonstrated inhibition of growth of *C. botulinum* type F in soil cultures of *Bacillus licheniformis*.

Smith (102) examined 31 soil samples for the presence of organisms capable of inhibiting growth and toxin production by strains of *C. botulinum* type A. Such organisms were found in eight samples of soil. Inhibiting strains of *B. perfringens* were found in five samples, *C. sporogenes* in three, and *Bacillus cereus* in three. Three of the *B. perfringens* strains produced an inhibitor effective on all 11 strains of type A against which they were tested, 7 of 8 proteolytic type B strains, 1 nonproteolytic type B strain, 5 of 9 type E strains and all 7 type F strains, whether proteolytic or nonproteolytic. They did not inhibit any of 26 type C strains, 6 type D strains...
and 24 *Clostridium sporogenes* strains. In mixed culture, an inhibitor strain of *C. perfringens* repressed growth and toxin production by a *C. botulinum* type A strain even though it was outnumbered by the latter about 40 times.

From 35 of 54 samples of mud, Graham (47) isolated 108 strains of bacteria which inhibited growth of *C. botulinum* type C, but did not denature preformed toxin. These strains fell into three groups: *Bacillus* sp. (73%), gram-positive non-sporing rods (11%), and gram-positive cocci (16%). Seven strains of *Bacillus* sp. were further investigated and found to produce antibiotics containing from 2 to 5 peptides.

Lactic acid bacteria in fermented foods inhibit growth of *C. botulinum* (97). Also, free fatty acids produced by *Brevibacterium linens* (49), nisin produced by *Streptococcus lactis* (50), boticin E produced by type E-like *C. botulinum* (37), and a tylosin-like compound produced by a *Moraxella* species (67) were shown to be inhibitory to *C. botulinum*.

Commercial raw milk inoculated with botulin spores remained non-toxic, which Edmondson et al. (34) attributed to the production of lactic acid. They also reported that cheese made from raw milk inoculated with *Streptococcus lactis* and a few spores of *C. botulinum* failed to become toxic.

Production of an antagonistic substance (boticin E) which is bacteriolytic for vegetative cells and bacteriostatic for spores of *C. botulinum* type E by organisms resembling type E, was reported by Kautter et al. (64). Of the clostridial species against which it had been tested, the spectrum of activity was limited to *C. botulinum* type E and, to a lesser extent, to *C. perfringens* and *C. acetobutylicum*; *C. botulinum* types A, B, and F were insensitive.

Anastasio et al. (2) reported boticin E to be sporostatic for all tested nonproteolytic *C. botulinum* (types B, E and F) strains. Proteolytic strains of *C. botulinum* (types A, B, and F) were resistant to boticin E.

Bacteriocin-like substances have also been found in cultures of *Clostridium sporogenes* by Betz and Anderson (17). To what extent these metabolic inhibitors play a role in competitive growth with *C. botulinum* in foods and in nature is largely unknown.

There are a few reports in the literature indicating that growth of certain organisms is stimulatory to growth of *C. botulinum*. *Salmonella typhimurium* and *C. perfringens* (82) and some lactic acid bacteria (1) have been reported to enhance the growth of *C. botulinum* by lowering the redox potential and supplying certain growth factors.

**Microbiology of canning cooling water**

Although many studies have been done on the quality of container cooling water, few have evaluated the water as to types of microorganisms present. Bean and Everett (15) reported on the taxonomy of 172 cultures of yellow-orange pigmented bacteria isolated from chlorinated can cooling waters or post-process can handling equipment. They found 132 (76.7%) were species of *Flavobacterium* and the remainder were species of *Xanthomonas, Bacillus*, *Corynebacterium*, or in the family *Enterobacteriaceae*.

Everton et al. (38) noted that the *Flavobacterium* isolates were resistant to germicidal action by virtue of their clumping. Clumping may have minimized the probability of entry through acceptable quality containers during container cooling, but these organisms could have gained access to containers during subsequent abusive container handling. They noted that spoilage did not occur with vegetable products, but did occur in milk-based products for reasons not fully explained by the authors.

Nambiar and Iyer (83) conducted a bacteriological survey of prawn canneries, examining for the presence of *Bacillus* spp. They found that most of the aerobic sporeformers were isolated from water samples, particularly the cooling water.

Can cooling water studies were started at the National Food Processors Association in 1976 (unpublished data). In the preliminary phase of the study, cooling systems in 5 canneries were surveyed and 87 cooling water samples were analyzed. In 1977, the second phase of the study was extended to cover 12 additional canneries from which 116 cooling water samples were collected; 72% of the water samples were from recirculated-type systems, whereas, the remainder of the samples were from single-pass systems. The aerobic plate count of 64% of the sam-

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**TABLE 1. Incidence of *C. botulinum* in various foods.**

<table>
<thead>
<tr>
<th>Food</th>
<th>No. positive/no. examined</th>
<th>% Positive</th>
<th>Toxin type(s)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fresh chickens</td>
<td>1/1078</td>
<td>0.1</td>
<td>C</td>
<td>52</td>
</tr>
<tr>
<td>Luncheon meat</td>
<td>1/73</td>
<td>1.4</td>
<td>A</td>
<td>109</td>
</tr>
<tr>
<td>Vacuum packed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>frankfurters</td>
<td>1/10</td>
<td>10</td>
<td>B</td>
<td>57</td>
</tr>
<tr>
<td>Smoked turkey</td>
<td>1/41</td>
<td>2.4</td>
<td>B</td>
<td>1</td>
</tr>
<tr>
<td>Cooked ham</td>
<td>5/100</td>
<td>5.0</td>
<td>A</td>
<td>1</td>
</tr>
<tr>
<td>Vacuum packed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>bacon (3)</td>
<td>6/110</td>
<td>5.4</td>
<td>3A; 3B</td>
<td>95</td>
</tr>
<tr>
<td>different lots</td>
<td>19/263</td>
<td>73</td>
<td>19A</td>
<td></td>
</tr>
<tr>
<td>Frozen spinach</td>
<td>6/50</td>
<td>12</td>
<td>A or B</td>
<td>56</td>
</tr>
</tbody>
</table>

*Only divalent A.B antitoxin was used in typing the toxic cultures.*
ples was in the range of less than 1 to 100/ml. Spores of aerobic mesophilic sporeformers were absent from 80% of the samples and the maximal count did not exceed 20/ml. Spores of anaerobic mesophilic sporeformers were only recovered from 5% of the samples and also at a very low concentration. In general, the anaerobic sporeformers showed a gradual increase with the general bacterial population when the counts exceeded 100/ml.

Put et al. (91) conducted a microbiological survey in 8 European canneries, 4 using chlorinated well water and 4 using chlorinated surface water. They found Streptococcus, Staphylococcus aureus, Bacillus spores and clostridial spores in the chlorinated well water. S. aureus was not found in the cooling water, but was found on the can handling equipment and in the double seam water, presumably due to the widespread practice of manually handling processed cans by canner workers in the plants surveyed. The canneries using chlorinated surface waters contained higher numbers of microorganisms as well as a greater variety, including Klebsiella sp., Pseudomonas aeruginosa and fairly large numbers of clostridial spores.

Graves et al. (48) examined 59 cooling water samples from 30 Midwestern canning plants. They found aerobic plate counts ranging from 2 to 320,000/ml, with a median of less than .03/ml for coliforms, enterococci and putrefactive anaerobic spores, a median of less than .01/ml for coagulase positive staphylococci and less than 1/ml for aerobic spores. They found no salmonella and no C. botulinum spores. Only one sample contained coagulase-positive staphylococci, and this water had no detectable germicide. Putrefactive anaerobic spores were found in 15.2% of the samples, with the count ranging from less than .03 to 9.3/ml, while aerobic spores were found in 42.4% of the samples, with the count ranging from less than 1 to 340/ml. As chlorine residuals became greater than 2 ppm, spores of putrefactive anaerobes were not detected.

Odlaug and Pflug (86) examined 171 cooling water samples from 17 food canning plants in Minnesota and Wisconsin. They found some influence of the kind of cooling system on the microbiological level in the cooling water. In general, hydrostatic cookers had the most microorganisms, followed by cooling cans, FMC Sterilizers, and in-retort-cooling with the least. Spores of anaerobes and aerobes were found in all systems with the mean for the anaerobes ranging from less than 0.1 to 0.5/ml, and the mean for the aerobes ranging from 0.2 to 2.3/ml for the various cooling systems.

Jacob (60) took 210 cooling water samples from 17 California canneries. He found that the mesophilic aerobic spore counts ranged from less than 1 to 20/ml, the anaerobic spore counts ranged from less than 1 to 4/ml, the staphylococci counts from less than 1 to 13/ml and the coliform counts from less than 1 to 950/ml. No coagulase-positive staphylococci were found. The aerobic sporeformers were present in about 4% of the samples. No anaerobic sporeformers were detected in waters with a residual chlorine concentration greater than 2 ppm.

The surveys indicate that a variety of microorganisms may be present in canning cooling waters, including spores of mesophilic anaerobes and aerobes. However, organisms are usually present in low numbers; their presence is dependent upon the source of the cooling water, the type of cooling system used, and the amount of effective germicide present.

**Distribution of leaker spoilage at the retail level**

An industry survey was made in 1967 and 1973 to determine the integrity of metal food containers with easy open ends. Out of almost 94,000 cans of various foods examined, 177 cans (0.19%) were leakers; 137 cans were microbiologically spoiled. No foodborne pathogens were recovered from the spoiled cans (8).

Pflug et al. (88) concluded that the examination of canned foods at the retail level for swells is a valid method to measure the performance of containers and food manufacuturing operations. They obtained swelled food cans which had been collected by two supermarket chains in the Minneapolis-St. Paul area over a 17-month period. From about 5 million cans inspected by store personnel, 1,104 swells were collected. Thus the overall swell rate was 20 per 100,000, or 0.02%. The incidence among the various food product categories ranged from 2.1 to 79.4 swells per 100,000 units. Major can defects were observed in 314 (28.4%) of the swelled cans, or about 0.006% of the total number of cans from the survey. The remaining 790 cans were examined microbiologically with the following results: (a) typical leaker spoilage, 86%; (b) typical underprocessing spoilage, 7%; (c) thermophilic spoilage, 1%; and (d) non-microbial swells, 6%. Based on vacuum testing and double seam measurements, the causes of leakage were determined as follows: (a) poor or questionable quality canner's end double seam, 51%; (b) leaks at locations other than the double seam, 26%; and (c) poor or questionable can manufacturer's end double seam, 4%.

In an extension of the work discussed above, Davidson and Pflug (31) determined the probable path of leakage in 764 of the 790 cans which had been examined microbiologically. They showed that the lap area at the side seam juncture was the point of greatest vulnerability. From can seam measurement data, insufficient seam tightness was found to be one of the primary double seam defects. By combining the seam measurement data with the vacuum leak test results, the investigators concluded that 81% of the swelled cans examined exhibited a potential for leakage.

Davidson et al. (30) determined the viability of organisms in swelled cans from their survey as well as the general kinds of microorganisms associated with the spoilage. Viable organisms were recovered from 47% of the 790 swelled containers examined; 91.6% were typical leaker spoilage organisms; 0.5% were thermophiles; and 7.9% were pure cultures of sporeforming organisms. Whether spoilage due to sporeformers can be attributed to underprocessing in instances where only a single container is involved is open to some question. Most labora-
tories which investigate canned food spoilage problems on a routine basis recognize that in some obvious leaker spoilage situations, occasional cans may show a pure culture of sporeforming organisms.

EFFECT OF SANITIZERS IN CANNERY COOLING WATER

A variety of antibacterial agents are known which reduce numbers of bacteria and endospores in cooling water. However, selection of the most effective sanitizer requires knowledge of the advantages and limitations of each. Relatively few disinfectants can be regarded as reliably sporicidal, readily applied and inexpensive. The following sections describe sanitizers that are presently in use for disinfection of canner cooling water.

Chlorine and chlorine compounds

Elemental chlorine (Cl₂), hypochlorites, chloramines, and chlorine dioxide (ClO₂) are all bactericidal and sporicidal to different degrees. But since approximately 15 times as much chloramine as free available chlorine is required to cause an effective bactericidal action (119), this group is not considered further.

Based on present usage, elemental chlorine may be the first choice for in-plant chlorination and cooling water treatment (42). This important commercial source of chlorine is supplied, by compressing and cooling it, as an amber liquid (1.5 times the density of water), for shipment in steel cylinders or tank cars (13). For industrial use where the daily requirements are less than 50 lb/24 h, the cylinders are recommended (89). Some advantages to use of elemental chlorine are: little to no effect on water pH level, purity, least cost on the basis of available chlorine, easy addition to cooling water, and ready control of concentration (42).

During the chlorination process, a certain portion of chlorine (or hypochlorite) reacts with organic and inorganic substances dissolved or suspended in water. When this initial demand is satisfied, a breakpoint is reached where any further additions of available chlorine result in a continual increase in free available chlorine in proportion to the dose (23). Breakpoint chlorination is applied to water as it enters a food plant to provide residuals of 2-7 ppm (42). Somers (104) reported a 4-5 ppm chlorine level was sufficient for normal operating conditions.

When elemental chlorine is added to water, its hydrolysis yields hydrochloric acid (HCl) and hypochlorous acid (HOCl). The HOCl is considered responsible for killing of organisms and spores (3). The disinfecting efficiency of chlorine decreases with increasing pH in parallel with the dissociation of HOCl (14). Even at low concentration, chlorine causes rapid and irreversible destruction of bacterial cell material, before formation of N-chloro derivatives within the cytoplasm (51,65). Thus inhibition of certain key enzymes is not the sole mechanism of killing. Friburg (43) demonstrated that very small amounts of chlorine cause leakage of nucleoproteins due to destructive permeability changes in the bacterial cell envelope.

Chlorine in solution is strongly affected by pH. Researchers Rideal et al. (92) and Johns (61) demonstrated that an increase in pH substantially reduced the biocidal activity of chlorine and a decrease in pH increased the activity. Spores of a mesophilic Bacillus species exposed to 100 ppm available chlorine at pH 8.2 were killed in the same time period as those exposed to a 1,000 ppm solution at pH 11.3 (25). More recently, Mercer and Somers (74) showed that a 15 ppm hypochlorite solution killed 99% of a test population of Bacillus macerans spores within 8.5 min at pH 6, but almost 42 min were required for a like kill at pH 8.

Temperature affects solubility and also biocidal efficiency of chlorine. Gaseous chlorine dissolved in water is depleted as temperature is increased, the solubility decreasing from 1.46% (w/w) at 0°C to 0.12% at 90°C, albeit, the lethal rate approximately doubles for each 10°C hike in temperature, in the temperature range of 5-50°C (117).

Bacterial vegetative cells are sensitive to free available chlorine at ambient temperatures so that they are readily killed. For example, Escherichia coli exposed to 0.055 ppm available chlorine at pH 7 was killed in 1 min while vegetative cells of C. botulinum, type A exposed to 0.5 ppm available chlorine at pH 7 were killed in 30 s (117). Endospores are more resistant; spores of C. botulinum, NFPA strains 62A, 213B, and Saratoga E require an exposure of 4-6 min to 4.5 ppm free available chlorine at pH 6.5 and 25°C for a 99.99% lethal result (58). Odlaugh and Pflug (85) reviewed the sporicidal effects of chlorine, analyzing data from several reports. They concluded that Bacillus spores are more resistant to chlorine than Clostridium spores and that numbers of C. botulinum and other sporeforming organisms will depend on water quality, pH, temperature and chlorine level. They anticipated that resistant Bacillus spores would predominate in cooling water where free available chlorine was maintained at 2-5 ppm in the pH range of 7-7.5.

Hypochlorites

Calcium and sodium hypochlorite salts [Ca(OCl)₂ and NaOCl] are extensively used for chlorination of industrial water. NaOCl is sold as a liquid solution while Ca(OCl)₂ is supplied as a solid. High test Ca(OCl)₂ provides up to 70% total available chlorine but is often diluted with Na₂CO₃ to yield useful concentrations such as 65%, 50%, 15%, and other percentages. NaOCl is provided for household use at about 5.25% and for industrial use at 10-18% total available chlorine (42).

The hypochlorites ionize in water to give alkaline solutions of the cations and the oxychloride ions which undergo hydrolysis to form some HOCl. Fair et al. (39) and Morris (80) calculated the relative lethality of HOCl and OCI⁻ to E. coli at various pH levels. The oxychloride ion was 1/80 as potent as HOCl under the test conditions. While OCI⁻ is considered bactericidal its mechanism of action remains obscure. According to Rudolph and Levine (96), the active germicidal agent permeates the bacterial cell barriers to form N-chloro compounds.
through reactions with nitrogenous elements of the cytoplasm that prove lethal to the organism.

The hypochlorites exercise lethal effects in response to pH in the same manner as do chlorine-water solutions, i.e., decreasing pH increases lethality. Rudolph and Levine (96) measured the time required to kill 99% of a Bacillus spore population at 25 ppm available chlorine solution. At pH 12.86, 465 min were required; at pH 10, 131 min; pH 9.35, 35.5 min; pH 9, 19.5 min; pH 8, 5 min; and pH 6.25 min. They attributed the lethality differences to increases in the HOCI concentration as the pH was lowered.

Weber and Levine (120), among others, measured the effect of temperature on the bactericidal action of hypochlorites. At 25 ppm available chlorine and three different pH levels, a rise of 10°C produced a reduction of 50-60% in kill time, whereas, a drop of 10°C increased the required exposure two-fold. Temperature coefficients were only slightly affected by pH. Temperature effects were especially noticeable at pH levels above 8.5 and also when chlorine residuals were low (0.02-0.03 ppm, Butterfield et al. 24). When either sodium or calcium hypochlorite is added to water, some free alkali results so that the pH is increased. Consequently, the amount of HOCI formed is diminished and bactericidal potency is lessened as seen in Table 2 (89).

In comparison to gaseous chlorine, some disadvantages of hypochlorites in container cooling water are evident: pH of water may become more alkaline thus reducing biocidal activity, less available chlorine per unit weight, disposal of the water containing increased salts concentration and difficulties in application and controlling the concentration (42).

Small amounts of organic matter in cooling water reduce the free residual chlorine, and this effect is most striking in solutions of low chlorine content. Mercer (73) demonstrated that an initial 5 ppm of free chlorine in water was reduced to 3.45 ppm by addition of 1 ml tomato juice for each liter of water. When added organic matter contains proteins, free chlorine reacts to form chloramines so that some antibacterial activity is retained even though the available chlorine levels are reduced. This explains some anomalous findings reported in early literature (71,114), wherein killing of cells and spores occurred in the absence of measurable free available chlorine. Single-pass and recycled cooling water, properly filtered or otherwise treated, should not contain organic material, dissolved or suspended at levels that would drastically reduce available free chlorine or the availability of any other disinfectants that react with organic matter.

**Chlorine dioxide**

Chlorine dioxide is used mainly for bleaching pulp in the manufacture of kraft paper, but has also found use as a bleaching agent in the textile industry. A relatively small application is needed to treat potable water for taste and odor control and to disinfect potable water (108).

Chlorine dioxide is a yellow-green to orange gas at room temperature and has an irritating odor resembling chlorine gas. It cannot be shipped in bulk due to its instability when compressed to a liquid, but is handled and shipped safely in a concentration of less than 10% by diluting with gas phase air or nitrogen. Most commonly, ClO₂ is generated on-site. For water treatment applications, sodium chlorite and chlorine are combined in aqueous solution to yield ClO₂ and sodium chloride (39).

Chlorine dioxide reacts through oxidation, not by substitution. Ingols and Ridenour (55) showed its oxidation capacity in terms of available chlorine to be about 2.5 times that of chlorine. The degree to which the oxidizing capacity is exercised depends on pH and the type of reducing agents present in the system (69). At high pH values, ClO₂ reduces to chlorite ion using only 20% of its oxidizing capacity. On the acid side, ClO₂ is reduced to chlorine ion using all of its oxidizing capacity. Between pH 7-10, the nature of the reactants plays the major role in how much oxidizing capacity of chlorine dioxide will be used. Chlorine dioxide differs from chlorine in how it reacts. Through substitution, chlorine forms chloramine with ammonia and amines. With phenols, chloro-derivatives are formed. Oxidation by-products and chloro-derivatives are formed with most other organic structures. Chlorine dioxide reacts with phenols to oxidize them and reacts not at all with ammonia and amines. Neither does it react with hydrocarbons, even unsaturated ones. It appears chlorine dioxide will maintain a residual in the presence of organic matter for longer periods than chlorine (121). This may be its major advantage over other chlorine compounds: in water systems or contaminated water containing substantial organic matter, the chlorine dioxide demand will be generally less than the chlorine demand of the same systems.

The bactericidal activity of chlorine dioxide diminishes with lowered temperature, according to Ridenour and Ingols (93). As previously seen, this behavior resembles that of the hypochlorites and gaseous chlorine.

Chlorine dioxide is similar to gaseous chlorine in its germicidal and sporicidal efficiency (115). A 0.1 ppm residual (as measured with 0-toluidine-arsenite) will destroy bacterial cells and a 0.25 ppm residual is sporicidal (93). Another study (94) demonstrated greater sporicidal activity for chlorine dioxide than chlorine at comparable 0-toluidine-arsenite residuals. The authors attribute this greater sporicidal activity to the fact that the 0-toluidine-arsenite residue only measured on-fifth of the chlorine dioxide potential valance change. Thus the full oxidizing

---

**Table 2. Lethality of hypochlorites and gaseous chlorine in clear water, pH 7.2.**

<table>
<thead>
<tr>
<th>Chlorine compound</th>
<th>Total residual chlorine (ppm)</th>
<th>pH</th>
<th>Time (min) to kill 99.9% of cells (asporogenous yeast)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cl₂</td>
<td>5</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Ca(OCl)₂</td>
<td>5</td>
<td>7.4</td>
<td>2</td>
</tr>
<tr>
<td>NaOCl</td>
<td>5</td>
<td>7.6</td>
<td>2.5</td>
</tr>
</tbody>
</table>
potential of the chlorine dioxide is not measured. The difference could be the reason for the greater activity.

**Iodine and iodine compounds**

Elemental iodine is the only halogen which is a solid at room temperature. It is sparingly soluble in water but very soluble in aqueous solutions containing iodides. Iodine is bactericidal and sporicidal over a wide pH range against a broad spectrum of microorganisms (45). The free element itself is the lethal agent, unlike chlorine or hypochlorites (19). Iodine, even at high dilution, kills quickly in water. Salle and Catlin (98) reported the highest dilution killing within 1 min at a concentration of 0.2 ppm was the same as for 10 min, i.e., killing was immediate rather than by a prolonged period of stasis.

Iodine is active against microorganisms and spores because of its oxidizing power but may cause lethal effects through substitution or addition. Free iodine in solution, i.e., I₂, is the disinfecting agent. Brandrick et al. (22) studied the reaction of radio-tagged iodine on *E. coli* cells. They found 90% of the iodine absorbed by the cells could be recovered as iodide ion in the suspending menstrum. This was taken as indicating that an oxidative reaction had occurred. But the retention of 10% iodine suggested addition or substitution may also have occurred.

Iodine in water of acid to slightly alkaline pH is most active bactericidally. At strongly alkaline pH, it exists as the iodate, iodide or triiodide ions, which are not actively lethal (117). Activity is accelerated at higher temperatures but the temperature coefficient is relatively low compared with most other bactericides. Thus iodine is more bactericidally effective than other agents at lower temperatures, according to Wilson et al. (124).

On a basis of available iodine in solution, some comparisons with hypochlorite have indicated that iodine is several times more effective than chlorine. Factors are quoted of a three- to eight-fold magnitude (41). Under use conditions, such as in disinfection of swimming pool water, iodine has been found more effective than chlorine against coliforms, fecal streptococci, enterococci and staphylococci from human sources (40).

In contrast to the findings with vegetative cells, sporidial activity of iodine is less than that of hypochlorite. To cite only one comparative study, Cousins and Allan (27) found that 800 ppm of iodine at pH 2 killed no *B. subtilis* spores (in suspension) during a 4-h exposure but only 100 ppm available Cl₂ from sodium hypochlorite killed them all in 1 h at pH 8.

**Iodophors**

The combination of iodine and a solubilizing agent or carrier, most frequently a surfactant which releases free iodine at a low rate when placed in water, is called an iodophor. Since the active agent released from the iodophor is iodine, conditions such as pH and temperature affect it in like manner, i.e., killing activity is increased with either increasing temperature or decreasing pH, within limits. At use concentrations, iodophors have as broad an antimicrobial spectrum as free iodine (68). Some reports, such as that of Blatt and Maloney (18), conclude that aqueous and alcoholic solutions of elemental iodine are as potent as commercially available iodophors and that the desirable properties of iodophors are essentially those of weak iodine solutions.

**Comparison of iodine and iodophors for cooling water treatment**

Jacob (60) reported that practical experience with iodophors for disinfecting container cooling water is extremely limited. He cited some shortcomings of iodophors in the few cooling systems that employed them in his study sample: (a) the drop in pH of 2.9-4.8 units in the cooling water due to the phosphoric acid content of the iodophor, and (b) the increase in chemical oxygen demand from the range of 0.281 ppm to 470-969 ppm generated by the organic surfactant content of the iodophor.

Iodophors had not been considered for the specific use of disinfecting can cooling water until the early 70s, thus practical experience is limited. Iodophor-treated water was used for can cooling and in-can sealing operations in a 1971 trial (99) with the result that bright clean cans free of water spots were obtained. These results were confirmed in a later study (100) at a different plant. A 0.1-0.3 ppm concentration of available iodine was maintained at the discharge exit of the can cooler. No microbiological data were given relative to spores of mesophilic aerobes and anaerobes surviving the treatment.

Odlaug and Pflug (86) reported that seven plants in their survey used chlorine and iodine in cooling water of FMC Sterilmatic continuous processing equipment. They found no relationship between the anaerobic spore count and the iodine or chlorine concentration. In 192 Sterilmatic systems where chlorine was used in the cooling water, anaerobic spore loads ranged from 0.01-2.8 ml. In 12 Sterilmatic systems where chlorine and iodine were used, spore loads ranged from less than 0.08-2.8 ml. These results, obtained from practical applications in the processing plant, do not support laboratory evidence (19) that addition of small amounts of iodine to chlorine enhances its lethality.

Graves et al. (48) recovered less than 0.03 putrefactive anaerobes/ml (MPN) from cooling water containing 1.2-5.0 ppm free residual iodine. At equivalent free residuals, chlorinated cooling water at other plants contained in the range of less than 0.23-0.3 putrefactive anaerobes with a median finding of less than 0.03/ml (MPN).

**Comparison of chlorine and chlorine compounds for cooling water treatment**

Depending on the type of processing equipment, water pH, amount of organic matter and the cooling system employed, different chlorine compounds are chosen. The agent of choice is gaseous chlorine, and it enjoys wide use. Hypochlorites find use in cooling canals and open coolers where they may be metered in with a solution feed pump or a drop feed device. However, drip feed
equipment may be clogged by carbonate deposits which form from calcium hypochlorite. Hence it is advisable to mix calcium hypochlorite solutions a few hours before use to permit sedimenting of carbonate deposits.

Put et al. (90) and Williams (123) agree that sanitization sufficient to reduce cooling water loads to less than 100 colony forming units per ml provides adequate protection against recontamination. However, concern has developed that the anaerobic spore load in cooling water may be an indicator of a public health hazard from *C. botulinum* (86). There are no published findings of *C. botulinum* in domestic can cooling water. Graves et al. (48) found no *C. botulinum* in 59 samples of cooling water from 30 canneries. Putrefactive anaerobe spores ranged from less than 0.03-9.3; the most common (median) value was less than 0.3 (MPN)/ml. Jacob (60) found that samples of can cooling water contained anaerobic spores in low concentrations, but tests for *C. botulinum* were negative. The 171 samples taken by Odlauq and Pflug (86) were negative for *C. botulinum* though the anaerobic spore populations of the cooling water ranged from less than 0.1-5.9/ml. Thompson and Griffith (112) examined 274 samples of recycled container cooling water taken over a 27-month period at one western cannery. Twenty-eight samples contained anaerobic spores ranging in number from 0.04-4.6/ml (MPN). All isolates were fully characterized and identified as *Clostridium* species. Eleven percent of isolates could not be matched with named species; 89% were identified to species level. *C. botulinum* was not detected.

If chlorination of water in cooling canals or still retorts or other systems allowing greater than a 1-min chlorine contact time is applied to the point that sterility is achieved, residual chlorine levels of approximately 20 ppm are required (124). Some corrosive effects might result. Disposal problems, irritation to personnel and heightened costs should be expected.

An approach more practical than sterilization of cooling water is suggested by investigations of Ito et al. (58). They studied reduction in spore viability of *C. botulinum* types A, B, and E, through application of chlorine gas or calcium hypochlorite to achieve a residual of 4.5 ppm. The pH was buffered at 6.5 and a temperature of 25°C was maintained. Between 3.8 and 8 min were required to cause a 4-log cycle reduction. They also noted the well-documented pH and temperature effects on death rate kinetics. Odlauq and Pflug (85) plotted the data from Ito et al. for *C. botulinum* type A and type E. They observed that 2 ppm of HOCl reduced a population of *C. botulinum* spores by 90% in 2-3 min. Thus it was anticipated from these data and other literature that maintenance of free chlorine levels of 2.5 ppm at a pH of 7.7-5 gives reasonably good sanitation control of cooling water.

**Recycled water**

Reclaimed water that is cooled in a cooling tower may become highly contaminated with microorganisms and rechlorination may be necessary. A free residual of at least 0.5 ppm should be achieved in water flowing from the tower for use in the plant. Screening and filtering should accompany the chlorination to reduce chlorine demand. The cooling tower should be directly chlorinated at 4.5 ppm for a few hours every week or two to eliminate any microbial buildup (89). An NFPA recommendation (84) for recirculated cooling water also calls for 0.5 ppm free chlorine residual. Ito and Seeger (59) recommended that “periodic high level application of germicides is needed to remove microorganisms buildup.” Put et al. (91) listed several recommendations to ensure wholesomeness and safety of canned foods, among them, the correct chlorination of water to a residual level of 1-2 mg of free chlorine/L measured in the water drained from the can seam after cooling.

Olivant and Shapton (87) believed that counts of less than 10 bacteria/ml can be achieved in cooling water by maintaining 1-2 ppm of free available chlorine at all times. They emphasized that satisfactory control of leaker spoilage depends on careful design, construction and operation of the chlorination (or other sanitizing) equipment.

It is clear that appropriate sanitation of cannery cooling water provides an effective way to minimize the incidence of leaker spoilage. Experience has also shown that an equally important consideration is the avoidance of filled container abuse especially after processing while the can double seams are still wet (16,116).

**BOTULISM INVOLVING U.S. CANNED FOODS IN METAL CONTAINERS**

*Outbreaks involving human illness*

To properly focus on the subject of human botulism caused by container leakage, it is appropriate to first examine the safety record of the U.S. canning industry, especially since 1940. The year 1940 is a logical starting point for good reasons. First, *C. botulinum* type E, which has been the only toxin type implicated in outbreaks involving container leakage, was not recognized as an etiologic agent until 1936. Furthermore, reliable data are available on the number of botulism outbreaks involving commercially processed foods from about 1940 and reasonably accurate estimates of the number of low acid foods produced since that time can be obtained from the Can Manufacturers Institute.

Table 3 summarizes all known human botulism outbreaks involving commercially canned foods in metal containers since 1940 to the present.

There have been only four human botulism outbreaks wherein the causative factor is alleged to be or accepted as leakage. These outbreaks involved a mushroom sauce in 1941, tuna fish in 1963, and salmon in 1978 and 1982. In 1941, a can of mushroom sauce was suspected to be a leaker, but this could not be proven (77). Leakage may have seemed more likely because the thermal process was judged to be adequate and the low heat resistance of spores of *C. botulinum* type E was known. A fact apparently not considered was that the mushrooms had been dried, canned and imported from Yugoslavia.

<table>
<thead>
<tr>
<th>Year</th>
<th>Product Description</th>
<th>Outbreaks</th>
<th>Casesb</th>
<th>Death</th>
<th>Toxin Type</th>
<th>Cause of Outbreak</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1941</td>
<td>Mushroom sauce (single can)</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>E</td>
<td>Suspected leakage</td>
<td>44,77</td>
</tr>
<tr>
<td>1963</td>
<td>Tuna fish</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>E</td>
<td>Alleged leakagec</td>
<td>62,106</td>
</tr>
<tr>
<td>1971</td>
<td>Vichyssoise soup</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>A</td>
<td>Under-processingd</td>
<td>7</td>
</tr>
<tr>
<td>1974</td>
<td>Beef stew (single can)</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>A</td>
<td>Unknown - can possibly missed retort</td>
<td>7</td>
</tr>
<tr>
<td>1978</td>
<td>Salmon (single can)</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>E</td>
<td>Leakage - can damaged after processing</td>
<td>9,10</td>
</tr>
<tr>
<td>1982</td>
<td>Salmon (single can)</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>E</td>
<td>Leakage - malfunctioning can reformer</td>
<td>13,113</td>
</tr>
<tr>
<td>1982</td>
<td>Peeled whole tomatoes (single can)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>A</td>
<td>Unknown - no evidence of container leakage</td>
<td>12</td>
</tr>
</tbody>
</table>

aNo botulism outbreak is recorded from 1926 to 1939 in commercially canned foods in metal containers (see Reference 77).
bNumber of persons afflicted.
c21 additional can recovered by FDA reportedly showed C. botulinum; container evaluation was not definitive.
dFour additional cans (swollen) of the same code showed type A toxin.

(44). If spores of the botulinal organism involved originated from the mushrooms, the adequacy of the thermal process for the mushroom sauce could only be certain if the mushrooms had been fully rehydrated at the start of the process.

The second alleged botulism outbreak attributed to container leakage occurred in 1963. Two of three patients died after consumption of canned tuna. The causative organism involved C. botulinum type E. The can which caused the outbreak was from one code of 2,592 packed on one date in 1963. A second can from this lot, found in a retail store, was swollen and contained type E toxin (106).

Results of can examinations suggested that the cause of the 1963 outbreak was defective double seams (62). Six codes produced in the same cannery involving nearly 650,000 cans were examined following the outbreak; about 3,300 (0.5%) of the cans were reported to have defective double seams at the canner’s end (106). In contrast, out of approximately 7 million cans of fish products examined throughout the State of California over the same period, only 0.13% were considered abnormal; none was shown to be toxic (77).

Evaluation of all of the information from the 1963 tuna fish outbreak prompted some investigators to believe that the toxic cans recovered had received little or no thermal processing. It is not our purpose to debate this issue, but merely to point out that the cause of the outbreak was not proven. This is acknowledged in a letter dated July 28, 1982 from John M. Taylor, Food and Drug Administration, to Charles Carey, National Food Processors Association.

The third botulism outbreak occurred in 1978 in Birmingham, England. The outbreak affected four elderly persons and resulted in two deaths due to consumption of canned salmon packed in Alaska. The cause of this outbreak was C. botulinum type E confirmed by the presence of toxin in the sera of four patients and from washings of the incriminated can. The outbreak involved a single can of salmon from among a production lot of 14,460 cans bearing the same embossed production code. British authorities stated that approximately 2,300 of the salmon cans were eventually recovered. None of the recalled cans showed damage similar in any way to the can involved in the outbreak. In addition, more than 24,000 cans of various codes from a consignment of nearly 114,000 cans from the 1977 salmon pack were examined at the importer’s warehouse. None showed damage similar to the incriminated can.

Structural examination of the incriminated can revealed that it was sound except for the damage area. The metal along the chuck wall at one end of the can had been gouged in some indeterminable way. Subsequently, corrosion created a small hole on the seaming panel (rim of double seam) at the damage site. Tests revealed that the can leaked only under about 5 lb of pressure. The interval between damage to the can and leakage of the botulinum organism(s) or spore(s) into the salmon is unknown, but some experts believed that the type E organism probably gained entrance late in the history of the can.

The cause of the damage to the incriminated can is unknown, although various theories were postulated. The National Food Processors Association stated that the unfamiliar damage could be reproduced with a hand saw (10). Thus it was suggested that the subject can might have been damaged by inadvertent use of stacked canned salmon cases to support wood being sawed. The FDA
investigation disclosed that employees working on the raw fish evisceration lines often placed their wet aprons and gloves on baskets coming out of the retorts at the end of the day. Such a practice was envisioned as one possible of *C. botulinum* type E contamination of the processed cans (9).

Following the 1978 botulism outbreak in canned salmon, the packer involved inspected visually and by dud detector over 14 million cans (nearly 99%) from the 1978 pack. In addition, over one million cans from the 1977 pack were similarly inspected. The packer conducted the inspections with assistance from the National Food Processors Association and the canner's container supplier and under supervision by the FDA. According to information supplied to the FDA in July of 1979, a total of 8,068 cans (defect level = 0.05%, or 5 cans/10,000) were sorted out representing defects such as false seams, droops, cable burns, cut-overs, dents and swells. Reportedly, 296 cans were cultured; only four cans contained viable organisms, but none was *C. botulinum*. Of the total number of cans sorted out, 3,515 cans were reported to have been screened for botulin toxin; none of these cans was toxin-positive.

The fourth botulism outbreak occurred in 1982. This outbreak followed consumption of U.S. canned salmon. Again, *C. botulinum* type E was the cause of the outbreak with one death which involved a Belgian couple. Information obtained by NFPA and FDA indicated that the Belgian couple had prepared a pâté from a 7-3/4 oz can of salmon. Reportedly, Belgian health officials demonstrated type E toxin in the pâté and from washings of the container in the home of the Belgian couple. Examination of the can showed a small triangular-shaped hole in the body near the double seam (13,113).

The triangular shaped opening in the can body at the double seam was caused by a malfunctioning can reforming machine. Economic considerations over the years have favored the use of reformed salmon cans by Alaskan canners. Flattened cylinders, shipped by the container manufacturers, are reformed, flanged and the bottom end attached by the salmon packers. Salmon canners are the only ones to use this type of container.

The single can involved in the 1982 botulism outbreak was from a lot consisting of about 24,000 cans. Soon after the outbreak, additional containers were discovered containing the so-called index defect. None of the containers contained toxic product.

The presence of these additional containers caused the FDA to visually examine containers warehoused in the Seattle area. In this initial series of examinations, the agency found 22 containers with index-type defects from about 500,000 cans examined. Nineteen of these containers were from canneries other than the one which produced the original index container. On the basis of these findings, the industry, working with NFPA and FDA agreed on a program to examine cans of 7-3/4 oz salmon to determine the need for product recall. As a result of these studies eight more canneries initiated recall of their product. When the 1982 salmon problem had been concluded, nearly 60 million cans from nine canneries had been recalled.

During the course of these investigations 131 index defective containers were found. The FDA also collected about 700 samples of obviously abnormal containers for analysis. In none of these samples was toxic product found.

As a means of clearing recalled product, the industry, NFPA and FDA conducted a study on the use of dud detectors and check-weighers to sort out defective containers. A total of about 208,000 7-3/4 oz cans from six companies who volunteered product were examined. The test was conducted by industry and can manufacturers representatives under supervision of NFPA with FDA inspectors acting as observers. This group also classified the defective containers found into categories based upon the severity of the defect or whether leakage had occurred.

Cans which showed signs of leakage were categorized as critical defects. The dud detector and check-weighing system removed 88% of this category of containers. The total critical defects found in the lot was 0.058%.

A portion of the abnormal containers found in this study were examined microbiologically. A total of 180 cans was analyzed by the NFPA, FDA Region X and American Can Company Laboratories. No toxic organisms were found in any of the cans.

During the entire course of the investigation of this incident, about 1000 cans were examined for the presence of botulinic toxin. None was found.

When all of the botulism outbreaks incriminating container leakage are considered, two facts emerge. First, all except the 1941 outbreak involved marine products; second, *C. botulinum* type E was the only toxin type involved in these outbreaks. Undoubtedly, a number of factors were involved. The wide distribution and comparatively high incidence of *C. botulinum* type E and/or its spores in marine and aquatic environments are well documented. In contrast, *C. botulinum* types A and B commonly exist in terrestrial environments. Since type E has the ability to grow at low temperatures, unlike type A and proteolytic type B, proliferation of type E in marine environments is considerably more likely than growth of the proteolytic types. Another consideration is the fact that all known strains of type E are nonproteolytic, hence, detection of spoilage based on odor is much less likely than when the proteolytic types with their putrid odor are involved.

*C. botulinum* in commercially canned foods not causing illness

It could be argued that the results presented in Table 3 show only one side of the proverbial coin. Besides those botulism outbreaks involving post-processing leakage, perhaps we should consider any incident in which *C. botulinum* and/or its toxin was detected in commercially canned foods in metal containers, but human illness was fortunately avoided.

Table 4 summarizes such botulism incidences in commercially canned foods in metal containers so far as
known. Of 13 separate incidents, only one involved post-processing leakage. In 1974, FDA analyzed one can of tuna fish that had been opened at home by a consumer processing leakage. In 1974, FDA analyzed one can of foods is between $10^{-7}$ and $10^{-10}$ based on probability consideration of post-processing leakage from canned C. botulinum. Further investigation showed that the container involved was a malformed can (turned or cocked body).

With the exception of the 1974 tuna fish incident, the remainder of the botulinal problems shown in Table 4 involve underprocessing situations. In fact, when all of the information in Tables 3 and 4 is considered, it is immediately obvious that the vast majority of the problems have been due to underprocessing rather than to container leakage.

**Probability assessment of human botulism from post-processing leakage**

Odlaug and Pflug (86) concluded that the likelihood of post-processing leakage from C. botulinum in canned foods is between $10^{-7}$ and $10^{-10}$ based on probability considerations. When the possibility of C. botulinum growing in canned foods and the likelihood of a consumer eating spoiled product are considered, the probability of human botulism from leakage increases to approximately $10^{-9}$ to $10^{-12}$.

Another, and perhaps more pragmatic assessment of the botulism risk from container leakage can be determined using the data in Tables 3 and 4. As shown, over the past 42 years, there have been only five incidents in which the presence of C. botulinum can be considered as due to post-processing leakage. Over this same period, based on information from the Can Manufacturers Institute, nearly 30 billion low-acid canned foods, on the average, have been consumed each year. Of this number, approximately one billion per year are canned seafoods.

Thus, the number of cans of low-acid foods consumed over the past 42 years approximates $1.3 \times 10^{12}$. Considering this number and the fact that there have been five botulinal incidents in which leakage is either accepted or alleged, the probability of botulism from container leakage is about $3.8 \times 10^{-12}$, or one chance in every 260 billion cans of food that are consumed. This translates to one potential botulism outbreak from leakage about every 9 years.

In considering the extremely low probability of botulism from leakage due to container defects, the remarks of Marth (72) with respect to general food safety seem appropriate:

"Because of the many ways by which a given food could become unsafe for a given consumer, it is impossible for the food industry or for regulatory agencies to control the food industry to guarantee that all foods will be completely safe for all consumers at all times. Consequently, those persons working in this field should invest their time and resources in those areas where the hazards are likely to be greatest and hence where such efforts are likely to benefit the health and welfare of the greatest number of people."

**CONCLUSIONS**

1. Current information does not suggest an increase in the incidence of leaker spoilage in canned foods due to container defects. While definitive data are largely unavailable, it is the conclusion of this committee that over the past 20 years there has been a considerable reduction in the incidence of leaker spoilage involving defective containers. This reduction can be largely attributed to the increased awareness and application of the Better Process Control procedures as required under GMP Regulation, Part 113 - Thermally Processed Low-Acid Foods Packaged in Hermetically Sealed Containers. Spoilage rate reductions are directly related to improved container closure control and post-processing handling practices, especially when the cans are still wet, and to improved container integrity as a result of newer can making technology.

2. C. botulinum has not been demonstrated in fruit, vegetable and meat plant cooling waters, according to published information. The widespread use of sanitizers in cooling water would also reduce the number

**TABLE 4. Presence of C. botulinum or its toxin not causing incidence of human illness in commercially canned foods in metal containers in the United States, 1940-1982.**

<table>
<thead>
<tr>
<th>Year</th>
<th>Canned product</th>
<th>Toxin type from product for culture</th>
<th>Cause of spoilage</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1965</td>
<td>Spinach</td>
<td>A</td>
<td>Underprocessinga</td>
<td></td>
</tr>
<tr>
<td>1970</td>
<td>Mushrooms</td>
<td>B</td>
<td>Underprocessinga</td>
<td>70</td>
</tr>
<tr>
<td>1971</td>
<td>Chicken vegetable soup</td>
<td>A, B</td>
<td>Underprocessing</td>
<td>4</td>
</tr>
<tr>
<td>1973-</td>
<td>Mushrooms - 7 U.S. packers</td>
<td>B (one involved both A &amp; B)</td>
<td>Underprocessinga</td>
<td>70</td>
</tr>
<tr>
<td>1974</td>
<td>Tuna fish (single can)</td>
<td>C</td>
<td>Presumed leakage of malformed container</td>
<td>6</td>
</tr>
<tr>
<td>1980</td>
<td>Mushrooms</td>
<td>B</td>
<td>Underprocessing</td>
<td>11</td>
</tr>
<tr>
<td>1981</td>
<td>Mushrooms</td>
<td>B</td>
<td>Underprocessinga</td>
<td></td>
</tr>
</tbody>
</table>

*a*Presence of botulinum toxin found in spoilage examination by canner's container supplier.

*b*Reportedly due to a malfunctioning flame sterilizer.
of microorganisms of public health significance, if present. Since nonpathogenic vegetative microorganisms constitute the typical microflora of cannery cooling water, and because little or no direct human contact is required in handling wet, cooled cans of low-acid foods in the United States, the likelihood of foodborne illness from leakage is extremely remote.

3. As shown by the data below, from 1940 through 1982, only four human botulism outbreaks were attributed to leakage or possible leaker contamination of U.S. commercially canned foods in metal containers. *Clostridium botulinum* type C was demonstrated in 1974, in a malformed container, opened by a consumer, but the product was not eaten.

<table>
<thead>
<tr>
<th>Year</th>
<th>Product</th>
<th>Toxin type</th>
<th>Cause of outbreak</th>
</tr>
</thead>
<tbody>
<tr>
<td>1941</td>
<td>Mushroom Sauce</td>
<td>E</td>
<td>Suspected leakage</td>
</tr>
<tr>
<td>1963</td>
<td>Tuna Fish</td>
<td>E</td>
<td>Alleged leakage</td>
</tr>
<tr>
<td>1978</td>
<td>Salmon</td>
<td>E</td>
<td>Leakage</td>
</tr>
<tr>
<td>1982</td>
<td>Salmon</td>
<td>E</td>
<td>Leakage</td>
</tr>
</tbody>
</table>

4. The number of outbreaks of botulism attributed to canned foods due to container leakage appear to be greater with those products in which (a) recontamination with nonproteolytic types of *C. botulinum* from the environment is more likely, (b) there is an increased incidence of *C. botulinum* in the environment, (c) detection of spoilage based on odor is much less likely, and (d) consumption occurs without cooking or heating sufficiently to inactivate botulinic toxin. Experience has shown that these factors are more often associated with canned fish products. Review of the botulism outbreaks and incidents not causing illness show that the known problems are predominately associated with underprocessing rather than container leakage.

5. Because of the rate and random nature of post-processing contamination there is no practical way to precisely predict the potential risk of botulism or other microorganisms of public health significance. However, based on historical data over the past 42 years, the probability of botulism from container leakage approximates one in 260 billion cans or, in other terms, one potential botulism incident about every 9 years. This probability compares well to the risk associated with the minimum acceptable thermal process for low-acid canned foods.

RECOMMENDATIONS

1. Container integrity should not be considered in terms of a “zero defects” concept. Such an approach in untenable for the food industry as it is elsewhere. A “zero defects” concept serves as a laudable goal, but it is technologically impossible for container manufacturers and food canners alike to achieve perfection in regard to container integrity. That being true, public health hazards from container leakage must be considered in terms of the more reasonable risk/benefit concept.

2. The *Compendium of Methods for the Microbiological Examination of Foods* (26) is recommended to determine the cause of spoilage of canned foods. The *Bacteriological Analytical Manual for Foods*, or BAM (Association of Official Analytical Chemists, 5th Edition), is recommended as a second choice to the *Compendium of Methods*, but not as an alternative to it.

3. Suspected defective containers should be examined by a trained analyst for leakage and defects. The container examination procedures described in the BAM are recommended.

4. The National Food Processors Association publication entitled *Recommendations for Post-Processing Can Handling* (NCA Information Letter, March 7, 1964) should serve as a guideline for food canners to minimize the incidence of leakage spoilage. Supplemental information can also be found in the Food Processors Institute book entitled *Canned Foods - Principles of Thermal Process Control and Container Closure Evaluation* (42). Specific recommendation for canners to detect and prevent post-processing container handling damage have also been published (16). Foremost, in minimizing the incidence of leaks, the avoidance of container defects, chlorination of cannery cooling water, and avoidance of excessive container abuse especially when wet during post process can handling.

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