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Type C Botulism Due to Toxic Feed Affecting 52,000 Farmed Foxes and Minks in Finland

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The largest reported outbreak of type C botulism in fur production animals is described. Epidemiological investigation of 117 out of 157 (response rate, 74.5%) farms revealed that 44,130 animals died or were euthanized, while 8,033 animals with milder symptoms recovered. The overall death rate in all animals at risk was 21.7%. The death rates were significantly higher in blue and shadow foxes (24.2 and 27.8%, respectively) than in silver and blue silver foxes and minks (below 4%). All minks had been immunized against botulinum toxin type C. Deaths were associated with feed manufactured by a local processor, 83 of whose customer farms (70.9%) reported dead or sick animals. Five feedlots out of 19 delivered to the farms on the day preceding the onset of the outbreak (day 2) were associated with a death rate higher than 40%. These feedlots consisted of fresh feed processed on day 2 and feed processed 1 day earlier (day 1). In laboratory analysis, the day 2 feed contained botulinum toxin type C (>600 minimum lethal doses/g), while the day 1 feed did not contain toxin. Toxin was not detected in feed raw-material samples. Clostridium botulinum type C was detected by PCR in some feed components and in feed. However, as the feed temperature was continuously 8°C or below and the pH was continuously 5.6 or below according to the manufacturer, it seems unlikely that spore germination and toxin formation occurred during overnight storage. Hence, the events leading to toxin formation were not determined.

Botulism is a paralytic disease caused by botulinum neurotoxin blocking neurotransmitter release in peripheral nerve endings. Botulinum neurotoxin is produced during exponential growth of the anaerobic bacterium Clostridium botulinum. Based on their serological properties, botulinum neurotoxins are classified into types A to G, of which types C and D, being produced by group III C. botulinum strains, cause disease in animals. Species sensitive to botulinum toxins include birds, horses, cattle, sheep, and fur production animals.

In the fur production industry, botulism outbreaks affecting hundreds or thousands of animals have caused considerable economical losses. Botulism outbreaks in fur-bearing animals are typically feed poisonings due to botulinum toxin formation in improperly chilled slaughter by-products that are used in feed manufacturing. Minks and ferrets are highly susceptible to botulinum toxins, type C toxin in particular, and reports of large outbreaks in these animals are available (3, 5, 7, 10, 19). Unlike minks, foxes are generally considered resistant to botulinum toxins (6, 20), and very few type C botulism outbreaks in foxes have been reported (6, 10, 17, 18). The oral toxicity of type C botulimum toxin to foxes has been reported to vary from 103 to higher than 108 minimum lethal doses (MLD, measured by intraperitoneal injection into mice) per animal (2, 6, 20, 29).

In order to control the microbiological hazards associated with fur animal feed production in Finland, national regulations regarding the safe handling of slaughter by-products have been set (13). The slaughter by-products are to be cut to a maximum final particle size of 50 mm and either acidified with an organic acid (typically formic or acetic acid) to yield a final pH of no higher than 4.0 or heat processed to a minimum internal temperature of 80°C in all parts of the product. In practice, however, all slaughter by-products are acidified rather than heat processed. The pH of the acidified slaughter by-product must be measured upon arrival at the feed processing plant, and it must not exceed 4.3 (13).

In order to inhibit the growth of yeasts and molds, the addition of sodium benzoate to slaughter by-products is also advised (13). Unless used in feed production immediately after arrival at the feed processing plant, the slaughter by-products must be stored at −18°C or below. Fur animal carcasses must be processed at 118°C for 20 min at 2 bars (13). In addition to these control measures, all minks are immunized against botulinum toxin type C at the age of 2 months. Foxes, however, have generally been considered immune to botulinum toxin and thus have not been vaccinated in Finland. Large botulism outbreaks in fur production animals have not been reported in Finland during the past decades.

This paper describes a recent massive outbreak of type C feed poisoning botulism affecting 52,000 farmed foxes in Finland in 2002. To our knowledge, this is the largest botulism outbreak ever reported in fur production animals.

MATERIALS AND METHODS

Background. The outbreak occurred in October 2002 in an area restricted to 80 by 120 km in the Central Ostrobothnian region in western Finland. On
Saturday, 5 October (day 3), and Sunday, 6 October (day 4), regional fur animal veterinarians were notified by farmers about illness and death of farmed foxes and minks (Table 1). By the end of the weekend, it became clear that dozens of farms had thousands of dead or sick animals. All of these farms were customers of the same feed processing plant.

Sixteen processing plants are responsible for fur animal feed production in Finland. The feed is mostly composed of acidified slaughter by-products with an approximate pH of 4.0, heat processed (118°C, 20 min, 2 bars) fur animal carcasses, ensiled fish and fish entrails, barley and soy four, vitamins, and fat. A total of 95% of slaughter by-products are of Finnish origin and thus mainly acidified, while the remaining 5%, imported mainly from Sweden, may be either acidified or frozen without any acid or heat treatment. The temperature of the acidified slaughter by-products is not controlled in the slaughterhouses, and depending on the time of year and thus the ambient temperature, it may vary greatly. However, due to its low pH and the sodium benzoate addition, the final feed is microbiologically very stable at ambient temperature. In practice, feed storage for longer than a few days is uncommon, but in the rare occasions of extended storage, the feed is normally frozen. The feed composition is regulated by the guidelines of the Finnish Fur Breeders' Association, and it may vary slightly with producer, time of year, and availability of food components.

The flow chart of the feed process routinely employed in the feed manufacturing plant associated with the botulism outbreak is presented in Fig. 1. In order to speed up feed production in the early morning of Friday, 4 October (day 2), this was started in the previous afternoon (day 1) at 1600 to 1700 h by the preparation of three batches of feed base mass. These were prepared in three large mixers (each with a capacity of 6,800 kg) by adding frozen acidified beef slaughter by-products, fresh and frozen fish, processed (118°C, 20 min, 2 bar) fur animal carcasses, mink fat, barley and soy flour, vitamins, and meat and bone powder (processed at 133°C, 20 min, 3 bars). The ingredients were roughly mixed but not homogenized at this stage, and the three 4,300-kg batches of such feed

<table>
<thead>
<tr>
<th>Date</th>
<th>Day</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thursday, 3 Oct</td>
<td>1</td>
<td>Routine feed production; a total of 46,000 kg of leftover feed stored overnight in two trucks and in the loading silo; 3 batches of base mass were prepared (4,300 kg each)</td>
</tr>
<tr>
<td>Friday, 4 Oct</td>
<td>2</td>
<td>Leftover feed from previous day transported to farms (43,000 kg) or mixed (3,000 kg) in the morning's first feed lot; later tests showed that the morning's first feed lots contained botulinum toxin type C</td>
</tr>
<tr>
<td>Saturday, 5 Oct</td>
<td>3</td>
<td>First signs of botulism in animals observed</td>
</tr>
<tr>
<td>Sunday, 6 Oct</td>
<td>4</td>
<td>Sick and dead animals observed at tens of farms</td>
</tr>
<tr>
<td>Monday, 7 Oct</td>
<td>5</td>
<td>Outbreak mentioned by the media; laboratory and epidemiological investigations of the outbreak begun</td>
</tr>
</tbody>
</table>

1. Base mass (pH 5.0 to 5.5; indicated in black) production. The first three base mass batches of day 2 feed were prepared on previous evening (day 1) and stored overnight at 5 to 8°C in three mixers.

2. Addition of other ingredients (indicated in grey) such as barley porridge (80°C, pH=5.3), acidified beef slaughter by-product (RT, pH≤5.2), non-acidified poultry slaughter by-product (<0°C, pH 6.8), and flours in the morning of day 2.

3. Mixing (20 min) and homogenisation (1 to 2 min).

4. Transfer to loading silo. 3000 kg of leftover feed from day 1 was stored overnight in the loading silo between days 1 and 2. This feed batch was mixed with the first batches of day 2 feed. Several feed batches mix with each other in the loading silo.

5. Loading into four trucks and delivery to farms. 43000 kg of leftover feed produced on day 1 was stored in the trucks overnight between days 1 and 2, and delivered to farms early in the morning of day 2. The outside air temperature was 4°C.
base mass were then stored in the mixers overnight. According to the manufacturer, the feed base mass had a typical pH of 5.0 to 5.5 and, due to the large amount of frozen raw material, an internal temperature of 5 to 8°C. No other chilling procedures were employed. The pH and temperature data of the feed base mass, however, were not available upon inspection at the processing plant.

The process continued early in the morning of day 2 with the addition of a total of 2,500 kg of barley porridge (normal pH range, 4.5 to 5.3; internal temperature, 95°C), crops, acidified beef slaughter by-product, and nonacidified frozen pork liver by-products into each of the three mixers (Table 2). The barley porridge was prepared overnight on-site by heating 6,000 liters of water to an internal temperature of 95°C, after which it was cooled down to 70°C and mixed with barley flour. After boiling for 30 min, this mixture was kept continuously at 70°C overnight and added to the feed mixture the next day. After addition of all ingredients, the complete feed mass was mixed for 20 min, followed by a quick homogenization to a final particle size of 10 mm. A total of 35 similar batches (238,000 kg total) were produced during day 2.

After homogenization, the feed was transferred to a loading silo (capacity, 75,000 kg) so that several different batches from the three mixers were mingled in the large loading silo. From this silo, the feed was loaded into four trucks (capacity, 75,000 kg) so that several different batches from the three mixers were loaded into the feed silo and further mixed with the first three feed lots (20,400 kg) produced on day 2. The first trucks (D and A) transporting the day 2 feed loaded their tanks with the mixture of day 1 and day 2 feed at 0655 h. A total of 19 drives were made during day 2, with the last feed lot being transported by 2330 h that night (Fig. 2).

According to the local meteorological observation center, the outside air temperature during the night between day 1 and day 2 was constantly below 4°C.

**Epidemiological investigation.** A questionnaire regarding the animal death rates, recovery rates, the time of feeding the suspect feed to the animals, the time of the first observation of clinical symptoms, and the description of the symptoms was sent to all 157 breeders that were customers of the plant that processed the suspect feed. Of these 157 breeders, 117 (74.5%) returned the questionnaire form with complete information. The animals at these farms were considered at risk. The 40 breeders not responding to the questionnaire included farms both with and without dead animals and were not included in the epidemiological investigation. However, an estimate of the number of dead animals on these farms was produced by using the mean farm size (1,736 animals) and the mean death rate of each feed transport lot.

The four truck drivers delivering feed to the farms on day 2 were interviewed, and their exact driving routes and departure times were recollected and analyzed.

**Sample analyses.** A total of 59 samples (one to nine samples per sample material) of feed and its raw material components were investigated for the presence of botulinum neurotoxin by the mouse bioassay (15). To determine the toxin type, extracts from 10-g samples were neutralized with type C and D botulinum toxin antisera (Centers for Disease Control and Prevention, Atlanta, Ga.) (15). The presence of C. botulinum types A, B, E, and F in the samples was investigated by botulinum neurotoxin gene-specific multiplex PCR (11), and type C was determined by seminested PCR (28). In addition to the feed samples, gastrointestinal samples from 12 dead foxes were investigated for the presence of C. botulinum and botulinum toxin as described above. Serum samples from five of these animals were also tested for toxin.

In order to detect C. botulinum, 10 l-g aliquots of each sample and 10 ethanol-treated (16) l-g aliquots of some samples were inoculated into 10-ml tubes containing tryptose-peptone-glucose-yeast extract (TPGY) broth (23), previously boiled for 15 min to remove the oxygen, and cooled to room temperature. The tubes were incubated in an anaerobic cabinet with an internal atmosphere of...
FIG. 2. Mean animal death rate (error bars indicate standard deviation) observed on farms receiving feed on day 2. Feed lots indicated in normal typeface were produced on day 1, and those indicated in bold were produced on day 2. The truck (A to D) used to deliver each feed lot and the amount of feed in each truck are indicated below the chart. Bold numbers in the columns indicate the cumulative amounts of day 2 feed delivered.
TABLE 3. Numbers of animals at risk and death rates (DR) at 117 fur farms responding to the epidemiological questionnaire*

<table>
<thead>
<tr>
<th>Age and sex group</th>
<th>Blue fox</th>
<th>Shadow fox</th>
<th>Silver fox</th>
<th>Blue silver fox</th>
<th>Minks</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young females and males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of animals at risk</td>
<td>140,274</td>
<td>8,566</td>
<td>1,747</td>
<td>4,513</td>
<td>13,719</td>
<td>168,819</td>
</tr>
<tr>
<td>No. of dead animals</td>
<td>30,951</td>
<td>2,364</td>
<td>0</td>
<td>158</td>
<td>87</td>
<td>33,560</td>
</tr>
<tr>
<td>DR (%)</td>
<td>22.1</td>
<td>27.6</td>
<td>0</td>
<td>3.5</td>
<td>0.6</td>
<td>19.9</td>
</tr>
<tr>
<td>Adult females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of animals at risk</td>
<td>27,465</td>
<td>889</td>
<td>557</td>
<td>12</td>
<td>3,111</td>
<td>32,034</td>
</tr>
<tr>
<td>No. of dead animals</td>
<td>9,529</td>
<td>240</td>
<td>1</td>
<td>12</td>
<td>142</td>
<td>9,924</td>
</tr>
<tr>
<td>DR (%)</td>
<td>34.7</td>
<td>27.0</td>
<td>0.2</td>
<td>100</td>
<td>4.6</td>
<td>31.0</td>
</tr>
<tr>
<td>Adult males</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of animals at risk</td>
<td>1,730</td>
<td>171</td>
<td>222</td>
<td>0</td>
<td>153</td>
<td>2,276</td>
</tr>
<tr>
<td>No. of dead animals</td>
<td>539</td>
<td>68</td>
<td>5</td>
<td>0</td>
<td>34</td>
<td>646</td>
</tr>
<tr>
<td>DR (%)</td>
<td>31.2</td>
<td>39.8</td>
<td>2.3</td>
<td>0</td>
<td>22.2</td>
<td>28.4</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of animals at risk</td>
<td>169,469</td>
<td>9,626</td>
<td>2,526</td>
<td>4,525</td>
<td>16,983</td>
<td>203,129</td>
</tr>
<tr>
<td>No. of dead animals</td>
<td>41,019</td>
<td>2,672</td>
<td>6</td>
<td>170</td>
<td>263</td>
<td>44,130</td>
</tr>
<tr>
<td>DR (%)</td>
<td>24.2</td>
<td>27.8</td>
<td>0.2</td>
<td>3.8</td>
<td>1.5</td>
<td>21.7</td>
</tr>
</tbody>
</table>

* All minks had been immunized against botulinum type C at the age of 2 months, while foxes were not immunized at all.
gastrointestinal samples but not in any of the serum samples. The *C. botulinum* organism was not detected in any of the clinical samples.

The pH of the toxic feed sample was 4.9, while that in the other feed samples varied from 4.6 to 5.6. The pH of the feed components varied between 4.3 and 6.9 (Table 2).

**DISCUSSION**

To our knowledge, the type C botulism outbreak described here is the largest botulism outbreak ever reported in fur production animals, affecting more than 52,000 animals in 83 breeding farms. The overall death rate at farms responding to the questionnaire was 21.7%. However, a large number of animals developing typical clinical symptoms were immediately euthanized for the sake of animal protection, and the fact that 8,000 animals with milder clinical symptoms actually recovered indicates that the natural death rate in all animals would have been lower than 21.7%. Also, taking into account the estimated number of dead animals at the farms not responding to the questionnaire, the overall death rate seems slightly lower (18.6%).

The outbreak affected mainly blue foxes and shadow foxes, while the death rate in silver foxes and in the related blue silver foxes as well as in minks was considerably lower. It is possible that the different fox species possess different susceptibilities to *botulinum* neurotoxins. The blue fox (*Alopex lagopus*) and its color variant shadow fox are of Arctic origin and consume frozen feedstuffs in wildlife, while the blue silver and related silver fox are related to the wild red fox (*Vulpes vulpes*), consuming mainly rotted carrion. In evolution, the different feeding habits of the two fox species may thus have resulted in the development of different resistance to botulinum toxin.

The most typical clinical symptoms observed in the sick animals included paralysis of the hind legs, total paralysis, and recumbent position. Foxes with milder symptoms were often seen in a “sitting” position, dragging the rear part of their bodies behind. Such a position is occasionally seen in otherwise healthy foxes and might serve as an indication of a subtle form of type C botulism in these animals. Hind leg paralysis and recumbence have also been observed as the main clinical feature of botulism in cattle (27), while the typical first symptoms of botulism in humans involve the cranial parts of the body. The reason for the different manifestation of botulism in humans and in animals is unknown but might be explained by different distribution of specific receptors for botulinum toxins in humans and animals.

The relatively high death rate in adult foxes compared to young foxes is explained by the lower body weight of the adults. During the off-reproduction period in late summer and autumn, the adult breeding animals are usually fed only once a day with a limited amount of feed, resulting in a body weight of approximately 6 to 8 kg, while the young fur animals fed ad libitum may weigh more than 10 kg. The adults fed a restricted diet also show an increased appetite and thus finish their meal immediately, while the younger fur animals take smaller quantities of feed now and then throughout the day. The low death rate observed in young minks was obviously due to recent immunization against type C botulimum toxin, while the older minks with a higher death rate, having only been immunized in their youth, apparently already had decreased antibody titers in their serum.

The type C toxin level measured in the feed sample investigated was >600 MLD/g of feed. This sample was collected on a farm that had a total death rate of 68% and received feed from truck A at 0700 h. As the death rate for each truck delivery was variable, it is probable that the toxin concentration in different feed lots delivered to the farms varied. Assuming the measured toxin concentration, an animal eating its entire 1-kg feed portion would ingest $6 \times 10^5$ MLD of toxin. This is in line with an earlier report on the lethal oral dose of type C toxin in blue foxes (2). However, the earlier findings on the susceptibility of foxes to botulimum toxins are controversial, and silver foxes (20) as well as blue foxes (6) have been reported to survive ingesting $10^7$ MLD of type C toxin. Thus, more information on the pathogenesis and lethality of type C toxin in foxes is required.

The fact that type C botulinum toxin was detected in the feed but not in any of its components leaves us with assumptions on the factors that allowed the outbreak to occur. Theoretically, four different events may explain the outbreak: (i) one or several of the feed raw material components contained preformed toxin; (ii) toxin formation from *C. botulinum* type C spores present in raw materials occurred in the feed base mass (three times 4,300 kg) during overnight storage; (iii) toxin formation occurred in the leftover feed produced on day 1 and stored overnight in the loading silo; or (iv) toxin was (deliberately) added to the feed during or prior to the day 2 production. These possible events are discussed below.

Botulinum toxin could not be detected in any of the feed raw materials investigated. However, the samples investigated represented only a very small fraction of all the raw materials used, and some of the feed components were not available for laboratory analysis. Therefore, in spite of the negative laboratory analysis results, it is possible that one or several of the feed components contained toxin. One of the most probable components to contain botulimum toxin was the nonacidified poultry slaughter by-product. The pH of this product was nearly neutral and thus theoretically could have allowed botulinum growth and toxin formation before freezing the lot. According to the manufacturer of this feed component, the raw material was collected in a slaughterhouse at daytime and taken else-

![Figure 3](https://example.com/figure3.png)  
**FIG. 3.** Correlation between the death rate and incubation times observed at fur animal farms.
where for freezing at the end of the day. However, documents describing the time elapsed between collecting and freezing the nonacidified poultry lots used in feed processing on day 2 were not available. In the worst case, the component was stored at room temperature for a whole working day.

*C. botulinum* type C is known to be present in the poultry production environment (4), causing large outbreaks in flocks (21, 24). In the present case, the organism was detected in the nonacidified poultry slaughter by-product at the level of 0.3 spore/g. Type C botulism in birds has been suggested to develop as a consequence of *C. botulinum* growth and toxin formation in the birds’ gastrointestinal tract (22). As the poultry slaughter by-products usually contain the birds’ viscera, such as guts, the contamination of poultry slaughter by-products with *C. botulinum* type C is highly probable. As only one 1,100-kg lot out of 34 similar lots of the suspected poultry by-product was available for laboratory analysis, the possibility that toxin was present in one or several of such batches could not be excluded.

Regarding the other raw material ingredients used in the production of the toxic feed, three components comprising fish entrails, acidified beef slaughter by-products, and barley porridge had a pH range of 4.3 to 5.3. Such a low pH is not likely to support the growth of and toxin formation by *C. botulinum*. Furthermore, the barley porridge was heated to boiling for 30 min, a heat treatment likely to destroy any possible preformed toxin. The barley and soy flours, feather powder, meat and bone powder, and meat powder would be expected to have such a low water activity that bacterial growth would be inhibited. The frozen fish that theoretically could have contained botulinum toxin were not available for laboratory investigation. However, the most commonly observed *C. botulinum* type in Finnish fishing waters and fish is nonproteolytic *C. botulinum* type E (8, 9), while type C strains have not been isolated from these samples. Thus, the source of the high concentration of type C toxin in the feed is likely to be elsewhere.

The growth of and toxin formation by *C. botulinum* type C present in the feed base mass is possible but improbable. *C. botulinum* type C cells were detected in the feed raw materials at levels of less than 0.5 spore/g. As a minimum of 500 kg of each type of slaughter-by-product was used to prepare the feed, the base mass could theoretically carry 10\(^5\) spores. Under optimal circumstances, this spore population might lead to toxin formation. However, the growth-inhibitory pH for *C. botulinum* type C strains has been reported to be 5.1 to 5.6 (23), while the pH measured in most of the base mass components was lower. Also, due to the addition of 1,000 kg of frozen components in each of the 4,300-kg feed base mass lots on the evening of day 1, the feed base mass temperature during the night between days 1 and 2 was constantly kept at 10°C or below according to the feed manufacturer. The growth-limiting temperature for *C. botulinum* type C is 10 to 15°C (23), thus rendering germination and subsequent toxin formation in the feed unlikely. Whether the feed contained foci with an increased temperature and pH due to possible failure in processing, allowing germination and subsequent toxin formation to occur, remains obscure. Nevertheless, this is improbable, as a large amount of toxin would be required to eliminate such an enormous number of animals.

The possibility of toxin formation in the feed base mass during overnight storage can also be excluded by estimating the amount of toxic feed required to cause such enormous damage. As indicated in Fig. 2, at least five transportation lots containing a total of 65,000 kg of day 2 feed were associated with high death rates. This corresponds to 9 to 10 full mixer lots, and only the base masses of three of these lots were stored overnight. Therefore, in addition to the mixer lots incubated overnight, at least two fresh lots prepared in each of the three mixers in the morning of day 2 still contained a high concentration of toxin. This suggests that toxin was present in one or several raw material components used in the production of the first 10 mixer lots (65,000 kg) between 0630 and 0750 h.

That the leftover feed produced on day 1 contained toxin and contaminated the first 68,000 kg of fresh feed produced on the morning of day 2 is very unlikely. First, the day 1 feed samples investigated were not shown to contain botulinum toxin in the laboratory analysis. Second, the 19,000-kg fraction of the leftover feed lot delivered to the farms early on the morning of day 2 did not cause an increased death rate in animals. Third, to make a 68,000-kg sample contain toxin at a level of 600 MLD/g, the 3,000-kg leftover feed mass mixed with day 2 production should theoretically have contained at least 4 \(\times 10^7\) MLD of toxin. The formation of such a high concentration within one night under the nonoptimal conditions described above is improbable.

The possibility of sabotage, i.e., intentional addition of toxic material to the feed or its components, could not be entirely excluded. However, in order to affect such a large number of animals, an enormous dose of toxin would have to have been added and thoroughly mixed into the feed. As toxin production in group III *C. botulinum* strains is mediated by a bacteriophage (14), the proper handling and effective preparation of toxic cultures in a laboratory is more challenging than with group I and II strains. Type C botulism has been reported previously in foxes, which might suggest that the outbreak was of natural origin.

The outbreak caused financial losses of 4 million euros to the government, the feed processing plant, and the fur animal farmers. The manufacturing plant was closed on the evening of day 3 for several weeks to allow thorough cleaning and disinfection. The toxin-containing carcasses and feed were considered high-risk material and subjected to incineration under the control of regional veterinary and environmental health authorities.

In conclusion, the factors leading to the type C botulism outbreak that killed more than 44,000 farmed fur animals remain unclear. However, by careful consideration and exclusion of other possible etiological factors, it may be assumed that one or several raw material components employed in feed production contained preformed toxin. In order to avoid such catastrophes in the future, a careful risk assessment of the factors affecting fur animal feed safety during feed production and storage is warranted.

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REFERENCES