Three Outbreaks of Foodborne Botulism Caused by Unsafe Home Canning of Vegetables—Ohio and Washington, 2008 and 2009†

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ABSTRACT

Foodborne botulism is a potentially fatal paralytic illness caused by ingestion of neurotoxin produced by the spore-forming bacterium Clostridium botulinum. Historically, home-canned vegetables have been the most common cause of botulism outbreaks in the United States. During 2008 and 2009, the Centers for Disease Control and Prevention (CDC) and state and local health departments in Ohio and Washington State investigated three outbreaks caused by unsafe home canning of vegetables. We analyzed CDC surveillance data for background on food vehicles that caused botulism outbreaks from 1999 to 2008. For the three outbreaks described, patients and their family members were interviewed and foods were collected. Laboratory testing of clinical and food samples was done at the respective state public health laboratories. From 1999 to 2008, 116 outbreaks of foodborne botulism were reported. Of the 48 outbreaks caused by home-prepared foods from the contiguous United States, 38% (18) were from home-canned vegetables. Three outbreaks of Type A botulism occurred in Ohio and Washington in September 2008, January 2009, and June 2009. Home-canned vegetables (green beans, green bean and carrot blend, and asparagus) served at family meals were confirmed as the source of each outbreak. In each instance, home canners did not follow canning instructions, did not use pressure cookers, ignored signs of food spoilage, and were unaware of the risk of botulism from consuming improperly preserved vegetables. Home-canned vegetables remain a leading cause of foodborne botulism. These outbreaks illustrate critical areas of concern in current home canning and food preparation knowledge and practices. Similar gaps were identified in a 2005 national survey of U.S. adults. Botulism prevention efforts should include targeted educational outreach to home canners.

Botulism is a serious neuroparalytic and sometimes fatal illness caused by potent neurotoxins produced by the gram-positive, anaerobic, spore-forming bacterium Clostridium botulinum and rare toxigenic strains of Clostridium baratii and Clostridium butyricum. Seven types of botulinum toxins are known (A through G), of which types A, B, E, and F cause virtually all cases of human botulism (8). Botulism is characterized by rapidly progressive cranial neuropathy and symmetric descending flaccid paralysis, which may progress to respiratory arrest requiring mechanical ventilation and intensive supportive care in ~60% of patients (24). Clinical recovery takes several weeks to months (24). While prompt administration of the specific antitoxin can halt progression, intensive-care supportive measures remain the mainstay of treatment (8, 24).

Foodborne botulism is caused by ingestion of foods contaminated with preformed botulinum toxin, and illness onset typically occurs 18 to 36 h after toxin ingestion (24). C. botulinum spores are ubiquitous and heat resistant to processes that kill nonsporulating organisms, but spore germination and toxin elaboration require high water activity (a_w > 0.955) and anaerobic, low-salt, low-sugar, and low-acid conditions at nonrefrigeration temperatures (3, 15, 16). In addition to being a clinical emergency, botulism is also a public health emergency because a single contaminated food can cause illness in many persons (23).

From 1950 through 1996, 1,087 cases and 444 outbreaks of foodborne botulism were reported in the United States, with a mean of 23 cases and 9 outbreaks per year (8). An outbreak was any occurrence of foodborne botulism associated with consumption of a specific contaminated food, whether a single case or a multicase cluster. Most U.S. outbreaks of foodborne botulism are caused by home-processed and home-canned foods (13, 29), with home-canned vegetables alone accounting for 56% of outbreaks in the United States in which a specific food

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vehicle was identified (8). Recent published accounts of botulism outbreaks have focused on outbreaks caused by commercial foods (7, 17, 25), novel or unusual food vehicles (31), and international outbreaks (9–11). Though home-canned vegetables have long been recognized as the most common cause of U.S. botulism outbreaks, few reports of these outbreaks have been published in recent decades. We summarize foodborne botulism outbreaks in recent years and describe three recent outbreaks caused by home-canned vegetables. Our investigations show the need to improve awareness and education among home canners regarding safe processing and preparation practices to reduce the risk of foodborne botulism.

MATERIALS AND METHODS

Botulism surveillance. Immediate reporting of suspect botulism patients to state public health authorities is mandatory in all 50 states. Additional botulism consultation for clinicians and state health authorities is available on a 24-h basis through the Centers for Disease Control and Prevention (CDC). Botulinum antitoxin for treatment of U.S. foodborne botulism patients is only available through CDC (and the states of California and Alaska), and confirmatory laboratory testing for botulism is available only through state health departments and CDC. The CDC National Surveillance Team compiles botulism surveillance information from state health departments and antitoxin release data into an electronic database.

Clinical and epidemiologic investigation. Botulism outbreak investigations were conducted by state health departments and CDC, and CDC collected additional demographic, clinical, and epidemiologic information from antitoxin recipients, using a standard Botulism Case Report form. CDC surveillance data were analyzed to provide background information about food vehicles that have been responsible for recent foodborne botulism outbreaks (1999 to 2008, the most recent 10 years for which data were available). For the three outbreaks presented in this report, hypothesis-generating interviews, including 5-day food histories, were administered to patients and their family members to identify suspect foods and other potentially exposed persons. Interviewers also collected detailed information about preservation (e.g., home canning), storage, and meal preparation of suspect foods. Where available, photocopies of relevant canning recipes were requested, as well as information about prior home canning experience, incentives for home canning, and knowledge regarding botulism from patients and family members. Up to three additional attempts were made to interview patients or family members who were initially unavailable for interview.

Laboratory investigation. Samples of serum, stool, and suspect foods were obtained from patients and forwarded to the respective Ohio and Washington state public health laboratories for confirmatory botulism testing. Laboratory isolation of C. botulinum was done by using directly inoculated agar media or enrichment cultures, and botulinum toxin was identified through diffusion-in-gel enzyme-linked immunosorbent assay and the mouse toxicity and neutralization bioassay, the laboratory methods for which are described elsewhere (8). Laboratory safety precautions for work with C. botulinum toxin were followed in accordance to the fifth edition of Biosafety in Microbiological and Biomedical Laboratories (2009), published by the CDC and the National Institutes of Health. Botulinum toxin is considered a select agent, and work with this toxin is regulated under 7 CFR Part 331, 9 CFR Part 121, and 42 CFR Part 73. All materials were handled under regulations using Biosafety Level 2 and level 3 practices, containment, and facilities.

RESULTS

Types of foods implicated in U.S. foodborne botulism outbreaks, 1999 to 2008. From 1999 to 2008, 116 outbreaks (201 cases) of foodborne botulism were reported, including 82 outbreaks with information about the source and preservation methods for the implicated food. Commercial and home-prepared foods were implicated in 9% (n = 7) and 91% (n = 75) of outbreaks, respectively. Among the 75 reported outbreaks caused by home-prepared foods, home-canned foods accounted for 44% (n = 33), traditional Alaska Native uncooked aquatic game foods for 36% (n = 27), and 20% (n = 15) were attributed to other home-prepared foods including a variety of meats, fermented tofu, and pruno. All outbreaks from Alaska Native aquatic game foods occurred in Alaska, and no Alaska outbreaks were attributed to other types of food vehicles. Excluding Alaska, home-canned vegetables were the most common single cause of outbreaks (18 of 48) from home-prepared foods in the United States.

Outbreak 1: Ohio, September 2008. On 12 September 2008, two family members (patient no. 1, a 76-year-old male, and patient no. 2, a 15-year-old male, grandson of patient no. 1) were hospitalized with gastrointestinal symptoms accompanied by bilateral cranial nerve palsies and progressive peripheral muscle weakness (Table 1). Paralysis progressed rapidly, and both patients required mechanical ventilatory support that day. On 13 September, four additional family members were evaluated at the same hospital: patient no. 3 (80-year-old female, wife of patient no. 1), who also progressively worsened and underwent intubation; patient no. 4 (38-year-old male, son of patients no. 1 and no. 3), who had milder symptoms and did not require mechanical ventilation; and two children, patients no. 5 and no. 6 (both 10-year-old females, grandchildren of patients no. 1 and no. 3), who were admitted for observation. Patient no. 5 developed mild subjective symptoms without objective neurologic findings, and patient no. 6 remained asymptomatic.

The investigation revealed that the four botulism patients were part of a group of seven family members who had shared a meal on the evening of 10 September, which included a home-canned blend of carrots and green beans (referred to as “blend”). The blend was “briefly heated in a microwave” before being served and was reported to have an unpleasant odor and taste by persons who ate it. Patient no. 1 (who reportedly “did not like wasting food”) and patient no. 2 ate most of the blend; patient no. 3 handled the blend and had it on her plate, though she was unsure whether she had eaten any; and patient no. 4 “tried a forkful” of the blend but stopped because of the bad taste. Patients no. 5 and no. 6 reported having the blend on their plates, patient no. 5 may have eaten some, and patient no. 6 did not consume any. The seventh family member, who was asymptomatic, neither consumed the blend nor had it on her plate. The meal of
# TABLE 1. Clinical and microbiological features among botulism patients, Ohio and Washington, 2008 and 2009

<table>
<thead>
<tr>
<th>Outbreak</th>
<th>Patient characteristics</th>
<th>Hospitalization</th>
<th>Mechanical ventilation prescribed</th>
<th>Antitoxin prescribed</th>
<th>Implicated food</th>
<th>Laboratory findings (botulinum toxin detected in:)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>Location</td>
<td>Clinical features</td>
<td></td>
<td></td>
<td></td>
<td>Clinical samples</td>
</tr>
<tr>
<td>1</td>
<td>Ohio State</td>
<td>Patient 1: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, hoarseness, dry mouth, difficulty swallowing, shortness of breath, subjective weakness, bilateral neurologic signs (extraocular palsy, ptosis, facial paralysis)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Home-canned green bean and carrot blend</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 2: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, shortness of breath, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, facial paralysis)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 3: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, dry mouth, difficulty swallowing, shortness of breath, subjective weakness, bilateral ptosis</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 4: Nausea, diarrhea, blurred vision, dizziness, difficulty swallowing</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 5: Subjective weakness</td>
<td>Yes (for observation)</td>
<td>No</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 6: Asymptomatic</td>
<td>Yes (for observation)</td>
<td>No</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Washington State</td>
<td>Patient 7: Nausea and vomiting, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, hoarseness, dry mouth, shortness of breath, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, facial paralysis, palatal weakness, impaired gag reflex)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Home-canned green beans</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 8: Slurred speech, thick tongue, change in sound of voice, hoarseness, dysphagia, subjective weakness, bilateral palatal weakness</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
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<td></td>
<td></td>
<td>Patient 9: Diplopia, slurred speech, change in sound of voice, subjective weakness, bilateral neurologic signs (extraocular palsy, palatal weakness, impaired gag reflex)</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Washington State</td>
<td>Patient 10: Nausea, vomiting, blurred vision, diplopia, dizziness, slurred speech, thick tongue, hoarseness, dysphagia, shortness of breath, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, dilated pupils)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Home-canned asparagus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient 11: Nausea, blurred vision, diplopia, dizziness, slurred speech, change in sound of voice, subjective weakness, fatigue, bilateral neurologic signs (extraocular palsy, ptosis, sluggish pupils)</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Patient 12: Blurred vision, diplopia, dry mouth, fatigue</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Residues obtained from jars containing the implicated asparagus</td>
</tr>
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</table>
10 September was the only shared meal among the ill family members, and none of the other foods during the meal were home canned.

*C. botulinum* toxin type A was detected in serum samples from patients no. 1 and no. 2 and in stool from patient no. 1. No leftover food remained from the jar of the blend consumed at the implicated meal, and the empty jar had been discarded. However, 10 unopened jars of the blend from the same canning batch were collected and tested. The jars were described by the laboratory as having “effervescence like opening cans of soda” with noticeable bubbles in several jars. Botulism toxin type A was detected in 5 of the 10 jars (Table 1).

The carrots and green beans for the blend were grown in the family’s home garden and were canned by patient no. 1 approximately 3 weeks prior to consumption. The canning recipe was reportedly obtained from an “at least 50-year-old edition” of *The Better Homes and Garden Cookbook*. The canning recipe for both vegetables suggested use of “pressure cooker” with processing times of 20 and 25 min for pint- and quart-sized jars, respectively, at a pressure of 10 lb in $^{-2}$ (68 kPa). However, patient no. 1 did not possess a pressure cooker and “was told by an old farmer’s wife that it was not needed.” Instead, he used a hot water bath for 1 h. Canning supplies included glass canning jars, of unknown brand, which were secured from a family home in a nearby town. Several jars were reported to have seal failure with lid centers “popped up” after canning had occurred. Patient no. 1 had home-canned pickled beets on a few previous occasions and no other foods.

**Outbreak 2: Washington State, January 2009.** On 21 January 2009, a 37-year-old female (patient no. 7) reported to a hospital emergency room with nausea and vomiting. She was evaluated and discharged home, but she returned to the hospital on 22 January with additional paralytic signs and symptoms consistent with botulism (Table 1), and she required intubation later that day. On 23 January, patient no. 8 (9-year-old female, daughter of patient no. 7) and patient no. 9 (7-year-old female, daughter of patient no. 7) were also evaluated for paralytic signs and symptoms consistent with botulism (Table 1).

Three botulism patients from a family shared a meal with one other family member on the evening of 20 January, which included three types of home-canned products: green beans, tomatoes, and pears. The fourth member did not develop symptoms. Patients no. 8 and no. 9 commented that the green beans “smelled like cat litter.” Patient no. 7 did not note an unusual odor or smell and ate more green beans than the others, including most of her daughters’ portions. The fourth member did not eat any green beans due to the “suspicious smell” but ate the other home-canned foods served during the meal. None of the other foods served were reported to have a suspicious appearance, odor, or taste.

Laboratory testing identified *C. botulinum* toxin Type A in serum and stool samples from patient no. 7 and patient no. 8 and from samples of the leftover green beans.

All home-canned foods consumed during the meal, including green beans, were grown in the family’s home garden. About 20 jars of green beans were canned shortly after harvesting by patient no. 7 in her home, approximately 5 months before the implicated meal. Recipe and processing details used for canning green beans, including whether or not a pressure cooker was used, were not available; however, patient no. 7 reported having shortened the recommended canning time due to lack of air-conditioning in the house. The jars of beans were stored at room temperature in the pantry, and the family had previously consumed other jars of green beans from the same canning batch. Limited cooperation by patients and family members precluded collection of additional details.

**Outbreak 3: Washington State, June 2009.** On 27 June 2009, two men (patient no. 10, a 54-year-old male, and patient no. 11, a 50-year-old male, friend of patient no. 10) sought care at a hospital in Tonasket, WA, with symptoms of nausea and progressively worsening blurred vision, double vision, and difficulty speaking (Table 1). After hospital admission, both patients developed respiratory distress and were intubated. On 28 June a third patient (patient no. 12, a 56-year-old male, brother of patient no. 10), also was evaluated at the hospital with blurred vision, mild dry mouth, and loss of appetite. His symptoms remained mild, and he did not need mechanical ventilation.

The three botulism patients identified in this outbreak had shared a meal on 26 June, which included home-canned asparagus. This was the only meal the three shared, and no others participated. The asparagus did not have an unusual odor, appearance, or taste when it was removed from the can, but several jar lids from the same canning batch were reported to have “popped up.” The asparagus was heated on a stove top (for unknown duration) before being served and consumed.

*C. botulinum* toxin type A was detected in serum and stool samples from patient no. 10 and from residues obtained from the jars containing the implicated asparagus.

The asparagus was a gift from a friend’s home garden, had been home canned by patient no. 10’s wife ~1 month before being eaten, and was stored at room temperature. Patient no. 10’s wife reported that she was a novice home canner and had only one prior canning experience with pickled asparagus and salsa. Instructions for canning were obtained from *The American Woman’s Cookbook*; the date of publication and edition could not be obtained. The canning recipe instructed cooking the asparagus in boiling water for 3 to 4 min, followed by processing immediately in a pressure cooker. However, patient no. 10’s wife did not use a pressure cooker but instead covered and processed the jars in a hot water bath for 15 min (details about the hot water bath were not available). The canner was also not aware of the risk of botulism with improper home canning before this illness episode.

**Antitoxin administration.** A presumptive clinical diagnosis of botulism was made in each of these outbreaks, and the CDC released botulism antitoxin immediately. Botulism antitoxin Bivalent (Equine) Types A and B were administered to 10 patients after sensitivity testing (Ta-
ble 1). Two patients experienced adverse reactions: patient no. 12 developed hypotension, which required supportive measures for anaphylaxis and discontinuation of antitoxin administration, and patient no. 7 developed facial edema after antitoxin administration, which resolved with corticosteroids.

**DISCUSSION**

Home-canned vegetables remain the most important single cause of foodborne botulism outbreaks in the contiguous United States. In these three outbreaks of foodborne botulism, epidemiologic and laboratory investigations confirmed home-grown, improperly home-canned vegetables as the source of each outbreak. The home canners did not follow recipe instructions to use pressure cookers, which is necessary for safe home canning of low-acid vegetables such as green beans and asparagus (30). Additionally, warning signs of food spoilage, including unpleasant odor or taste and/or popped lids, were unheeded in all three outbreaks. These findings indicate that awareness of the risk of botulism or other serious illness from improperly preserved vegetables was low among home canners and family and friends who consumed the implicated foods.

The recipes the three home canners used were obtained from cookbooks but then were adapted based on advice from family or friends and for personal convenience. Adherence to the available recipes would have been protective. Canners should refer to the most updated information regarding guidelines and recommendations for safe home canning (30). In these outbreaks, the food was not cooked again after the jars were opened. While heating suspect food is not recommended to salvage it (30), botulinum toxin is inactivated by heating to 85°C for at least 5 min (8, 28). Microwave warming in the absence of stirring is often nonuniform, leaving cold spots where toxin would not be inactivated (26).

The popularity of food gardening and home canning is increasing in the United States, a trend that is partly explained by economic forces as well as perceived health benefits of consuming home-grown foods (2, 4, 5, 18, 20–22). In a national survey conducted in 2004, about one in five U.S. households canned food, and 65% of those canned vegetables (2). Careful attention to safe canning practices is critical for all foods, but especially for pH-neutral vegetables like green beans, carrots, and asparagus that will permit toxin formation if they are contaminated with *C. botulinum* spores (30). This includes using a pressure canner and adhering to the recommended processing times. Boiling and cooking home-canned foods (≥10 min) before eating them provides an additional margin of safety. Alarmingly, errors similar to those that led to the three botulism outbreaks reported here were commonly reported in a national survey of home canners in 2005 (1, 2). Up to 57.5% of home canners used unsafe methods like oven canning, open-kettle canning, and boiling-water canning for preserving low-acid vegetables, rather than using a pressure canner or cooker; only 12% of respondents had the dial gauge on their pressure canner tested for accuracy; 32% reported having jars that did not seal properly after canning; and 12% served home-canned foods without reheating (2). Family or friends were the source of canning information for 51% of respondents. Home-canned foods are commonly distributed to friends (22), underscoring the risk of potentially widespread botulism outbreaks caused by unsafe home canning and food preparation practices.

Our investigations had several limitations. During the epidemiologic investigations some home canners were intubated, so some details about the food preparation process were obtained from family members, who had limited information on the specific home canning practices used; subsequent interviews were met with limited cooperation and yielded some inconsistencies. Detailed information about certain practices could not be obtained; for example, the exact microwave heating times and power settings were not available. Furthermore, the clinical data were limited to what is routinely collected during a public health investigation; thus, we did not track longer-term clinical outcomes such as total duration of ventilation, hospitalization, or symptoms.

These outbreaks highlight the importance of proper canning and food preparation practices and the severity of foodborne botulism for patients. Although we could not determine the exact duration of illness among patients associated with these outbreaks, reviews of the clinical severity of botulism are available elsewhere (6, 24, 27). Patients have required ventilatory support for up to 7 months before the return of normal function (8), and prolonged recovery and long-term outcomes have been documented (14, 19). Physicians should consider the diagnosis of botulism in patients who present with rapid-onset cranial neuropathy and descending flaccid paralysis, especially with a history of consumption of home-canned foods. The occurrence of acute paralysis among two or more persons who shared a common meal strongly suggests botulism. All suspected cases of botulism should be immediately reported to the local and state health departments and to the CDC. The state health departments and CDC maintain a 24-h emergency consultation service for clinical diagnostic, epidemiologic, and diagnostic laboratory services. Emergency contact information for state and local health departments is available with the CDC Emergency Operations Center, which can be reached 24 h a day, 7 days a week, every day of the year at 770-488-7100. Botulinum antitoxin is available through the States of California and Alaska and the CDC.

Although relatively rare, hypersensitivity reactions can occur with the equine antitoxin, as was seen with two patients during our investigation. Since 13 March 2010 an investigational heptavalent antitoxin (HBAT, Cangene Corporation) has replaced all licensed bivalent botulinum antitoxin AB and an investigational monovalent botulinum antitoxin E (BAT-AB and BAT-E, Sanofi Pasteur) through a CDC-sponsored U.S. Food and Drug Administration Investigational New Drug protocol (12). Investigational HBAT is composed of Fab and F(ab)’2 immunoglobulin fragments, contains <2% of intact immunoglobulin com-
ponents, and may cause fewer allergic reactions. However, preliminary data about the frequency of adverse events among investigational HBAT recipients are still being collected.

These outbreaks and national surveys of home canners identify critical areas of concern in current home canning and food preparation practices. Public health authorities should be alert for possible botulism cases and swiftly investigate them. Information with links to approved canning practices and warnings about the risk of botulism can be targeted directly to home canners, for example, thorough improved labeling of home canning supplies and the addition of information about botulism to cookbooks and Web sites about home canning. The most updated 2009 U.S. Department of Agriculture guidelines for safe home canning can be found at http://www.uga.edu/nchfp/publications/publications_usda.html. Foods with evidence of spoilage should be discarded. Additionally, all low-acid and tomato foods should be boiled in a saucepan for 10 min or longer based on altitudes, unless the preparer is sure that up-to-date, U.S. Department of Agriculture–recommended canning procedures were followed and there is no evidence of spoilage (30). More assessment of the prevalence of home canning practices in the United States and of the information sources most frequently used by home canners would be useful to further shape public health interventions.

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