Microbial Pathogens in Fresh Produce--the Regulatory Perspective

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ABSTRACT

The potential for microbial contamination of fruits and vegetables is high because of the wide variety of conditions to which the produce is exposed during growth, harvest, and distribution. Heat treatment may also destroy the protective barriers (peels, husks, rinds) of fruits and vegetables, permitting the entry of microbial pathogens into the produce and providing them access to nutrients essential for their growth and proliferation. Proper refrigeration, storage, and shipping conditions as well as removal of soil from fresh produce by washing with chlorinated water are recommended to prevent contamination.

TABLE 1. Outbreaks of gastrointestinal illness caused by consumption of various foods.

<table>
<thead>
<tr>
<th>Year</th>
<th>Fruits and vegetables</th>
<th>Meats and poultry</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>1983</td>
<td>13</td>
<td>5.5</td>
<td>35</td>
</tr>
<tr>
<td>1984</td>
<td>8</td>
<td>3.4</td>
<td>67</td>
</tr>
<tr>
<td>1985</td>
<td>18</td>
<td>8.1</td>
<td>39</td>
</tr>
<tr>
<td>1986</td>
<td>16</td>
<td>7.9</td>
<td>36</td>
</tr>
<tr>
<td>1987</td>
<td>4</td>
<td>2.6</td>
<td>24</td>
</tr>
</tbody>
</table>

As reported to CDC (4).

Outbreaks of gastroenteritis caused by the ingestion of fruits and vegetables occur less frequently than do those caused by other contaminated or improperly prepared foods (4) (Table 1). Although most fruits and vegetables contain the nutrients necessary to support the rapid and progressive growth of infectious or toxigenic microbes (the definition of a potentially hazardous food), they also possess a barrier that prevents microbes from entering and subsequently growing in the interior of the fruit or vegetable. The testae of seeds, the peels, husks, skins (cuticle) or rinds of fruits and vegetables, and the intact shells of nuts are such protective barriers. In fact, breaching of the natural barrier of fruits and vegetables by boring insects or bruising of the skin has not been demonstrated to make a raw fruit or vegetable potentially hazardous. The Retail Foods Branch, Division of Cooperative State Programs, Center for Food Safety and Applied Nutrition, Food and Drug Administration (FDA), issued a Model Code Interpretation on May 9, 1986, naming only one fresh plant food, seed sprouts, potentially hazardous. Contamination of this food with Bacillus cereus, Salmonella species, and Yersinia enterocolitica has been responsible for several cases of illness in past years (1,13).

Although penetration by potentially pathogenic microbes into the interior of a fruit or vegetable is prevented by a physical barrier, a heat process can destroy this barrier; it also denatures protein, breaks down complex carbohydrates into forms more readily usable by microbes, and destroys the normal microbial flora on the product which compete with pathogenic microbes for nutrients. Because of these changes, fruits and vegetables that receive a heat treatment (scalding, blanching, or cooking) have been designated as potentially hazardous by the FDA Retail Foods Branch. Most foodborne disease outbreaks traceable to fruits and vegetables are reportedly due to Clostridium botulinum and are associated with the consumption of improperly processed, home-canned products rather than the consumption of raw or commercially prepared foods (2,11). However, these reports may be misleading, either because C. botulinum is more universally reported by public health officials than are other microorganisms, or because the source of a foodborne illness is rarely traced back to contaminated fruits and vegetables. Salmonellosis has been a notifiable disease in the United States since 1943 and accounts for most of the foodborne microbes reported to the Centers for Disease Control (CDC) (19). Despite this fact, human salmonellosis was attributed to the consumption of fruits and vegetables in only 2.2% of all known cases reported to CDC between 1983 and 1987. In general, etiological agents responsible for gastroenteritis are identified in only 40% of gastroenteritis cases reported to CDC, and pathogens were identified in only 58% of the cases for which a food was implicated as the vehicle of transmission.

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Thus, the lack of illnesses attributable to fruits and vegetables may be due to artifacts inherent in the reporting process.

All green plants possess a resident microflora which normally subsists on the minute traces of carbohydrates, protein, and inorganic salts which dissolve in the water that exudes from the epidermis of the plant or condenses from the atmosphere onto the plant. The normal microbial flora of fruits and vegetables consists mainly of organisms commonly found in soil: coryneforms and sporeformers. Some gram-negative microbes exist as normal inhabitants of fruits and vegetables; they may be isolated from these foods regularly and may include coliforms, such as members of the genus Klebsiella.

The potential for fruits and vegetables to become contaminated with pathogenic microbes is high because of their exposure to a wide variety of conditions during growth, harvest, and distribution (Table 2). Soil appears to be responsible for most microbial contamination of fruits and vegetables, as evidenced by the isolation of soil-residing pathogenic bacteria from these products (8). The mere presence of C. botulinum on a food product will not cause illness; conditions must be such that spores of this microbe are not destroyed and are given the opportunity to germinate and produce toxin (16-18). The significance to human health of the finding of Listeria monocytogenes on fresh market produce is not known. However, outbreaks of listeriosis have been linked to the consumption of raw cabbage contaminated with the feces of sheep containing the organism (15), and lettuce, celery, and tomatoes were implicated as the vehicles of transmission in a cluster of listeriosis cases in Boston (9). The survival of pathogens on produce varies as to the microbe involved and the conditions under which the food is stored during shipment and before purchase. Refrigeration, storage, and shipment, whether in ice or dry, affect the survivability of resident microbes (Table 3).

Three large bacterial outbreaks of gastroenteritis were recently caused by the consumption of fresh, raw vegetables. Two of the outbreaks were due to the consumption of lettuce contaminated with Shigella sonnei. In one outbreak, contamination of the product may have occurred in the field or in a warehouse (10). It is speculated that the other S. sonnei outbreak was due to handling of the shredded product by a contaminated food handler, after which the shredded vegetable was incubated at elevated temperatures in a plastic bag (6). Subsequent experiments demonstrated that S. sonnei reproduces in shredded lettuce by rupturing the plant cell walls, thereby releasing cytosol, which contains enough moisture and nutrients to allow reproduction of this microbe (6). The third outbreak was due to the surface contamination of melons by the pathogenic microbe Salmonella chester (14). This outbreak involved 30 states and resulted in two deaths; it is estimated that more than 25,000 individuals were eventually infected (14). A high correlation of illness was associated with the consumption of cantaloupe from salad bars, and it was speculated that microbes on the unwashed rind of the melon came into contact with the interior of the fruit pieces after cutting (14). This was not the first outbreak of gastroenteritis attributed to the consumption of melons. A review of the literature revealed that three outbreaks of salmonellosis caused by contaminated melons have occurred in the United States; all of them were due to watermelon consumption (3,7). In all three outbreaks, the microbes were thought to be introduced from the melon rind into the fruit by the physical act of cutting the melon (3,6). Laboratory experiments have confirmed that the introduced microbes will multiply if temperatures are suitable for reproduction (3).

In an attempt to identify and eliminate the practice(s) responsible for the presence of microbes on the surface of melons, the FDA sampled imported melons from March 26 through April 13, 1990. Results of this sampling, which involved the culturing of 1,440 melons, revealed that only 11 melons (0.76% of those sampled) had Salmonella spp. on their surfaces. Of the eight different Salmonella serotypes isolated, none were S. chester. After the completion of this survey and further epidemiological work, it was determined that melons imported during the period of the outbreak (December 22, 1989 through March 23, 1990)
came from harvest areas different from those sampled. To pinpoint the harvest area from which the *S. chester*-contaminated melons were harvested, the FDA conducted a second sampling of melons from November 19, 1990 through January 3, 1991. This time frame coincided with the harvest of melons during the 1989-1990 outbreak; the melons examined were harvested from the same area as those that caused the previous year’s *S. chester* outbreak. For this survey, 24 (1.06%) of the 2,220 melons cultured contained *Salmonella* species. However, *S. chester* was not isolated from any of the melons examined; all of the isolates belonged to 12 other *Salmonella* serotypes. Although the methodology used by the FDA during the course of these surveys was qualitative rather than quantitative, the low incidence of melons positive for *Salmonella* within a sample (10-15 melons per sample) suggests that low numbers of the pathogen were present on the melon skins.

Epidemiological evidence further implicated cantaloupe as the vector of foodborne illness from June 2 through June 27, 1991; (5). Fifteen states and two Canadian provinces reported outbreaks of *Salmonella poona*, involving 185 confirmed cases in the United States and 56 in Canada. This outbreak was also associated with cantaloupe on salad bars, again indicating that the microbe multiplied while the cut melon was sitting in a bowl on the salad bar. No *S. poona* were recovered from any cantaloupes examined by either state or federal laboratory personnel; however, on July 17, 1991, FDA instructed food retailers to wash melons before cutting, to remove the rind from cut melons, to maintain the cut melons below 45°F after cutting, and not to display cut melons for more than 2 h after cutting.

The emergence of cholera in South America prompted the FDA to sample fruits and vegetables from South American countries beginning in March, 1991 (12,19). A similar sampling assignment of fruits and vegetables grown in Central America was issued in July, 1991, after cholera was reported in Mexico (Pan American Health Association, Washington, DC, personal communication). In both instances, sampling failed to reveal viable *Vibrio cholerae* on any imported fruits or vegetables. Although sampling of produce from South America for the presence of *V. cholerae* has now ceased, sampling of imports from Central America continues because of the small number of products analyzed to date and the possibility that *V. cholerae* may remain viable on produce between harvest and importation.

The actions taken by FDA regarding the possible *Salmonella* contamination of cantaloupe exteriors and the possibility of *V. cholerae* contamination of fresh fruits and vegetables are typical of past FDA actions. The agency attempts to identify the agricultural or shipping practice that led to the contamination, with the eventual goal of eliminating that practice. If a practice causing the contamination cannot be identified, FDA works with the Industry to develop a method, sometimes as simple as washing with chlorinated water, that will eliminate the microbial hazard associated with that particular produce.

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REFERENCES