Comprehensive REVIEWS in Food Science and Food Safety

Microbial Risks Associated with Cabbage, Carrots, Celery, Onions, and Deli Salads Made with These Produce Items

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Abstract: The microbiological safety of cabbage, carrots, celery, and onions/scallions as well as deli (mayonnaise-based) salads that contain these items is the subject of this review. Between 2000 and 2007, the number of outbreaks in the United States associated with these raw produce items ranged from 6 (celery) to 18 (carrots). For cases with confirmed etiologies involving these 4 types of produce as well as coleslaw, chicken, seafood, and other vegetable-based salads, more than 50% of the outbreaks were attributed to viral agents. In contrast, *Salmonella* spp. served as the major etiological agent in outbreaks associated with potato salad. Surveys conducted on these produce items within the United States and other developed countries found either an absence or infrequent contamination with foodborne pathogens. Despite this low prevalence, experimental studies have demonstrated the potential for preharvest contamination, and this event is more likely to occur when exposure is close to harvest. Postharvest contamination of these produce items has been documented in several cases with water, equipment, and incoming product serving as the principal cross-contamination agent. Survival of contaminated product during subsequent storage is dependent on the storage temperature, produce type, and presence of mayonnaise. Chemical interventions may be relied on to reduce cross-contamination during produce washing operations but are limited in their ability to inactivate pathogens on the produce surface. In contrast, irradiation at dosages (1.0 kGy) approved for use in the United States is an effective treatment for killing pathogenic bacteria in fresh-cut cabbage, carrots, and celery.

Introduction

Produce, especially fresh-cut produce items that are consumed raw, has been increasingly linked to foodborne outbreaks (Sivapalasingam and others 2004; DeWaal and Bhuiya 2007). Increased consumption of fresh produce, changes in production and distribution, and a growing awareness by public health officials have been cited as factors contributing to this trend (Lynch and others 2009). Several challenges have been advocated to thwart this trend and include identification of better methods for preventing contamination on the farm or during packing or processing, performing comprehensive and timely environmental assessments of outbreak investigations, and more research into the biology and ecology of pathogen produce interactions. Although these challenges are being addressed, as evidenced by the number of publications directed at produce, there is a disparity in the types of produce being investigated. Those produce items that are primarily targeted in food safety studies are those that have been involved in recurrent outbreaks including leafy greens (lettuce and spinach), tomatoes, melons, and sprouts. The large per capita consumption of these items, however, may contribute to greater exposure of outbreaks occurring with these commodities. The risk associated with commodities that have a lower per capita consumption, however, should not be overlooked. Hence, it is the purpose of this review to summarize data and issues regarding the microbiological safety associated with the produce items of cabbage, carrots, celery, and onions. Although each of these items may be consumed individually, they are more often consumed as an ingredient in a number of mayonnaise-based salads including ham salad, chicken salad, potato salad, coleslaw, and seafood salad. Consequently, this review will also focus on the issues associated with the microbiological safety of these multi-ingredient food items.

The 1st topic addressed in this review is a summary of the etiology of outbreaks within the United States from 2000 to 2007 that have been epidemiologically linked to the vehicles of cabbage, carrots, celery, onions, or mayonnaise-based salads in which these produce items are ingredients. The prevalence of foodborne pathogens in these same types of products, both domestically and internationally, is then presented as a significant portion of these foods is imported into the United States. These prevalence data are followed by a short discussion on the sources of contamination, both preharvest and postharvest. Survival and growth of pathogens in these food groups is then addressed along with factors that affect the organism's response to environmental conditions. Prior to describing intervention studies that have been undertaken to

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doi 10.1111/j.1541-4337.2010.00129.x

reduce or eliminate this contamination, a brief review of laboratory studies addressing the sites of contamination (surface versus internalized) is provided as the location of pathogen contamination could affect the effectiveness of different interventions. Finally, postharvest interventions are addressed with chemical and irradiation interventions summarized largely in tabular form followed by a brief discussion on the successes and failures of biological interventions.

Outbreaks

An important consideration when addressing the safety of any food product is the prevalence of pathogens and the number of outbreaks associated with those products. An outbreak is defined as 2 or more cases of the same illness in which an epidemiologic investigation implicates the same food item as the vehicle (CDC 2000). Many outbreaks are never recognized because of their small size, long incubation period, or geographic or temporal dispersions. In addition, although the Centers for Disease Control and Prevention (CDC) is charged with nationwide surveillance of outbreaks, outbreaks are first investigated by state and local departments and variation between states exist in the diseases tracked as well as their reporting requirements for health providers (GAO 2001). Implementation and collection of data within the Electronic Foodborne Outbreak Reporting System (eFORS) and more recently through the National Outbreak Reporting System (NORS) has facilitated improvements in reporting and tracking of outbreaks; however, epidemiologic investigations are complicated by the long incubation periods associated with *Cyclospora*, hepatitis A, and *Listeria* for which consumers are asked to recall specific food exposures weeks after the onset of illness. Also, consumer recall of exposures can be poor for inconspicuous ingredients in multifood items such as salads.

According to Sivapalasingam and others (2004), produce-associated outbreaks accounted for an increasing proportion of all reported foodborne outbreaks in the United States with a known food item, rising from 0.7% in the 1970s to 6% in the 1990s. More recent data from the Center for Science in the Public Interest (CSPI) database indicate produce outbreaks accounted for 13% of foodborne illness outbreaks in the United States between 1990 and 2005 (DeWaal and Bhuiya 2007). In contrast, fresh produce accounted for 4% of all foodborne outbreaks reported from 2001 to 2005 in Australia (Kirk and others 2008). A similar percentage of foodborne outbreaks in England and Wales between 1992 and 2006 were attributed to consumption of prepared salads (Little and Gillespie 2008). Several factors may contribute to an increase in reporting of produce outbreaks including changes in consumer food preference, food production and distribution practices, and the emergence of new foodborne pathogens (Lynch and others 2009). Implementation in 1996 of PulseNet, a molecular surveillance network for foodborne infections in the United States, has also been instrumental to an increased number of reported outbreaks (Gerner-Smidt and others 2006).

To better understand the sources of foodborne illness, the proportion of human cases of specific enteric diseases attributable to broad food categories has become a common practice (Greig and Ravel 2009; Painter and others 2009; Ravel and others 2009). Similarly, in an effort to focus on the specific produce items being addressed in this review, the raw CDC outbreak data from 2000 to 2007 were screened and information on outbreaks attributed to cabbage, carrots, celery, onions, and mayonnaise-based salads are summarized (Table 1 to 3). In compiling these data, multiple vehicles may have been identified with a specific outbreak; however, if cabbage, carrots, celery, or onions were identified in that list, they were included as an outbreak with each respective produce item. In addition, a separate vehicle category entitled "other vegetable-based salads" was included in Table 3, involving foodborne outbreaks associated with green, garden, house, vegetable-based, lettuce-based, prepackaged, pasta, macaroni, antipasta, Greek, Waldorf, Oriental, 7-layered, specialty, and multiple salads, as well as salad bars, mixed vegetables, and vegetable trays. This category was included because cabbage, celery, green onions, and carrots are all common components in these salad/vegetable items.

Very few outbreaks were linked directly to cabbage, carrots, celery, or onions/green onions (Table 1). Among these 4 produce types, carrots had the greatest number of outbreaks and celery had the least. Largely due to the outbreak associated with hepatitis A in 2003, the category of onions/green onions had the greatest number of illnesses. For all 4 produce types, more than 50% of the outbreaks with known etiology were attributed to either hepatitis A or norovirus, indicating the food was contaminated by infected humans, either through improper exposure to sewage during growing or processing or because of poor personal hygiene practices among food handlers. In cases of known

Table 1–Summary of outbreaks and illnesses (2000 to 2007) in the United States associated with cabbage, carrots, celery, and onions as a function of their etiology.[a]

	Cabbage		Carrots		Celery		Onion/green onions	
Etiology	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of Illnesses
Bacillus cereus, suspected	–	–	–	–	–	–	1	4
Campylobacter jejuni	–	–	–	–	–	–	1	28
Clostridium botulinum	–	–	1	2	–	–	–	–
Escherichia coli O157:H7[b]	1	41	–	–	–	–	–	–
Hepatitis A	1	16	–	–	–	–	5	967
Norovirus	2	52	4	126	4	168	1	13
Norovirus, suspected	1	26	7	199	2	23	2	77
Salmonella spp.	1	8	1	8	–	–	2	182
Salmonella spp., suspected	–	–	–	–	–	–	1	2
Shigella sonnei	–	–	1	7	–	–	–	–
Staphylococcus aureus, suspected	1	2	–	–	–	–	–	–
Multiple pathogens, suspected	–	–	1	6	–	–	–	–
Unknown[c]	2	10	3	57	–	–	2	26
Total	9	155	18	405	6	191	15	1299

[a] Data compiled from the CDC web site on outbreak surveillance (http://www.cdc.gov/outbreaknet/surveillance_data.html).
[b] Includes other Shiga toxin-producing *Escherichia coli*.
[c] Includes "Other bacterial, suspected" and "Viral, suspected."

Table 2–Summary of outbreaks and illnesses (2000 to 2007) in the United States associated with chicken, ham, seafood, and other meat-based salads as a function of their etiology.[a]

Etiology	Chicken salad		Ham salad		Seafood salad		Meat-based salads	
	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses
Campylobacter jejuni	–	–	–	–	1	136	–	–
Clostridium perfringens	3	145	–	–	–	–	–	–
Cyclospora cayetanensis	–	–	–	–	1	56	–	–
Giardia lamblia	1	6	–	–	–	–	–	–
Norovirus	11	414	–	–	11	783	2	30
Norovirus, suspected	9	89	–	–	7	154	–	–
Rotavirus	–	–	–	–	1	5	–	–
Salmonella spp.	4	92	–	–	4	611	–	–
Salmonella spp., suspected	5	49	–	–	2	20	–	–
Scombroid poisoning	–	–	–	–	2	5	–	–
Scombroid poisoning, suspected	–	–	–	–	1	2	–	–
Shigella sonnei, suspected	1	200	–	–	–	–	–	–
Staphylococcus aureus	3	129	–	–	–	–	–	–
Staphylococcus aureus, suspected	2	6	–	–	2	6	1	2
Vibrio parahaemolyticus	–	–	–	–	1	2	–	–
Vibrio spp., suspected	–	–	–	–	1	9	–	–
Yersinia enterocolitica	–	–	1	4	–	–	–	–
Multiple pathogens, suspected	1	21	–	–	–	–	–	–
Unknown[b]	18	78	1	3	16	147	2	4
Total	58	1229	2	7	51	1936	5	36

[a] Data compiled from the CDC web site on outbreak surveillance (http://www.cdc.gov/outbreaknet/surveillance_data.html).
[b] Includes "Other bacterial, suspected" and "Viral, suspected."

Table 3–Summary of outbreaks and illnesses (2000 to 2007) in the United States associated with coleslaw, potato salad, and other vegetable-based salads as a function of their etiology.[a]

Etiology	Coleslaw		Potato salad		Other vegetable-based salads[b]	
	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses	Nr of outbreaks	Nr of illnesses
Bacillus cereus	–	–	–	–	2	6
Bacillus cereus, suspected	1	4	–	–	3	9
Bacterial toxin, suspected	–	–	–	–	1	4
Campylobacter jejuni	–	–	4	74	3	26
Campylobacter jejuni suspected	–	–	–	–	3	12
Clostridium perfringens	–	–	1	34	1	22
Clostridium perfringens, suspected	–	–	–	–	3	39
Cryptosporidium parvum	1	8	–	–	–	–
Cyclospora cayetanensis	–	–	–	–	2	146
Escherichia coli O157:H7[c]	–	–	3	77	9	135
Escherichia coli O157:H7, suspected	–	–	–	–	1	7
Giardia lamblia	–	–	–	–	2	65
Hepatitis A	–	–	–	–	2	21
Norovirus	11	441	13	1037	145	7519
Norovirus, suspected	8	151	10	221	122	2983
Salmonella spp.	1	26	7	114	18	372
Salmonella spp., suspected	–	–	–	–	4	90
Sapovirus	–	–	–	–	1	21
Shigella sonnei	1	6	2	15	4	127
Shigella sonnei, suspected	–	–	–	–	1	12
Staphylococcus aureus	–	–	2	131	1	59
Staphylococcus aureus, suspected	3	18	7	113	5	62
Multiple pathogens	–	–	2	48	–	–
Multiple pathogens, suspected	–	–	3	91	3	8
Unknown[d]	16	98	10	260	104	1409
Total	42	752	64	2215	440	13154

[a] Data compiled from the CDC web site on outbreak surveillance (http://www.cdc.gov/outbreaknet/surveillance_data.html).
[b] Includes green, garden, house, vegetable-based, prepackaged, pasta, macaroni, antipasta, Greek, Waldorf, Oriental, 7-layered, specialty, and multiple salads, as well as salad bars, mixed vegetables, and vegetable trays.
[c] Includes other Shiga toxin-producing *Escherichia coli*.
[d] Includes "Other bacterial, suspected" and "Viral, suspected."

etiology, viral agents were also implicated in most of the outbreaks when coleslaw, chicken, seafood, and other vegetable-based salads were vehicles (Table 2 and 3). In contrast, the major known etiological agent in outbreaks associated with potato salad was bacteria, with *Salmonella* spp. being the dominant group (Table 2 and 3). The significance of the dominance of these food-pathogen combinations is still tentative because one-half of all outbreaks had unconfirmed etiologies. The large proportion of outbreaks associated with the multi-ingredient category of "other vegetable-based salads" is also evidence of our inability to identify a specific produce commodity as being a more dominant source of contamination. As an example where multiple produce items were implicated, 3 separate salad bar ingredients (whole carrots, grated carrots, and red peppers) were found to be likely sources of a food-borne outbreak of *Cryptosporidium hominis* (Ethelberg and others 2009).

Traceback to the source of contamination was uncommon for the vast majority of outbreaks associated with produce items. In many outbreaks, the suspected source of contamination was based on practices that were documented to have occurred in the food preparation facility. For example, in 1994, an outbreak of *Clostridium perfringens* gastroenteritis at a large teaching hospital affected at least 52 workers and the investigators attributed inadequate cooling of ingredients prior to mixing of tuna salad as the contributing factor to causing the outbreak (Khatib and others 1994). In another outbreak of gastroenteritis at the U.S. Air Force Academy, the celery component of chicken salad had been exposed to nonpotable water (Warner and others 1991). In more recent outbreak investigations, phenotypic and molecular techniques have been used to differentiate the outbreak strain from other strains circulating in the environment. For example, in May 1997, numerous cases of febrile gastrointestinal illness were reported among the students and staff of 2 primary schools in Northern Italy, all of whom had eaten at cafeterias served by the same caterer. Isolates of *Listeria monocytogenes*, serotype 4b, from both the caterer's sample of a tuna salad and from environmental specimens collected at the catering plant, were found to be indistinguishable based on DNA analysis (Aureli and others 2000). An outbreak of *Yersinia pseudotuberculosis* infection in Finland revealed 39 isolates from stool specimens and 5 isolates from 12 environmental samples (obtained from peeling and washing equipment at the production farm that contained carrot residue) were indistinguishable by pulsed-field gel electrophoresis (PFGE) (Jalava and others 2006). In a subsequent outbreak involving *Y. pseudotuberculosis* and grated carrots served at school lunches in Finland, however, traceback investigations only led to the carrot distributor's storage facility and the original source and the mechanism of the contamination remained unclear (Rimhanen-Finne and others 2009). Traceback to the farm using molecular techniques has been possible as exemplified in the following examples involving leafy greens: (1) lettuce contaminated with *Salmonella* Newport and responsible for 19 cases of infection in the United Kingdom was traced directly to the field lizard population at the source (Sagoo and others 2003); and (2) *Escherichia coli* O157:H7 isolates from 3 soil samples, 2 river water samples, 15 cattle feces samples, and 8 wild pig feces samples on one ranch used for growing spinach had indistinguishable PFGE patterns as the implicated outbreak strain in the 2006 U.S. spinach outbreak (Jay and others 2007).

Prevalence of Pathogens

Unlike commodities such as beef and chicken that are rigorously assayed with well-validated procedures, many methods to detect pathogens on fresh produce are not validated, and the sporadic nature of most produce contamination further limits the effectiveness of testing. In spite of these limitations, many studies have been conducted to determine the prevalence of pathogens on cabbage, carrots, celery, onions, and mayonnaise-based salads (Table 4 to 8). In general, within the United States and other developed countries, the pathogens tested were either absent or infrequent contaminants in these commodities. The exception was *L. monocytogenes* in mayonnaise-based salads, in which the mean percent prevalence of the 12 surveys was 10.4%. In contrast, there was generally a very low prevalence of *L. monocytogenes* on produce items used in these salads in the United States and other

Table 4–Prevalence of pathogens or surrogates in cabbage, postharvest.

Pathogen or surrogate	Country	Sampling target	Prevalence (nr of) positive/nr of samples	% prevalence	Reference
Ascaris eggs	Ghana	Retail fruit and vegetable markets	33/60	55.0	Amoah and others 2006
Campylobacter spp.	Ireland	Supermarkets	0/4	0	McMahon and Wilson 2001
C. botulinum	U.S.	Florida, New York, California, vegetable processors	1/337	0.3	Lilly and others 1996
Cryptosporidium	Costa Rica	Open markets	0/80	0	Monge and Chinchilla 1996
E. coli	U.S.	Minnesota/Wisconsin, organic farms	4/49	8.2	Mukherjee and others 2006
		Minnesota, certified organic farms	0/9	0	Mukherjee and others 2004
		Minnesota/Wisconsin, semiorganic farms	10/81	12.3	Mukherjee and others 2006
		Minnesota, noncertified organic farms	4/30	13.3	Mukherjee and others 2004
		Minnesota/Wisconsin, conventional farms	4/68	5.9	Mukherjee and others 2006
		Minnesota, conventional farms	0/15	0	Mukherjee and others 2004
		Southern U.S., farms, and packing sheds	17/58	29.3	Ailes and others 2008
	India	Street vendors	4/8	50.0	Viswanathan and Kaur 2001
	Nigeria	Abattoir and open traditional markets	48/72	66.7	Enabulele and Uraih 2009
E. coli O157:H7	Ireland	Supermarkets	0/4	0	McMahon and Wilson 2001
Helminth eggs	Turkey	Wholesale markets	0/14	0	Kozan and others 2005
L. monocytogenes	U.S.	Texas, packing sheds	7/150	4.7	Prazak and others 2002
	Chile	Ready-to-eat salads prepared at supermarkets	0/5	0	Cordano and Jacquet 2009
Listeria spp.	U.S.	Minnesota, supermarkets	2/92	2.2	Heisick and others 1989
	India	Local markets	4/4	100	Pingulkar and others 2001
	Ireland	Supermarkets	0/4	0	McMahon and Wilson 2001
Salmonella spp.	India	Street vendors	2/8	25.0	Viswanathan and Kaur 2001
	Ireland	Supermarkets	0/4	0	McMahon and Wilson 2001
	Mexico	Central Produce Supply Station, Mexico City	1/100	1.0	Quiroz-Santiago and others 2009
	Spain	Farms, wholesale and supermarkets	7/41	17.1	Garcia-Villanova Ruiz and others 1987
S. aureus	India	Street vendors	4/8	50.0	Viswanathan and Kaur 2001
Yersinia spp.	India	Local markets	4/4	100	Pingulkar and others 2001

Table 5–Prevalence of pathogens or surrogates in carrots, postharvest.

Pathogen or surrogate	Country	Sampling target	Prevalence (nr of) positive/nr of samples	% prevalence	Reference
Campylobacter spp.	Ireland	Supermarkets	0/13	0	McMahon and Wilson 2001
C. botulinum	U.S.	Florida, New York, California, vegetable processors	0/7	0	Lilly and others 1996
Cryptosporidium spp.	Costa Rica	Open markets	1/80	1.2	Monge and Chinchilla 1996
E. coli	U.S.	Texas, processing sheds	2/25	8.0	Endley and others 2003
	U.S.	Western states, retail outlets	3/36	8.3	Lopes and others 2007
	Canada	Farmers' markets	9/206	4.4	Bohaychuk and others 2009
	India	Mumbai market	6/8	75.0	Bandekar and others 2005
		Street vendors	6/8	75.0	Viswanathan and Kaur 2001
		Vegetable vendors	36/258	14.0	Singh and others 2007
	Spain	Retail establishments	0/18	0	Abadias and others 2008
E. coli O157:H7	India	Mumbai market	0/8	0	Bandekar and others 2005
	Ireland	Supermarkets	0/13	0	McMahon and Wilson 2001
Helminth eggs	Turkey	Wholesale markets	1/40	2.5	Kozan and others 2005
L. monocytogenes	Canada	Ontario, vegetable processor	0/35	0	Odumeru and others 1997
	Chile	Ready-to-eat salads prepared at supermarkets	0/6	0	Cordano and Jacquet 2009
	Spain	Retail establishments	0/18	0	Abadias and others 2008
Listeria spp.	U.S.	Western states, retail outlets	0/36	0	Lopes and others 2007
	India	Local markets	4/4	100	Pingulkar and others 2001
	Ireland	Supermarkets	0/13	0	McMahon and Wilson 2001
MS2 coliphage	U.S.	Texas, processing sheds	14/25	56.0	Endley and others 2003
Salmonella spp.	U.S.	Texas, processing sheds	0/75	0	Endley and others 2003
		Western states, retail outlets	0/36	0	Lopes and others 2007
	India	Mumbai market	0/8	0	Bandekar and others 2005
		Street vendors	2/8	25.0	Viswanathan and Kaur 2001
		Vegetable vendors	11/258	4.3	Singh and others 2007
	Ireland	Supermarkets	0/13	0	McMahon and Wilson 2001
	Spain	Retail establishments	0/18	0	Abadias and others 2008
Shigella spp.	U.S.	Texas, processing sheds	0/75	0	Endley and others 2003
S. aureus	India	Street vendors	2/8	25.0	Viswanathan and Kaur 2001
Yersinia spp.	India	Local markets	4/4	100	Pingulkar and others 2001

Table 6–Prevalence of pathogens or surrogates in celery, postharvest.

Pathogen or surrogate	Country	Sampling target	Prevalence (nr of) positive/nr of samples	% prevalence	Reference
Ascaris lumbricoides	India	Periurban area of Titagarh	7/28	25.0	Gupta and others 2009
Campylobacter	U.S.	Washington D.C. retail markets	0/12	0	Thunberg and others 2002
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
E. coli	U.S.	Washington D.C. retail markets	0/10	0	Thunberg and others 2002
		Southern U.S., farms and packing sheds	1/44	2.2	Ailes and others 2008
	India	Street vendors	0/8	0	Viswanathan and Kaur 2001
E. coli O157:H7	U.S.	Packers	0/95	0	FDA 2001
		Product imported from Canada and Mexico	0/84	0	FDA 1999
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
Hookworm	India	Periurban area of Titagarh	0/28	0	Gupta and others 2009
L. monocytogenes	Canada	Ontario, vegetable processor	0/35	0	Odumeru and others 1997
	Chile	Ready-to-eat salads prepared at supermarkets	2/13	15.4	Cordano and Jacquet 2009
Listeria spp.	U.S.	Washington, D.C. retail markets	3/6	50.0	Thunberg and others 2002
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
Salmonella spp.	U.S.	Washington D.C. retail markets	0/12	0	Thunberg and others 2002
		Product imported from Canada and Mexico	1/84	1.2	FDA 1999
		Packers	0/95	0	FDA 2001
	India	Street vendors	5/8	62.5	Viswanathan and Kaur 2001
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
	Mexico	Central Produce Supply Station, Mexico City	3/100	3.0	Quiroz-Santiago and others 2009
	Netherlands	Domestic and imported	0/20	0	Tamminga and others 1978
	Spain	Farms, wholesale, and supermarkets	2/26	7.7	Garcia-Villanova Ruiz and others 1987
Shigella spp.	U.S.	Product imported from Canada and Mexico	2/84	2.4	FDA 1999
		Packers	0/95	0	FDA 2001
S. aureus	India	Street vendors	8/8	100	Viswanathan and Kaur 2001
Trichuris trichura	India	Periurban area of Titagarh	1/28	3.6	Gupta and others 2009

Table 7–Prevalence of pathogens or surrogates in onions or scallions, postharvest.

Pathogen or surrogate	Country	Sampling target	Prevalence (nr of) positive/nr of samples	% prevalence	Reference
Ascaris eggs	Ghana	Retail fruit and vegetable markets	39/60	65.0	Amoah and others 2006
Campylobacter spp.	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
C. botulinum	U.S.	Florida, New York, California, vegetable processors	0/4	0	Lilly and others 1996
E. coli	U.S.	Minnesota, certified organic farms	0/1	0	Mukherjee and others 2004
		Minnesota, noncertified organic farms	3/8	37.5	Mukherjee and others 2004
		Minnesota, conventional farms	0/7	0	Mukherjee and others 2004
		Western states, retail outlets	2/25	8.0	Lopes and others 2007
	Canada	Retail distribution centers/farmers' markets	11/173	6.4	Arthur and others 2007
	Canada	Farmers' markets, Alberta	7/129	5.4	Bohaychuk and others 2009
E. coli O157:H7	U.S.	Product imported from Canada and Mexico	0/180	0	FDA 1999
		Packers	0/73	0	FDA 2001
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
Helminth eggs	Turkey	Wholesale markets	1/15	6.7	Kozan and others 2005
Listeria spp.	U.S.	Western states, retail outlets	0/25	0	Lopes and others 2007
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
Salmonella spp.	U.S.	Western states, retail outlets	0/25	0	Lopes and others 2007
		Product imported from Canada and Mexico	1/180	0.6	FDA 1999
		Packers	0/73	0	FDA 2001
	Ireland	Supermarkets	0/6	0	McMahon and Wilson 2001
	Mexico	Central Produce Supply Station, Mexico City	0/100	0	Quiroz-Santiago and others 2009
Shigella spp.	U.S.	Product imported from Canada and Mexico	2/180	1.1	FDA 1999
		Packers	3/73	4.1	FDA 2001

Table 8–Prevalence of pathogens in mayonnaise-based salads.

Mayonnaise-based salad	Pathogen	Country	Sampling target	Prevalence (nr of) positive/nr of samples	% prevalence	Reference
Chicken salad	*L. monocytogenes*	Belgium	Supermarkets	41/152	27.0	Uyttendaele and others 1999
		U.K.	Supermarkets, sandwich shops, and other retail stores	71/1070	6.6	Little and others 2007
Shredded coleslaw	*C. botulinum*	U.S.	Florida, New York, California, vegetable processors	0/72	0	Lilly and others 1996
Dry coleslaw mix	*L. monocytogenes*	Canada	Ontario, vegetable processor	1/35	2.8	Odumeru and others 1997
		Ireland	Supermarkets	10/80	12.5	Francis and O'Beirne 2006
Coleslaw	*L. monocytogenes*	Japan	Retail shops	0/6	0	Kaneko and others 1999
	B. cereus	Spain	Retail supermarkets	1/12	8.3	Valero and others 2007
Ham salad	*L. monocytogenes*	Belgium	Supermarkets	33/159	20.8	Uyttendaele and others 1999
		U.K.	Supermarkets, sandwich shops, and other retail stores	2/66	3.0	Little and others 2007
Potato salad	*L. monocytogenes*	Japan	Retail shops	0/26	0	Kaneko and others 1999
Seafood salad	*L. monocytogenes*	U.S.	Maryland/Calilfornia, retail markets	115/2446	4.7	Gombas and others 2003
		Belgium	Supermarkets	99/362	27.3	Uyttendaele and others 1999
		Iceland	Retail stores or factories	6/37	16.2	Hartemink and Georgsson 1991
		U.K.	Supermarkets, sandwich shops, and other retail stores	54/1418	3.8	Little and others 2007

developed countries. Produce in developing countries, however, had a much larger percentage prevalence of *L. monocytogenes* and other pathogens (*Ascaris* eggs, *Salmonella* spp., *S. aureus*, and *Yersinia* spp.). This prevalence likely reflects differences in agricultural and hygienic practices for those locations. Foods imported into the United States from those countries could have similarly high prevalences of contamination. Although the Food and Drug Administration has oversight of produce imported into the United States, currently only 1% and 0.2 to 0.3% of imported food shipments are given visual and laboratory inspections, respectively (Fortinne 2008). These statistics are cause for concern given that the quantities of imported produce have increased dramatically over the past decade and the major countries of origin are developing countries (Florkowski 2008). This trend is also the case for cabbage, carrots, and onions based on quantities imported in 2003 and 2007 (Table 9).

Notably absent from the surveys conducted to date on prevalence of pathogens in cabbage, carrots, celery, and onions (Table 4 to 8) are the prevalence of viruses or protozoan parasites. Limitations in methods for recovery and detection of these pathogens have likely been the major impediment to collecting these data; however, the large number of outbreaks associated with

Table 9–Quantities of cabbage, carrots, celery, and onions imported into the United States in 2003 and 2007 and the major countries of origin (USDA 2008).

Produce item	Countries of origin	million tons 2003	2007
Cabbage	World total	35272	52637
	Canada	21901	32427
	Mexico	12180	18148
	Costa Rica	1090	2003
Carrots	World total	84914	111355
	Canada	66954	72249
	Mexico	16393	33420
	Costa Rica	1434	4871
Celery	World total	27063	28859
	Mexico	21533	19597
	Canada	5527	8886
Onions	World total	296783	415201
	Mexico	170686	217352
	Canada	52318	70126
	Peru	43602	63413

viruses and the resistance of parasites to chlorine-based treatments (Erickson and Ortega 2006) warrants the investigation of these products. For example, in a study in which other types of produce (strawberries, raspberries, sprouts, parsley, lettuce, radish, leek, and dill) were assayed, there was a high prevalence (11.2%, 9/80) of human-virulent microsporidia contamination (Jedrzejewski and others 2007).

Sources of Contamination

Preharvest sources of contamination

Due to the low prevalence of pathogen contamination on retail produce, information regarding potential sources of preharvest contamination are mainly obtained from experimental studies. Several reviews have summarized these studies, describing sources such as contaminated manure, manure compost, sewage sludge, irrigation water, runoff water from livestock operations, and wild and domestic animals (Steele and Odumeru 2004; Beuchat 2006; Delaquis and others 2007; Doyle and Erickson 2008). In addition, a number of other indirect sources of contamination have been identified and include trophic interactions between plants and plant foragers (Sasaki and others 2000; Sela and others 2005; Kenney and others 2006; Sproston and others 2006). For the purposes of this review, those studies addressing cabbage, carrots, celery, or onions are summarized below.

(1) In fields, cabbage, onion, and carrots irrigated with highly polluted effluents had elevated cell numbers of indicator microorganisms. In contrast, vegetables irrigated with slightly polluted effluents had significantly lower cell numbers of indicator microorganisms (Armon and others 1994).

(2) *Escherichia coli* was isolated from cabbage roots but not from the edible portion, when cabbage plants were irrigated with contaminated creek water (Wachtel and others 2002).

(3) Contamination of carrots at harvest occurred when *S. enterica* and *E. coli*-containing manures were applied to soils under conditions simulating warm (daily average maximum temperature of >20 °C) summer conditions. In contrast, the pathogens were not present in carrots harvested in soil to which nonsterile manure had been applied and subjected to repeated freeze-thaw cycles (Natvig and others 2002).

(4) Three of 20 carrot samples were positive for *E. coli* when harvested from fields irrigated with feces-contaminated streams (Okafo and others 2003).

(5) Survival of *E. coli* O157:H7 in soil and on the plant is dependent on the type of plant grown, as exemplified when baby carrot and green onion seedlings were planted into a manure compost-amended soil contaminated with the pathogen. Within 64 d, populations of *E. coli* O157:H7 decreased by 3 log CFU/g in soil beneath the roots of green onions, while it decreased only 2.3 log in soil beneath carrots after 84 d. On plants, populations of *E. coli* O157:H7 decreased by 2 log CFU/g on onions (64 d) and 1.7 log CFU/g on carrots (84 d) (Islam and others 2004a).

(6) At least one enrichment-*E. coli*-negative carrot sample was obtained ≤ 100 d after manure application for 63% of the treatments; however, 100% of the treatments had one or more enrichment-*E. coli*-negative carrot sample when sampled at 120 d or more after manure application (Ingham and others 2004).

(7) In a series of field studies, *E. coli* O157:H7-contaminated composts were applied to plots prior to sowing either carrots or onions, while *Salmonella*-contaminated composts were applied to plots prior to sowing carrots. Using separate plots, contaminated irrigation water was also applied 3 wk after the plants were sown. Survival profiles of *E. coli* O157:H7 and *Salmonella* spp. on vegetables and soil samples from plots contaminated via these 2 routes were similar. In soil, *E. coli* O157:H7 survived for 154 to 196 d and was detected for 74 and 168 d on onions and carrots, respectively (Islam and others 2005). Salmonellae persisted for a longer time, with detection of the pathogen in soil samples for 231 d and detection on carrots for 203 d after sowing (Islam and others 2004b).

(8) When introduced on carrot seeds, *E. coli* O157:H7, *S. typhimurium*, and *L. monocytogenes* became rapidly established shortly after germination, attaining cell densities of 5.5 to 6.5 log CFU/g. Based on scanning electron micrographs of these germinated root seedlings, *E. coli* O157:H7 preferentially colonized the root junctions (Jablasone and others 2005).

(9) Chemical interventions (ozone gas, acidified sodium chlorite, or quaternary ammonium) were ineffective in inactivating *L. monocytogenes* inoculated on celery seeds. In spite of this failure, no *L. monocytogenes* bacteria were recovered on any of the seedlings, possibly because of the growth-suppressing effect of endogenous microflora. In contrast, ozone inactivated *E. coli* inoculated on celery seeds (Warriner and others 2005).

(10) Based on quantitative microbial risk assessment models for enteric virus infections, the annual risk of infection associated with consuming raw cabbage ranged from 10^{-2} to 10^{-3} when reclaimed water irrigation ceased 1 d before harvest, and from 10^{-6} to 10^{-8} when it ceased 2 wk before harvest (Hamilton and others 2006).

Postharvest sources of contamination

Upon harvest of field crops, a series of processing steps are applied to the product that in general includes trimming, peeling, cutting, shredding, washing, dewatering, and packaging. Such fresh-cut processing operations are critical to converting the crop into a consumable product; however, they also create surfaces upon which enteric pathogens can more easily attach as well as release large amounts of nutrient-laden liquids that are more readily utilized by the attached microbes. For example, a study comparing the attachment of 24 *Listeria* strains to cabbage tissue revealed all strains had a preference to attach to cut tissues at levels of 1.0 to 1.2 log CFU/cm^2 greater than those on intact tissue (Ells and Hansen

2006). In addition to increased cell numbers at cut edges, scanning electron microscopy revealed numerous cells within folds and crevices, whereas cells on the intact surface appeared to be randomly distributed with no apparent affinity for specialized surface structures such as stomata. Attachment, however, appeared to be dependent on the strain as well as each strain's prior growth temperature (Ells and Hansen 2006).

Although a product containing pathogens may enter packing sheds or fresh-cut processing operations, it may also become contaminated within these processing facilities through contact with tainted water or contaminated equipment surfaces. For example, in one study more than 3 log plaque forming unit (PFU) of the surrogate murine norovirus was transmitted to onion bulbs, when these vegetables were washed in water containing approximately 5 log PFU/mL (Baert and others 2008). In another study, common sources of contamination were implicated when isolates from environmental sponge samples of conveyor belts in packing sheds and from cabbage samples had indistinguishable PFGE profiles (Prazak and others 2002). Contamination within the packing shed or processing plant, however, can also come from incoming products. For example, in a fresh celery packinghouse, bacterial genera isolated from equipment were similar to those found on celery plants sampled at the packinghouse entrance (Robbs and others 1996). Hence, cross-contamination events can be common during the processing of fresh-cut vegetables, and a small amount of contaminated product could eventually lead to contamination of a large lot.

Once the product leaves the fresh-cut processing facility, the potential for contamination continues at food service outlets (restaurants and delis) and in the consumer's home where it may be handled frequently. Under these conditions, transfer of pathogens from hands to food may occur as found in the transfer of hepatitis A virus from artificially contaminated fingerpads of adults to pieces of fresh lettuce (Bidawid and others 2000). Pathogen transfer may be minimized by using antibacterial soap compared to plain soap (Fischler and others 2007), but it requires that the handler engage in proper hygiene that does not always occur. As an example of improper hygienic practices, a pilot study recently revealed that in 5 delis surveyed, employees did not wash their hands properly or at appropriate times during production and service. In addition, even when hands were washed, most of the employees only used 1 or 2 steps of the 3-step handwashing procedure described in the 2005 Food Code (Paez and others 2007). A similar observation was made in a study of 2000 randomly selected households in the United States where almost half of the respondents indicated that they did not wash their hands before handling fresh produce (Li-Cohen and Bruhn 2002). Given these practices, a panel of scientists with expertise in microbial safety of fresh produce concluded that sealed bags of leafy green salads, labeled "washed" or "ready-to-eat," and produced in a facility inspected by a regulatory authority and operated under good manufacturing practices, do not need additional washing at the time of use unless specifically directed on the label (Palumbo and others 2007). Additional washing of these products would not enhance safety and, in fact, may confer additional risk due to cross-contamination by food handlers.

Survival and Growth of Pathogens

Produce (cabbage, carrots, celery, and onions)

The significance of contamination of product during preharvest or processing is greatly influenced by the pathogen's infectious dose and its fate during storage. With pathogens that have a low infectious dose, such as *E. coli* O157:H7, their presence is unacceptable and contaminated products should not be distributed regardless of the pathogen's fate. With pathogens whose infectious dose is high, such as *B. cereus*, distribution of product contaminated with low populations may not be harmful unless storage conditions contribute to the pathogen's growth.

In general, factors that have been demonstrated to impact the fate of enteric pathogens on produce during storage include storage temperature, relative humidity, atmosphere gas composition, nutrient availability, and presence of competitive bacteria or antimicrobial compounds. Specific studies that have addressed the impact of many of these factors as well as others on the response of pathogens or pathogen surrogates contaminating cabbage, carrots, celery, and onions are presented in Table 10 to 12. No studies addressing the fate of protozoan parasites or helminthes-contaminating cabbage, carrots, celery, or onions were found.

The fate of pathogens is dependent on the type of produce as exemplified by greater survival in cabbage than raw carrots or dry coleslaw mix. (Table 10 to 12). Mechanisms responsible for the inhibitory activity of raw carrots on pathogens have been ascribed to both the antimicrobial activity of carrot tissue as well as the antagonistic action of associated microflora (Liao 2007). Similar antimicrobial mechanisms could be responsible for inactivation of pathogens in other types of produce. For example, *L. monocytogenes* increased 7-fold during storage at 4 °C on white cabbage and China cabbage, whereas in red cabbage and Savoy cabbage, a decrease in population was observed (Breer and Baumgartner 1992). Slight differences in the microarchitectural structure of the vegetable may also contribute to differences in pathogen attachment and survival. Supporting this statement, higher numbers of *Salmonella* spp. attached to Romaine lettuce compared to cabbage surfaces (Patel and Sharma 2010). Surface features have also been suggested to be important factors in growth of *L. monocytogenes* on fresh-cut cabbage (Ongeng and others 2007).

Variation in the response of a specific pathogen between experimental studies may be due to a variety of factors including strain selection. For example, in a study with coleslaw mixes, there were significant differences in survival of different strains of *L. monocytogenes*, with populations of most strains decreasing during storage (Francis and O'Beirne 2005). However, in one case, populations of serotype 1/2a strain 269 increased on coleslaw during storage at 8 °C for 10 d. Another factor likely to influence experimental results, but is often uncontrollable in an experiment, is the composition and level of background microbial flora. For example, coinoculating carrot disks with *Pseudomonas viridiflava* (spoilage organism) and *S. typhimurium* or with *S. typhimurium* only resulted in *Salmonella* populations in the coinoculated samples being approximately 3 times the levels found in samples inoculated with the pathogen only (Wells and Butterfield 1997).

Mayonnaise-based salads

Adding mayonnaise to salads that contain cabbage, carrots, celery, or onions as a major or minor ingredient can influence the fate of pathogens. Mayonnaise, by itself, is not a medium in which pathogens survive. The highest manufacturing target pH for mayonnaise-based dressings and sauces is 4.4, which is below the 4.75 pK_a of acetic acid and below the reported inhibitory pH of 4.5 for foodborne pathogens in the presence of acetic acid (Smittle 2000). Hence, the most important factors in destroying pathogenic bacteria in mayonnaise-based products are pH as adjusted with acetic acid and concentration of acetic acid in the water phase (Smittle 2000). Incorporation of other organic acids reduces

Table 10–Studies evaluating the survival and/or growth of foodborne pathogens or surrogates in cabbage.

Pathogen or surrogate	Temperature (°C)	Initial population[a] (log CFU, PFU, or TCID/g)	Population log response	Product form	Storage atmosphere	Reference
B. cereus	5	2.3	↑[b] 1.3 (5 d)	Shredded	Nitrogen or air	Koseki and Itoh 2002
E. coli	4	8	↓[c] 1.0 (5 d)	Cut pieces	Air	Allwood and others 2004
	25		↓ 1.0 (6.5 d)			
	37		↓ 1.0 (5.5 d)			
Cronobacter sakazakii	4	2.7	↓ 0.3 (6 d)	Cut pieces	Air	Kim and Beuchat 2005
	12		↑ 3.0 (3 d)			
	25		↑ 5.9 (3 d)			
Listeria innocua	3	4.2	↓ 1.3 (12 d)	Shredded	MA[d]	Bourke and O'Beirne 2004
	8	4.1	No change (12 d)			
Salmonella hadar	4	4.2	↓ 0.1 (10 d)	Shredded	MA	Piagentini and others 1997
	12		↑ 3.6 (10 d)			
	20		↑ 6.8 (10 d)			
F-specific coliphage MS2	4	6	↓ 1.0 (5.0 d)	Cut pieces	Air	Allwood and others 2004
	25		↓ 1.0 (3.5 d)			
	37		↓ 1.0 (3.0 d)			
Feline calicivirus	4	8	↓ 1.0 (1.5 d)	Cut pieces	Air	Allwood and others 2004
	25		↓ 1.0 (1.0 d)			
	37		↓ 1.0 (1.0 d)			
MS2 bacteriophage	4	7.2	↓ 1.4 (87 d)	Leaves	Air	Dawson and others 2005
Poliovirus	4	5.6	↓ 1.0 (14 d)	Leaves	Air	Kurdziel and others 2001

[a] CFU = colony forming unit; PFU = plaque forming unit; TCID = tissue culture infectious dose.
[b] ↑ = increase.
[c] ↓ = decrease.
[d] Modified atmosphere created by respiration.

Table 11–Studies evaluating the survival and/or growth of foodborne pathogens or surrogates in carrots, celery, or onions.

Food product	Pathogen or surrogate	Temperature (°C)	Initial population[a] (log CFU, PFU, or TCID/g)	Population log response	Product form	Storage atmosphere	Reference
Carrots	*C. jejuni*	7	6.5 to 6.9	↓[b] 0.8 to 1.7 (3 d)	Grated	Air	Kärenlampi and Hänninen 2004
		21	6.5 to 6.9	↓ 4.6 to 5.0 (3 d)			
	C. sakazakii	4	2.8	↓ 1.4 (6 d)	Cut pieces (1 cm^2)	Air	Kim and Beuchat 2005
		12	2.8	↓ 0.6 (3 d)			
		25	2.8	↑[c] 1.7 (3 d)			
	E. coli	8	6.0	↓ 2.1 to 2.3 (9 d)	Machine blade sliced, blunt or sharp knife	MA (respiration)	Gleeson and O'Beirne 2005
		8	5.8	↓ 3.7 (9 d)	Discs, razor blade sliced.	MA (respiration)	
	E. coli O157:H7	5	5.3	↓ 5.2 (7 d)	Shredded	MAP (3:97 O_2:N_2)	Abdul-Raouf and others 1993a
	L. innocua	3	3.1	↓ 1.0 (12 d)	Shredded	MA (respiration)	Bourke and O'Beirne 2004
		8	2.9	No change (12 d)			
	L. monocyto-genes	4	5.1	↓ 1.2 to 1.6 (15 d)	Shredded	Air or MAP (4.9:2.1:93 CO_2:O_2:N_2)	Kakiomenou and others 1998
		4	2.3	↓ 1.6 (9 d)	Chopped	MA (respiration)	Farber and others 1998
	Salmonella enteritidis	4	5.8	↓ 1.7 to 1.9 (15 d)	Shredded	Air or MAP (4.9:2.1:93 CO_2:O_2:N_2)	Kakiomenou and others 1998
	Hepatitis A	4	3.4	↓ >2.4 (4 d)	Cut pieces (<9 cm^2)	Air	Croci and others 2002
	MS2 bacterio-phage	4	7.1	↓ 1.9 (87 d)	Slices	Air	Dawson and others 2005
	Rotavirus	4	4.5	↓ 4.5 (30 d)	Cut pieces (5 to 15 cm^2)	Air	Badawy and others 1985
		25	4.5	↓ 4.5 (20 d)			
Celery	*E. coli*	5	5.2	↓ 1.2 (20 d)	Diced (0.5 cm thick)	Air	Prakash and others 2000
Onion	*L. monocyto-genes*	4	3.8	↓ 0.3 (9 d)	Slices	MA (respiration)	Farber and others 1998
Green onion	Poliovirus	4	3.7-4.1	No change (15 d)	Individual	Air	Kurdziel and others 2001

[a] CFU = colony forming unit; PFU = plaque forming unit; TCID = tissue culture infectious dose.
[b] ↓ = decrease.
[c] ↑ = increase.

Table 12–Studies evaluating the survival and/or growth of foodborne pathogens or surrogates in mixed vegetables.[a]

Food product	Pathogen or surrogate	Temperature (°C)	Initial population (log CFU/g)	Population log response	Reference
Dry coleslaw mix[b]	*B. cereus*	7	4.6	↓[d] 4.6 (6 d)	Finn and Upton 1997
	C. perfringens	7	5.6	↓ 3.7 (8 d)	
	E. coli O157:H7	4	4.1	↓ 1.2-1.4 (12 d)	Francis and O'Beirne 2001
	L. innocua	3	4.1	↓ 1.8 (12 d)	Bourke and O'Beirne 2004
		8	4.2	↓ 1.4 (12 d)	
		7	6.0	↓ 1.7 (8 d)	Finn and Upton 1997
	L. monocytogenes	4	2.6	↑[e] 1.0 (9 d)	Farber and others 1998
		4	4.1	↓ 1.7-2.5 (12 d)	Francis and O'Beirne 2001
		3	4.6	↓ 2.9 (12 d)	Bourke and O'Beirne 2004
		8	4.6	↓ 4.3 (12 d)	
	S. aureus	7	5.9	↓ 2.3 (8 d)	Finn and Upton 1997
Stir-fry vegetables[c]	*L. monocytogenes*	4	2.5	↑ 0.5 (9 d)	

[a]Stored under modified atmosphere.
[b]Shredded carrot and cabbage.
[c]Broccoli, cauliflower, carrots, and celery.
[d]↓ = decrease.
[e]↑ = increase.

Table 13–Studies evaluating the survival and/or growth of foodborne pathogens in mayonnaise-based salads.

Food product	Pathogen	Temperatue (°C)	pH	Initial population (log CFU/g)	Population log response	Reference
Coleslaw	*E. coli* O157:H7	4	4.3 to 4.5	5.3	↓[a] 0.1 to 0.2 (3 d)	Wu and others 2002
		11			↓ 0.1 to 0.2 (3 d)	
		21			↓ 0.4 to 0.5 (3 d)	
Coleslaw	*L. monocytogenes*	5	4.8	3.1	↓ 1.2 (14 d)	Burnett and others 2005
		7			↓ 1.8 (14 d)	
		10			↓ 2.5 (14 d)	
Chicken salad	*L. monocytogenes*	5	4.0	6.0	↓ 7.5 (24 d)	Guentert and others 2005
			4.6		↓ 4.0 (119 d)	
			5.2		↓ 4.0 (199 d)	
		7.2	4.0		↓ > 3.0 (119 d)	
			4.6		↓ > 3.0 (119 d)	
			5.2		↓ 1.1 (119 d)	
		21.1	4.0		↓ 6.0 (14 d)	
			4.6		↓ 6.0 (52 d)	
			5.2		↓ 6.0 (38 d)	
Ham salad	*L. monocytogenes*	4	3.8	2	↑[b] 4.0 (26 d)	Hwang 2005
			4.2		↑ 4.0 (17 d)	
			4.6		↑ 4.0 (21 d)	
		8	3.8		↑ 4.0 (7 d)	
			4.2		↑ 4.0 (8 d)	
			4.6		↑ 4.0 (9 d)	
		12	3.8 to 4.6		↑ 4.0 (4 d)	
Potato salad	*L. monocytogenes*	4	3.8	2	↓ 2.0 (10 d)	Hwang 2005
			4.2		↓ 2.0 (18 d)	
			4.6		↓ 2.0 (15 d)	
		8	3.8		↓ 2.0 (7 d)	
			4.2		↓ 2.0 (18 d)	
			4.6		↓ 2.0 (15 d)	
		12	3.8-4.6		↓ 2.0 (3 d)	
	S. sonnei	2.5	5.2	6	↓ 0.5 (28 d)	Warren and others 2007
		8			↓ 0.3 (28 d)	
Shrimp salad	*L. monocytogenes*	5	4.5	3.0	↓ 0.4 (14 d)	Burnett and others 2005
		7			↓ 0.8 (14 d)	
		10			↓ 1.4 (14 d)	
Seafood salad, mayonnaise pH 3.7	*L. monocytogenes*	4	6.3	1.5	↑ 4.5 (22 d)	Hwang and Tamplin 2005
Seafood salad, mayonnaise pH 4.0-5.1	*L. monocytogenes*	4	6.5-6.7	1.5	↑ 5.5 (22 d)	

[a]↓ = decrease.
[b]↑ = increase.

the inhibitory or lethal activity of mayonnaise against pathogens, with the order of effectiveness of acids being acetic acid > lactic acid > citric acid (Abdul-Raouf and others 1993b). Similarly, acetic acid is a much more effective acidulant than citric acid for inactivation of *S. aureus* and *L. monocytogenes* in mayonnaise-based surimi salads (Bornemeier and others 2006).

Mayonnaise-based salads contain a wide variety of components that vary in their physical and chemical properties as well as their microbiological profiles. Hence, the survival and growth response of foodborne pathogens in these mayonnaise-based salads can vary dramatically (Table 13). In coleslaw and potato salads, *L. monocytogenes* populations decreased with storage, while they increased during storage in ham and seafood salads. This growth response is likely a result of the product pH. Although acetic acid concentrations decrease in coleslaw and potato salads as a result of absorption of the acid by the vegetable tissue (Brocklehurst and Lund 1984), there is much greater capacity of protein-based meats and seafoods to buffer the pathogens from the harmful effects

Table 14–Pathogen or surrogate reduction by chlorine interventions applied to cabbage, carrots, and onions.

Food product	Treatment conditions	Pathogen or surrogate	Log reduction (CFU/g)	Reference
Cabbage	Cut Chinese cabbage leaves (3 cm^2, 100 g) were mixed with 1 L sodium hypochlorite wash solution (100 ppm) for 15 min at room temperature.	*E. coli* O157:H7	2.0	Inatsu and others 2005a
	Inoculated shredded cabbage was immersed in 100 mL of 100 ppm sodium hypochlorite, pH 6.0, for 10 min at room temperature.	*E. coli* O157:H7 *Salmonella* spp. *S. aureus*	2.7 2.4 2.5	Fukuyama and others 2009
	Inoculated shredded cabbage was immersed in 100 mL of a treatment solution containing 100 ppm sodium hypochlorite and 0.1% calcinated calcium, pH 6.0, for 20 min at room temperature.	*E. coli* O157:H7 *Salmonella* spp. *S. aureus*	3.8 >4.3 3.5	Fukuyama and others 2009
	Cut cabbage (200 g) was dipped for 10 min in a sodium chlorite solution (100 or 200 ppm) at 4° or 22 °C, stirring being done every minute, drained for 1 min, and then spin-dried for 4 min.	*L. monocytogenes*	1.3 to 1.7	Zhang and Farber 1996
	Cabbage (100 g) was washed with agitation for 5 min in a beaker containing 1 L of 100 ppm free chlorine.	MS2 bacteriophage	0.4 to 0.5	Dawson and others 2005
Carrots	Carrot shreds were dipped at a sample/wash water ratio of 1:20 (w/v) into a sodium hypochlorite solution (200 ppm) prepared in fresh tap water or simulated processing water with a chemical oxygen demand of about 3500 mg/L. Carrots were submerged for 2 min, drained, then dried in a salad spinner.	*E. coli* O157:H7	1.6 (tap water) 0.0 (process water)	Gonzalez and others 2004
	Carrots (100 g) were washed with agitation for 5 min in a beaker containing 1 L of 100 ppm free chlorine.	MS2 bacteriophage	0.8 to 1.3	Dawson and others 2005
	At a sample to sanitizer solution of 1:10 (w/v), shredded carrots were dipped with constant stirring for 1 min into a sodium hypochlorite sanitizer solution (200 ppm, pH 6.5) prepared in fresh tap water or simulated processing water with a chemical oxygen demand of about 3500 mg/L. The carrots were subsequently drained for 30 s and then spin dried for 30 s.	*E. coli* O157:H7 *Salmonella* spp. *L monocytogenes*	3.4 (tap water) 1.0 (process water) 3.4 (tap water) 1.3 (process water) 1.9 (tap water) 0 (process water)	Ruiz-Cruz and others 2007a
Coleslaw mix, dry	Coleslaw mix (250 g, 80% cabbage, 20% shredded carrot) were submerged in 100 ppm chlorine for 5 min, and then rinsed for 5 min with distilled water.	*L. innocua* *E. coli*	0.5 0.4	Francis and O'Beirne 2002
Green onions	Inoculated green onions were soaked in 500 mL of acidic electrolyzed water (free available chlorine of 37.5 ppm) for 3 min.	*E. coli* O157:H7 *S. typhimurium* *L. monocytogenes*	5.8 5.6 4.8	Park and others 2008
Spring onions	Spring onions (100 g) were washed with agitation for 5 min in a beaker containing 1 L of 100 ppm free chlorine.	MS2 bacteriophage	0.9 to 1.0	Dawson and others 2005

of acetic acid (Hwang 2005). Hence, increasing the acidity of mayonnaise in seafood salad reduces the growth rate of *L. monocytogenes*; however, significant growth still occurs to warrant concern (Table 13). To assist with growth predictions of *L. monocytogenes* under conditions typical for mayonnaise-based salads, Gysemans and others (2007) developed growth/no growth models. Similarly, to model the behavior of *L. monocytogenes* in chicken salad, a nonlinear model was developed; however, further improvements to the model were foreseen, if data could be collected and incorporated into the model to take into account the adaptive nature of *L. monocytogenes* (Guentert and others 2006).

A number of other factors can also affect the survival and/or growth of pathogens in mayonnaise-based salads, including the type of mayonnaise used. While Erickson and others (1993) concluded that no microbiological safety differences existed between salads prepared with real mayonnaise and reduced-calorie mayonnaise dressings, Hathcox and others (1995) observed that *E. coli* O157:H7 survived longer in salads prepared with real mayonnaise than with reduced-calorie mayonnaise dressings. The type of oil used to manufacture mayonnaise also impacts pathogen survival, with olive oil providing the greatest reductions in populations followed by soybean, grapeseed, rapeseed, groundnut (peanut), sunflower, hazelnut oils, and a blended olive oil (Lock and Board 1995a). Prior exposure of *Salmonella* to nonlethal acid environments or low temperatures enhances survival of the pathogen in mayonnaise (Lock and Board 1995b; Tosun and Gonul 2003) and would likely also occur in mayonnaise-based salads.

Interventions to Reduce Pathogen Populations in Cabbage, Carrots, Celery, and Onions

Internalized versus surface contamination

A critical issue that influences the effectiveness of many interventions is whether the pathogen resides on the surface or at internal locations within the product. Internalization of pathogens may arise through both preharvest and postharvest exposures. In preharvest exposure, an attenuated hepatitis A vaccine placed on either the soil or the plant itself was found within growing 2-mo-old green onions, but not on the outside after 1 and 3 wk (Chancellor and others 2006). Other studies documenting internalization have been conducted primarily with leafy greens (lettuce or spinach). Based on field studies, however, demonstration of internalization required exposure to high concentrations of *E. coli* O157:H7 (Erickson and others 2010a, 2010b) and it is unlikely that such high concentrations would be encountered naturally.

Postharvest exposures can also lead to internalization. Using confocal laser microscopy, *E. coli* O157:H7 was found entrapped 20 to 200 μm below the surface in stomata and cut edges of lettuce leaves (Seo and Frank 1999), while in sliced carrots, the pathogen was found mainly at cell junctions and in intracellular spaces up to 50 μm deep (Auty and others 2005). The location of pathogens at those depths is of concern because of the inability of

Table 15–Pathogen reduction by chemical oxidant interventions (excluding chlorine) applied to cabbage, carrots, and onions.

Food product	Chemical agent	Treatment conditions	Pathogen	Log reduction (CFU/g)	Reference
Cabbage	Chlorine dioxide	Pieces (4 cm by 4 mm) were exposed to gaseous ClO_2 (4.1 mg/L) for periods ranging from 20.5 min to 30.8 min at 22 °C.	*E. coli* O157:H7 *S. enterica* *L. monocytogenes*	3.1 4.4 3.6	Sy and others 2005
		Cut cabbage (200 g) was dipped for 10 min in a ClO_2 solution (5 ppm) at 4° or 22 °C, stirring every minute, drained for 1 min, and then spin-dried for 4 min.	*L. monocytogenes*	0.8 to 1.1	Zhang and Farber 1996
Carrot		Pieces (5 cm by 3 mm by 2 mm) were exposed to gaseous ClO_2 (4.1 mg/L) for periods ranging from 20.5 min to 30.8 min at 22 °C.	*E. coli* O157:H7 *S. enterica* *L. monocytogenes*	5.6 5.2 5.9	Sy and others 2005
Onion, Vidalia		One whole onion was exposed to gaseous ClO_2 (4.1 mg/L) for 20 min at 22 °C.	*S. enterica*	1.9	
Cabbage	Acidified sodium chlorite	Cut Chinese cabbage leaves (3 cm^2, 100 g) were mixed with 1 L of an acidified sodium chlorite wash solution (0.5 g/L) for 15 min at room temperature.	*E. coli* O157:H7	2.9	Inatsu and others 2005a
		Chinese cabbage leaves (500 g) were mixed with 5 L of acidified sodium chlorite wash solution (0.5 g/L) for 15 min at room temperature.	*E. coli* O157:H7 *S.* Enteritidis. *L. monocytogenes* *S. aureus*	2.8 2.8 2.2 2.4	Inatsu and others 2005b
Carrots		At a sample-to-sanitizer solution of 1:10 (w/v), shredded carrots were dipped into an acidified sodium chlorite sanitizer solution (100, 250, or 500 ppm; pH 2.71, 2.55, or 2.47, respectively) for one min with constant stirring, followed by draining for 30 s and spin drying for 30 s.	*E. coli* O157:H7 *Salmonella* spp. *L. monocytogenes*	4.8 4.8 2.5	Ruiz-Cruz and others 2007a
		Carrot shreds were dipped at a sample/wash water ratio of 1:20 (w/v) into an acidified sodium chlorite solution (1000 ppm) prepared in fresh tap water or simulated processing water with a chemical oxygen demand of about 3500 mg/L. Carrots were submerged for 2 min, drained, then dried in a salad spinner.	*E. coli* O157:H7	5.25	Gonzalez and others 2004
Cabbage	Ozone	Cut cabbage pieces were exposed to ozone (1.00 ppm) for 5 min at 24 °C.	*L. monocytogenes*	8.0	Fisher and others 2000
Carrots	Peroxyacetic acid	Carrot shreds were dipped at a sample/wash water ratio of 1:20 (w/v) into a peroxyacetic solution (80 ppm) prepared in fresh tap water or simulated processing water with a chemical oxygen demand of about 3500 mg/L. Carrots were submerged for 2 min, drained, then dried in a salad spinner.	*E. coli* O157:H7	1.6	Gonzalez and others 2004
		At a sample-to-sanitizer solution of 1:10 (w/v), shredded carrots were dipped into a peroxyacetic acid sanitizer solution (40 ppm, pH 3.72) for 2 min with constant stirring, followed by draining for 30s and spin-drying for 30 s.	*E. coli* O157:H7 *Salmonella* spp. *L. monocytogenes*	1.2 2.1 0.8	Ruiz-Cruz and others 2007a

most chemical and biological intervention agents to penetrate to those internalized sites using typical processing exposure periods.

Chemical interventions

Chlorine is the most common sanitizing agent used by the fresh-cut produce industry in the United States. The hypochlorite form is essentially the antibacterial agent. The recommended concentrations of chlorine range from 50 to 200 ppm with a contact time of 1 to 3 min (WHO 2008). Results of studies to evaluate the efficacy of chlorine on inactivation of pathogens on cabbage, carrots, and onions are presented in Table 14. Chlorine has minimal effect, achieving pathogen reductions primarily in the range of 1 to 2 log CFU/g. Organic matter in the wash water particularly reduces effectiveness (Ruiz-Cruz and others 2007a; Park and others 2009). Strong attachment of the pathogen to the tissue and waxy cuticle, formation of barriers (such as biofilms) that reduce the opportunity for contact of the sanitizer with the microorganisms, and inability to access the site (pores, cut surfaces, indentations, and other irregularities) where the pathogen is lodged are factors that reduce the effectiveness of chlorine or the ability to wash away the pathogen with the disinfectant (Keskinen and others 2009). Another disadvantage of using chlorine as a sanitizer is that there are no residual antimicrobial effects, therefore, the subsequent growth rates of pathogens on chlorine-treated cabbage were greater than nontreated cabbage (Koseki and Itoh 2001). Hence, the primary purpose for chlorine treatments has been to limit cross-contamination from occurring during produce washing operations.

Due to the limited effectiveness of chlorine, alternative sanitizers have been evaluated on cabbage, carrots, and onions, including chlorine dioxide, acidified sodium chlorite, ozone, peroxyacetic acid, organic acids, plant essential oils, and purified bacteriocins (Table 15 and 16). In some cases, the mode of delivery influences the effectiveness of the chemical agents such as for chlorine dioxide of which the gaseous form was much more effective than the aqueous form. Effectiveness of acidified sodium chlorite was also dependent on the type of product being washed (reduced pathogen inactivation occurred on cabbage compared to carrots) and the type of pathogen (reduced inactivation occurred with *L. monocytogenes* compared to *E. coli* O157:H7 and *Salmonella* spp.). Similarly, efficacy of essential oils is dependent on the pathogen target with gram-negative bacteria more resistant than gram-positive ones to the antagonistic effects of essential oils (Oussalah and others 2007; Romeo and others 2010).

Table 16–Pathogen reduction by organic acids, plant essential oils, or bacteriocins applied to cabbage, carrots, and onions.

Food product	Chemical agent	Treatment conditions	Pathogen	Log reduction (CFU/g)	Reference
Carrots	Lemon juice[a]	Shredded carrots (10 g) were exposed at 20 °C to 50 mL of lemon juice (100%, 75%, or 50%) for 60 min.	*S. typhimurium* *Y. enterocolitica*	2.6 to 4.0 7.2	Sengun and Karapinar 2004, 2005b
Spring onions		Shredded spring onions (10 g) were held in 50 mL of fresh lemon juice for 60 min at room temperature.	*S. typhimurium*	2.9	Sengun and Karapinar 2005a
Carrots	Vinegar[b]	Shredded carrots (10 g) were exposed at 20 °C to 50 mL of vinegar for 60 min.	*S. typhimurium*	3.3 to 3.6	Sengun and Karapinar 2004
Carrots	Lemon juice: vinegar (1:1)	Shredded carrots (10 g) were exposed at 20 °C to 50 mL of a lemon juice:vinegar mixture (1:1) for 30 min.	*S. typhimurium*	5.7 to 6.0	Sengun and Karapinar 2004, 2005b
Spring onions		Shredded spring onions (10 g) were held in 50 mL of vinegar for 60 min at room temperature.	*S. typhimurium*	2.1 to 2.9	Sengun and Karapinar 2005a
Cabbage	Bergamot, citral, or linalool essential oils	Squares (2 × 2 cm) were exposed to vapors from 1.85 mL of essential oil.	*Arcobacter butzleri* (Water isolate) (Chicken isolate)	 0.5 to 1.0 7.0	Fisher and others 2007
Carrots	Lemon verbena, cypress, or lemon-balm essential oils	Grated carrots (60 g) were exposed to 0.3 mL of 1% solution of essential oil and held in sealed bag at 8 °C for 5 d.	*E. coli* *S. aureus* *L. innocua*	1.7 to 3.0[c] 6.0 to 6.8[c] 8.0	Romeo and others 2010
Celery	Cinnamaldehyde or carvacrol, 1%	Pieces (10 g) were immersed in 1% plant extract for 10 min, then stored at 4 °C for 3 d.	*S. enterica*	2.3-7.0	Ravishankar and others 2010
Cabbage	Nisin	Cabbage pieces (25 g) were washed vigorously with agitation in 50 mL of nisin (50 μg/mL) for 1 min.	*L. monocytogenes*	2.7	Bari and others 2005a
Cabbage	Pediocin	Cabbage pieces (25 g) were washed vigorously with agitation in 50 mL of pediocin (48 mg/mL) for 1 min.	*L. monocytogenes*	1.9	Bari and others 2005a
Cabbage	Nisin and phytic acid	Cabbage pieces (25 g) were washed vigorously with agitation in 50 mL of an antimicrobial solution (nisin, 50 μg/mL; phytic acid, 0.02%) for 1 min.	*L. monocytogenes*	4.4	Bari and others 2005a

[a] 4.46%, v/v, citric acid.
[b] 4.03%, v/v, acetic acid.
[c] Similar decreases were seen in nonexposed (control) samples.

In selecting chemical interventions, consideration should be given to potential adverse effects on sensory or nutritional quality. For example, sliced carrots treated with 3% whey permeate and chlorine scored lower in acceptability due to higher surface whiteness, although these samples had lower microbial loads (Martin-Diana and others 2006). In addition, high concentrations of oxidizing agents (such as hydrogen peroxide) and/or organic acids (lactic acid) can adversely affect produce, and these effects are often magnified during storage (McWatters and others 2002; Martínez-Sánchez and others 2006). The appropriateness of a sanitizing agent, however, is dependent on the manner in which the agent is delivered as well as the target produce item. For example, gaseous chlorine dioxide caused browning of cabbage but aqueous chlorine dioxide did not affect the sensory properties of carrots (Gómez-López and others 2008). In another case where the sanitizing agent had minimal effects on nutritional and visual quality of produce, shredded carrots were sanitized with acidified sodium chlorite at 100, 250, and 500 ppm (Ruiz-Cruz and others 2006, 2007b). Improvements to the nutritional quality upon treatment with a sanitizing agent, however, have also been documented. Zhang and others (2005) observed that the vitamin C content of low-dose (0.03 to 0.08 ppm) ozonated water-treated fresh-cut celery was significantly higher than that of nontreated celery at the end of 9 d of storage. The effect of sanitizing agents on quality, though, will be impacted by the state of the product being treated. For example, washing shredded carrots resulted in increased sugar leaching and loss of sensory quality, whereas prewashing uncut carrots with chlorine ensured sugar retention and sensory quality, while providing some microbiological inactivation (Klaiber and others 2004).

Physical interventions

Due to the limited effectiveness of chemical interventions for eliminating foodborne pathogens on fresh-cut cabbage, carrots, celery, and onions, physical interventions, such as irradiation (Table 17), high-pressure processing (Arroyo and others 1999; Kingsley and others 2005), intense light pulses (Gómez-López and others 2005), and ultraviolet light (Fino and Kniel 2008), have been evaluated. Irradiation is an approved treatment of fruits and vegetables at a maximum dose of 1.0 kGy (IFT 1983). Low-dose ionizing radiation (1 to 2 kGy) may be an effective treatment for killing pathogenic bacteria in fresh-cut cabbage, carrots, and celery (Table 17). Based on data in Table 17, *L. monocytogenes* is more radiation resistant than *E. coli* in both carrots and celery. In confirmation of this order of susceptibility, D_{10} values of *E. coli* and *Y. enterocolitica* in carrot paste were in a lower range (0.12 to 0.26 kGy) compared with that of *L. monocytogenes* (0.3 to 0.5 kGy) (Kamat and others 2005). Dosages required for inactivation, however, are diminished if packages are held in a modified atmosphere during irradiation (Caillet and others 2006a, 2006b; Lacroix and others 2009), or if an antimicrobial coating is applied to the produce item prior to irradiation (Caillet and others 2006a; Lacroix and others 2009).

Similar to chemical intervention studies, irradiation studies have also addressed the effect of the treatment on sensory and nutritional qualities. For example, in the case of fresh-cut onions, samples treated with 1 kGy of radiation had similar or better sensory qualities and a reduced microbial population than the controls, whereas samples treated with 2 and 3 kGy of radiation eliminated the microflora but had increased loss of aroma and deterioration of visual quality (Fan and others 2003). As another example, carrots

Table 17–Pathogen or surrogate reduction by irradiation applied to cabbage, carrots, and celery.

Produce item	Pathogen or surrogate	Treatment dose(s) (kGy)	Initial population (log CFU/g)	Log reduction (CFU/g)	Reference
Cabbage	*Enterobacteriaceae*	1.0	5.3	3.8	López and others 2005
	E. coli	0.8	6 to 8	4 to 5	Khattak and others 2005
	L. monocytogenes	1.0	5.2	5.2	Bari and others 2005b
	Salmonella Paratyphi	1.0	6 to 8	3 to 4	Khattak and others 2005
Carrots	*E. coli* O157:H7	0.3 to 0.9	6.1	>6	Lacroix and Lafortune 2004
	L. innocua	0.25	3.2	0.6	Caillet and others 2006a
		0.50		1.3	
	L. monocytogenes	1.0	5.3	2.6	Kamat and others 2005
		2.0		>5.2	
		2.4	6.5	6.5	Caillet and others 2006b
		2.2	6.0	6.0	Lacroix and others 2009
Celery	*Enterobacteriaceae*	0.5	5.1	3.4	Prakash and others 2000
		1.0		5.1	
	E. coli	1.0	4.9	3.6	López and others 2005
	L. monocytogenes	0.5	5.5	3.1	Prakash and others 2000
		1.0		5.5	

were more tolerant than onions of higher radiation doses in that there was no significant effect on the sensory ratings following a 2-kGy dose (Hajare and others 2006); however, appearance scores decreased significantly in samples receiving a 3-kGy dose (Chaudry and others 2004).

Biological interventions

In an attempt to capitalize on the competitive advantage that indigenous bacteria may have in the suppression of undesirable microorganisms, research has been directed toward isolating and characterizing potential beneficial bacteria and applying lactic acid bacteria to salads and fresh-cut produce. For example, 14 isolates of potential beneficial bacteria were obtained from carrots, celery, green and purple cabbage, and green onions and, of these isolates, 5 had antimicrobial activity against *S. aureus*, *E. coli* O157:H7, *L. monocytogenes*, and *S.* Montevideo (Schuenzel and Harrison 2002). Studies addressing the inhibitory properties of lactic acid bacteria on foodborne pathogens in fresh-cut produce have had variable success. When *S.* Enteritidis was present in grated carrots, addition of *Lactobacillus* sp. failed to eliminate the pathogen (Tassou and Boziaris 2002). Similarly, both *E. coli* O157:H7 and *L. monocytogenes* survived on carrots treated with a cell suspension of *Lactobacillus delbrueckii* subsp. *lactis* (Harp and Gilliland 2003). Further testing in the latter study revealed that there was apparently sufficient catalase activity in the cut vegetables to destroy the antagonistic activity of hydrogen peroxide produced by the lactobacilli. The lactic acid culture was more effective as an antimicrobial in coleslaw, when used in combination with nisin in which case multiplication of *Listeria* cells was not prevented by nisin (500 to 2000 IU/g) alone but was with the protective culture added (Schillinger and others 2001).

Biocontrol of foodborne pathogens through application of virulent bacteriophage to produce is another biological intervention that shows promise. For example, phage A511 applied to sliced cabbage at 3×10^8 PFU/g decreased populations of *L. monocytogenes* from 3 log CFU/g to below 10 CFU/g (Guenther and others 2009). Unfortunately, the effect was limited as the concentration of infective particles decreased with storage and growth of *Listeria* then occurred.

Summary

Based on the number of outbreaks within the United States and the prevalence of pathogen contamination in cabbage, carrots, celery, and onions that are grown in developed countries, these commodities would appear to have minimal microbial risk associated with their consumption. This conclusion may not be warranted, however, as there is ample evidence demonstrating the potential for preharvest and postharvest contamination of these commodities. Given the ineffectiveness of chemical interventions to eliminate surface contamination and the unwillingness of consumers to purchase irradiated produce, additional research should be directed to reducing the risk of preharvest contamination and to developing more effective chemical, physical, and biological interventions.

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