

**RISKS ASSOCIATED WITH BACTERIAL
PATHOGENS IN EXPORTED
FRUIT AND VEGETABLES**

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by

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PATHOGENS IN EXPORTED
FRUIT AND VEGETABLES**

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1 INTRODUCTION

In 2001 a report was submitted to the Ministry of Agriculture and Forestry (MAF) with the intention of providing generic HACCP models to underpin food assurance programmes in the New Zealand fresh produce industry (Faulkner *et al.*, 2001). These programmes would in turn provide quality assurance to importing countries of New Zealand's fresh produce. The report highlighted the lack of data available to identify food safety hazards in fresh produce exported from New Zealand, although several potential food/hazard combinations were suggested. There was recognition of a need for research to provide information on identification and levels of food safety pathogens on fresh produce, encompassing both organic and conventional production methods.

The large cost involved in producing meaningful scientific data covering all of the fresh produce and hazards suggested in the 2001 report lead to the proposal to MAF Policy that a qualitative risk assessment process should be applied to determine which food/hazard combinations appear to pose the greatest risk for the health of overseas consumers. This report represents the output from Milestone 2 of the MAF Policy project FMA128, "Identification and establishment of the levels of food safety pathogens on fresh produce in New Zealand and intended for export". It uses a risk assessment approach to identify food/hazard combinations among the range of bacterial pathogens and produce foods that are the most likely to result in human disease.

The purpose of this milestone is "Prepare a report on risk assessment and ranking to recommend 2 produce/hazard combinations (organic vs conventional). Appropriate scientific comment on the characteristics of the identified hazards and their public health significance of their presence on the produce, will be noted and mentioned in this report". As suggested in the Expression of Interest, the risk assessment process takes careful consideration of the following points (listed in order of final discussion):

- i. Presence or absence of the pathogen in New Zealand
- ii. Availability of testing methodology
- iii. The risk the pathogen poses to human health in New Zealand
- iv. Likelihood of the pathogen occurring on a product
- v. Export value of the produce likely to host the pathogen

The report follows the general format of the "Risk Profiles" produced by ESR for the NZFSA. Normally these consider only a single food/hazard combination. Due to the multiple combinations considered here, and the short time frame, the level of detail is lower in this report. Information on potential pathogenic hazards associated with various fresh produce was collected predominantly through review of published literature.

2 HAZARD IDENTIFICATION: BACTERIAL PATHOGENS IN FRUIT AND VEGETABLES

Pathogens associated with fruit and vegetables are predominantly those from human or animal faecal sources and include *Shigella*, *Salmonella*, *E. coli* (specifically *E. coli* O157:H7) *Campylobacter* (rarely), *Yersinia enterocolitica*, *Aeromonas*, *L. monocytogenes*, *Staphylococcus*, *C. botulinum* (although only with cooked preserved vegetables), enteric viruses such as Norwalk-like virus, and parasites such as *Giardia*. A review of the association between pathogens and produce has been published (Beuchat, 1996).

Brief descriptions of produce-associated bacterial pathogens follow. This section is not intended to be extensive as such descriptions are readily available elsewhere. In accordance with the MAF Policy Expression of Interest parasites, viruses and *Y. enterocolitica* are not considered further.

Aeromonas

This taxonomically complex group of organisms is of equivocal pathogenicity. There is a lot of circumstantial evidence supporting some species' role as a pathogen (e.g. *A. hydrophila*), but only a single case has yielded a plausible link between isolates from food and from the clinical case. Many of these organisms can grow at refrigeration temperatures.

Aeromonads are usually regarded as a waterborne organism, but they are also commonly found on raw foods of many types. They are also readily isolated from ready-to-eat foods. The greatest association with foods is with seafoods, reflecting their aquatic habitat. It has been isolated from produce in a number of surveys.

Since even its status as a pathogen is unclear there is no information on dose response.

Bacillus cereus

While normally associated with starchy foods, this organism has also been isolated from intact vegetables at medium levels (2.0 – 2.7 log₁₀/g, Kaneko *et al.*, 1999), and at low prevalences (2.5%, Thunberg *et al.*, 2002).

Campylobacter

Campylobacter is the most commonly reported cause of bacterial food poisoning in New Zealand. The two species most often associated with disease are *C. jejuni* and *C. coli*. Most cases of disease are sporadic and outbreaks are relatively rare.

Campylobacter is unlikely to grow in foods unless they are severely temperature abused. Therefore most outbreaks are caused by cross contamination or inadequate cooking. This organism is associated with undercooked chicken meat, in particular, as well as undercooked red meats and offal, raw milk and untreated or inadequately treated drinking water. Infection can also result from contact with pets and farm animals, particularly calves.

This organism has been isolated from vegetables (Kumar *et al.*, 2001; Park and Sanders, 1992) and mushrooms (Doyle and Schoeni, 1986), and has been associated with an outbreak of campylobacteriosis linked to the consumption of cucumber salad, although this was probably as a result of cross contamination during preparation (Kirk *et al.*, 1997).

Campylobacter survives well at refrigeration temperatures but less well as the temperature increases. On watermelon, *Campylobacter* reduced in numbers by 38-87 % over six hours at room temperature. A reduction under the same conditions on papaya was in excess of 90% (Castillo and Escartin, 1994).

Clostridium botulinum

This organism causes disease through the production of a toxin. While the case fatality rate from this organism has been reduced to around 8%, those that suffer from its effects often require hospitalisation and long periods of treatment involving supportive therapy and the neutralisation of the toxin. However, the disease is rare and there has only been one outbreak in New Zealand caused by improperly prepared Tiroi (Puha and mussels).

The organism is a spore-former and is often thought of as being able to grow only in oxygen-free environments. Despite this the organism has been recorded as growing in some environments which would not normally be considered oxygen-free, such as in between aluminium foil and the surface of a baked potato, and under a layer of fat and onion left on a grill overnight (References in Notermans 1993). The organism is an inhabitant of the soil and so may be detected on produce.

***Escherichia coli* O157:H7 (and other shiga-toxigenic *Escherichia coli*)**

Disease caused by *E. coli* O157:H7 can be serious and is fatal in a small percentage of cases, especially when children become infected. A number of long-term illnesses can be caused by this organism, with kidney dialysis being required for some cases.

The organism is associated overseas with beef products, especially hamburgers, but has also caused outbreaks while present on foods as diverse as venison jerky, beansprouts and non-alcoholic cider. However, person-to-person (or person-to-food) spread can also occur. A number of outbreaks of *E. coli* O157:H7 infections have involved lettuce as the vehicle.

The presence of even a few organisms (levels of 0.3-0.4 cells per gram) has caused outbreaks in the past.

Listeria monocytogenes

This organism is one of the most problematic facing the food industry today since infections can lead to death in around 30% of cases. There are very few cases of listeriosis in New Zealand every year (usually less than 20), but the case fatality rate, and the demographics of those people most at risk (old, young, pregnant and immunocompromised) give it its high profile with the public and the media.

The main distinguishing feature of this bacterium is its ability to grow at refrigeration temperatures, i.e. down to -1.5°C (Hudson *et al* 1994). Although growth under refrigeration is slow, products which have shelf lives measured in weeks (such as meats) may contain *L. monocytogenes* at high numbers at the end of that shelf life. The organism will grow well if contaminated food is temperature abused.

The organism has been isolated from produce (e.g. field cress and potatoes, Thunberg *et al.*, 2002) and this is expected because of the association of the organism with soil. It can grow on a number of specific fruit and vegetable products at refrigeration temperatures; for example on cabbage stored at 5°C (Beuchat *et al.*, 1986).

Salmonella

There have been numerous outbreaks of salmonellosis linked to produce consumption; for example with melons (Velaudapillai *et al.*, 1969). This organism has also been isolated from produce in a number of studies (Beuchat, 1996).

There is little remarkable about the physiology of this organism and its growth parameters (minimum 7°C, optimum 35-37°C, maximum 49.5°C) are quite like those of many other organisms. However, some of its survival characteristics are noteworthy. It survives very well on food and in the environment, for example the organism survived 18 days of storage at 10°C on the surface of tomatoes (Zhuang *et al.*, 1995).

Shigella

Cases of shigellosis are normally associated with contamination by a food handler, but infections via contaminated water do occur. The organism is regarded as a good survivor of acidic conditions and so the death rate on acidic fruit will be less than for other, more sensitive organisms. A slow decline in numbers of *Shigella* has been shown when the organism was inoculated onto green peppers, onions and cabbages stored at 4°C (Rafii and Lunsford, 1997).

The probability of infection at low dose is relatively high, and so the presence of even a few cells of this organism in a ready-to-eat food is significant.

Staphylococcus aureus

This organism is usually introduced on to food during handling. There have been isolations from produce, for example Thunberg *et al.*, (2002) reported isolation from lettuce. Illness results from consuming toxins produced when the population of *S. aureus* exceeds 1×10^5 CFU/g. Small numbers of *S. aureus* in food are not a direct hazard to health.

3 HAZARD IDENTIFICATION: FRUITS AND VEGETABLES

3.1 Relevant Characteristics of the Food

3.1.1 Fruit

Fruits contain high concentrations of organic acids and so are of low pH. However there is a great deal of variability, for example passionfruit are approximately pH 2, oranges around pH 3-4 and melons around pH 6.5. Because of these low pH values fruits do not generally allow the growth of bacteria.

Fruits have an associated microflora, and initial contamination comes from the field and during harvesting and transport operations. A source of contamination of fruits is the use of contaminated water for their irrigation or washing prior to consumption.

Pathogens which may have been introduced to raw fruits during growth or harvest will not grow on most fruits, but they may survive for some time depending on the pathogen and the fruit. This is important with some organisms, such as *Salmonella*, that survive well in the environment. *E. coli* O157:H7 is also adept at surviving under low pH conditions, as are encountered in fruit pulps (Marques *et al.*, 2001).

Some bacteria can grow on higher pH fruits such as melons, apples and tomatoes. *E. coli* O157:H7 has been shown to grow on cut apple surfaces in air at 15-20°C (Gunes and Hotchkiss, 2002). Growth has also been shown in the rind of both canteloupe and watermelon at 25°C, but not at 5°C (Del Rosario and Beuchat, 1995).

3.1.2 Vegetables

The pH of vegetables varies between 5 and 7 (e.g. 5.73 in green peppers, 5.95 in coleslaw mix, and 6.54 in cauliflower florets) and nutrients are available so that they provide a medium on which microbial growth can occur.

Contaminants can be acquired at any stage in the production process, from growing, through distribution to handling prior to use. Foodborne disease outbreaks associated with vegetables usually occur because the vegetable was exposed to faecal material during growth or handling. Some vegetables may be fertilised with manure or compost that may not have been correctly produced. Irrigation water may be contaminated, and people handling produce who practice poor personal hygiene following defecation may also contaminate vegetables.

Vegetables may be subjected to decontamination or washing after harvest but this does not guarantee the removal of pathogens despite the fact that their numbers should be reduced 10-100 fold.

Fresh ready-to-eat vegetables are usually stored for relatively short periods and under refrigeration to maintain quality. Although growth of most organisms under these conditions will be minimal, cool, moist conditions are likely to favour the survival of some organisms such as *Campylobacter*.

Extension of shelf life increases the potential for the growth of *L. monocytogenes* to high numbers. The first outbreak caused by this organism occurred because extended storage of cabbage, which was made into coleslaw, allowed for growth of the organism.

Table 1 gives some data on the growth and survival of bacterial pathogens on vegetables and vegetable products. Growth occurs largely where it would be expected. An exception is the inability for *L. monocytogenes* to grow on carrots, the anti-listerial effect of which is now well documented. Also of possible note is the inability of two from three pathogens tested to grow on tomatoes at temperatures which might be considered to be permissive to growth.

3.1.3 Sites of Contamination

Until very recently it has been assumed that contamination of produce by bacteria was a surface phenomenon, perhaps complicated by the potential for organisms to grow at wound sites. However, this view has been changing because of a small number of studies indicating that pathogens may be internalised into produce under some circumstances.

The contamination of the core of warm apples immersed into medium containing *Escherichia coli* O157:H7 has been shown (Buchanan *et al.*, 1999). Dye uptake studies showed that 6% of Golden Delicious apples would take up dye from the blossom end of the fruit into the core region. Apples are often moved and sorted using flumes and so apples might take up pathogens if the water is contaminated. High rates of internalisation of coliforms, yeasts and moulds and “aerobic bacteria” in apples and pears has been reported (Riordan *et al.*, 2001). The phenomenon has also been shown for tomatoes and *Salmonella* (Zhuang *et al.*, 1995), and *A. hydrophila* (Velázquez *et al.*, 1998).

Two papers reported on the colonisation of lettuces by *E. coli* O157:H7 when grown in contaminated soils or hydroponic systems (Solomon *et al.*, 2002; Watchell *et al.*, 2002). The former paper focused on the fact that *E. coli* O157:H7, while also colonising stomatal pores can be found on the internal surfaces of lettuce leaf tissue. The latter paper describes adherence to roots and the seed coat, but also “Remarkably, we observed fluorescent EHEC moving within the vasculature of a hypocotyl. The bacteria were most likely located within the xylem, since this is the only open tissue within the hypocotyl....”.

Table 1 **Growth and Survival of Bacterial Pathogens on Vegetables.**

Food	Organism	Temperature (°C)	Growth	Reference
Asparagus	<i>A. hydrophila</i>	4 and 15	+	Berrang et al., 1989a
	<i>L. monocytogenes</i>	4 and 15	+	Berrang et al., 1989b
Broccoli	<i>A. hydrophila</i>	4 and 15	+	Berrang et al., 1989a
	<i>L. monocytogenes</i>	4 and 15	+	Berrang et al., 1989b
Cabbage	<i>L. monocytogenes</i>	5	+	Beuchat et al., 1986
	<i>L. monocytogenes</i>	5	+ ¹	Kallander et al., 1991
		25	+ ¹	
	<i>Shigella</i>	5	-	Rafii and Lunsford, 1997
Carrots	<i>L. monocytogenes</i>	5 and 15	-	Beuchat and Brackett, 1990a
Cauliflower	<i>A. hydrophila</i>	4 and 15	+	Berrang et al., 1989a
	<i>L. monocytogenes</i>	4	-	Berrang et al., 1989a
		15	+	
Green pepper	<i>Shigella</i>	4	-	Rafii and Lunsford, 1997
Lettuce	<i>E. coli</i> O157:H7	13 and 22	+	Diaz and Hotchkiss, 1996
	<i>L. monocytogenes</i>	5	-(18 d) ²	Li et al., 2002
		15	+	
	<i>L. monocytogenes</i>	5, 12 and 15	+	Steinbruegge et al., 1988
	<i>L. monocytogenes</i>	5 and 10	+	Beuchat et al., 1990b
Onion	<i>Shigella</i>	4	-	Rafii and Lunsford, 1997
Tomatoes	<i>A. hydrophila</i>	6, 25 and 35	+ ¹	Velázquez et al., 1998
	<i>L. monocytogenes</i>	10	-(20d)	Beuchat and Brackett, 1991
		21	+	
	<i>Salmonella</i>	10	-(18d)	Zhuang et al., 1995
		20 and 30	+	

+ = growth, - = no growth.

¹ Notable declines in numbers followed the peaks.² Duration of incubation given where growth did not occur.

4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

4.1 Disease Severity and Frequency

Table 2 compares the disease caused by infection/intoxication by the pathogens to reflect the severity of the consequent clinical outcomes. The aetiological agents that result in the highest proportions of serious clinical outcomes are *Clostridium botulinum*, *Listeria monocytogenes* and *E. coli* O157:H7.

Table 2 Table of Disease Severity of Bacteria of Concern

(Data are for New Zealand from Lake *et al.* (2000) except where indicated)

Organism	Symptoms	Hospitalisation (% cases)	Deaths (% cases)
<i>Aeromonas</i>	Watery diarrhoea, nausea, abdominal pain, 'flu-like' symptoms	No data found for gastrointestinal disease	No data found for gastrointestinal disease
<i>Bacillus cereus</i>	Nausea and vomiting or abdominal pain and diarrhoea	0.6*	0*
<i>Campylobacter</i>	Fever, abdominal pain, diarrhoea	0.3	0.001
<i>Clostridium botulinum</i>	Nausea, vomiting, neurological symptoms, paralysis	80.0*	7.7*
<i>Escherichia coli</i> O157:H7	Bloody diarrhoea, kidney failure	29.5*	0.8*
<i>Listeria monocytogenes</i>	Either febrile diarrhoea or meningitis, septicaemia	56.3	2.5
<i>Salmonella</i>	Diarrhoea, abdominal pain, vomiting, nausea and fever	1.0	0.007
<i>Shigella</i>	Abdominal pain, diarrhoea, malaise, fever	2.6	0
<i>Staphylococcus aureus</i>	Nausea, vomiting	18.0*	0.02*

* Data from Mead *et al.*, 1999 (USA)

4.2 Dose Response

Recent developments in quantitative risk assessment, and in particular risk characterization have lead to the development of mathematical models which assign probability of infection to a given dose. A consequence of this is that there is no dose (other than 0) that gives zero probability of disease, although the probability might be very low. Similarly, there is no dose where $p \text{ disease} = 1.0$, but again there may be doses that get very close to this value. The

concept of an “infectious dose” is now therefore largely historical, although this rule of thumb is still applied to those organisms producing toxins.

Table 3 shows probabilities of disease at given dose levels derived from published dose/response models.

Table 3 Modelled Probability of Disease at Various Doses for Organisms of Concern

Organism	Dose (cells)			
	10^2	10^4	10^6	10^8
	Probability of Disease			
<i>L. monocytogenes</i> - more susceptible population ¹	1.06×10^{-10}	1.06×10^{-8}	1.06×10^{-6}	1.06×10^{-4}
<i>L. monocytogenes</i> - less susceptible population ¹	2.37×10^{-12}	2.37×10^{-10}	2.37×10^{-8}	1.06×10^{-6}
<i>Campylobacter</i> ²	1.50×10^{-1}	2.17×10^{-1}	1.38×10^{-1}	1.59×10^{-2}
<i>Salmonella</i> ²	6.87×10^{-10}	6.73×10^{-6}	2.17×10^{-2}	9.58×10^{-1}
<i>Escherichia coli</i> O157:H7 ³	2.56×10^{-4}	2.47×10^{-2}	5.92×10^{-1}	9.53×10^{-1}
<i>Escherichia coli</i> O157:H7 ⁴	2.52×10^{-3}	1.55×10^{-1}	6.50×10^{-1}	8.73×10^{-1}
<i>Shigella</i> ⁴	3.22×10^{-1}	6.66×10^{-1}	8.38×10^{-1}	9.21×10^{-1}

Models from ¹FAO/WHO 2001, ²Teunis *et al.*, 1999, ³Haas *et al.*, 2000, ⁴ Powell *et al.*, 2000.

It should be noted that not all models give 95% confidence limits and only the median values are given here for the sake of clarity. Given the biological variability in both pathogen and host, as well as other considerations such as the effect of any food on the pathogen, some variability around the values given in the table above is to be expected.

Accepting these caveats it is apparent that the probability of disease from consuming *L. monocytogenes* is low compared with the other organisms. *Campylobacter* and *Shigella* are comparable in that there is a significant but largely flat probability of disease over the 6 log₁₀ range modelled, while the probability of salmonellosis increases a billion fold over the same dose range. The data for the two models for *E. coli* O157:H7 are similar, except at lower dose levels where they diverge. The probability for infection by this organism becomes high at doses above 10⁴ cells in both models.

Doses for the toxin-producing organisms *S. aureus* and *B. cereus* fit better with the conventional “infectious dose” model, in that they must reach numbers sufficient for production of a physiologically active toxin level. The level is around 10⁵/g for both of these organisms (although again variability will make individuals less or more prone to intoxication at any given dose).

5 EXPOSURE ASSESSMENT

5.1 Prevalence and Quantitative Data for Pathogens in Fruit and Vegetables

5.1.1 New Zealand

No information concerning the prevalence of pathogens on New Zealand fruit and vegetables could be located. Consumer magazine has published an article reporting that *L. monocytogenes* was present in 14 of 110 supermarket salad samples (Anonymous, 1997), but contamination may have been introduced during processing.

5.1.2 Overseas

A literature search was conducted to compile any international data where research was carried out to detect any of the pathogens of interest on whole, fresh produce such as that which is exported from New Zealand. Only data from whole and fresh fruits and vegetables were included, and it was found that much of this originated from the US (both locally grown and imported produce). Tables 4 and 5 summarise the results of this literature search, and Tables 6 and 7 present in detail this information.

In the following information, avocados and tomatoes are treated as vegetables due to their common use in savoury foods. When available, data from organic produce are included and specified.

Table 4 Summary of the Association of Bacterial Pathogens with Fruit from International Data

7= tested for and not detected

3= tested for and detected

		No data ¹	<i>Aeromonas</i>	<i>Bacillus cereus</i>	<i>Campylobacter</i> spp.	<i>Clostridium botulinum</i>	<i>E. coli</i>	<i>E. coli</i> O157:H7	<i>Listeria</i> spp.	<i>Listeria monocytogenes</i>	<i>Salmonella</i> spp.	<i>Shigella</i> spp.	<i>Staphylococcus</i> spp.	<i>Staphylococcus aureus</i>
<i>Berry fruit</i>	Blackberries	⊖												
	Blackcurrants	⊖												
	Blueberries						3							
	Raspberries						7							
	Strawberries							7		3	3		3	
	Boysenberries	⊖												
<i>Citrus</i>	Lemons	⊖												
	Oranges						3				7			
	Mandarins	⊖												
	Tangelos	⊖												
	Grapefruit	⊖												
<i>Summerfruit</i>	Apricots	⊖												
	Cherries						3							
	Nectarines	⊖												
	Peaches						3				3			
	Plums	⊖												
	Tamarillos	⊖												
<i>Other Fruit</i>	Apples						3	7					3	
	Apples (organic)							7						
	Feijoa	⊖												
	Kiwifruit	⊖												
	Melons							7		3	3	3		
	Nashi	⊖												
	Passionfruit	⊖												
	Pears						3	7						
	Persimmons	⊖												

¹ No data was found concerning this food and the selected pathogens.

Table 5 Summary of the Association Between Bacterial Pathogens and Vegetables from International Data

7= tested for and not detected
3= tested for and detected

	No data ¹	<i>Aeromonas</i>	<i>Bacillus cereus</i>	<i>Campylobacter</i> spp.	<i>Clostridium botulinum</i>	<i>E. coli</i>	<i>E. coli</i> O157:H7	<i>Listeria</i> spp.	<i>Listeria monocytogenes</i>	<i>Salmonella</i> spp.	<i>Shigella</i> spp.	<i>Staphylococcus</i> spp.	<i>Staphylococcus aureus</i>	Sample frequency where <i>E. coli</i> count >10 ³ (2)
Asparagus						3			7					2/3
Asparagus (organic)		3		7		7	7	7		7				
Avocados	⊖													
Broccoflower	⊖													
Broccoli		3	3	7		7	7	3	7	7	7		7	
Broccoli (organic)		3		7		7	7	7		7				
Brussels sprouts						3								1/2
Cabbages				7		3	3	3	3	3				5/41
Cabbages (organic)		7		7		7	7	7		7				
Capsicums				7				3		3				
Capsicums (organic)		3		7		7	7	7		7				
Carrots				7	7	3		3	7	3		3		
Carrots (organic)		3		7		7	7	7		7				
Cauliflower			7	7		3		7	7	3			7	4/23
Cauliflower (organic)		3		7		7	7	7		7				
Celery		3	7	7		3	3	3	7	3	3		7	10/26
Celery (organic)		3		7		7	7	7		7				
Corn									7					
Courgettes/zucchini										7				
Courgettes (organic)		3		7		7	7	7		7				
Cucumbers				7				3	3	7				
Cucumbers (organic)		3		7		7	7	7		7				
Garlic	⊖													
Green beans													7	
Leeks				3			7			7	3			
Lettuce		3	7	3	7	3	7	3	3	3	3	3	3	31/80
Lettuce (organic)		3		7		7	7	7		7				
Mushrooms				3	7	7		3	3			3	7	
Mushrooms (organic)		3		7		7	7	7		7				
Onions					3					3				
Onions (organic)		7		7		7	7	7		7				
Parsnips	⊖													

Potatoes				3	7			3	7	7				
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Table 5 continued

7= tested for and not detected 3= tested for and detected	No data ¹	<i>Aeromonas</i>	<i>Bacillus cereus</i>	<i>Campylobacter</i> spp.	<i>Clostridium botulinum</i>	<i>E. coli</i>	<i>E. coli</i> O157:H7	<i>Listeria</i> spp.	<i>Listeria monocytogenes</i>	<i>Salmonella</i> spp.	<i>Shigella</i> spp.	<i>Staphylococcus</i> spp.	<i>Staphylococcus aureus</i>	Sample frequency where <i>E. coli</i> count >10 ³ ⁽²⁾
Potatoes (organic)		7		7		7	7	7		7				
Pumpkins	⊖													
Scallops	⊖													
Shallots	⊖													
Spring onions (organic)		7		7		7	7	7		7				
Squash	⊖													
Swedes	⊖													
Taro	⊖													
Tomatoes						7	7	3	3	7	7	3		
Tomatoes (organic)		3		7		7	7	7		7				
Turnips (organic)		3		7		7	7	7		7				
Witloof/endive		3				3		7		3				1/1
Yams				7				3		7				

¹ No data was found concerning this food and the selected pathogens.

⁽²⁾ Where counts recorded

Table 6 Detailed Listing of Associations Between Bacterial Pathogens and Indicators with Fruit from International Data

Produce	Pathogen	No. tested	Frequency	Count	Country	Reference
Apples	Aerobic Plate Count (APC)	169 (over 10 orchards)		4.2 log CFU/g (2.73 (n=10) -5.29 (n=6))	US	Riordan <i>et al.</i> (2001)
	APC (unwashed)	? (>1x10g)		0-4x10 ⁴ CFU/g	US	Riordan <i>et al.</i> (2001)
	Coliforms	169 (over 10 orchards)		1.5 log CFU/g (0.33 (n=10)-1.77(n=6))	US	Riordan <i>et al.</i> (2001)
	<i>Escherichia coli</i>	169 (over 10 orchards)	1/169 (0.6%)	1.19 log CFU/g , apple on tree	US	Riordan <i>et al.</i> (2001)
	<i>Escherichia coli</i> O157:H7	169 (over 10 orchards)	Not detected		US	Riordan <i>et al.</i> (2001)
	<i>Staphylococcus</i> spp.	? (>1x10g)	4.3%		Lebanon	Abdelnoor <i>et al.</i> (1983)
Apples (organic)	APC	18 (1 orchard)		3.94 log CFU/g	US	Riordan <i>et al.</i> (2001)
	APC	20 (1 orchard)		4.93 log CFU/g	US	Riordan <i>et al.</i> (2001)
	Coliforms	18 (1 orchard)		0.71 CFU/g	US	Riordan <i>et al.</i> (2001)
	Coliforms	20 (1 orchard)		0.86 CFU/g	US	Riordan <i>et al.</i> (2001)
	<i>E. coli</i> O157:H7	38 (2 orchards)	Not detected		US	Riordan <i>et al.</i> (2001)
<i>Berryfruit</i>						
Blueberries	<i>E. coli</i> (SLT)	1x10g	Detected (by DNA probe)		US	Samadpour <i>et al.</i> (1990)
Raspberries	<i>E. coli</i> (SLT)	1x10g	Not detected (by DNA probe)		US	Samadpour <i>et al.</i> (1990)
Strawberries	APC (unwashed)	? (>1x10g)		0.01-3x10 ⁵ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)
	Coliforms (thermotolerant)	173	1 (0.6%)		Norway	Johannessen <i>et al.</i> (2002)
	<i>E. coli</i> O157	173	Not detected		Norway	Johannessen <i>et al.</i> (2002)
	<i>E. coli</i> O157:H7	143	Not detected		US	USFDA (2001)
	<i>E. coli</i> O157:H7	121	Not detected		US	Stier & Nagle (2001)

	<i>Listeria monocytogenes</i>	173	1 (0.6%)	Norway	Johannessen <i>et al.</i> (2002)
	<i>Salmonella</i> spp.	143	1 (0.7%)	US	USFDA (2001)
	<i>Salmonella</i> spp.	121	Not detected	US	Stier & Nagle (2001)
	<i>Salmonella</i> spp.	173	Not detected	Norway	Johannessen <i>et al.</i> (2002)
	<i>Staphylococcus</i> spp.	173	26 (15.0%)	Norway	Johannessen <i>et al.</i> (2002)
<i>Citrus</i>					
Oranges	APC	10 (unwashed)	150 CFU/cm ²	US	Parish (1998)
(Juice	APC	10 (unwashed)	112,000 CFU/cm ²	US	Parish (1998)
factories)	APC	10 (washed)	6 CFU/cm ²	US	Parish (1998)
	APC	10 (washed)	3,600 CFU/cm ²	US	Parish (1998)
	Coliforms (fecal)	10 (unwashed)	20 MPN/100cm ²	US	Parish (1998)
	Coliforms (fecal)	10 (unwashed)	1,200 MPN/100cm ²	US	Parish (1998)
	Coliforms (fecal)	10 (washed)	<1 MPN/100cm ²	US	Parish (1998)
	Coliforms (fecal)	10 (washed)	300 MPN/100cm ²	US	Parish (1998)
	<i>E. coli</i>	10 (unwashed)	<1 MPN/cm ²	US	Parish (1998)
	<i>E. coli</i>	10 (unwashed)	300 MPN/cm ²	US	Parish (1998)
	<i>E. coli</i>	10 (washed)	<1 MPN/cm ²	US	Parish (1998)
	<i>E. coli</i>	10 (washed)	<40 MPN/cm ²	US	Parish (1998)
	<i>Salmonella</i> spp.	10 (unwashed)	Not detected	US	Parish (1998)
	<i>Salmonella</i> spp.	10 (washed)	Not detected	US	Parish (1998)
Oranges &	APC	84	4.0 log CFU/cm ²	US	Pao & Brown (1998)
tangerines	Coliforms (total)	84	35.2MPN/cm ²	US	Pao & Brown (1998)
(pack houses)	Coliforms (fecal)	84	5.0MPN/cm ²	US	Pao & Brown (1998)
<i>Other fruit</i>					
Cantaloupe	<i>E. coli</i> O157:H7	115	Not detected	US	Stier & Nagle (2001)
	<i>E. coli</i> O157:H7	151	Not detected	US	USFDA (2001)
	<i>L. monocytogenes</i>	30	Not detected	Pakistan	Vahidy <i>et al.</i> (1992)
	<i>Salmonella</i> spp.	115	3 (2.6%)	US	Stier & Nagle (2001)
	<i>Salmonella</i> spp.	151	8 (5.3%)	US	USFDA (2001)

	<i>Shigella</i> spp.	115	1 (0.9%)		US	Stier & Nagle (2001)
	<i>Shigella</i> spp.	151	3 (2.0%)		US	USFDA (2001)
Pears	APC	12		3.81 log CFU/g	US	Riordan <i>et al.</i> (2001)
	APC	11		2.93 log CFU/g	US	Riordan <i>et al.</i> (2001)
	APC (unwashed)	? (>1x10g)		6-9x10 ³ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)
	Coliforms	12		1.53 CFU/g	US	Riordan <i>et al.</i> (2001)
	Coliforms	11		0.63 CFU/g	US	Riordan <i>et al.</i> (2001)
	<i>E. coli</i>	12	1 (8.3%)	0.85 log CFU/g (dropped)	US	Riordan <i>et al.</i> (2001)
	<i>E. coli</i>	11	2 (18.2%)	0.4 (damaged on tree) & 0.7 log CFU/g (dropped)	US	Riordan <i>et al.</i> (2001)
	<i>E. coli</i> O157:H7	23	Not detected		US	Riordan <i>et al.</i> (2001)
Watermelon	<i>L. monocytogenes</i>	30	1 (3.3%)		Pakistan	Vahidy <i>et al.</i> (1992)
<i>Summerfruit</i>						
Cherries	APC (unwashed)	? (>1x10g)		0.01-3x10 ⁵ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)
	<i>E. coli</i>	? (>1x10g)	6.25%		Lebanon	Abdelnoor <i>et al.</i> (1983)
Peaches	APC (unwashed)	? (>1x10g)		0.01-3x10 ⁵ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)
	<i>E. coli</i>	? (>1x10g)	10.5%		Lebanon	Abdelnoor <i>et al.</i> (1983)
	<i>Salmonella</i> spp.	? (>1x10g)	5.3%		Lebanon	Abdelnoor <i>et al.</i> (1983)
Plums	APC (unwashed)	? (>1x10g)		0-3x10 ⁵ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)

Table 7 Detailed Listing of the Association Between Bacterial Pathogens and Indicators and Vegetables from International Data

Produce	Pathogen	No. tested	Frequency	Count	Country	Reference
Asparagus	Aerobic Plate Count (APC)	3		MPN per 100g: 10 ⁴ -10 ⁶ (0), 10 ⁶ -10 ⁸ (3), 10 ⁸ -10 ¹⁰ (0), >10 ¹⁰ (0)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	APC	3		5.04 log CFU/g	Spain	Arroyo <i>et al.</i> (1999)
	Coliforms (bacilli)	3		MPN per 100g: <10 ³ (1), >10 ³ (2)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>Escherichia coli</i>	3		MPN per 100g: <10 ³ (1), >10 ³ (2)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>Listeria monocytogenes</i>	1300g	Not detected		US	Berrang <i>et al.</i> (1989a)
Asparagus (organic)	<i>Aeromonas</i> spp.	1	1 (100%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	1	Not detected		UK	McMahon & Wilson (2001)
Broccoli	APC	10		6.3 log CFU/g (range 3.4-6.9)	US	Thunberg <i>et al.</i> (2002)
	APC	1		3.2x10 ⁴	US	Callister & Agger (1987)
	<i>Aeromonas</i> spp.	16	5 (31.3%)		US	Callister & Agger (1987)
	<i>Bacillus cereus</i> (enterotoxigenic)	7	1 (14%)		US	Thunberg <i>et al.</i> (2002)
	<i>Campylobacter</i> spp.	13	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>E. coli</i>	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>E. coli</i> O157:H7	36	Not detected		US	USFDA (2001)
	<i>Listeria</i> spp.	1		<10 ²	US	Callister & Agger (1987)

	<i>Listeria</i> spp.	13	Not detected	US	Thunberg <i>et al.</i> (2002)
	<i>L. monocytogenes</i>	25g stem	Not detected	US	Petran <i>et al.</i> (1988)
	<i>L. monocytogenes</i>	1300g	Not detected	US	Berrang <i>et al.</i> (1989)
	<i>Salmonella</i> spp.	36	Not detected	US	USFDA (2001)
	<i>Salmonella</i> spp.	13	Not detected	US	Thunberg <i>et al.</i> (2002)
	<i>Shigella</i> spp.	36	Not detected	US	USFDA (2001)
	<i>Staphylococcus aureus</i> (enterotoxigenic)	7	Not detected	US	Thunberg <i>et al.</i> (2002)
Broccoli (organic)	<i>Aeromonas</i> spp.	4	1 (25%)	UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	4	Not detected	UK	McMahon & Wilson (2001)
Brussels sprouts	APC	2	MPN per 100g: 10 ⁴ -10 ⁶ (0), 10 ⁶ - 10 ⁸ (2), 10 ⁸ -10 ¹⁰ (0), >10 ¹⁰ (0)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	Coliforms (bacilli)	2	MPN per 100g: <10 ³ (0), >10 ³ (2)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>E. coli</i>	2	MPN per 100g: <10 ³ (1), >10 ³ (1)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
Cabbage	APC	1?	160 CFU/g	US	Rafii & Lunsford (1997)
	APC	41	MPN per 100g: 10 ⁴ -10 ⁶ (5), 10 ⁶ - 10 ⁸ (19), 10 ⁸ - 10 ¹⁰ (17), >10 ¹⁰ (0)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	APC	4 (stem)	5.3x10 ⁶ CFU/g	India	Pingulkar <i>et al.</i> (2001)

	APC	4 (outer leaves)	4 (100%)	5.9 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	11 (mid-leaves)	5 (45%)	4.9 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	3 (inner leaves)	1 (33%)	2.9 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	<i>Campylobacter</i> spp.	130	Not detected		Canada	Park & Sanders (1992)
	<i>Campylobacter jejuni</i>	9	Not detected		India	Kumar <i>et al.</i> (2001)
	Coliforms	4		<3 MPN/g	India	Pingulkar <i>et al.</i> (2001)
	Coliform (bacilli)	41		MPN per 100g: <10 ³ (16), >10 ³ (25)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>E. coli</i>	41		MPN per 100g: <10 ³ (36), >10 ³ (5)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>E. coli</i> O157:H7	4	1 (25%)		Mexico	Zepeda-Lopez <i>et al.</i> (1995)
	<i>Listeria</i> spp.	92	2 (2.2%)		US	Heisick <i>et al.</i> (1989)
	<i>Listeria</i> spp.	4	Present		India	Pingulkar <i>et al.</i> (2001)
	<i>L. monocytogenes</i>	425	20 (4.7%)		US	Prazak <i>et al.</i> (2002)
	<i>L. monocytogenes</i>	25g (outer leaves)	Not detected		US	Petran <i>et al.</i> (1988)
	<i>L. monocytogenes</i>	18	6 (33%)		Sri Lanka	Gunaseena <i>et al.</i> (1995)
	<i>Salmonella</i> spp.	41	7 (17.1%)		Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>Salmonella</i> spp.	18	Not detected		Netherlands	Tamminga <i>et al.</i> (1978)
Cabbage (organic)	<i>Aeromonas</i> spp.	4	Not detected		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	4	Not detected		UK	McMahon & Wilson (2001)

Capsicums/ green peppers	APC	6		4.23 log CFU/g	US	Liao & Fett (2001)
	APC	4		1.0x10 ⁶ CFU/g	India	Pingulkar <i>et al.</i> (2001)
	APC	2	2 (100%)	3.9 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	<i>Campylobacter</i> spp.	2	Not detected		US	Thunberg <i>et al.</i> (2002)
	Coliforms	4		<3 MPN/g	India	Pingulkar <i>et al.</i> (2001)
	<i>Listeria</i> spp.	4	Present		India	Pingulkar <i>et al.</i> (2001)
	<i>Listeria</i> spp.	2	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Salmonella</i> spp.	8	6/8 (75%)		US	Martin & Katz (1991)
	<i>Salmonella</i> spp.	2	Not detected		US	Thunberg <i>et al.</i> (2002)
Capsicum (organic)	<i>Aeromonas</i> spp.	6	3 (50%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	6	Not detected		UK	McMahon & Wilson (2001)
Carrots	APC	3		5.4 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC (unwashed)	? (>1x10g)		3-100x10 ⁵ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)
	APC	4		2.0x10 ⁷ CFU/g	India	Pingulkar <i>et al.</i> (2001)
	APC	6x25g (baby carrots)		6.27 log CFU/g	US	Liao & Fett (2001)
	<i>Campylobacter</i> spp.	149	Not detected		Canada	Park & Sanders (1992)
	<i>Campylobacter jejuni</i>	6	Not detected		India	Kumar <i>et al.</i> (2001)
	<i>Clostridium botulinum</i>	18	Not detected		Hungary	Notermans (1993)
	Coliforms	4	4 (100%)		Japan	Kaneko <i>et al.</i> (1999)
	Coliforms	4		<3 MPN/g	India	Pingulkar <i>et al.</i> (2001)
	<i>E. coli</i>	? (>1x10g)	7.1%		Lebanon	Abdelnoor <i>et al.</i> (1983)
	<i>Listeria</i> spp.	4	Detected		India	Pingulkar <i>et al.</i> (2001)
	<i>L. monocytogenes</i>	3	Not detected		Scotland	Fenlon <i>et al.</i> (1996)
	<i>L. monocytogenes</i>	15	Not detected		Pakistan	Vahidy <i>et al.</i> (1992)

	<i>L. monocytogenes</i>	8x50g (unwashed)	Not detected	<1 log CFU/g	US	Beuchat & Brackett (1990a)
	<i>L. monocytogenes</i>	25g peel	Not detected		US	Petran <i>et al.</i> (1988)
	<i>Salmonella</i> spp.	49	1 (2%)		US	Rude <i>et al.</i> (1984)
	<i>Staphylococcus</i> spp.	? (>1x10g)	14.3%		Lebanon	Abdelnoor <i>et al.</i> (1983)
Carrots (organic)	<i>Aeromonas</i> spp.	13	9 (69%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	13	Not detected		UK	McMahon & Wilson (2001)
Cauliflower	APC	23		MPN per 100g: 10 ⁴ -10 ⁶ (4), 10 ⁶ - 10 ⁸ (13), 10 ⁸ - 10 ¹⁰ (5), >10 ¹⁰ (1)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	APC	10		7.4 log CFU/g (range 4.7-8.3)	US	Thunberg <i>et al.</i> (2002)
	APC	3		5.13 log CFU/g	Spain	Arroyo <i>et al.</i> (1999)
	<i>B. cereus</i> (enterotoxigenic)	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Campylobacter</i> spp.	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>C. jejuni</i>	9	Not detected		India	Kumar <i>et al.</i> (2001)
	Coliform (bacilli)	23		MPN per 100g: <10 ³ (7), >10 ³ (16)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>E. coli</i>	23		MPN per 100g: <10 ³ (19), >10 ³ (4)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>E. coli</i>	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Listeria</i> spp.	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>L. monocytogenes</i>	25g stem	Not detected		US	Petran <i>et al.</i> (1988)
	<i>L. monocytogenes</i>	1300g	Not detected		US	Berrang <i>et al.</i> (1989b)

	<i>Salmonella</i> spp.	23	1 (4.3%)		Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>Salmonella</i> spp.	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Salmonella</i> spp.	13	1 (7.7%)		Netherlands	Tamminga <i>et al.</i> (1978)
	<i>S. aureus</i> (enterotoxigenic)	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Aeromonas</i> spp.	1	1 (100%)		UK	McMahon & Wilson (2001)
Cauliflower (organic)	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	1	Not detected		UK	McMahon & Wilson (2001)
Celery	APC	26		MPN per 100g: 10 ⁴ -10 ⁶ (3), 10 ⁶ -10 ⁸ (14), 10 ⁸ -10 ¹⁰ (8), >10 ¹⁰ (1)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	APC	3	3 (100%)	5.8 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	10		7.5 log CFU/g (range 6.6-8.3)	US	Thunberg <i>et al.</i> (2002)
	APC	2		1.2x10 ⁶	US	Callister & Agger (1987)
	APC	8		4.5 log CFU/g (3.0-6.0)	US	Robbs <i>et al.</i> (1996)
	<i>Aeromonas</i> spp.	1		3.6x10 ³	US	Callister & Agger (1987)
	<i>Aeromonas</i> spp.	8	3 (37.5%)		US	Robbs <i>et al.</i> (1996)
	<i>B. cereus</i> (enterotoxigenic)	9	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Campylobacter</i> spp.	150	Not detected		Canada	Park & Sanders (1992)
	<i>Campylobacter</i> spp.	12	Not detected		US	Thunberg <i>et al.</i> (2002)
	Coliforms	3	1 (33%)		Japan	Kaneko <i>et al.</i> (1999)
	Coliforms (bacilli)	26		MPN per 100g: <10 ³ (3), >10 ³ (23)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)

	<i>E. coli</i>	26		MPN per 100g: <10 ³ (16), >10 ³ (10)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>E. coli</i>	10	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>E. coli</i>	8	Not detected		US	Robbs <i>et al.</i> (1996)
	<i>E. coli</i> O157:H7	84	Not detected		US	USFDA (2001)
	<i>E. coli</i> O157:H7	34	6 (17.6%)		Mexico	Zepeda-Lopez <i>et al.</i> (1995)
	<i>Listeria</i> spp.	12	3 (25%)		US	Thunberg <i>et al.</i> (2002)
	<i>Listeria</i> spp.	2		<10 ²	US	Callister & Agger (1987)
	<i>L. monocytogenes</i>	30	Not detected		Canada	Farber <i>et al.</i> (1989)
	<i>Salmonella</i> spp.	26	2 (7.7%)		Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>Salmonella</i> spp.	84	1 (1.2%)		US	USFDA (2001)
	<i>Salmonella</i> spp.	20	Not detected		Netherlands	Tamminga <i>et al.</i> (1978)
	<i>Salmonella</i> spp.	12	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Salmonella</i> spp.	48	1 (2.1%)		US	Rude <i>et al.</i> (1984)
	<i>Shigella</i> spp.	84	2 (2.4%)		US	USFDA (2001)
	<i>S. aureus</i> (enterotoxigenic)	9	Not detected		US	Thunberg <i>et al.</i> (2002)
Celery (organic)	<i>Aeromonas</i> spp.	3	1 (33%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	3	Not detected		UK	McMahon & Wilson (2001)
Corn	<i>L. monocytogenes</i>	25g (husks)	Not detected		US	Petran <i>et al.</i> (1988)
Courgettes	<i>Salmonella</i> spp.	11	Not detected		Netherlands	Tamminga <i>et al.</i> (1978)
Courgettes (organic)	<i>Aeromonas</i> spp.	3	1 (33%)		UK	McMahon & Wilson (2001)

	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	3	Not detected		UK	McMahon & Wilson (2001)
Cucumbers	APC	3		6.3 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	4		1.7x10 ⁶ CFU/g	India	Pingulkar <i>et al.</i> (2001)
	<i>Campylobacter</i> spp.	123	Not detected		Canada	Park & Sanders (1992)
	<i>Campylobacter</i> spp.	2	Not detected		US	Thunberg <i>et al.</i> (2002)
	Coliforms	4		<3 MPN/g	India	Pingulkar <i>et al.</i> (2001)
	<i>Listeria</i> spp.	2	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Listeria</i> spp.	92	9 (10.9%)		US	Heisick <i>et al.</i> (1989)
	<i>Listeria</i> spp.	4	Detected		India	Pingulkar <i>et al.</i> (2001)
	<i>L. monocytogenes</i>	15	1 (6.7%)		Pakistan	Vahidy (1992)
	<i>L. monocytogenes</i>	5	4 (80%)		Malaysia	Arumagaswaby (1994)
	<i>Salmonella</i> spp.	2	Not detected		US	Thunberg <i>et al.</i> (2002)
Cucumbers (organic)	<i>Aeromonas</i> spp.	6	4 (67%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	6	Not detected		UK	McMahon & Wilson (2001)
Endive (witloof)	APC	1		MPN per 100g: 10 ⁴ -10 ⁶ (0), 10 ⁶ -10 ⁸ (0), 10 ⁸ -10 ¹⁰ (1), >10 ¹⁰ (0)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	APC	2		1.5x10 ⁷	US	Callister & Agger (1987)
	<i>Aeromonas</i> spp.	20	8 (40%)	3.9x10 ⁴ -5.3x10 ⁵	Italy	Villari <i>et al.</i> (2000)
	Coliforms (bacilli)	1		MPN per 100g: <10 ³ (0), >10 ³ (1)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)

	<i>E. coli</i>	1		MPN per 100g: <10 ³ (0), >10 ³ (1)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	<i>Listeria</i> spp.	2		<10 ²	US	Callister & Agger (1987)
	<i>Salmonella</i> spp.	26	2 (7.7%)		Netherlands	Tamminga <i>et al.</i> (1978)
Green beans	<i>S. aureus</i>	20x50g samples		<1.00 log count/g	US	Silbernagel & Lindberg (2001)
Leeks	<i>Campylobacter</i> spp.	160	1 (0.6%)		Canada	Park & Sanders (1992)
	<i>Campylobacter</i> spp.	76	2 (2.6%)		Canada	Park & Sanders (1992)
	<i>E. coli</i> O157:H7	73	Not detected		US	Stier & Nagle (2001)
	<i>Salmonella</i> spp.	73	Not detected		US	Stier & Nagle (2001)
	<i>Shigella</i> spp.	73	3 (4.1%)		US	Stier & Nagle (2001)
Lettuces	APC	100g		5.3x10 ³ CFU/g	Nebraska	Steinbruegge <i>et al.</i> (1988)
	APC	100g		5.9x10 ² CFU/g	Nebraska	Steinbruegge <i>et al.</i> (1988)
	APC	10g		4.3 log CFU/ml	Spain	Arroyo <i>et al.</i> (1997)
	APC	10g		3.0 log CFU/ml	Spain	Arroyo <i>et al.</i> (1997)
	APC	80		MPN per 100g: 10 ⁴ -10 ⁶ (5), 10 ⁶ - 10 ⁸ (35), 10 ⁸ - 10 ¹⁰ (39), >10 ¹⁰ (1)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
	APC	4 (outer leaves)	4 (100%)	5.9 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	2 (mid-leaves)	2 (100%)	6.9 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	5 (inner leaves)	5 (100%)	5.0 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	10		8.6 log CFU/g (range 6.9-9.2)	US	Thunberg <i>et al.</i> (2002)
	APC (unwashed)	? (>1x10g)		2-120x10 ⁵ CFU/g	Lebanon	Abdelnoor <i>et al.</i> (1983)
	APC	3		5.57 log CFU/g	Spain	Arroyo <i>et al.</i> (1999)

APC	6		7.14 log CFU/g	US	Liao & Fett (2001)
APC	2		6.8x10 ⁵ CFU/g	US	Callister & Agger (1987)
APC	4		1.8x10 ⁶ CFU/g	India	Pingulkar <i>et al.</i> (2001)
APC	144		3.01-7.81 log CFU/g	Spain	Soriano <i>et al.</i> (2000)
APC	120		6.59x10 ⁷ /100g	Italy	Ercolani (1976)
<i>Aeromonas</i> spp.	2		3.7x10 ³ CFU/g	US	Callister & Agger (1987)
<i>Aeromonas</i> spp.	20	9 (45%)	1x10 ⁴ -4.5x10 ⁵ CFU/g	Italy	Villari <i>et al.</i> (2000)
<i>B. cereus</i> (enterotoxigenic)	10	Not detected		US	Thunberg <i>et al.</i> (2002)
<i>Campylobacter</i> spp.	165	2 (1.2%)		Canada	Park & Sanders (1992)
<i>Campylobacter</i> spp.	82	3 (3.7%)		Canada	Park & Sanders (1992)
<i>Campylobacter</i> spp.	10	Not detected		US	Thunberg <i>et al.</i> (2002)
<i>Campylobacter</i> spp.	151	Not detected		UK	Little <i>et al.</i> (1999)
<i>Clostridium botulinum</i>	34	Not detected		US	Riser <i>et al.</i> (1984)
Coliforms	144		<0.47->3.38 log MPN/g	Spain	Soriano <i>et al.</i> (2000)
Coliforms	120		5.95x10 ⁴ /100g	Italy	Ercolani (1976)
Coliforms	4		>1000 MPN/g	India	Pingulkar <i>et al.</i> (2001)
Coliforms (fecal)	120		6.13x10 ³ /100g	Italy	Ercolani (1976)
Coliforms (bacilli)	80		MPN per 100g: <10 ³ (9), >10 ³ (71)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
Coliforms (thermotolerant)	200	5 (2.5%)		Norway	Johannessen <i>et al.</i> (2002)
Coliforms (<i>Klebsiella pneumoniae</i>)	40	4 (10%)		Spain	Soriano <i>et al.</i> (2001)
Coliforms (<i>Klebsiella oxytoca</i>)	40	2 (5%)		Spain	Soriano <i>et al.</i> (2001)
Coliforms (<i>Klebsiella ozaenae</i>)	40	2 (5%)		Spain	Soriano <i>et al.</i> (2001)
<i>E. coli</i>	80		MPN per 100g: <10 ³ (49), >10 ³ (31)	Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)
<i>E. coli</i>	10	1 (10%)		US	Thunberg <i>et al.</i> (2002)

<i>E. coli</i>	40	4 (10%)	<20 CFU/g	Spain	Soriano <i>et al.</i> (2001)
<i>E. coli</i>	34	Not detected		US	Riser <i>et al.</i> (1984)
<i>E. coli</i>	151			UK	Little <i>et al.</i> (1999)
<i>E. coli</i>	? (>1x10g)	28.6%		Lebanon	Abdelnoor <i>et al.</i> (1983)
<i>E. coli</i>	144	37 (25.7%)		Spain	Soriano <i>et al.</i> (2000)
<i>E. coli</i> O157	200	Not detected		Norway	Johannessen <i>et al.</i> (2002)
<i>E. coli</i> O157:H7	116	Not detected		US	USFDA (2001)
<i>E. coli</i> O157:H7	40	Not detected		Spain	Soriano <i>et al.</i> (2001)
<i>E. coli</i> O157:H7	151	Not detected		UK	Little <i>et al.</i> (1999)
<i>E. coli</i> O157:H7	114	Not detected		US	Stier & Nagle (2001)
<i>Listeria</i> spp.	10	2 (20%)	<20 CFU/g	US	Thunberg <i>et al.</i> (2002)
<i>Listeria</i> spp.	151			UK	Little <i>et al.</i> (1999)
<i>Listeria</i> spp.	92	1 (1.1%)		US	Heisick <i>et al.</i> (1989)
<i>Listeria</i> spp.	4	Present		India	Pingulkar <i>et al.</i> (2001)
<i>L. monocytogenes</i>	200	1 (0.5%)		Norway	Johannessen <i>et al.</i> (2002)
<i>L. monocytogenes</i>	50	Not detected		Canada	Farber <i>et al.</i> (1989)
<i>L. monocytogenes</i>	151			UK	Little <i>et al.</i> (1999)
<i>L. monocytogenes</i>	6	Not detected		Scotland	Fenlon <i>et al.</i> (1996)
<i>L. monocytogenes</i>	25g	Not detected		US	Li <i>et al.</i> (2002)
<i>L. monocytogenes</i>	25g (outer leaves)	Not detected		US	Petran <i>et al.</i> (1988)
<i>L. monocytogenes</i>	20	10 (50%)	<20 CFU/g	Sri Lanka	Gunaseena <i>et al.</i> (1995)
<i>L. monocytogenes</i>	28	1 (3.6%)		Kuala Lumpur	Tang <i>et al.</i> (1994)
<i>Salmonella</i> spp.	200	Not detected		Norway	Johannessen <i>et al.</i> (2002)
<i>Salmonella</i> spp.	10	Not detected		US	Thunberg <i>et al.</i> (2002)
<i>Salmonella</i> spp.	40	Not detected		Spain	Soriano <i>et al.</i> (2001)
<i>Salmonella</i> spp.	151	Not detected		UK	Little <i>et al.</i> (1999)
<i>Salmonella</i> spp.	34	Not detected		US	Riser <i>et al.</i> (1984)
<i>Salmonella</i> spp.	116	1 (0.9%)		US	USFDA (2001)
<i>Salmonella</i> spp.	80	5 (6.25%)		Spain	Garcia-Villanova Ruiz <i>et al.</i> (1987)

	<i>Salmonella</i> spp.	114	2 (1.8%)		US	Stier & Nagle (2001)
	<i>Salmonella</i> spp.	28	2 (7.1%)		Netherlands	Tamminga <i>et al.</i> (1978)
	<i>Salmonella</i> spp.	120	68.3%		Italy	Ercolani (1976)
	<i>Shigella</i> spp.	151	Not detected		UK	Little <i>et al.</i> (1999)
	<i>Shigella</i> spp.	114	Not detected		US	Stier & Nagle (2001)
	<i>Shigella</i> spp.	116	1 (0.9%)		US	USFDA (2001)
	<i>Staphylococcus</i> spp.	? (>1x10g)	14.3%		Lebanon	Abdelnoor (1983)
	<i>Staphylococcus aureus</i>	144	33 (22.9%)		Spain	Soriano <i>et al.</i> (2000)
	<i>S. aureus</i>	34	Not detected		US	Riser <i>et al.</i> (1984)
	<i>S. aureus</i>	40	1 (2.5%)		Spain	Soriano <i>et al.</i> (2001)
	<i>S. aureus</i> (enterotoxigenic)	10	1 (10%)		US	Thunberg <i>et al.</i> (2002)
Lettuces (organic)	<i>Aeromonas</i> spp.	8	2 (25%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	8	Not detected		UK	McMahon & Wilson (2001)
Mushrooms	<i>C. jejuni</i>	200	3 (1.5%)		US	Doyle & Schoeni (1986)
	<i>Clostridium botulinum</i>	50	Not detected	<0.08-0.16 MPN/100g	Netherlands	Notermans (1993)
	Coliforms (fecal)	40x(25g from 12 mushrooms)		<2 log CFU/g	Spain	Gonzalez-Fandos <i>et al.</i> (2000)
	Coliforms (fecal)	28x(25g from 12 mushrooms)		<1 CFU/g	Spain	Gonzalez-Fandos <i>et al.</i> (2001)
	Coliforms (thermotolerant)	156	6 (3.8%)		Norway	Johannessen <i>et al.</i> (2002)
	<i>E. coli</i>	40x(25g from 12)	Not isolated		Spain	Gonzalez-Fandos <i>et al.</i> (2000)

	<i>E. coli</i>	mushrooms) 28x(25g from 12	Not isolated		Spain	Gonzalez-Fandos <i>et al.</i> (2001)
		mushrooms)				
	<i>Listeria</i> spp.	92	11 (12%)		US	Heisick <i>et al.</i> (1989)
	<i>L. monocytogenes</i>	25g stems	Not detected		US	Petran <i>et al.</i> (1988)
	<i>L. monocytogenes</i>	156	1 (0.6%)		Norway	Johannessen <i>et al.</i> (2002)
	<i>L. monocytogenes</i>	28x(25g from 12	Not isolated		Spain	Gonzalez-Fandos <i>et al.</i> (2001)
		mushrooms)				
	<i>Staphylococcus</i> spp.	156	35 (22.4%)		Norway	Johannessen <i>et al.</i> (2002)
	<i>S. aureus</i>	40x(25g from 12	Not isolated		Spain	Gonzalez-Fandos <i>et al.</i> (2000)
		mushrooms)				
Mushrooms (organic)	<i>Aeromonas</i> spp.	12	1 (8%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E.</i> <i>coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	12	Not detected		UK	McMahon & Wilson (2001)
Onions	APC	3 (outer leaves)	1 (33%)	6.5 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	3 (mid-leaves)	1 (33%)	4.0 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	3 (inner leaves)	1 (33%)	4.5 log CFU/g	Japan	Kaneko <i>et al.</i> (1999)
	APC	3		3.54 log CFU/g	Spain	Arroyo <i>et al.</i> (1999)
	<i>Clostridium botulinum</i>	75	5 (6.7%)		US	Notermans (1993)
	<i>Salmonella</i> spp.	6	6 (100%)		US	Martin & Katz (1991)
Onions (organic)	<i>Aeromonas</i> spp.	3	Not detected		UK	McMahon & Wilson (2001)

	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	3	Not detected	UK	McMahon & Wilson (2001)
Potatoes	<i>Campylobacter</i> spp.	153	1/153 (0.7%)	Canada	Park & Sanders (1992)
	<i>Campylobacter</i> spp.	75	1/75 (1.3%)	Canada	Park & Sanders (1992)
	<i>Campylobacter</i> spp.	10	Not detected	US	Thunberg <i>et al.</i> (2002)
	<i>Clostridium botulinum</i>	26	Not detected	Hungary	Notermans (1993)
	<i>Listeria</i> spp.	10	4/10 (40%)	US	Thunberg <i>et al.</i> (2002)
	<i>Listeria</i> spp.	132	34 (25.8%)	US	Heisick <i>et al.</i> (1989)
	<i>L. monocytogenes</i>	25g peel	Not detected	US	Petran <i>et al.</i> (1988)
	<i>Salmonella</i> spp.	10	Not detected	US	Thunberg <i>et al.</i> (2002)
Potatoes (organic)	<i>Aeromonas</i> spp.	3	Not detected	UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	3	Not detected	UK	McMahon & Wilson (2001)
Spring onions (organic)	<i>Aeromonas</i> spp.	2	Not detected	UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E. coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	2	Not detected	UK	McMahon & Wilson (2001)
Tomatoes	APC	10g	4.1 log CFU/ml	Spain	Arroyo <i>et al.</i> (1997)
	APC	10g	3.3 log CFU/ml	Spain	Arroyo <i>et al.</i> (1997)
	APC	12	5.0x10 ³ CFU/g	India	Pingulkar <i>et al.</i> (2001)
	APC	3	3.55 log CFU/g	Spain	Arroyo <i>et al.</i> (1999)

	Coliforms	12		3-240 MPN/g	India	Pingulkar <i>et al.</i> (2001)
	<i>E. coli</i> O157:H7	123	Not detected		US	Stier & Nagle (2001)
	<i>E. coli</i> O157:H7	20	Not detected		US	USFDA (2001)
	<i>Listeria</i> spp.	12	Present		India	Pingulkar <i>et al.</i> (2001)
	<i>L. monocytogenes</i>	15	2 (13.3%)		Pakistan	Vahidy (1992)
	<i>L. monocytogenes</i>	20	Not detected		Canada	Farber <i>et al.</i> (1989)
	<i>Salmonella</i> spp.	20	Not detected		US	USFDA (2001)
	<i>Salmonella</i> spp.	123	Not detected		US	Stier & Nagle (2001)
	<i>Shigella</i> spp.	20	Not detected		US	USFDA (2001)
	<i>Shigella</i> spp.	123	Not detected		US	Stier & Nagle (2001)
	<i>Staphylococcus</i> spp.	50 (10x5 lots)		0 - 9.62x10 ⁵ CFU/g	India	Pingulkar <i>et al.</i> (2001)
Tomatoes (organic)	<i>Aeromonas</i> spp.	4	1 (25%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E.</i> <i>coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	4	Not detected		UK	McMahon & Wilson (2001)
Turnips (organic)	<i>Aeromonas</i> spp.	3	1 (33%)		UK	McMahon & Wilson (2001)
	<i>Salmonella</i> spp., <i>Campylobacter</i> spp., <i>E.</i> <i>coli</i> , <i>E. coli</i> O157:H7, <i>Listeria</i> spp.	3	Not detected		UK	McMahon & Wilson (2001)
Yams	<i>Campylobacter</i> spp.	5	Not detected		US	Thunberg <i>et al.</i> (2002)
	<i>Listeria</i> spp.	5	1 (20%)		US	Thunberg <i>et al.</i> (2002)
	<i>Salmonella</i> spp.	5	Not detected		US	Thunberg <i>et al.</i> (2002)
Various organic	<i>Campylobacter</i> spp.	3200	Not detected			
	<i>E. coli</i>	3200	48 (1.5%)		UK	Sagoo <i>et al.</i> (2001)

vegetables*	<i>E. coli</i> O157:H7	3200	Not detected	UK	Sagoo <i>et al.</i> (2001)
	<i>Listeria</i> spp.	3200	6 (0.2%)	UK	Sagoo <i>et al.</i> (2001)
	<i>L. monocytogenes</i>	3200	Not detected	UK	Sagoo <i>et al.</i> (2001)
	<i>Salmonella</i> spp.	3200	Not detected	UK	Sagoo <i>et al.</i> (2001)

* Includes: Broccoli (209), cabbage (159), carrot (478), cauliflower (70), celery (193), lettuce (415), mushrooms (425), spring onions (87), cucumber (221), pepper (184), tomato (428), other (incl. Leeks and shallots) (208)

5.2 Likelihood of Pathogen Growth

Some information on the growth of pathogens in produce has been given above. Growth is unlikely on most fruits, an exception being melon because of its relatively high pH. Where produce is stored at temperatures $<7^{\circ}\text{C}$ the growth of most of the pathogens considered would be inhibited, with the exception of *L. monocytogenes* and *Aeromonas*. At higher temperatures the likelihood of growth will be dependent on the organism and the food, making it difficult to generalise. However, growth of pathogens on vegetables and high pH fruit is likely to occur at temperatures above 7°C .

5.3 Fruit and Vegetable Export Volumes

The information presented in this section indicates the importance to the New Zealand economy of each type of fresh produce exported. In the context of this risk assessment, the export values are indicative of export volumes, which is proportional to the extent of New Zealand derived hazard that overseas consumers are exposed to. Export values were used in this section due to the lack of data available on actual export volumes. Conventionally produced fruits and vegetables are considered separately, followed by what is known regarding the values and volumes of exported organic produce.

5.3.1 Conventional

The export value of conventionally grown fruits and vegetables are given in Table 8, and presented graphically in Figures 1 and 2.

Table 8 Export Values of Fresh Fruits and Vegetables for the Year Ended June, 2001 (conventional)

(Sources: <http://www.maf.govt.nz>, <http://tradenz.govt.nz>, HortResearch (2001))

Fruit produce		NZ\$ million	Vegetable produce		NZ\$ million
<i>Berry fruit</i>	Blueberries	5.9	Asparagus		13.9
	Strawberries	13.7	Avocado		26.1
	Other berry fruit ¹	0.5	Beans		0.6
<i>Citrus</i>	Lemons	2.9	<i>Brassicas</i> ³		0.8
	Oranges	0.7	Capsicums		22.6
	Mandarins	4.1	Carrots		20.2
	Other citrus fruit ²	0.3	<i>Cucurbits</i> ⁴		1.8
<i>Summerfruit</i>	Apricots	6.8	Garlic		3.6
	Cherries	7.7	Lettuces		0.5
	Nectarines	0.3	Mushrooms		2.3
	Peaches	0.1	Onions		96.8
	Plums	0.2	Potatoes		16.7
	Tamarillos	0.7	Squash		70.3
<i>Other Fruit</i>	Apples	339.0	Sweet corn		3.1
	Feijoa	0.4	Tomatoes (greenhouse)		0.8
	Kiwifruit	585.2	Other vegetables ⁵		3.4
	Melons	4.4			
	Nashi	0.6			
	Passionfruit	0.6			
	Pears	6.2			
	Persimmons	9.9			

¹ Includes blackberries, blackcurrents, boysenberries and raspberries.

² Includes tangelos and grapefruit.

³ Includes broccoli, cabbages and cauliflower.

⁴ Includes courgette/zucchini, scallopini, cucumber, and pumpkin (1999 export value).

⁵ Includes celery, leek, parsnip, shallots, spring onion, swede, taro and witloof (endive) and yam.

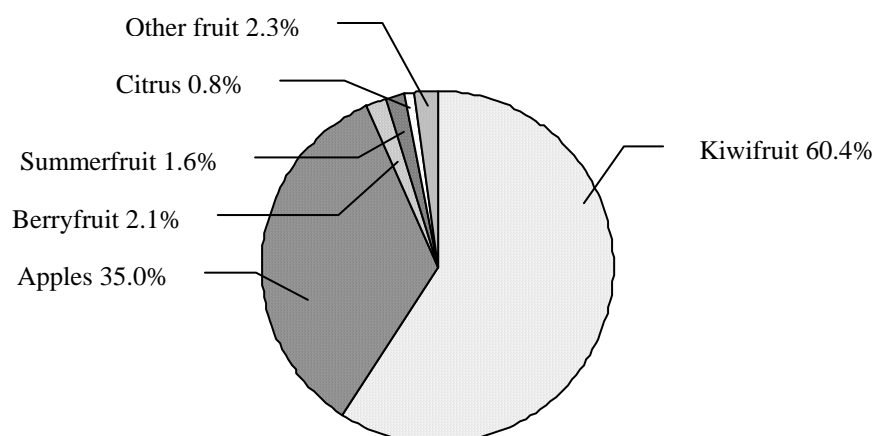


Figure 1 Export value of Fruit in 2001 (NZ\$million)

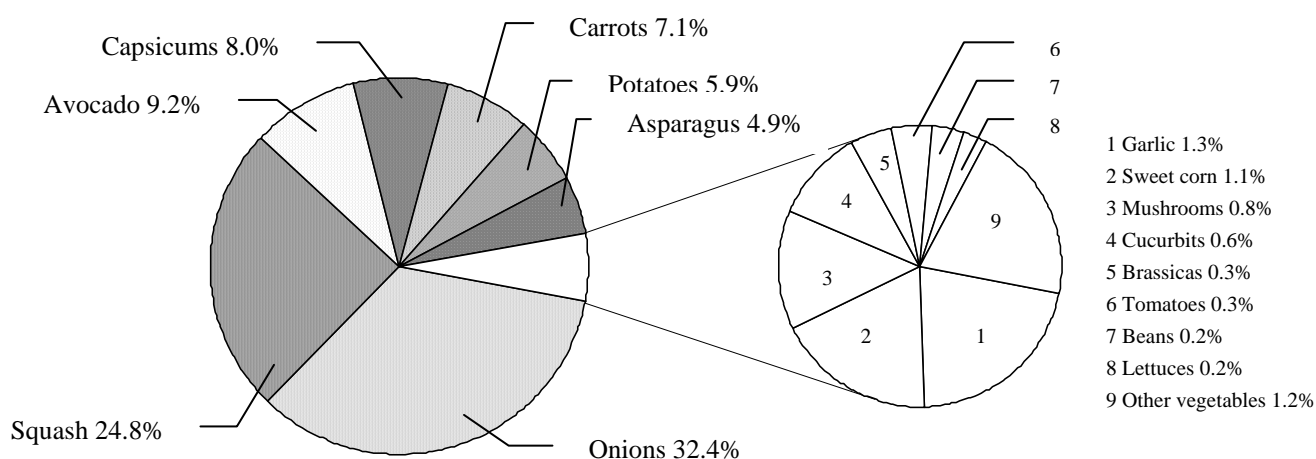


Figure 2 Export value of Vegetables in 2001 (NZ\$million)

5.3.2 Organic

In the year ended June 2001, fresh fruit accounted for 71% (approx. NZ\$49.7 million) of all exported organic products, and fresh vegetables 3% (approx. NZ\$2.1 million) (Hortresearch, 2001).

The majority of exported fresh organic produce consists of apples and kiwifruit with export values of approximately \$20 million and \$30 million respectively for the year ended June 2002 (S. Mason, pers. comm.). Other organic produce exported includes berryfruit (blueberries, strawberries), pears, summerfruit (apricots, cherries, nectarines, peaches), nashi, persimmons, feijoa, asparagus, onions, squash, and avocados (www.freshco.co.nz; S. Mason, pers. comm.). A complete list of organic fresh produce exported from New Zealand and their relative value was not attainable.

5.4 Likelihood Of Pathogen Removal by Consumer Preparation Prior To Consumption

It seems reasonable to assume that the majority of fruit is eaten raw, although a proportion will be processed before consumption. For fruits such as kiwifruit and citrus, peeling before consumption is a common practice which is likely to remove external pathogens that may be present. Fruit that is not peeled may or may not be washed before consumption depending on consumer behaviour.

A number of the vegetables, e.g. onions, squash, asparagus, potatoes and sweetcorn will be most commonly cooked prior to consumption, and this is likely to be enough to inactivate the vegetative cells of most bacterial pathogens. Many other vegetables may or may not be cooked (e.g. capsicums), and others rarely cooked (e.g. lettuce). Some vegetables, though eaten raw, are commonly peeled (e.g. carrots and avocados), but washing prior to consumption of raw vegetables depends again on consumer behaviour. The extent of removal of any pathogens that may be present is directly proportional to the degree of preparation by the consumer.

6 RISK CHARACTERISATION

6.1 Adverse Health Effects in New Zealand

Table 9 shows the most recently available data (to September, 2002) for notifiable foodborne disease in New Zealand. Fortunately the trend is for disease with the most serious clinical outcomes to occur less frequently than those with less severe clinical consequences. Some information can be inferred from these data. For example, the absence of cases of botulism suggests that New Zealand-grown produce (or any other food) is not a significant contributor of foodborne disease from this pathogen.

Table 9 Incidence of Disease Caused by Organisms of Concern in New Zealand

Disease	Number of cases (1 Jan - 30 Sept 2002)	Reported incidence (cases/100,000/year)
<i>Aeromonas</i> infection		Not a notifiable disease
<i>Bacillus cereus</i> intoxication		Not a notifiable disease
Campylobacteriosis	9,011	349.2
<i>Clostridium botulinum</i> intoxication	0	0
STEC infection	60	1.9
<i>Listeria monocytogenes</i> infection	13	0.5
Salmonellosis	1,486	59.7
Shigellosis	91	3.0
<i>Staphylococcus aureus</i> intoxication		Not a notifiable disease

Source: ESR data (www.esr.cri.nz/surveillance/monthly_surveillance.html)

Compared to international incidence rates, New Zealand is notable for the very high rates of campylobacteriosis. Rates of infection with other organisms in Table 9, where known, are similar to comparable countries overseas. In terms of disease transmission routes there are no known major differences here compared to overseas except for the case of *E. coli* O157:H7. In the USA infection by this organism is often transmitted through undercooked meat, especially hamburgers. In New Zealand there is, so far, no evidence to support transmission by this route. The organism has been found in New Zealand cattle and so transmission via produce that has been contaminated by (or fertilised using) cow faeces is plausible.

A search of ESR data from 1995 to date concerning outbreaks of disease and cases of acute gastroenteritis (in its communicable disease definition) revealed no incidents of foodborne disease that could be unequivocally linked to fresh fruits or vegetables as a vehicle.

The reported data for nine outbreaks (three of salmonellosis, three of campylobacteriosis and one each of *B. cereus*, *S. aureus* and *C. perfringens*) included fruits or vegetables as suspected food vehicles. The fruits and vegetables included were avocado (4 outbreaks), lettuce (2), salad (2), garlic (2; mayonnaise and bread), vegetables (2; raw and “dish”) (more than one food may be suspected for each outbreak). However, in all cases other foods were consumed at the same meal and in many cases these included rather more plausible sources of the possible infections/intoxications.

Similarly equivocal data were found for acute gastroenteritis notifications (defined as gastroenteritis in a food handler, healthcare worker, childcare worker, or child in an early childhood centre, or gastroenteritis in 2 or more people with a history of common exposure to food or water). Of these 23 were attributed to salads, 18 to salad-containing sandwiches/rolls, six to coleslaw, three to sushi (two of which contained avocado), and one each to avocado, blueberry ice cream, courgettes, fruit salad, grapes and pineapple. Nearly all of these foods were handled in some way, introducing the possibility contamination may have been introduced rather than being on the produce initially. Unequivocal links between produce and disease cannot therefore be readily made given these data.

6.2 Adverse Health Effects Internationally

Outbreaks of foodborne disease associated with produce from international data are summarised in Table 10. Much of the data available pertained to the US, though information on some outbreaks elsewhere were located. There is a clear pattern whereby *Salmonella* is the most commonly implicated pathogen across several fruits and vegetables, with the exception of lettuce where outbreaks have generally been caused by *E. coli* O157:H7.

Table 10 Overseas Outbreaks of Bacterial Foodborne Disease where Raw Produce was the Vehicle

Product	Year	Location	Agent	Vehicle	Cases	Deaths	Reference
<i>Fruit</i>							
Melons	1990	US	<i>Salmonella</i> Chester	Cantaloupes (from Mexico)	245	2	Beuchat (1996); D'Aoust (1994)
	1991	US and Canada	<i>Salmonella</i> Poona	Cantaloupes	>400		CDC, 1991
	1991	US	<i>Salmonella</i> Javiana	Watermelons	39		Nutritionaction website ^a
	1991	England	<i>Salmonella</i> Enteritidis	Cantaloupes			D'Aoust (1994)
	1991	US	<i>E. coli</i> O157:H7	Cantaloupes	27		Nutritionaction website
	1997	US	<i>Salmonella</i> Saphra	Cantaloupes (from Mexico)	24		Mohle-Boetani et al. (1999)
			<i>Shigella sonnei</i>	Watermelons			Fredlund et al. (1987)
	2000	US	<i>Salmonella</i> Poona	Cantaloupes	46		FSNet ^b 15/05/01
	2001	US	<i>Salmonella</i> Poona	Cantaloupes (from Mexico)	30	2	FSNet 25/05/01, 15/05/01
	2002	US	<i>Salmonella</i> Poona	Cantaloupes (from Mexico)	58		FSNet 21/11/02
<i>Vegetables</i>							
Cabbage	1981	Canada	<i>Listeria monocytogenes</i>	Cabbage (in coleslaw)	7+34 ^c		Schlech et al. (1983)
Cucumber		Australia	<i>Campylobacter</i>	Cucumbers	78		Kirk et al. (1997)
Green beans		British Columbia	<i>Bacillus cereus</i>	Green bean salad (beans cooked?)	300		Schmitt et al. (1976)
Lettuce	1983	US	<i>Shigella sonnei</i>	Lettuce (in salad)	2 o/bks ^d		Martin et al. (1986)
	1993	US	<i>Salmonella</i> Senftenberg	Lettuce (cross-contamination)	22		L'Ecuyer et al. (1996)

	1994	NW Europe	<i>Shigella sonnei</i>	Iceberg lettuce (from Spain)	>100 adults		Frost et al. (1995)
	1995	Canada	<i>E. coli</i> O157:H7	Iceberg lettuce (imported)	21		Canada Communicable Disease Report, 23(5), 1997
	1995	US	<i>E. coli</i> O157:H7	Lettuce	>70		Nutritionaction website
	1995	US	<i>E. coli</i> O157:H7	Romaine lettuce	20		Nutritionaction website
	1995	US	<i>E. coli</i> O157:H7	Iceberg lettuce	30		Nutritionaction website
	1996	US	<i>E. coli</i> O157:H7	Mesclun lettuce (faecal contamination)	61		Hilborne et al. (1999); FSNet 12/08/99
	1996	US	<i>Campylobacter jejuni</i>	Lettuce	14		Nutritionaction website
	1998	US	<i>Shigella sonnei</i>	Lettuce	160		Nutritionaction website
	1999	US	<i>E. coli</i> O157:H7	Iceberg lettuce	72		Nutritionaction website
	2002	US	<i>E. coli</i> O157:H7	Romaine lettuce	50		FSNet 03/08/02
Potatoes	1985	UK	<i>E. coli</i> O157:H7	Handling of soiled potatoes	24	1	Morgan et al. (1988)
Tomatoes	1990	US	<i>Salmonella</i> Javiana	Raw tomatoes (water bath contamination)	176		Hedberg et al. (1999)
	1993	US	<i>Salmonella</i> Montevideo	Raw tomatoes (water bath contamination)	100		Hedberg et al. (1999)
	1999	US	<i>Salmonella</i> Baildon	Raw tomatoes	86	1	Cummings et al. (2001); FSNet 29/09/99
	2001	US	<i>Shigella flexneri</i>	Bruised tomatoes	900		FSNet 26/10/02
	2002	US	<i>Salmonella</i> Javiana	Diced tomatoes	141		CDC, 2002a

^a Nutritionaction website, <http://www.nutritionaction.org/new/prodhark.html>.

^b FSNet, Food Safety Network, <http://www.foodsfetynetwork.ca/#>.

^c 7 adults, 34 perinatal cases.

^d o/bks, outbreaks.

6.3 Qualitative Estimate of Risk

As suggested in the Expression of Interest, this risk assessment considers the following points:

1. Presence or absence of the pathogen in New Zealand
2. Availability of testing methodology
3. The risk the pathogen poses to human health in New Zealand
4. Likelihood of the pathogen occurring on a product
5. Export value of the produce likely to host the pathogen

This discussion addresses these points.

6.3.1 Presence or absence of the pathogen in New Zealand and Availability of testing methodology

All of the pathogens of interest are present in the New Zealand environment and may potentially be present on fresh produce, and all may be tested for with current laboratory methods.

6.3.2 The risk the pathogen poses to human health in New Zealand

There is a lack of data regarding the risk each of the pathogens pose to human health in New Zealand where produce is the vehicle of infection. Dose response models indicate that *Shigella*, *Campylobacter* and *E. coli* O157:H7 have the highest likelihood of causing disease at low dose. Of all the pathogens considered in this report *E. coli* O157:H7 is least likely to be found on fresh produce due to its low environmental prevalence relative to the others (as far as is known) which, excepting *Shigella*, are more environmentally ubiquitous.

Most of the pathogens of interest can be given a low priority because of their lack of association with foodborne disease involving raw produce (*Staphylococcus aureus*, *Bacillus cereus*, *Clostridium botulinum*), because they are equivocal pathogens (*Aeromonas hydrophila*), or where they are usually introduced through poor hygiene prior to consumption (*Shigella*, *Staphylococcus aureus*).

The lack of New Zealand data requires assumptions to be made from comparable overseas information. *L. monocytogenes* needs some discussion in particular. This organism is detected in produce at varying prevalences and is an organism that has been shown to grow in a variety of vegetables. However, apart from the landmark Canadian outbreak in 1981 due to contaminated coleslaw, there has been no proven subsequent association between disease and the food/hazard combination. Given that the organism is associated with soil and vegetation, it is one that is likely to be found in vegetables, and unlikely to be removed completely in subsequent washing/disinfection procedures. The low probability of infection at a given dose compared with other bacterial pathogens probably explains why the exposure of the population to *L. monocytogenes* contaminated produce (which must occur) does not result in large numbers of cases. Given this information we propose that the presence of *L.*

monocytogenes in raw produce is inevitable, largely uncontrollable by acceptable CCPs and unlikely to result in disease in anything other than exceptional circumstances. Detection of the organism in produce is therefore likely, but of little public health concern.

New Zealand is noted for its very high rates of campylobacteriosis compared to other similar countries. *Campylobacter* is frequently associated with the faeces of ruminant animals and so may come into contact with produce if improperly composted manure is used as a fertiliser. However, there is very little information to implicate produce as a vehicle of campylobacteriosis except where cross contamination has occurred, and surveys rarely detect this organism on produce. In addition *Campylobacter* survives less well than other pathogenic bacteria (although it survives best under cool moist conditions), and so initially contaminated produce may be free of the organism before it is consumed. There is no reason to suppose that *Campylobacter* is present in the New Zealand environment any more than in any other country. For example the prevalence and numbers of *Campylobacter* in New Zealand river water are very similar to data from Europe. Given these considerations, testing for *Campylobacter* is not suggested.

Salmonella seems to be the dominant aetiological agent among outbreaks of bacterial infections where fruit and vegetables have been implicated as the vehicle, followed by *E. coli* O157:H7. In outbreak data, *Salmonella* has been closely associated with cantaloupe melons and tomatoes. In survey data *Salmonella* has been detected on a wider variety of fruits and vegetables indicating its high prevalence, though it has not been detected on tomatoes. *E. coli* O157:H7 has rarely been found in microbiological surveys of fresh produce, however it is prevalent as a cause of outbreaks associated with fruit and vegetables, particularly where lettuces have been implicated.

The overseas outbreak data given in Table 10 suggest that the highest risks lie with lettuces, melons and tomatoes. The hazards associated with these foods include *Salmonella*, especially in melons and tomatoes, and *E. coli* O157:H7 with lettuces.

Lettuces are commonly eaten raw and may or may not be adequately washed before consumption. Though the volume exported from New Zealand is relatively small, the association with outbreaks in many countries, particularly of *E. coli* O157:H7, suggests this vegetable as a subject for further research. Both conventional and organic methods expose lettuces to pathogens present in soil and, potentially, in agricultural applications such as compost. Lettuces will be similar to other leafy ground-grown vegetables such as cabbages and witloof in terms of risk of contamination, and may in some ways be representative to these latter products when they are also consumed raw.

Melons have also been associated with outbreaks overseas with *Salmonella* as the pathogen most frequently implicated. However, where information was available it appears that the outbreaks overseas were primarily associated with poor handling, both in the source country (Mexico and central America) where the fruit was not rinsed and decontaminated, and at the retail end where cross-contamination and temperature abuse of the pre-cut product resulted in outbreaks. There is also a suspicion that melons grown in Mexico may be contaminated by reptiles, such as iguanas, that feed on melons (CDC 2002b). Melons grown in New Zealand are considered to be of low risk.

Tomatoes have also been associated with several salmonellosis outbreaks. Their relative risk compared to other combinations is reduced however, when the following are considered. Of the five outbreaks recorded, four resulted from poor handling. Two outbreaks were attributed to the same producer who did not prevent contamination in the packing shed water bath, and another occurred from fresh cut tomatoes (although the cause of contamination has not yet been established). In New Zealand, tomatoes intended for export are produced only in hothouses, thus removing many of the environmental sources that could lead to *Salmonella* contamination. There is the potential for *Salmonella* to be introduced via the irrigation system, but there have been no documented cases of this occurring.

6.3.3 Likelihood of the pathogen occurring on a product

The Tables presented in the sections above summarise information regarding the detection of pathogens and indicator organisms on produce resulting from surveillance studies. There is a need for caution as no survey has been truly comprehensive for a range of pathogens or range of foods; the data are therefore somewhat *ad hoc*. The data are also not for New Zealand, and there are no data of which we are aware for New Zealand produce.

6.3.4 Export value of the produce likely to host the pathogen

The fruit and vegetable products that New Zealand exports in the greatest value (apples, kiwifruit, squash and onions) are those which have little outbreak or survey data recorded to indicate they are major food safety risks. Exceptions occur where they have been further processed and microbial controls have been insufficient, such as unpasteurised apple juice which has been associated with several *E. coli* O157:H7 outbreaks (Dingman, 2000).

Of the largest value export vegetables (Figure 2), onions and squash will likely be cooked prior to consumption and are considered to be low risk. Carrots, capsicums and asparagus are also considered low risk as they are likely to meet with some degree of pre-consumption preparation (peeling, washing, cooking) and have not been associated with outbreaks. Avocados represent an unknown risk as no microbiological data were available. New Zealand data have suggested that avocados were implicated in several incidents of food-borne illness. However, the aetiological agents of these incidents were varied (*Clostridium*, *Salmonella*, *Campylobacter*) and the avocado was only one ingredient among others which were more likely to be the vehicles of illness. Due to the absence of outbreaks and illness that can be unequivocally linked with the product, plus the peeling involved in preparation, they are considered low risk. Greatest risk must be assigned to fruits and vegetables that are eaten raw with minimal preparation. This is evident in overseas outbreaks that have largely been associated foods that are (relatively) low value exports for New Zealand yet predominantly eaten raw, specifically melons, lettuces and tomatoes.

Kiwifruit, the largest export fruit (Figure 1), is usually peeled prior to consumption and considered low risk. Given the large volumes of apples exported and the limited data on pathogen prevalence (Table 4), this fruit may pose a currently unrecognised risk. There is very little information on the microbiological characteristics of apples, though there are several factors that may make them susceptible to contamination. Apples are grown outside where they are exposed to environmental sources of contamination such as dust, insects and birds, all of which may carry pathogens onto the fruit. Birds are likely to be of greater importance since they are known to be carriers of *Salmonella*. Faecal material from birds is

likely to reach the fruit, and there is opportunity for cross-contamination to other apples while being handled in water flumes. Apples are more commonly eaten raw without peeling and with or without washing, and in many aspects represent other orchard grown fruit such as pears, nashi, and summerfruit.

6.4 Response of Regulators to Survey Data

As part of this project, MAF requested ESR to consider what the response of food safety regulatory authorities may be to survey work which revealed the existence of a significant hazard on fresh produce that posed a risk to human health. Dr. Richard Whiting, USFDA, was asked to comment on the regulatory response to survey data becoming available. The full text of the question posed and response are contained in Appendix 1. Dr. Whiting's comments focus on largely what would happen with US-produced data, but the general message is that regulators such as USFDA and USDA FSIS try to work with industry to obtain data that are useful, especially for quantitative risk assessment. He gives a number of examples where industry has collected data and provided them to regulators in an form where data could not be attributed to individual companies. Data may also be provided once a product's shelf life has expired and hence is no longer available for purchase.

There is obviously a balance to be struck between the need for real data and protecting public health, and Dr. Whiting indicates that data of immediate public health concern could be acted on.

7 CONCLUSIONS AND RECOMMENDATIONS

Given the qualitative assessment of risk above it is recommended that survey work should be carried out on:

- *Escherichia coli* O157:H7 and lettuces

and either

- *Salmonella* and apples, or
- *Salmonella* and tomatoes

The choice between these last two food/hazard combinations can be made on the basis of which criteria MAF considers to be most important for the end-users of this work: Export value and volume, hence exposure, (apples), or the estimate of risk derived from overseas outbreak data (tomatoes). It is recognised that the export trade in organic products of tomatoes and lettuces is minimal, and so it will be necessary to sample local organic product and to assess the possible differences in handling that domestic and organic products might receive.

The detection of *E. coli* O157:H7 in any sample would be of public health significance because of the relatively high probability of disease at even small doses, and the serious consequences of disease. If this organism was present at numbers high enough for enumeration then this could be regarded as a result of significant public health consequence. The detection of *Salmonella* is less of a concern, as at low concentrations the probability of disease is very low. However, the presence of *Salmonella* in numbers high enough to be enumerated should be regarded as a result of public health significance.

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APPENDIX 1

Email correspondence with Dr. R. Whiting, USFDA.

Question:

Dear Dick,

I hope you don't mind me taking a few moments of your time to pose a question! We are doing some work for a client who would like to know the regulatory response might be to the results of a survey. For example, if a postgrad student tested 200 strawberry samples for foodborne pathogens (as surveillance, not outbreak response) and produced a few positives, what might the response (or range of responses) that a regulator such as the FDA might implement ? Are there any documented instances of regulatory activity following such a survey ?

If you are unable to help, or feel that there may be someone better placed to provide an answer I'd appreciate being pointed in their direction.

I hope to catch up with you at a conference sometime in the future!

Regards,

Andrew Hudson

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Response:

Andrew,

Good to hear from you. I'll take a crack at answering your question, we've faced it here in Washington with both FDA and FSIS. If samples are taken by the food inspectors and the inspection-enforcement people, they usually feel they must take action if a positive sample turns up. That's their culture and mission. What we've done is move the survey out of the inspection divisions and have another group to it. This is not usually a problem for the inspectors because they are fully occupied with their ongoing work and don't have time or resources to add a major survey to their work schedule.

For example, John Luchansky is finishing a major hot dog study for *Listeria*

desired by FSIS. He is in the Agricultural Research Service, the research agency of USDA, not FSIS, the inspection agency. To further industry cooperation, a third party was contracted to collect the samples, remove company identification and then forward the samples to John's lab for analysis. This means that some information about the source of the product is deliberately lost.

When doing our Listeria risk assessment the lack of quantitative data for most foods was very apparent. The Int'l. Dairy Foods Assoc volunteered to have milk surveyed (and would pay for it). They came to us to discuss the survey and testing protocol to insure the data would meet our needs and criteria. They then contracted an independent lab (who we knew) who collected the samples, ran the analyses, and gave IDFA the data. They passed the tabulated results on to us. For 7 other RTE foods the National Food Processors Assoc did the same thing. They did two foods on their own and then got a USDA grant to fund the other five. The survey typically has 4000 to 5000 samples per food, quantitative tests are run when a positive is detected. Again, they discussed the survey and microbiological methods with us, then hired a consumer survey company to collect samples from the supermarkets and two independent testing labs to do the analyses. They then passed the results on to us (and plan to publish the data). Specific company data was not retained. We had some additional limitations (compromises) that I thought were unnecessary such as not specifying whether the store was a major supermarket or small convenience store or whether the deli meats were sliced/packaged by the manufacture or handled in the store. However, this removed the survey out of the regulatory arena and protected the individual companies. We have a "Freedom of Information Act" where anyone can ask for data from the government and unless there are good security or confidentiality reasons it must be made public. But with this structure, the government doesn't have any of the background and raw data and thus can't make public what it doesn't have.

One other way to minimize the regulatory issue is to clearly specify in the protocol a time line for sample analysis and data handling the will not have results ready until after the food has gone through the marketplace.

We've done several surveys like this and I think their success has built up confidence with the industry that this is a good way to get high quality data that is useful to everyone. We haven't used a university yet but I think they could function as the third party between the industry and regulatory agencies.

This does not mean that the government will not take regulatory actions based upon the survey, public health problems may become evident that necessitate some action. However, it will be addressed on an industry-wide basis and can't be traced back to a specific company in the survey.

Hope this helps, I could provide additional information or pass this on to others for comment if you wish.

Regards,

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