MINIREVIEW

Salmonella, a cross-kingdom pathogen infecting humans and plants

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Abstract

Infections with non-typhoidal *Salmonella* strains are constant and are a non-negligible threat to the human population. In the last two decades, salmonellosis outbreaks have increasingly been associated with infected fruits and vegetables. For a long time, *Salmonellae* were assumed to survive on plants after a more or less accidental infection. However, this notion has recently been challenged. Studies on the infection mechanism in vegetal hosts, as well as on plant immune systems, revealed an active infection process resembling in certain features the infection in animals. On one hand, *Salmonella* requires the type III secretion systems to effectively infect plants and to suppress their resistance mechanisms. On the other hand, plants recognize these bacteria and react to the infection with an induced defense mechanism similar to the reaction to other plant pathogens. In this review, we present the newest reports on the interaction between *Salmonellae* and plants. We discuss the possible ways used by these bacteria to infect plants as well as the plant responses to the infection. The recent findings indicate that plants play a central role in the dissemination of *Salmonella* within the ecosystem.

Introduction

Numerous pathogenic bacteria appear to have a fairly broad spectrum of host organisms. Among them, *Salmonella* spp. efficiently infect animal and plant organisms. In this review, we focus on *Salmonella enterica*, a Gram-negative enteropathogenic bacteria and one of the main causes of food-borne poisoning today. Salmonellosis is unfortunately a constant threat to human health in developed as well as developing countries. A large study conducted in 2007 revealed that in the UK, the Netherlands, Germany and Ireland, 0.1–2.3% of pre-cut products were contaminated with *Salmonella* bacteria (Westrell *et al.*, 2009). In the USA, an estimated one of six citizens will become infected with food-borne pathogens (Centers for Disease Control & Prevention, 2011). In cases related to domestic food poisoning, salmonellosis was responsible for 11% of infections in the USA, 35% of these resulting in hospitalization and 28% in death (Centers for Disease Control & Prevention, 2011). The incidence of *Salmonella* infections has not declined in the USA in the last 15 years, making the non-typhoidal strains the second most common cause (after Norovirus) of food poisoning, with around 1 million infections per year (Centers for Disease Control & Prevention, 2011). Poultry and eggs are commonly associated with *Salmonella* outbreaks; however, 33% of infections in 2004–2008 were linked to other sources including: vine vegetables, fruits, nuts, sprouts, leafy greens, roots, and beans (Centers for Disease Control & Prevention, 2011). The assumption that *Salmonellae* passively survive on plants after occasional contamination has changed in the last few years. Research on the interaction between plants and these bacteria revealed an active infection process (for review see Brandl, 2006; Holden *et al.*, 2009; Schikora *et al.*, 2012a). Additionally, numerous reports point to suppression of the plant immune system prior to infection (Barak *et al.*, 2005, 2008, 2011; Iniguez *et al.*, 2005; Klerks *et al.*, 2007; Schikora *et al.*, 2008, 2011; Kroupitski *et al.*, 2009; Golberg *et al.*, 2011; Shirron & Yaron, 2011). Consequently, the consumption of *Salmonella*-contaminated raw vegetables and fruits has been identified as the source of infection in many recent out-

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Table 1. Salmonellosis outbreaks related to the consumption of contaminated fruits or vegetables during the last 14 years. The table is not an exhaustive list of outbreaks; we considered only those that were traced back to a vegetal contamination source

Year	Strain	Affected regions	Fruit/vegetable	Reference
1999	*S.* Mbandaka	USA	Alfalfa sprout	Gill *et al.* (2003)
2000	*S.* Typhimurium DT204b	Iceland, Netherlands, UK and Germany	Lettuce	Crook *et al.* (2003)
2000	*S.* Typhimurium DT104	UK	Lettuce	Horby *et al.* (2003)
2000	*S.* Enteritidis Phage type 4b	Netherlands	Bean sprouts	van Duynhoven *et al.* (2002)
2001	*S.* Enteritidis	Canada	Mung bean sprouts	Honish & Nguyen (2001)
2001	*S.* Baildon	USA	Tomatoes	Cummings *et al.* (2001)
2003	*S.* Newport	USA	Mangoes	Sivapalasingam *et al.* (2003)
2004	*S.* Thompson	Norway	Ruccola	Nygard *et al.* (2007)
2004	*S.* Newport	England, Scotland, Isle of Man and Ireland	Lettuce	Irvine *et al.* (2009)
2004	*S.* Mbandaka, *S.* Virchow	Serbia	Sesame seeds	Ilic *et al.* (2010)
2004	*S.* Braenderup	USA	Tomatoes	Gupta *et al.* (2007)
2004	*S.* Braenderup *S.* Javiana	USA	Tomatoes	Centers for Disease Control & Prevention C (2005)
2005	*S.* Newport	USA	Tomatoes	Greene *et al.* (2008)
2006	*S.* Enteritidis NST3	Sweden	Almonds	Ledet Muller *et al.* (2007)
2006	*S.* Typhimurium	USA	Tomatoes	Behravesh *et al.* (2012)
2007	*S.* Seftenberg	UK, Denmark, Netherlands and USA	Basil	Pezzoli *et al.* (2008)
2007	*S.* Weltvreden	Norway, Denmark, FInland	Alfafa sprouts	Emberland *et al.* (2007)
2007	*S.* Stanley	Sweden	Alfalfa sprouts	Werner *et al.* (2007)
2007	*S.* Paratyphi B variant Java	Sweden, UK Denmark	Spinach	Denny *et al.* (2007)
2008	*S.* Newport and *S.* Reading	Finland	Iceberg lettuce,	Lienemann *et al.* (2011)
2010	*S.* Bareilly	UK	Bean sprouts	Cleary *et al.* (2010)

breaks. Table 1 lists examples of *Salmonella* outbreaks related to plants in recent years.

Different ways to reach the vegetal host

Salmonella bacteria are able to persist in soil. Studies on native *Conzattia multiflora*, a legume tree indigenous to Mexico, revealed the existence of *Salmonella* plant-borne lines isolated from nodule-like structures (Wang *et al.*, 2006). *Salmonella enterica* ssp. *enterica* serovar Typhimurium (*S.* Typhimurium) was detected in the rhizosphere of several crop plants including: wheat (*Triticum sativum*), oilseed rape (*Brassica napus*) and strawberry (*Fragaria x ananassa*) (Berg *et al.*, 2005). An additional infection route could be water, which was in contact with human or animal waste. Contaminated water is a well-known dissemination path for numerous pathogens, including *Salmonella* (Islam *et al.*, 2004; Kisluk & Yaron, 2012). In this way, bacteria can move throughout the water supply system and subsequently reach crop fields where the contaminated water is used for irrigation (see Fig. 1 for different infection routes). *Salmonella enterica* populations, for instance, were bigger in the phyllosphere of tomato plants irrigated with contaminated water than in plants grown from seeds in pre-infested soil (Barak *et al.*, 2011). Infection of plants with *Salmonellae* may occur also with the help of other organisms. Co-inoculation with the nematode *Caenor-*

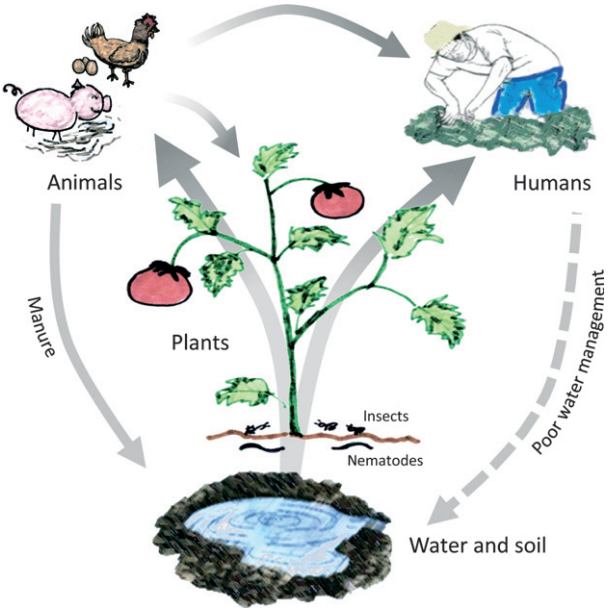

Fig. 1. Infection routes of *Salmonella enterica* in ecosystem. Plants play a crucial, although not yet fully recognized, role in the dissemination of *Salmonellae*. The rising awareness of the ability of *S. enterica* to infect plants and the probability that also other human pathogenic bacteria actively infect plant hosts opens a new view on how pathogenic bacteria spread within an ecosystem. This also places plants in a diversified picture of potential sources for human infection and argues for reconsideration of the currently used prevention practices.

habditis elegans and *Salmonella* Newport of lettuce, strawberries or carrots placed on the top of animal or composted manure resulted in plant infection with these bacteria; conversely, the plant tissues were not infected with the pathogen in the absence of the nematode (Kenney *et al.*, 2006). In addition, the free-living *C. elegans* has been found to ingest *S. enterica* (Aballay *et al.*, 2000). *Salmonellae* can also be transported *via* insects, e.g. by the pharaoh ant (*Monomorium pharaonis*), moving from indoor facilities to the environment, and therefore coming in contact with a range of possible hosts, including plants (Beatson, 1972). A recently published report on the interaction between *Salmonellae* and arbuscular mycorrhzal fungi (AMF) has demonstrated a higher persistence of *Salmonella* in plants colonized by AMF, which indicates another layer of interactions in the rhizosphere (Gurtler *et al.*, 2013).

Susceptibility to contamination by *S. enterica* via soil differs among agricultural crops. Members of the *Brassicaceae* family presented higher bacterial populations than tomato and lettuce (Barak *et al.*, 2008). However, lettuce was shown to have a higher contaminated phyllosphere, suggesting that other contamination routes, such as irrigation water, are also effective (Barak *et al.*, 2008). Important to note is the possibility that the plant itself might contribute to infection. Study performed on alfalfa sprouts demonstrated that during seed germination, the endosperm breakdown causes the release of reducing sugars and other organic molecules, which can be found in the irrigation water and, in consequence, produce a growth medium for *S. enterica*. Since bacteria metabolize these molecules as a source of nutrients, plant exudates seem to be a suitable nutrient source. The high multiplication rates of some of the *Salmonella* strains in effluxes of germinating seeds, prompted the suggestion of saprophytic growth (Howard & Hutcheson, 2003). Furthermore, other plant pathogenic bacteria may contribute to the infection with *Salmonella.* Pectinolytic bacterial pathogens, which cause soft rot and in this way mobilize nutrients, are frequently associated with infection of fruits and vegetables by *S. enterica* (Barak & Schroeder, 2012).

Adhesion and attachment to plant host

Successful attachment of bacteria to plant surfaces is the first step in the infection process. Studies on *Salmonella* attachment to diverse crop plants revealed that both the host plant and the bacterial genetic equipment influence the efficiency with which bacteria adhere to plants. Lettuce leaves, for example, show distinct attachment properties between older leaf parts and leaf regions near the petiole (Kroupitski *et al.*, 2011). Bacterial attachment to basil, lettuce or spinach leaves can differ among *S. enterica* serovars. Whereas *S.* Typhimurium, Enteritidis and Senftenberg are efficient in attaching to those plants, other serovars including Agona, Heidelberg or *Salmonella arizonae* are less effective (Berger *et al.*, 2011).

Biofilm formation could also influence the success of attachment to plant tissues. Biofilms are formed of a matrix of exopolymers, thin aggregative fimbriae (Tafi or curli), cellulose and lipopolysaccharide *O*-polysaccharide (also known as *O*-antigen) capsules that are involved in multicellular behavior and persistence under harsh environmental conditions (for review see Barnhart & Chapman, 2006; Latasa *et al.*, 2006). AgfD, a transcription regulator from the *Tafi* operon, positively regulates expression of those structural elements and confers two distinct *Salmonella* morphotypes: red dry and rough (rdar) or smooth and white (saw). Thus, the *Tafi* operon supports the persistence of the biofilm-associated bacteria in response to environmental conditions (Gibson *et al.*, 2006). In comparison with the saw morphotype, the rdar morphotype, which was isolated during tomato-originated outbreaks, adhered and attached better to tomato leaflets (Cevallos-Cevallos *et al.*, 2012). Similarly, on parsley plants, attachment and resistance to disinfection treatments are improved in biofilm-associated bacteria. The efficiency with which a biofilm protects bacteria from disinfecting agents seems to increase during storage and food processing because no significant difference was observed between the wild-type and mutants failing to produce biofilm when treated with chlorine at the early stages of infection (Lapidot *et al.*, 2006). A large screen of 6000 *S.* Newport mutants identified 20 mutants with lower attachment ability to alfalfa sprouts (Barak *et al.*, 2005). Interestingly, genes identified in this study code for the surface-exposed aggregative fimbria nucleator curli (*agfB*) and for the global stress regulator *rpoS*. Both proteins regulate the production of curli, cellulose and adhesins, important for biofilm formation. *AgfD* was also identified in this study. In addition, Barak *et al.* (2007) showed that *yihO* (involved in *O*-antigen capsule formation) and *bcsA* (coding for a cellulose synthase) are important for adhesion to alfalfa sprouts (Barak *et al.*, 2007), whereas cellulose and curli are involved in the transmission of *S.* Typhimurium from water to parsley leaves (Lapidot & Yaron, 2009). Another report characterized two additional genes (STM0278 and STM0650) as important factors for the infection of alfalfa sprouts, due to their essential role in formation of biofilm and swarming (Barak *et al.*, 2009).

The disease-like symptoms appearing on leaves in response to the attachment and recognition of bacteria (wilting and chlorosis) seem to depend on the structure of the bacterial capsule. Strains such as *S.* Senftenberg, *S.* Cannstatt, *S.* Krefeld and *S.* Liverpool, all of which belong to the serogroup E_4 (O: 1, 3, 19), possess the *O*-antigen and induce rapid wilting and chlorosis in

Arabidopsis plant. In contrast, infiltration with serovars lacking the *O*-antigen does not provoke such symptoms (Berger *et al.*, 2011). In contrast to *Arabidopsis*, infection with *S.* Typhimurium seems to cause no disease-like symptoms in tobacco plants (Shirron & Yaron, 2011). Whether this difference depends on the different immune responses remains to be verified.

Salmonella gain access to plant interior

Several natural entry points used by *Salmonellae* to infect plant organisms were described in recent reports. Stomata are natural openings responsible for gas exchange. *Salmonella enterica* was shown to aggregate near the open stomata of iceberg lettuce leaves and subsequently invade the inner leaf tissues under light conditions, suggesting that the pathogen is attracted to nutrients produced in photosynthetic active cells (Kroupitski *et al.*, 2009), and even competes for carbon sources with the natural endophytic microflora (Klerks *et al.*, 2007). Internalization via stomata was also documented by electron microscopy in arugula, basil and parsley (Golberg *et al.*, 2011). However, differences in *Salmonella* internalization were observed in different seasons; summer had the highest frequency of internalization (Golberg *et al.*, 2011). In addition, the type I glandular trichomes and hydratodes were shown to be a potential colonization site in tomato leaves, as confirmed by fluorescence and confocal laser microscopy (Barak *et al.*, 2011; Gu *et al.*, 2011, 2013).

The movement of bacteria within plants grown in fields contaminated with *Salmonellae* and the colonization of fruits is a very important agronomical concern. If present in the soil, *S.* Typhimurium systemically infects tomato plants including the fruits, without inducing any disease symptoms except for a slight reduction in plant growth (Gu *et al.*, 2011). Since plant-originated bacteria retain the ability to infect animals (Schikora *et al.*, 2011), plants infected before the harvest and storage/processing procedures are likely to be responsible for at least a part of the salmonellosis outbreaks associated with raw fruits and vegetables.

A very intriguing question is whether, as in the case of animals, *Salmonella* gains access to the intracellular region of plant host. Observations on *Arabidopsis* roots inoculated with *S.* Typhimurium strain 14028s expressing the green fluorescent protein (GFP) showed that bacteria are present within root hair cells already 3 h after infection (Schikora *et al.*, 2008); 20 h later, bacteria were also visible in other rhizodermal cells. The internalization rate observed in *Arabidopsis* protoplasts and tobacco cells was relatively low (Schikora *et al.*, 2008; Shirron & Yaron, 2011). Other reports that focused on mesophyll cells, indicated only the extracellular presence of *Salmonella* (Kroupitski *et al.*, 2009, 2011). Thus, bacterial internalization in root cells, or in leaf or fruit cells, needs to be examined further.

Bacteria require T3SS for efficient infection

Recent findings regarding the mechanism of plant infection indicate that the mechanisms used for plant and animal infections are similar (Schikora *et al.*, 2012b). Infiltration of *Arabidopsis* plants with the wild type *S.* Typhimurium and type III secretion system (T3SS) mutants demonstrated that both T3SSs encoded on *Salmonella* genome are important for a successful colonization (Schikora *et al.*, 2011, 2012b; Shirron & Yaron, 2011). The isogenic T3SS mutants of *S.* Typhimurium 14028s *prgH* and *invA* (encoded by *Salmonella* pathogenicity island (SP-1), and *ssaV* and *ssaJ* (encoded by SPI-2) had lower proliferation rates and enhanced hypersensitive response (HR)-related symptoms in *Arabidopsis* plants, suggesting that these mutants are unable to suppress HR-like symptoms (Schikora *et al.*, 2012b). Moreover, a transcriptome comparison of responses to infection with *Salmonella* wild-type and *prgH* mutant revealed that highly conserved *Arabidopsis* genes involved in defense are up-regulated upon infection with the *prgH* mutant (Schikora *et al.*, 2011). Another study analyzed the oxidative burst after exposure to *S.* Typhimurium in *Nicotiana tabacum* plants and cell suspensions. Living *Salmonella* did not trigger an oxidative burst, whereas heat-killed or chloramphenicol-treated bacteria were effective elicitors, indicating that the pathogen actively suppresses this immune response. Furthermore, deletion of *invA* reduced the ability of *Salmonella* to suppress the oxidative burst (Shirron & Yaron, 2011). In summary, all three reports imply that a functional secretion system is required for the suppression of the plant defense mechanism.

Plant responses to *Salmonella* infection

The involvement of the plant immune system in the response to a *Salmonella* attack was demonstrated in several independent publications. The previously mentioned oxidative burst in response to treatment with killed bacteria or the *invA* mutant (Shirron & Yaron, 2011), or the transcriptional reprogramming in response to the *prgH* mutant (Schikora *et al.*, 2011) are good examples of immune responses suppressed by wild-type bacteria. Another recently published example is the recognition of the *Salmonella* SseF effector protein (Ustün *et al.*, 2012). SseF elicits HR-like symptoms in *Nicotiana benthamiana*, and a loss of symptoms upon silencing of the SGT1 suppressor indicates that SseF is recognized in an R protein-

mediated mechanism. This recognition activates the effector-triggered immunity of the host plant (Ustün *et al.*, 2012) otherwise induced by a disarmed pathogen (Schikora *et al.*, 2012b). Additionally, plant hormones usually associated with defense responses (ethylene and jasmonic acid) were found to be of major importance; the JA-mediating pathway seems to be crucial for restricting *Salmonella* proliferation in *Arabidopsis* (Schikora *et al.*, 2008). However, salicylic acid and ethylene are also important in the resistance to *Salmonella* (Iniguez *et al.*, 2005). Furthermore, the mitogen-activated protein kinase (MAPK) cascades are involved in the response to *Salmonella* infection. Three MAP kinases commonly associated with immune response (MPK3, MPK4 and MPK6) are activated 15 min after contact with *Salmonella* bacteria (Schikora *et al.*, 2008). The role of MAPK cascades in the defense against these bacteria is further supported by the fact that the *mpk3* and *mpk6* mutants are highly susceptible to *S.* Typhimurium infection (Schikora *et al.*, 2008).

Conclusions

The infection potential of *Salmonella*-infected *Arabidopsis* leaves towards mice (Schikora *et al.*, 2011) and the increasing number of outbreaks related to raw fruit and vegetables (Table 1 and references within) are clear indications of the role of plants as dissemination vectors. In addition, recent findings support the notion that *Salmonellae* infect plants in an active process, which resembles the infection of animal hosts. The characterization of invasion mechanism(s), immune suppression and cellular reprogramming during infection of plant hosts is a very intriguing question, still requiring answers. The knowledge gained will surely increase our understanding of the bacterial infection mechanism and also provide suggestions for future prevention and food protection strategies.

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References

Aballay A, Yorgey P & Ausubel FM (2000) *Salmonella* Typhimurium proliferates and establishes a persistent infection in the intestine of *Caenorhabditis elegans*. *Curr Biol* **10**: 1539–1542.

Barak JD & Schroeder BK (2012) Interrelationships of food safety and plant pathology: the life cycle of human pathogens on plants. *Ann Rev Phytopathol* **50**: 12.11–12.26.

Barak JD, Gorski L, Naraghi-Arani P & Charkowski AO (2005) *Salmonella enterica* virulence genes are required for bacterial attachment to plant tissue. *Appl Environ Microbiol* **71**: 5685–5691.

Barak JD, Jahn CE, Gibson DL & Charkowski AO (2007) The role of cellulose and O-antigen capsule in the colonization of plants by *Salmonella enterica*. *Mol Plant Microbe Interact* **20**: 1083–1091.

Barak JD, Liang A & Narm K-E (2008) Differential attachment to and subsequent contamination of agricultural crops by *Salmonella enterica*. *Appl Environ Microbiol* **74**: 5568–5570.

Barak JD, Gorski L, Liang AS & Narm KE (2009) Previously uncharacterized *Salmonella enterica* genes required for swarming play a role in seedling colonization. *Microbiology* **155**: 3701–3709.

Barak JD, Kramer LC & Hao LY (2011) Colonization of tomato plants by *Salmonella enterica* is cultivar dependent, and type 1 trichomes are preferred colonization sites. *Appl Environ Microbiol* **77**: 498–504.

Barnhart MM & Chapman MR (2006) Curli biogenesis and function. *Annu Rev Microbiol* **60**: 131–147.

Beatson SH (1972) Pharaoh's ants as pathogen vectors in hospitals. *Lancet* **299**: 425–427.

Behravesh CB, Blaney D, Medus C *et al.* (2012) Multistate outbreak of *Salmonella* serotype Typhimurium infections associated with consumption of restaurant tomatoes, USA, 2006: hypothesis generation through case exposures in multiple restaurant clusters. *Epidemiol Infect* **140**: 2053–2061.

Berg GE, Eberl L & Hartmann A (2005) The rhizosphere as a reservoir for opportunistic human pathogenic bacteria. *Environ Microbiol* **7**: 1673–1685.

Berger CN, Brown DJ, Shaw RK, Minuzzi F, Feys B & Frankel G (2011) *Salmonella enterica* strains belonging to O serogroup 1,3,19 induce chlorosis and wilting of *Arabidopsis thaliana* leaves. *Eviron Microbiol* **13**: 1299–1308.

Brandl MT (2006) Fitness of human enteric pathogens on plants and implications for food safety. *Annu Rev Phytopathol* **44**: 367–392.

Centers for Disease Control and Prevention (2011) *Making Food Safer to Eat: Reducing Contamination from the Farm to the Table*. Centers for Disease Control and Prevention, Atlanta, GA.

Centers for Disease Control and Prevention C (2005) Outbreaks of *Salmonella* infections associated with eating Roma tomatoes, United States and Canada, 2004. *MMWR Morb Mortal Wkly Rep* **54**: 325–328.

Cevallos-Cevallos JM, Gu G, Danyluk MD & van Bruggen A (2012) Adhesion and splash dispersal of *Salmonella enterica* Typhimurium on tomato leaflets: Effects of rdar morphotype and trichome density. *Int J Food Microbiol* **160**: 58–64.

Cleary P, Browning L, Coia J *et al.* (2010) A foodborne outbreak of *Salmonella* Bareilly in the United Kingdom, 2010. *Euro Surveill* **15**: 1–3.

Crook PD, Aguilera JF, Threlfall EJ *et al.* (2003) A European outbreak of *Salmonella enterica* serotype Typhimurium definitive phage type 204b in 2000. *Clin Microbiol Infect* **9**: 839–845.

Cummings K, Barrett E, Mohle-Boetani JC *et al.* (2001) A multistate outbreak of *Salmonella enterica* serotype Baildon associated with domestic raw tomatoes. *Emerg Infect Dis* **7**: 1046–1048.

Denny J, Threlfall J, Takkinen J *et al.* (2007) Multinational *Salmonella paratyphi* B variant Java (*Salmonella* Java) outbreak, August–December 2007. *Euro Surveill* **12**: E071220–E071222.

van Duynhoven YT, Widdowson MA, de Jager CM *et al.* (2002) *Salmonella enterica* serotype Enteritidis phage type 4b outbreak associated with bean sprouts. *Emerg Infect Dis* **8**: 440–443.

Emberland KE, Ethelberg S, Kuusi M *et al.* (2007) Outbreak of *Salmonella* Weltevreden infections in Norway, Denmark and Finland associated with alfalfa sprouts, July–October 2007. *Euro Surveill* **12**: E071129–E071124.

Gibson DL, White AP, Snyder SD *et al.* (2006) *Salmonella* produces an *O*-antigen capsule regulated by AgfD and important for environmental persistence. *J Bacteriol* **188**: 7722–7730.

Gill CJ, Keene WE, Mohle-Boetani JC, Farrar JA, Waller PL, Hahn CG & Cieslak PR (2003) Alfalfa seed decontamination in a *Salmonella* outbreak. *Emerg Infect Dis* **9**: 474–479.

Golberg DK, Kroupitski Y, Belausovl E, Pinto R & Sela S (2011) *Salmonella* Typhimurium internalization is variable in leafy vegetables and fresh herbs. *Int J Food Microbiol* **145**: 250–257.

Greene SK, Daly ER, Talbot EA *et al.* (2008) Recurrent multistate outbreak of *Salmonella* Newport associated with tomatoes from contaminated fields, 2005. *Epidemiol Infect* **136**: 157–165.

Gu G, Hu J, Cevallos-Cevallos JM, Richardson SM, Bartz JA & van Bruggen AH (2011) Internal colonization of *Salmonella enterica* serovar Typhimurium in tomato. *PLoS ONE* **6**: e27340.

Gu G, Cevallos-Cevallos JM & van Bruggen AH (2013) Ingress of *Salmonella enterica* Typhimurium into tomato leaves through hydathodes. *PLoS ONE* **8**: e53470.

Gupta SK, Nalluswami K, Snider C *et al.* (2007) Outbreak of *Salmonella* Braenderup infections associated with Roma tomatoes, northeastern United States, 2004: a useful method for subtyping exposures in field investigations. *Epidemiol Infect* **135**: 1165–1173.

Gurtler JB, Douds DD Jr, Dirks BA, Quinlan JJ, Nicholson AM, Phillips JG & Niemira BA (2013) Survival of *Salmonella* and *E. coli* O157:H7 in soil and translocation into leek (*Allium porrum*) as influenced by arbuscular mycorrhizal fungi (*G. intraradices*). *Appl Environ Microbiol* **79**: 5.

Holden N, Pritchard L & Toth I (2009) Colonization outwith the colon: plants as an alternative environmental reservoir for human pathogenic enterobacteria. *FEMS Microbiol Rev* **33**: 689–703.

Honish L & Nguyen Q (2001) Outbreak of *Salmonella enteritidis* phage type 913 gastroenteritis associated with mung bean sprouts, Edmonton, 2001. *Can Commun Dis Rep* **27**: 151–156.

Horby PW, O'Brien SJ, Adak GK *et al.* (2003) A national outbreak of multi-resistant *Salmonella enterica* serovar Typhimurium definitive phage type (DT) 104 associated with consumption of lettuce. *Epidemiol Infect* **130**: 169–178.

Howard M & Hutcheson SW (2003) Growth dynamics of *Salmonella enterica* strains on alfalfa sprouts and in waste seed irrigation water. *Appl Environ Microbiol* **63**: 548–553.

Ilic S, Duric P & Grego E (2010) *Salmonella* Senftenberg infections and fennel seed tea, Serbia. *Emerg Infect Dis* **16**: 893–895.

Iniguez AL, Dong Y, Carter HD, Ahmer BM, Stone JM & Triplett EW (2005) Regulation of enteric endophytic bacterial colonization by plant defenses. *Mol Plant Microbe Interact* **18**: 169–178.

Irvine WN, Gillespie IA, Smyth FB, Rooney PJ, McClenaghan A, Devine MJ & Tohani VK (2009) Investigation of an outbreak of *Salmonella enterica* serovar Newport infection. *Epidemiol Infect* **137**: 1449–1456.

Islam M, Morgan J, Doyle MP, Phatak SC, Millner P & Jiang X (2004) Persistence of *Salmonella enterica* serovar Typhimurium on lettuce and parsley and in soils on which they were grown in fields treated with contaminated manure composts or irrigation water. *Foodborne Pathog Dis* **1**: 27–35.

Kenney SJ, Anderson GL, Williams PL, Millner PD & Beuchat LR (2006) Migration of *Caenorhabditis elegans* to manure and manure compost and potential for transport of *Salmonella* Newport to fruits and vegetables. *Int J Food Microbiol* **106**: 61–68.

Kisluk G & Yaron S (2012) Presence and persistence of *Salmonella enterica* serotype Typhimurium in the phyllosphere and rhizosphere of spray-irrigated parsley. *Appl Environ Microbiol* **78**: 4030–4036.

Klerks MMFE, van Gent-Pelzer M, Zijlstra C & van Bruggen AH (2007) Differential interaction of *Salmonella enterica* serovars with lettuce cultivars and plant-microbe factors influencing the colonization efficiency. *ISME J* **1**: 620–631.

Kroupitski YG, Golberg D, Belausov E, Pinto R, Swartzberg D, Granot D & Sela S (2009) Internalization of *Salmonella enterica* in leaves is induced by light and involves chemotaxis and penetration through open stomata. *Appl Environ Microbiol* **75**: 6076–6086.

Kroupitski YP, Pinto R, Belausov E & Sela S (2011) Distribution of *Salmonella* Typhimurium in romaine lettuce leaves. *Food Microbiol* **28**: 990–997.

Lapidot A & Yaron S (2009) Transfer of *Salmonella enterica* serovar Typhimurium from contaminated irrigation water to parsley is dependent on curli and cellulose, the biofilm matrix components. *J Food Prot* **72**: 618–623.

Lapidot AR, Romling U & Yaron S (2006) Biofilm formation and the survival of *Salmonella* Typhimurium on parsley. *Int J Food Microbiol* **109**: 229–233.

Latasa C, Solano C, Penades JR & Lasa I (2006) Biofilm-associated proteins. *CR Biol* **329**: 849–857.

Ledet Muller L, Hjertqvist M, Payne L, Pettersson H, Olsson A, Plym Forshell L & Andersson Y (2007) Cluster of *Salmonella enteritidis* in Sweden 2005–2006 – suspected source: almonds. *Euro Surveill* **12**: E9–E10.

Lienemann T, Niskanen T, Guedes S, Siitonen A, Kuusi M & Rimhanen-Finne R (2011) Iceberg lettuce as suggested source of a nationwide outbreak caused by two *Salmonella* serotypes, Newport and Reading, in Finland in 2008. *J Food Prot* **74**: 1035–1040.

Nygard K, Lindstedt BA, Wahl W *et al.* (2007) Outbreak of *Salmonella* Typhimurium infection traced to imported cured sausage using MLVA-subtyping. *Euro Surveill* **12**: E070315–E070315.

Pezzoli L, Elson R, Little CL *et al.* (2008) Packed with *Salmonella* – investigation of an international outbreak of *Salmonella* Senftenberg infection linked to contamination of pre-packed basil in 2007. *Foodborne Pathog Dis* **5**: 661–668.

Schikora A, Carreri A, Charpentier E & Hirt H (2008) The dark side of the salad: *Salmonella* Typhimurium overcomes the innate immune response of *Arabidopsis thaliana* and shows an endopathogenic lifestyle. *PLoS ONE* **3**: e2279.

Schikora A, Virlogeux-Payant I, Bueso E *et al.* (2011) Conservation of *Salmonella* infection mechanisms in plants and animals. *PLoS ONE* **6**: e24112.

Schikora A, García AV & Hirt H (2012a) Plants as alternative hosts for *Salmonella*. *Trends Plant Sci* **17**: 245–249.

Schikora M, Neupane B, Madhogaria S *et al.* (2012b) An image classification approach to analyze the suppression of plant immunity by the human pathogen *Salmonella* Typhimurium. *BMC Bioinformatics* **13**: 171.

Shirron N & Yaron S (2011) Active Suppression of early immune response in tobacco by the human pathogen *Salmonella* Typhimurium. *PLoS ONE* **6**: e18855.

Sivapalasingam S, Barrett E, Kimura A *et al.* (2003) A multistate outbreak of *Salmonella enterica* serotype Newport infection linked to mango consumption: impact of water-dip disinfestation technology. *Clin Infect Dis* **37**: 1585–1590.

Ustün SM, Müller P, Palmisano R, Hensel M & Börnke F (2012) SseF, a type III effector protein from the mammalian pathogen *Salmonella enterica*, requires resistance-gene-mediated signalling to activate cell death in the model plant *Nicotiana benthamiana*. *New Phytol* **194**: 1046–1060.

Wang ET, Tan ZY, Guo XW, Rodriguez-Duran R, Boll G & Martinez-Romero E (2006) Diverse endophytic bacteria isolated from a leguminous tree *Conzattia multiflora* grown in Mexico. *Arch Microbiol* **186**: 251–259.

Werner S, Boman K, Einemo I *et al.* (2007) Outbreak of *Salmonella* Stanley in Sweden associated with alfalfa sprouts, July–August 2007. *Euro Surveill* **12**: E071018–E071012.

Westrell T, Ciampa N, Boelaert F *et al.* (2009) Zoonotic infections in Europe in 2007: a summary of the EFSA-ECDC annual report. *Euro Surveill* **14**: 1–3.