

Special Issue: Specificity of plant–enemy interactions

# Plants as alternative hosts for *Salmonella*

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**Recent findings show that many human pathogenic bacteria can use multiple host organisms. For example, *Salmonella* Typhimurium can use plants as alternative hosts to humans and other animals. These bacteria are able to adhere to plant surfaces and actively infect the interior of plants. Similarly to the infection of animal cells, *S. Typhimurium* suppresses plant defense responses by a type III secretion mechanism, indicating that these bacteria possess a dedicated multi-kingdom infection strategy, raising the question of host specificity. In addition, evidence is accumulating that the interaction of *Salmonella* with plants is an active process with different levels of specificity, because different *Salmonella* serovars show variations in pathogenicity, and different plant species reveal various levels of resistance towards these bacteria.**

## Plant-originated salmonellosis

Several reports indicated that bacteria, which are pathogenic to humans and other mammals, are able to infect plants. *Salmonella enterica*, *Pseudomonas aeruginosa*, *Burkholderia cepacia*, *Erwinia* spp., *Staphylococcus aureus*, *Escherichia coli* O157:H7 and *Listeria monocytogenes* infect animals and plants [1–5]. Amongst these pathogens, *Salmonella* bacteria are the major cause of food poisoning. These Gram-negative enteropathogenic bacteria can successfully colonize animals, humans and plants. Their genus is divided into two species: *Salmonella bongori* and *Salmonella enterica*, encompassing several hundred isolates, which are typically named after the place of origin [6]. The species *S. enterica* is additionally divided into seven subspecies, one of them, *S. enterica* subsp. *enterica*, is the major cause of salmonellosis in humans. The most common mode of infection is ingestion of contaminated food or water. Moreover, many reports have linked food poisoning with the consumption of *Salmonella*-contaminated raw vegetables and fruits (for review see [2,7]). Studies in various European countries revealed that in 2007, 0.3–2.3% of raw vegetables were infected with *Salmonella* bacteria [8]. In the USA, the proportion of raw food-associated salmonellosis outbreaks increased from 0.7% in the 1960s to 6% in the 1990s [9], and crossed 25% in recent years [10]. Most studies on *Salmonella*–plant interactions suggested an epiphytic lifestyle of *Salmonella* on plants. However, a growing body of evidence

points to a directed process in which the bacteria infect various plants and use them as viable hosts (Table 1) [11–22]. The ability to infect and grow on such diverse hosts is a remarkable example of the lack of specificity seen in so many other microbes (Figure 1).

## Do plants serve as alternative hosts or are they part of the *Salmonella* life cycle?

Adhesion is typically the first step of an infection by *Salmonella*. Diverse *S. enterica* serovars have been shown to adhere to plant surfaces, and many *Salmonella* serovars bind to plants significantly better than for instance the pathogenic *E. coli* strain O157:H7 [23]. Evidence suggests that *Salmonella* actively attach to plant tissues and only viable bacteria can successfully colonize plants [19]. In a screen of 6000 *S. Newport* mutants, 20 mutants were identified with lower attachment ability to *Medicago sativa* (alfalfa) sprouts [12]. Interestingly, some of the genes identified in this study code for the surface-exposed aggregative fimbria nucleator curli (*agfB*) and for the global stress regulator *rpoS* which regulates the production of curli, cellulose and other adhesins that are important also for animal pathogenicity. *AgfD*, which was also identified in this study, plays not only a central role in the ability to attach to plant surfaces [24], but also in the environmental fitness and the pathogenicity of the bacteria toward animals [25]. In addition, it was shown that *yihO* (involved in O-antigen capsule formation) and *bcsA* (coding for a cellulose synthase) are also important for adhesion to alfalfa sprouts [24], whereas cellulose and curli are involved in transmission of *S. Typhimurium* from water onto parsley (*Petroselinum hortense*) leaves [26]. In another study, two previously uncharacterized genes (STM0278 and STM0650) were characterized as important factors for the infection of alfalfa sprouts, due to their essential role in biofilm formation and swarming [11]. It is thus becoming evident that the genetic equipment of *Salmonella*, previously thought to be animal-infection specific, plays an important role in the infection of animals and plants alike. Surprisingly, a comparative study on the internal colonization in lettuce (*Lactuca sativa*) leaves by five *S. enterica* serovars (Dublin, Enteritidis, Montevideo, Newport and Typhimurium) indicated significant differences between the different serovars, indicating that distinct genetic backgrounds have an impact on the pathogenic behavior towards plants [16]. A similar study conducted on the

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**Table 1. Known interactions between *Salmonella* and plants<sup>a</sup>**

<i>Salmonella</i> strain	Infected plant	Main finding	Refs
<i>S. Anatum</i> DMST 19600	Cabbage	Temperature-dependent susceptibility to infection	[46]
<i>S. enterica</i> Dublin	Lettuce	Colonization of lettuce and transcriptome of response to infection	[47]
<i>S. enterica</i> , diverse serovars	Lettuce	Different serovars vary in their colonizing capacity	[16]
<i>S. enterica</i> , diverse serovars	<i>Arabidopsis</i>	Strains from O-serogroup induce chlorosis and wilting in <i>Arabidopsis</i>	[30]
<i>S. enterica</i> , diverse serovars	Tomato, pepper	Cultivar-dependent colonization, trichomes as infection point	[13]
<i>S. enterica</i> , diverse serovars	Lettuce, cabbage	Serovar-dependent divergences in attachment to leaves	[27]
<i>S. Newport</i>	Alfalfa	Identification of two new genes required for attachment to plants	[11]
<i>S. Newport</i>	Alfalfa	Screen of 6000 mutants for their ability to attach to plant surface	[12]
<i>S. Newport</i> , Enteriditis, mutants	Alfalfa	Cellulose and O-antigen capsule play role in the attachment to plants	[24]
<i>S. Thompson</i> RM1987	Lettuce	Increased infection was observed in elderly leaves	[48]
<i>S. Typhimurium</i>	Barley ( <i>Hordeum vulgare</i> )	Colonization of barley roots	[49]
<i>S. Typhimurium</i>	Potato ( <i>Solanum tuberosum</i> )	Attachment to plant surface is an active process	[19]
<i>S. Typhimurium</i> 14028	Tomato fruits	Screen for bacterial genes expressed upon plant infection	[18]
<i>S. Typhimurium</i> 14028	<i>Arabidopsis</i>	Plants induce defense mechanisms after infection, bacteria internalized in plants cells	[20]
<i>S. Typhimurium</i> 14028	<i>Arabidopsis</i>	Suppression of plant immune system is T3SS-dependent	[21]
<i>S. Typhimurium</i> 14028, 1344	Tobacco	Wild type bacteria suppress plant defense reactions	[45]
<i>S. Typhimurium</i> DT104	Lettuce	A passage via lettuce increased attachment capacity to epithelial cells	[50]
<i>S. Typhimurium</i> MAE110, MAE119	Tomato	Bacteria spread systemically and colonize non-infected leaves and fruits	[22]
<i>S. Typhimurium</i> SL1344	Lettuce	Internalization via stomata is light dependent and requires chemotaxis	[17]
<i>S. Typhimurium</i> SL1344	Diverse	Internalization of bacteria varies between leafy vegetables	[14]
<i>S. Typhimurium</i> 14028	<i>Arabidopsis</i>	Plant defense is required for resistance towards <i>Salmonella</i>	[15]

<sup>a</sup>The majority of the studies focus on different serovars of *S. enterica* subspecies *enterica* interacting with *Arabidopsis* or plants traditionally associated with salmonellosis outbreaks such as lettuce, tomato and alfalfa. The list presented here summarizes the research on the interaction between *Salmonella* and the plant immune system, as well as the genetic requirement to infect plants. Due to length restrictions, it is impossible to cover comprehensively the broad literature of different plant-originated outbreaks and the anti-microbial activity of diverse plants.

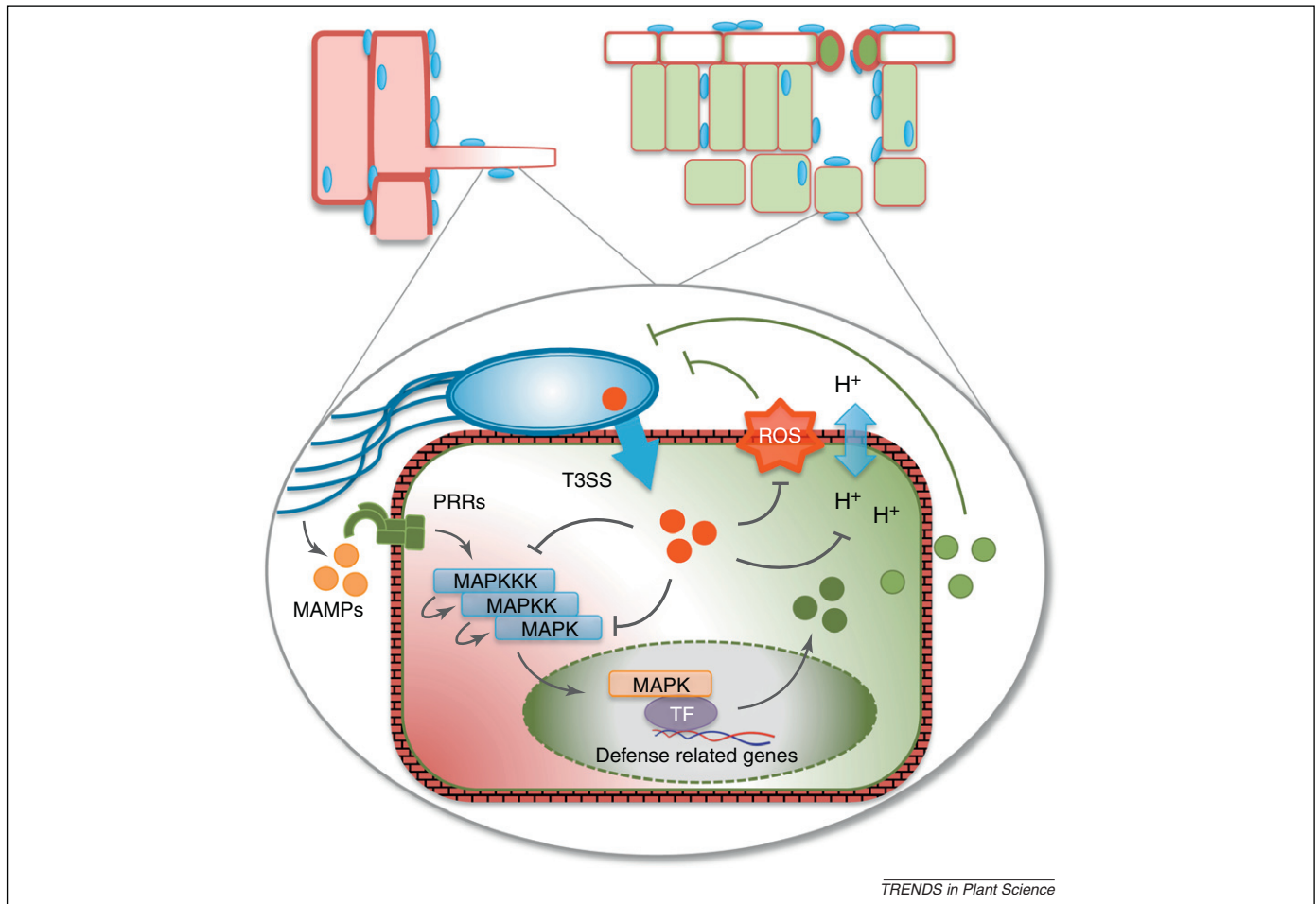
serovars Braenderup, Negev, Newport, Tennessee and Thompson, likewise revealed differences between the tested serovars [27]. Interestingly, the authors pointed out a correlation between the capacity to produce biofilms and the attachment to leaves, with *S. Thompson* producing the strongest biofilms and showing the most efficient adhesion to lettuce leaves [27].

### ***Salmonella* can live inside plants**

In animals, *Salmonella* actively enter epithelial and other cell types in order to replicate and spread through the organism. The question whether *Salmonella* use a similar strategy to infect plants is therefore of great interest. *Salmonella* were found to form biofilm-like structures on the surface of roots, preferentially colonizing regions around emerging lateral roots and wounded tissues [15,20]. The formation of biofilms of *Salmonella* on leaves was also reported. Recently, three reports presented the possible entry points of bacteria to the inner layers of leaves [13,14,17] and it was postulated that trichomes are preferential colonization sites [13]. By contrast, it was shown that *Salmonella* use stomata as entry points in order to penetrate lettuce leaves [17]. Moreover, bacterial aggregation near stomata occurs only under light conditions when the stomata are open. Artificial opening of the stomata in the dark had no impact on the bacterial behavior, suggesting that bacteria are attracted to photosynthesis-dependent products. Previously, we showed that the GFP-marked *S. Typhimurium* 14028s bacteria can be observed inside root hairs at 3 h, and bacterial titers increased at 20 h after inoculation of *Arabidopsis* plants [20].

Additional tests revealed that motility and the ability of chemotaxis are essential for *Salmonella* to colonize the interior of lettuce leaves [17]. In a follow-up report, the same group demonstrated that not all plants are equally susceptible (or resistant) to *Salmonella* internal infection. Using GFP-marked bacteria, the authors analyzed the internalization of the *S. Typhimurium* strain 1344 in many leafy vegetables and herbs [14]. In the same year, another study reported that *S. Typhimurium* strain MAE110 is able to translocate within tomato (*Solanum lycopersicum*) plants, infecting distal, non-infected leaves and fruits without visible symptoms and only slightly reducing plant growth [22]. Interestingly, while some plant species [e.g. arugula (*Diplotaxis tenuifolia*)], allowed *Salmonella* to internalize, some others (e.g. parsley), seemed to have effective means to prevent infection [14]. Studies on lettuce, cabbage (*Brassica oleracea*) and tomato demonstrated significant differences in the susceptibility to *Salmonella* infection [13,16], pointing to an important role of plant innate immunity in modulating the response to infection by these bacteria.

By contrast, pathogenic bacteria often use type III secretion system (T3SS)-dependent injection of effector proteins in order to modulate host physiology and suppress the immune system. To answer the question whether *Salmonella* rely on T3SS for infection of plants, mutants in two *Salmonella* T3SS were tested for their performance on plants. Both of the T3SS mutants are unable to inject effector proteins into host cells and are therefore not virulent for animal hosts [28,29]. Although these T3SS mutant strains showed normal proliferation rates when grown in standard medium, their proliferation in



**Figure 1.** Wild type *Salmonella* are able to attach to plant surfaces and infect plants via stomata openings or roots. Upon infection, *Salmonella* hinder the enhanced production of reactive oxygen species (ROS) and prevent pH changes in the apoplast. Moreover, *Salmonella* actively prevent the transcriptional activation of defense-related genes. Abbreviations: MAMP, microbe-associated molecular pattern (yellow circles); MAPK, mitogen-activated protein kinase; PRR, pattern recognition receptor; T3SS, type III secretion system; TF, transcription factor; red circles represent *Salmonella* effectors; green circles represent products of defense-related genes.

*Arabidopsis* (*Arabidopsis thaliana*) plants was compromised, indicating that both SPI-1- and SPI-2-encoded type III secretion systems are needed for successful plant infection [21].

### Plant responses to *Salmonella* infection

Upon inoculation, *Arabidopsis* responds to *Salmonella* with a rapid induction of defense responses, including the activation of mitogen-activated protein kinases MPK3, MPK4 and MPK6 that is followed by the expression of a number of defense genes, such as *PDF1.2* or the pathogenesis-related genes *PR2* and *PR4* [20]. Transcriptome analysis of *Arabidopsis* plants showed differential expression of about 250 and 1300 genes at 2 and 24 h after *Salmonella* infection, respectively. With the exception of 32 genes, the *Salmonella*-induced differentially expressed genes were also affected by inoculation with the non-pathogenic *E. coli* laboratory strain DH5 $\alpha$  and the pathogenic *Pseudomonas syringae* strain DC3000 [21]. Among the genes that were induced by *E. coli* DH5 $\alpha$ , *S. Typhimurium* 14028 and *P. syringae* DC3000, about 160 (including various WRKY and bZIP transcription factors as well as protein kinases and phosphatases) could be identified as a core set of *Arabidopsis* genes responsive to common bacterial exposure [21].

### Towards identification of the plant *Salmonella* receptors

A recent study examined the macroscopic symptoms of wilting and chlorosis in *Arabidopsis* plants after infiltration with different serovars of *S. enterica* subsp. *enterica*, as well as *S. enterica* subsp. *arizonae* and *diarizonae* [30]. Infiltration with *S. Senftenberg* and also with *S. Cannstatt*, Krefeld and Liverpool, all of which belong to the serogroup E<sub>4</sub> (O: 1, 3, 19) possessing the O-antigen, resulted in rapid wilting and chlorosis. By contrast, infiltration with serovars lacking the O-antigen provoked no symptoms [30]. In addition, the authors stated that the response to *Salmonella* infiltration is independent of the most prominent and studied pattern recognition receptors, suggesting that specific receptors for *Salmonella* O-antigen could exist in *Arabidopsis*.

### *Salmonella* factors interacting with the plant immune system

In humans, salmonellosis develops after the bacteria enter epithelial cells of the intestine [31]. Although a typical infection usually leads to a self-limiting gastroenteritis, *Salmonella* can cause systemic infections by invading spleen, liver and other organs in susceptible hosts. Studies of the infection mechanisms in animals have shown that

*Salmonella* actively remodel the host cell physiology and architecture, and suppress the host immune system by injecting a cocktail of effectors delivered by T3SS. A recently published literature survey revealed a standard list of 62 protein–protein interactions between 22 *Salmonella* proteins and numerous human proteins [32]. *Salmonella enterica* subsp. *enterica* has two distinct T3SSs, T3SS-1 and T3SS-2, encoded by the Salmonella Pathogenicity Islands (SPI) SPI-1 and SPI-2, respectively [33,34]. T3SS-1 secretes at least 16 proteins of which six were shown to interact with the host signaling cascades and the cytoskeleton. T3SS-2 secretes at least 19 *S. enterica*-specific effector proteins that are involved in survival and multiplication within the host cell [35,36]. The expression and the secretion of SPI-1 and SPI-2 encoded effectors are tightly regulated. Recently, the cytoplasmic SpaO–OrgA–OrgB complex was identified as the sorting platform for T3SS effectors that determines the appropriate hierarchy for protein secretion [37]. This complex enables the sequential delivery of translocases before the secretion of the actual effectors. The authors also described the role of specific chaperones in the recognition and loading of effectors into the sorting SpaO–OrgA–OrgB complex, and postulated that similar sorting platforms might exist in other bacteria [37]. Even though many reports suggest that the mechanisms used by *Salmonella* to infect animal and plant hosts could be similar, the role of *Salmonella* T3SS effectors during plant infections remains unclear. To date, 44 *Salmonella* effectors have been described to be injected into animal and human cells through one or both T3SSs (reviewed in [38]). Several of these effectors target MAPK cascades, which are important regulators of the immune response in animals and plants. SpvC from *Salmonella* spp. belongs to the OspF family initially identified in *Shigella* spp. OspF encodes a phosphothreonine lyase that dephosphorylates the pTXpY double phosphorylated activation loop in the ERK1/2 kinases [39–41]. Interestingly, *P. syringae* HopAI1 is a homolog of SpvC/OspF, and encodes a phosphothreonine lyase that dephosphorylates the threonine residue in the activation loop of activated MAPKs [42]. When expressed in *Arabidopsis*, HopAI1 directly interacts with MPK3 and MPK6, attenuating flg22-induced MAPK activation and downstream defense responses [40–42]. Besides OspF/SpvC/HopAI1, also the *Pseudomonas* effector HopPtoD2 has homologs in human pathogenic bacteria. HopPtoD2 is a tyrosine phosphatase which inhibits pathogen-triggered programmed cell death [43], while its homolog from *Salmonella* SptP, inhibits phosphorylation and membrane localization of Raf kinase and therefore the activation of ERK2 [44]. It is tempting to speculate that the biochemical features of these effectors are conserved between animal and plant hosts, providing *Salmonella* and other pathogenic bacteria with efficient tools for suppressing the host immune systems. A suppression of the defense responses was recently reported during the infection of tobacco (*Nicotiana tabacum*) plants with *S. Typhimurium*. In contrast to living *Salmonella*, dead or chloramphenicol-treated bacteria elicited an oxidative burst and pH changes in tobacco cells [45], indicating that *Salmonella* actively engages in the suppression of the

plant defense responses. Similar conclusions were reached when comparing the *Arabidopsis* responses against *S. Typhimurium* wild type and the T3SS mutants *invA* or *prgH*, which lack a functional T3SS [21,45]. Whereas *Salmonella* wild type and *prgH* mutants provoke changes in more than 1600 *Arabidopsis* genes after 24 h, a group of 649 genes is specifically induced by infection with the T3SS mutant. Many of these *prgH*-specific genes encode proteins related to pathogen responses and ubiquitin-mediated protein degradation. This group of genes also includes *BAK1*, *BIK1*, *WRKY18* and *WRKY33*, *EIN3*, *PR4* and *PUB23*, all of which are marker genes that are upregulated upon plant pathogen infections. The lower expression level of those genes upon infection with wild type *Salmonella* suggests that the T3SS mutant is unable to employ an effective immune suppression mechanism. These results suggest that *Salmonella* depend on the T3SS during plant infection and actively suppress immune responses.

### Concluding remarks

Along with *E. coli*, *Salmonella* belong to the best-studied bacteria today. The growing number of human infections with pathogenic bacteria derived from vegetables and fruits raise the question of the host specificity mechanisms of these bacteria. Recent reports clearly demonstrate that *Salmonella* not only passively survive, but also actively infect plants. Moreover, infection of plants depends on the active suppression of the host immune responses by *Salmonella*. Further studies are clearly warranted to uncover the extent to which the factors and mechanisms employed by *Salmonella* to infect animals are also used against plants and will likely lead to a better understanding of the evolution of specificity.

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