Phytopathogenic bacteria suppress plant innate immunity and promote pathogenesis by injecting proteins called type III effectors into plant cells using a type III protein secretion system. These type III effectors use at least three strategies to alter host responses. One strategy is to alter host protein turnover, either by direct cleavage or by modulating ubiquitination and targeting the 26S proteasome. Another strategy involves alteration of RNA metabolism by transcriptional activation or ADP-ribosylation of RNA-binding proteins. A third major strategy is to inhibit the kinases involved in plant defence signaling, either by the removal of phosphates or by direct inhibition. The wide array of strategies that bacterial pathogens employ to suppress innate immunity suggest that circumvention of innate immunity is crucial for bacterial pathogenicity of plants.

The virulence activity of several T3Es is thought to be because of their ability to suppress plant innate immunity, as well as uncovering new enzyme activities. These studies provide insight into how pathogen virulence and plant innate immunity work, as well as uncovering new enzyme activities.
Widespread screens to determine the effect of T3Es on plant immunity outputs demonstrate that certain T3Es can suppress the plant innate immunity [8–10] or upregulate hormones that are antagonistic to SA and other defence responses [11–13]. This review will focus on recent progress in elucidating T3Es activities, plant targets, and mechanisms of action. The impact of this new information on the guard hypothesis will also be addressed. The review is divided into sections on the basis of the following T3E activities: protein turnover, RNA expression or stability, protein phosphorylation and dephosphorylation, and clues about T3E activities on the basis of interactions with host proteins. Their sites of action and enzymatic activities are summarized in Figure 1 and Table 1.

**Figure 1**

Plant targets and activities of type III effectors from phytopathogenic bacteria. Bacterial plant pathogens inject many different type III effectors (T3Es) into plant cells via the type III secretion system. The activities of T3Es can be recognized by plant resistance (R) proteins inducing effector-triggered immunity (ETI). The R protein RPM1 causes ETI by recognizing the phosphorylation (P) of RIN4 by the T3E AvrRpm1 and AvrB, while RPS2 causes ETI upon the cleavage of RIN4 by the T3E AvrRpt2. The T3E AvrPphB degrades the PBS1 kinase inducing RPSS-dependent ETI. The R protein Prf recognizes the interaction of the Pto kinase with AvrPto or AvrPtoB to elicit ETI; however, AvrPtoB ubiquinates (Ub) the Fen kinase targeting it for degradation and preventing recognition by Prf. Plants can also use receptor kinases such as EFR or FLS2 to detect pathogen-associated molecular patterns (PAMPs). This leads to PAMP-triggered immunity (PTI). AvrPto inhibits the kinase activity of Pto, FLS2, and EFR. The HopAI1 T3E is a phosphothreonine lyase that suppresses MAPKs. The HopAO1 T3E is a protein tyrosine phosphatase whose target is unknown. The HopU1 T3E is a mono-ADP-ribosyltransferase that modifies GRP7 glycine-rich RNA-binding protein and probably prevents it from binding to RNA. The HopM1 T3E causes the ubiquination and degradation via the 26S proteasome of AtMIN7, which may be involved in vesicle trafficking. The GALA T3Es contain F-box domains and can interact with plant ASK proteins (part of an SCF-type E3 ubiquitin ligase complex). GALAs are predicted to change the ubiquitination status of host proteins. The T3Es XopD and AvrXv4, which function in different locations in the plant cell, are isopeptidases that remove SUMO (Su) from host proteins. The chloroplast localized, J domain-containing T3E HopP1, suppresses salicylic acid (SA) production and may associate with Hsp70. T3Es AvrBs3, PthXo6/7, and HsvG/B bind to specific promoters in the nucleus inducing the transcription of genes favoring pathogenesis. Broken lines indicate plant responses and solid lines T3E activities.
T3Es that impact host protein turnover

One way for T3Es to suppress plant innate immunity is to remove or inactivate its components. Certain T3Es are proteases that can remove these components by degradation. For example, AvrPphB from *P. syringae* is a papain-like cysteine protease that cleaves the *Arabidopsis* protein kinase PBS1 [14]. PBS1 exists in a complex with the R protein RPS5, which recognizes its cleavage and initiates ETI [18,19]. This recognition makes AvrPphB an obvious protein RPS5, which recognizes its cleavage and initiates PTI, which would apparently have a negative impact on pathogenic success. However, RIN4 interacts with at least two other T3Es and two R proteins suggesting that its inactivation may destabilize this complex and benefit the pathogen. One of these R proteins, RPS2, recognizes the cleavage of RIN4 by AvrRpt2 and induces ETI [18,19]. This was one of the first findings to provide molecular evidence in support of the guard hypothesis. AvrRpt2 probably targets additional host proteins since it can cleave other plant proteins and *P. syringae* virulence is increased by its presence in plants that lack RIN4 [20,21]. Additionally, AvrRpt2 was recently shown to be capable of altering auxin physiology [12], whether this is a direct or indirect effect remains to be determined.

Two families of T3Es (XopD and YopJ) are cysteine proteases/isopeptidases that specialize in the cleavage of ubiquitin or small ubiquitin-like modifiers (SUMOs) from proteins. As the addition of a single ubiquitin or SUMO to proteins can alter their activity, it is probably that the deubiquitinating/deSUMOylating activity of these T3Es alters the activity or stability of host proteins. XopD is a *X. campesstris pv. vesicatoria* (Xcv) T3E that is translocated to the nucleus where it is believed to mimic endogenous PTI, which would apparently have a negative impact on pathogenic success. However, RIN4 interacts with at least two other T3Es and two R proteins suggesting that its inactivation may destabilize this complex and benefit the pathogen. One of these R proteins, RPS2, recognizes the cleavage of RIN4 by AvrRpt2 and induces ETI [18,19]. This was one of the first findings to provide molecular evidence in support of the guard hypothesis. AvrRpt2 probably targets additional host proteins since it can cleave other plant proteins and *P. syringae* virulence is increased by its presence in plants that lack RIN4 [20,21]. Additionally, AvrRpt2 was recently shown to be capable of altering auxin physiology [12], whether this is a direct or indirect effect remains to be determined.

Table 1

<table>
<thead>
<tr>
<th>T3E</th>
<th>Species</th>
<th>Activity</th>
<th>Target</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>AvrB</td>
<td><em>P. syringae</em> pv. glycinearace 0</td>
<td>Induces phosphorylation</td>
<td>RIN4/RAR1</td>
<td>[50,52]</td>
</tr>
<tr>
<td>AvrB3</td>
<td>X. campestris pv. vesicatoria race 1</td>
<td>Transcription activator-like</td>
<td>upa20/Bs3</td>
<td>[35**,36**]</td>
</tr>
<tr>
<td>AvrPphB</td>
<td><em>P. syringae</em> pv. phaseolicola race 3</td>
<td>Cysteine protease</td>
<td>PBS1</td>
<td>[14]</td>
</tr>
<tr>
<td>AvrPtoB</td>
<td><em>P. syringae</em> pv. tomato JL1065</td>
<td>Kinase inhibitor</td>
<td>Pto/ER/FLS2</td>
<td>[47**,49**]</td>
</tr>
<tr>
<td>AvrPtoB</td>
<td><em>P. syringae</em> pv. tomato DC3000</td>
<td>E3 ubiquitin ligase</td>
<td>Fen</td>
<td>[29,31**,32]</td>
</tr>
<tr>
<td>AvrRpm1</td>
<td><em>P. syringae</em> pv. glycinea race 0</td>
<td>Induces phosphorylation</td>
<td>RIN4</td>
<td>[48]</td>
</tr>
<tr>
<td>AvrRpt2</td>
<td><em>P. syringae</em> pv. tomato T1</td>
<td>Cysteine protease</td>
<td>RIN4</td>
<td>[18,19]</td>
</tr>
<tr>
<td>AvrXa27</td>
<td>X. oryzae pv. oryzae PXO99A</td>
<td>Transcription activator-like</td>
<td>Xa27</td>
<td>[38]</td>
</tr>
<tr>
<td>AvrXv4</td>
<td>X. campestris pv. vesicatoria T3</td>
<td>DeSUMOylating cysteine protease</td>
<td>Unknown</td>
<td>[24]</td>
</tr>
<tr>
<td>GALA</td>
<td>R. solanacearum GM1000</td>
<td>F-box and LRR domains</td>
<td>ASK(s)</td>
<td>[28*]</td>
</tr>
<tr>
<td>HopAl1</td>
<td><em>P. syringae</em> pv. tomato DC3000</td>
<td>Phosphothreonine lyase</td>
<td>MPK3/MPK6</td>
<td>[42**]</td>
</tr>
<tr>
<td>HopAO1</td>
<td><em>P. syringae</em> pv. tomato DC3000</td>
<td>Protein tyrosine phosphatase</td>
<td>Unknown</td>
<td>[44,45]</td>
</tr>
<tr>
<td>HopI1</td>
<td><em>P. syringae</em> pv. maculicola ES4326</td>
<td>J-domain protein (possible</td>
<td>AtMIN7 and others</td>
<td>[34**]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hsp70 cochaperone)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HopM1</td>
<td><em>P. syringae</em> pv. tomato DC3000</td>
<td>Unknown</td>
<td>GRP7 and other RNA-binding proteins</td>
<td>[41**]</td>
</tr>
<tr>
<td>HopU1</td>
<td><em>P. syringae</em> pv. tomato DC3000</td>
<td>Mono-ADP-ribosyltransferase</td>
<td>Unknown</td>
<td>[40]</td>
</tr>
<tr>
<td>HsvB</td>
<td>Pantoea agglomerans pv. betae 4188</td>
<td>Transcriptional activator-like</td>
<td>Unknown</td>
<td>[40]</td>
</tr>
<tr>
<td>HsvG</td>
<td>Pantoea agglomerans pv. gypsophilae 824-1</td>
<td>Transcriptional activator-like</td>
<td>Unknown</td>
<td></td>
</tr>
<tr>
<td>PthXo1</td>
<td>X. oryzae pv. oryzae PXO99A</td>
<td>Transcriptional activator-like</td>
<td>Os8N3</td>
<td>[39]</td>
</tr>
<tr>
<td>PthXo6/7</td>
<td>X. oryzae pv. oryzae PXO99A</td>
<td>Transcriptional activator-like</td>
<td>OsTFX1 OsTFIIAγ1</td>
<td>[37*]</td>
</tr>
<tr>
<td>XopD</td>
<td>X. campesstris pv. vesicatoria 85-10</td>
<td>DeSUMOylating cysteine protease</td>
<td>Unknown</td>
<td>[22]</td>
</tr>
</tbody>
</table>
A T3E from *P. syringae*, AvrPtoB (also known as HopAB2), contains a C-terminal E3 ligase domain [29] that ubiquitinates the tomato protein kinase Fen (originally identified as a cause of sensitivity to the pesticide fenthion [30]) leading to the degradation of Fen by the 26S proteasome [31**]. However, ETI occurs when the R protein Prf recognizes the interaction of Fen with AvrPtoB truncations that lack the E3 ligase domain. Thus, AvrPtoB has apparently evolved to evade ETI by acquiring an E3 ligase domain that marks Fen for degradation [31**]. Interestingly, some plants seem to have countered AvrPtoB’s E3 ligase activity by evolving the Pto kinase, which cannot be ubiquitinated by AvrPtoB and induces Prf-mediated ETI. AvrPtoB appears to have multiple plant targets since it also suppresses PTI-induced MAPK pathways in *Arabidopsis* [32]. It will be important to determine whether Fen and Pto are virulence targets or virulence decoys — if they are decoys evolved to ‘trick’ T3Es it would be tempting to speculate that, in a manner similar to AvrPto described below, PAMP receptor kinases may be the ‘real’ virulence targets of AvrPtoB.

A final example of a T3E that alters protein turnover is HopM1 (formerly HopPtoM) from *P. syringae* [33]. It is present in the endomembrane fraction of plant cells where it enhances AtMIN7 degradation by targeting it to the 26S proteasome [34**]. AtMIN7 is an adenosine diphosphate ribosylation factor-guanine nucleotide exchange factor (ARF-GEF). HopM1 may target AtMIN7 for degradation in order to disrupt vesicle transport as ARF-GEFs have been implicated in vesicle trafficking. Vesicle transport is necessary for the export of defence compounds to the cell wall and apoplast, so its disruption would impair cell-wall-based defences [34**]. HopM1 has no obvious similarity to known components of the host ubiquitination machinery, so exactly how it targets AtMIN7 to the proteasome remains to be established.

**Targeting host transcription or RNA stability**

T3Es can also modify protein levels by altering host transcription, which can lead to increased host susceptibility. T3Es that target host transcription include AvrBs3, PthXo6, and PthXo7 from *Xanthomonas* spp., and HsvG and HsvB from *Pantoea agglomerans*. AvrBs3 alters transcription by binding to the promoter region of the pepper transcription factor *upa20* and increasing its expression. This upregulation of *upa20* is partly responsible for the hypertrophy of mesophyll cells seen when AvrBs3 is overexpressed in plants [35**]. In resistant plants AvrBs3 also binds to the promoter and activates the transcription of its cognate R gene, Bss, leading to ETI [36**]. The encoded product of Bss is a flavin-dependent monoxygenase rather than an NB-LRR protein and it causes ETI by an unknown mechanism [36**]. The case of AvrBs3 recognition could be considered as a variation of the guard hypothesis, in that instead of guarding a protein the R gene is guarding a DNA sequence. This could work by the promoter of Bss acting as a decoy target for AvrBs3 by mimicking the promoter of *upa20*, which is apparently a ‘real’ virulence target.

AvrBs3 belongs to a family of transcription activator-like T3Es. Two members of this family, PthXo6 and PthXo7 increase the expression of the rice genes *OsTFX1* (a bZIP transcription factor) and *OsTFIHA1* (a small subunit of the transcription factor IIA), respectively [37]. Loss of virulence in *PthXo6* mutants can be rescued by overexpression of *OsTFX1* but it is not yet known if *PthXo6* directly binds to the promoter of *OsTFX1* [36**]. Two other host genes *Xa27* [38] and *Os8N3* [39] are specifically upregulated by the T3Es AvrXa27 and PthXo1, respectively. Rice plants silenced for *Os8N3* RNA result in plants that are less susceptible to *X. oryzae pv. oryzae* supporting the idea that *Os8N3* is a susceptibility gene [39]. Although less established than with the *Xanthomonas* T3Es similar activities are emerging for the *P. agglomerans* T3Es HsvG and HsvB [40]. Collectively, these examples suggest that the use of T3Es to upregulate expression of genes such as transcription factors that coregulate many genes is a common virulence strategy employed by bacterial pathogens. These T3E-induced proteins probably represent susceptibility factors that make the plant more favorable.
to bacterial colonization. Whether they cause the suppression of PTI or modify the cellular environment to benefit the pathogen remains to be determined.

The *P. syringae pv. tomato* HopU1 (formerly HopPtoS2) T3E targets RNA metabolism rather than mRNA production. HopU1 is a mono-ADP-ribosyltransferase (ADPRT) that ADP ribosylates several *Arabidopsis* RNA-binding proteins in vitro, including the glycine-rich RNA-binding protein GRP7 [41**]. Furthermore, this ADPRT activity is necessary for HopU1's ability to suppress PTI and ETI, and *Arabidopsis* plants lacking GRP7 are more susceptible to *P. syringae* [41**]. ADP-RTs are well-characterized toxins of animal pathogens but not previously shown to be associated with RNA-binding proteins. The ADP-ribosylation of RNA-binding proteins by HopU1 possibly decreases their ability to bind, stabilize or process RNA [41**]. It is not known how this alteration of RNA-binding leads to the suppression of innate immunity or if it alters specific or general RNA metabolism. Nevertheless, the post-transcriptional control of RNA by T3Es represents an exciting new pathogenic strategy to suppress innate immunity.

**Phosphorylation or dephosphorylation of host proteins**

PTI and ETI use kinase-based signaling pathways, perhaps most prominently MAPK pathways. These kinases represent ideal T3E targets as multiple signal transduction pathways often converge at them and disabling them results in the suppression of many downstream responses. As these pathways work by sequentially phosphorylating downstream components, modifying their phosphorylation states inhibits them preventing activation of host defences.

Two T3Es that seem to use this strategy are HopAII and HopAO1 (formerly HopPmaI2). HopAII from *P. syringae* has phosphothreonine lyase activity and irreversibly inactivates MAPKs [42**]. HopAII binds to animal MAPKs [42**], and MPK3 and MPK6 of *Arabidopsis* that are involved in defence responses to *P. syringae* [43]. HopAII’s broad specificity leaves open the possibility that its *in vivo* targets may include other MAPKs.

HopAO1 is a protein tyrosine phosphatase that contributes to virulence of *P. syringae* by suppressing PTI and ETI [44–46]. Because MAPKs are phosphorylated on tyrosine residues these are attractive putative targets for HopAO1. However, HopAO1 does not appear to act on MPK3 and MPK6 [46]. Therefore, its direct target(s) remain(s) to be identified.

A slightly different strategy for modifying phosphorylation is used by the *P. syringae* T3E AvrPto, which interacts with the PAMP receptor kinases FLS2 and EFR, inhibiting their ability to autophosphorylate and activate MAPK-signaling cascades [47**]. Similarly to AvrPtoB discussed above, AvrPto induces Pto-Prf-mediated ETI [48]. AvrPto inhibits the kinase activity of Pto [49*]. However, its induction of Prf-mediated ETI appears to be independent of this inhibition [49*]. Currently, the virulence target of AvrPto appears to be PAMP receptor kinases even though it also inhibits the kinase activity of Pto. This suggests that Pto may be an R-protein-guarded decoy and that other T3Es that interact with it, such as AvrPtoB, may also target PAMP receptor kinases.

**Clues about T3E activities on the basis of interactions with host proteins**

Host proteins that interact with T3Es have been identified through a variety of methods. Sometimes these interactors have provided clues to the T3E function. In other cases the protein–protein interaction itself is vital to the activity of the T3E by either stabilizing or destabilizing host protein complexes. This class of T3Es includes AvrB, AvrRpm1, and HopI1 from *P. syringae*. In pioneering research, AvrB and AvrRpm1 were found to interact with RIN4 leading to its hyperphosphorylation, which apparently allows it to be recognized by the R protein RPM1 inducing ETI [50,51]. AvrB has also been found to interact *in vivo* with RAR1 [52]. Moreover, RAR1 was identified in a genetic screen for *Arabidopsis* mutants insensitive to AvrB [52]. RAR1 is an attractive candidate target for an AvrB target because, together with SGT1 and HSP90, it stabilizes R proteins and, therefore, is a primary component of ETI.

Protein–protein interactions also appear to be important for HopI1 (formerly HopPmaI1) a chloroplast-targeted T3E with a potential J domain [53]. J domains are often found in the cochaperones of Hsp70, which direct proteins to Hsp70 and activate the ATPase activity and protein-folding ability of Hsp70 [54]. The J domain of HopI1 is necessary for its virulence activity and over-expression of HopI1 in *Arabidopsis* causes thylakoid remodeling and the suppression of SA accumulation [55]. However, how the possible Hsp70 cochaperone activity of HopI1 leads to these outcomes remains to be established. The targets and activities of the T3Es discussed here are summarized in Figure 1 and Table 1.

**Concluding remarks**

It is clear that though much remains to be understood about how T3Es interfere with plant innate immunity several underlying themes are becoming apparent. The first is that the guard hypothesis probably accurately describes the perception of T3E activities inside plant cells. The open question now seems to be whether the guarded T3E targets identified such as Pto or RIN4 represent real virulence targets or decoys. If they indeed represent decoys, this information could be exploited to identify their actual targets using similar logic employed
for AvrPto’s targets (i.e. PAMP receptor kinases). In the fascinating coevolution that has occurred between bacterial pathogens and plants it seems clear that PTI evolved before ETI. Did the evolution of PTI suppression provide the selection pressure necessary for ETI evolution? If so, it seems plausible that other R proteins may be guarding decoys of PAMP receptor kinase complexes.

A second emerging theme is that T3Es often have multiple targets many of which alter different aspects of innate immunity. These targets seem to function at every possible level of PTI and ETI pathways including the receptors themselves, downstream signal transduction pathways, and transcriptional and post-transcriptional responses. In this manner T3Es have apparently evolved to have the highest chance of suppressing sufficient innate immune responses to allow for the success of the bacterial pathogen. T3Es probably coevolved to target a particularly important node of regulation or component of innate immunity. Because T3Es are targeting the innate immune response at seemingly every level, studying T3E targets promises to identify important unknown components of innate immunity and represent exciting tools for exploring plant biology.

Acknowledgements
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References and recommended reading
Papers of particular interest, published within the period of review, have been highlighted as:

• of special interest
•• of outstanding interest


This study showed that T3Es from *Ralstonia solanacearum* called GALA proteins contain F-box domains and interact with Arabidopsis Skp1-like proteins that are part of E3 ligases involved in protein ubiquitination and turnover. A *R. solanacearum* mutant deficient in all seven GALAs lost its ability to cause disease on *Arabidopsis*.


The authors discovered that the *Pseudomonas syringae* T3E HopM1 causes the ubiquitination and degradation of ATMIN7 via 26S proteasome. ATMIN7 encodes a member of the adenosine diphosphate (ADP)-ribosylation factor (ARF) guanine nucleotide exchange factor (GEF) protein family that functions in vesicle trafficking. This suggests HopM1 promotes the ubiquitination of *P. syringae* by interfering with the host’s vesicle trafficking.


This paper reported that the T3E AvrBs3 from *Xanthomonas campestris* binds to the promoter and induces the expression of *upa20*, a bHLH transcription factor and master regulator of cell enlargement, to cause hypertrophy in mesophyll cells.

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37. Sugio A, Yang B, Zhu T, White FF: Two type III effector genes of *Xanthomonas oryzae pv. oryzae* control the induction of the host genes OsTFL1a and OsTFL1 in *Xanthomonas oryzae* transcontinentaly activates OsTFX1 and OsTFX1α, respectively. OsTFX1, when ectopically expressed in the host, substitutes for the role of ptoX06 in virulence, acting as a host susceptible gene.


41. Fu ZQ, Guo M, Jeong B-J, Tian F, Elthon TE, Cerny RL, Stigler D, Alfano JR: A type III effector ADP-ribosylates RNA-binding proteins and quells plant immunity. Nature 2007, 447:284-289. This study identified a T3E from *Pseudomonas syringae*, HopU1, as a mono ADP-ribosyltransferase (ADP-RT). This class of enzyme has never been reported in plant pathogens or plants. HopU1 suppresses the outputs of plant innate immunity dependent on its ADP-RT catalytic site. Using a proteomic approach, the authors identified HopU1 substrates to be glycine-rich RNA-binding proteins, which are new substrates of ADP-RT. More importantly, this paper suggests a novel strategy employed by a bacterial pathogen where the ADP-ribosylation of plant RNA binding proteins results in posttranscriptional inhibition of host innate immunity.


The *Pseudomonas syringae* T3E AvrPto was found to interact with the PRRs FLS2 and EFR and inhibit their kinase activities to block plant innate immunity. In the resistant host, Pto competes with FLS2 for AvrPto binding, and the binding of Pto with AvrPto triggers a Prf-dependent defence.


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