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Review

The roles of plant phenolics in defence and communication during *Agrobacterium* and *Rhizobium* infection

AMITA BHATTACHARYA[1,†], PRIYANKA SOOD[1,†] AND VITALY CITOVSKY[2,*]

[1]*Institute of Himalayan Bioresource Technology (Council of Scientific and Industrial Research), Palampur-176061, Himachal Pradesh, India*
[2]*Department of Biochemistry and Cell Biology, State University of New York, Stony Brook, NY 11794-5215, USA*

SUMMARY

Phenolics are aromatic benzene ring compounds with one or more hydroxyl groups produced by plants mainly for protection against stress. The functions of phenolic compounds in plant physiology and interactions with biotic and abiotic environments are difficult to overestimate. Phenolics play important roles in plant development, particularly in lignin and pigment biosynthesis. They also provide structural integrity and scaffolding support to plants. Importantly, phenolic phytoalexins, secreted by wounded or otherwise perturbed plants, repel or kill many microorganisms, and some pathogens can counteract or nullify these defences or even subvert them to their own advantage. In this review, we discuss the roles of phenolics in the interactions of plants with *Agrobacterium* and *Rhizobium*.

INTRODUCTION

Phenolics are one of the most ubiquitous groups of secondary metabolites found throughout the plant kingdom (Boudet, 2007; Harborne, 1980). They encompass a very large and diverse group of aromatic compounds characterized by a benzene ring (C_6) and one or more hydroxyl groups. Generally, the classification of phenolics is based on the number of carbon atoms present in the molecule (Harborne and Simmonds, 1964).

Phenolics are formed by three different biosynthetic pathways: (i) the shikimate/chorizmate or succinylbenzoate pathway, which produces the phenyl propanoid derivatives (C_6–C_3); (ii) the acetate/malonate or polyketide pathway, which produces the side-chain-elongated phenyl propanoids, including the large group of flavonoids (C_6–C_3–C_6) and some quinones; and (iii) the acetate/mevalonate pathway, which produces the aromatic terpenoids, mostly monoterpenes, by dehydrogenation reactions (for more details on these metabolic pathways, see www.plantcyc.org). Here, we focus on the classes of phenolics that are involved in interactions of plants with microbes, *Agrobacterium* and *Rhizobium*, belonging to the Rhizobiaceae family.

*Correspondence: Email: vitaly.citovsky@stonybrook.edu
†Equal authorship.

PLANTS SYNTHESIZE PHENOLICS IN RESPONSE TO BIOTIC AND ABIOTIC STRESS

Phenolics are often produced and accumulated in the subepidermal layers of plant tissues exposed to stress and pathogen attack (Clé *et al*., 2008; Schmitz-Hoerner and Weissenbock, 2003). The concentration of a particular phenolic compound within a plant tissue is dependent on season and may also vary at different stages of growth and development (Lynn and Chang, 1990; Ozyigit *et al*., 2007; Thomas and Ravindra, 1999). Several internal and external factors, including trauma, wounding, drought and pathogen attack, affect the synthesis and accumulation of phenolics (Kefeli *et al*., 2003; Zapprometov, 1989). Furthermore, the biosynthesis of phenolics in chloroplasts and their accumulation in vacuoles are enhanced on exposure to light (Kefeli *et al*., 2003). Photoinhibition, as well as nutrient stresses, such as deficiencies in nitrogen, phosphate, potassium, sulphur, magnesium, boron and iron, also trigger the synthesis of phenylpropanoid compounds in some plant species (Dixon and Paiva, 1995). These may include members of the flavonoid biosynthetic pathway (Balasundram *et al*., 2006; Hollman and Katan, 1999).

PHENOLICS IN PLANT DEFENCE

Phenolics serve a dual function of both repelling and attracting different organisms in the plant's surroundings (Table 1). They act as protective agents, inhibitors, natural animal toxicants and pesticides against invading organisms, i.e. herbivores, nematodes, phytophagous insects, and fungal and bacterial pathogens (Dakora and Phillips, 1996; Lattanzio *et al*., 2006; Ravin *et al*., 1989). Simple phenolic acids, complex tannins and phenolic

Table 1 Examples of different types of phenolics and diverse functions that they perform for plants and bacteria.

	Examples	Functions
Phenolics	Coniferyl alcohol, sinapinic acid, cinnamic acid	*vir* gene inducers, determinants of scent and attractants of pollinators and symbiotic microbes in plants, etc.
	Hydroxybenzoate, hydroxycinnamates, 5-hydroxyanthraquinones	Allelochemicals for plant competition
	Umbelliferone, *p*-hydroxybenzoic acid, vanillyl alcohol, isoflavones	Chemoattractants in *Rhizobium*
	Sinapinic acid, syringic acid, ethylsyringamide, propylsyringamide, carbethoxyethylensyringamide, parahydroxybenzoate, ferulic acid	*vir* gene inducers in *Agrobacterium*
	Vanillyl alcohol, bromo acetosyringone	Inhibitors of *vir* gene induction in *Agrobacterium*
	Acetosyringone, α-hydroxyacetosyringone, *p*-hydroxybenzoate	Chemoattractants in *Agrobacterium* and *Rhizobium*, and *vir* gene inducers in *Agrobacterium*
	Salicylic acid	Quorum quencher in *Agrobacterium*
	Hydroquinones	Allelochemical for plant competition
	Coumarins, xanthones, anthocyanidins	Determinants of colour and attractants of pollinators in plants
	Caffeic acid	*vir* gene inducer in *Agrobacterium*
	3,4-Dihydroxybenzoic acid	Chemoattractant in *Agrobacterium* and *Rhizobium*
	Protocatechuic acid, β-resorcylic acid, protocatechuate, *p*-resorcylate, catechol	*vir* gene inducer in *Agrobacterium*
	Chlorogenic acid	Precursor for lignin and suberin synthesis in plants
	Lignin, tannins and suberins	Structural components of plant cells
	Catechins	Plant defence
	Flavonoids, flavonols, flavones, genistein, daidzein, *O*-acetyldaidzein, 6-*O*-malonylgenistin, 6-*O*-malonyl daidzin, glycitin, 6-*O*-malonylglycitin	*nod* gene inducers in *Rhizobium*
	Apigenin, naringenin, luteolin	Chemoattractants in *Agrobacterium* and *Rhizobium*, and *nod* gene inducers in *Rhizobium*
	Gallate, gallic acid, pyrogallic acid, syringic acid, kaempferol	*vir* gene inducers in *Agrobacterium*
	Flavanones, quercetin	*nod* gene inducers in *Rhizobium*
	Isoflavonoids	Chemoattractants and *nod* gene inducers in *Rhizobium*
	Cajanin, medicarpin, glyceoline, rotenone, coumestrol, phaseolin, phaseolinin, limonoids, tannins, flavonoids	Phytoalexins, phytoanticipins and nematicides in plant defence

resins on the plant surface deter birds by interacting with the gut microflora and diminishing their digestive ability. The scent and pigmentation conferred by low-molecular-weight phenylpropanol derivatives attract symbiotic microbes, pollinators and animals that disperse fruits (Ndakidemi and Dakora, 2003; Vit *et al*., 1997).

Phenolics have long been recognized as phyto-estrogens in animals (Adams, 1989) and as allelochemicals for competitive plants and weeds (Weir *et al*., 2004; Xuan *et al*., 2005). Mainly, the volatile terpenoids, the toxic water-soluble hydroquinones, hydroxybenzoates, hydroxycinnamates and the 5-hydroxynapthoquinones are widely effective allelochemicals.

A number of simple and complex phenolics accumulate in plant tissues and act as phytoalexins, phytoanticipins and nematicides against soil-borne pathogens and phytophagous insects (Akhtar and Malik, 2000; Lattanzio *et al*., 2006). Therefore, phenolic compounds have been proposed for some time to serve as useful alternatives to the chemical control of pathogens of agricultural crops (Langcake *et al*., 1981).

Most known effects of polyphenols on microbes are negative (Cushnie and Lamb, 2005; Ferrazzano *et al*., 2009; Taguri *et al*., 2006). Plants respond to pathogen attack by accumulating phytoalexins, such as hydroxycoumarins and hydroxycinnamate conjugates (Karou *et al*., 2005; Mert-Türk, 2002). The synthesis, release and accumulation of phenolics—in particular, salicylic acid (Boller and He, 2009; Koornneef and Pieterse, 2008; Lu, 2009; Tsuda *et al*., 2008)—are central to many defence strategies employed by plants against microbial invaders.

Phenolics are synthesized when plant pattern recognition receptors recognize potential pathogens (Newman *et al*., 2007; Ongena *et al*., 2007; Schuhegger *et al*., 2006; Tran *et al*., 2007) by conserved pathogen-associated molecular patterns (PAMPs), leading to PAMP-triggered immunity (Zipfel, 2008). As a result, the progress of the infection is restricted long before the pathogen gains complete hold of the plant (Bittel and Robatzek, 2007; Nicaise *et al*., 2009).

PHENOLICS—MOLECULES FOR CROSS-TALK IN THE RHIZOSPHERE

The plant rhizosphere is a dynamic ecosystem of different species, representing flora, fauna and microbes that interact with each other in a variety of complex reactions (Whipps, 2001). These interactions are mainly governed by a diverse array of phenolics exuded by the growing roots of plants, together with a host of other chemicals (Bais *et al*., 2006; Bertin *et al*., 2003; Dakora and Phillips, 2002). The functions performed by phenolics in the plant rhizosphere (Dakora, 2003) have been aptly termed the 'rhizosphere effect' (Hiltner, 1904). The root exudates generally include ions, free oxygen, water, enzymes, mucilage, a

number of carbon-containing primary and secondary metabolites and, most importantly, plant phenolics. The released phenolics differ from species to species and also with time, space and location. Their concentrations in the soil range from 2.1 to 4.4% in monocotyledonous plants and 0.1 to 0.6% in dicotyledonous plants (Hartley and Harris, 1981). Phenolics trigger redox reactions in soils and selectively influence the growth of soil microorganisms that colonize the rhizosphere. These then influence the hormonal balance, enzymatic activity, availability of phytonutrients and competition between neighbouring plants (Hättenschwiler and Vitousek, 2000; Kraus *et al*., 2003; Northup *et al*., 1998). As a result of this dynamic and ever-changing interaction, the structure and chemistry of the soil are altered significantly depending on the quantity and identity of phenolics released by different plant species. The composition of the microorganism species in different root locations is also persistently modified and shaped. Moreover, as phenolics move through the rhizosphere, they are bound by soil organic matter and metabolized by the bacterial flora of the soil (Kefeli *et al*., 2003).

Microorganisms break down phenolics into elements that contribute towards the mineralization of soil nitrogen and the formation of humus (Halvorson *et al*., 2009). The phenolics chelate metals and improve soil porosity, providing active absorption sites and increasing the mobility and bioavailability of elements, such as potassium, calcium, magnesium, copper, zinc, manganese, molybdenum, iron and boron, for plant roots (Seneviratne and Jayasinghearachchi, 2003). Some phenolic metabolites, such as *trans*-cinnamic acid, salicylic acid, coumarin, benzoic acid, parahydroxybenzoic acid and syringic acid, are phytotoxic. For example, the accumulation of phenolics in the soil can inhibit seed germination and seedling growth (Baleroni *et al*., 2000). This effect may be caused by interference with cell division and the normal functioning of cellular enzymes. Indeed, phenolics have been shown to inhibit phosphatase and prolyl aminopeptidase involved in seed germination (Madhan *et al*., 2009). In addition, phenolics affect the process of mineral uptake by the plants (Lodhi *et al*., 1987).

Many of the phenolic root exudates serve as chemotactic signals for a number of soil microorganisms that recognize them and move towards plant roots in the carbon-rich environment of the rhizosphere (Perret *et al*., 2000; Taylor and Grotewold, 2005). Based on the nature and type of root-derived chemicals, both positive and negative cross-talk pathways are initiated between roots, roots and insects, and roots and microbes. Different organisms are repelled from or attracted to the same chemical signal, which then elicits different responses in different recipients. One specific example is the isoflavones from soybean roots which serve as a chemoattractant for both the symbiotic *Bradyrhizobium japonicum* and the pathogenic *Phytopthora sojae* (Morris *et al*., 1998). The number and activity of soil microorganisms around the root increase as a result of root–microbe cross-talk (Bais *et al*., 2004). This subsequently leads to the colonization of roots. Generally, the zone of root elongation, just behind the tip, supports the growth of primary root colonizers that utilize the easily degradable sugars and organic acids. On the other hand, fungi and bacteria that are adapted to crowded, oligotrophic conditions inhabit the older root zones, comprising sloughed cells with lignified cellulose, hemicellulose and carbon deposits. Mature communities of fungi colonize relatively nutrient-rich environments provided by the newly emerging lateral roots and the secondary non-growing root tips. Colonization of vesicular–arbuscular mycorrhizal fungi in response to isoflavonoids from soybean roots leads to greater phosphorus acquisition for plant nutrition, improved water relations and, consequently, better plant growth (Bagayoko *et al*., 2000; Siqueira *et al*., 1991). During phosphate deficiency, plant roots also exude strigolactones. These apocarotenoid molecules are detected as host-derived signalling compounds at the presymbiotic stage of beneficial fungal symbionts (Akiyama, 2007; Akiyama *et al*., 2005). Unlike various flavonoids that induce hyphal branching only in a limited number of hosts, strigolactones represent primary signalling factors for hyphal branching and growth of arbuscular mycorrhizal fungi (Steinkellner *et al*., 2007).

Other positive effects of root colonizers include the symbiotic associations with epiphytes and mycorrhizal fungi, the fixation of atmospheric nitrogen by different classes of proteobacteria (Moulin *et al*., 2001), increased biotic and abiotic stress tolerance imparted by the presence of endophytic microbes (Schardl *et al*., 2004), and several direct and indirect advantages caused by various plant growth-promoting rhizobacteria (Gray and Smith, 2005). Some colonizing bacteria also interact with plants to produce protective biofilms or antibiotics, functioning as effective biocontrol against potential pathogens (Bais *et al*., 2004).

Although some types of colonization may lead to associations with symbiotic microorganisms, others result in plant infection by soil-borne pathogens. The symbiotic *Rhizobium* and the pathogenic *Agrobacterium* spp. (except *A. radiobacter*) of the Rhizobiaceae family are examples of plant colonizers with positive and negative effects on their hosts, respectively. Specifically, rhizobia are important for their nitrogen-fixing ability and endosymbiotic associations with leguminous plants, whereas most *Agrobacterium* species are phytopathogens. As members of the same family, these two bacteria exhibit similarities and differences in their basic mechanisms of symbiosis or infection, each enjoying a special ecological niche.

HOST PHENOLICS IN THE INFECTION CYCLES OF *AGROBACTERIUM* AND *RHIZOBIUM*

The type and concentration of phenolics in the surroundings govern the interactions of *Agrobacterium* and *Rhizobium* with their host plants (Table 2). These interactions may range from

Table 2 Summary of events in host–bacteria (Rhizobiaceae) interactions affected by phenolics.

Processes	Phenolics	Bacterial species	Bacterial factors
Chemotaxis	Acetosyringone, hydroxyacetosyringone, parahydroxybenzoic acid, 3,4-dihydroxybenzoic acid, vanillyl alcohol, etc.	*Agrobacterium tumefaciens*, *A. rhizogenes, A. vitis*	VirA
	Isoflavonoids, flavonoids, apigenin, luteolin, vanillyl alcohol, parahydroxybenzoic acid, 3,4-dihydroxybenzoic acid, acetosyringone, umbelliferone, naringenin, etc.	*Rhizobium meliloti, R. leguminosarum* bv. *phaseoli*, *viciae, trifolii*	NodD
Gene inducers	Acetosyringone, catechol, gallate, *p*-resorcylate, protocatechuate, parahydroxybenzoate, vanillin, α-hydroxyacetosyringone, ferulic acid, gallic acid, pyrogallic acid, protocatechuic acid, β-resorcylic acid, syringic acid, kaempferol, ethylsyringamide, propyl syringamide, carbethoxyethylensyringamide, sinapinic acid	*Agrobacterium* spp.	VirA and VirG
	Flavonoids, flavones, flavonols, vanillin, genistein, daidzein, *O*-acetyl daidzin, 6-*O*-malonyl genistein, 6-*O*-malonyl daidzin, glycitin, 6-*O*-malonyl glycitin, genistin, apigenin, naringenin, luteolin	*Rhizobium melitloti, R. leguminosarum* bv. *phaseoli*, *viciae, trifolii*, *Bradyrhzobium japonicum*, *Sinorhizobium meliloti*	NodD
Detoxification/mineralization	Phenolic *vir* gene inducers (high concentrations)	*Agrobacterium* spp.	VirH2
	Flavonoids, quercetin, daidzein, genistein, etc.	*Rhizobium* and *Bradyrhzobium* spp.	Enzymes that cleave C-ring
Quorum signalling	Negative influence of flavonoids	*Rhizobium* spp.	CinR/CinI, RhiI, RhiA, RhiB, RhiC, RhiR, RaiI, RaiR, TraI, TraB-I, BisR and TraR
	Indirect influence of phenolic *vir* gene inducers (via opines)	*Agrobacterium* spp.	TraI/R, products of *tra* and *repABC* operons
Quorum quenching	Flavonoids	*Rhizobium*	BisR
	Salicylic acid, phenolic *vir* gene inducers	*Agrobacterium* spp.	TraM, AttM, AttL, AttK

strong to weak to transient (Bais *et al*., 2006). Although plants exude different phenolic compounds that are toxic to most microorganisms, *Agrobacterium* and *Rhizobium* have evolved mechanisms to counteract, nullify and even utilize these defences for their own advantage (Figs 1 and 2) (Hartmann *et al*., 2009; Matilla *et al*., 2007). The most significant of these mechanisms that involve phenolics are: (i) chemotaxis; (ii) activation of the bacterial nodulation (*nod*) and virulence (*vir*) gene networks; (iii) xenobiotic detoxification; and (iv) quorum signalling (Fig. 2).

Chemotaxis

As in several other soil bacteria, phenolics play a pivotal role in the chemotactic responses of *Agrobacterium* and *Rhizobium* in their search for growth substrates and hosts. These serve as excellent models for signal transduction and plant–microbe interactions (Palmer *et al*., 2004; Samac and Graham, 2007). Diverse plant phenolic compounds with varying substitution patterns determine the chemotactic movement of *Agrobacterium* or *Rhizobium* across chemical gradients towards higher levels of potential nutrients and lower levels of inhibitors. For example, umbelliferone, vanillyl alcohol, *p*-hydroxybenzoic acid, 3,4-dihydroxybenzoic acid and acetosyringone evoke a strong chemotactic response in *Rhizobium leguminosarum* bv. *phaseoli*; *R. leguminosarum* bv. *trifolii* and *Sinorhizobium meliloti* are also chemoattracted by apigenin and luteolin (Brencic and Winans, 2005). In contrast, naringenin evokes only a weak to no chemotactic response in *R. leguminosarum* bv. *viciae* and *trifolii* (Zaat *et al*., 1987). It also suppresses the strong chemotaxis of *S. meliloti* by luteolin (Caetano-Anolles *et al*., 1988). Although acetosyringone and umbelliferone are inhibitors of *nod* gene inducers, *R. leguminosarum* exhibits an exaggerated chemotactic response to high concentrations of these compounds (Aguilar *et al*., 1988). Probably, the complex nature of the root exudates of different plants in the rhizosphere (Djordjevic *et al*., 1987) necessitates such negative regulation by some phenolics. For example, it might be required to prevent competing rhizobia from targeting the same host plant and also for creating a favourable ecological niche for each of the species. Preventing nodule initiation in the vicinity of clover root tips by umbelliferone is a good example of such negative rhizospheric interactions (Djordjevic *et al*., 1987).

A variety of phenolic compounds affect directly the *virA/G* genes on the Ti plasmid of different *Agrobacterium* species (Lee *et al*., 1996; McCullen and Binns, 2006; Sheng and Citovsky, 1996). These, in turn, influence chromosomal genes, such as the 8-kb chemotaxis operon, beginning with *orf1* (Harighi, 2009; Wright *et al*., 1998) and the 7205-bp putative operon involved in the formation of flagellar rods and associated proteins (Deakin *et al*., 1999; Shaw *et al*., 1991). Acetosyringone and hydroxyacetosyringone exuded from plant wounds are potent chemoattractants at very low concentrations, and they also act to induce the *vir* genes of *Agrobacterium* (Escobar and Dandekar, 2003). In

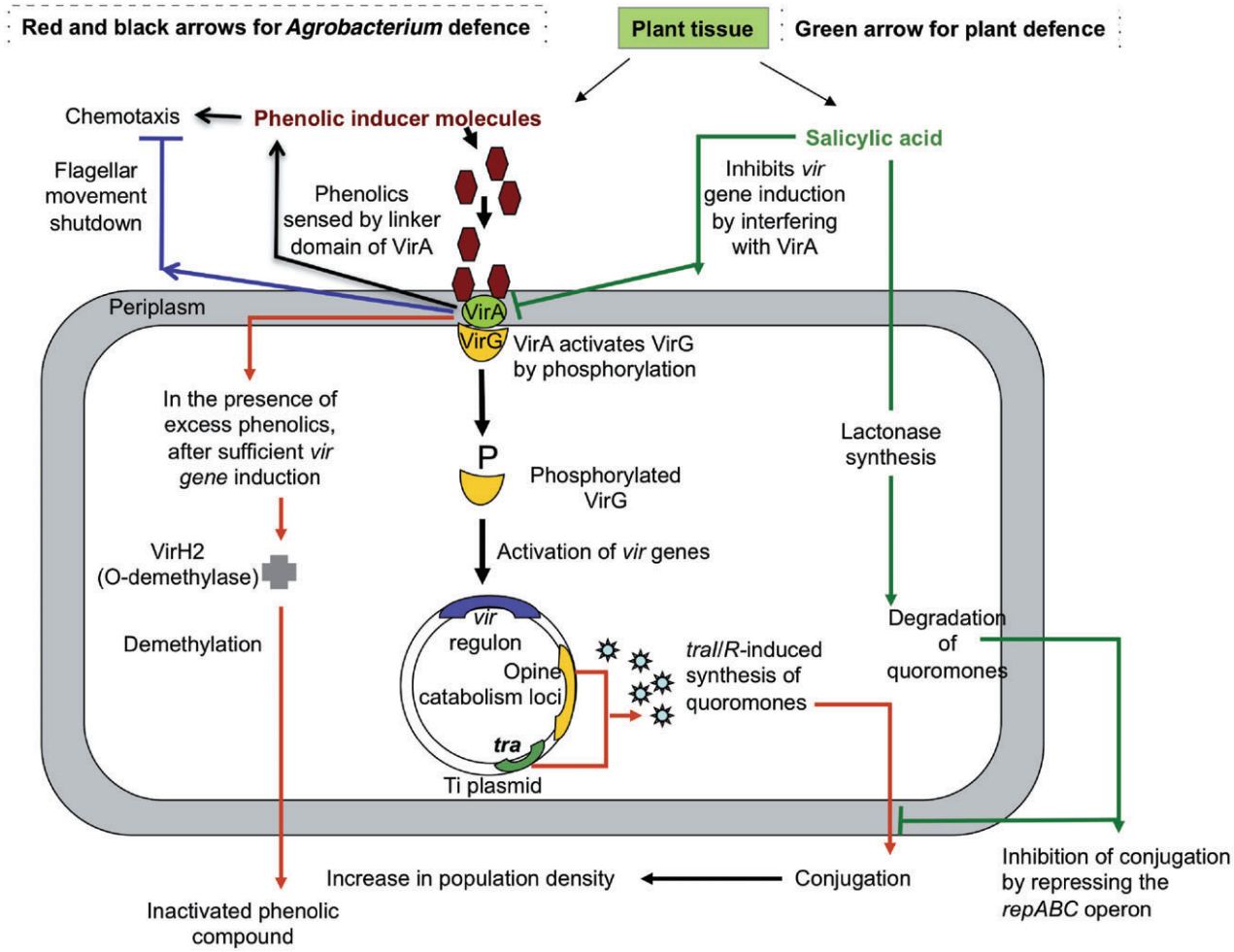

Fig. 1 Phenolics in the *Agrobacterium*–plant interaction. Black arrows indicate how *Agrobacterium* uses phenolics to initiate pathogenesis in host plants for opine synthesis and nutrition, and red arrows indicate how it inactivates the excess amounts of the same phenolics using its own specific *O*-demethylase system. Blue arrow indicates the involvement of the phenolic-sensing bacterial VirA protein in negative chemotaxis. Green arrows show how plants synthesize and use some phenolics, such as salicylic acid, to interfere with VirA and hence pathogenesis, and also to degrade the quoromones that give ecological advantage to *Agrobacterium* over other bacteria in competition for the opines synthesized by the infected plant.

addition to acetosyringone, a number of other phenolics and sugars are effective as chemoattractants (Brencic and Winans, 2005; Palmer *et al*., 2004; Peng *et al*., 1998). Even the non-*vir* gene-inducing vanillyl alcohol is a strong chemoattractant (Ashby *et al*., 1988). On the other hand, high concentrations of some polyphenols may have bacteriostatic or even bactericidal effects, and may block *Agrobacterium*'s access to plant wound sites by inhibiting its chemotactic movement.

Activation of the bacterial *nod* and *vir* gene networks

Microbial gene expression following chemotaxis is influenced by a diverse array of substituted plant phenols. These plant-derived signals are termed 'host recognition factors' or 'xenognosins' (Campbell *et al*., 2000). In both the symbiotic *Rhizobium* and pathogenic *Agrobacterium*, the same phenolics that act as chemoattractants may also regulate the expression of *nod* and *vir* genes, respectively (Djordjevic *et al*., 1987). For several decades, flavonoids were presumed to be the sole chemoattractants and inducers of *nod* gene expression in rhizobia (Cohen *et al*., 2001; Stougaard, 2000). Until the late 1980s, practically almost every study conducted on this subject revolved around the isoflavonoids from soybean (D'Arcy-Lameta and Jay, 1987). It was only in the late 1990s that the *nod* gene-inducing ability of other compounds, such as flavones and flavonols from broad beans, gained importance (Bekkara *et al*., 1998). With time, however, other organic molecules, mainly phenolics, were identified as potent inducers of *nodABC* genes, and the effect was dependent on the presence of a functional *nodD* gene (Perret *et al*., 2000;

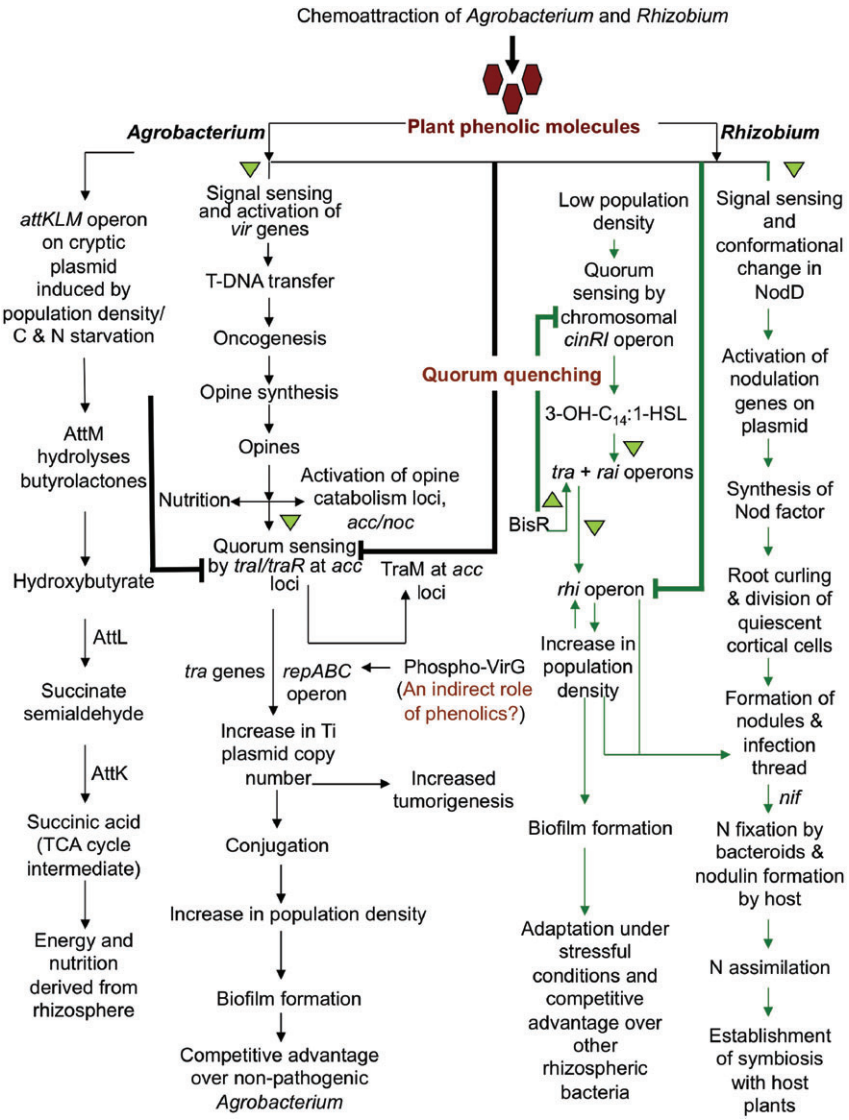

Fig. 2 The use of phenolics by *Agrobacterium* and *Rhizobium* for survival and infection of the host plant. Black arrows indicate how *Agrobacterium* uses phenolics to initiate a complex process of pathogenesis, culminating with opine synthesis. In addition to their nutritional value, opines help *Agrobacterium*'s competition with nonpathogenic bacteria, such as *A. radiobacter*, by increasing its population density and biofilm formation through quorum sensing. *Agrobacterium* also uses the *attKLM* operon to regulate its population density during times of nutritional starvation and to synthesize alternative sources of nutrients and energy by degrading γ-butyrolactones produced by other rhizospheric bacteria. Green arrows indicate the use of phenolics by *Rhizobium leguminosarum* bv. *viciae* for the induction of *nod* genes followed by the process of symbiosis. Under stress conditions, phenolics also regulate the increase in population density, biofilm formation and effective nodulation by repressing the quorum-sensing *rhi* operon. An increase in population density as a result of quorum sensing provides a competitive edge to rhizobia over other rhizospheric bacteria. Bold lines indicate the quorum-quenching mechanisms, whereas triangles represent the steps of activation. TCA, tricarboxylic acid.

Subramanian *et al*., 2006). The *nodD* genes from different *Rhizobium* species are optimally responsive to specific phenolics (Gagnon and Ibrahim, 1998; Peck *et al*., 2006). Although some phenolics influence *nod* genes positively, others may have a negative effect. For example, the *nod* genes of some strains of *Bradyrhizobium japonicum* are induced by daidzein, genistein and isoflavonoids from soybean, whereas the *nod* genes of *S. meliloti* are inhibited by the same phenolics and, instead, are induced by luteolin (Begum *et al*., 2001; Kosslak *et al*., 1987). Some phenolics may also perform a dual function of serving as both *nod* inducers and chemoattractants, whereas others may perform only one of the two functions. One such example is isoliquiritigenin (2′,4′,4-trihydroxychalcone), which is a strong *nod* gene inducer, but not a chemoattractant (Kape *et al*., 1992).

In addition to inducing the biosynthesis of Nod signals, flavonoids inhibit the transport of auxins at the site of rhizobial infection, once the rhizobia have entered the host cells (Brown *et al*., 2001). In *Arabidopsis*, two flavonoid-binding protein complexes, AtAPM and AtMDR, are believed to regulate the polar transport of auxins through the *PIN* gene homologues (Subramanian *et al*., 2007). In legumes, the inhibition of flavonoid-regulated auxin transport is critical for the formation of indeterminate nodules (Wasson *et al*., 2006). In contrast, the inhibition of auxin transport is not required for the formation of determinate nodules, which can develop even in the presence of very low levels of isoflavones (Grunewald *et al*., 2009; Subramanian *et al*., 2007).

In the case of *Agrobacterium*, a variety of phenolic compounds, one of the most potent of which is acetosyringone, are known to induce *vir* gene expression (Table 2) (Gelvin, 2009). These phenolics are detected by the VirA/VirG two-component sensor–transducer system, which then induces all *vir* loci that encode most components of the protein machinery for T-DNA transfer (Zupan *et al*., 2000). In addition to phenolics, VirA, with the help of a chromosomally encoded glucose/galactose-binding protein ChvE, senses sugar components of the cell wall released from wound sites of susceptible hosts (Ankenbauer and Nester, 1990; He *et al*., 2009). A conformational change that ensues as a result of the binding of ChvE to VirA allows the latter to interact even with poor *vir* gene inducers, such as 4-hydroxyacetophenone, *p*-coumaric acid and phenol (Peng *et al*., 1998).

A specific structure of phenolic molecules is essential for *vir* gene induction. The aromatic hydroxyl group, together with several other structural features, is absolutely essential. Particularly, the phenolics bearing an unsaturated lateral chain have comparatively higher *vir* gene-inducing ability (Joubert *et al*., 2002). The monomethoxy derivatives are more active than those that lack these methoxy substitutions on the phenol ring. The dimethoxy derivatives are invariably the most active of these three classes of phenolics. In addition, a chiral carbon at the centre of the phenolic molecule is critical for *vir* gene-inducing activity. The polarity or acidity created by the *para* position of the aromatic hydroxyl group bound to the hydrogen bond is associated with a higher induction potential (McCullen and Binns, 2006).

As the introduction of an amide group in syringic acid enhances its *vir* gene-inducing activity quite strongly, several new types of phenolic compounds, including three phenol amides, have been synthesized on the basis of syringic acid (Dye *et al*., 1997). Of these, the highest *vir* gene-inducing activity was observed with ethylsyringamide, followed by propylsyringamide, carbethoxyethylensyringamide and syringic acid itself. Recently, benzene rings with a hydroxyl group at position 4, methoxy group at position 3 and another methoxy group at position 5 in a phenolic molecule have been shown to increase the induction of the *vir* gene significantly (Brencic and Winans, 2005). In addition to phenolics, other compounds, such as D-glucose, D-galactose and D-xylose, are known to enhance *vir* gene induction (Wise *et al*., 2005).

Detoxification and biotransformation of xenobiotics into inactive and/or utilizable forms

Phenolics act as antimicrobial compounds because of their ability to disrupt nonspecifically the structural integrity of bacterial membranes and to inhibit specifically bacterial enzymes involved in electron transport (Hirsch *et al*., 2003). It is natural that bacteria would counteract or even nullify these toxic compounds. Many bacteria are endowed with the ability to degrade and utilize toxic phenols as a source of carbon (Dua *et al*., 2002; Lovely, 2003; Wackett, 2000; Wackett *et al*., 1987; Watanabe, 2001). Often the mechanisms for phenol detoxification are closely associated with those involved in establishing plant–bacteria interactions. For example, in the case of *Agrobacterium*, the *virH* (formerly *pinF*) locus that encodes factors for detoxification of harmful phenolics is itself located in the phenolic-inducible *vir* regulon of the Ti plasmid (Fig. 1). This regulation ensures that the metabolism or inactivation of phenolics does not commence until the *vir* regulon is induced (Kalogeraki and Winans, 1998; Sheng and Citovsky, 1996). One of these factors, VirH2, bears strong resemblance to the xenobiotic detoxification enzymes, i.e. the cytochrome P450-dependent mixed-function oxidases (Brencic *et al*., 2004). VirH2 quenches or detoxifies wound-released plant phenolics by catabolism, mineralization or conversion into sources of carbon, energy or nutrients (Fig. 1). The detoxification of xenobiotics generally involves the modification of xenobiotic compounds by transfer of polar or reactive groups (Guengerich, 2001). The modified compound is then conjugated with a charged species, such as glutathione sulphate (GSH), and metabolized by glutathione *S*-transferases (GSTs), γ-glutamyl transpeptidases and dipeptidases into easily removable acetylcysteine conjugates or mercapturic acids (Boyland and Chasseaud, 1969). These are finally removed from cells by a family of ATP-binding cassette transporters (Konig *et al*., 1999). Both cytochrome P450-dependent oxidases, such as VirH2, and a new class of GSTs exemplified by AtGST1 are present in *Agrobacterium* species (Kosloff *et al*., 2006).

What are the specific detoxification reactions catalysed by VirH2? *O*-Demethylase encoded by *virH2* (Brencic *et al*., 2004; Kalogeraki *et al*., 2000) is a specific detoxification enzyme that demethylates ferulic acid into caffeic acid (Fig. 3). VirH2-dependent mineralization and *O*-demethylation of 16 other *vir*-inducing methoxyl group-containing phenolics have also been detected (Brencic *et al*., 2004). Furthermore, VirH2 mediates the oxidation of vanillyl alcohol and vanillin into vanillate, which is then mineralized into protocatechuate via the β-ketoadipate pathway (Shaw *et al*., 2006).

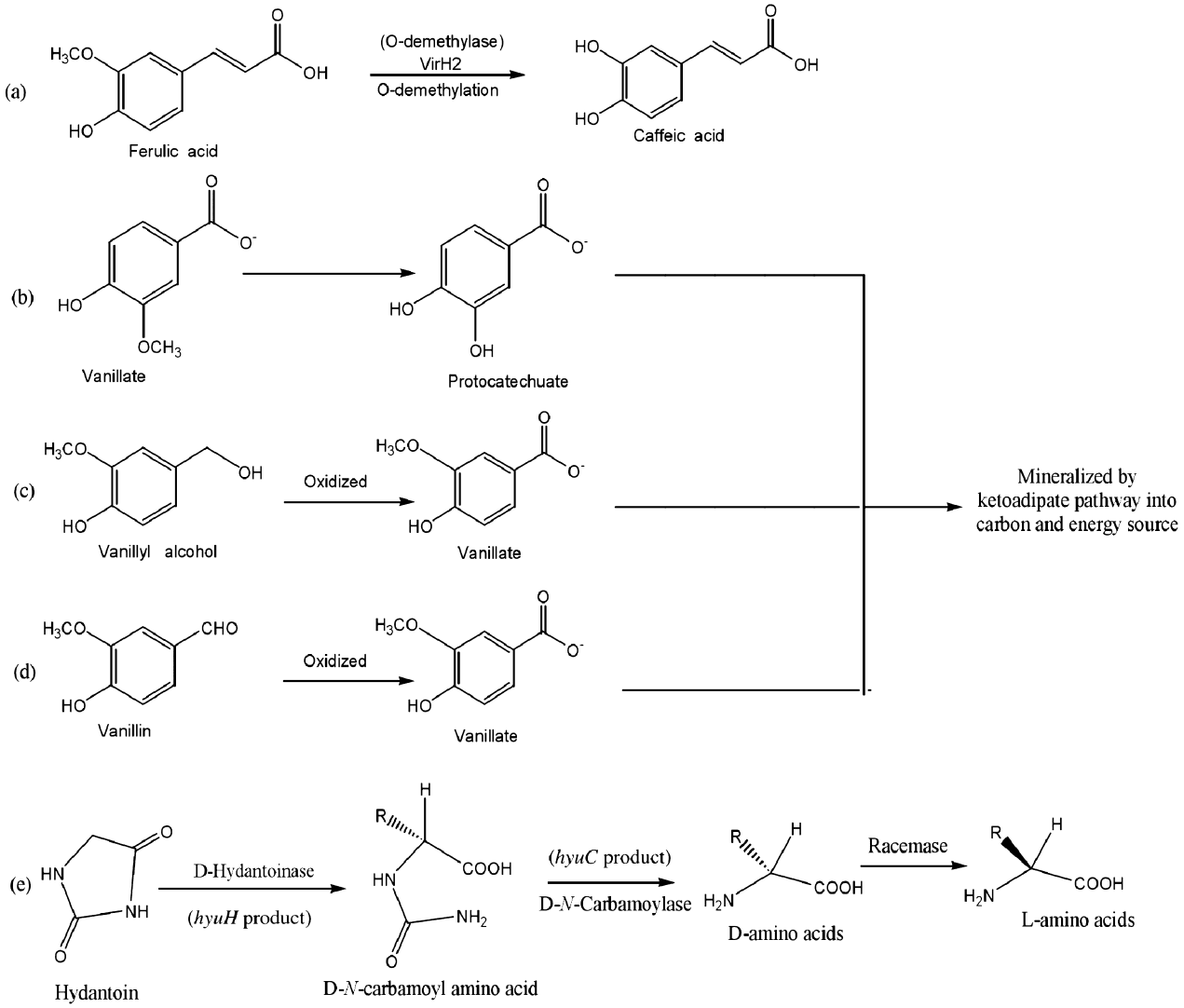

Fig. 3 The xenobiotic detoxification reactions by *Agrobacterium*. (a) Conversion of the excess and toxic amounts of the *vir*-inducing ferulic acid into its relatively less toxic caffeic acid by the VirH2 demethylase. (b–d) Oxidation and mineralization of vanillate, vanillyl alcohol and vanillin into sources of carbon and energy. (e) Biotransformation of hydantoin by hydantoinase into D-*N*-carbamoyl amino acid and further into D-amino acids by D-*N*-carbamoylase. The D-amino acid is finally converted into its easily utilizable L-form of amino acid by racemase.

The detoxification of diverse xenobiotics is common in different species of pathogenic as well as nonpathogenic strains of *Agrobacterium*. Detoxification of hydantoins or glycolylurea is an important example in which the phenolic-inducible *hyuH* gene encodes a hydantoinase that cleaves the amide bond at the second position of the hydantoin ring to produce D-*N*-carbamoylamino acid. The *N*-carbamoylase enzyme encoded by the *hyuC* gene then converts the D-*N*-carbamoylamino acid into its corresponding D-amino acid (Fig. 3) (Burton and Dorrington, 2004). The racemase enzyme present in some *Agrobacterium* strains can also convert the 5-monosubstituted hydantoin from the L- to its D-form (Martinez-Rodriguez *et al*., 2004). Although both *hyuH* and *hyuC* genes have been cloned and characterized from most strains of *Agrobacterium* (Chao *et al*., 2000; Jiang *et al*., 2007; Jiwaji *et al*., 2009; Martinez-Gomez *et al*., 2007), some strains contain only one of these genes (Hils *et al*., 2001). The activities of these detoxifying enzymes are tightly regulated by growth conditions and are particularly sensitive to nitrogen catabolism (Hartley *et al*., 2001). Although the molecular basis of *hyu* gene regulation is unknown, cellular glutamine levels regulate the reversible post-translational modification of the hydantoinase, representing the pathway by which many Gram-negative bacteria assimilate nitrogen under high-nitrogen conditions (Jiwaji and Dorrington, 2009).

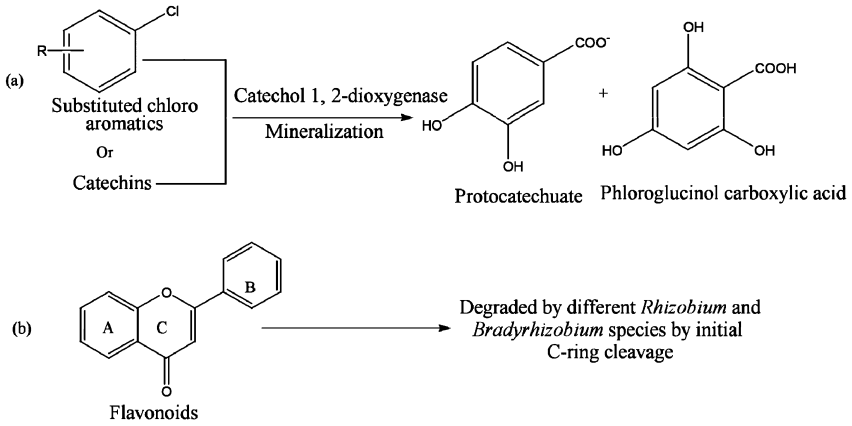

Fig. 4 Reactions showing how rhizospheric phenolics are utilized as sources of carbon and energy by *Rhizobium* spp. for saprophytic and symbiotic survival in soil and host. (a) Mineralization of catechins and substituted chloro-aromatics by catechol-1,2-dioxygenase into phloroglucinolcarboxylic acid and protocatechuate. (b) Degradation of flavonoids by C-ring cleavage into utilizable forms.

Both *Agrobacterium* and *Rhizobium* also possess hydrophobic efflux pumps for actively excluding toxic levels of flavonoids (Burse *et al*., 2004; González-Pasayo and Martínez-Romero, 2000; Palumbo *et al*., 1998; Parniske *et al*., 1991). Such flavonoid-inducible efflux pumps confer enhanced resistance to phaseolin in *R. etli* (González-Pasayo and Martínez-Romero, 2000) and pump out isoflavonoids, such as medicarpin, coumestrol and formononetin in *A. tumefaciens* (Palumbo *et al*., 1998). It has been proposed that these pumps function by capturing hydrophobic or amphiphilic substrate molecules from the cytoplasmic membrane using a transporter protein (Bolhuis *et al*., 1997).

Although not threatened by plant-derived toxic phenolics, rhizobia possess mechanisms for detoxifying various xenobiotics, mainly nonphenolic compounds, present in their rhizosphere. For example, four different *Rhizobium* species rapidly degrade glyphosate from their environment (Parker *et al*., 1999). Interestingly, *Agrobacterium* can also degrade glyphosate and other broad-spectrum phosphonates and utilize them as a source of phosphorus (Liu *et al*., 1991). In most species of rhizobia and bradyrhizobia, phenolics are converted into forms that can be used as sources of carbon, nitrogen or energy (Vela *et al*., 2002). This confers selective advantage on the bacteria for their saprophytic and symbiotic survival in soil and host. The mineralization of catechins and substituted chloro-aromatics by catechol-1, 2-dioxygenase into phloroglucinolcarboxylic acid and protocatechuate are good examples of such relationships (Fig. 4) (Latha and Mahadevan, 1997). Other phenolics, such as the flavonoids quercetin, daidzen and genistein, are also degraded by different *Rhizobium* and *Bradyrhizobium* species via the initial C-ring cleavage (Fig. 4) (Brencic and Winans, 2005; Rao and Cooper, 1994). Even xenobiotic degradation of an industrial chemical, polychlorinated biphenyl (PCB), can occur during the catabolism of the flavonoids naringin and apigenin (Dzantor, 2007; Fletcher and Hedge, 1995). Such biodegradation of xenobiotic compounds presents attractive opportunities for rhizo-engineering or rhizosphere manipulations for soil bioremediation.

Quorum signalling for attaining infection

An important mechanism by which members of Rhizobiaceae monitor their environment is quorum sensing (Bjarnsholt and Givskov, 2007; Gonzalez and Marketon, 2003). The production, release and sensing of homoserine lactones (HSLs) or their acylated forms (AHL) are important for quorum sensing (Parsek and Greenberg, 2000; Steidle *et al*., 2001). Quorum sensing allows bacterial cell–cell communication and promotes an advantageous lifestyle for both the survival and maintenance of pathogenic or symbiotic relationships within a range of environmental niches (Joint *et al*., 2002). In quorum sensing, cell density-dependent regulation of gene expression enables bacteria to coordinate certain adaptive processes that cannot be performed by an individual microbe.

Quorum sensing helps rhizobia to synchronize themselves to phenolic signals on a population-wide scale and to function as multicellular organisms for successful symbiosis (Fig. 2). The *Rhizobium* quorum sensing enhances nodulation efficiency, symbiosome development, exopolysaccharide production, nitrogen fixation and adaptation to stress (Danino *et al*., 2003; Gonzalez and Marketon, 2003). The extensively studied *R. leguminosarum* bv. *viciae* has four quorum sensing operons. Of these, only the *cin* operon, comprising *cinrli*, is located on the chromosome (Lithgow *et al*., 2000) and regulates the synthesis of the long-chain ($C_{14.1}$) quoromones, i.e. AHL. Of the four operons involved in quorum sensing, these AHLs induce only the *rai* and *tra* operons located on two different plasmids. The plasmid pIJ9001 contains the *raii/rair* operon, which encodes the synthesis of short-chain AHLs and 3-OH-C8-HSL, whose functions are unknown. The *raii/rair* operon itself is regulated by AHLs produced by the *cin* operon and by the *tra* operon of the pRL1J1 plasmid (Wisniewski-Dye and Downie, 2002). The *tra* operon

encodes the synthesis of 3-oxo-C8-HSL and influences the expression of *rai*, *rhi* and *tra* operons (Wilkinson *et al*., 2002). It also transcribes the repressor of the *cin* operon, i.e. the BisR regulator. The *rhii* genes of the *rhiilr/abc* operon located on the symbiotic plasmid pRL1J1 encode short-chain (C_6–C_8) AHLs and induce their own *rhi* operon (Sanchez-Contreras *et al*., 2007). The expression of the *rhiilr/abc* operons is repressed by flavonoids in *R. leguminosarum* (Economou *et al*., 1989), but, beyond this observation, little is known about the role of phenolics in the regulation of quorum sensing in rhizobia.

Quorum sensing in *A. tumefaciens* is regulated by the specific *acc* and divergent *arc* operon, closely linked to opine catabolism loci, and also by the *tra* (conjugal transfer genes) and *repABC* operons of the Ti plasmid (White and Winans, 2007). Mainly, TraI/R and TraM, in conjunction with the diffusible quoromones, *N*-3-(oxooctanoyl)-L-homoserine lactone (AAI) and 3-oxo-C8 HSL, regulate quorum sensing (Pappas *et al*., 2004; Zhu and Winans, 1999). In this mechanism, the *N*-acylhomoserine lactone synthase or TraI, encoded by the first gene of a cluster located on the Ti plasmid (Hwang *et al*., 1994), synthesizes AAI, which then binds to the signal receptor and transcription regulator TraR to form a stable dimer (White and Winans, 2007). This, in turn, binds to the *tra* boxes (18 bp sequences) and activates the transcription of the Ti plasmid *tra* genes and *repABC* operon for conjugal transfer (White and Winans, 2007). The *traR* genes are expressed only in the presence of specific conjugal opines, indicating the indirect role of phenolics (Oger and Farrand, 2002; Pappas, 2008). The products of the *repABC* operon, in turn, increase the copy number of the Ti plasmid to lessen the metabolic burden during saprophytic growth (Cho and Winans, 2005). TraM is another important regulator of quorum sensing, and the gene encoding it is just adjacent to *traR*. Although AAI induces TraR at low cell density, the phenolic-inducible TraM sequesters and inhibits TraR, resulting in quorum quenching (Chen *et al*., 2004; White and Winans, 2007). In addition to TraM, the products of the *attKLM* operon on the cryptic plasmid of *Agrobacterium* also inhibit quorum sensing specifically, either when the population density is low or under conditions of carbon and nitrogen starvation at high population density. AttM, AttL and AttK are also important for the utilization of other rhizospheric quoromones, such as the γ-butyrolactones produced by other soil bacteria, as alternative nutrition and energy sources in times of food scarcity (Fig. 2).

Quorum sensing in *Agrobacterium* is also inhibited by salicylic acid (SA) which upregulates the *attKLM* operon, the products of which, in turn, degrade the bacterial quormone *N*-acylhomoserine lactone (Yuan *et al*., 2007). Transcriptome analysis of *A. tumefaciens* revealed that, in this quorum-quenching effect, SA functions additively with indole-3-acetic acid (IAA) and γ-aminobutyric acid (GABA) (Yuan *et al*., 2008). In a complementary approach, a recent analysis of the *Arabidopsis* transcriptome also indicated the roles of SA and IAA, as well as of ethylene, in defence against *Agrobacterium* infection (Lee *et al*., 2009). The protective effect of SA against *Agrobacterium* is not limited to quorum quenching. SA can also shut down the expression of the *vir* genes (Anand *et al*., 2008; Yuan *et al*., 2007), mainly by attenuating the function of the VirA kinase domain and shutting down the *virA*/*G* regulatory system (Yuan *et al*., 2007). Interestingly, these inhibitory effects of SA on plant genetic transformation by *Agrobacterium* do not appear to be related to the classical role of SA in plant–pathogen interactions, i.e. the SA-induced expression of pathogenesis-related (PR) genes (Lee *et al*., 2009).

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