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Compatible plant-aphid interactions: How aphids manipulate plant responses

Interactions compatibles plantes-pucerons: comment les pucerons manipulent les réponses des plantes

Philippe Giordanengo[a,*], Laurence Brunissen[a], Christine Rusterucci[a], Charles Vincent[b], Aart van Bel[c], Sylvie Dinant[d], Christine Girousse[e], Mireille Faucher[f], Jean-Louis Bonnemain[f]

[a] *Plant Biology and Insect Pest Control (EA 3900), université de Picardie Jules-Verne, 33, rue Saint-Leu, 80039 Amiens cedex, France*
[b] *Centre de recherche et de développement en horticulture, agriculture et agroalimentaire Canada, 430, Boulevard Gouin, Saint-Jean-sur-Richelieu, QC J3B 3E6, Canada*
[c] *Plant Cell Biology Research Group, Department of General Botany, Justus Liebig University, Senckenbergstrasse 17-21, 35390 Giessen, Germany*
[d] *UR501, institut Jean-Pierre-Bourgin, Institut national de la recherche agronomique, route de Saint-Cyr, 78026 Versailles, France*
[e] *INRA, UMR1095, Genetics, Diversity and Ecophysiology of Cereals, 234, avenue du Brézet, 63100 Clermont-Ferrand, France*
[f] *Physiologie moléculaire du transport des sucres chez les végétaux (FRE 3091), université de Poitiers, 40, avenue du Recteur-Pineau, 86022 Poitiers cedex, France*

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ABSTRACT

To access phloem sap, aphids have developed a furtive strategy, their stylets progressing towards sieve tubes mainly through the apoplasmic compartment. Aphid feeding requires that they overcome a number of plant responses, ranging from sieve tube occlusion and activation of phytohormone-signalling pathways to expression of anti-insect molecules. In addition to bypassing plant defences, aphids have been shown to affect plant primary metabolism, which could be a strategy to improve phloem sap composition in nutrients required for their growth. During compatible interactions, leading to successful feeding and reproduction, aphids cause alterations in their host plant, including morphological changes, modified resource allocation and various local as well as systemic symptoms. Repeated salivary secretions injected from the first probe in the epidermal tissue up to ingestion of sieve-tube sap may play a crucial role in the compatibility between the aphid and the plant.

RÉSUMÉ

Mots clés :
Interaction compatible plante-puceron
Défense des plantes
Manipulation métabolique
Réponse locale et systémique
Métabolisme primaire
Occlusion des tubes criblés

Pour accéder à la sève phloémienne, les pucerons ont développé une stratégie furtive, leurs pièces buccales différenciées en stylets souples s'insinuant entre les cellules. Malgré les faibles dégâts cellulaires occasionnés, leur alimentation nécessite l'inhibition des réponses des plantes, depuis l'occlusion des tubes criblés et l'activation des voies de signalisation phytohormone-dépendantes jusqu'à l'expression des molécules anti-insectes. Parallèlement, les pucerons peuvent manipuler le métabolisme primaire de leur plante hôte afin

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[*] Corresponding author.
E-mail addresses: philippe.giordanengo@u-picardie.fr (P. Giordanengo), laurence.brunissen@u-picardie.fr (L. Brunissen), christine.rusterucci@u-picardie.fr (C. Rusterucci), charles.vincent@agr.gc.ca (C. Vincent), aart.v.bel@bot1.bio.uni-giessen.de (A. van Bel), dinant@versailles.inra.fr (S. Dinant), girousse@clermont.inra.fr (C. Girousse), mireille.faucher@univ-poitiers.fr (M. Faucher), jl.bonnemain@voila.fr (J.-L. Bonnemain).

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que la composition de la sève qu'ils ingèrent couvre mieux leurs besoins nutritionnels. Dans le cas d'une interaction compatible, les pucerons induisent de multiples altérations de leur plante hôte, incluant des modifications morphologiques, une réallocation des ressources ainsi que des réponses locales et systémiques variées. Injectées dès la première piqure dans les tissus épidermiques et durant la phase d'ingestion de sève, les sécrétions salivaires des pucerons semblent jouer un rôle majeur dans l'établissement d'une interaction compatible avec leur plante hôte.

1. Introduction

Aphids (Sternorrhyncha: Aphididae) are exclusive phloem feeders distributed worldwide. They cause serious losses to cultivated plants. Their economic impact is related to highly efficient colonization and settlement, due to several biological characteristics. Firstly, parthenogenesis confers a double intrinsic rate of increase and a shortened pre-reproductive time. Secondly, winged adults colonize new host plants while wingless adults invest more resources in reproduction [1]. Thirdly, high population densities lead to significant nutrient withdrawal from sieve tubes and, fourthly, they vector numerous phytoviruses.

To counteract herbivore attacks, plants have developed chemical and physical defences, either constitutive or inducible. While most herbivores may leave a damaged plant to exploit a naive or healthy one, insects feeding on phloem must have prolonged interactions to ingest sufficient amounts of their dietary solution because of their small body size and their soft cuticle that makes them vulnerable to rapid dehydration. Aphid survival therefore relies on their ability to access phloem bundles, avoiding or sabotaging plant defence responses, and to withdraw their liquid diet while keeping the phloem cells alive. Contrary to grazing insects that remove large pieces of plant tissues, aphids only inflict slight physical damage. To access sieve tubes, they insert long and flexible stylets that mainly progress in the cell wall apoplasm between cells. In the course of this extracellular transit to sieve tubes, stylets also realize intracellular punctures to assess their internal chemistry [2].

The early plant responses to attacks by phytophagous insects or by pathogens share common events such as protein phosphorylation, membrane depolarization, calcium influx and release of reactive oxygen species (ROS, such as hydrogen peroxide (H_2O_2)) [3]. This leads to activation of phytohormone-dependent pathways. Shortly, ethylene- (ET) and jasmonate- (JA) dependent responses are activated by necrotrophic pathogens [4] and grazing insects [5], whereas the salicylate- (SA) dependent response is triggered by biotrophic pathogens [4]. However, plants are able to fine-tune differential production of SA, JA and ET signalling molecules to adapt their response to the type of bioagressor [6,7]. Phytohormone accumulation triggers both local and systemic plant responses, leading to production and accumulation of defense proteins and secondary metabolites with antixenotic or antibiotic properties in damaged and non-damaged parts of the plant. Interestingly, in the case of plant-aphid compatible interactions, a plant SA-dependent response appears to be activated, while expression of JA-dependent genes appears repressed [8–11].

From the very first insertion of stylets in epidermal tissues up to the prolonged feeding on sieve tubes, aphids continuously inject salivary secretions within plant tissues. The effects of aphid saliva on plant metabolism and defence responses are partially understood. However, saliva injections are probably required to counteract plant defence. Indeed, despite their furtive strategy, phloem feeders cause alterations in their host plants, including morphological changes, modified resource allocation and various local as well as systemic symptoms [12]. These alterations are potential inducers of plant defence responses and the continuous production of saliva, believed to counteract plant defence responses, may also participate in the manipulation of plant metabolism to ensure compatible interactions between aphids and host plants.

Advances in genomics, transcriptomics and proteomics during the last decade enabled numerous studies to decipher the complexity of plant-aphid interactions. This review will first address events occurring during probing and stylet penetration into sieve elements. Secondly the reprogramming of plant responses will be treated. We focused on situations where compatible plant-aphid interactions occur, i.e., when aphids successfully feed on plants.

2. Probing and stylet penetration into the sieve elements

2.1. Plant acceptance and search for sieve tubes

After landing on plants, aphids rapidly insert long flexible stylets composed of two outer mandibules and two inner maxillae, forming a salivary and a food canal within leaf tissues to assess internal chemistry. As this occurs both in host [13] and non-host [14] plants, it suggests that plant surface signals have little or no influence in this probing behaviour [13]. The first insertions of the stylets last less than one minute and are probably only epidermal. These first probes seem to provide enough information for plant rejection and induction of aphid flight or, alternatively, for plant acceptance [1,13] as parturition occurs before stylets reach the phloem [14]. Stylet insertion in the plant occurs between two epidermal cells, then the mouthparts follow an apoplastic pathway between the primary and secondary cell wall layers [15]. Gelling saliva is continuously secreted during this apoplastic transit and forms a lubricating and hardening sheath around the stylets. That sheath remains within plant tissues after stylet withdrawal [2,15]. The gel sheath would facilitate stylets transit via the extracellular pathway and stabilize the coaptation of the mandibules and the maxillae [16]. On the way to the phloem, stylets briefly puncture many cells then are withdrawn a few seconds later. They do not run through the punctured cells. After stylet withdrawal the punctured site is readily sealed by gelling saliva [2,15]. Concomitantly to stylet insertion through plasmalemma, a small quantity of watery saliva is injected within the cytosol, and, few seconds later, about 25 fL (M. Drucker, pers. comm.) of a saliva/cytoplasm mixture is ingested [2,15,17]. These intracellular probes are not only decisive for plant acceptance [1,18], but also to locate the position of the stylets within the plant tissues. The more numerous punctured cells near the phloem vessels [15] indicate that aphids find sieve tubes by assessing the internal chemistry of the punctured cells [19]. While intracellular probing seems to be related with plant rejection and stylet withdrawal [1], apoplastic factors such as pH or saccharose gradients may also be perceived along the extracellular pathway [20]. Cues provided by the peripheral tissues leading to orientation towards the sieve tubes remain unclear. Whiteflies, other Sternorrhyncha that feed on phloem sap, perform only few cell punctures [21], suggesting that different mechanisms occur to locate sieve tubes. When reached, sieve elements are directly injected with watery saliva before phloem uptake. Such saliva injection, which may periodically recur during the whole phloem-feeding period [22], probably counteracts plant defence mechanisms [23,24].

2.2. Saliva composition and its antagonistic effects

Current knowledge on aphid saliva contents suggests that a common pattern may be found in gelling saliva composition between different aphid species [20,25,26], whereas watery saliva compositions strongly differ [24,27]. Gelling saliva is primarily composed of proteins (including phenoloxidases, peroxidases, pectinases, β-glucosidases), phospholipids, and conjugated carbohydrates [20,25,26]. Watery saliva is a more complex mixture of enzymes and other components [20,24–28]. Proteomic analyses revealed that watery saliva composition differs between aphid species [24,25,27,29] and within the same species according to the diet [25]. This suggests that host-plant range depends on variations on watery saliva composition [27]. This hypothesis is supported by strong alterations in feeding behaviour and survival of *Acyrtosiphon pisum* on its host *Vicia fabae* following RNA-i repression of one single salivary protein (C002) expression [30]. Although the function of C002 protein is unknown, the drastic effects of its silencing suggest its essential role in aphid feeding [31].

Pectinase, pectinmethylesterase, polygalacturonase and cellulase activities have been found in the watery saliva of several aphid species [25,29,32,33]. These enzymes may facilitate stylet progress by degrading cell walls. However, stylet penetration seems to go faster than the enzyme activity would allow [20,25].

Oligogalacturonides (α-1,4 D-galactosyluronic acid oligomers), fragments of homogalacturonans released after primary cell wall damage, are known to induce plant defence [34]. These oligogalacturonides can be produced through cell wall degradation by hydrolytic activities of salivary secretions [35]. Such aphid recognition is reported in wheat, where the exogenous application of pectinases on wounded leaves increases attractiveness of the treated plant towards parasitoid specialists of the wheat aphid *Sitobion avenae* [36].

Polyphenoloxidases and peroxidases constitute the second class of enzymes whose activities have been identified in salivary secretions of several aphid species [25,26]. As the toxicity of phenolic compounds depends on their redox state, most of the phenolics and their derivatives released by the damaged tissues may be absorbed by the salivary sheath, where both oxidase types convert them into less toxic substances [26,37–40]. Their further polymerization causes browning of cells in contact with the saliva, as observed in alfalfa infested by *Therioaphis trifolii* [37].

Another oxidase, a glucose-oxidase, has been recently shown in the salivary secretions of the green peach aphid *Myzus persicae* [29]. Reported in the saliva of the Lepidoptera *Heliothis zea* and *Spodoptera exigua*, this enzyme oxidizes D-glucose releasing H_2O_2, which may stimulate SA accumulation [41].

JA-dependent and SA-dependent pathways may have reciprocal antagonistic effects [42]. Such strategy, where SA antagonizes with JA, is used by the whitefly *Bemisia tabaci* [43]. In contrast, Bostock [44] reported that SA accumulation does not influence or even synergizes with JA synthesis, and therefore amplifies plant defence response. Glucose-oxidase has been reported to interfere with early steps of the wound-activated mevalonate and 2C-methyl erythritol 4-phosphate pathways, leading to the biosynthesis of anti-insect secondary metabolites, terpenoids and saponins respectively [45–47]. Glucose-oxidase is a potent inhibitor of lipoxygenase activity that can, in turn, inhibit JA production, as observed on glucose-oxidase treated tobacco plants [47]. Irrespective of the exact function of glucose-oxidase, the presence of this enzyme in their saliva may allow the aphids to reroute plant signalling, thus leading to the weak induction of JA-dependent response observed on aphid-infested plants [8–11].

Compared to these proteins whose enzymatic activity is believed to depress plant responses, De Vos and Jander [48] reported that *Arabidopsis* local defence was elicited by *M. persicae* salivary peptides in the 3–10 kDa fraction. Interestingly, these peptides are in a lower fraction range than that of proteins identified in the saliva of different aphid species, including *M. persicae*, reported above to suppress plant defence [25,27,29,49].

Elicitors of plant defence have been identified in some chewing insects, particularly in Lepidoptera: volicitin (N-(17-hydroxylinolenoyl)-L-glutamine) from *S. exigua* [50], glucose-oxidase in *Helicoverpa zea* [45], β-glucosidase from *Pieris brassicae* [51]; and in Orthoptera: caeliferins (disulfo-oxy fatty acids) from *Schistocerca americana* [51]. Although no elicitor or suppressor has been formerly identified in aphid salivary secretions, aphids have been found to greatly interfere with different metabolisms, including cell wall modelling, photosynthetic activity and source-sink relations, and secondary metabolism [11,52–54].

An alternative mechanism of bioagressor recognition involving molecules from both plant and its bioagressor is the indirect perception of the pathogen attack in which the plant recognizes its own molecules altered by the bioagressor as foreign molecules [55]. Chloroplastic ATP synthase γ-subunit proteins from *Vigna unguiculata* previously ingested by caterpillar of the fall armyworm *Spodoptera frugiperda* and regurgitated in the form of derived peptides termed inceptins have been identified as potent elicitors triggering cowpea defence [56]. Such a recognition mechanism, never reported in plant-aphid interactions, would be consistent with the successive saliva injections and cytoplasm ingestions during the numerous cell punctures realized throughout the progress of the stylets towards sieve tubes.

2.3. Control of sieve tube occlusion

In response to mechanical damage, plants immediately occlude injured sieve elements to prevent sap loss (e.g. [57]). Occlusion may involve callose (a β-1,3 glucan polymer) deposition, constricting the sieve pores and plugging by phloem-specific proteins, which coagulate or disperse in response to injury signals [57,58]. In legumes, spindle-like protein bodies termed forisomes were reported to disperse resulting in sieve tube occlusion [59,60].

As demonstrated by remote burning, which triggers electrical potential waves, protein plugging (a few seconds) is much faster than callose deposition (a few minutes) [57]. Both occlusion mechanisms are activated by a sudden influx of Ca^{2+} ions [23,58,59] from sieve element storage compartments such as the endoplasmic reticulum and cell wall by activation of voltage- and mechano-sensitive Ca^{2+} channels [61]. Coagulation of proteins may also be induced by a change in the redox state of the sieve-tube sap [62].

Whereas piercing a sieve element with a microcapillary immediately induces sieve plate occlusion [58], stylet penetration does not affect sap flow. It indicates that watery saliva secreted before and during feeding into sieve tubes [2] prevents sieve tube occlusion so that sap flow continues [23]. As with the cells along the stylet penetration pathway towards the sieve tubes [63], the gelling sheath saliva may seal sieve elements to minimize loss of phloem sap, influx of Ca^{2+} through the puncture and a decline of turgor pressure, which in turn activates influx by mechano-sensitive Ca^{2+} channels [2,23,49]. After penetration of the mouthparts, watery saliva is immediately injected during a few minutes into the sieve element prior to any sap ingestion. Such phases of intense salivation may periodically be repeated during the whole ingestion phase [22], suggesting a competition between mechanisms controlling sieve tubes occlusion and preventing effects of injected saliva.

Experimentally induced forisome plugging of sieve tubes in major veins of *V. fabae* led to a shift in *Megoura viciae* behaviour from ingestion to salivation for several minutes [49]. The authors showed, by *in vitro* treatments, that concentrated watery saliva impeded Ca^{2+}-induced dispersion of forisomes. On the basis of western blotting they also concluded to calcium-binding properties of at least two proteins in *M. viciae* salivary secretions [49]. Recently, a homolog to regucalcin (a putative calcium-binding protein) was characterized by mass spectrometry in *Schizaphis graminum* salivae [27]. Thus, salivary proteins reduce Ca^{2+} availability, which inhibits sieve tube occlusion by protein coagulation [49] as well as stylet canal plugging during sap ingestion [2,15]. Translocation of salivary secretions in plants has been demonstrated [64], so that mobile salivary compounds may prevent clogging of sieve plates distant from the feeding site [35].

Although the exact mode of action of salivary proteins in preventing sieve tube occlusion awaits further elucidation, phloem specific proteins and aphid salivary proteins are likely compete for free calcium [49]. The ability of salivary proteins to bind Ca^{2+} may thus play a crucial role to ensure a compatible interaction between aphids and host plants [2].

3. Reprogramming plant responses

3.1. Early events

Despite the physical damage inflicted by stylet penetration into plant tissues and the putative release of elicitors from both plant and aphid, it is assumed that only slight cellular disorders are induced [15].

ROS, readily released upon injury, are toxic to insects [65]: they induce a hypersentive response and trigger the plant defence pathways locally and in remote tissues [52]. Although cell death has been reported limited around the sites of feeding [66], hypersensitive responses have been rarely observed in plants infested by aphids, suggesting a role of salivary secretions in preventing ROS production, probably through enzymes such as NADH-dehydrogenases [29]. Transcriptional studies of *Arabidopsis thaliana* infested by the cabbage aphid *Brevicoryne brassicae* showed up-regulation of several genes encoding proteins involved in ROS detoxification (e.g. ascorbate reductases and L-ascorbate oxidase, copper protein precursor, glutathione S-transferases and glutathione S-conjugate transporters, peroxidase precursors) while transcript-coding proteins involved in ROS generation were moderately induced, such as copper amine oxidase and NADPH oxidase or even down-regulated, such as superoxide dismutase and polygalacturonase RNAs. At the same time, expression of two genes (*BAP1* and *BAP2*) encoding inhibitors of H_2O_2-generated cell death [67] were induced [68].

Mechanical damage to cells causes a sudden calcium influx believed to induce local and systemic signalling cascade [69,70]. Several transcripts of calcium-binding proteins, including calmodulin-binding proteins [52], calmodulin-regulated nucleotide-gated ion channels, calcium-dependent protein kinases, calcium-binding calreticulin and pinoid-binding protein, as well as calcium-transporting ATPases, were up-regulated from the sixth hour in *Arabidopsis* infested by the cabbage aphid *B. brassicae* [68]. Several genes encoding transcription factors, including some associated with pathogen resistance or senescence [71], were also induced by the cabbage aphid [68].

Aphids thus modulate the initial steps of plant responses, especially signals related to oxidative stress and calcium. This emphasizes the importance of an early suppression of the plant response to ensure the compatibility of the plant-aphid interactions.

3.2. Systemic responses

In addition to local reactions, aphid infestation induces a systemic effect on the expression of genes involved in cell wall modification, water transport, vitamin biosynthesis, photosynthesis, and carbon and nitrogen assimilation [52]. This systemic effect may reflect either plant defensive responses to limit subsequent infestations on other parts of the plant or a manipulation of plant responses that can facilitate the feeding of other aphids. Systemic responses are molecular, chemical or morphological events that occur in non-damaged parts of an attacked plant. They include responses remotely transmitted via vascular tissues as well as those related to priming by self-released volatiles [72].

3.2.1. Primary metabolism

As exclusive phloem feeders, aphids receive all their nutrients from phloem sap. Generally free of toxins and feeding deterrents, phloem sap contains high concentrations of sugars, providing an abundant source of carbon and nitrogen, essentially in the form of free amino acids. The amount of essential amino acids (5–25% of whole amino acid content) made available by phloem sap is insufficient to meet the aphid's requirements [73]. This shortfall is partly compensated for by the endosymbiotic coccoid γ-proteobacterium *Buchnera aphidicola*, which biosynthesizes lacking essential amino acids [74] from sucrose and aspartate present in the phloem sap [75]. Besides these adaptations specific to Hemiptera, strikingly the sole insect order comprising of phloem feeders, aphids may alter plant metabolism, which could be a strategy to adapt phloem sap composition to their own nutritional requirements. Two aphids inducing chlorotic lesions, *S. graminum* and *Diuraphis noxia*, were reported to affect nitrogen allocation when they feed on grasses [76,77]. In contrast, *Rhopalosiphum padi*, which does not cause any visible macroscopic changes, did not modify phloem sap amino acids composition [76]. Girousse et al. [78] reported an apparent shift from nitrogen sinks to nitrogen sources after 24 h of aphid severe infestation. Interestingly, such shift was also observed in non-infested tissues of attacked plants, suggesting a long-distance impact on nitrogen metabolism induced by aphid infestation. We recently found evidence of aphid ability to interfere with nitrogen and sugar metabolisms (Sené et al., unpublished data). Using enzymatic and immunohistochemical bioassays, we found that *M. persicae* infestation on a potato plant strongly increases glutamine synthase and glutamate dehydrogenase activities at the site of feeding, while an enhanced glutamine synthase activity was also measured in distant leaves. Our results are consistent with those of Divol et al. [52] who reported the induction of several genes involved in nitrate and sugar remobilisation in celery, including glutamine synthase by *M. persicae*. Voelckel et al. [79] reported induction of glutamate synthase in *Nicotiana attenuata* infested by *Myzus nicotianae*. It has been suggested that aphids can breakdown leaf proteins, inducing senescence-like changes, and take advantage of the increased translocation [80,81]. This hypothesis is in agreement with an increased glutamine synthase activity mainly involved in controlling amino acid transport and recycling ammonium released as a product of protein catabolism [82]. It appears that aphids feed on amino acids translocated from various tissues as well as those rerouted from metabolic sinks, at least under severe infestation [78].

Expression of genes encoding enzymes involved in carbon assimilation, including carbonic anhydrase, glycolate oxidase, ascorbate oxidase, glyceraldehyde-3-phosphate dehydrogenase, and S-adenosyl methionine were induced by *M. persicae* feeding on celery. However, such changes in the expression of genes associated with photosynthesis probably reflect plant stress status [52].

3.2.2. Cell walls

Transcriptional reprogramming by aphids includes several genes involved in cell wall metabolism and remodelling [52,68]. Feeding respectively on *Arabidopsis* and *Apium graveolens*, *B. brassicae* and *M. persicae* induced systemic over-expression of cell wall-associated protein kinases and pectin esterases, pectin acetyl esterase, expansin and cellulose synthase, whereas transcription of pectin esterase inhibitors was down-regulated [52,68]. Changes in transcript accumulation of xyloglucan endotransglycosylase/hydrolases (XTH) were also reported following an aphid infestation in *Beta vulgaris* [83], *A. graveolens* [52] and *Arabidopsis* [68,84]. These enzymes, involved in the metabolism of xyloglucans, linking adjacent cellulose microfibrils, are essential in structuring the primary cell wall [85]. A mutant *Arabidopsis* line with disrupted *XTH33* provided evidence that cell wall changes affect plant-aphid interactions [86]. Alteration of the expression pattern of such genes would favour the progeny to feed by facilitating stylet penetration of nymphs through cell walls in cooperation with salivary pectinases and cellulases. Nevertheless, variations in the expression of many genes involved in cell wall remodelling may be involved in adjustment of turgor pressure variations due to aphid feeding [52] or may strengthen cell wall barriers and reinforce defence mechanisms.

3.2.3. Hormone-signalling pathways

Contrary to grazing insects, phloem feeders activate both SA- and JA-regulated genes, and induce transcription of phytohormones involved in disease responses, including ET, abscissic acid and giberellic acid [11]. *S. graminum* [10], *Macrosiphum euphorbiae* [87] and *M. persicae* [84,88] induced a strong up-regulation of the SA-dependent pathway and reduced the expression of JA-dependent genes. Strikingly, up-regulation of SA expression in tomato and *Arabidopsis* plants exhibited deleterious effects on *M. euphorbiae* [89] and *M. persicae* [88] respectively, whereas the SA-insensitive *npr1 Arabidopsis* mutant reduced the performance of *M. persicae* and *B. brassicae* [90]. Although the latter aphid species exhibited improved population growth on the JA-insensitive *coi1* mutant [90,91], the constitutive expressing JA- and ET-signalling pathways *cev1 Arabidopsis* mutants restricted aphid population growth [91]. Exogenous application of methyl jasmonate inducing JA-dependent gene expression strongly altered performance of several aphid species [9,10,89,91,92]. Apparently, aphids inhibit efficient plant defence conferred by JA-regulated genes [9,10,88,93], while allowing the SA-regulated pathway to spread. This could be a decoy strategy, rerouting plant response towards an inefficient defence against the feeding aphids [8]. Moreover, induced expression of SA-regulated genes may prevent pathogen infections and ensure host-plant suitability during prolonged plant-aphid interactions. However, it appears that the role of SA, JA and ET varies among plant species and may differ depending on aphid species. Therefore understanding the involvement of phytohormones and their contribution in limiting aphid infestation in the case of compatible interactions, still needs further work.

3.2.4. Growth

When aphids induce phytotoxicoses, plant damage is usually ascribed to a toxic effect of some substances delivered by salivary secretions [94]. On the contrary, when no toxic effect is visible on host plants, it is generally assumed that aphid effect on growth is mainly due to removal of phloem sap from their host plants ([94] and references therein). In fact, when aphids, like the pea aphid *A. pisum*, do not induce visible toxic effect, the relationships between aphid feeding and plant growth inhibition are complex. Using different combinations of pea aphid numbers and locations on alfalfa stems, a quantitative relationship between reduction in stem elongation rate (SER) and ^{14}C-assimilate withdrawal due to aphid feeding was found [95]. SER reduction does not depend on this sole parameter, as changes in allocation only explain half the variance of this relationship. Aphid settlement on stem-growing zone induces a systemic reduction of C and N fluxes in the stem, notably a negative nitrogen deposition rate in the apical zone under severe infestation [78]. Moreover, a short-term infestation (24 h) by pea aphids on alfalfa stems induces a reversible (within 24 h) reduction of SER when aphids feed on non-elongating internodes, but a dramatic long-lasting reduction of SER (at least 8 days, i.e., the duration of the experiment) when aphids feed on elongating internodes (Girousse et al., unpublished data). Together with observations of a smaller reduction of stem radial growth than stem elongation, these data suggest that signals associated with aphid feeding induce a reprogramming of plant growth to the aphid's advantage.

4. Conclusion: it takes two to tango

Establishing a compatible interaction requires two complementary components. Aphids must fine-tune both early local and later systemic events, but also resource allocations to adapt phloem sap to their trophic requirements.

Aphid salivary secretions appear fully involved in plant reprogramming, while the underlying mechanisms remain partially unknown. Phloem sap is believed to coordinate plant response as several molecules related to stress and signalling have been reported in the sieve tubes of aphid attacked plants [65]. Interestingly, it can also be used by aphids to distantly manipulate plant metabolism, as suggested by the translocation of salivary compounds [64]. Whatever the plant or aphid origin of the molecules involved in plant responses, phloem sap composition thus appears to highly vary in response to aphid attack. Because phloem sap is the main diet of aphids, such variations in its content may affect aphid performance and colonization. Several studies have reported beneficial or adverse effects of previous conspecific or heterospecific infestations on aphid behaviour and performance on secondary colonizers [22,96,97]. In the case of interspecific interactions, variations depending on the sequence of plant colonization by aphid species were also reported, emphasizing the specificity of the systemic induced response [97].

While direct competition implies extensive damage to the plant, indirect competition may occur following a brief interaction causing slight macroscopic damage. Unlike the acknowledged theory postulating that competition between herbivore insects is negligible because they are not limited by resources [98], induced plant responses arise as key factors governing interactions between phytophagous insects [99], particularly within phloem feeders [100]. As competition is thought to be the mainspring of community distribution, abundance and diversity, aphid induced responses then would play a crucial role in structuring phloemophagous and, more widely, insect communities.

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