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Mechanisms and evolution of plant resistance to aphids

Tobias Züst† and Anurag A. Agrawal

Aphids are important herbivores of both wild and cultivated plants. Plants rely on unique mechanisms of recognition, signalling and defence to cope with the specialized mode of phloem feeding by aphids. Aspects of the molecular mechanisms underlying aphid–plant interactions are beginning to be understood. Recent advances include the identification of aphid salivary proteins involved in host plant manipulation, and plant receptors involved in aphid recognition. However, a complete picture of aphid–plant interactions requires consideration of the ecological outcome of these mechanisms in nature, and the evolutionary processes that shaped them. Here we identify general patterns of resistance, with a special focus on recognition, phytohormonal signalling, secondary metabolites and induction of plant resistance. We discuss how host specialization can enable aphids to co-opt both the phytohormonal responses and defensive compounds of plants for their own benefit at a local scale. In response, systemically induced resistance in plants is common and often involves targeted responses to specific aphid species or even genotypes. As co-evolutionary adaptation between plants and aphids is ongoing, the stealthy nature of aphid feeding makes both the mechanisms and outcomes of these interactions highly distinct from those of other herbivore–plant interactions.

Aphids are specialized herbivores that feed on the phloem of vascular plants, especially in the temperate regions of the world. They are potent pests on virtually all crops[1]. Phloem sap contains an abundance of simple sugars (produced in 'source' leaves by photosynthesis and transported to 'sinks' of plant growth), as well as nutrients and plant secondary metabolites (PSMs), but contains few essential amino acids[2]. To cope with the overabundance of sugars in their diet and maintain their osmotic balance, aphids have evolved morphological modifications to their intestinal tract, and exude large quantities of sugars in the form of honeydew[3]. To meet all their nutritional requirements, aphids obligatorily associate with endosymbiotic *Buchnera* bacteria for the synthesis of essential amino acids[4]. Plants in turn have evolved a range of mechanisms to protect the valuable resources contained in their phloem sap, and natural selection exerted by aphids has been an important contributor to plant–herbivore co-evolution[5–7].

Compared to chewing herbivores, aphid feeding typically causes relatively little harm to a plant. Furthermore, aphids produce and inject specific compounds ('effectors') intended to modulate and suppress the phytohormonal and defensive response of susceptible plants[8]. In resistant plants, aphid salivary compounds ('elicitors') may be recognized by plants and activate targeted defences[8], including the induction of PSMs and other mechanisms of resistance. Negative impacts of sustained aphid feeding often arise from the rapid clonal reproduction of aphids and subsequent depletion of the plant's resources. In addition, a large number of predominantly generalist aphid species are known to transmit plant pathogenic viruses[9] and, for these species, the detrimental effects of virus transmission often exceed the direct effects of aphid feeding.

Recognition of a plant as a suitable host and subsequent feeding initiation by an aphid depends on a complex interaction between aphid and plant traits (Box 1). Aphids extensively probe and salivate into potential host plants, and frequently reject non-hosts after initial sampling of epidermal cell contents, or subsequent sampling of mesophyll cell contents[10]. Plants are rejected as non-hosts by aphids for a number of reasons, including unsuitable nutrient composition and high levels of PSMs. For host plants, a deciding factor for their susceptibility or resistance is their ability to recognize aphid feeding and mount rapid defences. Plants may recognize aphid feeding using general associated cues, such as mechanical damage to cells and shifts in turgor[11]. However, many aphids feed in a highly stealthy manner and actively suppress this recognition[11–13]. In contrast, specific recognition of aphid feeding by a plant can, for example, be mediated by the recognition of aphid effectors by resistance (*R*) genes[8,14], or of other aphid-specific compounds by pattern recognition receptors (PRRs)[15]. As we discuss below, specific versus general recognition results in different plant responses and creates variation in host plant resistance[16].

Aspects of plant–aphid interactions have been well-studied over the past decade, including some of the molecular mechanisms underlying plant resistance and defence induction[16–18], processes involved in the feeding establishment of aphids[11], and ecological interactions between aphids and other organisms[19]. In this Review, we track recent progress in understanding key stages of the aphid–plant interaction, particularly the recognition of aphid feeding and the induction of defence mechanisms by the plant. We focus on contrasting the mechanisms that act at these key stages of the interaction by linking findings from molecular and whole-organism approaches, and placing them within a co-evolutionary framework.

Phytohormonal signalling

Generalized plant responses to aphid feeding are mediated by phytohormonal signalling. Leaf damage by chewing herbivores and, to some extent, cell damage by aphids induces jasmonic acid (JA) and ethylene production across a broad swathe of plants species[16,20]. However, except for a few specific aphid–plant interactions[21], aphid feeding mostly induces salicylic acid (SA), which is otherwise typically associated with plant responses to microbial pathogens[22].

Interestingly, aphids are commonly susceptible to externally induced JA-mediated defences. For example, the exogenous

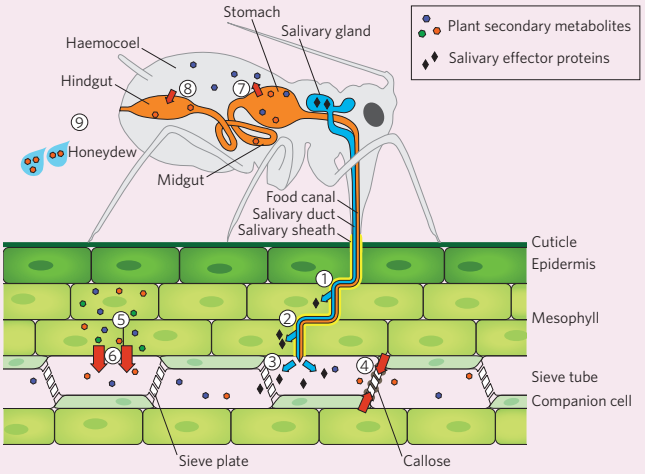
Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, New York 14853, USA. †Present address: Institute of Plant Sciences, University of Bern, Bern, CH-3013, Switzerland.
e-mail: tobias.zuest@ips.unibe.ch; aa337@cornell.edu

Box 1 | A primer on host plant selection.

With the exception of a few highly generalist aphid species, most aphids specialize on just one or a few closely related plant species[3]. Part of this specialization is due to physiological constraints, as plant-specific phloem depth must be closely matched by the aphid's stylet length[3]. However, tolerance of toxins and the ability to suppress host plant defences, as well as the composition of the aphid's endosymbiont community[90], are likely to be important drivers of specialization. Selection, settling and feeding establishment on a new host plant is thus not only critical from the perspective of an aphid's fitness, but is also a focal point in the evolution of plant resistance.

Locating new host plants is highly hazardous for aphids, and up to 99% of all winged dispersing aphids fail to find a host[91]. Winged aphids may use both visual and volatile information to detect host plants[92]. After alighting on a potential host plant, aphids may assess plant suitability based on surface molecules, including lipids and secondary metabolites[93]. A few plant species manage to repel or kill the aphid at this stage by exuding toxic compounds from glandular trichomes[94]. On plants that lack such defences, aphids secrete saliva onto the surface and imbibe it again immediately (without piercing the cuticle) to evaluate dissolved surface molecules with the chemoreceptors in their proboscis[95]. Plants thus potentially have multiple opportunities to avoid or deter aphids even before feeding begins, even though it is unclear how important or general these pre-penetration cues are for aphid host selection.

After initial acceptance, the aphid penetrates the cuticle and moves its stylet across the extracellular space toward the sieve tubes of the phloem (see figure). Throughout probing, aphids will puncture most cells along the stylet's pathway for host quality assessment as well as orientation[10,96]. Aphids secrete distinct types of saliva at different stages of feeding establishment. A thick, congealing saliva ('gelling saliva') is continuously secreted as the stylet moves through the apoplast, encasing the stylet in the salivary sheath[67]. During penetration of epidermal cells, mesophyll cells and sieve elements, as well as during established phloem feeding, 'watery saliva' is secreted alternatingly with sap ingestion[67], perhaps as a means to suppress plant defence. Several of these steps leading up to successful feeding can act as signals to elicit more- or less-targeted plant responses. For example, even though the damage to plant cells caused by aphids is relatively minimal compared to leaf-chewing herbivores, plants still upregulate genes involved in responses to wounding following aphid probing[27]. In addition, both types of saliva that aphids inject into the inter- or intracellular space and directly into the phloem contain effectors that manipulate plant responses in the aphid's favour, but at the same time represent prime targets for the recognition of aphid feeding by a plant[11,17].



A schematic of aphid feeding and plant responses. Red arrows indicate key processes for the plant–aphid interaction. Aphids penetrate the apoplast with their stylet and move it between individual cells while exuding gelling saliva into the intercellular space (1), encasing the stylet in a salivary sheath and sealing off any cell leaks caused by the insertion process. During insertion, aphids puncture mesophyll cells and inject small amounts of watery saliva containing effector proteins (2) before sucking back some liquid to assess plant quality. After the phloem is reached, aphids alternate between sap ingestion and secretion of watery saliva containing effector proteins into the phloem (3) to prevent callose deposition at sieve plates leading to phloem sealing (4). Plant cells synthesize defensive secondary metabolites (5), of which a subset is transported into the phloem (6). Secondary metabolites are ingested during feeding, and may be taken up into the haemocoel by passive or active transport across the gut membrane (7). Secondary metabolites either accumulate in the haemocoel or are excreted back into the hindgut (8) and exuded with the aphid's honeydew together with all remaining metabolites (9).

application of JA to tomato plants impaired aphid population growth[23], and previous damage by leaf-chewing herbivores that induces JA was shown to have a negative effect on aphid performance both in milkweed and tomato[5,24,25]. In contrast, SA-mediated defences have less-consistent effects on aphid performance. For example, induction of the SA pathway by a pathogen on tobacco did not impact subsequent feeding by the aphid *Myzus nicotianae*[26], but SA signalling and exogenous application of SA analogues did reduce performance of *Myzus persicae* on *Arabidopsis thaliana* and *Macrosiphum euphorbiae* on tomato[27,28].

Phytohormonal signalling is evolutionarily highly conserved, and the two hormones SA and JA are natural antagonists, most likely as part of the plant's strategy to fine-tune its defence[29]. By inducing the plant's SA pathway, aphids may be able to use this hormonal 'crosstalk' to suppress a potentially more detrimental JA response. In support of this hypothesis, mutant *Arabidopsis* plants that are deficient in SA signalling (and thus unaffected by such manipulation) are more resistant to aphids than wild-type plants[30]. It is also important to note that activation of the SA pathway by aphids commonly induces unique plant responses compared to exogenous SA application[30,31], suggestive of a finely tuned manipulation of plant responses by the aphid.

To summarize, specific aphid–plant interactions may have distinct SA/JA responses, which may reflect different stages of co-evolutionary history. We hypothesize that those aphids triggering a JA response that is detrimental to themselves would be species that have infrequent contact with their host plant (such as generalist aphids on marginal hosts), or that have not been a major agent of selection. However, the majority of aphid species are specialized to just a few host plants[3], and these aphids seem to have gained the ability to manipulate plant defences to their benefit using the plant's own hormonal crosstalk.

Co-evolution is ongoing (Box 2), and so we can expect that plants will respond to this manipulation of SA and JA responses by altering their hormonal signalling pathways or developing alternative means of aphid detection. A better understanding of the relative

importance of JA and SA signalling pathways across more plant–aphid systems is not only important for gaining a more general view of aphid resistance, but also for understanding complex community interactions among herbivores of different guilds[5,24–26]. The extent of aphid specialization and the evolutionary history of plant–aphid interactions may well serve as predictors of the relative induction and impact of the JA and SA pathways.

Plant secondary metabolites

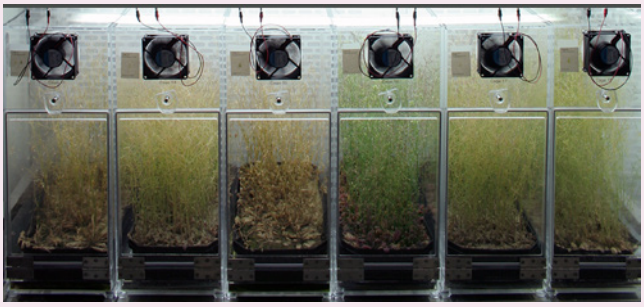
PSMs play a key role in resistance against many chewing herbivores, but their role against aphids is less clear. Even though aphids may occasionally come into contact with PSMs before feeding (Box 1), the efficacy of these compounds is mostly dependent on ingestion. Defensive PSMs are produced inside plant cells, and even though defensive compounds can be translocated via the phloem, only some of these compounds are likely to be phloem-mobile in their active form[32]. Thus, perhaps with the exception of short periods of mesophyll cell probing during feeding establishment, aphids may never be exposed to PSMs located outside the phloem. In addition, aphids cause little structural damage to a plant compared to chewing herbivores, yet for many defensive compounds such structural damage is required to bring inactive precursors into contact with a spatially separated activating enzyme. Finally, because the majority of aphid species are specialists on a single or a few closely related plant species[3], aphids are likely to have evolved tolerance to PSMs in their diet. PSM tolerance mechanisms are rarely studied in aphids, but are likely to involve avoidance of PSM uptake by the gut, active elimination from the body cavity, degradation by detoxifying enzymes after uptake, or development of insensitive target sites for plant toxins[33]. Below we provide an overview of the major types of PSMs and review the current knowledge on their activity against aphids.

Cardiac glycosides (cardenolides). These are steroidal compounds that are specific inhibitors of animal Na^+/K^+-ATPase, and have repeatedly evolved in a wide range of plants (but are dominant in the Apocynaceae)[34]. A range of cardenolide compounds are present in the phloem[35,36], and at least some studies have found negative correlations between performance of specialist aphids and foliar cardenolide levels across and within species[36,37]. In addition, several aphid species sequester host plant cardenolides for their own defence[38]. In a recent study it was shown that out of the full range of cardenolides present in plant leaves, aphids predominantly accumulate apolar cardenolides, and exude polar cardenolides in honeydew (Fig. 1)[36]. This pattern was identical across the specialist milkweed aphids *Aphis nerii*, *Aphis asclepiadis* and *Myzocallis asclepiadis*, and the extremely polyphagous green peach aphid *M. persicae*. Because low polarity of a compound allows its passive diffusion across cellular membranes, polarity of a PSM may be an important determinant of its (mostly passive) uptake in the aphid gut. All four of the aphid species tested contained some plant cardenolides in their bodies, but sequestration increased with dietary specialization. Surprisingly, the most specialized and highest sequestering aphid species was also the most susceptible to variation in plant cardenolide content, in contrast to the expected correlation between specialization and tolerance of toxins[36].

Alkaloids. These are an extremely diverse group of cyclical, nitrogen-containing compounds with a large range of targets and biological activities, including interference with neurotransmitters, disruption of DNA replication and inhibition of protein synthesis[33]. Alkaloids are produced by 20–30% of all higher plant species, with often significant impacts on herbivore feeding[33], yet again effects on aphids are variable. For example, the pea aphid *Acyrtosiphon pisum*, a specialist on many legumes (Fabaceae)[39], was shown to be only mildly deterred by pyrrolizidine alkaloids in an artificial diet, but strongly deterred by indolizidine and quinolizidine alkaloids[40].

Box 2 | Evolution of resistance.

Co-evolution between plants and aphids, among other herbivores, is continuously shaping the traits and behaviours of both interaction partners, and selection acts quickly to remove non-adaptive strategies from a natural community. For example, although most aphids undergo sexual recombination before overwintering, the high genetic diversity in spring rapidly declines to a few genotypes as selection acts to select the most adapted genotypes[97]. Similarly, exposure to a diverse community of herbivores selects for a diversity of defensive traits in plants. If aphids co-occur with other, more damaging herbivores, the selective pressure imposed by aphids might be masked by the more severe natural selection imposed by the other herbivores[98].



Selection experiments provide valuable tools to study the selective pressures imposed by aphid herbivory. In the study of Züst *et al.*[6] it is visually apparent that different aphid species had markedly different effects on plant populations (selection treatments from left: *L. erysimi*, *B. brassicae*, *L. erysimi*, control, *B. brassicae*, *M. persicae*).

Experimental manipulation of herbivore communities, and especially experimental evolution, are powerful tools to disentangle effects of multiple selective agents and to identify plant traits involved in aphid resistance. For example, two specialist aphids were shown to exert opposing selection on the glucosinolate chemotype of the model plant *Arabidopsis*, and over just five generations caused significant change in the relative frequency of the major-effect glucosinolate locus *GS-ELONG*[6]. This locus had not previously been implicated in aphid resistance. However, the results concur with those in another recent study, in which small differences in glucosinolate structure under the control of *GS-ELONG* seemed to have direct effects on the aphid's host preference, even though further genetic loci are likely to be important as well[53].

As plant resistance is shaped by co-evolutionary processes, future studies should attempt to combine the reciprocal approaches of measuring genetic changes in plants[6] and aphids[97] to simultaneously allow evolution in both interaction partners, thereby experimentally replicating natural co-evolutionary dynamics. In an interesting example, changes in plant phenotype associated with the domestication of crops, particularly the increase in nutritional quality, were shown to reduce the rate of evolutionary change in aphids through increased population size and a decrease in random drift[99]. Comparative approaches can thus provide important insights into ongoing plant–aphid co-evolution, especially if species comparisons are placed within a phylogenetic framework. The comparison of aphid performance on a set of closely related species can help to identify traits related to plant defence and aphid performance[100]. Such comparative studies can help to inform future experiments that address specific mechanisms of plant resistance.

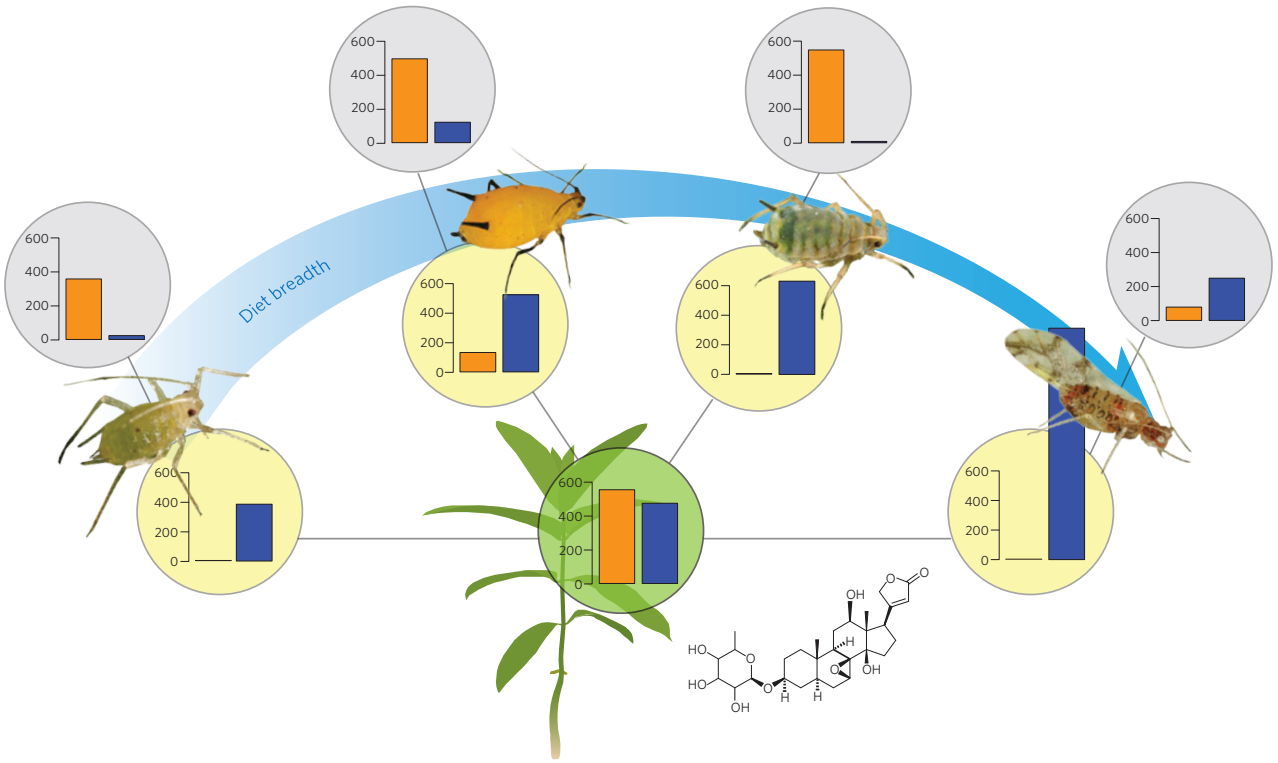


Figure 1 | A schematic of cardenolide sequestration and exudation by four aphid species on common milkweed (*Asclepias syriaca*, middle). Left to right: *Myzus persicae*, *Aphis nerii*, *Aphis asclepiadis* and *Myzocallis asclepiadis*. Aphids differ in their host specialization, ranging from broad generalism to monophagy (blue arrow). Cardenolides are grouped into polar (orange bars) and apolar (dark blue bars) compounds based on HPLC (high-performance liquid chromatography) retention times. Units are ng cardenolides per mg dried sample. The shared host plant has similar levels of polar and apolar cardenolides in leaves (green circle). All four aphids predominantly contain apolar cardenolides in their bodies (yellow circles), with evidence for increasing content with specialization. Aphids predominantly exude polar cardenolides in their honeydew (grey circles), with the exception of the most specialized aphid. Data simplified from ref. 36.

Interestingly, the least active compound in this study, and the only compound detected in exuded honeydew, was the polar indolizidine alkaloid swainsoine[40]. Similarly, the sensitivity of the broadly generalist potato aphid *Macrosiphum euphorbiae* (whose native host plants include alkaloid-producing Solanaceae[39]) to alkaloids also depends on the specific compound tested. When offered a number of glycoalkaloids and their aglycones from potato in an artificial diet, only the aglycones proved to be a deterrent to aphid feeding or lethal, at least at the highest natural concentrations, whereas the glycoalkaloids conversely acted as feeding stimulants[41]. It is interesting to note that aglycones are far less polar than their glycoalkaloids, which again suggests that the aphid gut may not be able to avoid the uptake of apolar compounds.

Alkaloid sequestration by aphids has been well-studied in the highly specialized aphids *Macrosiphum albifrons*, *Aphis cytisorum* and *Aphis jacobeae*, where alkaloid accumulation in the aphid's bodies provides a clear defensive benefit[42–45]. All three aphids require alkaloids as feeding stimulants, yet they prefer plants with lower alkaloid contents and are not found on plants with very high alkaloid levels[44]. Three of these studies reported relative alkaloid content in plant tissue and aphid bodies, making it possible to compare the predicted polarity of compounds (logP; http://pubchem.ncbi.nlm.nih.gov/) with their uptake. In two of the studies[43,45], apolar alkaloids were preferentially accumulated in aphid bodies, whereas there was no relation in the third study[42]. Similarly, *M. albifrons* aphids feeding on legumes excreted more polar alkaloids in their honeydew and sequestered non-polar compounds[45].

A number of similarities emerge between cardenolide and alkaloid uptake and their effects on aphids. Compound polarity is an important determinant of accumulation in aphid bodies, indicative of a mostly passive mode of sequestration that is shared across generalist and specialist aphids. Apolar compounds entering the aphid's body may be tolerated at low concentrations, as their accumulation provides a defensive benefit. However, both cardenolides and alkaloids seem to be toxic at high (natural) concentrations, making increased production a potentially costly but effective defensive strategy for plants.

Benzoxazinoids. Whereas cardenolides and alkaloids are transported and accumulated in their toxic form, many PSMs are maintained as inactive compounds (for example, through glycosylation), and rely on activation by enzymes following damage or ingestion. Benzoxazinoids are defensive compounds of maize and other grasses that are stored as glucosides and enzymatically activated following plant tissue damage[46]. The most abundant benzoxazinoid in maize seedlings is 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one glucoside (DIMBOA-Glc)[47]; glucosidases break this compound down into insect-deterrent compounds (such as 6-methoxybenzoxalin-3-one)[48]. In contrast, the related compound HDIMBOA-Glc breaks down more rapidly and, as a consequence, has significantly higher toxicity for aphids *in vitro*[47]. Some maize varieties constitutively express proportionally more HDIMBOA-Glc, whereas other varieties respond to aphid feeding by transforming DIMBOA-Glc to HDIMBOA-Glc, indicating genetic variation for this trait. Despite the increased toxicity of HDIMBOA *in vitro*, aphids perform better on plants high in HDIMBOA and low in DIMBOA[47], most likely because DIMBOA also acts as a signal for callose deposition (a protein involved in sieve-plate blockage; see figure in Box 1) and

may increase the effectiveness of phloem-sealing mechanisms[49]. Thus PSMs seem to be acting not only as direct agents of resistance, but also as signalling molecules that feedback on other resistance mechanisms.

Glucosinolates. Analogous to benzoxazinoids, glucosinolates are major defensive compounds of the Brassicaceae that require activation by a specific glucosidase (the enzyme myrosinase). Aphids avoid activation by imposing minimal damage to cells, and thus consume and exude mostly intact glucosinolates with little negative effects[50]. Accordingly, the addition of the aliphatic glucosinolate sinigrin to an artificial diet had no effect on the performance of *M. persicae*, unless supplemented with myrosinase[50]. However, some Brassicaceae plants also synthesize indole glucosinolates in addition to aliphatic glucosinolates. These compounds are thought to be less stable, and activate spontaneously in the absence of myrosinase[50]. Consequently, indole glucosinolates alone have been shown to impair the growth of the generalist aphid *M. persicae* when added to an artificial diet or overexpressed in host plants[50,51]. In response to feeding by *M. persicae*, *Arabidopsis* specifically induces indole glucosinolates to increase resistance, and this induction is largely independent of a functioning SA pathway[50]. More specialized aphids such as *Brevicoryne brassicae* accumulate aliphatic glucosinolates at up to 16-fold higher concentrations than found in the host plant, whereas indole glucosinolates are hardly accumulated at all[52]. Recent work suggests that sequestration of glucosinolates may be even more specific, with *B. brassicae* preferentially sequestering the glucosinolate sinigrin from cabbage while exuding the structurally similar progoitrin[53]. Although there is some evidence of aphids avoiding plants accumulating progoitrin[53], the performance of *B. brassicae* was shown to be positively correlated with the concentration of both progoitrin and sinigrin, but negatively correlated with that of indole glucosinolates[54]. Whereas aliphatic glucosinolates thus have mostly negligible or even beneficial effects on aphids (but are likely to be maintained as effective defences against other herbivores), indole glucosinolates potentially represent the plant's evolutionary response to the aphid's evasion of 'conventional' JA-mediated defences.

Across the classes of defensive compounds described above, PSMs requiring enzymatic activation seem to be less efficient in defending against aphids. In response to selective pressure, a common strategy by plants seems to be the supplementation of the defensive arsenal with compounds that are more likely to activate spontaneously (such as indole glucosinolates and the benzoxazinoid HDIMBOA-Glc). Such compounds are probably more costly for the plant, both due to autotoxicity and the higher turnover rate of these compounds in the absence of herbivores. Both benzoxazinoids and glucosinolates show evidence of inducibility following feeding by aphids, whereas the inducibility of cardenolides and alkaloids is less certain. Specialist aphids often have little impact on cardenolide levels in milkweed plants[24], perhaps due to their successful suppression of JA signalling. As co-evolution is ongoing, we expect plants to regain the ability to induce effective defences, such as the production of cardenolides and alkaloids, or to respond with other means, for example through improved mechanisms of phloem sealing.

Phloem-sealing mechanisms

As the transport system for highly valuable nutrients, the phloem is protected with a specific set of mechanisms intended to immediately seal damaged sieve tubes. These mechanisms have recently been summarized in a comprehensive review[11], and so here we only provide a short overview of potential links to other types of defence. Phloem sealing seems to be a highly efficient mechanism to prevent aphid establishment (see figure in Box 1), even though it cannot prevent probing and aphids may still spread plant viruses. Phloem sealing is tied to other lines of defence, such as species-specific aphid receptors[15]. In addition, there is some evidence that the efficiency and speed of phloem sealing can vary in the same host plant species in response to different aphid species[55]. Given the importance of phloem sealing in the plant–aphid interaction, there is a need to focus on the general patterns of variation in phloem-sealing mechanisms and their interactions with aphid recognition and plant signalling. Large-scale screening of both intra- and inter-population variation in these mechanisms in wild plants and the use of signalling mutants in model species may well provide the means by which to understand the relevance of phloem sealing in the sequence of events leading to aphid infestation.

Species-specific recognition of aphid feeding

As aphids circumvent general plant mechanisms of detecting herbivores, plants should respond with more aphid- specific mechanisms. Highly effective plant resistance to specific aphids has been linked to single *R* gene loci or alleles in a variety of plants, including several crops[14]. For example, the presence of the *Mi-1.2* allele in tomato conveys resistance to aphids, psillids, whiteflies and nematodes[14]. Similarly, the *Vat* locus is linked to resistance against *Aphis gossypii* in melon[56]. Interestingly, both resistance genes belong to the same receptor family[16], which suggests a shared response mechanism to a specific aphid elicitor — even though no such elicitor has yet been identified. Similar to *R* genes in specificity, PRRs recognize specific conserved molecule patterns of compounds (pathogen-associated molecular patterns, PAMPs) in aphid saliva[15,57].

Although PRR- and *R*-gene-mediated recognition are distinct processes, they are both highly specific mechanisms for the recognition of aphid feeding and allow the mounting of targeted immune responses by the plant (reviewed in ref. 58). Even though specific receptors or *R* genes for the detection of aphids have only been identified in a few systems, there are several plant species in which specific plant genotypes or cultivars are completely resistant against aphids[59,60]. The exact molecular mechanism in these examples remains to be elucidated, but such patterns are strongly suggestive of more widespread prevalence of *R*-gene-like mechanisms.

If plants recognize aphid elicitors or PAMPs, highly targeted defensive responses are activated[61,62]. For example, PRR activation has been linked to increased rates of phloem sealing[15]. Even though there is no direct evidence for the involvement of phloem sealing in *R* gene resistance, aphids feeding on resistant cultivars of peach significantly increased their amount of probing and sustained shorter periods of feeding, indicative of increased rates of phloem sealing[63]. Interestingly *Mi-1.2*-mediated resistance of tomato against the potato aphid has been shown to be highly dependent on the SA pathway[64]; this may be interpreted as a strategy by the plant to counter the aphid's phytohormonal manipulation through the linkage of the SA pathway with an *R* gene.

Specific recognition of aphid feeding allows the plant to rapidly mount defences and protect its resources. However, as this form of plant resistance may often be based on a single or a few genes, it is likely to break down with small changes in the salivary proteins, and thus *R* gene evolution should be under strong selective pressure in both plants and aphids. The presence of such genes within natural plant populations is likely to have important consequences for aphid community dynamics, as it determines the efficiency of plants' defence responses. Although the evolutionary dynamics of *R* genes and PRRs are well understood in plant–pathogen interactions, their importance for plant–herbivore interactions is only just beginning to emerge[58]. Future studies that specifically focus on this aspect of plant resistance will be valuable in understanding plant–aphid co-evolution.

Aphid-induced plant resistance

Nearly 50 years ago, Dixon[65] demonstrated that the intensity of aphid feeding on sycamore trees in spring was negatively correlated with aphid performance on the same plants in autumn,

Table 1 | Evidence for aphid-induced plant defences or susceptibility at a local and systemic scale.

Plant	Aphids	Methods used	Local effects (within leaf)	Systemic effects (between leaves)	Results
(Fabaceae) *Vicia faba*	*Aphis fabae*	Honeydew production, EPG	+	NA	Honeydew exudation and time to successful feeding is positively affected by previous feeding of *A. fabae* on the same leaf; no effect for *R. padi*[86].
(Poaceae) *Triticum aestivum*	*Rhopalosiphum padi*		0	NA	
(Fabaceae) *Lupinus* spp.	*Myzus persicae*	Choice assay, EPG	NA	–	Avoidance of pre-infested plants, but plant varieties differ in strength of induced effects[87].
(Poaceae) *T.aestivum*	*R.padi*	Aphid fecundity	–	–	Fecundity on leaves is reduced by pre-infestation at the local and systemic scale[88].
(Poaceae) *T.aestivum*	*R.padi*, *Sitobion avenae*	Aphid fecundity	NA	–	Fecundity is reduced by pre-infestation of heterospecific aphids, with stronger effects of *S.avenae* on *R.padi* than vice versa[70].
(Poaceae) *T.aestivum*	*R.padi*	Aphid fecundity, choice assay	(–)	–	Fecundity is reduced by pre-infestation at the systemic scale; local scale not explicitly tested. Pre-infested plants are also avoided[89].
(Rosaceae) *Prunus persica*	*M.persicae*	Aphid survival and fecundity, EPG	NA	–/+	Effects of previous feeding on survival, fecundity and feeding behaviour ranged from negative to positive, depending on plant genotype[59].
(Solanaceae) *Solanum tuberosum*	*M.persicae*, *Macrosiphum euphorbiae*	Choice assay, EPG	+	NA	Preference for pre-infested leaves; pre-infestation has positive effect on nymph survival[69].
(Solanaceae) *S.tuberosum*	*M.persicae*, *M.euphorbiae*	EPG	+	–	Enhanced feeding on pre-infested leaves, but reduced feeding on systemic leaves[68].
(Solanaceae) *Capsicum annuum*	*M.persicae*	Choice assay, EPG	NA	–	Avoidance of pre-infested plants for all three species; weakest effect for *T.aestivum*. Pre-infested leaves are as equally attractive as control leaves without aphids[67].
(Brassicaceae) *Brassica oleracea*	*Brevicoryne brassicae*		+	–	
(Poaceae) *Triticum aestivum*	*R.padi*		NA	–	

Minus sign (–), negative effect on performance; plus sign (+), positive effect on performance; 0, no effect on performance; NA, effect not tested or not controlled. EPG, electrical penetration graph.

suggestive of long-lasting host plant manipulations by aphids. Similarly, although on a shorter timescale, initial infestation of cotton plants by *A. gossypii* was shown to reduce population growth of subsequently colonizing aphids by 30% compared with uninfested controls[66]. These studies demonstrate induced plant resistance that may be mediated by either one or a combination of defensive mechanisms — although the mechanisms of resistance are typically not identified in such ecological studies, and induced effects cannot be separated from deteriorating nutritional quality. In Table 1 we summarize the literature on sequential aphid infestation and induced plant resistance, specifically avoiding studies that used intense infestations that may have led to plant deterioration. All the studies listed plants that are infested with aphids and measured the effects of previous infestation on subsequent colonization by con- or heterospecifics over a short timescale. Aphid responses to previous induction by aphids are measured as choice, performance, probing behaviour (monitored by the electrical penetration graph technique, EPG), or a combination of the three.

Most of the studies distinguish between effects of previous aphid infestation at the local scale (within the same leaf) and at the systemic scale (between leaves of the same plant). Remarkably, all studies report negative effects of previous aphid infestation at the plant scale (Table 1), which is in accordance with systemically induced plant resistance. In contrast, studies that specifically address local effects typically found increased aphid performance (induced susceptibility) within the same leaf, perhaps as a result of local suppression of defence induction and phloem sealing by salivary effector proteins accumulating within the leaf[67]. Studies that considered multiple aphid species typically found similar effects between con- and heterospecifics, although there is some evidence for asymmetric relationships between aphid species[68–70]. From this set of relatively highly controlled studies (Table 1), it is evident that aphids may suppress plant defences at a very local scale. However, we would expect to find this pattern only in susceptible plants that lack specific aphid recognition, whereas aphid saliva should elicit defensive responses in resistant plants[15]. Interestingly, even in the set of mostly susceptible plants used in these studies (Table 1), plants were still able to perceive aphid feeding and mount a systemic response that subsequently enhanced resistance, indicating the multifaceted nature of plant resistance to aphids.

Genetic variation in the plant response to aphid induction has been demonstrated[59] and increased resistance in induced plants may be due to more rapid phloem sealing[63]. However, we currently have little understanding of the inducibility of phloem sealing and its relative importance compared with PSMs, nutritional effects and other mechanisms, especially under natural conditions in the field. Different mechanisms are likely to interact with each other. For instance, competitive interaction between aphid species may be

mediated by host plant changes. In a recent example from a natural system, three co-occurring aphid species were shown to have different preferences for plants occupied by con- and heterospecifics (or aphid-free plants), depending on their relative competitive ability[71]. Many aphid species might thus actively avoid previously colonized plants, which could represent a potential source for novel aphid resistance traits in pest management. To begin to disentangle such complex interactions, known and future plant mutants with impaired traits (for instance in phytohormonal signalling, PSMs and phloem sealing) are likely to prove invaluable.

Aphid-induced plant sinks

Separate from the body of work showing induced systemic resistance, several studies report beneficial effects of previous aphid feeding for aphids, suggesting induced susceptibility (that is, aphid manipulation of the plant). For example, two aphids on pine exhibited increased survival and fecundity if they fed together[72]. Similarly, three different aphid species feeding on wheat significantly increased amino acid concentrations in the phloem[73]. Connected to these phenomena, aphids have long been reported to manipulate plants and induce physiological sinks that are beneficial for their performance[74–76]. Localized sink induction might thus interact with localized defence suppression to increase the benefit of previous aphid infestation at a very local scale.

In an extreme case of host plant manipulation, some aphid species induce senescence and manipulate plant cells to release nutrients for the aphid's benefit, which often results in visible signs of foliar chlorosis or necrosis, similar to the effects of plant viruses[73,77]. Aphids causing visible changes in their host plant ('symptomatic' aphids), such as *Diuraphis noxia* and *Schizaphis graminum* feeding on wheat and barley, significantly increase the amount of free essential amino acids in sieve tube sap[73]. In contrast, the more typical 'asymptomatic' aphids, such as the co-occurring *Rhopalosiphum padi*, have little effect on amino acids in sieve tube sap[73]. The symptomatic *Melanocallis caryaefolia* induces senescence of leaves of its pecan host (*Carya illinoinensis*) but, importantly, this induction does not benefit the co-occuring asymptomatic *Monellia caryella*; in fact, previous feeding by *M. caryella* suppresses senescence induction by *M. caryaefolia*[77]. The ability to cause foliar chlorosis or senescence by symptomatic aphids is often dependent on matching aphid–plant genotype combinations[78], indicating the involvement of genotype-specific aphid effectors. Symptomatic aphids thus seem to use a markedly different strategy to asymptomatic aphids: they manipulate the plant into increasing the available nutrients, especially essential amino acids in the phloem, but this is most likely to accelerate plant death and requires a higher host switch frequency. Given their highly divergent modes of interacting with plants, it is unclear whether the contrast of symptomatic and asymptomatic aphids will reveal insights into aphid–plant co-evolution; nonetheless, where they co-occur, strong ecological interactions are likely to ensue.

Signals in plant resistance

Although increased resistance to aphids has been best studied within a single plant, resistance can also be induced through signals from neighbouring plants. Volatiles elicited by aphid-infested plants influence surrounding plants in a way that lowers their attractiveness to subsequent aphid colonizers[67,79]. Furthermore, there is evidence that resistance in neighbouring plants can also be induced through signals transmitted via their shared mycorrhizal network[80].

Plants not only use signals to 'warn' their neighbours, but also to 'cry for help' by releasing volatile compounds when damaged, which attract natural enemies of their herbivores[22,81]. Even though aphids may not extensively damage plant tissue, plants emit volatile blends that act as long-range attractant cues for parasitoids[17,82]. The emission of these signals occurs systemically from both aphid-infested and un-infested leaves, and there is evidence that signals are sufficiently specific to allow parasitoids to discriminate between host and non-host aphid species[83]. Finally, several plants release (*E*)-β-farnesene when damaged[84], a volatile compound that is generally used by aphids as an alarm pheromone that elicits dropping off plants or moving away, and that also acts as an attractant for natural enemies of aphids such as parasitoid wasps[85]. Plants may therefore trick aphids into fleeing their feeding site, or alternatively increase the apparency of aphids to their enemies. Such volatile and other signals are thus likely to be of key importance in understanding community dynamics at a larger scale.

Ongoing evolution of plant–aphid interactions

Plant–aphid co-evolution is likely to be continuously shaping both the defensive phenotypes of plants and the coping mechanisms of aphids (Box 2). Multiple mechanisms act at the different stages of the plant–aphid interaction, and are likely to differ for species pairs at different stages of co-evolution. Where plants are 'ahead', conserved defences mediated by JA are probably an efficient means to control aphid infestation. However, due to their specific mode of feeding, aphids are predisposed to circumvent this defence, gaining the upper hand through the use of effectors to further manipulate the phytohormonal response of their host plants. In response, plants have evolved the capability to turn aphid salivary components into elicitors of defence responses, and, particularly in these interactions, phloem-sealing mechanisms emerge as key resistance traits. Finally, plant secondary metabolites seem to be important, especially at early stages of co-evolution, but aphid specializations and sequestration of these compounds may undermine the plant's efforts. Although there has been substantial progress in our understanding of these mechanisms in recent years, the conceptual and empirical links between physiological changes, defence induction and ecological impacts on aphid populations continues to emerge.

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Author contributions

T.Z. and A.A.A developed the project and wrote the paper.

Additional information

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Competing interests

The authors declare no competing financial interests.