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INVITED REVIEW

Mechanisms and ecological consequences of plant defence induction and suppression in herbivore communities

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• **Background** Plants are hotbeds for parasites such as arthropod herbivores, which acquire nutrients and energy from their hosts in order to grow and reproduce. Hence plants are selected to evolve resistance, which in turn selects for herbivores that can cope with this resistance. To preserve their fitness when attacked by herbivores, plants can employ complex strategies that include reallocation of resources and the production of defensive metabolites and structures. Plant defences can be either prefabricated or be produced only upon attack. Those that are ready-made are referred to as constitutive defences. Some constitutive defences are operational at any time while others require activation. Defences produced only when herbivores are present are referred to as induced defences. These can be established via *de novo* biosynthesis of defensive substances or via modifications of prefabricated substances and consequently these are active only when needed. Inducibility of defence may serve to save energy and to prevent self-intoxication but also implies that there is a delay in these defences becoming operational. Induced defences can be characterized by alterations in plant morphology and molecular chemistry and are associated with a decrease in herbivore performance. These alterations are set in motion by signals generated by herbivores. Finally, a subset of induced metabolites are released into the air as volatiles and function as a beacon for foraging natural enemies searching for prey, and this is referred to as induced indirect defence.
• **Scope** The objective of this review is to evaluate (1) which strategies plants have evolved to cope with herbivores and (2) which traits herbivores have evolved that enable them to counter these defences. The primary focus is on the induction and suppression of plant defences and the review outlines how the palette of traits that determine induction/suppression of, and resistance/susceptibility of herbivores to, plant defences can give rise to exploitative competition and facilitation within ecological communities "inhabiting" a plant.
• **Conclusions** Herbivores have evolved diverse strategies, which are not mutually exclusive, to decrease the negative effects of plant defences in order to maximize the conversion of plant material into offspring. Numerous adaptations have been found in herbivores, enabling them to dismantle or bypass defensive barriers, to avoid tissues with relatively high levels of defensive chemicals or to metabolize these chemicals once ingested. In addition, some herbivores interfere with the onset or completion of induced plant defences, resulting in the plant's resistance being partly or fully suppressed. The ability to suppress induced plant defences appears to occur across plant parasites from different kingdoms, including herbivorous arthropods, and there is remarkable diversity in suppression mechanisms. Suppression may strongly affect the structure of the food web, because the ability to suppress the activation of defences of a communal host may facilitate competitors, whereas the ability of a herbivore to cope with activated plant defences will not. Further characterization of the mechanisms and traits that give rise to suppression of plant defences will enable us to determine their role in shaping direct and indirect interactions in food webs and the extent to which these determine the coexistence and persistence of species.

Key words: Herbivory, plant defence, herbivore, jasmonate, salicylate, induction, suppression, manipulation, detoxification, sequestration, resistance, community interactions, facilitation, adaptation, plant–animal interaction.

INTRODUCTION

At a first glimpse, plants seem subject to the caprices of their environment, being constantly confronted with stresses such as drought, heat or ultraviolet radiation while frequently being challenged by pathogens or herbivores. However, these stresses have posed selection pressures on plants that have resulted in numerous adaptations, ranging from stress tolerance and resistance to the ability to manipulate their environment. Many of these adaptive traits have multiple functions. For example, plants are equipped with a waxy cuticle that prevents evaporation of water, but also forms an efficient barrier against phytopathogens (Eigenbrode and Espelie, 1995). Moreover, many

species have epidermal leaf hairs (trichomes), some of which carrying a special gland at the tip, and for which a broad diversity of functions have been reported. For example, they form structural barriers that hinder small arthropods in their mobility (Simmons and Gurr, 2005), but also guide light to the leaf surface of the leaf so that it can be used optimally for photosynthesis (Wagner *et al.*, 2004) while filtering out UV-A and -B (Karabourniotis and Bornman, 1999). Moreover, glandular hairs also secrete protective coatings that prevent fungal spores from germinating (Shepherd *et al.*, 2005), and contain glues and toxins that obstruct and intoxicate plant-surface-dwelling arthropods when ruptured (Glas *et al.*, 2012). Preformed structural barriers are referred to as 'constitutive defences' and they augment the so-called induced defences, which become operational only in response to an attack.

Defence against herbivores requires measures different from defence against pathogens. In general, phytopathogens are less mobile than herbivores, and migration across or through their host plant is often passive and occurs over relatively short distances. For example, plant-infecting viruses usually need plant proteins for tissue-to-tissue transport, plant-infecting fungi can only relocate by growing longer hyphae and plant-infecting flagellar bacteria can only travel short distances by taxis in fluid media. Hence, the infected host can attack such relatively immobile pathogens on the spot; at the site of infection, plants will often respond by producing structural reinforcements (e.g. cell wall thickening and callose deposition) or toxins (e.g. phytoalexins or alkaloids), or by initiating programmed local cell death (apoptosis) to isolate and possibly kill the pathogen (Dangl and Jones, 2001). Programmed cell death, orchestrated by the hypersensitive response, is highly efficient in preventing pathogens from spreading and is therefore one of the most common anti-parasite defence strategies found in nature. Herbivores, however, are not very susceptible to this isolate-and-kill strategy because they are mobile. Therefore, anti-herbivore defences generally come down to a go-away-or-die strategy or a slow-them-down strategy, and these two strategies share many physiological characteristics. In both instances, plants will mount a sequence of defence programmes that serve to interfere with herbivore growth and development on the one hand and to reallocate resources on the other, in order to delay growth of the herbivores into larger individuals, stages or populations, which consume more plant tissue. For example, well-known anti-herbivore defence proteins include proteinase inhibitors (PIs) and polyphenol oxidases (PPOs), both believed to interfere with digestive processes in the herbivore gut (Zhu-Salzman *et al.*, 2008). Hence, the simultaneous reallocation of resources may not only serve to rescue resources so that the plant can use them later for growth and reproduction, but may also serve to deprive the herbivore of food. This may be an effective strategy, especially when the plant is attacked by small herbivores or relatively immobile (immature) stages. Resource allocation is characterized by reallocation of nutritious carbon-containing (Schwachtje *et al.*, 2006; Babst *et al.*, 2008) and nitrogen-containing (Newingham *et al.*, 2007; Gomez *et al.*, 2012) substances to either the reproductive tissues or the storage organs, such as roots (Anten and Pierik, 2010). Hence, it can give rise to rapid flowering or to a period of dormancy (Stowe *et al.*, 2000) to regrow later, depending on the life-history characteristics of both plant and herbivore. Consequently,

herbivores that decide to stay on defended plants select plant tissues where defences are lower (Paschold *et al.*, 2007; Shroff *et al.*, 2008; Stork *et al.*, 2009) and/or increase their feeding intensity to gain sufficient biomass, thus compensating for the decreased efficiency of food conversion (Gomez *et al.*, 2012). Plants, in turn, often initiate systemic responses (Pieterse *et al.*, 2009) to decrease the chance that the herbivore will simply move to undefended tissues (Paschold *et al.*, 2007) and furthermore produce secondary metabolites to constrain compensatory feeding responses (Steppuhn and Baldwin, 2007).

The sequence of defence programmes executed by plants under attack appears not to be fully hard-wired, suggesting a certain degree of herbivore-specific tailoring by the plant, and while the early responses seem usually aimed at rescuing the attacked tissue, they may shift towards senescence and tissue death (reminiscent of the hypersensitive response) after a couple of days (Steinbauer *et al.*, 2014). It is unknown whether, how and when the plant decides to switch from the 'rescue' response to the 'scorched earth'-like tactic and whether this is characterized by a partial (Kahl *et al.*, 2000) or complete induced shut-down of local metabolic activity, e.g. to initiate senescence (Gross *et al.*, 2004). Moreover, it is unclear whether, and if so for how long, tissues that are being sacrificed are supported by photosynthates (Ferrieri *et al.*, 2013) and by defensive products from distal tissues (Nabity *et al.*, 2009). From the herbivore's point of view, resource depletion at the feeding site may represent a more difficult problem to deal with than toxins, because they cannot develop resistance to an absence of nutrients. However, some herbivores such as gall-makers have evolved abilities to manipulate plant resource flows and turn their feeding site into a sink for resources (Tooker *et al.*, 2008), but also leaf-cutters and trenchers (Dussourd and Denno, 1991) that prevent plants from transporting defence compounds to the feeding site and possibly also from transporting resources away from it.

Whereas the role of resource allocation remains under-studied, induced plant defences and their effects on herbivores have been analysed in great detail. In this review, we evaluate what determines the susceptibility of arthropod herbivores to plant defences and which herbivore traits counteract these defences. We focus on the induction and suppression of plant defences and outline how traits determining the susceptibility of parasites to plant defences can give rise to exploitative competition and facilitation in the ecological communities inhabiting the plant. First, we describe the diversity of herbivore feeding modes and life styles with which plants have to cope, and how plants detect the presence of herbivores through saliva and oral secretions.

HERBIVORE FEEDING AND PLANT DEFENCE STRATEGIES

Herbivore feeding styles

Early in evolution, herbivorous arthropods were mandibulate and chewed on vegetation (Budd and Teldford, 2009). After vascular plants had emerged, many different forms of feeding evolved, such as sap-sucking, leaf-mining, gall-forming and nectar-feeding (Grimaldi and Engel, 2005). Among the arthropods, many differently shaped mouthparts can be found which are suitable for chewing, siphoning, piercing–sucking and

sponging, or a combination of these. Chewing insects, such as many lepidopteran larvae, but also beetles and grasshoppers, have two mandibles, surrounded by sensory organs, which they use for crushing or cutting food while mixing it with salivary secretions before swallowing it. This type of feeding removes relatively large quantities of leaf material. Siphoning insects, typically adult lepidopterans, feed by sucking without piercing. Often, these insects have a long coiled proboscis that can be extended to reach food such as nectar. Siphoning itself does not cause much damage, although flowers may be damaged severely by an agile siphoning insect (Kessler *et al.*, 2008). The piercing–sucking arthropods feed by means of stylets, which are needle-shaped mouthparts. Stylets are common among insects and mites, although they probably evolved independently in these groups and possibly even more than once among mites (Manton and Harding, 1964; Caravas and Friedrich, 2010). The insect stylet is a single anatomical structure, often equipped with two separate channels, one for releasing saliva and the other for ingesting food (Miles, 1999). Mite mouthparts are composed of two or more stylets, which can be grouped together in pairs to form a piercing structure with an internal duct, which probably serves to release saliva (Ragusa and Tsolakis, 2000). While stylet-feeding insects such as whiteflies, psyllids and aphids usually feed from vascular tissue (Miles, 1999), herbivorous mites feed on the contents of mesophyll cells (Park and Lee, 2002). Insects such as thrips are usually also considered stylet–mesophyll feeders, but have an asymmetrical mouth cone, i.e. they have a single mandibular stylet, which is used for piercing and rasping plant tissue, after which their acinial stylet is inserted in the wound to take up food (punch-and-suck feeding) (Coll and Guershon, 2002; Kindt *et al.*, 2003). Finally, sponging insects have non-functional mandibula but an extended food channel that ends in a sponge-like labellum. They secrete saliva, which is dabbed onto their (solid) food to liquefy it, after which the labium channels the food to the oesophagus. This mode of feeding is typical of many species of flies (Vijaysegaran *et al.*, 1997). Thus, arthropods have diverse feeding styles and, especially among insects, different life stages can make use of distinct modes of feeding.

Different herbivore life stages feed differently

Different feeding styles accompany diverse life styles of herbivorous arthropods. These can differ considerably across stages of the life cycle, especially in holometabolous arthropods, but also in many hemimetabolous species. For example, adult thrips are winged and feed mostly from plant tissues. They deposit their eggs under the epidermis of the host plant and the wingless larvae that emerge from these eggs move across the plant surface to more protected areas and feed from pollen, nectar and/or leaf tissue until they develop into the nymphal stage (the third larval stage) and move into the soil to pupate (Broadbent *et al.*, 2003). Many other herbivores also spend part of their life cycle inside the host plant and/or the soil. Many lepidopteran and dipteran species have larval stages called leaf miners; they live inside a leaf or in needle tissue, where they feed and find protection. The adults of leaf miners are free-living but deposit their eggs underneath the leaf cuticle, where the larvae usually stay to graze the mesophyll until they

develop into pupae. This feeding behaviour causes the formation of distinct blotches or serpentine tunnels on plant leaves (Connor and Taverner, 1997). In addition, leaf-rollers are moth larvae that feed and pupate within the protection of rolled-up leaves; these leaves are either cut and folded or rolled up and wrapped by the larvae in their silk to create a shelter and feeding site (Gaston *et al.*, 1991). Leaf miners and leaf-rollers usually cause minor damage, i.e. mostly aesthetic, but can cause defoliation and fruit malformation when densities are too high (Witzgall *et al.*, 2008). Another group of herbivores are the stem and root borers, which deposit their eggs in stems or roots and whose larvae consume these tissues. They can be very damaging, not only because they often cause stunted growth, but also because they weaken the plant structure. Some species of borers have specialized in specific plant tissues such as fruits and seeds (frugivores) or bark (Mainali, 2014). Whereas leaf rollers physically create their shelter, galling insects induce somatic plant tissues to form domatia (Stone and Shonrogge, 2003). Many species of gall midges (Harris *et al.*, 2003) and gall mites (Van Leeuwen *et al.*, 2010*a*) manipulate the plant into producing tumorous outgrowths by mitosis to form hollow structures in which they spend a substantial part of their life cycle and which often serve as a feeding site, reminiscent of galls induced by root-knot nematodes (Caillaud *et al.*, 2008) or *Agrobacterium* (Zhu *et al.*, 2000). Many eriophyids do not induce protective cavities but other types of external malformations, like tufts or hairy outgrowths (erineum), in which they can seek shelter, while others induce deterioration rather than formation of plant leaf hairs (Karioti *et al.*, 2011; Van Houten *et al.*, 2013). Finally, most sap feeders, such as aphids, whiteflies and psyllids, spend their whole life cycle on the plant surface. However, the juvenile stages of these homopterans can be relatively immobile and sometimes cover themselves with honeydew for protection (VanDoorn *et al.*, 2015). Because phloem lacks essential amino acids, many homopteran species possess bacteriomes which harbour symbiotic bacteria that provide these and vitamins in return for nutrients (Schwemmler, 1989). Thus, plants encounter a wide range of different attackers and are therefore in need of defence systems that allow a degree of tailoring.

Plant defence theories

Understanding the mechanisms that plants have evolved to defend themselves and identification of the ecological drivers of this evolution have been major challenges during recent decades. Whereas research focusing on plant physiological aspects of these defences mostly worked from a scenario in which a single plant species is attacked by a single species, it has become increasingly clear that the diversity of ecological interactions within plant-inhabiting communities is an important determinant of the evolution of plant defence strategies. This notion gave rise to several theoretical frameworks revolving around the central dilemma that plant defences require resources that would otherwise be available for growth and reproduction (Mattson, 1980; Stamp, 2003). These frameworks can be classified into two groups: one of these attempts to explain the distribution of plant defences based on defensive function and plant life history, whereas the other group attempts to

explain this based on resource availability. The first theoretical framework is the *optimal defence theory*, which hypothesizes that plants with limited resources will defend different tissues differently, depending on the chance they will be attacked, the fitness value of the tissue and the cost of the defence. It was suggested that such a strategy will impose an 'evolutionary dilemma' leading to selection for defences at intermediate levels when plants are frequently attacked by generalists and specialists, because the latter will generally be more resistant to the plant's defences than the former (Zangerl and Rutledge, 1996). This theory is related to the *plant apparency hypothesis* (Feeny, 1976), which poses that plants that are attacked by a relatively large diversity of herbivores will need to display a larger diversity of defences than plants that are targeted by few species. It assumes that, compared with short-lived species, long-lived species are more likely to encounter generalists as well as specialists during their lives and hence have been under selection to display a broader range of defences. Third is the *carbon:nutrient balance hypothesis*, which hypothesizes that plant defences are constrained by nutrient variation in the environment and posits that the C:N ratio will dictate which secondary metabolites are synthesized (Bryant *et al*., 1983). This hypothesis predicts that changes in available nutrients will change the palette of defences. The fourth is the *resource availability hypothesis*, which posits that defence strategies are determined by the inherent growth rate of the plant, which is assumed to be constrained by resource availability (Coley *et al*., 1985). This theory implies that a trade-off between growth rate and defences will restrict species to particular habitats. Finally, the fifth theory is based on the *growth–differentiation balance hypothesis* and it subsumes elements of the previous hypotheses (Herms and Mattson, 1992). It posits that the ecological costs of the physiological trade-off between growth and secondary metabolism (defence) vary across environments. Since plants must protect their acquired resources, which are needed for growth in order to be able to compete for new resources, natural selection has shaped their secondary metabolism to be flexible and their life histories to vary across environments. Hence, this theory aims to explain patterns of phenotypic and genetic variation in secondary metabolism in response to environmental variation and resource gradients. It needs to be said that it has been difficult to generate testable hypotheses from these defence theories, not only because the magnitudes of the costs and benefits of defences have proved to be very difficult to measure, but also since they often fail both to distinguish among evolutionary, ecological and physiological levels of analysis and to clearly distinguish between genetic and environmental influences.

Plant defence strategies

Plant defences are often divided into three basal strategies: *deterrence* (antixenosis), *resistance* (antibiosis) and *tolerance* (a compensation strategy to reduce the detrimental effects of herbivory). Deterrence traits are usually constitutively expressed and can emanate from colours, odours or textures (such as hairs) that demotivate a herbivore from feeding on the plant, or from the absence of feeding stimuli that otherwise would stimulate the attacker. Resistance traits are those that can injure or kill a herbivore or slow its development and reproduction.

Finally, tolerance comes from those traits that do not primarily serve to negatively interact with the herbivore, but to compensate for damage through changes in assimilation rate, compensatory growth, phenological shifts, resource allocation or morphological changes. These three strategies are not mutually exclusive and can overlap mechanistically and functionally. Hence, it will often be difficult to tell these three strategies apart (Stout, 2013) and it is doubtful whether deterrence as a stand-alone defence strategy will be evolutionarily stable since it offers ample opportunities for herbivores to adapt.

Constitutive plant defences

Plants cannot simply accumulate all the defences that have emerged during the course of evolution within a 'super-genotype' because defensive structures, compounds or processes such as the inducible defences (Baldwin, 1998) cost energy to form and maintain. Hence, only those defences for which selection pressure has been constant and strong enough have been retained. Moreover, the optimal defence theory predicts that plants with limited resources are selected to arrange the relatively costly and less costly defences across tissues based on the fitness value of these tissues. Moreover, assuming that the induced defences are overall less costly than constitutive defences, this theory predicts that tissues with a higher probability of being attacked will rely more on constitutive defences, whereas tissues with a lower probability of being attacked will depend more on induced defences. Indeed, the reproductive parts of wild parsnip (*Pastinaca sativa*) had the highest levels of constitutive furanocoumarins with low inducibility and had the highest probability of being attacked, while in the roots, which were less frequently attacked, constitutive levels were relatively low but highly inducible (Zangerl and Rutledge, 1996).

Discriminating experimentally between constitutive and induced defences is often not easy since there can be considerable overlap. For example, the size and density of physical barriers such as spines and plant hairs (Glas *et al*., 2012), which are commonly considered to function as constitutive defences, can also be increased by induction (Traw and Dawson, 2002). For example, formation of more and longer thorns was induced by giraffes feeding on acacia (*Acacia seyal*) trees (Milewski *et al*., 1991). Moreover, not only are leaf hairs mechanical barriers (Pott *et al*., 2012), but glandular trichomes (Tissier, 2012) are important production sites of a wide variety of constitutive and induced secondary metabolites with defensive functions, including terpenoids, phenylpropenes, flavonoids, methylketones, acyl sugars and defensive proteins, and some of these compounds are inducible. For instance, the emission of terpenoids (Van Schie *et al*., 2007) and the production of acyl sugars and defensive proteins (Hare and Walling, 2006) can be induced in trichomes by treating plants with methyl jasmonate, a compound that activates the jasmonate (JA) signal transduction cascade, also induced by herbivores. Even mere contact of insects with a plant was found to suffice to induce the expression of PIs in glandular trichomes (Peiffer *et al*., 2009). The contents of glandular trichomes, such as sticky acyl sugars and polyphenols, can be excreted or released after they are ruptured by insect movement, and cause entrapment of small herbivores,

often followed by death (Simmons *et al.*, 2004). Trichomes of tobacco (*Nicotiana tabacum*) also produce defensive proteins that are secreted to the leaf surface and inhibit germination of oomycete spores (Shepherd *et al.*, 2005). Conversely, some pathogens, like *Pseudomonas syringae* on tomato, have adapted to use trichomes as their habitat (Schneider and Grogan, 1977), and damaged trichomes may be used as an entry point by these bacteria (Huang, 1986). Thus, trichomes are important components of both the constitutive and the inducible defence system, and trichome secretions function to hinder herbivore feeding and the germination of fungal spores. Other forms of constitutive defence involve the plant's waxy cuticle; this primarily serves to prevent the evaporation of water (Buschhaus and Jetter, 2012), but wax morphology and chemistry contribute to a plant's resistance by restraining herbivore foraging behaviour (Eigenbrode and Shelton, 1990). Also, leaf toughness, which includes cell wall lignification, is known to deter herbivore feeding (Choong, 1996) and is positively correlated with resistance to pathogens (Bhuiyan *et al.*, 2009).

Induced plant defence

Induced defences are often subdivided into direct and indirect defences. *Direct defence* includes the activation or production of antifeedants, such as toxins and inhibitors of digestion, which negatively affect the growth and/or survival of herbivores (Howe and Jander, 2008). The existence of induced defences has been known for more than 100 years (early work is reviewed by Chester, 1933). A well-known example of a herbivore-induced plant defence is increased PI gene expression and enzyme activity. Although most plant PIs have regulatory roles in the plant's endogenous protein metabolism, the herbivore-induced PIs inhibit proteases in the gut of herbivores, thereby decreasing the plant's palatability and increasing its resistance (Hartl *et al.*, 2010). Defences may also be induced in the phloem (Will *et al.*, 2013). For instance, feeding on rice by the brown planthopper (*Nilaparvata lugens*) induces the deposition of callose on the plant's sieve plates to block further transport of sap through the attacked phloem tissues (Hao *et al.*, 2008). Another mechanism of such sieve tube occlusion is dependent on large protein bodies called forisomes, which can seal off sieve elements in a Ca^{2+}-dependent manner (Furch *et al.*, 2007) to obstruct herbivore feeding.

Indirect defence refers to plant traits that enhance attraction or arrestment of natural enemies of the herbivore, such as predators and parasitoids (Sabelis *et al.*, 2001). Often, this type of defence is inducible. That natural enemies of herbivores use plant odours for locating prey has been suggested several times (reviewed by Vinson, 1976), and Dicke and Sabelis (1988) outlined a framework for the mode of action and the evolution of indirect defence strategies, mediated by so-called infochemicals, which forms the basis for our current view of the phenomenon. Since then, induced indirect defences have been reported for many plant species under laboratory conditions, including *Arabidopsis* (van Poecke *et al.*, 2001), cotton (De Moraes *et al.*, 1998), tomato (Kant *et al.*, 2004) and maize (Schnee *et al.*, 2006). In 1999, Thaler showed that indirect defences can act in the field while, in 2001, Kessler and Baldwin showed that plant volatiles can establish indirect

defences under natural conditions. They supplemented *Nicotiana attenuata* plants with synthetic volatiles and some of these increased the natural predation of herbivore eggs and repelled adult moths. In a later study with transgenic plants that were silenced for genes involved in volatile production, the same group showed that indirect defences can actually promote a plant's fitness under natural conditions (Schuman *et al.*, 2012). Moreover, it was found that hyperparasitoids also respond to herbivore-induced plant volatiles; volatiles released by plants infested with parasitized caterpillars attracted more hyperparasitoids than volatiles emitted by plants infested with healthy caterpillars (Poelman *et al.*, 2012). Indirect defence is known to occur below ground as well. A well-known example is the release of the volatile *β*-caryophyllene by maize roots into the soil when attacked by larvae of the beetle *Diabrotica virgifera virgifera*; this compound was shown to function as an attractant for entomopathogenic nematodes that attack the beetle larvae (Rasmann *et al.*, 2005). Finally, restoring this function in maize varieties deficient in the release of *β*-caryophyllene from roots also increased attraction of the nematodes (Degenhardt *et al.*, 2009).

Volatiles are not the only means by which plants can increase the abundance of natural enemies in their vicinity. Natural enemies can be arrested by providing them with food, e.g. extrafloral nectar (Pemberton and Lee, 1996) or food bodies (Fischer *et al.*, 2002). Also, dead insects entrapped on sticky plants were shown to attract predatory insects such that overall herbivore damage decreased and fruit production increased (Krimmel and Pearse, 2013). Finally, an alternative means by which plants establish indirect defence is to provide shelter (domatia) such as cavities or tufts of hair, for small natural enemies, which these can use to moult and/or to protect their eggs (Walter, 1996).

HERBIVORE DIGESTION, SALIVA AND REGURGITATION

A herbivore's host-plant range is closely linked to its digestive physiology (Pearse *et al.*, 2013). The midgut and salivary glands are important organs for establishing interaction between a phytophagous insect and its host plant (Shukle *et al.*, 2010). For example, the ability of insects to tolerate ingested tannins, which are among the most abundant secondary plant metabolites, is determined by a variety of biochemical and physiological features of their midgut (Fig. 1). These include the release of surfactants and antioxidants, the maintenance of a high pH and the formation of a protective peritrophic membrane envelope lining the midgut such that it functions as a barrier (Barbehenn and Constabel, 2011). The more a herbivore can maintain flexible and diverse digestion and detoxification pathways, the more host plants may eventually become available to it, as is the case for the spider mite *Tetranychus urticae*, which has been found to live on well over 1000 plant species (Dermauw *et al.*, 2013a).

Herbivore symbionts to aid digestion

In many cases, herbivores rely on the help of microorganisms to overcome some of the problems that come with feeding on plant tissues. Phloem is a troublesome food source for insects,
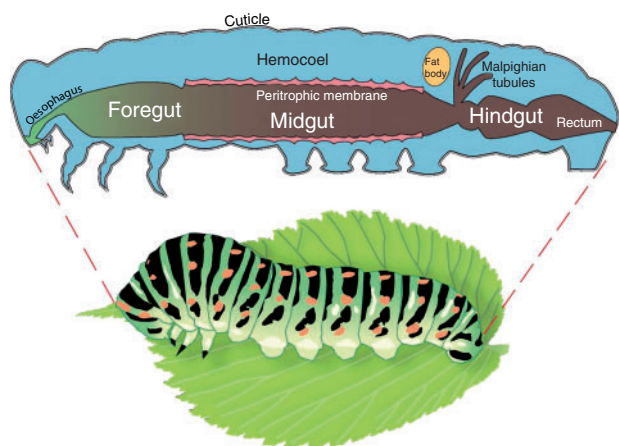
Fig. 1. The herbivore digestive system. The insect digestive system has two openings, the mouth and the anus. The digestive tract has three sections: the foregut, the midgut (stomach) and the hindgut. Food is stored in the foregut and mixed with saliva to help digestion; sometimes this mixture is regurgitated, but most digestion takes place in the midgut. Nutrients are absorbed in the midgut and the hindgut. Nitrogen-containing waste from metabolic processes is excreted from the Malpighian tubules and the remaining waste from the anus. The midgut is lined with a semipermeable membrane, called the peritrophic membrane, which is composed of proteins and chitin and allows the passage of liquids while blocking the passage of solid food particles and microorganisms.

not only because it lacks essential components, but also because its sugar content and osmotic pressure are high. Many phloem feeders have adapted tolerance to this and display sucrose trans-glucosidase activity in their gut, which can transform the excess ingested sugar into long-chain oligosaccharides, which in turn can be excreted as honeydew (Douglas, 2006). Especially homopteran insects such as aphids, which live solely on plant sap (i.e. predominantly phloem), have evolved associations with microbes to compensate for the deficiencies in this food source. Phloem is not only rich in sugars but also lacks several amino acids and vitamins. Hence, insects such as aphids have evolved intimate endosymbiotic relationships with bacteria, which supply them with essential amino acids and vitamins and receive other nutrients in return (Engel and Moran, 2013). These bacteria seldomly reside within the gut, but instead are usually found in specialized cells called bacteriocytes (Schwemmler, 1989).

Chewing insects in particular have to deal with the poorly digestible cell wall components that they ingest. The major structural components of the primary plant cell wall are cellulose, hemicellulose and pectin, forming a complex and organized structure, and proper degradation of these components requires a range of enzymes, including cellulases, hemicellulases and pectinases (Cosgrove, 2005; Vilanova *et al.*, 2012). Microbial symbionts present in the digestive tract of insects are known to contribute significantly to the digestion of plant cell wall components. The prevailing view was that herbivorous insects are completely dependent on these symbionts, but it was found that some herbivorous insects also produce their own endogenous plant cell-wall-degrading enzymes (Caldéron-Cortés *et al.*, 2012). These enzymes are usually secreted by the epithelial cells of the insect's midgut and move forward to the foregut or are secreted by the salivary glands (Terra and Ferreira, 1994). In some cases, these endogenous cell-wall-degrading enzymes have been acquired by insects from microbes by horizontal gene transfer. Xylanases, for example, were transferred from gammaproteobacteria to the mustard leaf beetle *Phaedon cochleariae* or its ancestor (Pauchet and Heckel, 2013). Other cases of horizontal gene transfer that enable arthropods to feed on plants or plant tissues with an unfavourable nutrient composition show that such transfers may be important drivers of herbivore evolution. Examples of these have been found for the spider mite *T. urticae* (Grbić *et al.*, 2011; Wybouw *et al.*, 2012) and the coffee berry borer beetle *Hypothenemus hampei* (Acuña *et al.*, 2012).

The importance of amino acid availability

Herbivorous animals have a lower tissue carbon-to-nitrogen ratio than plants. Hence, they must eat an excess of carbon-rich plant material to acquire sufficient nitrogen, making nitrogen one of the central determinants of herbivore foraging behaviour and population growth (Fagan *et al.*, 2002). Proteins (i.e. amino acids) are the main macronutrients containing nitrogen and are commonly considered to strongly affect the growth rates of arthropod herbivores (Mattson, 1980). The nutritional value of a host plant is not solely based on its protein quantity, as protein quality is hypothesized to be equally important. Protein quality depends on the essential amino acid composition and can be quantified based on which essential amino acids have the lowest abundance relative to the composition required by a herbivore (Barbehenn *et al.*, 2013).

Dietary protein is broken down into peptides and amino acids in the midgut region of the insect digestive tract, a process catalysed by the abundant gut proteases. There are two types of proteases: proteinases, which cleave protein chains at specific peptide bonds, and exopeptidases, which remove amino acids from the C- or N-terminus of a protein (Jongsma and Bolter, 1997). Such free amino acids, but also di- or tripeptides, are subsequently absorbed via transporter proteins in the midgut. Plants, in turn, have evolved mechanisms to disturb the uptake of amino acids by herbivores as an integral part of their defence system (Chen *et al.*, 2005; Yang *et al.*, 2013). For example, essential amino acids in the insect gut can be bound to quinones, highly reactive molecules generated by the ingested plant material. This compromises the plant's nutritional value, thereby reducing insect performance (Felton, 2005). In addition, the digestion of dietary plant proteins by the insect's gut proteases may be hampered by ingested plant PIs, which rapidly accumulate in herbivore-damaged plant tissues. In turn, some herbivores have evolved novel proteases that are largely insensitive to plant PIs or have adapted by increasing their overall protease gene expression levels and thereby the total amount of gut protease activity (Jongsma and Bolter, 1997).

Herbivore saliva and regurgitant

Herbivore oral secretions play an important role in plant–herbivore interactions. Oral secretions are a mixture of secretions from the labial and mandibular salivary glands and regurgitant (Vadassery *et al.*, 2012). Regurgitation is the expulsion of material from the herbivore's oesophagus and this gut reflux is usually composed of partially digested food and gut juices. Some species regurgitate to digest food, e.g. some species have life

stages that cannot chew and that repeatedly draw regurgitated liquid in and out of their proboscis, whereas for others regurgitation may have evolved as a defence mechanism against natural enemies (Rhainds *et al.*, 2011). Chewing caterpillars may regurgitate during feeding (Vadassery *et al.*, 2012), but their tendency to do so differs across species of herbivore and host plant. Regurgitant can be collected from caterpillars by gently squeezing them (Peiffer and Felton, 2009) and regurgitant of several herbivore species has been shown to contain components that alter the plant defence response when applied to wounded tissues. Digestive enzymes in regurgitant may emanate from the gut or from the saliva (Afshar *et al.*, 2013; Chen *et al.*, 2013) and some constituents of the saliva of caterpillars (Musser *et al.*, 2002) and aphids (Rodriguez and Bos, 2012), were found to modulate the host's defence responses.

Elicitors of defences from herbivore saliva and regurgitant

An elicitor of plant defences can be any substance that provokes a specific defence-related response in a host plant after exposure to it. Four groups of elicitors have been distinguished in the literature: plant-derived (endogenous) elicitors, herbivore-derived elicitors, conjugates of these two, and synthetic elicitors.

Plant-derived elicitors are molecules produced or released upon injury or infection and are responsible for induction or amplification of the plant's defence responses (either local or systemic) against the attacking organism. Such molecules can include cell wall fragments, phytohormones, reactive oxygen species (ROS) and peptides (Albert, 2013; Gozzo and Faoro, 2013). Many elicitors act in concert or in sequence with other elicitors or signalling molecules (Kessler and Baldwin, 2002). Six groups of plant-borne peptide elicitors that play a role in plant–herbivore interactions have been identified: (1) peptides derived from preproteins such as systemin; (2) hydroxyproline-rich systemin (HypSys); (3) a group referred to as 'plant elicitor peptides' (Peps); (4) cryptic peptides, derived from 'preproteins' that have their own (unrelated) primary functions, such as inceptin, which is derived from a chloroplastic ATP synthase; (5) SubPep, which is derived from a subtilisin-like protease (Yamaguchi and Huffaker, 2011); and (6) CAPE1 (CAP-derived peptide 1), which is derived from PR-1 (Pathogenesis-related protein 1) (Chen *et al.*, 2014). All these plant peptides were shown to act upstream of a subset of defence responses and to modulate these responses. Receptors for the Peps have been identified (Yamaguchi *et al.*, 2010). Inceptins are disulphide-bridged peptides, originally described from cowpea (*Vigna unguiculata*), and are derived from the chloroplast ATP synthase γ subunit following digestion by proteolytic enzymes in the gut of the fall armyworm (*Spodoptera frugiperda*) larva. Hence, inceptin is considered a plant-derived elicitor and it induces and amplifies local and systemic defence responses (Yamaguchi *et al.*, 2011). Inceptin recognition shows that plants have evolved the means to sense insects not only directly via their secretions or movements, but also indirectly by monitoring the emergence of catabolic products indicative of an insect that successfully feeds and digests (Schmelz *et al.*, 2009). Elicitor activity of inceptins seems to be specific to legumes in the genera *Phaseolus* and *Vigna* (Yamaguchi *et al.*, 2011).

Interestingly, the inceptin-related peptide of the velvet bean caterpillar, *Anticarsia gemmatalis*, has a C-terminal truncation and does not induce but rather antagonizes defences (Schmelz *et al.*, 2012). This indicates that plants and herbivores may be involved in an arms race reminiscent of plants and pathogens. The elicitor 2-hydroxyoctadecatrienoic acid (2-HOT) is generated during *Manduca sexta* feeding on *N. attenuata* by a dioxygenase from the plants using plant linolenic acid as a substrate. However, this conversion occurs mainly locally at the feeding site, possibly because the dioxygenase has a high pH optimum and may increase its activity under the alkaline conditions at the feeding sites or potentially in the insect's mouth during chewing and regurgitation (Gaquerel *et al.*, 2009). Also, plant hormones, such as the linolenic acid-derived jasmonic acid (JA) and the phenolic salicylic acid (SA), elicit specific defences when applied to plants as pure compounds. Moreover, plants that are attacked by herbivores release distinct volatile blends that can contain methyl jasmonate (the volatile form of JA) and methyl salicylate (the volatile form of SA), which, together with a small subset of additional volatiles, are potent elicitors of defences in systemic uninduced leaves of the same plant or in nearby plants (Kessler and Baldwin, 2002).

It may not come as a surprise that pure (synthetic) plant defence signalling molecules, or substances closely related to them, elicit such defences when applied manually to uninduced plants. However, molecules derived directly from herbivores have also been found to elicit specific defence-related processes. Such elicitors can originate from organs associated with feeding (gut, salivary glands, etc.) and can be present in saliva, regurgitant or other secretions such as honeydew. In lepidopterans, saliva is proposed to be a more important source of elicitors than regurgitant, whereas the opposite may be true for Coleoptera (Kim *et al.*, 2011). Herbivore saliva has been studied in detail (Miles, 1999). Some lepidopteran larvae also possess a ventral eversible gland, whose secretions have been associated with silk strengthening, defences against predators and the production of anti-aggregation pheromones (Zebelo and Maffei, 2012). The secretions may, however, also interact with the plant host because the tip of the everted gland can reach the mouthparts of the larvae, allowing them to mix with the oral secretions. Research by Zebelo and Maffei (2012) suggests that the ventral eversible gland of *Spodoptera littoralis* might contain elicitors that are able to trigger early plant defences in *Arabidopsis*. Phloem-feeding herbivores deposit secretions onto the leaf surface when attaching their stylet and coat the stylet trajectory with a protective sheet. Subsequently, they inject saliva in a pierced vascular bundle (Hogenhout and Bos, 2011). Two salivary proteins (Mp10 and Mp42) of the green peach aphid *Myzus persicae* were found to act as elicitors in *Nicotiana benthamiana* and to reduce aphid fecundity when expressed in plants. Furthermore, Mp10 overexpression in *N. benthamiana* resulted in chlorosis and local cell death. The involvement of the plant chaperone protein SGT1 suggest that aphid elicitor recognition is mediated by proteins encoded by R (Resistance) genes: these are sensory proteins that are known to recognize pathogen elicitors. Induction of chlorosis was not observed in tomato, implying that the Mp10 response may be specific for *N. benthamiana*. Mp10 was also able to suppress the ROS response induced by the well-known bacterial elicitor flagellin 22, but not by the putative insect elicitor chitin

(Bos *et al*., 2010). Hence, mechanisms of recognition and signal transduction of aphid salivary proteins are unclear at this stage (Hogenhout and Bos, 2011).

The interface between the plant and the feeding insect will often contain a mixture of substances of both herbivore and plant origin. Sometimes plants specifically recognize substances that originate from the plant but only after they are processed by the herbivore, reminiscent of inceptin. Some lepidopteran larvae harbour fatty acid amino acid conjugates (FACs) in their digestive system. The fatty acid moiety of these conjugates originates in the plant (Felton and Tumlinson, 2008) and is conjugated to amino acids, such as glutamine, in the insect gut. These conjugates may play a primary role in the regulation of glutamine supply for nitrogen assimilation (Yoshinaga *et al*., 2010). The oral secretions and regurgitant of caterpillar larvae may also contain FACs, which can act as elicitors of specific defence responses after recognition by the host plant (Bonaventure *et al*., 2011). The FACs have a broad taxonomic distribution in insects (Felton and Tumlinson, 2008). The first FAC found was named volicitin [*N*-(17-hydroxylinolenoyl)-L-glutamine]. Together with several related substances, such as *N*-linolenoyl-L-glutamine and *N*-linolenoyl-glutamic acid, it constitutes a significant fraction of the elicitor pool of lepidopteran larvae, responsible for the production and release of induced plant volatiles (Alborn *et al*., 1997). Apart from inducing volatiles, FACs are also known to induce an increase in activity of the salicylic-induced protein kinase and the wound-induced protein kinase in *N. attenuata* leaves when attacked by caterpillars or treated with oral secretions (Wu *et al*., 2007). Other elicitors of plant volatiles with a basal fatty acid moiety are caeliferins. These are disulphooxy fatty acids originally isolated from *Schistocerca americana* regurgitant and were found in grasshoppers of the suborder Caelifera. These compounds elicit defence responses in corn (Alborn *et al*., 2007) and *Arabidopsis* (Schmelz *et al*., 2009). Furthermore, synthetic caeliferin A16:0 was shown to strongly induce ethylene production in *Arabidopsis* (O'Doherty *et al*., 2011). However, application of synthetic caeliferin A16:0 to puncture wounds in *Arabidopsis* did not induce any of the responses observed on treatment with grasshopper oral secretions (Schäfer *et al*., 2011). Regurgitant of *Pieris brassicae* caterpillars also contains the enzyme *β*-glucosidase. This molecule is the first reported herbivore-associated elicitor and triggers the same emission of volatiles in cabbage plants as that induced by feeding caterpillars (Mattiacci *et al*., 1995). Another well-known enzyme from caterpillar saliva with elicitor properties is glucose oxidase (GOX). In some cases, however, GOX was found to act neutrally or in favour of the herbivore (Tian *et al*., 2012; Musser *et al*., 2002).

Together, these elicitors activate defensive responses of the host plant. Hence, they are referred to as herbivore-associated molecular patterns (HAMPs), a term that covers all herbivore-derived signalling compounds that might come into contact with host plants during any stage of their life cycle and elicit defence reactions (Felton and Tumlinson, 2008). The HAMPs are presumably recognized by pattern recognition receptors that evolved to recognize conserved, generally occurring pathogen- and herbivore-derived molecules or motifs, but so far no specific HAMP receptors have been identified (Erb *et al*., 2012), with the exception of a putative volicitin receptor (Truitt *et al*., 2004). However, the additional perception of specific individual herbivore-associated elicitors may allow the plant to distinguish the type of attacking herbivore (Poelman *et al*., 2011).

Non-oral elicitors of defences

HAMPs also include those elicitors that do not directly result from feeding activities. These include the secretions from the ventral eversible gland of *S. littoralis*, but the fluids secreted by female pea weevils (*Bruchus pisorum*), which are used to attach the eggs to the plant surface and can also contain substances perceived by the plant. These fluids contain mono- and bis-(3-hydroxypropanoate) esters of long chain α,ω-diols ('bruchins') and increase cell division and induce neoplasm formation in several legume hosts. Also, benzyl cyanides from the oviposition fluids of mated female *Pieris rapae* can elicit transcriptional changes in defence-related genes (Fatouros *et al*., 2008). The presence of feeding herbivores can also be detected by the plant through components present in the excreted honeydew (VanDoorn *et al*., 2015). In addition to HAMPs, non-molecular signals can also alert a host plant. For example, herbivore larvae can betray their presence to plants by their crawling, which stimulates the synthesis of 4-aminobutyrate (GABA), while imprints of their footsteps lead to increases in chlorophyll fluorescence or superoxide production. This possibly represents early defence signalling events (Hall *et al*., 2004). Plant trichomes can also operate as sensors of herbivore movements after being touched (Peiffer *et al*., 2009).

REGULATION OF PLANT DEFENCES AGAINST HERBIVORES

Detection of herbivores

Plants recognize herbivores by their molecular patterns or their elicitors. It is hypothesized that polyphosphoinositides generated at the plasma membrane play an important role as second messengers, just as they do during pathogenesis (Munnik and Nielsen, 2011). The most rapid measured responses are ion (e.g. Ca^{2+} and K^+) fluxes across the plasma membrane, followed by changes in the plasma membrane potential. Subsequently, a protein kinase cascade can activate the production of ROS such as hydrogen peroxide by activating an NADPH-dependent oxidase. Hydrogen peroxide can have a direct effect on herbivores or enter the cell, thus changing its redox status. The rapid increase in cytosolic Ca^{2+} can also give rise to increased nitric oxide-mediated processes that precede the upregulation of JA levels (Zebelo and Maffei, 2015). These responses occur not only locally, but also in unattacked neighbouring cells and in distal tissues. Herbivory, the application of oral secretions to wounded leaves and aphid probing have been shown to give rise to membrane depolarization due to an electrochemical gradient between the interior and the exterior of the attacked plants cells. This membrane depolarization can travel with a speed of up to $40 \, \text{cm s}^{-1}$ through the entire plant and mutant plants with attenuated wound-induced surface potential changes exhibit a reduced JA response in distal leaves (Mousavi *et al*., 2013). Moreover, ablation of the ventral

eversible gland of *S. littoralis* reduced this depolarization as well as the Ca^{2+} and hydrogen peroxide bursts and downstream defence responses (Zebelo and Maffei, 2012). The relationship between Ca^{2+} levels, ion channel activity and the oxidative burst is correlative but may depend more strongly on K^+ than on Ca^{2+}. Additional to these rapid electric signals (Mousavi *et al.*, 2013), slower chemical signals are also transmitted to distal tissues, either via the vascular tissues (Schilmiller and Howe, 2005) or via the air (Sugimoto *et al.*, 2014) (Fig. 2).

Defence-regulating plant hormones

Three phytohormones play a primary role in regulating defence responses (Pieterse *et al.*, 2009): JA (Wasternack and Hause, 2013), SA (Vlot *et al.*, 2009) and the volatile ethylene (Adie *et al.*, 2007). The central roles of JA and SA are substantiated by the fact that biosynthesis mutants are hypersensitive to a wide range of attackers. Several other phytohormones are known to play a secondary role in plant defence by modulating it, including abscisic acid (ABA) (Dinh *et al.*, 2013), auxin (Kazan and Manners, 2009), cytokinin (Choi *et al.*, 2011), gibberellic acid (GA) (Yang *et al.*, 2012) and brassinosteroids (Nakashita *et al.*, 2003) and possibly strigolactones (Torres-Vera *et al.*, 2013). In concert with these hormones, a small set of signalling peptides, such as systemin (Ryan, 2000) and the Peps from *Arabidopsis* (Huffaker *et al.*, 2006), are also involved in orchestrating plant defences. The peptide systemin of tomato, *Solanum lycopersicum* (Pearce *et al.*, 1991), functions upstream of JA biosynthesis and may facilitate priming of the plant's JA response (Kandoth *et al.*, 2007). Moreover, homologues from different plant species were found to have different (non-defence related) functions (Schmidt and Baldwin, 2006). In contrast, some of the Peps appeared to have similar functions across different plant species, because Pep3 from maize (*Zea mays*) induces accumulation of JA and ethylene as well as their downstream responses, including the emission of volatiles (Huffaker *et al.*, 2013).

Jasmonate as a regulator of plant defences against herbivores

Jasmonic acid regulates the core defences of dicots against herbivorous arthropods (Howe and Jander, 2008) and necrotrophic pathogens (Glazebrook, 2005). The biosynthesis of JA was elucidated by Vick and Zimmermann (1984) and seems quite conserved across species. In tomato, JA biosynthesis was shown to take place in the chloroplast and peroxisomes of the phloem companion cells (Howe, 2004). Briefly, the first step in JA biosynthesis comprises the formation of α-linolenic acid, which is released from the galactolipids of chloroplast membranes by the action of one or more phospholipases, although it
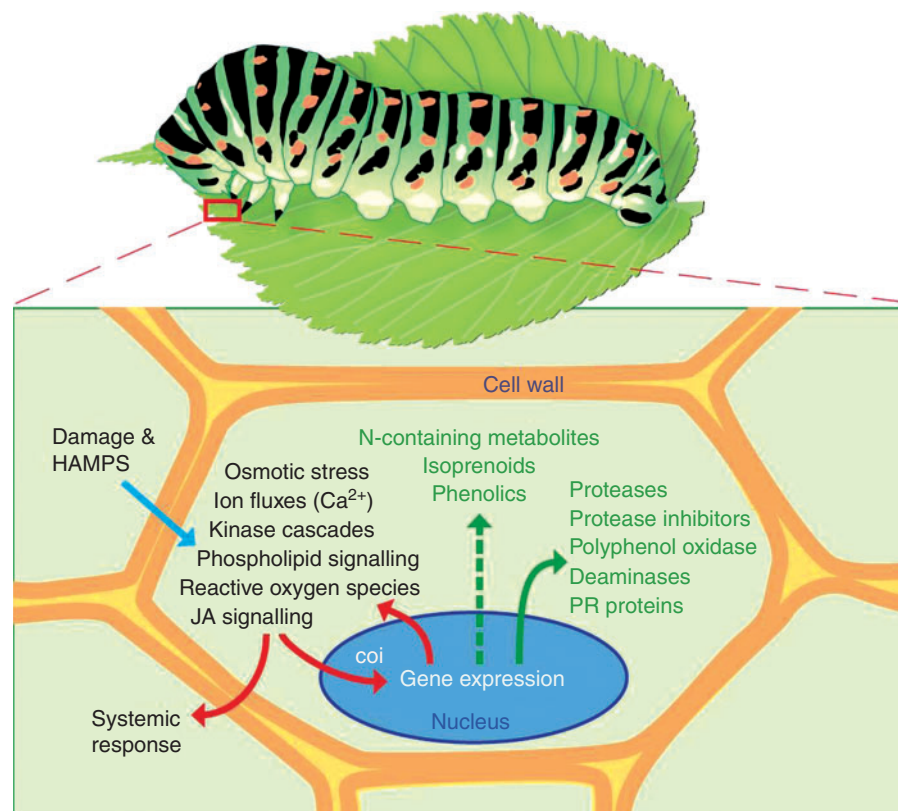
FIG. 2. The plant defence response. Mechanical damage and exposure to oral secretions from herbivores lead to osmotic stress, cause ion fluxes and elicit signalling cascades leading to rapid accumulation of jasmonates (JAs). Jasmonoyl isoleucine diffuses into the nucleus, where it associates with a protein complex including its receptor encoded by the *coi* (coronatine insensitive) gene. Binding to this complex initiates degradation of transcriptional suppressor proteins, thereby stimulating defence gene expression. Jasmonate also stimulates increased accumulation of secondary metabolites (phytotoxins). Together, these changes make plant tissues less palatable. Upstream processes are shown in black, the defence substances produced by the plant in green.

is still unclear which roles the different lipase candidates play in α-linolenic acid formation during different plant–herbivore interactions (Wasternack and Hause, 2013). Subsequently, α-linolenic acid is converted via three enzymatic steps into 12-oxophytodienoic acid (OPDA), and dinorOPDA is also formed in *Arabidopsis* (Stintzi *et al.*, 2001). OPDA is then imported into the peroxisomes, where it is converted by OPDA reductase OPR3, followed by three cycles of β-oxidation into JA. Finally, JA diffuses into the cytosol, after which a range of JA conjugates and derivatives are formed (Yan *et al.*, 2013), among which is jasmonoyl isoleucine (JA-Ile), which is the main bioactive form of JA (Fonseca *et al.*, 2009). Although JA-Ile has a well-established role in regulating defence gene expression, OPDA may also function as such independently (Taki *et al.*, 2005).

Before induction, JA-dependent responses are constitutively blocked due to repressor proteins, called jasmonate ZIM domain (JAZ) proteins, bound to transcription factors that otherwise would promote defence gene expression (Thines *et al.*, 2007), including several MYC (Chini *et al.*, 2007; Fernández-Calvo *et al.*, 2011) and MYB (Qi *et al.*, 2011) transcription factors. The JAZ proteins have two types of functional domain: ZIM domains and Jas domains. The ZIM domains establish homo- or heterodimerization among individual JAZ proteins but also interactions with additional (co)-suppressors, such as TOPLESS and NINJA (Pauwels *et al.*, 2010). The Jas domains establish the interaction with the transcription factors, which prevents these from functioning. Transcriptional (de)repression also regulates the synergistic action of JA and ethylene since JAZ proteins repress not only the transcriptional activity of the ethylene-stabilized transcription factors EIN3 and EIL1 but also interfere with their transcription by promoting histone acetylation. However, induced JA-Ile interrupts the interaction between the JAZ proteins and EIN3/EIL1 to enhance their transcriptional activity (Zhu *et al.*, 2011) by promoting the ubiquitination–degradation of JAZ proteins via a protein complex called the SCF^{COI1} complex. Hence, activation of JA-responsive genes largely is obtained by derepression of transcription.

In *Arabidopsis*, the JA responses downstream of SCF^{COI1} are executed via two different branches: one branch that is dependent on MYC transcription factors (referred to as the MYC branch) (Dombrecht *et al.*, 2007) and the other depending on transcription factors like ETHYLENE RESPONSE FACTOR1 (ERF-1) and OCTADECANOID-RESPONSIVE ARABIDOPSIS 59 (ORA59), which is referred to as the ERF/ORA59 branch (Pré *et al.*, 2008; Zhu *et al.*, 2011). These branches are known to antagonize each other: the MYC2 transcription factor suppresses expression of ERF-dependent JA-responsive genes and vice versa (Lorenzo *et al.*, 2004; Dombrecht *et al.*, 2007). The levels of JA in *Arabidopsis* leaves can start to rise within 30 s after wounding (Glauser *et al.*, 2009). The burst is transient: levels decrease again after a few hours (Reymond *et al.*, 2000; Schittko *et al.*, 2000); however, two consecutive bursts have been observed in *S. nigrum* (VanDoorn *et al.*, 2011). In *N. attenuata*, large veins can constrain the spatial spread of JA bursts, and while a second elicitation can suppress a (second) burst, a third elicitation can induce it again (Stork *et al.*, 2009). Subsequently, induction of JA accumulation can also occur in distal leaves (Glauser *et al.*,

2008). Spatiotemporal variability in JA accumulation may be a defensive tactic by itself because it makes it difficult for herbivores to anticipate which tissues are defended poorly and which strongly (Stork *et al.*, 2009). Finally, it has been shown that plants synchronize the JA response with the feeding activities of a generalist herbivore across day–night cycles (Goodspeed *et al.*, 2012), although different plant ecotypes challenged by different kinds of herbivores may exhibit different circadian interactions (Jander, 2012).

Ethylene as a regulator of plant defences against herbivores

Ethylene is a gaseous hormone and is involved in development, senescence and defence against necrotrophic pathogens (Chen *et al.*, 2005). Endogenous ethylene concentrations in plant tissues depend on the activities of two biosynthetic enzymes, 1-aminocyclopropane-1-carboxylic acid synthase (ACS) and 1-aminocyclopropane-1-carboxylic acid oxidase (ACC oxidase), which convert *S*-adenosyl-Met to ethylene, but also on the rates of outward diffusion and metabolization (Wang *et al.*, 2002). Transcription factors that control ethylene-responsive genes are constitutively repressed by proteins such as JAZ (Zhu *et al.*, 2011) and ethylene perception controls the ethylene response. *Arabidopsis* contains five ethylene transmembrane receptors, located in different organelles (Kendrick and Chang, 2008). These receptors are active in the absence of ethylene (Hua and Meyerowitz, 1998), and suppress the ethylene response by constitutively stimulating phosphorylation of the ethylene signalling hub EIN2 (ETHYLENE INSENSITIVE2), leading to its degradation (Qiao *et al.*, 2009). Upon binding to ethylene, the receptors become inactive, allowing unhindered accumulation of EIN2 in the cytosol. This initiates degradation of the ethylene transcriptional repressors and thus the activation of ethylene-responsive genes in the nucleus (An *et al.*, 2010).

Salicylate as a regulator of plant defences against herbivores

Salicylate mediates defences against biotrophic pathogens (Glazebrook, 2005) and phloem-feeding herbivores (Kaloshian and Walling, 2005). During pathogen infections, defence responses can spread systemically, so are also expressed in uninfected tissues, and this is referred to as systemic acquired resistance. Several candidate signals have been reported to play a role in systemic acquired resistance, including the SA-derivative methyl salicylate. However, SA is the central local regulator because plants that are unable to accumulate SA are often highly susceptible to pathogen infections (Dempsey and Klessig, 2012). In rice (a monocot), the JA and SA pathways are thought to regulate a common set of defence genes that are effective against both biotrophic and necrotrophic pathogens (De Vleesschauwer *et al.*, 2013). Salicylate is derived from chorismate, the end-product of the shikimate pathway. From there it can be synthesized in plants via at least two distinct biosynthetic routes. The first route delivers SA in two steps and depends on the enzymes isochorismate synthase, which is induced upon pathogen infection (Wildermuth *et al.*, 2001), and isochorismate pyruvate lyase. The second route depends on the phenylpropanoid pathway. This is a pathway responsible for a variety

of products, such as flavonoids and lignins, but also for SA, and there may be parallel sub-branches within the branch leading to SA (Boatwright *et al.*, 2013). Which of these pathways or branches determines induced SA levels most strongly may also differ across plant species. Once formed, SA may be modified further by glucosylation, methylation or amino acid conjugation. Most of these derivatives are inactive and may serve to fine-tune local and systemic SA accumulation and function or may provide safe storage. Methyl salicylate is inactive but easier to transport to distal tissues, either actively via the phloem or passively via the air (Dempsey *et al.*, 2011).

A central role is played by the NONEXPRESSOR OF PR GENES (NPR) protein family. It has recently been discovered that NPR3 and NPR4 are SA receptors, whereas NPR1 acts a master regulator of SA-mediated responses (Yan and Dong, 2014). NPR1 proteins are constitutively present in the cytosol of the cell as oligomers (Tada *et al.*, 2008) and their concentration increases upon induction (Spoel *et al.*, 2009). Accumulation of SA causes an increase in the levels of reduced glutathione (the antioxidant form of glutathione), thereby changing the redox status of the cell, i.e. the balance between oxidants and antioxidants (Spoel and Loake, 2011), and this generates NPR1 monomers by the thioredoxin-catalysed reduction of monomeric disulphate bridges. Subsequently, NPR1 monomers migrate into the nucleus (Mou *et al.*, 2003; Tada *et al.*, 2008). Without NPR1, the expression of the SA-responsive genes is repressed by TGA transcription factors. After NRP1 has arrived in the nucleus, a portion of it is phosphorylated. Phosphorylated NPR1 binds to the TGA transcription factors and this complex allows the expression of target genes such *PR-1*. Unphosphorylated NPR1 may assemble together with different transcription factors and give rise to TGA-independent expression of other target genes. After a round of transcription initiation, the NPR1 protein complexes are degraded via the proteasome and new monomeric NPR1 proteins need to enter the nucleus from the cytosol to keep the response going (Mukhtar *et al.*, 2009).

Hormonal crosstalk in plant defences against herbivores

The distinct defence signalling pathways that are regulated by phytohormones interact directly and indirectly, forming complex networks, and these interactions can be additive, antagonistic or synergistic (Koornneef and Pieterse, 2008). Of all the interactions that occur between hormonal defence signalling pathways, crosstalk between the JA and SA pathways has received most attention, after it was discovered that SA can inhibit the plant's wound response (Doherty *et al.*, 1988) and indications were found for the opposite (Sano *et al.*, 1994). Under most conditions, crosstalk between SA and JA is antagonistic (Thaler *et al.*, 2012), but when applied to plants in specific ratios synergistic interactions were also observed (Mur *et al.*, 2006), and other plant hormones may modulate this crosstalk (Robert-Seilaniantz *et al.*, 2011). Suppression of the JA response by SA occurs downstream of JA-Ile perception, depending on ORA59 (Van der Does *et al.*, 2013). Other regulators include NPR1 and some of its interacting partners, including the TGA and WRKY transcription factors (Pieterse *et al.*, 2012). Nuclear localization of NPR1 is not required for

the suppression of JA signalling, and it has therefore been suggested that the role of NPR1 in mediating JA/SA crosstalk depends on a function executed in the cytosol (Spoel *et al.*, 2003). The adaptive value of the JA/SA (antagonistic) crosstalk is not clear. It has frequently been suggested that it allows plants to fine-tune the balance between different defensive strategies, depending on the type of attacker, or in case the plant is attacked by multiple attackers, depending on the timing and sequence of infestation (Pieterse and Dicke, 2007). However, whether JA/SA antagonism is adaptive remains an open question (Thaler *et al.*, 2012).

MOLECULES USED BY PLANTS TO RESIST HERBIVORES

The collective hormonal responses and their interactions induced by herbivores determine which defences are established in which host plant tissues and to what extent. Induced plant defences upon herbivory are seldom lethal: the fact that herbivores can move away from defended tissues will usually prevent them from ingesting a fatal dose. Hence, plant defences induced by herbivores will cause them to depart or, alternatively, slow down their development and population growth because bigger herbivores, or higher herbivore densities, consume and reproduce more, thus causing more damage. Many of these herbivore-induced plant defences rely on the direct antagonistic action of enzymes that interfere with feeding activities, (gut) digestive processes and gut integrity (Carlini and Grossi-de-Sa, 2002) (Fig 2).

Defence proteins

Protease inhibitors Most organisms produce a range of enzymes belonging to the protease class (also called 'peptidases'). Proteases are enzymes that perform proteolysis on target proteins and can thereby regulate enzymatic activities (Rawlings *et al.*, 2014). A subset of these proteases are the endopeptidases, which cleave the chain of amino acids of the target protein, and are referred to as 'proteinases'. The active site of a protease can be centred on a particular amino acid, e.g. a serine protease has its active site at a serine, and these 'active site' amino acids are used for their classification. Proteases themselves are regulated by PIs, which are also commonly occurring enzymes across the tree of life. Most plants upregulate a subset of their PIs, often measured as PI activity, upon herbivory, and some of these are associated with resistance to herbivores (Ryan, 1990; Lison *et al.*, 2006). These typical herbivore-induced plant PIs are believed to act on proteases in the herbivore's gut and have been suggested to have a dual role: to reduce the efficiency of proteinase activity during the herbivore's digestion of plant proteins but also to protect co-ingested defensive proteins of the plants against herbivore proteases (Macintosh *et al.*, 1990). Proteases and their inhibitors often form couples: a serine protease can be inhibited by a serine PI, although these functional annotations can be less strict than the name suggests. PIs can have different modes of action, but in general mimic the substrate of the protease and establish a strong bond with the enzyme, thus creating a protease–PI complex and delaying or blocking its proteolytic activity (Bateman and James, 2011). Different plant PI families

have been associated with defence against specific families of herbivores (Ryan, 1990). For instance, serine PIs have a primary role in defence (Hartl *et al*., 2010) and affect the performance of some lepidopteran species (Duan *et al*., 1996; Yeh *et al*., 1997). In contrast, cysteine PIs are effective against coleopteran species such as the southern corn rootworm, *Diabrotica undecimpunctata howardi* (Fabrick *et al*., 2002), and the spider mite *T. urticae* (Santamaria *et al*., 2012).

Peptidases Several peptidases/proteases are associated with plant defence responses, but it is not always clear what their functions in defence responses are and whether they directly interact with the physiology of attackers or have regulatory roles within the plant's defence network (Harrison and Bonning, 2010). While subtilisin-like proteinases are associated with anti-pathogen defences and depend on SA signalling (Jorda and Vera, 2000), cysteine proteases (such as papain) are typically induced by herbivory and are associated with disruption of the peritrophic matrix in the insect gut (Pechan *et al*., 2002; Fescemyer *et al*., 2013). Some insects have evolved adaptations to protect the peritrophic membrane against these proteases (Li *et al*., 2009; Zhu-Salzman and Zeng, 2015). Interestingly, overexpression of a cotton cysteine protease in *Arabidopsis* improved the effect of plant-delivered small RNAs, designed to trigger gene silencing via RNAi in *Helicoverpa armigera*, possibly by increasing the permeability of the peritrophic matrix (Mao *et al*., 2013). Finally, herbivory induces strong JA-dependent expression of leucine aminopeptidase (LAP) genes. The LAPs are exopeptidases that hydrolyse the terminal amino acid (leucine, methionine and arginine) residues of proteins (Gu *et al*., 1999). Herbivore-induced accumulation of LAPs in the chloroplasts depends on the JA pathway and it is assumed that they have a direct role in defence against plant consumers, probably by inactivating herbivore digestive enzymes by proteolysis (Fowler *et al*., 2009; Lomate *et al*., 2013). However, they may also have regulatory roles in the plant itself and modulate the expression of defence genes (Scranton *et al*., 2013).

Amino acid degrading proteins Digestion of proteins in the intestinal lumen results in free amino acids and peptides, available for absorption by the insect. Transepithelial transport of (essential) amino acids in the alimentary canal (predominantly the midgut) often requires energy. Some plant defensive enzymes degrade such amino acids in the arthropod gut before they can be taken up. *Manduca sexta* feeding on tomato (*S. lycopersicum*) was found to induce an arginase and a threonine deaminase, which degrades the amino acids arginine and threonine respectively, which could be found back in the gut of the larvae. Interestingly, the optimal pH for enzymatic activity of both enzymes was found to be similar to the pH found in the midgut of *M. sexta*. Moreover, threonine deaminase was inactive in tomato itself due to an activation domain that was cleaved off in the herbivore gut, thereby activating the enzyme (Chen *et al*., 2005; Gonzales-Vigil *et al*., 2011). This suggests that some plant-defensive enzymes have evolved to be activated only after having reached the herbivore gut.

Polyphenol oxidases The PPOs have been associated with plant defences: they are among the major gene/enzyme families induced by wounding, herbivory or after application of JA or JA derivatives (Thaler *et al*., 1996; Constabel and Ryan, 1998;

Stout *et al*., 1998). While PPOs can be found in diverse phylogenic groups (Tran *et al*., 2012), such as green algae, mosses and gymnosperms, *Arabidopsis* does not have them. Their mode of action and their site of action during plant–herbivore interactions is still under debate. Polyphenol oxidases catalyse the oxidation of ortho-oriented dihydroxy phenolic compounds, thereby generating quinones, which are highly reactive molecules that can either spontaneously polymerise or damage proteins, amino acids and nucleic acids via an alkylation reaction (Constabel and Barbehenn, 2008). Polyphenol oxidases have been implicated in at least two distinct defence responses. First, polyphenolics on the plant surface hinder crawling herbivores. For instance, Yu *et al*. (1992) and Kowalski *et al*. (1993) found a correlation between PPO activity, i.e. phenol polymerization activity, in glandular trichomes of different solanaceous species and the entrapment of small arthropods between such trichomes. Second, PPO-mediated generation of quinones in the herbivore's gut, i.e. after ingesting and mixing plastidial plant PPOs in combination with vacuolar plant phenolics, damages amino acids and proteins and thus interferes with food digestion (Constabel and Barbehenn, 2008).

Activity of PPOs has been associated with defence against herbivores such as coleopterans (Castañera *et al*., 1996) and lepidopterans (Felton *et al*., 1992*a*). Moreover, PPO overexpression in different plant species was also shown to increase resistance to several herbivores. Overexpression of a potato PPO in tomato increased resistance to the common cutworm *Spodoptera litura* (Mahanil *et al*., 2008) and overexpression of a poplar (*Populus trichocarpa*) PPO in *Populus tremula* increased resistance to the forest tent caterpillar *Malacosoma disstria*. Interestingly, this poplar PPO is latent in plant cells, whereas it was fully active when isolated from the caterpillar's frass, suggesting activation in the insect's gut (Wang and Constable, 2004). However PPO overexpression does not always have an effect on herbivores (Barbehenn *et al*., 2007), indicating that PPO effectiveness could depend on specific physiological conditions in the herbivore gut (Felton *et al*., 1992*b*) or on the availability of specific PPO substrates. Finally, other oxidases, such as peroxidase and lipoxygenase, may play a similar functional role in plant defences by creating potent electrophiles or interfering with the accumulation of essential nutrients (Zhu-Salzman *et al*., 2008).

Lectins Plant defensive lectins are associated with the disruption of several processes involved in the digestion of food and nutrient uptake in herbivores (Michiels *et al*., 2010). Lectins comprise a diverse family of proteins that bind specifically with mono- and oligosaccharides (Komath *et al*., 2006). Although the mode of action is not well understood, there are several cases in which herbivorous insects that propagated on artificial diets with lectins or on plants that overexpressed lectin genes showed delayed or impaired development (Gatehouse *et al*., 1996; Sauvion *et al*., 2004). Due to their high affinity with oligosaccharides, it is assumed that lectins can interact with glycoproteins in the digestive tract of the herbivore and bind to the insect's intestinal epithelial cells or its peritrophic membrane and disrupt these tissues (Macedo *et al*., 2004).

Pathogenesis-related proteins Pathogenesis-related (PR) proteins include a wide variety of proteins with diverse functions,

predominantly associated with resistance to pathogens. These proteins can be classified into 17 families and they are often used as defence marker genes, though not all of them are functionally understood: most of them are classified as a glucanase, chitinase, thaumatin, PI or peroxidase. They are defined as pathogen-induced proteins and for the majority of them evidence is largely lacking that they play a significant role in anti-herbivore defences. For example, PR-2 proteins have β-1,3-endoglucanase activity and are associated with (fungal) cell wall degradation. However, some of these proteins could also be active against herbivores. The families PR-3, 4, 8 and 11 are chitinases (Sels *et al.*, 2008). Insect chitinases, key enzymes for arthropod morphogenesis, have insecticidal activity when delivered via the plant (Kramer and Muthukrishnan, 1997). However, the effects of plant chitinases on insects are less clear. Carnivorous plants secrete chitinases to digest arthropod prey (Paszota *et al.*, 2014) and purified chitinases from mulberry latex were found to have insecticidal activity (Kitajima *et al.*, 2010). In addition, overexpression of a poplar chitinase in tomato inhibited the development of Colorado potato beetles (Lawrence and Novak, 2006). Moreover, larvae of *Orgyia antiqua* showed a lower growth rate when feeding on transgenic birch (*Betula pendula*) expressing a sugar beet chitinase (Vihervuori *et al.*, 2013), but transgenic plants in the field were more susceptible to aphids (Vihervuori *et al.*, 2008). Thus, there are indications that plant chitinases are active against insects and have the potential to damage the exoskeleton and chitin-rich peritrophic membrane of arthropods, but these activities could be limited to some species and life stages of herbivores. Other PR proteins could also have insecticidal activities. PR-1 is found in the digestive fluids of pitcher plants (Buch *et al.*, 2014) and PR-2 in the digestive fluid of sundew, although possibly in order to utilize pollen grains, fungal spores or detritus as nutritional source (Michalko *et al.*, 2013). However, there is not much evidence for herbivore-induced foliar PR proteins other than chitinases that affect herbivores directly, although PR proteins are ingested by them and can be found in their frass (Chen *et al.*, 2007).

Small cysteine-rich defence proteins There are two families of small cysteine-rich proteins that are suggested to play a role in plant defence against herbivores: defensins (e.g. PR-12) and cyclotides. Defensins are small cysteine-rich proteins, commonly synthesized in plants but also by animals. They are proteins of 45–54 amino acids that contain eight conserved cysteine residues and are similar to thionins (Thomma *et al.*, 2002). Most defensins operate by binding to cell membranes, resulting in pore-like membrane defects, causing efflux of essential ions and nutrients. Plant defensins are predominantly active against fungi (Stotz *et al.*, 2009), but some defensins inhibit α-amylase activity and have no effect on fungi (Osborn *et al.*, 2009). α-Amylase is a typical insect gut enzyme and there are indications that particular defensins reduce the digestibility of plant material (Shade *et al.*, 1994; Shiau *et al.*, 2006). Cyclotides are peptides of typically 28–37 amino acids, derived from longer precursor proteins, such as metallocarboxy peptidase inhibitors (Cavallini *et al.*, 2010). They contain six conserved cysteine residues connected by three intermolecular disulphide bonds that form a knotted structure. Their mode of action is poorly understood, but is also associated with

disrupting membrane integrity. Several cyclotides were found to exhibit insecticidal activities (Poth *et al.*, 2011; Pinto *et al.*, 2012), although they may also find applications as therapeutic drugs (Smith *et al.*, 2011) (Fig. 3).

Defence metabolites

While defensive plant proteins play a significant role in direct interactions between herbivores and plants, the role of non-protein secondary metabolites is just as big. The term 'secondary' is used to contrast them with metabolites that are directly involved in growth, development or reproduction, although it is not always possible to determine the precise physiological role of each metabolite. Across the plant kingdom, there is a staggering diversity of secondary metabolites, and they can be distinct for small phylogenetic groups. Many secondary metabolites have been implicated in plant defences or are stress-related. Despite the rich diversity of secondary metabolites, their biosynthetic origins allow them to be classified into three basal groups: (1) the phenolics; (2) the isoprenoids; and the (3) N-containing compounds (Fig. 2).

Phenolics Phenolics consist of an aromatic ring with one or more hydroxyl groups. Two pathways are responsible for the majority of plant phenolics: (1) the phenylpropanoid pathway (Cheynier *et al.*, 2013), which converts the aromatic amino acid phenylalanine into phenolics, and (2) the acetate/malonate (polyketide) pathway (Quideau *et al.*, 2011). Single phenolics can be polymerized to form polyphenols and both can be subjected to additional modifications, giving rise to a vast quantity (>9000) of chemically diverse metabolites, which include benzoquinones, phenolic acids (such as SA), coumarins, flavonoids, lignins and tannins (Balasundram *et al.*, 2006). Phenolics have various functions in primary metabolism; for instance, they protect plants from UV radiation (Landry *et al.*, 1995) or form, as lignins, an integral part of the secondary cell wall in vascular plants (Boerjan *et al.*, 2003). Furthermore, flavonoids are crucial for reproduction because they are required for pollen development (Van der Meer *et al.*, 1992) and provide many of the visual and volatile cues used by flowers (Hoballah *et al.*, 2007) and fruits (Jaakola *et al.*, 2002) to attract pollinators and seed dispersers, respectively.

Plants use phenolics to resist attacks from herbivores because of their deterrent (Kessler *et al.*, 2012*b*) and toxic (Lindroth and Peterson, 1988) nature. Hence, they are often constitutively present at or near the cell surface or stored as inactive compounds away from activating enzymes (e.g. in vacuoles or specialized cells, or bound to the cell wall) (Pourcel *et al.*, 2007). Inactive phenolics can be activated when a herbivore disrupts plant cells, thereby mixing them with activating enzymes such as glycosidases, PPOs and peroxidases to produce toxic (free) phenolics and quinones (Constabel and Barbehenn, 2008). On top of this, herbivory can induce accumulation of phenolic compounds and PPO and peroxidase activity (Stout *et al.*, 1999; Constabel *et al.*, 2000), thereby amplifying the defence response. Upregulation of chemical defences can coincide with the deposition of lignin at the site of infection or attack, creating an extra physical barrier, which is especially effective against small organisms such as nematodes and relatively immobile arthropods (Valette *et al.*, 1998). Finally, some volatile phenolics,
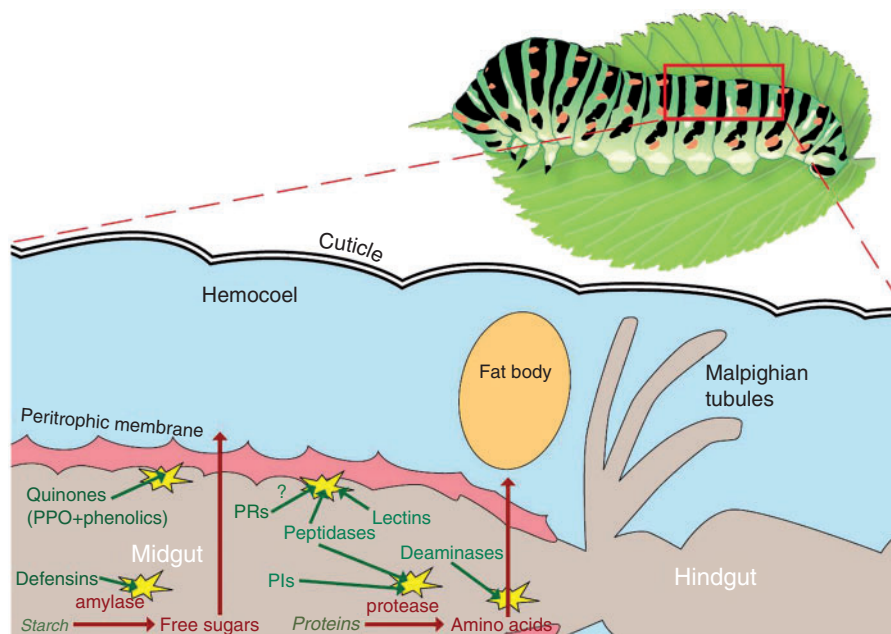
FIG. 3. Mode of action of plant defence proteins in the herbivore gut. Herbivores utilize plant material predominantly to obtain sugar and amino acids. They digest proteins into amino acids via proteases and starch/sucrose into free sugars via amylases/invertases. Plants produce special proteins that are co-ingested and interfere with digestive processes in the gut. Defensins inhibit α-amylase activity. Deaminases degrade amino acids. Proteinase inhibitors and peptidases inhibit the arthropod's proteases. Peptidases, lectins and possibly some PR proteins damage the peritrophic membrane. Plant polyphenol oxidases in combination with plant phenolics are believed to generate quinones in the arthropod gut. These quinones may damage soluble and membrane proteins and DNA. Digestive processes of the herbivore are shown in red and counter-measures of the plant in green.

such as methyl salicylate, are involved in the indirect defence response (Ament *et al.*, 2004, 2010).

Isoprenoids Isoprenoids constitute the largest and structurally most diverse class of metabolites with over 55 000 known structures. The universal isoprene [C_5] precursors isopentenyl diphosphate (IPP) and its isomer dimethylallyl diphosphate (DMAPP) are produced via two pathways in plants: the cytosolic/peroxisomal mevalonate (MVA) and the plastidial 2-methylerythritol 4-phosphate (MEP) pathway (Lange *et al.*, 2000; Sapir-Mir *et al.*, 2008). DMAPP (C_5) is a precursor for the synthesis of cytokinins and isoprene. It also serves as the substrate for successive head-to-tail condensations with one or more IPP units to obtain precursors of longer-chain isoprenoids (Akhtar *et al.*, 2013). Isoprenoids are building blocks for several phytohormones and are essential components of membranes (Schaeffer *et al.*, 2001) and the photosynthetic (Fraser *et al.*, 2000) and respiratory (Ducluzeau *et al.*, 2012) machinery. Terpenoids are derived from isoprenoid precursors through the action of terpene synthases and subsequent modifications lead to a large diversity of molecules (Schilmiller *et al.*, 2009), some of which are volatile and emitted in large quantities (Kant *et al.*, 2009). Volatile terpenoids are present in relatively large quantities in most plant-derived scent bouquets (Knudsen *et al.*, 2006). As such, they are cues for pollinators (Wright *et al.*, 2005) and seed dispersers (Hodgkison *et al.*, 2007), but also function in responses against biotic and abiotic stresses (Gershenzon and Dudareva, 2007). Since volatile terpenoids are highly lipophilic, they can penetrate plasma membranes and increase their permeability (Sikkema *et al.*, 1995) to exert direct

toxic (and repellent) effects on arthropods (Bleeker *et al.*, 2012; Vaughan *et al.*, 2013). Moreover, terpenoids serve as key compounds in the indirect defence against herbivores as attractants of their natural enemies both above- and below-ground (Kant *et al.*, 2009). Their production and release is regulated in a spatiotemporal manner (Köllner *et al.*, 2004) through defence-associated phytohormones such as JA and SA (Van Poecke and Dicke, 2002). Although considered highly effective (Kessler and Baldwin, 2001), terpenoid-mediated indirect defence can also backfire as it might attract even more herbivores (Carroll *et al.*, 2006) and can prime defence responses in competing neighbouring plants (Kessler *et al.*, 2006) and guide parasitic plants to a new host (Runyon *et al.*, 2006).

Nitrogen-containing compounds Nitrogen-containing secondary metabolites are thought to have primarily defensive purposes. Alkaloids constitute the biggest subgroup (>12 000 compounds). They bear at least one nitrogen atom in a heterocyclic ring and have been identified in ~20 % of all plant species (Levin, 1976). Several amino acids as well as purine nucleotides and isoprenoids are precursors of alkaloids (Itkin *et al.*, 2013). Their biosynthesis is often initiated in roots, followed by phloem and usually xylem transport (Courdavault *et al.*, 2014). Alternatively, the final steps of their *de novo* biosynthesis can take place above ground (Miettinen *et al.*, 2014). Alkaloids are constitutively present in plants, but their production and transport can increase upon herbivory (Baldwin, 1988) and exogenous application of methyl jasmonate (Baldwin, 1996). They can react with DNA, membranes and enzymes, and are therefore potent toxins for many organisms, including arthropods

(Wink *et al*., 1998). Interestingly, the nectar of some plants contains sublethal amounts of alkaloids, which not only protects them from nectar robbers (Kessler *et al*., 2008), but also improves their reproductive success by manipulating the behaviour of their natural pollinators (Kessler *et al*., 2012*a*).

Many plant species accumulate cyanogenic glucosides (α-hydroxynitrile glucosides), which protect them against herbivores because they can release volatile hydrogen cyanide (HCN), which inhibits cellular respiration (Way, 1984). Because plants are vulnerable to high concentrations of HCN as well, cyanogenic glucosides are stored in vacuoles, whereas the HCN-liberating enzymes β-glucosidase and α-hydroxynitrile lyase are localized in plastids (Thayer and Conn, 1981), the apoplast (Frehner and Conn, 1987) or in intracellular protein bodies (Swain *et al*., 1992). Upon herbivory, the cyanogenic glucosides become exposed to β-glucosidases. Depending on the pH, the resulting α-hydroxynitriles will either dissociate spontaneously into HCN or will be enzymatically converted to it by α-hydroxynitrile lyases (Siritunga *et al*., 2004).

Like cyanogenic glucosides, glucosinolates are derived from amino acids (Hansen *et al*., 2001). They are N- and S-containing defensive metabolites mainly found in Brassicaceae (Halkier and Gershenzon, 2006). Glucosinolates can be activated by the enzyme myrosinase (Husebye *et al*., 2002), from which they are separated by compartmentalization. Herbivory mixes the two (Barth and Jander, 2006), thereby triggering the production and release of various reactive hydrolysis products, mainly isothiocyanates and nitriles (Bones and Rossiter, 1996), which can be directly toxic and repellent to herbivores (Lazzeri *et al*., 2004) but also attract specialist herbivores (Beran *et al*., 2011) and serve as attractants of parasitoids (Mumm *et al*., 2008). Consistently, glucosinolate biosynthesis can be induced by herbivory (Hopkins *et al*., 2009) and is controlled by JA, SA and ethylene (Schweizer *et al*., 2013) (Fig. 3).

PLANT DEFENCES IN NON-ATTACKED TISSUES

Induced plant defensive substances accumulate not only locally at the feeding site, but also in undamaged tissues, and these 'systemic' responses have distinct spatiotemporal dynamics. There are several cases showing that distinct defence-associated changes can be observed in tissues neighbouring the damaged area within 1 min after wounding or herbivory (Schittko *et al*., 2000; Glauser *et al*., 2009), and these changes are typically the signalling events upstream of the actual defence response. A steady increase in the levels of JA and JA-Ile can be observed after a few minutes of damaging, with a peak after ~30 min (Glauser *et al*., 2008; Stork and Baldwin, 2009; VanDoorn *et al*., 2011). The extent to which the JA from this JA burst is synthesized *de novo* or released from storage is not always clear; it probably differs across different plant species and with the number of elicitations. The JA burst is preceded by a fast signal (Schittko *et al*., 2000), which most probably is electric, i.e. transmitted via membrane depolarizations, and which also travels to distal leaves (Glauser *et al*., 2009; Koo *et al*., 2009; Mousavi *et al*., 2013). However, molecular signals also travel from leaf to leaf, via either the phloem or the xylem (Malone and Alarcon, 1995; Rhodes *et al*., 1999). However, the identity

of these signals is still elusive. Grafting experiments with tomato plants showed that JA biosynthesis, but not perception, was required for initiating the systemic signal while the downstream defence responses required perception (Schilmiller and Howe, 2005). Peptides such as systemin and Peps, and phytohormones such as ABA, auxin and cytokinins (Soler *et al*., 2013), are closely associated with local and systemic signalling, but especially JA and usually JA derivatives play a critical role (Wu and Baldwin, 2010).

Temporal and spatial heterogeneity of signalling within and between leaves

The spatiotemporal patterns of systemic responses differ across plant species, depending on differences not only in their size or age but also in plant-specific (vascular) architecture. Because not all leaves of a plant are connected to the same degree, the induction of defensive compounds is higher in undamaged leaves with the most direct vascular connection to the attacked leaf, the so-called orthostichous leaves (Orians *et al*., 2000), and these are less vulnerable to herbivores than leaves with a less direct connection (Viswanathan and Thaler, 2004). Heterogeneity in the levels of defensive compounds in damaged tissues can stimulate herbivores to move to other areas in an attempt to avoid the induced defences (Shroff *et al*., 2008). For example, caterpillars of *M. sexta* might be able to escape from the induced resistance of a plant through 'induced movement' (Paschold *et al*., 2007). Also, the foraging behaviour of the generalist insect *Helicoverpa armigera* depends on JA: the larvae leave induced areas quickly and move to non-induced distant parts. Interestingly, *Plutella xylostella*, which is known to be resistant to defensive chemicals in some Brassicaceae plants, did not display this behaviour (Perkins *et al*., 2013). Finally, heterogeneity of leaf systemic response (Stork *et al*., 2009) or plant systemic response due to heterogeneity of vascular connections was suggested to be adaptive because it makes it more difficult for herbivores to learn which tissues will be least defended. Rodriguez-Saona and Thaler (2005) used normal tomato plants and JA-deficient (def-1) tomato plants to analyse local and systemic induced JA responses in relation to patterns of caterpillar feeding damage. They observed that the systemic response in leaves with a stronger vascular connection was stronger than in leaves with a weaker connection, but at a similar physical distance from the damaged area. Hence the extent to which a herbivore can avoid induced defences is determined by the strength of the vascular connection of the induced leaf and the newly selected (distal) leaf.

It was shown that electric and vascular signals can act in synergy with airborne signals to optimize the systemically expressed resistance within a plant (Heil and Ton, 2008). Moreover, exposure of plants to relatively high amounts of synthetic volatiles was shown to induce defences. For example, exposure of tomato plants to methyl jasmonate results in the accumulation of PIs in systemic leaves of the plant, in neighbouring tomato plants and even in plants of different species, such as tobacco and alfalfa (Farmer and Ryan, 1990). Similarly, methyl salicylate may act as an airborne signal to activate resistance in uninfected tissues of an infected plant and in neighbouring plants (Shulaev *et al*., 1997). In contrast, corn

plants previously exposed to the natural volatiles emitted by neighbouring plants accumulated more JA when damaged mechanically or induced with caterpillar regurgitant compared with corn plants not pre-exposed to volatiles (Engelberth *et al.*, 2004). It has been shown that plant volatiles elicit responses not only in neighbouring plants but also in their own distal tissues (Heil and Silva Bueno, 2007) and, apart from elicitation, volatiles can also 'prime' defences: priming means that the actual defence response is not established yet but is preprepared in such a way that it is displayed faster and/or more strongly upon actual induction by a herbivore later on (Kessler *et al.*, 2006; Heil *et al.*, 2007). Airborne signalling has at least two advantages over vascular signalling. First, priming the induction of plant defences through the air overcomes vascular restrictions resulting from the plant's orthostichy; second, airborne signalling can reach distal plant parts faster than compounds that are transported through vascular tissues (Heil and Ton, 2008). This is especially relevant for bushy plants in which signals transported via the vascular system have to travel long distances. For example, systemic induced resistance in sagebrush depends on air contact, possibly due to restrictions in vascular connections (Karban *et al.*, 2006). Similarly, undamaged leaves of hybrid poplar (*Populus deltoides* × *nigra*) exposed to volatiles from wounded leaves with little or no vascular connection were primed to defend against larvae of the gypsy moth, *Lymantria dispar* (Frost *et al.*, 2007). Thus, there is ample evidence that plant volatiles can facilitate signalling between leaves with weak vascular connections and facilitate priming in synergy with signals transmitted directly via plant tissues.

HOW HERBIVORES COPE WITH DEFENCES

Plants and herbivores have coevolved for over 400 million years. While plants have evolved signalling networks to regulate induced defences and diversity in their palette of secondary metabolites, herbivores have been under pressure to evade defences (reviewed in Alba *et al.*, 2011). Hence, behavioural adaptions have evolved that allow herbivores to avoid defended plant tissues (Paschold *et al.*, 2007; Shroff *et al.*, 2008; Perkins *et al.*, 2013) as much as possible or to dismantle defensive structures such as trichomes (Cardoso, 2008) and latex channels (Rodrigues *et al.*, 2010). However, herbivores have also evolved a variety of mechanisms to cope with deterrent substances produced by their host plants. Given the enormous economic impact of herbivore resistance to agrochemicals, a large part of our knowledge of adaptations to xenobiotics comes from the field of pesticide resistance (Despres *et al.*, 2007). However, the mechanisms at play are similar to those that enable them to resist defensive phytotoxins, and a functional overlap between adaptation to agrochemicals and to phytotoxins has been suggested (Dermauw *et al.*, 2013*a*).

Two general mechanisms allow herbivores to cope with the xenobiotics from their environment: mechanisms that decrease exposure (pharmacokinetic responses) and mechanisms that decrease sensitivity (pharmacodynamic responses). Pharmacokinetic responses comprise a variety of adaptations that reduce uptake, increase catabolism and allow sequestration, whereas the pharmacodynamic response types comprise adaptations at the level of interactions between allelochemicals and

their target-site(s) (Taylor and Feyereisen, 1996; Kennedy and Tierney, 2013). Together, these mechanisms determine the level of tolerance of herbivores to xenobiotics (Fig. 4).

Pharmacokinetic responses

Mechanisms of decreased exposure The detoxification of xenobiotics usually occurs in three phases. In phase I, the xenobiotic is modified by reactions that incorporate a nucleophilic functional group (a hydroxyl, carboxyl or amine group) and this often results in a more polar/hydrophilic substance. In phase II, the metabolite resulting from phase I is conjugated to endogenous molecules such as glutathione or a sugar molecule, which further increases the compound's polarity/hydrophilicity. In phase III, the phase II conjugated xenobiotic is transported out of the cell by cellular transporters. In several cases, these transporters can also act as a first line of defence, preventing allelochemicals entering the cell by rapid efflux without the need for modifications (sometimes this is referred to as phase 0).

Enzymes that operate during phase I are often cytochrome P450 monooxygenases (P450s) and carboxyl/choline esterases, whereas enzymes such as glutathione-*S*-transferases (GSTs) and UDP-glycosyltransferases (UGTs) typically operate during phase II. Finally, transport of phase II metabolites out of the cell is often performed by ATP-binding cassette (ABC) and solute carrier (SLC) family proteins. Adaptations that allow herbivores to enhance their detoxification of particular target xenobiotics often occur via mutations that increase the production of specific detoxification enzymes and transporters as well as by mutations that improve their catalytic or transport properties (Brattsten, 1992; Despres *et al.*, 2007; Kennedy and Tierney, 2013).

Phase I detoxification A wide range of allelochemicals, including furanocoumarins, terpenoids, glucosinolates, flavonoids and alkaloids can be metabolized by P450s of herbivorous arthropods (Despres *et al.*, 2007; Feyereisen, 2012). The involvement of P450s within the lepidopteran genus *Papilio* in furanocoumarin resistance is one of the first documented examples relating to plant–arthropod interactions (Berenbaum, 1982). The P450s are by far the best studied enzymes of phase I, and have been associated with resistance to plant allelochemicals and pesticides in a wide range of species (Feyereisen, 2012; Schuler, 2012). Although esterases have been linked to pesticide resistance in a number of cases, their role in plant allelochemical defence remains elusive (Despres *et al.*, 2007; Li *et al.*, 2007). Notably, esterases were found not always to operate via hydrolysis but also to confer resistance to pesticides by sequestration, i.e. by binding to the target substance without modifying it. Remarkably, esterase genes were duplicated in some aphid species, which gave rise to elevated expression, such that 3 % of their total protein content consisted of these enzymes (Devonshire and Sawicki, 1979; Devonshire and Moores, 1982). It is well imaginable that such esterases are involved in resistance or tolerance to phytotoxins as well.

Phase II detoxification Conjugation of xenobiotics by GSTs has been linked to allelochemical tolerance in a number of cases, although most evidence has been obtained from *in vitro* assays. The expression of GSTs in arthropod herbivores can be induced
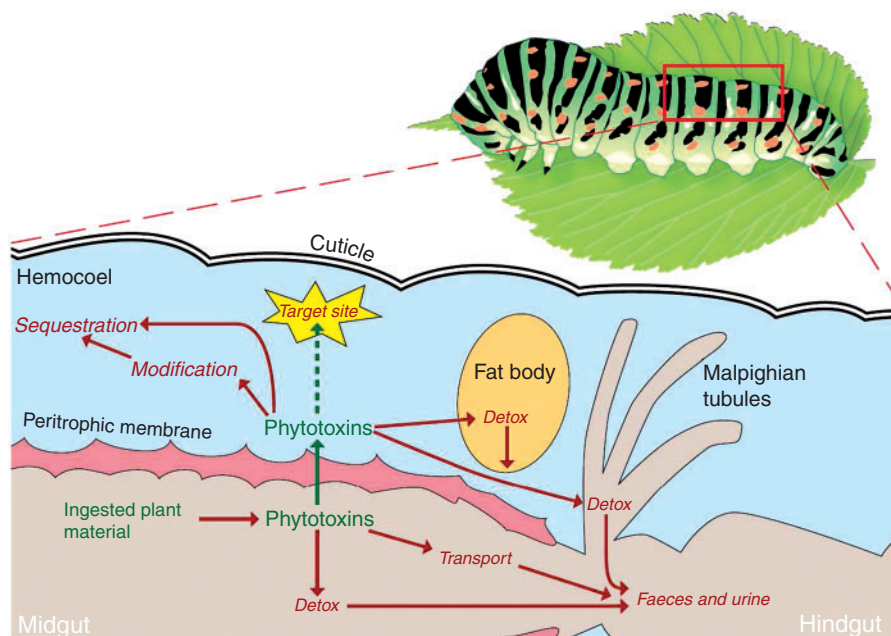
Fig. 4. The herbivore resistance response. Plant secondary metabolites can have diverse target sites. Two general mechanisms allow arthropod herbivores to cope with plant secondary compounds: mechanisms that decrease exposure via detoxification, transport and/or sequestration (shown in this figure) but also mechanisms that decrease sensitivity (see main text). Detoxification (indicated as 'Detox' in this figure) usually occurs in three phases: in phase I, enzymes such as P450s and CCEs (carboxyl/cholinesterases) modify the metabolite; in phase II GSTs and UGTs conjugate it and in phase III the conjugated metabolites are transported out of the cell, typically by ABC transporter and SLC family proteins. The midgut, fat body and Malpighian tubes are the insect tissues where these detoxification phases occur (Harrop *et al.*, 2014). Metabolites are excreted via faeces and urine. Some metabolites, or modified metabolites, are stored in the cuticle or in other organs and are used by the arthropod for its own protection. Plant substances are indicated in green and arthropod responses in red.

by a number of allelochemicals, and these enzymes are generally believed to make up an important component of the overall pharmacokinetic response. Some GSTs of the polyphagous insect *Spodoptera frugiperda* are able to metabolize a variety of thiocyanate conjugates. Moreover, the diversity of glucosinolate-derived thiocyanates that insects such as the larvae of *S. frugiperda*, *Trichoplusia ni* and *Anticarsia gemmatalis* can metabolize correlates with their host plant range (Li *et al.*, 2007), whereas the major glucosinolate-derived thiocyanate was found to be a glutathione-conjugated derivative in several generalist insect herbivore species (Schramm *et al.*, 2012). Recently, a role for GSTs in tolerance to glucosinolates was also suggested for the whitefly *Bemisia tabaci* and in the mustard-feeding specialist *Scaptomyza nigrita* (Elbaz *et al.*, 2012; Gloss *et al.*, 2014). Finally, an enzyme called GST16 inactivates the phytohormone OPDA in the gut of *Helicoverpa armigera* by isomerization to inactive iso-OPDA (Dąbrowska *et al.*, 2011). However, it is unclear whether this OPDA modification has adaptive significance (Shabab *et al.*, 2014).

The second important class of conjugation enzymes is that of the UGTs, which convert lipophilic aglycones into more hydrophilic glycosides by conjugation with UDP-glucose. UGTs have been shown to be involved in resistance of lepidopterans to the alkaloid capsaicin and the detoxification of benzoxazinoids (Ahn *et al.*, 2011; Wouters *et al.*, 2014), and they may be widespread enzymes that allow herbivorous arthropods to adapt to xenobiotics (Ahn *et al.*, 2014). Some insects have evolved traits that in principle allow them to prevent the activation of protoxins, such as glucosinolates, by plant enzymes. For

example, aphids prevent glucosinolates from mixing with myrosinases by avoiding rupturing myrosinase-containing plant cells. Moreover, the insect degrades these glucosinolates in the gut and conjugates the breakdown product to ascorbate, glutathione and cysteine. However, artificial feeding assays suggested that these conjugates also have an antifeeding effect in *M. persicae*. Hence, glucosinolates may also have defensive functions independent of myrosinase, via post-ingestive breakdown processes occurring in the aphid (Kim *et al.*, 2008).

Phase III detoxification While phases I and II have been characterized in some detail, phase III has been documented in far less detail (Sorensen and Dearing, 2006; Dermauw and Van Leeuwen, 2014). Gaertner *et al.* (1998) suggested that the efflux of nicotine and other alkaloids by the Malpighian tubules, the main excretory organs of insects, of *M. sexta* is mediated by an ABC transporter. This finding was based on the observation that verapamil, a known inhibitor of the ABC transporter, blocks nicotine transport in the Malpighian tubules of *M. sexta*. In addition, Petschenka *et al.* (2013) found an ABC transporter that probably functions as cardenolide efflux carrier, thereby protecting the nervous tissues of lepidopterans. Other transporter families are also implicated in mediating the efflux of plant allelochemicals. Govind *et al.* (2010) showed that transporters of the major facilitator superfamily, which include many transporters of the SLC family, were downregulated in *M. sexta* larvae after feeding on wild tobacco mutants that were unable to produce JA. Interestingly, other members of the same superfamily were upregulated in arthropod herbivores that were

transferred from their preferred host to a more challenging, less suitable host (de la Paz Celorio-Mancera *et al.*, 2013), e.g. in the non-insect arthropod *T. urticae* (Dermauw *et al.*, 2013*b*). Regardless of the identity of the transporters or transport mechanisms, a number of cases have been described in which the evolution of transport systems has determined herbivore success. For example, the ability to selectively transport plant glycosides has been suggested to stand at the basis of the evolution of life styles and host ranges of leaf beetles (Kuhn *et al.*, 2004; Strauss *et al.*, 2013).

Miscellaneous resistances to xenobiotics

Some insects, especially host-plant specialists, have developed resistance mechanisms that differ from those described above. For example, a flavin-dependent mono-oxygenase in arctiid moths was shown to be involved in detoxifying pyrrolizidine alkaloids (Sehlmeyer *et al.*, 2010; Wang *et al.*, 2012). These enzymes usually have general functions in an insect's primary metabolism but here evolved, after gene duplication, to catalyse N-oxidation of pyrrolizidine alkaloids (Langel and Ober, 2011). Moreover, specialist crucifer-feeding insects have developed the means to redirect the formation or to overcome the toxic action of glucosinolate breakdown products (Pentzold *et al.*, 2013) by evolving novel enzymes and proteins into general detoxification pathways. *Pieris rapae* was shown to use a nitrile-specifier protein to divert the degradation of glucosinolates in the toxic isothiocyanates to less toxic nitriles (Wittstock *et al.*, 2004; Stauber *et al.*, 2012). Another specialist, the diamondback moth, *Plutella xylostella*, desulphates glucosinolates and thereby generates inactive metabolites (Ratzka *et al.*, 2002).

Other examples of adaptations to xenobiotics in a number of lepidopterans include the detoxification of cyanogenic glucosides by converting cyanide into *β*-cyanoalanine (Zagrobelny and Møller, 2011). It was shown recently that the *β*-cyanoalanine synthase genes of lepidopterans as well as mites stand at the basis of this adaptation and were originally obtained from bacteria by horizontal transfer into the ancestral genomes (Wybouw *et al.*, 2014). This enzyme might also be crucial for countering the negative effects of glucosinolate in *P. rapae* because their breakdown also generates cyanide (Stauber *et al.*, 2012; Wybouw *et al.*, 2014). In addition, some lepidopterans have evolved the means to convert cyanide into nitrogen (Engler *et al.*, 2000; Pentzold *et al.*, 2014).

Sequestration

Some herbivores make use of plant allelochemicals for their own defence against predators by storing ingested chemicals in specialized tissues or in the integument. A variety of mechanisms have been described that allow herbivores to exploit these toxic substances without suffering from their latent detrimental effects. Compounds can be sequestered either directly or after biotransformation, such as by oxidation and conjugation. More than 250 insect species have been shown to sequester plant metabolites covering a wide range of chemical classes, such as alkaloids, cyanogenic glucosides, glucosinolates, isoprenoids and cucurbitacins (Nishida, 2002; Opitz and Müller,

2009). In some cases, insects have evolved the ability to synthesize these compounds *de novo* by convergent evolution of the biosynthetic pathways (Jensen *et al.*, 2011). Also, some insect species have evolved additional means to efficiently utilize sequestered compounds for their own defence. For example, some aphid and flea beetle species have evolved a specific myrosinase that allows them to convert glucosinolates into their toxic breakdown products in a similar way as plants do and to release these as toxic and repellent volatiles into the air (Beran *et al.*, 2014; Rahfeld *et al.*, 2014).

Pharmacodynamic responses: mechanisms of decreased sensitivity

While many adaptations of herbivores to xenobiotics depend on mechanisms that directly or indirectly divert these substances from their target sites, some adaptations are due to reduced target-site sensitivity. Although alterations in the target site of pesticides have been associated with herbivore resistance to pesticides (Ffrench-Constant, 2013; Van Leeuwen *et al.*, 2010*b*; Feyereisen *et al.*, 2015), few studies have linked such mutations to insect resistance to plant allelochemicals. This lack of documented target-site resistance to phytochemicals is probably due to the fact that many of these have multiple modes of action and our knowledge of these mechanisms is limited (Berenbaum, 1987; Despres *et al.*, 2007). The best documented example of target-site insensitivity to phytotoxins is that of amino acid substitutions in the α subunit of Na$^+$/K$^+$-ATPase from four different insect orders (Lepidoptera, Coleoptera, Heteroptera and Diptera), conferring resistance to the plant toxin ouabain, a cardenolide produced by several members of the Apocynaceae. Remarkably, the same substitution was found in different insect species, all of which specialized in cardenolide-containing plant species, suggesting that this replacement must have evolved independently several times (Agrawal *et al.*, 2012; Dobler *et al.*, 2012; Zhen *et al.*, 2012; Dalla *et al.*, 2013). Finally, tolerance to L-canavanine, a non-proteinogenic amino acid of leguminous plants, can also be considered as a target-site-based resistance mechanism in some insect species. The toxicity of L-canavanine is caused by its incorporation into proteins, replacing L-arginine. Insects such as the bruchid beetle *Caryedes brasiliensis* have specialized in feeding on the L-canavanine-rich seeds of *Dioclea megacarpa* and have evolved an arginyl-tRNA synthetase that can discriminate between L-canavanine and L-arginine, thereby effectively avoiding the adverse biochemical effect of L-canavanine (Rosenthal *et al.*, 1976; Leisinger *et al.*, 2013). In addition to target-site insensitivity, mechanisms of decreased exposure to L-canavanine have also been reported for the tobacco budworm *Heliothis virescens* (Melangeli *et al.*, 1997) (Fig 4).

Plant defence suppression by herbivores

Because many defensive actions of plant are induced or maintained by ongoing physiological processes, some herbivores have evolved the means to interfere with these processes. In this way, these herbivores may manipulate resource flows (Clark and Harvell, 1992) or suppress defences (Musser *et al.*, 2002). The suppression of defences is distinct from defence

avoidance, such as 'stealthy feeding' or vein cutting, which serve to prevent detection or to prevent physical contact with defensive substances. However, these two processes will not always be easy to separate experimentally (Karban and Agrawal, 2002; Stireman and Cipollini, 2008; Alba *et al.*, 2011). Suppression of defences is characterized by lowering the rate of production of defensive compounds. Hence, suppression can operate upstream or downstream of a defensive pathway and block it altogether or dampen it to intermediate levels, although assessing the latter can be difficult without having a suppression-free control experiment as a benchmark (Alba *et al.*, 2015). Finally, downregulation of plant defences qualifies as suppression when it is paralleled by an increase in (reproductive) performance of the herbivore (Table 1).

Suppression of plant defences is a well-known phenomenon in plant pathogens such as pathogenic bacteria (Abramovitch *et al.*, 2006), rust fungi (Voegele and Mendgen, 2003), oomycetes (Kamoun, 2006) and viruses (Kasschau and Carrington, 1998). However, nematodes and mites were also found to suppress defences. Several phytophagous nematode species interfere with host plant resistance (Haegeman *et al.*, 2012) and the cyst nematode *Meloidogyne incognita* was found to suppress SA- and JA-dependent systemic acquired resistance in *Arabidopsis thaliana* (Hamamouch *et al.*, 2011). In addition, two spider mite species were found to suppress the defences downstream of both JA and SA simultaneously in tomato (Kant *et al.*, 2008; Sarmento *et al.*, 2011*a*; Alba *et al.*, 2015), whereas an eriophyid mite was found to suppress only the downstream JA defences, independently from hormonal crosstalk (Glas *et al.*, 2014). Other examples of suppression are from insects and can be attributed to hormonal crosstalk in the majority of cases. Hemipteran phloem feeders such as the mealybug *Phenacoccus solenopsis* (Zhang *et al.*, 2011) and the whitefly *B*. *tabaci* were found to suppress JA defences (Zhang *et al.*, 2009), possibly by inducing antagonist SA defences (Zarate *et al.*, 2007; Walling, 2009). In addition, the aphid *Megoura viciae* inhibits defensive phloem clogging (Will *et al.*, 2007), whereas other aphid species were found to suppress the oxidative burst (Bos *et al.*, 2010). The leafhopper *Macrosteles quadrilineatus* suppresses JA defences indirectly via an effector derived from a vectored phytoplasma (Sugio *et al.*, 2011). Also, chewing larvae of several lepidopteran species have been found to interfere with induced defences (Bede *et al.*, 2006). On *A. thaliana*, larvae of *Spodoptera exigua* inhibit JA-mediated defence responses via the systemic acquired resistance pathway (Weech *et al.*, 2008) whereas *Pieris brassicae* suppresses defences independently of the JA and SA pathways (Consales *et al.*, 2012). In addition, *Helicoverpa zea* was found to suppress nicotine accumulation in *N. tabacum* (Musser *et al.*, 2002, 2005) and to suppress JA- and ethylene-regulated genes via salivary enzymes in tomato (Wu *et al.*, 2012). This is reminiscent of the downregulation of nicotine by *M. sexta* feeding on *N. attenuata*, although it may reflect a plant-adaptation rather than a herbivore-adaptive trait (Kahl *et al.*, 2000; Voelckel *et al.*, 2001). Moreover, the Colorado potato beetle, *Leptinotarsa decemlineata*, suppresses transcription of PI genes in tomato (Lawrence *et al.*, 2011). Finally, larvae of virulent strains of the hessian fly, *Mayetiola destructor*, secrete substances via their vestigial mouthparts into plant tissues, thereby suppressing the expression of PI and lectin genes (Stuart *et al.*, 2012).

Thus, evidence for suppression of plant defensive processes is found across herbivorous insects, plant-eating mites and nematodes.

Whereas induction of plant defences often results from elicitor or HAMP recognition, the suppression of induced or constitutive defences is often attributed to so-called effector molecules. These molecules are especially well known from phytopathogens and had been discovered already in the 1970s (Shiraishi *et al.*, 1978), but the notion that herbivores may secrete molecules with similar properties arose after the discovery that GOX from the saliva of *H. zea* caterpillars counteracts the production of nicotine in *N. tabacum* (Musser *et al.*, 2002). Although our knowledge of herbivore effectors is still limited, a staggering diversity of effectors from pathogens (Deslandes and Rivas, 2012; Rovenich *et al.*, 2014) and nematodes (Mitchum *et al.*, 2013; Kazan and Lyons, 2014) has been discovered. Roughly, these effectors comprise the following functional groups (although these are not mutually exclusive).

- Metabolites secreted into the host to manipulate particular physiological processes such as hormonal signalling. Some strains of *P. syringae* produce the JA mimic coronatine, which puts JA defences into overdrive to suppress SA defences (Zhao *et al.*, 2003).
- Enzymes that interfere with the host's ability to control infected tissues encoded by transgenes inserted into the host's genome by the pathogen. The best known example is *Agrobacterium tumefaciens*, which injects its transfer DNA (which carries genes that facilitate gall formation) into plant cells, where it integrates into the host's genome and is expressed by the host (Zhu *et al.*, 2000).
- Enzymes secreted by pathogens to perform a metabolic conversion in the host that affects its defences. Fungi such as *Septoria lycopersici* produce tomatinase, which not only detoxifies the defensive alkaloid tomatine but also generates hydrolysis products that suppress the hypersensitive response (Bouarab *et al.*, 2002).
- Secreted proteins that interfere with transcription factors or that act as transcription factors of defence genes of the host. The first is the case for an effector called SAP11, produced by phytoplasmas vectored by leafhoppers. SAP11 destabilizes the host's CIN-TCP transcription factors, leading to downregulation of the JA response (Sugio *et al.*, 2011). The second is the case for the transcription activator-like (TAL) effectors produced by the *Xanthomonas* bacterium. TAL effectors are translocated to the host's nucleus, where they modulate the expression of specific target genes to facilitate the infection (Boch and Bonas, 2010).
- Secreted proteins that interfere with host receptors involved in defences. For example, the *P. syringae* HopF2 effector suppresses plant immunity by targeting the R gene co-receptor BAK1 (Zhou *et al.*, 2014).
- Secreted proteins that interfere with defence signalling cascades downstream of receptor recognition. This is the case with AvrB of *P. syringae*, which phosphorylates the signalling hub RIN4 to block PAMP (Pathogen-Associated Molecular Pattern) triggered immunity (Mackey *et al.*, 2002).
- Secreted proteins that manipulate proteasome functioning in defensive processes. This is the case for the E3 ubiquitin

TABLE 1. *Defence suppression by arthropod herbivores*

Arthropod	Common name	Plant	Defence suppressed	Mode of action	Biological readout	Reference
Helicoverpa zea (Insecta)	Corn earworm	*Nicotiana tabacum*	Nicotine production	Salivary enzyme glucose oxidase	Caterpillar weight gain	Musser *et al.*, 2002, 2005
Helicoverpa zea (Insecta)	Corn earworm	*Solanum lycopersicum*	JA defence response	Salivary enzymes		Wu *et al.*, 2012
Spodoptera exigua (Insecta)	Beet armyworm	*Medicago truncatula*	Genes involved in terpenoid biosynthesis	Saliva-derived factors		Bede *et al.*, 2006
Spodoptera exigua (Insecta)	Beet armyworm	*Arabidopsis thaliana*	JA defence	SA–JA antagonism	Larval weight gain	Weech *et al.*, 2008
Spodoptera littoralis, Pieris brassicae (Insecta).	African cotton leafworm, cabbage butterfly	*Arabidopsis thaliana*	Wound-inducible genes	Unknown		Consales *et al.*, 2012
Pieris rapae, Spodoptera exigua (Insecta)	Small white; beet armyworm	*Arabidopsis thaliana*	Green leaf volatiles	Unknown	Feeding choice	Savchenko *et al.*, 2013
Manduca sexta (Insecta)	Tobacco hornworm	*Nicotiana attenuata*	Nicotine production	Ethylene		Kahl *et al.*, 2000; Voelckel *et al.*, 2001
Frankliniella occidentalis (Insecta)	Western flower thrips	*Arabidopsis thaliana*	JA defence	SA–JA antagonism	Host plant preference	Abe *et al.*, 2012
Mayetiola destructor (Insecta)	Hessian fly	*Triticum* spp.	Protease inhibitors, lectins	Unknown		Liu *et al.*, 2007; Wu *et al.*, 2008
Bemisia tabaci (Insecta)	Silverleaf whitefly	*Arabidopsis thaliana*	JA defence response	SA–JA antagonism	Nymphal developmental speed	Zarate *et al.*, 2007
Bemisia tabaci (Insecta)	Silverleaf whitefly	*Phaseolus lunatus*	(E)–β-ocimene production, JA defence	SA–JA antagonism	Attraction of predatory mites, preference, oviposition	Zhang *et al.*, 2009
Bemisia tabaci (Insecta)	Silverleaf whitefly	*Nicotiana tabacum*	JA defence	Vectored virus	Population growth	Zhang *et al.*, 2012
Bemisia tabaci (Insecta)	Silverleaf whitefly	*Solanum lycopersicum*	JA defence	Saliva via endogenous bacterium	Survival, oviposition	Su *et al.*, 2015
Megoura viciae (Insecta)	Aphid	*Vicia fabae*	Phloem clogging	Ca^{2+} chelators	Behaviour	Will *et al.*, 2007
Acyrthosiphon pisum (Insecta)	Pea aphid	*Vicia faba*	Unknown	Salivary effector protein C002	Survival, behaviour	Mutti *et al.*, 2008
Myzus persicae (Insecta)	Green peach aphid	*Nicotiana benthamiana*	Oxidative burst	Salivary effector proteins C002, Mp10, Mp42	Reproductive performance	Bos *et al.*, 2010
Myzus persicae (Insecta)	Green peach aphid	*Arabidopsis thaliana*	Unknown	Salivary effector proteins C002, PIntO1, PIntO2	Reproductive performance	Pitino and Hogenhout, 2013
Macrosiphum euphorbiae (Insecta)	Potato aphid	*Nicotiana benthamiana, Solanum lycopersicum*	Unknown	Salivary effector proteins Me10, Me23	Reproductive performance	Atamian *et al.*, 2013
Myzus persicae (Insecta)	Green peach aphid	*Nicotiana tabacum, Nicotiana benthamiana, Arabidopsis thaliana*	Indole glucosinolate, callose, hydrogen peroxide	Salivary effector protein Mp55	Reproductive performance	Elzinga *et al.*, 2014
Brevicoryne brassicae (Insecta)	Cabbage aphid	*Brassica oleracea*	JA defence	Interference with JA signal transduction	Caterpillar developmental speed, caterpillar weight gain	Soler *et al.*, 2012
Macrosteles quadrilineatus (Insecta)	Aster leafhopper	*Arabidopsis thaliana*	JA defence	Vectored phytoplasma	Reproductive performance, oviposition rate	Sugio *et al.*, 2011
Phenacoccus solenopsis (Insecta)	Cotton mealybug	*Gossypium hirsutum*	Gossypol production, JA defence response	Unknown	Development al speed, adult weight gain, feeding choice	Zhang *et al.*, 2011
Leptinotarsa decemlineata (Insecta)	Colorado potato beetle	*Solanum lycopersicum*	Proteinase Inhibitors	Unknown		Lawrence *et al.*, 2011
Leptinotarsa decemlineata (Insecta)	Colorado potato beetle	*Solanum lycopersicum*	JA defence	Oral bacteria	Larval weight gain	Chung *et al.*, 2013
Tetranychus urticae (Acari)	Two-spotted spider mite	*Solanum lycopersicum*	JA and SA defence, terpenoids and methyl salicylate	Unknown	Oviposition rate	Kant *et al.*, 2008
Tetranychus evansi (Acari)	Red spider mite	*Solanum lycopersicum*	JA and SA defence, terpenoids and methyl salicylate	Unknown	Oviposition rate, population growth	Sarmento *et al.*, 2011a, Alba *et al.*, 2015
Aculops lycopersici (Acari)	Tomato russet mite	*Solanum lycopersicum*	JA defence	Unknown	Population growth	Glas *et al.*, 2014

ligase AvrPtoB of *P. syringae*, which initiates degradation of a kinase that is essential for innate immunity (He *et al.*, 2006).

- Secreted proteins that perform proteolysis of plant defence proteins. This was found for an extracellular PI of *Phytophthora infestans*, which targets a tomato PR protein (Tian *et al.*, 2004) and for AvrRpt2 of *P. syringae*, which acts as protease to eliminate RIN4 (Axtell *et al.*, 2003).
- Secreted proteins that interfere with host vesicle trafficking during immune responses. This is the case for HopM1 of *P. syringae*, which mediates degradation of the vesicle trafficking regulatory protein MIN7 (Nomura *et al.*, 2011).
- Secreted proteins that interfere with RNAi, such as the 2b protein of the cucumber mosaic virus. This protein protects the virus against RNAi-mediated degradation and also suppresses SA defences (Ji and Ding, 2001) and JA defences induced by their aphid vectors (Westwood *et al.*, 2014).
- Secreted small RNAs that manipulate a host's RNAi machinery, such as a small RNA transferred by *Botrytis cinerea*, which silences the protein Argonaute 1 in *Arabidopsis* and tomato and thereby suppresses host immunity (Weiberg *et al.*, 2013).

Effectors may be powerful weapons whereby pathogens and nematodes interfere with plant defences, but plants have evolved a range of R genes in return, which specifically serve to recognize effectors and bypass suppression (Bergelson *et al.*, 2001). These molecular sensors are used in plant breeding to obtain pathogen-resistant crops but R gene resistance is often broken again shortly after its introduction due to counter-adaptations in pathogens (Bent and Mackey, 2007). Hence, plant breeders have redirected their focus to the effector targets, also referred to as susceptibility genes or S genes (van Schie and Takken, 2014), since effector-resistant breeding targets are promising tools for obtaining more durable resistance (Gawehns *et al.*, 2013).

Effectors involved in plant defence suppression by herbivores

There are indications that plant-defence-suppressing herbivorous arthropods also secrete effectors via their saliva into their host, similar to pathogens and nematodes. The first of such salivary components that was discovered was the enzyme GOX, which is the most abundant molecule in the oral secretions of the caterpillar of *H. zea* (Musser *et al.*, 2002). This enzyme catalyses the oxidation of glucose to D-gluconic acid and thereby generates hydrogen peroxide. The amount of GOX applied to *T. tabacum* plants correlates with an increase in SA and a decrease in the accumulation of nicotine. Possibly, GOX suppresses or attenuates JA and ethylene responses by crosstalk with SA (Diezel *et al.*, 2009; Eichenseer *et al.*, 2010). GOX has been found in many more caterpillar species (Eichenseer *et al.*, 2010) and other herbivorous insects, such as aphids and non-herbivores such as honeybees (Iida *et al.*, 2007; Harmel *et al.*, 2008). A comprehensive study by Eichenseer *et al.* (2010) showed large variation in GOX activity within families and subfamilies of 88 caterpillar species, but these activities depended on the host plant species as well. Moreover, a recent report showed that *H. zea* GOX elicits the JA pathway in tomato (Tian *et al.*, 2012). Taken together, these studies suggest that some plants, such as tomato, may have evolved a recognition mechanism for GOX, resembling R-gene-mediated recognition of effector proteins in plant–pathogen interactions.

Advances in genomics and proteomics have greatly facilitated the discovery of more effector proteins in insects. After the *Acyrthosiphon pisum* (peach aphid) salivary glands were sequenced, the first aphid effector was discovered. This protein is a 22-kDa salivary-secreted protein of unknown function called C002 (Mutti *et al.*, 2008). RNAi-mediated knockdown of C002 expression affected *A. pisum* foraging and feeding behaviour and reduced aphid fitness. Bos *et al.* (2010) used *N. benthamiana* to ectopically express C002 from *M. persicae* (green peach aphid) and showed that aphid fecundity increased on these plants. Transient overexpression of a second aphid protein, Mp10, sufficed to suppress the flagellin-triggered oxidative burst in *N. benthamiana*, but aphid reproduction was lower on these plants. A subsequent study characterized two additional aphid proteins, Mp1 (PIntO1) and Mp2 (PIntO2), which correlate positively with aphid fecundity on *Arabidopsis*. Interestingly, the performance of *M. persicae* did not improve on *Arabidopsis* plants expressing the *A. pisum* orthologues of both effectors (Pitino and Hogenhout, 2013). Finally, two putative effectors of *Macrosiphum euphorbiae* were found, Me10 and Me23, both of which increased aphid fecundity on *N. benthamiana*, whereas only Me10 increased their fecundity on tomato (Atamian *et al.*, 2013).

Research on gall midges has provided independent evidence for a role of effector proteins in plant–herbivore interactions. Early larval stages of the hessian fly, *M. destructor*, are plant parasites. When they colonize wheat (*Triticum* spp.), the larvae induce feeding cells in their host, which provide them with food until they develop into adults (Harris *et al.*, 2006). More than 30 hessian fly resistance genes have been found in wheat, some of which are predicted to encode typical R proteins (Liu *et al.*, 2005). On resistant wheat, hessian fly larvae are unable to induce feeding cells, but instead induce a hypersensitive-like response that prevents them from eating (Harris *et al.*, 2010). One *M. destructor* gene, *vH13*, functions as an avirulence factor on wheat carrying the *H13* resistance gene. In contrast, larvae from populations that are virulent on *H13* wheat did not express *vH13*, while RNAi-mediated knockdown of *vH13* in avirulent larvae made some of them virulent (Aggarwal *et al.*, 2014). These data suggest that *vH13* may function as an effector on non-resistant wheat varieties.

Thus, there are indications that herbivores may make use of effectors, just as pathogens do. This notion is strengthened by the existence of anti-herbivore R genes such as *Mi-1*, *Vat* and *Bph14* (Rossi *et al.*, 1998; Dogimont *et al.*, 2008; Du *et al.*, 2009). The high diversity found among pathogen effectors discourages the use of protein homology as a strategy to identify herbivore effectors (Rep, 2005). Nevertheless, most effector proteins share structural features that can be easily recognized, such as an amino-terminal signal peptide, the absence of transmembrane domains and a small protein size. Furthermore, effectors that operate in the plant apoplastic space are usually rich in cysteine residues (Rooney *et al.*, 2005). Several studies have exploited these common properties to find novel effector-encoding genes from sequenced pathogen genomes or transcriptomes. Comprehensive datasets on herbivore transcriptomes and proteomes (Grbić *et al.*, 2011; Su *et al.*, 2012;

DeLay, 2012) will probably give rise to the discovery of new effectors in the near future (Table 1).

HOW COMMUNITY INTERACTIONS SHAPE THE ADAPTIVE PROCESS

Plants are often attacked by a diverse community of enemies, including herbivores and pathogens. Interspecific competition within phytophagous communities can be direct (e.g. interference through fighting) or indirect (e.g. using a resource deprives others of also using it) (Denno *et al.*, 1995). Because induction and suppression of defences manifest themselves in distal tissues (Kant *et al.*, 2008; Alba *et al.*, 2015), it is predicted that herbivores and pathogens will also interact indirectly through the changes in the plant elicited by their feeding (Ohgushi, 2005). These interactions may lead to decreased performance, but also to facilitation: herbivores were sometimes found to benefit from the presence of conspecific or heterospecific attackers (Faeth, 1986; Harrison and Karban, 1986; Karban, 1989).

Indirect interactions in phytophagous communities via induced defences

Responses induced by one particular species can result in resistance to another (Long *et al.*, 2007; Poelman *et al.*, 2008*a*; Mouttet *et al.*, 2013), whereas the order of herbivore arrival on the plant can influence the performance and number of herbivore species occurring later in the season (Van Zandt and Agrawal, 2004; Viswanathan *et al.*, 2005; Erb *et al.*, 2011). Hence, inducible plant defences can be major determinants of ecological interactions; in particular, defences depending on JA and SA appear to play important roles in determining community composition. For example, JA-deficient wild tobacco plants in the field were colonized by herbivores that normally ignore these plants (Kessler *et al.*, 2004), and studies in which plants were sprayed with synthetic phytohormones to assess the effect of induced defences on their ecology showed that such artificial induction can decrease the abundance of herbivores when JA is applied (Thaler *et al.*, 2001) or of pathogens when SA or SA mimics are applied (Inbar *et al.*, 1998). However, treating tomato plants with JA increased the growth of the pathogen *P. syringae*, whereas induction of SA responses enhanced the performance of the beet armyworm (*S. exigua*) (Thaler *et al.*, 1999). Moreover, when JA and SA responses were induced simultaneously the performance of the cabbage looper caterpillar (*T. ni*) increased, but not that of the thrips *Frankliniella occidentalis*, the spider mite *T. urticae* and *M. sexta* larvae (Thaler *et al.*, 2002). In addition to these field studies, experiments have also been carried out in the laboratory with model organisms to reveal the mechanisms that underlie indirect interactions. Using *Arabidopsis* mutants impaired in the JA, ethylene or SA-pathway, de Vos *et al.* (2006) found that feeding by the JA-inducing caterpillar *P. rapae* did not, contrary to expectation, induce resistance against the necrotrophic fungus *Alternaria brassicicola*. In contrast, it did reduce disease symptoms caused by *P. syringae*, but this effect was not (exclusively) dependent on JA, ethylene or SA. It also reduced infectiousness of the biotroph turnip crinkle virus due to an

ethylene-primed SA response (de Vos *et al.*, 2006). In addition Thaler *et al.* (2010) showed that *P. syringae* induced JA, SA and increased the activity of PIs in tomato plants and thereby reduced the growth of *S. exigua* caterpillars. Conversely, infection with tobacco mosaic virus induced only an SA response, causing induced susceptibility to *S. exigua* caterpillars and induced resistance to aphids (*M. persicae*). Herbivores feeding on different plant organs can also affect each other through the induction of plant responses (Soler *et al.*, 2013). Larvae of *P. rapae* grew more slowly on plants that were also infested with root-feeding nematodes (*Pratylenchus penetrans*) and this correlated with higher foliar glucosinolate levels (Van Dam *et al.*, 2005). Similarly, Soler *et al.* (2005) found that *P. brassicae* larvae developed more slowly on wild mustard (*Brassica nigra*) plants that were infested with the cabbage root fly (*Delia radicum*), and this in turn affected developmental rates of *Cotesia glomerata*, which is a parasitoid of the herbivore. Systemic signals that have been proposed to be involved in above–belowground interactions include the phytohormones JA, ABA, ethylene, auxin and cytokinin, but for none of these has a clear role been unequivocally established. This suggests that the simultaneous induction of different defences may affect community members differently.

Indirect interactions in phytophagous communities via suppressed defences

Not only induction, but also suppression of defences can affect interactions between herbivores and pathogens. In principle, the benefits of suppression by a single herbivore species can be shared by other species in the community. Within the spider mite species *T. urticae*, there is intraspecific variation in the ability to induce and suppress defences (Kant *et al.*, 2008). Some spider mites are very sensitive to the JA defences they induce in tomato plants. However, when these mites share their feeding site with other types that can suppress JA defences, their reproductive performance increases dramatically (Alba *et al.*, 2015). In addition, suppressor mites of the species *Tetranychus evansi* as well as non-suppressor mites of the species *T. urticae* perform better when they are introduced on leaves already suppressed by *T. evansi* than on uninduced control leaves (Sarmento *et al.*, 2011*a*,*b*). Similarly, whiteflies (*B. tabaci*) were found to suppress JA defences, and this was shown to improve the performance of spider mites on Lima bean (Zhang *et al.*, 2009) and to disrupt the attraction of natural enemies (Zhang *et al.*, 2009, 2013*a*). In turn, some parasitoids have adapted to locate hosts using SA-induced volatiles, the emission of which is not suppressed by whiteflies (Zhang *et al.*, 2013*b*). Soler *et al.* (2012) showed that the cabbage aphid (*Brevicoryne brassicae*) inhibited the production of JA in cabbage (*Brassica oleracea*) plants, and this led to increased growth and development of the large cabbage white butterfly (*P. brassicae*) on plants that had been co-infested with the two species. Finally, field-grown tomato plants were frequently infested with the two-spotted spider mite *T. urticae* and the tomato russet mite, *Aculops lycopersici*; the first of these induces JA and SA defences simultaneously, whereas the second induces only SA and suppresses the JA response. Spider mites had much higher reproductive performance on plants infested with

russet mites, an effect that was not due to the russet mite-suppressed JA response but to the antagonistic effect of the doubled SA response induced by both species. However, this same SA response inhibited infection by *P. syringae*. Hence, the overall effect of selective suppression one type of defence is determined by the presence of competing plant parasites and the distinct palette of defences these induce (Glas *et al*., 2014).

Indirect interactions among plant defence, herbivores and predators

During recent decades, the study of interactions between plants and other organisms has developed from the investigation of relatively simple interactions between plants and herbivores to that of more complex multitrophic interactions and their importance for the structure of communities of plants and arthropods. In nature, plants are part of complex food webs in which the sum of direct and indirect interactions between organisms, either allies or enemies, determines the selection pressures on plants. Plant defences against herbivores play an important role in these interactions (Hairston *et al*., 1960, Price *et al*., 1980, Janssen *et al*., 1998, Poelman *et al*., 2012). Plant defences comprise not only traits that interfere with herbivores directly (Walling, 2000), but also traits that operate indirectly by facilitating foraging predators and host-seeking parasitoids. These indirect plant defences often rely on the release of volatile compounds into the atmosphere, which can act as cues for prey-searching natural enemies (Dicke *et al*., 1990; Turlings *et al*., 1991).

Plant toxins ingested by herbivores may interfere with their natural enemies. Hence plant defence may actually provide a herbivore with additional protection. While sequestration is adaptive and entails storage of ingested plant toxins in specialized tissues or eggs (Duffey, 1980; Tooker and De Moraes, 2007), in principle any plant substance present anywhere in a herbivore can serve as antipredator protection. For example, tomatine incorporated in the diet of *H. zea* resulted in prolonged larval development, reduced pupal eclosion and size, while it reduced adult longevity of its parasitoid *Hyposoter exiguae* (Campbell and Duffey, 1979). Nicotine in the diet of *M. sexta* decreased survival of its parasitoid *Apanteles congregates* (Barbosa *et al*., 1982), while there is evidence that plants stop producing nicotine when attacked by nicotine-tolerant herbivores (Kahl *et al*., 2000; Voelckel *et al*., 2001), possibly to facilitate parasitoids. Alternatively, sometimes predators may themselves evolve resistance to plant toxins such as nicotine (Kumar *et al*., 2014). In addition, glucosinolates were found to affect the performance of the second and the third trophic level (Poelman *et al*., 2008b; Hopkins *et al*., 2009; Kos *et al*., 2012). Furthermore, Kauffman and Kennedy (1989) found that a high concentration of the methyl ketone 2-tridecanone in a particular accession of *Lycopersicon hirsutum* was toxic to the herbivore *H. zea* but much more to the herbivore's parasitoid *Campoletis sonorensis*. Moreover, the stinkbug *Podisus maculiventris* reared on *M. sexta* caterpillars avoided prey that had been fed on tomatine and chlorogenic acid (a phenolic) in an artificial diet (Traugott and Stamp, 1997). This shows that not only growth, development and mortality but also the behaviour of natural enemies can be modulated by plant toxins ingested by

their prey. There are also indications that JA-dependent plant metabolites can constrain herbivore predation. Thaler (1999) showed that treating tomato with JA increased parasitism of *S. exigua* by *H. exiguae* in the field, but the parasitoids developed more slowly on these caterpillars and gained less weight than control wasps. In addition, Kaplan and Thaler (2010) observed lower predation of the caterpillar of *M. sexta* by the predaceous stinkbug *P. maculiventris* on plants overexpressing JA-related induced defences in field experiments. However, manual application of JA forces the constitutive display of defences that are normally induced, and this may result in overestimation of the magnitude of the predator response.

Another layer of complexity is added when communities harbour one or more herbivore species that can suppress plant defences (Alba *et al*., 2011). Suppression of defences by one species can facilitate another competing species (Sarmento *et al*., 2011b; Alba *et al*., 2015). For instance, suppression of defences by specialized strains of the two-spotted spider mite *T. urticae* (Kant *et al*., 2008) by the red spider mite *T. evansi* (Sarmento *et al*., 2011b) or by the tomato russet mite *A. lycopersici* (Glas *et al*., 2014) benefits not only these species themselves but also defence-sensitive competing species inhabiting the same leaf or plant. Hence, suppression of plant defence can indirectly mediate competition between herbivores, forcing the suppressor to adopt strategies to reduce competition with opportunistic herbivores. For instance, the spider mite *T. evansi* produces more web in the presence of *T. urticae*, and this behaviour results in elimination of the competitor (Sarmento *et al*., 2011b). Suppression of plant defences also reduces the emission of induced plant volatiles (Rodriguez-Saona *et al*., 2003; Kant *et al*., 2008; Zhang *et al*., 2009; Schwarzberg *et al*., 2011; Sarmento *et al*., 2011a), although this does not always reduce the attraction of predators (Sarmento *et al*., 2011a). Moreover, suppression of plant defences by spider mites can potentially backfire in the presence of predatory mites, because these prefer the eggs of prey derived from plants with suppressed JA defences to those from plants with activated JA defences (L. M. S. Ataide, University of Amsterdam, Netherlands, unpubl. res.).

Like suppression, induction also affects the performance of competitors. When plants are simultaneously attacked by more than one herbivore species, the palette of plant defences these induce together will determine their mutual interactions and those with their natural enemies. For example, cutting and trenching prevent the plant from transporting photosynthates away from, and defence compounds towards, the tissue where the herbivore is feeding (Dussourd and Denno, 1991; Delaney and Higley, 2006; Oppel *et al*., 2009). This allows the herbivore to exploit a nutrient-rich part of the plant without having to deal with elevated plant defences. However, if the isolated tissue is large enough, other herbivores can profit from this resource as well, without having to invest in cutting a vein or digging a trench. Similarly, density-dependent feeding efficiency is also observed with gregarious feeding (Prokopy and Roitberg, 2001). For example, gregarious aphids can create sinks in plant tissue, which are preferentially supplied with nutrients by the plant compared with parts where individual aphids feed (Larson and Whitham, 1991). Also, the adult mass of froghoppers, a predictor of fecundity, peaks at intermediate juvenile group size (Wise *et al*., 2006). In this way, gregarious feeding

provides clear benefits because multiple herbivores can feed more efficiently than single herbivores. Interactions with the third trophic level may also shift when multiple herbivore species attack a plant simultaneously. For instance, when cabbage plants are simultaneously attacked by more than one herbivore species, the blend of volatiles released from these plants is different from that released by a singly infested plant. This new blend is less attractive to natural enemies of one of the herbivores and consequently adults of these herbivores preferred to oviposit on cabbage plants previously attacked by the other herbivore species, thus reducing the risk of parasitism of their offspring (Shiojiri *et al.*, 2002).

Finally, induced plant defences can also mediate indirect intra- or interspecific interactions among plants, and this possibly arises from competition between plants for enemy-free space. It has long been known that non-attacked plants close to attacked plants can be warned of imminent attacks through the volatile cues released by the neighbours (Baldwin and Schultz, 1983; Bruin *et al.*, 1992; Karban and Maron, 2002; Baldwin *et al.*, 2006). These volatiles prime the defences of neighbouring plants such that herbivores attacking these plants will trigger the induction of defences more rapidly and usually more strongly than in non-primed plants (Engelberth *et al.*, 2004). Since well-defended plants will deflect herbivores onto their less defended neighbour plants (McNickle and Dybzinski, 2013), the ability of plants to eavesdrop on attacked neighbour plants and be primed to an imminent herbivore attack can be considered an adaptation to increase the plant's competitive ability.

The examples mentioned above illustrate the importance of incorporating the indirect interactions between plants, herbivores and natural enemies in the study of the evolution of plant defences. Besides the interaction between plants and their herbivores, many other organisms can benefit or suffer, either directly or indirectly, from the defensive products induced by herbivores. The net effects of plant defences on plant fitness thus depend not only on the interaction of the plant with the inducing herbivore, but also on effects on plant fitness through the interaction web associated with the plant. The true fitness effects of plant defences can therefore only be evaluated in the natural environment in which the various interacting species have evolved.

Evolutionary consequences of defence suppression

Defence suppression is perhaps the most striking example of a herbivore strategy that affects the performance of other herbivores living on the same host plant. There are indications that defence suppression is not always restricted to the site of the suppressor, and may act systemically throughout leaflets (Kant *et al.*, 2008; Glas *et al.*, 2014; Alba *et al.*, 2015). For example, the performance of a non-suppressing strain of the spider mite *T. urticae* increased when feeding from a leaflet co-infested with a suppressor *T. urticae* strain (Kant *et al.*, 2008) or with the suppressor species *T. evansi* (Alba *et al.*, 2015), even when spatially separated from one another through a lanolin barrier. In addition, the performance of such non-suppressing *T. urticae* mites also increased on plants on which suppressor mites had been feeding previously but had been manually removed

(Sarmento *et al.*, 2011a). This demonstrates that the beneficial effects of defence suppression for competing herbivores are not restricted to the suppressor's feeding site or to the moment at which the suppressor is feeding. This raises a key question: why did herbivores evolve the ability to suppress plant defences beyond their feeding site, while the alternative, i.e. the evolution of resistance to plant defences, seems an equally effective but competitively much more attractive trait for herbivores living in communities?

There is no straightforward answer to this question yet, but ecological theory does allow speculation on the adaptive benefits of systemic and long-lasting defence suppression. First of all, herbivores may simply have to work hard to keep the monopoly of their feeding site, and limit the negative effects of interspecific competition as much as possible. For example, *T. evansi* produces massive amounts of dense webbing, impenetrable for competing species (Sarmento *et al.*, 2011b) and interferes with the reproduction of competitors (Sato *et al.*, 2014), whereas tomato russet mites (*A. lycopersici*) may avoid competitors by feeding preferentially between the dense leaf-hair forests of tomato (van Houten *et al.*, 2013; Glas *et al.*, 2014). These strategies will be effective against competing herbivore species, but not against intraspecific competition. One potential benefit for herbivores of extending plant defence suppression beyond their own feeding site could be kin selection (Hamilton, 1964): when the individuals that profit from the suppressed plant defence are the suppressor's relatives, inclusive fitness benefits for the suppressor can outweigh the costs of resource investment. Indeed, when individuals are more related to their neighbours, kin selection can theoretically increase such 'helping' behaviour, even when taking the negative fitness effects of stronger competition among kin into account (Mitteldorf and Wilson, 2000). However, a defence-suppressed area on a host plant is a public good (Rankin *et al.*, 2007) when accessible to all conspecifics, and this will allow unrelated individuals to take advantage of the situation as well. These 'cheaters' do not have to invest resources in suppressing plant defence but do take the benefit of increased performance. In general, the evolutionary stability of public goods depends on the degree of relatedness at the local site as well as the probability of unrelated individuals dispersing to this site. A potential model system for studying defence-suppressed host plants as public goods is the defence-suppressing spider mite *T. evansi*. It usually feeds and reproduces in the same area, which increases the chance that individuals are surrounded by relatives. Indeed, high relatedness is frequently observed in *T. evansi* populations (Boubou *et al.*, 2012).

Apart from intraspecific competition, interspecific competition can evidently also constrain the evolution of defence suppression when suppressors do not succeed in fully monopolising their feeding site (Glas *et al.*, 2014). Herbivores may promote competing species not only through feeding, but also through oviposition. For example, *A. thaliana* leaves treated with egg extracts of the butterfly *P. brassicae* had suppressed JA-induced defence responses, resulting in increased performance of the larvae of their competitor *Spodoptera littoralis* (Bruessow *et al.*, 2010). Interspecific competition can impose serious fitness costs on herbivores (Kaplan and Denno, 2007), and although some herbivores display behaviour (such as massive web production) associated with minimizing the

ecological costs of suppressing plant defences (Sarmento *et al.*, 2011*b*), such strategies can be costly physiological investments. Previous research suggests that web production by *T. urticae* spider mites shows a trade-off with reproduction (Tien *et al.*, 2009), which would indicate a direct fitness cost of web production. In addition, like defence suppression, the web is a public good as it protects not only individual web builders but conspecifics too. Web builders could therefore also be subject to intraspecific cheaters that do not invest in web production but nevertheless benefit from its protection (Oku *et al.*, 2009).

Given that the fitness benefits of defence suppression can vary with the ecological context, one may ask whether there is genetic variation for defence suppression within species. Kant *et al.* (2008) and Alba *et al.* (2015) obtained nearly isogenic lines from single populations of spider mites that were either suppressing or non-suppressing. Also, different populations of *H. zea* caterpillars were found to produce different amounts of glucose oxidase (Eichenseer *et al.*, 1999). This suggests that genetic variation for defence suppression can be present at the species level. Hence, considering that defence suppression is potentially adaptive, what maintains this variation? First, it is likely that plants from different families differ in their molecular pathways of direct defence (Schulz, 1988). Therefore, generalist herbivores are expected to adapt to their local host plants (Agrawal, 2000), which maintains genetic variation among different herbivore populations on different host plant species. Second, defence suppression could show a trade-off with life-history traits such as oviposition and web production. In this case, the shape of the trade-off determines whether disruptive selection on defence suppression may maintain genetic variation. Third, varying ecological circumstances, such as the presence of competitors or predators, intraspecific cheaters, the relatedness of neighbours and food availability, can alter the direct fitness effects of defence suppression. Because herbivores typically cannot control such factors, it is likely that the benefits of defence suppression vary dynamically over time and space, allowing the maintenance of genetic variation within populations.

Finally, it is appealing to think of defence suppression as an adaptation of the herbivore. However, some studies suggest this may not always be the case. For example, mathematical models suggest that hosts can evolve not to mount an immune response upon infection with a pathogen if the pathogen reacts to the immune response with increased virulence (Restif, 2013). In a plant–herbivore context such 'mafia behaviour' (Soler *et al.*, 1995) would mean that herbivores increase feeding upon experiencing induced plant defence. Although empirical studies of mafia behaviour are scarce (Ponton *et al.*, 2006), the costs of mounting an induced defence response under certain circumstances can be higher than its benefits (Fagerström *et al.*, 1987; Herms and Mattson, 1992). For example, when exposed to herbivores, *N. attenuata* plants typically initiate a JA-induced accumulation of nicotine, but the accumulation of nicotine is costly and decreases the competitive ability of the plant (Baldwin *et al.*, 1998). Hence, when larvae of the nicotine-tolerant herbivore *M. sexta* feed on *N. attenuata*, the plant responds by downregulating the accumulation of nicotine (Kahl *et al.*, 2000). Voelckel *et al.* (2001) argue that this response likely reflects an adaptation of the plant to shut down an inefficient and costly defence response, because using this energy to

compete with conspecifics may be more rewarding. Furthermore, when these plants attract parasitoids of *M. sexta*, any nicotine present in the herbivore may decrease the efficiency of this indirect defence response. Viewed this way, not mounting a direct defence response may be beneficial for plants because it prevents intoxication of their bodyguards (Fordyce, 2001; Kumar *et al.*, 2014). Indeed, whereas continued *M. sexta* feeding might downregulate nicotine accumulation in *N. attenuata*, the emission of volatile terpenoids that attract the parasitoids is upregulated (Kahl *et al.*, 2000). These examples show that investigating defence suppression from the plant's perspective can provide important new insights in the power balance between the plant and the herbivore. If defence suppression was solely an adaptation of the plant, there would not be a tragedy of the commons situation involved for the herbivore. We expect, however, that both plant and herbivore play an active role in this complex phenomenon, which would call for a coevolutionary approach to understand the evolution of defence suppression.

OUTLOOK

Plant defence suppression by herbivores is a largely unexplored phenomenon, but, together with induction, it may play a profound role in the plant-mediated indirect interactions that determine community structure in the phyllosphere. The vast majority of studies on defence suppression by plant eaters have focused on plant–pathogen interactions. These studies have predominantly focused on the mechanisms of defence suppression and the counter-adaptions of plants to undo such suppression. Although there may be many similarities in the mechanisms of defence suppression and the eco-evolutionary dynamics of traits underlying suppression, we do expect fundamental differences between the interactions of plants with immobile (pathogens) and mobile (herbivores) organisms. In this review we call attention to three main themes for future research.

1. How do herbivores suppress defences? Answering this question requires the transfer of molecular biology tools from phytopathology to the field of plant–herbivore interactions, especially to identify herbivore effectors and their *in planta* targets. This may deliver not only genetic markers that facilitate the study of their presence in (natural) populations, but also plant breeding targets to decrease the vulnerability of crops to key pest species that suppress plant defences, such as mites, aphids and whiteflies.
2. What are the consequences of suppression for community interactions? Answering this question requires long-term laboratory, greenhouse and field studies to assess the fitness costs and benefits in simple and more complex communities, not only for the herbivores, but also for the host plants on which they live. This knowledge may also have consequences for plant resistance breeding in conjunction with biological control strategies, e.g. when plant toxins decrease the beneficial effect of natural enemies more than is gained by decreasing herbivore performance directly.
3. Which conditions cause suppression to emerge, to persist or to disappear from populations? Answering this question requires ecological and evolutionary theory to make predictions on the invasion and population dynamics of

herbivores with such traits, as well as a more detailed knowledge of the traits that allow either suppression or resistance to align these predictions with their dynamics under experimental and natural conditions.

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