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Cross Tolerance to Biotic and Abiotic Stresses in Plants: A Focus on Resistance to Aphid Infestation

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Highlight Summary: Within natural environments plants respond to multiple biotic and abiotic stresses simultaneously, using an integrated signalling and response network that involves multiple points of reciprocal control. Here we explore how these multiple stress response pathways are managed and co-ordinated at a molecular

level to create short/medium term defences and long term memories of environmental hazards with a specific focus on how other biotic and abiotic stresses impact on plant-aphid interactions.

Abstract

Plants co-evolved with an enormous variety of microbial pathogens and insect herbivores under daily and seasonal variations in abiotic environmental conditions. Hence, plant cells display a high capacity to respond to diverse stresses through a flexible and finely balanced response network that involves components such as reduction-oxidation (redox) signalling pathways, stress hormones and growth regulators, as well as calcium and protein kinase cascades. Biotic and abiotic stress responses use common signals, pathways and triggers leading to cross tolerance phenomena, whereby exposure to one type of stress can activate plant responses that facilitate tolerance to several different types of stress. While the acclimation mechanisms and adaptive responses that facilitate responses to single biotic and abiotic stresses have been extensively characterised, relatively little information is available on the dynamic aspects of combined biotic/abiotic stress response. In this review, we consider how the abiotic environment influences plant responses to attack by phloem-feeding aphids. Unravelling the signalling cascades that underpin cross tolerance to biotic and abiotic stresses will allow the identification of new targets for increasing environmental resilience in crops.

Key words: aphids, secondary metabolites, nitrogen deficiency, drought, high light stress, UV irradiation, oxidative stress, reactive oxygen species

Introduction

Stress may be defined as any external factor that has a negative influence on plant growth and/or reproduction (Madlung and Comai, 2004). Environmental stresses have a significant negative impact on current agriculture. Under field conditions, commercially grown crops achieve an average of only about 50% of their potential yield due to the negative effects of abiotic environmental stresses such as drought, poor soil quality, temperature extremes and flooding (Hatfield and Walthall, 2015). Biotic stress also contributes significantly to the yield gap with field losses to insect pests estimated at more than 10% (Kerchev et al., 2012a) a figure that rises to 50-80% in the absence of control measures (Bruce, 2010).

In order to mitigate these diverse agricultural limitations, extensive effort has been expended examining the signalling and response pathways of plants to biotic and abiotic stresses. The majority of this work has necessarily focussed on single stresses in highly controlled environments in order to build our understanding of key processes and signalling elements. To date much less focus has been placed on the integrated response of plants to multiple stresses typically encountered under field conditions, however fundamental knowledge is now sufficiently advanced to tackle these questions. It is clear from studies on single stresses that there is significant overlap in signalling and response pathways to different biotic and abiotic stresses that include cellular redox status, reactive oxygen species, hormones, protein kinase cascades and calcium gradients as common elements (Atkinson and Urwin, 2012). This overlap in signalling pathways is associated with cross tolerance phenomena in which exposure to one type of stress enhances plant resistance to other biotic or abiotic stresses (Pastori and Foyer, 2002). These observations imply the possibility

of engineering or breeding for multiple stress resistance in crop plants. However, to achieve these goals a thorough understanding of how plants integrate information from multiple signals and optimise response to simultaneous stresses is required. In the present review we discuss knowledge concerning plant signalling and response to multiple stresses with particular reference to the impact of abiotic stresses on plant resistance to aphids.

Factors that underpin multiple stress resistance

Plants are able to withstand multiple mild and even severe environmental stresses simultaneously (Atkinson and Urwin, 2012). In a competitive growing environment a key factor underlying such resistance is the capacity of sessile plants to recognise, integrate and respond to biotic and abiotic environmental variables by constantly adjusting physiology and metabolism to optimise growth and reproduction in an ever changing environment. This capacity is facilitated by cross-tolerance phenomena, in which enhanced tolerance to a range of different environmental stresses is triggered by exposure to a single stress (Pastori and Foyer, 2002; Mittler, 2006). Cross-tolerance is achieved by the synergistic co-activation of the plant innate immune system involving a network of non-specific stress-responsive pathways that cross biotic-abiotic stress boundaries (Bostock, 2005). The innate immune system of plants additionally facilitates the induction of the systemic acquired immune response (SAR) that primes the immune response of tissues distal to the site of pathogen attack in a process analogous to that of cross-tolerance. Both cross-tolerance and SAR comprise an arsenal of inducible defences activated by stress perception and associated cell signalling pathways (Figure 1). Cross-tolerance and

SAR are linked in many cases to an enhanced production of reactive oxygen species (ROS) such as superoxide (O_2^-) and hydrogen peroxide (H_2O_2) that are perceived via thiol-modulated redox- and nitric oxide-mediated (NO) signalling pathways (Foyer and Noctor, 2009). For example, exposure to the atmospheric pollutant ozone generates ROS in the apoplast of plant cells, activating a plethora of biotic and abiotic stress responses through interactions with plant hormones such as ethylene (ET), salicylic acid (SA), abscisic acid (ABA), auxin and JA (Baier et al., 2005; Fujita et al., 2006; Bartoli et al., 2013a). This redox-hormone signalling hub, which receives and integrates information from a wide range of environmental stimuli is linked to downstream changes in gene expression that are presumably optimised to meet the prevailing environmental conditions as well as the developmental stage of the plant. The concept that the transcriptional response of a plant results from integration of all of the prevailing external factors is often overlooked in experiments where researchers focus on responses to a single variable. This is illustrated by a recent meta-analysis of the transcriptional response of Arabidopsis to aphid infestation (Foyer et al., 2015). Highly limited overlap in gene expression changes were observed between experiments even when comparing identical interactions between *M. persicae* and the Col-0 genotype and these were reflective of variability in other environmental variables such as growth temperature, day length, light intensity and relative humidity between laboratories (Foyer et al., 2015).

Changes in gene expression result in downstream consequences in plant developmental and defence programmes mediated via changes in protein and metabolite content. Plant metabolite composition is strongly impacted by the prevailing abiotic environment affecting the quality of plants as hosts for insect herbivores through both direct effects on the quality of the insect diet as well as

126 indirectly via their signalling function in plants. Conversely infestation by pathogens
127 or pests can induce specific compounds that may play a role in abiotic stress
128 signalling or adaptation.

129 The metabolite composition of leaves and other organs is considered to be an
130 important determinant of the success of aphid infestation. Aphid feeding can exert a
131 strong influence on leaf metabolite profiles (Foyer et al., 2012), greatly increasing
132 the contents of some stress-associated primary and secondary metabolites such as
133 trehalose (Hodges et al., 2013). Trehalose metabolism is important in sugar
134 signalling and underpins the regulation of carbon partitioning during plant responses
135 to abiotic stress (Nuccio et al., 2015). It also influences the resistance of *A. thaliana*
136 plants to *M. persicae* where loss of *TREHALOSE PHOSPHATE SYNTHASE11*
137 (*TPS11*) gene function, which is required for sugar signalling activities, prevented
138 trehalose accumulation in aphid-infested leaves and decreased resistance to aphid
139 infestation through modulation of the PAD4-dependent biotic stress response
140 pathways (Singh et al., 2011). Interestingly trehalose plays a role in starch
141 metabolism, where the external application of trehalose results in the accumulation
142 of starch in plant tissues and in addition to promoting trehalose accumulation, *M.*
143 *persicae* infestation of Arabidopsis results in local starch accumulation. In *tps11*
144 mutant lines that exhibited lower resistance to *M. persicae* than wild-type lines,
145 starch accumulation was impaired in response to aphid infestation. Similarly *pgm1*
146 mutant plants that were unable to accumulate starch due to impaired glucose
147 metabolism exhibited reduced *M. persicae* resistance (Singh et al., 2011). Taken
148 together these data suggest that changes at the primary metabolic level can have
149 broad pleiotropic effects on aphid susceptibility. Polyphenols are well known to
150 respond to abiotic stresses such as nutrient availability, drought, salinity, light and

151 temperature (Nakabayashi and Saito, 2015). Polyphenols and their oxidation
152 products are also considered important in aphid resistance (Miles and Oertli, 1993;
153 Lattanzio *et al*., 2000; Kerchev *et al*., 2012b) through the nonspecific formation of
154 radicals or by crosslinking of cell walls suggesting that certain abiotic environments
155 might induce a relatively broad and non-specific basal aphid resistance. Conversely,
156 many secondary metabolites are specific in their anti-aphid action. For example, the
157 aphid-induced indole alkaloid gramine accumulated only in response to infestation
158 by the aphid *Schizaphis graminum* on different barley genotypes with varying
159 resistance characteristics. Feeding by the russian wheat aphid (*Diuraphis noxia*), the
160 rose-grain aphid (*Metopolophium dirhodum*) or the bird cherry-oat aphid
161 (*Rhopalosiphum padi*) failed to elicit gramine accumulation. Gramine accumulation
162 was additionally triggered by exposure to abiotic drought stress or the addition of
163 ABA (Larsson et al., 2011) and drought stressed barley plants were a poorer host for
164 *Schizaphis graminum* than control plants (Cabrera et al., 1995).

165 Non-protein amino acids, such as 5-hydroxynorvaline that is induced in
166 maize (*Zea mays*) leaves in response to herbivory by aphids (*Rhopalosiphum maidis*,
167 the corn leaf aphid) and caterpillars (*Spodoptera exigua*, beet armyworm), and by
168 abotic stresses such as drought stress, can impede aphid reproduction (Yan et al.,
169 2015). Moreover, glucosinolates and the products of their hydrolysis by myrosinases
170 play important roles in constitutive and inducible defences in crucifers. In the
171 absence of stress, myrosinases and their substrates are not localised in the same cell
172 types, the enzymes are transported to the cells that contain glucosinolates in response
173 to mechanical damage and other triggers such as jasmonic acid (Thangstad et al.,
174 2004; Redovniković et al., 2008). Furthermore, it has been demonstrated that
175 feeding by *Myzus persicae* on Arabidopsis induces the accumulation of indole

glucosinolates and that the addition of indole glucosinolates to artificial diets reduces the fecundity of *M. persicae* (Kim and Jander, 2007). However the association between indole glucosinolates in plant tissues and aphid performance is less clear. For example, *atr1D* mutants of Arabidopsis that contain elevated levels of indole glucosinolates supported slower reproduction of *M. persicae* than wild-type plants (Kim *et al*., 2008). Similarly, drought caused a significant increase in the indole glucosinolate content of Arabidopsis plants as well as reduced aphid fecundity (Pineda *et al*., 2016). However a similar negative impact of drought treatment was observed in knockout mutant lines that were blocked in the production of indole glucosinolates (Pineda *et al.*, 2016). In our own experiments, treatment of kale with 1 mM methyl-jasmonate (Me-JA) resulted in increases of glucobrassicin (indol-3-ylmethylglucosinolate) and neoglucobrassicin (1-methoxy-indol-3-ylmethylglucosinolate) of more than 35- and 550-fold, respectively. However, aphid fecundity on Me-JA treated plants was significantly higher (Student's t-test, $p<0.05$) than that on untreated plants (Figure 2).

Camalexin, a characteristic indole alkaloid of Arabidopsis, is considered to be important in plant defences against bacteria, fungi and insects (Rogers et al., 1996; Kettles et al., 2013). For example, aphid reproductive performance was decreased on the *dcl1* Arabidopsis mutants, which accumulate high levels of camalexin (Kettles et al., 2013). However, aphid fecundity was increased in the *A. thaliana* phytoalexin-deficient *pad3* relative to the wild type plants (Glazebrook and Ausubel, 1994; Kettles et al., 2013). Camalexin accumulation has been observed under conditions that cause amino acid starvation or those inducing oxidative stress (Zhao et al., 1998).

201 **Plant responses to aphid infestation**

202 Aphids, which are the largest group of phloem feeding insects, are major
203 agricultural pests causing extensive damage to crop, garden and wild plants (Foyer et
204 al., 2015). During feeding, aphids secrete metabolites, proteins, pathogenic bacteria
205 and viruses into the host plant (Furch et al., 2015; Sugio et al., 2015; Whitfield et al.,
206 2015). While the feeding process is thought to cause relatively little damage to the
207 host plant tissues, the impact of feeding on vigour and productivity depend largely on
208 the intensity of infestation. In agricultural environments, aphid-induced damage
209 generally results in crop losses of about 15% (Leather et al., 1989). The majority of
210 damage is associated with their role as vectors for more than 100 disease-causing
211 viruses such as potato leaf roll virus and cucumber or cauliflower mosaic virus (van
212 Emden et al., 1969). Furthermore, because aphids feed exclusively on the phloem,
213 their diet is rich in sugar but relatively poor in nitrogen requiring the ingestion of
214 large volumes so that the insects can acquire sufficient nitrogen (Douglas 2006).
215 These large volumes of phloem sap are secreted as honeydew, which attracts
216 saprophytic fungi which colonise the leaf surface inhibiting photosynthetic
217 performance (Dedryver *et al.*, 2010).

218 In order to feed, aphids penetrate the leaf epidermis and probe between the
219 mesophyll cells with their piercing-sucking mouthparts that are called stylets to reach
220 the phloem sieve elements from which they feed (Figure 3). Along the stylet track
221 mesophyll cells are regularly probed and small amounts of cell content are ingested,
222 a behaviour that is believed to orientate the aphid stylet towards the phloem (Hewer
223 et al., 2011). Aphids produce a rapidly-gelling "sheath saliva" around the stylets
224 during probing activity which is rich in conjugated carbohydrates, phospholipids,
225 pectinases, phenoloxidases and β-glucosidases, all of which have the potential to

induce plant defence responses (Miles, 1999) however it remains unclear whether aphid sheath components are recognised by plant hosts (Bak *et al*., 2013). In addition to the rapidly-gelling sheath saliva, aphids also secrete "watery saliva" at the puncture points and feeding locations (Tjallingii, 2006). The enzymes present in the watery saliva prevent the induction of the plant wound responses in the penetrated tissues and so impede the repair of feeding-associated damage (Will et al., 2009). However, aphid saliva also contains components that act as elicitors that induce plant defence responses (Miles, 1999; de Vos and Jander, 2009). For example, Mp10, an elicitor present in green peach aphid saliva induces chlorosis and local cell death in *Nicotiana benthamiana* (Bos et al., 2010). Moreover, oligogalacturonides are released from the plant cell walls as a result of the action of enzymes secreted by the stylet sheath. Oligogalacturonides and other products of the cell wall breakdown have the potential to induce defence responses that limit aphid infestation (Heil, 2009). Proteins derived from endosymbiotic bacteria that have been found in aphid saliva may also participate in the elicitation of plant defence responses, for example GroEL a chaperonin associated with the obligate aphid endosymbiont *Buchnera aphidicola* triggers plant immunity resulting in reduced aphid fecundity on hosts (Chaudhary et al., 2014).

Plants perceive the presence of fungal pathogens mainly through the presence of chitin in the fungal cell wall, which acts as an elicitor. Chitin is also a major constituent of the insect exoskeleton and chitin oligosaccharides act as microbe-associated molecular patterns (MAMP), inducing a suite of responses which play important roles in defence against fungal pathogens (Boller and Felix, 2009; Wan et al., 2008). In Arabidopsis, plant perception of chitin is dependent on LysM RECEPTOR-LIKE KINASE 1 (LysM RLK1) that specifically binds

chitooligosaccharides released from fungal cell walls and insect exoskeletons by the action of chitinases. These important pathogenesis-related (PR) proteins are induced not only by biotic but also by abiotic stress (Ahmed et al., 2012). It has been suggested that in addition to catalysing chitin oligosaccharide release from pests and pathogens, plant chitinases may also release similar polysaccharides from endogenous glycoproteins. In support of this hypothesis Arabidopsis plants engineered to express *Trichoderma* endochitinase and hexoaminidase exhibit enhanced tolerance to several abiotic stresses however tolerance was lost in a LysM RLK1 mutant background (Brotman et al., 2012). The significance of endogenous chitinases was highlighted in a study in *Malus hupehensis* where infection by the fungal pathogen *Botryosphaeria berengeriana*, infestation by the apple aphid *Aphis citricota*, as well as treatment with SA, methyl jasmonate, and 1-aminocyclopropane-1-carboxylic acid increased the expression of *MHCHIT1*, a class I chitinase gene (Zhang et al., 2012). Transgenic tobacco plants that constiutively over-expressed *MHCHIT1* had enhanced resistance to *Botrytis cinerea* and to treatment with the drought-inducing compound, polyethylene glycol, suggesting that the pathways induced by the *MHCHIT1* gene product were involved in cross tolerance responses to abiotic and biotic stresses (Zhang et al., 2012).

The induction of MAMP-type responses is not specific to the detection of micro-organisms. Similar molecular patterns and related responses such as the hypersensitive response (HR) can be triggered by a range of abiotic and biotic stresses, including aphid feeding (Klinger et al., 2009; Villada et al., 2009). The oxidative burst that is characteristic of HR involves the production and accumulation of reactive oxygen species (ROS) as well as changes in calcium fluxes, leading to the production of pathogenesis-related (PR) proteins linked to genetically-programmed

276 cell suicide responses (Smith and Boyko, 2007). Relatively little is known about the
277 resistance responses that are mediated by the plant disease resistance (R) genes
278 involved in aphid resistance. Incompatible plant-pathogen interactions involve the
279 recognition of the products of avirulence genes produced by the attacking or
280 invading organism by R genes, most of which encode nucleotide-binding site
281 leucine-rich repeat (NBS-LRR) proteins (Martin et al., 2003; McHale et al., 2006).
282 For example, an NBS-LRR gene is thought to be involved in the incompatible
283 interaction between potato aphid (*Macrosiphum euphorbiae*) and tomato that leads to
284 poor aphid growth and reproductive performance (Rossi et al., 1998). Furthermore,
285 the *AIN* gene that mediates the hypersensitive response of M*edicao trunculata* to
286 *Acyrthosiphon kondoi* and *A. pisum* as well as the *AKR* and *TTR* genes which
287 specifically provide resistance to *A. kondoi* and *Therioaohis maculate* respectively all
288 map to a genomic region containing a cluster of NBS-LRR coding sequences
289 (Klinger et al., 2009). Similarly, the *VAT* gene encodes a NBS-LRR protein, which is
290 implicated in the resistance response of melon to the aphid *Aphis gossypii* (Villada et
291 al., 2009). Resistance responses dependent on the presence of the *VAT* gene included
292 apoplastic callose production, lignin decomposition and localised programmed cell
293 death (Villada et al., 2009; Dogimont et al., 2014).

294 In other plant species although R genes against insects have been defined
295 through genetic studies individual genes have not been identified and cloned. For
296 example, while several genes that confer resistance to fungi and rusts have been
297 cloned from wheat and mostly identified as NBS-LRRs, none of the 65 R genes
298 providing resistance to insects have been identified (Harris et al., 2015). In contrast,
299 a number of genes that act downstream of R genes in wheat have been identified and
300 functionally characterised. For example, the wheat genes *Hfr-1* and *Wci-1* encoding

lectins thought to interfere with feeding are expressed in response to Hessian fly (*Mayetiola destructor*) or bird cherry-oat aphid (*Rhopalosiphum padi*) in a biotype specific manner. Both of these genes additionally respond to treatment with SA or its analogue benzothiadiazole while *Wci-1* was also responsive to MeJA and ABA (Subramanyam et al., 2006). Within the context of abiotic-biotic stress crosstalk an interesting additional observation was that expression of *Wci-1* was upregulated by mechanical wounding while *Hfr-1* upregulation was observed following water-deficit. Similarly, the presence of the *Rag1* aphid resistance gene in soybean led to the constitutive expression of many defence-related transcripts, including those associated with ABA signalling. In resistant cultivars containing the *Rag1* gene, aphid feeding triggered the significant expression of only one additional gene, whereas aphid feeding in the susceptible cultivar caused increased abundance of many transcripts (Studham and MacIntosh, 2013).

The SA, ABA and JA signalling network is considered to be particularly important in triggering appropriate responses against herbivory (de Vos et al., 2005; Kerchev et al., 2013; Studham and MacIntosh, 2013; Hillwig *et al*., 2016). While each hormone has a defined role to play in activating defences, the dynamic adjustment of the relative contribution of each pathway is required to ensure that elicited defence responses are appropriate to prevailing biotic and abiotic environments. SA is required for the induction of effective defences against biotrophic and hemi-biotrophic pathogens. Plants exposed to pathogens, herbivores and to abiotic stresses accumulate SA and PR proteins such as β-1,3-glucanase (Loake and Grant, 2007). However, SA does not provide an effective defence against necrotrophic pathogens (Coquoz et al., 1995; Yu, et al., 1997), which require activation of JA-dependent responses. The NON-EXPRESSOR OF PR1

(NPR1) protein is important in the elaboration of SA-mediated defence responses (Kinkema et al., 2000; Mou et al., 2003). *NPR1* and *NPR1*-related transcripts such as *MhNPR1* in apple were increased in response to a range of different abiotic and biotic stresses including aphid infestation (Zhang et al., 2014). Although overexpression of AtNPR1 decreased dehydration and salt tolerance in rice (Quilis et al., 2008), the constitutive expression of *MhNPR1* in tobacco enhanced tolerance to salinity and drought stresses, together with increasing resistance to *Botrytis cinerea* (Zhang et al., 2014).

In addition to its functions in protection against invasion by necrotrophic pathogens, the JA-dependent pathways of defence are associated with wounding and responses to herbivory (Creelman and Mullet, 1995; Devoto and Turner, 2005). Although many studies show that JA and SA act in an antagonistic manner in the regulation of plant defences (Spoel et al., 2003), abiotic stress-associated oxidative signalling can induce both pathways together (Han et al., 2013a). ABA has roles in oxidative signalling and protection against aphids (Kerchev et al., 2013, Studham and MacIntosh, 2013). ABA, which can act antagonistically to SA (Ton et al., 2009; Zabala et al., 2009), is important in drought and key physiological responses such as stomatal closure, via the activation of NADPH oxidases (Kwak et al., 2003; Petrov and Van Breusegem, 2012). Mutants defective in ABA biosynthesis such as *aba2* fail to accumulate JA or associated oxylipins following pathogen challenge (Adie et al., 2007). Furthermore *aba2* mutants support smaller aphid colonies than the wild type controls (Kerchev et al., 2013). The *aba1* mutant also supported reduced aphid colonisation associated with increased accumulation of the indole glucosinolates glucobrassicin and 4-methoxy glucobrassicin (Hillwig *et al*., 2016). Both of these compounds and particularly 4-methoxy glucobrassicin are toxic when provided in

351 artificial diets (Kim and Jander, 2007). However, as discussed above *M. persicae* can
352 tolerate high concentrations of indole glucosinolates *in planta* (Figure 2).

353 Many hormones such as auxin and ABA promote ROS production as part of
354 their mechanism of action through the activation of superoxide-producing enzymes
355 such as NADPH oxidases, also called respiratory burst oxidase homologues (RBOH)
356 (Bartoli et al., 2013b; Xia et al., 2015). For example, the RbohD and RbohF proteins
357 were found to be important in generating a ROS burst and long-distance systemic
358 signal following aphid infestation (Miller et al., 2009; Jaouannet et al., 2015).
359 Together with cell wall peroxidases that also produce ROS, germin-like oxalate
360 oxidases, and polyamine oxidases these enzymes generate an oxidative burst in the
361 apoplast (Bolwell et al., 2002; Torres et al., 2002; Apel and Hirt, 2004; Sierla et al.,
362 2013). The steady state concentrations of ROS within the cytoplasm are generally
363 very low because of the activity of an elaborate network of low molecular weight
364 antioxidants and antioxidant enzymes (Noctor and Foyer, 1998; Foyer and Noctor,
365 2009). Plant cells contain many low molecular weight antioxidants such as ascorbic
366 acid and glutathione. The capacity of the cellular antioxidant defences including the
367 activities of enzymes such as superoxide dismutases (SOD), ascorbate peroxidases
368 (APX), glutathione peroxidases (GPX), catalases (CAT) and peroxiredoxins (PRX)
369 (Noctor and Foyer, 1998; Foyer and Noctor, 2005) are important in regulating the
370 innate immune response to aphids and other pathogens. Mutants that are defective in
371 antioxidant enzymes, or that have a low abundance of ascorbate, show enhanced
372 resistance to biotrophic pathogens (Pavet et al., 2005). For example, mutants lacking
373 the major leaf form of catalase (*CAT2*) exhibit enhanced resistance to bacterial
374 pathogens (Chaouch et al., 2010), together with constitutive activation of
375 pathogenesis-related (PR) genes and lesion development linked to SA accumulation

376 (Chen et al., 1993; Chamnongpol et al., 1998). Similarly, leaves with low ascorbate
377 show enhanced resistance to aphid infestation (Kerchev et al., 2013).

378 ROS signals are in part mediated through GSH-dependent post-translational
379 modifications of signalling proteins (Mhamdi et al., 2013; Han et al., 2013 a, b) as
380 well as through protein kinase signalling cascades (Apel and Hirt, 2004; Foyer et al.,
381 2015). Different components of mitogen-activated protein kinase (MAPK) cascades
382 that comprise of MAPK, MAPK kinase (MAPKK/MKK) and MAPKK kinase
383 (MAPKKK/MEKK) are activated by H_2O_2. For example, the MEKK1–
384 MKK4/MKK5–MPK3/MPK6 signalling cascades that regulate pathogen defences
385 via regulation of transcription factors such as WRKY22 and WRKY29 (Asai et al.,
386 2002) are responsive to oxidative signalling (Rentel et al., 2004; Nakagami et al.,
387 2005; Xing et al., 2008; Pitzschke et al., 2009;). There are more than 80 MAPKKK
388 genes in the *A. thaliana* genome and most have been implicated in plant defence
389 responses (Taj et al., 2010). Furthermore, systematic transcriptional analyses of
390 aphid infestation in Arabidopsis revealed a significant role for MAPK cascades in
391 plant responses to this stress (Foyer et al., 2015). The roles of cell wall associated
392 kinases (WAKS) and Domain of Unknown Function (DUF)26 receptor-like kinases
393 in the responses of Arabidopsis leaves to aphid infestation was highlighted in a
394 recent metadata analysis of available transcriptome responses to aphid infestation
395 (Foyer *et al*., 2015).

396 Protein phosphatases, which regulate the degree of protein phosphorylation,
397 participate in cell signalling, particularly in oxidative and stress-regulated pathways
398 (He et al. 2004; Nakagami et al., 2005; Segonzac et al. 2014), as well as in wounding
399 responses (Rojo et al. 1998). Protein phosphatase (PP)2A, which has been shown to
400 regulate oxidative signalling leading to the elaboration of pathogen responses (Li et

al., 2014), also plays a role in plant resistance to aphids (Rasool et al., 2014). PP2A-B'γ was found to function downstream of metabolic ROS signals and act as a negative control of SA-linked responses in *A. thaliana* (Trotta et al., 2011; Li et al., 2014). Moreover, metabolite profiling analysis indicated that PP2A-B'γ modulates amino acid and secondary metabolism, particularly camalexin synthesis under oxidative stress (Li et al., 2014).

Biotic and abiotic factors alter the expression of a large number of transcription factors. For example, the *A. thaliana* homeodomain-leucine zipper (HD-Zip) transcription factor, ATHB13 influences resistance to both biotic and abiotic stresses (Gao et al., 2014; Cabello et al., 2012; Cabello and Chan, 2012). While Arabidopsis plants in which this transcription factor was overexpressed by activation tagging had a similar susceptibility to *Pseudomonas syringae*, they were found to exhibit a higher resistance to *M. persicae* and downy mildew (Gao et al., 2014). Similarly, the WRKY53 transcription factor, which is expressed in response to biotic and abiotic stress triggers in cereals, contains upstream *cis*-acting regulatory elements responsive to environmental cues such as drought and ultraviolet radiation (Van Eck, et al., 2014). Downstream targets of WRKY53 include components involved in HR such as the ORK10/LRK10 Ser/Thr receptor kinase and the apoplastic peroxidase POC1, which are expressed in response to pathogens and aphids (Van Eck, et al., 2014).

The expression of Redox Responsive Transcription Factor1 (RRTF1) is increased by ROS-generating necrotrophic pathogens but not by biotrophic and mutualistic infections (Matsuo et al., 2014). Moreover, transgenic lines overexpressing RRTF1 showed increased susceptibility to *Alternaria brassicae* infection (Matsuo et al., 2014). In contrast, aphid fecundity was reduced on null

426 mutants deficient in the RRTF1 protein compared to that on the wild type controls,
427 even though RRTF1 transcripts were greatly increased within the first hours of aphid
428 feeding (Kerchev et al., 2014).
429
430 **Effects of the abiotic environment on plant responses aphids**

431 While it has been postulated that abiotic stress increases herbivory, a meta-
432 analysis of insect performance on woody plants subjected to drought, pollution,
433 and/or shading, showed that overall these stresses had few significant effects on
434 insect growth rates or other parameters such as colonization density (Koricheva et
435 al., 1998). However, this analysis also revealed that abiotic stresses adversely
436 affected chewing insects and that the reproductive potential of phloem feeding
437 insects was reduced by drought (Koricheva et al., 1998). Moreover, much uncertainty
438 remains concerning how climate change will alter the performance of insect
439 herbivores and influence plant resistance to aphids and other insects. The
440 development of the *Brassica* specialist feeder, *Brevicoryne brassicae* was not greatly
441 altered by elevated plant growth temperatures, such as those that might be predicted
442 to occur as a result of climate change. However, the weights of *M. persicae* adult and
443 progeny were lower at the higher temperatures and the development time was
444 decreased (Himanen et al., 2008).

445 Like global temperatures, atmospheric carbon dioxide (CO_2) levels are
446 increasing annually. Growth under high atmospheric CO_2 typically decreases plant
447 tissue N contents while increasing C/N ratios but these effects had little impact on
448 aphid performance on oilseed rape (Himanen et al., 2008). However, in a free air
449 enrichment (FACE) study of pea aphid (*Acyrthosiphon pisum*) performance on *Vicia*

450 *faba*, the atmospheric composition had a significant impact on aphid performance in
451 a genotype-dependent manner. One genotype was unaffected by enrichment of
452 either CO_2, O_3 or both gasses together however, a second genotype was significantly
453 more abundant when CO_2 and O_3 were enriched simultaneously although enrichment
454 of either gas in isolation had either no (CO_2) or a negative (O_3) impact on aphid
455 abundance (Mondor et al., 2005). However, it was unclear whether differences in
456 aphid fecundity were due to the direct impact of altered atmospheres or via indirect
457 influences on the host plant. In a recent report, *M. trunculata* plants grown at
458 ambient temperature (26^{o}C) with CO_2 fertilisation (640 μmol mol^{-1}) were observed
459 to have a significant increase in both total and essential amino acids relative to plants
460 grown at ambient CO_2 (400 μmol mol^{-1}) concentrations. Plants grown under
461 elevated CO_2 were more suitable hosts for *A. pisum* than those grown at ambient
462 CO_2; however, when plants were grown at elevated temperature (30^{o}C) the effect of
463 CO_2 fertilisation on amino acid content was lost as was the enhanced susceptibility of
464 plants to aphid infestation (Ryalls et al., 2015) suggesting that at least under some
465 conditions effects may be plant mediated. These data further illustrate the complexity
466 of biotic-abiotic crosstalk under variable environmental conditions and demonstrate
467 potential difficulties in predicting herbivore pest status under changing
468 environments. Considerable cross talk exists between plant responses to ozone and to
469 aphids leading to speculation that future selection of ozone-resistsnt cultivars may
470 also influence the ability of plant defences to prevent infestation (Menendez et al.,
471 2009).

472 While the relative importance of abiotic and biotic soil components can differ
473 between plants and their herbivores, a study of the interactions between the aphid
474 *Schizaphis rufula* and its host dune grass *Ammophila arenaria* revealed that aphid

475 population characteristics were dependent on the abiotic properties of the soils in
476 different growing regions, irrespective of whether soil biota were present
477 (Vandegehuchte et al., 2010). Moreover, herbivore-induced resistance is likely to be
478 constrained in plants growing on degraded soils because of JA-linked responses to
479 prevailing abiotic and biotic stresses (Held and Baldwin, 2005). Of the abiotic
480 properties of the soils, the availability of water and essential nutrients such as
481 nitrogen and phosphate are the most important in determining plant growth and
482 productivity (Comadira et al., 2015).

483 *Nitrogen availability*

484 Soil nitrogen contents can have a strong influence on aphid fecundity (Gash,
485 2012), as well as influencing the competition between phytophagous species. For
486 example, the presence of leaf-chewing insects had a negative impact on aphid
487 infestation on plants growing on all fertilizer treatments, except for ammonium
488 nitrate fertilizer treatment (Staley, et al., 2011). The availability of essential nutrients
489 in the soil is likely to have a significant impact on the success of herbivores because
490 of direct effects of host nutrient availability on the diet, as well as on plant
491 composition of secondary metabolites and on the nature of preformed and inducible
492 defences. Herbivore feeding itself can cause carbon and nitrogen allocation changes
493 in plants that are exacerbated under conditions of nitrogen deficiency. Moreover, the
494 emission of volatiles is decreased in plants grown with low fertilization (Gouinguene
495 and Turlings, 2002). In situations where essential resources such as nitrogen and
496 phosphate are scarce, one might predict that the plant response to aphids is adjusted
497 by shortages in essential metabolites. The specialist aphid *Rhopalosiphum padi*
498 performed more poorly on N-limited barley seedlings, with aphids taking longer to
499 locate the phloem (Ponder *et al*., 2000). Similarly, the generalist feeder *M. persicae*

500 was unable to establish a successful infestation of nitrogen-deficient barley plants
501 even though the leaves were found to be rich in amino acids, sugars and tricarboxylic
502 acid cycle intermediates (Comadira et al., 2015). Nitrogen deficiency has a large
503 impact on leaf transcriptome profiles, such that transcripts encoding cell wall, sugar
504 and nutrient signalling, protein degradation and secondary metabolism are over-
505 represented in nitrogen-deficient leaves. The extensive reorganisation of leaf
506 metabolism and gene expression that occurs under nitrogen deficiency induces
507 defences that protect the metabolite-rich nitrogen-deficient leaves from *M. persicae*
508 attack (Comadira et al., 2015). Some significant similarities were observed between
509 the gene expression profiles of N-deficient barley leaves and those of *A. thaliana*
510 leaves infested by *M. persicae* (Foyer *et al*., 2015). For example, transcripts encoding
511 WRKY 18, 33, 40, 51 and 53 were significantly induced following either N-
512 limitation in barley or by aphid infestation in Arabidopsis leaves. Conversely, while
513 the transcript data show that N-limitation resulted in higher levels of flavonoid
514 metabolism transcripts in barley, flavonoid metabolism was effectively suppressed
515 by *M. persicae* feeding in *A. thaliana* leaves (Foyer *et al*. 2015). Transcripts
516 encoding WAKs and DUF26 kinases were significantly abundant in both stress
517 situations, adding support to the hypothesis that WAKs, DUF26 kinases and WRKY
518 transcription factors play important roles for basal resistance to aphids (Foyer *et al*.,
519 2015).

520 The presence of the root nematode, *H. schachtii* decreased aphid performance
521 on *A. thaliana* when nitrate levels were low but not under conditions of higher nitrate
522 fertilization (Kutyniok et al., 2014). While host choice by the aphids was not
523 influenced by the presence of nematodes under the higher nitrate fertilization regime,
524 the aphids preferred nematode-free plants to nematode-infested plants under the

525 lower nitrogen conditions (Kutyniok et al., 2014). The presence of aphids on the
526 shoots enhanced nematode infestation compared to controls under the low but not
527 high nitrate availability, (Kutyniok and Müller, 2013), suggesting that the carbon-
528 nitrogen interactions in the roots and shoots exert a strong influence on herbivore
529 preferences and the susceptibilities of roots and shoots to herbivory.

530 *Water availability, drought and salinity*

531 Drought can have a strong negative influence on the success of phloem
532 feeding insects (Koricheva et al., 1998) although it has additionally been proposed
533 that under conditions of pulsed water stress such insects can perform better than on
534 unstressed plants (Huberty and Denno, 2004). Interestingly, aphid performance was
535 found to be highest in *Brassica* plants subjected to moderate drought stress (Tariq et
536 al., 2013). Moreover, plant water status in *B. oleracea* did not have a great influence
537 on the ability of the specialist *Brevicoryne brassicae* to induce leaf glucosinolate
538 accumulation although it was significant with respect to glucosinolate accumulation
539 following feeding by the generalist *M. persicae*. While the responses of plants
540 infested with *B. brassicae* were not changed by water availability (flooding or
541 drought), the ability of plants to induce this response following *M. persicae*
542 infestation was negatively affected by both treatments (Khan et al., 2011). High
543 salinity led to a significant decrease in aphid fecundity on cotton plants, an effect that
544 was linked to increased levels of secondary metabolites such as flavonoids (Wang et
545 al., 2015).

546 Any negative impact of drought on aphid performance is likely to be related
547 to increases in ABA and ABA-signalling pathways that are known to decrease aphid
548 fecundity (Kerchev et al., 2013). Protein elicitors such as harpin are able to induce
549 plant SAR and HR responses, including resistance to the green peach aphid and can

550 also trigger drought tolerance through ABA-dependent pathways. For example,
551 constitutive over-expression of the harpin-encoding gene, *HRF1* in rice enhanced
552 drought tolerance through abscisic acid (ABA) signalling (Zhang et al., 2011).
553
554 *Light intensity and quality, including UV irradiation*
555 Although light is an essential driving force for photosynthesis, excess light
556 has a damaging impact on photosynthetic efficiency by inducing photoinhibition and
557 producing transcriptome changes indicative of a wide-ranging stress response (Foyer
558 et al., 1994; Niyogi, 1999; Suzuki et al., 2012). Signals concerning light availability
559 arise in the chloroplast and are transmitted to the nucleus in order to regulate gene
560 expression (Karpinski et al., 2013). High light stress triggers oxidative signalling,
561 MPK3/MPK6, lipoxygenase and hormone signalling, particularly through SA, ABA
562 and auxin-dependent pathways (Mühlenbock et al. 2008; Suzuki et al. 2012). For
563 example, singlet oxygen ($^1O_2$) generated by the photosynthetic electron transport
564 chain triggers signalling pathways leading to defence responses including
565 programmed cell death (Lee et al., 2007). The plant response to high light is
566 qualitatively similar to HR (Chang et al. 2009; Frenkel et al. 2009) leading to SAR
567 (Nomura et al., 2012) and systemic acquired acclimation (SAA) responses (Mateo et
568 al. 2004; Rossel et al. 2007). Moreover, plants pre-treated with high light retain a
569 "memory" of the high light stress that persists when plants are returned to low light
570 conditions (Szechyńska-Hebda et al., 2010; Zhao et al., 2014). The creation of such
571 "light memory" signalling pathways is poorly understood but ROS, hormonal and
572 electrophysiological signalling are thought to have important roles (Szechyńska-
573 Hebda et al., 2010). Growth under high light also increases the levels of secondary
574 metabolites, raffinose, polyamines and glutamate in leaves (Edreva et al., 2008;

575 Zavala and Ravetta, 2001; Wulff-Zottele et al., 2010; Jänkänpää et al., 2012) and
576 light quality also has a marked effect on leaf metabolite profiles (Kopsell and Sams,
577 2013). *M. persicae* fecundity was similar on tobacco plants when infestation
578 occurred in plants grown under high or low light levels, presumably because the
579 high-light grown leaves had more amino acids and sugars compared to those grown
580 under low light (unpublished data). In contrast, aphid fecundity was decreased when
581 infection took place on Arabidopsis plants that had previously been grown under
582 high light (1000 μmol m^{-2} s^{-1}) and returned to low light (250 μmol m^{-2} s^{-1}) conditions
583 (Rasool et al., 2014).

584 The content of UV-B radiation within the light spectrum can also have an
585 impact on herbivory. Growth under UV-irradiation altered the attractiveness *B.*
586 *oleracea* plants to herbivorous insects such as thrips, whiteflies, and aphids
587 (Kuhlmann and Müller, 2009). However, the fecundity of the green peach aphid was
588 significantly decreased on the *B. oleracea* plants grown under high (80%) and low
589 (4%) UV-B levels compared to ambient UV-B (Kuhmann and Müller, 2010). In
590 contrast, the reproduction of specialist cabbage aphid (*Brevicoryne brassicae*) was
591 decreased only under high UV-B (80%) levels (Kuhmann and Müller, 2010).

592

593 **Discussion and perspectives**

594 It has long been supposed that plants experiencing adverse environmental
595 conditions are likely to be more susceptible to attack by herbivores and pathogens.
596 Certainly herbivore performance and behaviour are affected by the quality of their
597 host plants, which in turn is determined by the prevailing environmental conditions.
598 However, in many cases even mild exposures to abiotic stresses trigger innate
599 immune responses and so enhance plant defences. Each stress influences the

morphological, metabolic, transcript and protein landscapes of the leaves and other organs in ways that show a high degree of overlap with the responses to other stresses allowing for cross tolerance phenomena. In reality, relatively few stress-specific signalling pathways have been found in plant responses to biotic and abiotic triggers. The use of common signalling pathways that enhance general defences to a wide range of stresses dictates that exposure to a single environmental stress is sufficient to trigger rapid defence responses to a range of stresses as well as generating epigenetic memories of stress that can persist from generation to generation. Plant responses to aphids therefore involve overlap and interaction points between hormone, redox, nitric oxide, kinase and calcium signalling pathways that have common features with abiotic stress responses. The analysis of current literature discussed above suggests that few stresses pre-dispose plants to aphid infestation.

Most of the common plant defence responses to the imposition of abiotic stress such as decreased growth and enhanced production of secondary metabolites are likely to have a negative impact on the ability of aphids to colonise and thrive on their plant hosts. In particular, abiotic stresses that lead to strengthening of the cell wall and/or altered accumulation of assimilate in the phloem are likely to impede aphid feeding. It is therefore important to understand the impact of abiotic stress on factors that are crucial to aphid success. Climate change factors such as elevated atmospheric CO_2 concentrations might diminish aphid success, particularly if the higher capacity for carbon gain achieved by the inhibition of photorespiration is accompanied by nitrogen limitation and limitations on primary nitrogen assimilation (Foyer et al., 2009).

624 Redox regulation and signalling through different pathways, particularly
625 thiol-mediated post-translational modification processes, is important in the
626 regulation of growth and defence responses because it is intrinsically linked to the
627 action of hormones such as ABA, SA and JA that facilitate resistance to different
628 pathogens and herbivores. Redox regulation is also likely to participate in a raft of
629 different epigenetic control mechanisms that influence the plant response to aphids.
630 For example, processes such as S-glutathionylation of histones and GSTs, together
631 with GSH - and glutaredoxin-dependent mechanisms for the reductive activation of
632 methionine sulfoxide reductases that facilitate the reduction of methionine sulfoxide
633 to methionine, provide an additional layer of stress-mediated control of gene
634 regulation. We have previously highlighted the importance of ascorbate as a major
635 redox buffer in priming leaf local and systemic transcript profile responses to aphids
636 (Kerchev et al. 2013). Ascorbate is also an important co-factor for the 2-oxoglutarate
637 dehydrogenase family of enzymes that includes the ten-eleven translocation (TET)
638 methylcytosine dioxygenases. These enzymes catalyze the conversion of 5-methyl
639 cytosine (5-mC) to 5-hydroxmethyl cytosine (5-hmC), which is considered to be the
640 initial step of active DNA methylation. Moreover, ascorbate has been shown to be a
641 reprogramming enhancer in animals because of its ability to induce a blastocyst-like
642 state in embryonic stem cells, promoting widespread DNA demethylation in gene
643 promoters by modulating epigenetic modifiers (Gao et al., 2015). In this way, the
644 impacts of biotic and abiotic stresses on the cellular ascorbate pool and the redox
645 state of the cell can therefore generate molecular memories of stress with lasting
646 consequences.

647 Recent advances in molecular genetic techniques mean that we are close to
648 the identification of receptors and the associated cell signalling pathways that

underpin many stress-induced responses that influence aphid performance. For example, a better understanding of the stress-dependent regulation and functions of wall-associated kinases is likely to provide new insights into the biotic/abiotic stress interactions that influence aphid fecundity.

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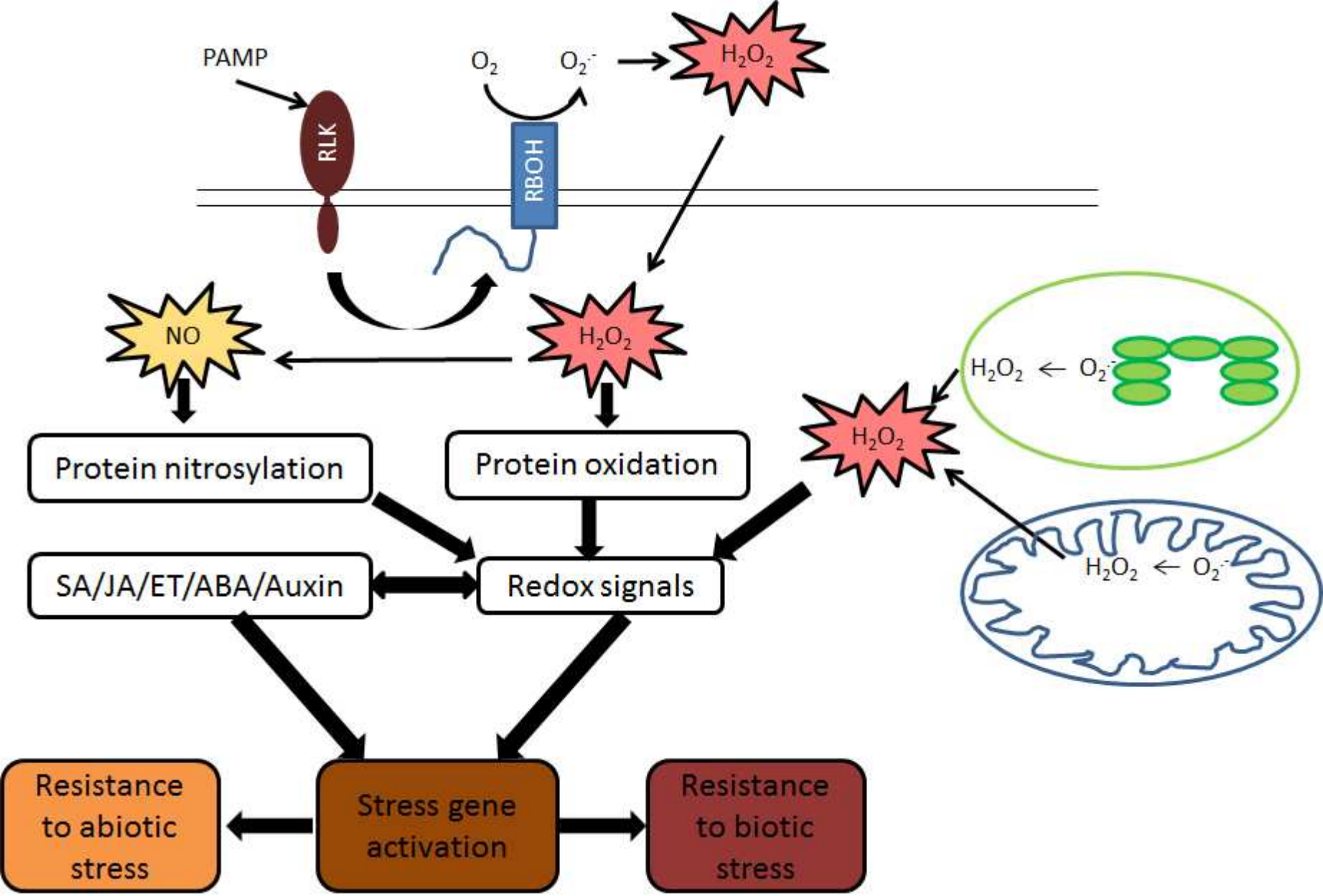
Figure Legends

Figure 1 Schematic model of the perception and common signalling pathways that trigger enhanced biotic and abiotic stress cross tolerance. Biotic threats are frequently perceived by the recognition of pathogen associated molecular patterns (PAMP) by receptor-like kinases (RLK) which in turn activate respiratory burst oxidase homologues (RBOH) via Ca^{2+} and kinase signalling cascades (not shown) leading to the accumulation of apoplastic ROS that diffuse across the plasma membrane to enter the cytoplasm. Unfavourable abiotic environments similarly result in the accumulation of ROS primarily produced in organelles such as the chloroplasts (Chl), mitochondria (Mit) and peroxisomes (not shown). ROS accumulation promotes NO production and NO can react with $O_2^{\cdot-}$ to produce other reactive nitrogen species (RNS). ROS and RNS react with protein thiol groups providing one of the perception mechanisms for redox signals that promote hormonal signalling leading to the combined activation of stress associated genes and subsequent tolerance to biotic and abiotic stress.

Figure 2 The impact of methyl-jasmonate treatment on indole glucosinolate content and aphid fecundity on curly kale. Plants were grown under glass for three weeks prior to treatment with 1 mM methyl-jasmonate or water (control) as a foliar spray. Five days after treatment 5 plants were harvested and the relative quantity of indole glucosinolate estimated by LC/MS as previously described (Panel A, Viger *et al*., 2015). Ten further plants were transferred to controlled environment chambers and a single one-day *M. persicae* nymph (genotype G) was applied to each plant which were caged as previously described (Kerchev *et al.*, 2012b). Following 15 days, the total number of aphids present were recorded (panel B). Bars represent mean values ± SE.

Figure 3 Schematic of potential elicitor release during aphid feeding. Hydrolytic enzymes in gelling sheath saliva have the capacity to release cell wall oligosaccharides allowing 'damaged self' recognition and furthermore, sheath proteins and peptides can be recognised by the plant immune system and will be present both in the apoplast and through the function of sheath saliva in sealing cell puncture wounds small amounts will also be present intracellularly. proteins and

peptides from watery saliva will be primarily present in cells punctured epidermal, mesophyll and companion cells punctured during location of the sieve element as well as within the sieve elements themselves. Similarly, proteins of bacterial symbiont origin will be localised within these cells.


PAMP
RLK
RBOH
O_2
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H_2O_2
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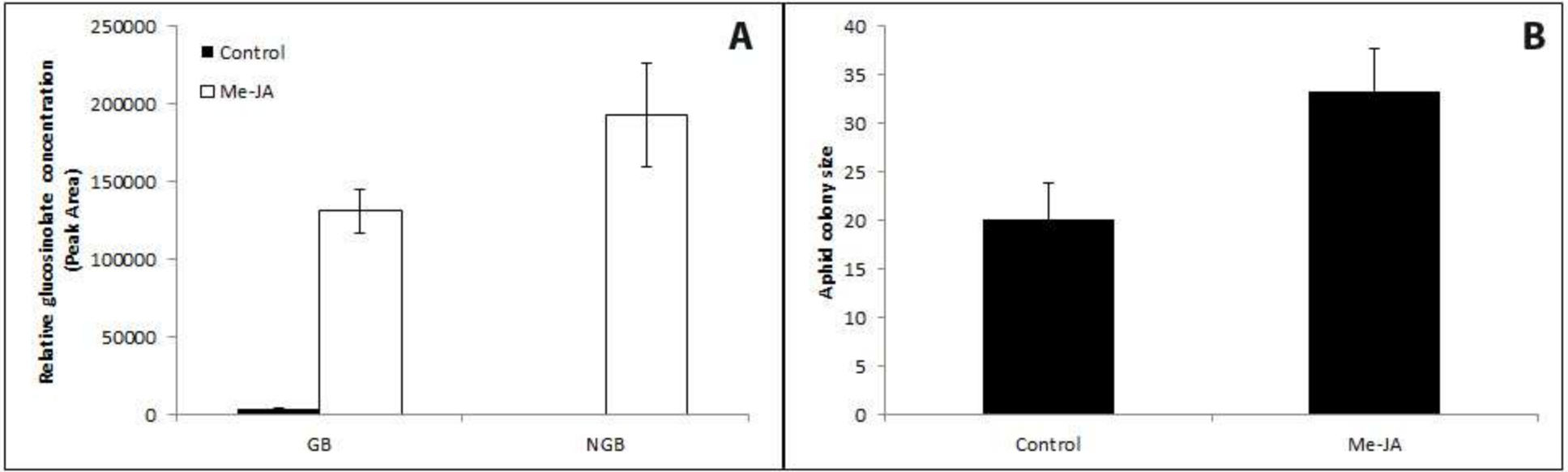
Protein oxidation
SA/JA/ET/ABA/Auxin
Redox signals
Resistance to abiotic stress
Stress gene activation
Resistance to biotic stress


A
Relative glucosinolate concentration (Peak Area)
250000
200000
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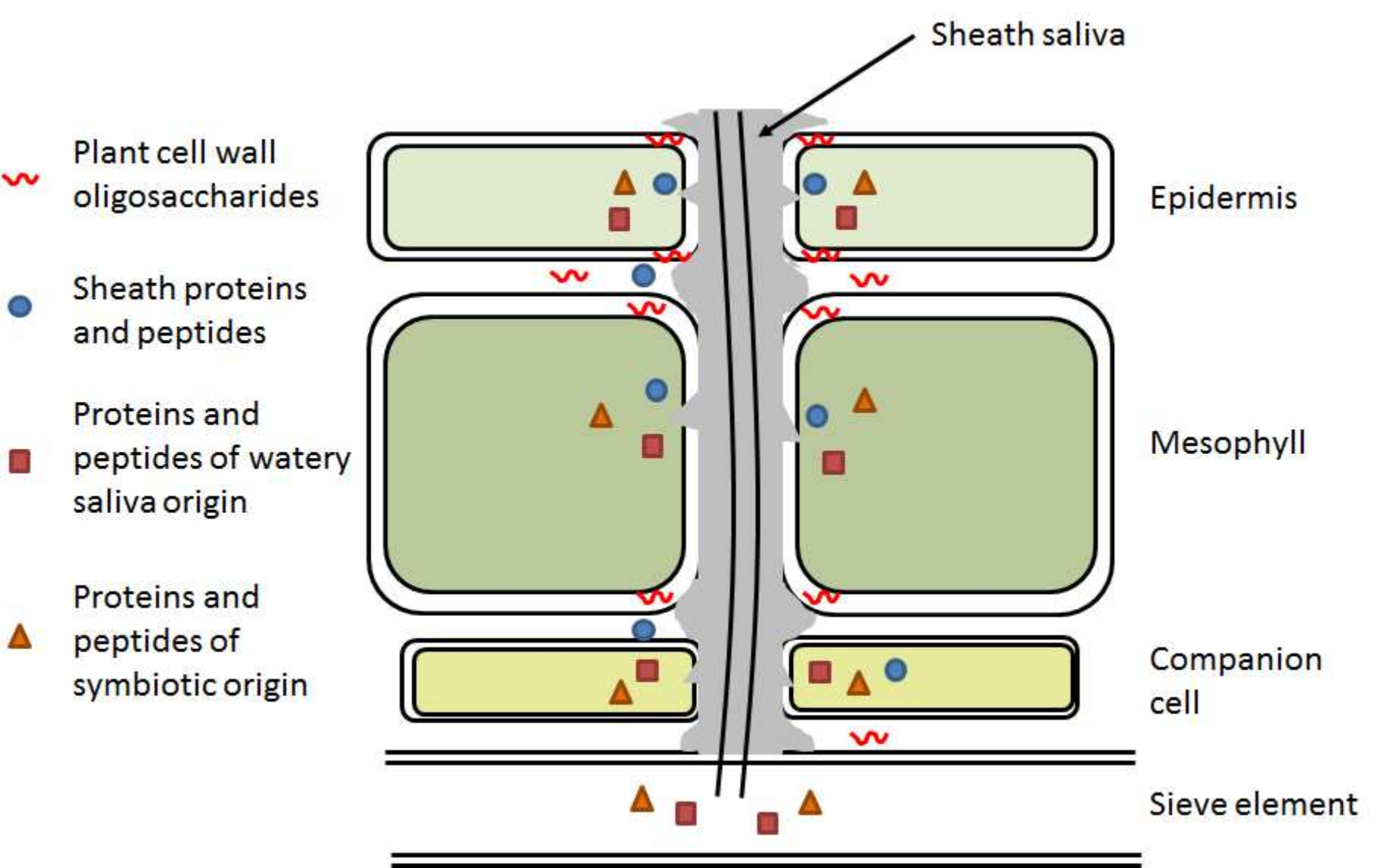
Me-JA


Sheath saliva
Plant cell wall oligosaccharides
Sheath proteins and peptides
Proteins and peptides of watery saliva origin
Proteins and peptides of symbiotic origin
Epidermis
Mesophyll
Companion cell
Sieve element