Cross-tolerance to biotic and abiotic stresses in plants: a focus on resistance to aphid infestation — Source link ↗

Christine H. Foyer, Brwa Rasool, Jack W. Davey, Robert D. Hancock

Institutions: University of Leeds, James Hutton Institute

Related papers:

- The interaction of plant biotic and abiotic stresses: from genes to the field

- Analysis of relative gene expression data using real-time quantitative pcr and the 2(-delta delta c(t)) method

- Abiotic and biotic stress combinations

- The interactive effects of simultaneous biotic and abiotic stresses on plants: Mechanistic understanding from drought and pathogen combination

- Plant Responses to Simultaneous Biotic and Abiotic Stress: Molecular Mechanisms

Share this paper:

This is a repository copy of *Cross-tolerance to biotic and abiotic stresses in plants: a focus on resistance to aphid infestation*.

White Rose Research Online URL for this paper:
http://eprints.whiterose.ac.uk/95165/

Version: Accepted Version

Article:

1 **Cross Tolerance to Biotic and Abiotic Stresses in Plants: A Focus on Resistance**

2 **to Aphid Infestation**

3

4 Christine H. Foyer[1*], Brwa Rasool[1], Jack Davey[2] and Robert D. Hancock[2*]

5

6 [1] Centre for Plant Sciences, School of Biology, Faculty of Biological Sciences,

7 University of Leeds, LS2 9JT. United Kingdom.

8 [2] Cell and Molecular Sciences, The James Hutton Institute, Invergowrie, Dundee,

9 DD2 5DA. United Kingdom.

10

11 Email addresses: c.foyer@leeds.ac.uk, ml10bmar@leeds.ac.uk,

12 rob.hancock@hutton.ac.uk

13

14 *Corresponding Authors: CH Foyer Tel: +44 (0)113 343 1421, Fax: +44 (0)113 343

15 2882; RD Hancock Tel: +44 (0)1382 568 779, Fax: +44 (0)1382 568 704

16

17 **Running title**: Aphid-abiotic stress interactions

18 **Number of tables**: 0

19 **Number of figures**: 3

20 **Word count**: 7083

21

22 **Highlight Summary**: Within natural environments plants respond to multiple biotic

23 and abiotic stresses simultaneously, using an integrated signalling and response

24 network that involves multiple points of reciprocal control. Here we explore how

25 these multiple stress response pathways are managed and co-ordinated at a molecular

26   level to create short/medium term defences and long term memories of

27   environmental hazards with a specific focus on how other biotic and abiotic stresses

28   impact on plant-aphid interactions.

29   **Abstract**

30

31   Plants co-evolved with an enormous variety of microbial pathogens and insect

32   herbivores under daily and seasonal variations in abiotic environmental conditions.

33   Hence, plant cells display a high capacity to respond to diverse stresses through a

34   flexible and finely balanced response network that involves components such as

35   reduction-oxidation (redox) signalling pathways, stress hormones and growth

36   regulators, as well as calcium and protein kinase cascades. Biotic and abiotic stress

37   responses use common signals, pathways and triggers leading to cross tolerance

38   phenomena, whereby exposure to one type of stress can activate plant responses that

39   facilitate tolerance to several different types of stress. While the acclimation

40   mechanisms and adaptive responses that facilitate responses to single biotic and

41   abiotic stresses have been extensively characterised, relatively little information is

42   available on the dynamic aspects of combined biotic/abiotic stress response. In this

43   review, we consider how the abiotic environment influences plant responses to attack

44   by phloem-feeding aphids. Unravelling the signalling cascades that underpin cross

45   tolerance to biotic and abiotic stresses will allow the identification of new targets for

46   increasing environmental resilience in crops.

47

48

49   **Key words**: aphids, secondary metabolites, nitrogen deficiency, drought, high light

50   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

76   of engineering or breeding for multiple stress resistance in crop plants. However, to

77   achieve these goals a thorough understanding of how plants integrate information

78   from multiple signals and optimise response to simultaneous stresses is required. In

79   the present review we discuss knowledge concerning plant signalling and response to

80   multiple stresses with particular reference to the impact of abiotic stresses on plant

81   resistance to aphids.

82

83   **Factors that underpin multiple stress resistance**

84

85   Plants are able to withstand multiple mild and even severe environmental

86   stresses simultaneously (Atkinson and Urwin, 2012). In a competitive growing

87   environment a key factor underlying such resistance is the capacity of sessile plants

88   to recognise, integrate and respond to biotic and abiotic environmental variables by

89   constantly adjusting physiology and metabolism to optimise growth and reproduction

90   in an ever changing environment. This capacity is facilitated by cross-tolerance

91   phenomena, in which enhanced tolerance to a range of different environmental

92   stresses is triggered by exposure to a single stress (Pastori and Foyer, 2002; Mittler,

93   2006). Cross-tolerance is achieved by the synergistic co-activation of the plant innate

94   immune system involving a network of non-specific stress-responsive pathways that

95   cross biotic-abiotic stress boundaries (Bostock, 2005). The innate immune system of

96   plants additionally facilitates the induction of the systemic acquired immune

97   response (SAR) that primes the immune response of tissues distal to the site of

98   pathogen attack in a process analogous to that of cross-tolerance. Both cross-

99   tolerance and SAR comprise an arsenal of inducible defences activated by stress

100 perception and associated cell signalling pathways (Figure 1). Cross-tolerance and

101   SAR are linked in many cases to an enhanced production of reactive oxygen species

102   (ROS) such as superoxide (O_2^-) and hydrogen peroxide (H_2O_2) that are perceived via

103   thiol-modulated redox- and nitric oxide-mediated (NO) signalling pathways (Foyer

104   and Noctor, 2009). For example, exposure to the atmospheric pollutant ozone

105   generates ROS in the apoplast of plant cells, activating a plethora of biotic and

106   abiotic stress responses through interactions with plant hormones such as ethylene

107   (ET), salicylic acid (SA), abscisic acid (ABA), auxin and JA (Baier et al., 2005;

108   Fujita et al., 2006; Bartoli et al., 2013a). This redox-hormone signalling hub, which

109   receives and integrates information from a wide range of environmental stimuli is

110   linked to downstream changes in gene expression that are presumably optimised to

111   meet the prevailing environmental conditions as well as the developmental stage of

112   the plant. The concept that the transcriptional response of a plant results from

113   integration of all of the prevailing external factors is often overlooked in experiments

114   where researchers focus on responses to a single variable. This is illustrated by a

115   recent meta-analysis of the transcriptional response of Arabidopsis to aphid

116   infestation (Foyer et al., 2015). Highly limited overlap in gene expression changes

117   were observed between experiments even when comparing identical interactions

118   between *M. persicae* and the Col-0 genotype and these were reflective of variability

119   in other environmental variables such as growth temperature, day length, light

120   intensity and relative humidity between laboratories (Foyer et al., 2015).

121   Changes in gene expression result in downstream consequences in plant

122   developmental and defence programmes mediated via changes in protein and

123   metabolite content. Plant metabolite composition is strongly impacted by the

124   prevailing abiotic environment affecting the quality of plants as hosts for insect

125   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 abiotic 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).

Plant responses to aphid infestation

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

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

226 induce plant defence responses (Miles, 1999) however it remains unclear whether

227 aphid sheath components are recognised by plant hosts (Bak *et al*., 2013). In addition

228 to the rapidly-gelling sheath saliva, aphids also secrete "watery saliva" at the

229 puncture points and feeding locations (Tjallingii, 2006). The enzymes present in the

230 watery saliva prevent the induction of the plant wound responses in the penetrated

231 tissues and so impede the repair of feeding-associated damage (Will et al., 2009).

232 However, aphid saliva also contains components that act as elicitors that induce plant

233 defence responses (Miles, 1999; de Vos and Jander, 2009). For example, Mp10, an

234 elicitor present in green peach aphid saliva induces chlorosis and local cell death in

235 *Nicotiana benthamiana* (Bos et al., 2010). Moreover, oligogalacturonides are

236 released from the plant cell walls as a result of the action of enzymes secreted by the

237 stylet sheath. Oligogalacturonides and other products of the cell wall breakdown

238 have the potential to induce defence responses that limit aphid infestation (Heil,

239 2009). Proteins derived from endosymbiotic bacteria that have been found in aphid

240 saliva may also participate in the elicitation of plant defence responses, for example

241 GroEL a chaperonin associated with the obligate aphid endosymbiont *Buchnera*

242 *aphidicola* triggers plant immunity resulting in reduced aphid fecundity on hosts

243 (Chaudhary et al., 2014).

244   Plants perceive the presence of fungal pathogens mainly through the presence

245 of chitin in the fungal cell wall, which acts as an elicitor. Chitin is also a major

246 constituent of the insect exoskeleton and chitin oligosaccharides act as microbe-

247 associated molecular patterns (MAMP), inducing a suite of responses which play

248 important roles in defence against fungal pathogens (Boller and Felix, 2009; Wan et

249 al., 2008).   In Arabidopsis, plant perception of chitin is dependent on LysM

250 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

301 lectins thought to interfere with feeding are expressed in response to Hessian fly

302 (*Mayetiola destructor*) or bird cherry-oat aphid (*Rhopalosiphum padi*) in a biotype

303 specific manner. Both of these genes additionally respond to treatment with SA or its

304 analogue benzothiadiazole while *Wci-1* was also responsive to MeJA and ABA

305 (Subramanyam et al., 2006). Within the context of abiotic-biotic stress crosstalk an

306 interesting additional observation was that expression of *Wci-1* was upregulated by

307 mechanical wounding while *Hfr-1* upregulation was observed following water-

308 deficit. Similarly, the presence of the *Rag1* aphid resistance gene in soybean led to

309 the constitutive expression of many defence-related transcripts, including those

310 associated with ABA signalling. In resistant cultivars containing the *Rag1* gene,

311 aphid feeding triggered the significant expression of only one additional gene,

312 whereas aphid feeding in the susceptible cultivar caused increased abundance of

313 many transcripts (Studham and MacIntosh, 2013).

314 The SA, ABA and JA signalling network is considered to be particularly

315 important in triggering appropriate responses against herbivory (de Vos et al., 2005;

316 Kerchev et al., 2013; Studham and MacIntosh, 2013; Hillwig *et al*., 2016). While

317 each hormone has a defined role to play in activating defences, the dynamic

318 adjustment of the relative contribution of each pathway is required to ensure that

319 elicited defence responses are appropriate to prevailing biotic and abiotic

320 environments. SA is required for the induction of effective defences against

321 biotrophic and hemi-biotrophic pathogens. Plants exposed to pathogens, herbivores

322 and to abiotic stresses accumulate SA and PR proteins such as β-1,3-glucanase

323 (Loake and Grant, 2007). However, SA does not provide an effective defence against

324 necrotrophic pathogens (Coquoz et al., 1995; Yu, et al., 1997), which require

325 activation of JA-dependent responses. The NON-EXPRESSOR OF PR1

326 (NPR1) protein is important in the elaboration of SA-mediated defence responses

327 (Kinkema et al., 2000; Mou et al., 2003). *NPR1* and *NPR1*-related transcripts such as

328 *MhNPR1* in apple were increased in response to a range of different abiotic and

329 biotic stresses including aphid infestation (Zhang et al., 2014). Although

330 overexpression of AtNPR1 decreased dehydration and salt tolerance in rice (Quilis et

331 al., 2008), the constitutive expression of *MhNPR1* in tobacco enhanced tolerance to

332 salinity and drought stresses, together with increasing resistance to *Botrytis cinerea*

333 (Zhang et al., 2014).

334   In addition to its functions in protection against invasion by necrotrophic

335 pathogens, the JA-dependent pathways of defence are associated with wounding and

336 responses to herbivory (Creelman and Mullet, 1995; Devoto and Turner, 2005).

337 Although many studies show that JA and SA act in an antagonistic manner in the

338 regulation of plant defences (Spoel et al., 2003), abiotic stress-associated oxidative

339 signalling can induce both pathways together (Han et al., 2013a). ABA has roles in

340 oxidative signalling and protection against aphids (Kerchev et al., 2013, Studham

341 and MacIntosh, 2013). ABA, which can act antagonistically to SA (Ton et al., 2009;

342 Zabala et al., 2009), is important in drought and key physiological responses such as

343 stomatal closure, via the activation of NADPH oxidases (Kwak et al., 2003; Petrov

344 and Van Breusegem, 2012). Mutants defective in ABA biosynthesis such as *aba2* fail

345 to accumulate JA or associated oxylipins following pathogen challenge (Adie et al.,

346 2007). Furthermore *aba2* mutants support smaller aphid colonies than the wild type

347 controls (Kerchev et al., 2013). The *aba1* mutant also supported reduced aphid

348 colonisation associated with increased accumulation of the indole glucosinolates

349 glucobrassicin and 4-methoxy glucobrassicin (Hillwig *et al*., 2016). Both of these

350 compounds and particularly 4-methoxy glucobrassicin are toxic when provided in

15

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

16

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*

21

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

649 underpin many stress-induced responses that influence aphid performance. For

650 example, a better understanding of the stress-dependent regulation and functions of

651 wall-associated kinases is likely to provide new insights into the biotic/abiotic stress

652 interactions that influence aphid fecundity.

653

References

Adie BAT, Perez-Perez J, Perez-Perez MM, Godoy M, Sanchez-Serrano JJ, Schmelz EA, Solano R. 2007. ABA is an essential signal for plant resistance to pathogens affecting JA biosynthesis and the activation of defenses in Arabidopsis. *Plant Cell* **19**, 1665-1681.

Ahmed NU, Park J-I, Jung H-J, Kang K-K, Hur Y, Lim Y-P, Nou I-S. 2012. Molecular characterization of stress resistance-related chitinase genes of *Brassica rapa*. *Plant Physiology and Biochemistry* **58**, 106-115.

Apel K, Hirt H. 2004. Reactive oxygen species: Metabolism, oxidative stress, and signal transduction. *Annual Review of Plant Biology* **55**, 373-399.

Asai T, Tena G, Plotnikova J, Willmann MR, Chiu WL, Gomez-Gomez L, Boller T, Ausubel FM, Sheen J. 2002. MAP kinase signalling cascade in Arabidopsis innate immunity. *Nature* **415**, 977-983.

Atkinson NJ, Urwin PE. The interaction of plant biotic and abiotic stresses: from genes to field. *Journal of Experimental Botany* **63**, 3523-3544.

Baier M, Kandlbinder A, Golldack D, Dietz KJ. 2005. Oxidative stress and ozone: perception, signalling and response. *Plant, Cell and Environment* **28**, 1012-1020.

Bak A, Martinière A, Blanc S, Drucker M. 2013. Early interactions during the encounter of plants, aphids and arboviruses. *Plant Signaling and Behavior* **8**, e24225.

Bartoli G, Forino LMC, Tagliasacchi AM, Durante M. 2013a. Cell death induced by ozone stress in the leaves of *Populus deltoides* x *maximowiczii. Biologia Plantarum* **57**, 514-524.

Bartoli CG, Casalongue CA, Simontacchi M, Marquez-Garcia B, Foyer CH. 2013b. Interactions between hormone and redox signalling pathways in the control of growth and cross tolerance to stress. *Environmental and Experimental Botany* **94**, 73-88.

Boller T, Felix G. 2009. A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. *Annual Review of Plant Biology* **60**, 379-406.

Bolwell GP, Bindschedler LV, Blee KA, Butt VS, Davies DR, Gardner SL, Gerrish C, Minibayeva F. 2002. The apoplastic oxidative burst in response to biotic stress in plants: a three-component system. *Journal of Experimental Botany* **53**, 1367-1376.

Bos JIB, Prince D, Pitino M, Maffei ME, Win J, Hogenhout SA. 2010. A functional genomics approach identifies candidate effectors from the aphid species *Myzus persicae* (green peach aphid). *PLoS Genetics* **6**, e1001216.

Bostock RM. 2005. Signal crosstalk and induced resistance: Straddling the line between cost and benefit. *Annual Review of Phytopathology* **53**, 545-580.

Brotman Y, Landau U, Pnini S, Lisec J, balazadeh S, Mueller-Roeber B, Zilberstein A, Willmitzer L, Chet I, Viterbo A. 2012. The LysM receptor-like kinase LysM RLK1 is required to activate defense and abiotic-stress responses induced by overexpression of fungal chitinases in Arabidopsis plants. *Molecular Plant* **5**, 1113-1124.

Bruce TJA. 2010. Tackling the threat to food security caused by crop pests in the new millennium. *Food Security* **2**, 133-141.

Cabello JV, Chan RL. 2012. The homologous homeodomain-leucine zipper transcription factors HaHB1 and AtHB13 confer tolerance to drought and salinity stresses via the induction of proteins that stabilize membranes. *Plant Biotechnology Journal* **10**, 815-825.

Cabello JV, Arce AL, Chan RL. 2012. The homologous HD-Zip I transcription factors HaHB1 and AtHB13 confer cold tolerance via the induction of pathogenesis-related and glucanase proteins. *Plant Journal* **69**, 141-153.

Cabrera HM, Argandoña VH, Zúñiga GE, Corcuera LJ. 1995. Effect of infestation by aphids on the water status of barley and insect development. *Phytochemistry* **40**, 1083-1088.

Chamnongpol S, Willekens H, Moeder W, Langebartels C, Sandermann H, Van Montagu A, Inze D, Van Camp W. 1998. Defense activation and enhanced pathogen tolerance induced by H_2O_2 in transgenic tobacco. *Proceedings of the National Academy of Sciences USA* **95**, 5818-5823.

Chang CCC, Slesak I, Jorda L, Sotnikov A, Melzer M, Miszalski Z, Mullineaux PM, Parker JE, Karpinska B, Karpinski S. 2009. Arabidopsis chloroplastic glutathione peroxidases play a role in cross talk between photooxidative stress and immune responses. *Plant Physiology* **150**, 670-683.

Chaouch S, Queval G, Vanderauwera S, Mhamdi A, Vandorpe M, Langlois-Meurinne M, Van Breusegem F, Saindrenan P, Noctor G. 2010. Peroxisomal hydrogen peroxide is coupled to biotic defense responses by ISOCHORISMATE SYNTHASE1 in a daylength-related manner. *Plant Physiology* **153**, 1692-1705.

Chaudhary R, Atamian HS, Shen Z, Briggs SP, Kaloshian I. 2014. GroEL from the endosymbiont *Buchnera aphidicola* betrays the aphid by triggering plant defense. *Proceedings of the National Academy of Sciences USA* **111**, 8919-8924.

Chen ZX, Silva H, Klessig DF. 1993. Active oxygen species in the induction of plant systemic acquired-resistance by salicylic-acid. *Science* **262**, 1883-1886.

Comadira G, Rasool B, Karpinska B, Morris J, Verrall SR, Hedley PE, Foyer CH, Hancock RD. 2015. Nitrogen deficiency in barley (*Hordeum vulgare*) seedlings

induces molecular and metabolic adjustments that trigger aphid resistance. *Journal of Experimental Botany* **66**, 3639-3655.

Coquoz JL, Buchala AJ, Meuwly P, Metraux JP. 1995. Arachidonic-acid induces local but not systemic synthesis of salicylic-acid and confers systemic resistance in potato plants to *Phytophthora infestans* and *Alternaria solani. Phytopathology* **85**, 1219-1224.

Creelman RA, Mullet JE. 1995. Jasmonic acid distribution and action in plants – regulation during development and response to biotic and abiotic stress. *Proceedings of the National Academy of Sciences USA* **92**, 4114-4119.

Dedryver C-A, Le Ralec A, Fabre F. 2010. The conflicting relationships between aphids and men: a review of aphid damage and control strategies. *Comptes Rendus Biologies* **333**, 539-553.

Devoto A, Turner JG. 2005. Jasmonate-regulated Arabidopsis stress signalling network. *Physiologia Plantarum* **123**, 161-172.

Dogimont C, Chovelon V, Pauquet J, Boualem A, Bendahmane A. 2014. The *Vat* locus encodes for a CC-NBS-LRR protein that confers resistance to *Aphjis gossypii* infestation and *A. gossypii*-mediated virus resistance. *Plant Journal* **80**, 993-1004.

Douglas AE, Price DRG, Minto LB, Jones E, Pescod KV, Francois CLMJ, Pritchard J, Boonham N. 2006. Sweet problems: insect traits defining the limits to

dietary sugar utilisation by the pea aphid, *Acyrthosiphon pisum. Journal of Experimental Biology* **209**, 1395-1403.

Edreva A, Velikova V, Tsonev T, Dagnon S, Gurel A, Aktas L, Gesheva E. 2008. Stress-protective role of secondary metabolites: diversity of functions and mechanisms. *General and Applied Plant Physiology* **34**, 67-78.

van Emden HF, Eastop VF, Hughes RD, Way MJ. 1969. The ecology of *Myzus persicae*. *Annual Review of Entomology* **14**, 197–270.

Foyer CH, Verrall SR, Hancock RD. 2015. Systematic analysis of phloem-feeding insect-induced transcriptional reprogramming in *Arabidopsis* highlights common features and reveals distinct responses to specialist and generalist insects. *Journal of Experimental Botany* **66**, 495-512.

Foyer CH, Kerchev PI, Hancock RD. 2012. The ABA-INSENSITIVE-4 (ABI4) transcription factor links redox, hormone and sugar signalling pathways. *Plant Signaling and Behaviour* **7**, 276-281.

Foyer CH, Bloom A, Queval G, Noctor G. 2009 Photorespiratory metabolism: genes, mutants, energetics, and redox signaling. *Annual Reviews of Plant Biology*. **60,** 455-484.

Foyer CH, Noctor G. 2009. Redox regulation in photosynthetic organisms: Signaling, acclimation, and practical implications. *Antioxidants and Redox Signaling* **11**, 861-905.

Foyer CH, Noctor G. 2005. Redox homeostasis and antioxidant signaling: A metabolic interface between stress perception and physiological responses. *Plant Cell* **17**, 1866-1875.

Foyer CH, Lelandais M, Kunert KJ. 1994. Photooxidative stress in plants. *Physiologia Plantarum* **92**, 696-717.

Frenkel M, Kulheim C, Jankanpaaa HJ, Skogstrom O, Dall'Osto L, Agren J, Bassi R, Moritz T, Moen J, Jansson S. 2009. Improper excess light energy dissipation in Arabidopsis results in a metabolic reprogramming. *BMC Plant Biology* **9**, 12.

Fujita M, Fujita Y, Noutoshi Y, Takahashi F, Narusaka Y, Yamaguchi-Shinozaki K, Shinozaki K. 2006. Crosstalk between abiotic and biotic stress responses: a current view from the points of convergence in stress signaling networks. *Current Opinion in Plant Biology* **9**, 436-442.

Furch ACU, van Bel AJE, Will T. 2015. Aphid salivary proteases are capable of degrading sieve-tube proteins. *Journal of Experimental Botany* **66**, 533-539.

Gao DL, Appiano M, Huibers RP, Chen X, Loonen AEHM, Visser RGF, Wolters AMA, Bai YL. 2014. Activation tagging of ATHB13 in Arabidopsis thaliana confers broad-spectrum disease resistance. *Plant Molecular Biology* **86**, 641-65.

Gao, Y, Han Z, Li1 Q, Wu Y, Shi X, Ai Z, Du J, Li W, Guo Z . Zhang Y. 2015. Vitamin C induces a pluripotent state in mouse embryonic stem cells by modulating microRNA expression. *FEBS Journal.* **282**, 685–699

Gash AFJ. 2012. Wheat nitrogen fertilisation effects on the performance of the cereal aphid *Metopolophium dirhodum*. *Agronomy* **2**, 1–13.

Glazebrook J, Ausubel FM. 1994. Isolation of phytoalexin-deficient mutants of *Arabidopsis thaliana* and characterization of their interactions with bacterial pathogen. *Proceedings of the National Academy of Sciences USA* **91**, 8955-8959.

Gouinguene SP, Turlings TCJ. 2002. The effects of abiotic factors on induced volatile emissions in corn plants. *Plant Physiology* **129**, 1296-1307.

Han Y, Mhamdi A, Chaouch S, Noctor G. 2013a. Regulation of basal and oxidative stress-triggered jasmonic acid-related gene expression by glutathione. *Plant, Cell and Environment* **36**, 1135-1146,

Han Y, Chaouch S, Mhamdi A, Queval G, Zechmann B, Noctor G.. 2013b. Functional analysis of Arabidopsis mutants points to novel roles for glutathione in coupling H_2O_2 to activation of salicylic acid accumulation and signaling. *Antioxidants and Redox Signaling* **18**, 2106-2121.

Harris MO, Friesen TL, Xu SS, Chen MS, Giron D, Stuart JJ. 2015. Pivoting from *Arabidopsis* to wheat to understand how agricultural plants integrate responses to biotic stress. *Journal of Experimental Botany* **66**, 513-531.

Hatfield JL, Walthall CL. 2015. Meeting global food needs: realizing the potential by genetics x environment x management interactions. *Agronomy Journal* **107**, 1215-1226.

He XH, Anderson JC, del Pozo O, Gu YQ, Tang XY, Martin GB. 2004. Silencing of subfamily I of protein phosphatase 2A catalytic subunits results in activation of plant defense responses and localized cell death. *Plant Journal* **38**, 563-577.

Heil M. 2009.Damaged-self recognition in plant herbivore defence. *Trends in Plant Science* **14**, 356-363.

Held M, Baldwin IT. 2005. Soil degradation slows growth and inhibits jasmonate-induced resistance in *Artemisia vulgaris. Ecological Applications* **15**, 1689-1700.

Hewer A, Becker A, van Bel AJE. 2011. An aphid's Odyssey – the cortical quest for the vascular bundle. *Journal of Experimental Biology* **214**, 3868-3879.

Hillwig MS, Chiozza M, Casteel CL, Lau ST, Hohenstein J, Hernández E, Jander G, MacIntosh GC. 2016. Abscisic acid deficiency increases defence responses against *Myzus persicae* in Arabidopsis. *Molecular Plant Pathology* **17**, 225-235.

Himanen SJ, Nissinen A, Dong WX, Nerg AM, Stewart CN, Poppy GM, Holopainen JK. 2008. Interactions of elevated carbon dioxide and temperature with aphid feeding on transgenic oilseed rape: Are *Bacillus thuringiensis* (Bt) plants more susceptible to nontarget herbivores in future climate? *Global Change Biology* **14**, 1437-1454.

Hodges S, Ward JL, Beale MH, Bennett M, Mansfield JW, Powell G. 2013. Aphid-induced accumulation of trehalose in *Arabidopsis thaliana* is systemic and dependent upon aphid density. *Planta* **237**, 1057-1064.

Huberty AF, Denno RF. 2004. Plant water stress and its consequences for herbivorous insects: a new synthesis. *Ecology* **85**, 1383-1398.

Jankanpaa HJ, Mishra Y, Schroder WP, Jansson S. 2012. Metabolic profiling reveals metabolic shifts in Arabidopsis plants grown under different light conditions. *Plant, Cell and Environment* **35**, 1824-1836.

Jaouannet M, Morris JA, Hedley PE, Bos JIB. 2015. Characterization of Arabidopsis transcriptional responses to different aphid species reveals genes that

contribute to host susceptibility and non-host resistance. *PLoS Pathogens* **11**, e1004918.

Karpinski S, Szechynska-Hebda M, Wituszynska W, Burdiak P. 2013. Light acclimation, retrograde signalling, cell death and immune defences in plants. *Plant, Cell and Environment* **36**, 736-744.

Kerchev PI, Karpińska B, Morris JA, Hussain A, Verrall SR, Hedley PE, Fenton B, Foyer CH, Hancock RD. 2013. Vitamin C and the abscisic acid-insensitive 4 transcription factor are important determinants of aphid resistance in Arabidopsis. *Antioxidants and Redox Signaling* **18**, 2091-2105.

Kerchev PI, Fenton B, Foyer CH, Hancock RD. 2012a. Plant responses to insect herbivory: interactions between photosynthesis, reactive oxygen species and hormonal signalling pathways. *Plant, Cell and Environment* **35**, 441-453.

Kerchev PI, Fenton B, Foyer CH, Hancock RD. 2012b. Infestation of potato (*Solanum tuberosum* L.) by the peach-potato aphid (*Myzus persicae* Sulzer) alters cellular redox status and is influenced by ascorbate. *Plant, Cell and Environment* **35**, 430-440.

Kettles GJ, Drurey C, Schoonbeek HJ, Maule AJ, Hogenhout SA. 2013. Resistance of *Arabidopsis thaliana* to the green peach aphid, *Myzus persicae*, involves camalexin and is regulated by microRNAs. *Plant Journal* **198**, 1178-1190.

Khan MAM, Ulrichs C, Mewis I. 2011. Water stress alters aphid-induced glucosinolate response in *Brassica oleracea* var. italica differently. *Chemoecology* **21**, 235-242.

Kim JH, Jander G. 2007. *Myzus persicae* (green peach aphid) feeding on Arabidopsis induces the formation of a deterrent indole glucosinolate. *Plant Journal* **49**, 1008-1019.

Kim JH, Lee BW, Schroeder FC, Jander G. 2008. Identification of indole glucosinolate breakdown products with antifeedant effects on *Myzus persicae* (green peach aphid). *Plant Journal* **54**, 1015-1026.

Kinkema M, Fan WH, Dong XN. 2000. Nuclear localization of NPR1 is required for activation of PR gene expression. *Plant Cell* **12**, 2339-2350.

Klinger JP, Nair RM, Edwards OR, Singh KB. 2009. A single gene, *AIN*, in *Medicago trunculata* mediates a hypersensitive response to both bluegreen aphid and pea aphid, but confers resistance only to bluegreen aphid. *Journal of Experimental Botany* **60**, 4115-4127.

Kopsell DA, Sams CE. 2013. Increases in shoot tissue pigments, glucosinolates, and mineral elements in sprouting broccoli after exposure to short-duration blue light from light emitting diodes. *Journal of the American Society for Horticultural Science* **138**, 31-37.

Koricheva J, Larsson S, Haukioja E. 1998. Insect performance on experimentally stressed woody plants: A meta-analysis. *Annual Review of Entomology* **43**, 195-216.

Kuhlmann F, Müller C. 2010. UV-B impact on aphid performance mediated by plant quality and plant changes induced by aphids. *Plant Biology* **12**, 676-684.

Kuhlmann F, Müller C. 2009. Development-dependent effects of UV radiation exposure on broccoli plants and interactions with herbivorous insects. *Environmental and Experimental Botany* **66**, 61-68.

Kutyniok M, Persicke M, Müller C. 2014. Effects of root herbivory by nematodes on the performance and preference of a leaf-infesting generalist aphid depend on nitrate fertilization. *Journal of Chemical Ecology* **40**, 118-127.

Kutyniok M, Müller C. 2013. Plant-mediated interactions between shoot-feeding aphids and root-feeding nematodes depend on nitrate fertilization. *Oecologia* **173**, 1367-1377.

Kwak JM, Mori IC, Pei ZM, Leonhardt N, Torres MA, Dangl JL, Bloom RE, Bodde S, Jones JDG, Schroeder JI. 2003. NADPH oxidase AtrbohD and AtrbohF genes function in ROS-dependent ABA signaling in Arabidopsis. *EMBO Journal* **22**, 2623-2633.

Larsson KAE, Saheed SA, Gradin T, Delp G, Karpinska B, Botha CEJ, Jihnsson LMV. 2011. Differential regulation of 3-aminomethylindole/N-methyl-3-

aminomethylindole N-methyltransferase and gramine in barley by both biotic and abiotic stress conditions. *Plant Physiology and Biochemistry* **49**, 96-102.

Lattanzio V, Arpaia S, Cardinali A, Di Venere D, Linsalata V. 2000. Role of endogenous flavonoids in resistance mechanism of Vigna to aphids. *Journal of Agricultural and Food Chemistry* **48**, 5316-5320.

Leather SR, Walters KFA, Dixon AFG. 1989. Factors determining the pest status of the bird cherry-oat aphid, *Rhopalosiphum padi* (L) (Hemiptera, aphididae), in Europe – a study and review. *Bulletin of Entomological Research* **79**, 345-360.

Lee KP, Kim C, Landgraf F, Apel K. 2007. EXECUTER1- and EXECUTER2-dependent transfer of stress-related signals from the plastid to the nucleus of *Arabidopsis thaliana. Proceedings of the National Academy of Sciences USA* **104**, 10270-10275.

Li SC, Mhamdi A, Trotta A, Kangasjarvi S, Noctor G. 2014. The protein phosphatase subunit PP2A-B'γ is required to suppress day length-dependent pathogenesis responses triggered by intracellular oxidative stress. *New Phytologist* **202**, 145-160.

Loake G, Grant M. 2007. Salicylic acid in plant defence - the players and protagonists. *Current Opinion in Plant Biology* **10**, 466-42.

Luna E, Bruce TJA, Roberts MR, Flors V, Ton J. 2012. Next-generation systemic acquired resistance. *Plant Physiology* **158**, 844-853.

Luna E, Ton J. 2012. The epigenetic machinery controlling transgenerational systemic acquired resistance. *Plant Signaling and Behavior* **7**, 615-618.

Pastori GM, Foyer CH. 2002. Common components, networks, and pathways of cross tolerance to stress. The central role of "redox" and abscisic acid-mediated controls. *Plant Physiology* **129**, 460-468.

Madlung A, Comai L. 2004. The effect of stress on genome regulation and structure. *Annals of Botany* **94**, 481-495.

Martin GB, Bogdanove AJ, Sessa G. 2003. Understanding the functions of plant disease resistance proteins. *Annual Review of Plant Biology* **54**, 23-61.

Mateo A, Muhlenbock P, Rusterucci C, Chang CCC, Miszalski Z, Karpinska B, Parker JE, Mullineaux PM, Karpinski S. 2004. LESION SIMULATING DISEASE 1 is required for acclimation to conditions that promote excess excitation energy. *Plant Physiology* **136**, 2818-2830.

Matsuo M, Johnson JM, Hieno A, *et al.* 2014. High redox responsive transcription factor 1 levels result in accumulation of reactive oxygen species in *Arabidopsis thaliana* shoots and roots. *Molecular Plant* **8,** 1253-1273.

McHale L, Tan XP, Koehl P, Michelmore RW. 2006. Plant NBS-LRR proteins: adaptable guards. *Genome Biology* **7**, 212.

Menendez AI, Romero AM, Folcia AM, Martinez-Ghersa MA. 2009. Getting the interactions right: Will higher O_3 levels interfere with induced defenses to aphid feeding? *Basic and Applied Ecology* **10**, 255-264.

Mewis I, Khan MAM, Glawischnig E, Schreiner M, Ulrichs C. 2012. Water stress and aphid feeding differentially influence metabolite composition in *Arabidopsis thaliana* (L.). *PLoS One* **7**, e48661.

Mhamdi A, Han Y, Noctor G. 2013. Glutathione-dependent phytohormone responses: teasing apart signaling and antioxidant functions. *Plant Signaling and Behavior* **8**, e24181.

Miles PW. 1999. Aphid saliva. *Biological Reviews of the Cambridge Philosophical Society* **74**, 41-85.

Miles PW, Oertli JJ. 1993. The significance of antioxidants in the aphid-plant interaction – the redox hypothesis. *Entomologia Experimentalis et Applicata* **67**, 275-286.

Miller G, Schlauch K, Tam R, Cortes D, Torres MA, Shulaev V, Dangl JL, Mittler R. 2009. The plant NADPH oxidase RBOHD mediates rapid systemic signaling in response to diverse stimuli. *Science Signaling* **2,** ra45.

Mittler R. 2006. Abiotic stress, the field environment and stress combination. *Trends in Plant Science* **11**, 15-19.

Mondor EB, Tremblay MN, Awmack CS, Lindroth RL. 2005. Altered genotypic and phenotypic frequencies of aphid populations under enriched CO_2 and O_3 atmospheres. *Global Change Biology* **11**, 1990-1996.

Mou Z, Fan WH, Dong XN. 2003. Inducers of plant systemic acquired resistance regulate NPR1 function through redox changes. *Cell* **113**, 935-944.

Muhlenbock P, Szechynska-Hebda M, Plaszczyca M, Baudo M, Mullineaux PM, Parker JE, Karpinska B, Karpinski S. 2008. Chloroplast signaling and LESION SIMULATING DISEASE1 regulate crosstalk between light acclimation and immunity in Arabidopsis. *Plant Cell* **20**, 2339-2356.

Nakabayashi R, Saito K. 2015. Integrated metabolomics for abiotic stress responses in plants. *Current Opinion in Plant Biology* **24**, 10-16.

Nakagami H, Pitzschke A, Hirt H. 2005. Emerging MAP kinase pathways in plant stress signalling. *Trends in Plant Science* **10**, 339-346.

Niyogi KK. 1999. Photoprotection revisited: Genetic and molecular approaches. *Annual Review of Plant Physiology and Plant Molecular Biology* **50**, 333-359.

Noctor G, Foyer CH. 1998. Ascorbate and glutathione: keeping active oxygen under control. *Annual Review of Plant Physiology and Plant Molecular Biology* **49**, 249-279.

Nomura H, Komori T, Uemura S, *et al.* 2012. Chloroplast-mediated activation of plant immune signalling in Arabidopsis. *Nature Communications* **3**, 926.

Nuccio ML, Wu J, Mowers R, Zhou H-P, Meghji M, Primavesi LF, Paul MJ, Chen X, Gao Y, Haque E, Basu SS, Lagrimini LM. 2015. Expression of trehalose-6-phosphate phosphatase in maize ears improves yield in well-watered and drought conditions. *Nature Biotechnology* **33**, 862-869.

Pastori GM, Foyer CH. 2002. Common components, networks, and pathways of cross-tolerance to stress. The central role of "redox" and abscisic acid-mediated controls. *Plant Physiology* **129**, 460-468.

Pavet V, Olmos E, Kiddle G, Mowla S, Kumar S, Antoniw J, Alvarez ME, Foyer CH. 2005. Ascorbic acid deficiency activates cell death and disease resistance responses in Arabidopsis. *Plant Physiology* **139**, 1291-1303.

Petrov VD, Van Breusegem F. 2012. Hydrogen peroxide-a central hub for information flow in plant cells. *AOB Plants* **2012**, pls014.

Pineda A, Pangesti N, Soler R, van Dam NM, van Loon JJA, Dicke M. 2016. Negative impact of drought stress on a generalist leaf chewer and a phloem feeder is associated with, but not explained by an increase in herbivore-induced glucosinolates. *Environmental and Experimental Botany* **123**, 88-97.

Pitzschke A, Djamei A, Bitton F, Hirt H. 2009. A major role of the MEKK1-MKK1/2-MPK4 pathway in ROS signalling. *Molecular Plant* **2**, 120-137.

Ponder KL, Pritchard J, Harrington R, Bale JS. 2000. Difficulties in location and acceptance of phloem sap combined with reduced concentration of phloem amino acids explain lowered performance of the aphid *Rhopalosiphum padi* on nitrogen deficient barley (*Hordeum vulgare*) seedlings. *Entonologia Experimentalis et Applicata* **97**, 203-210.

Quilis J, Penas G, Messeguer J, Brugidou C, Segundo BS. 2008. The Arabidopsis AtNPR1 inversely modulates defense responses against fungal, bacterial, or viral pathogens while conferring hypersensitivity to abiotic stresses in transgenic rice. *Molecular Plant-Microbe Interactions* **21**, 1215-1231.

Rasool B, Karpinska B, Konert G, Durian G, Denessiouk K, Kangasjarvi S, Foyer CH. 2014. Effects of light and the regulatory B-subunit composition of protein phosphatase 2A on the susceptibility of *Arabidopsis thaliana* to aphid (*Myzus persicae*) infestation. *Frontiers in Plant Science* **5**, 405.

Redovniković IR, Peharec P, Krsnik-Rasol M, Delonga K, Brkić K, Vorkapić-Furač J. 2008. Glucosinolate profiles, myrosinase and peroxidase activity in horseradish (*Amoracia lapathifolia* Gilib.) plantlets, tumour and teratoma tissues. *Food Technology and Biotechnology* **46**, 317-321.

Rentel MC, Lecourieux D, Ouaked F, *et al.* 2004. OXI1 kinase is necessary for oxidative burst-mediated signalling in Arabidopsis. *Nature* **427**, 858-861.

Rogers EE, Glazebrook J, Ausubel FN. 1996. Mode of action of the *Arabidopsis thaliana* phytoalexin camalexin and its role in Arabidopsis-pathogen interactions. *Molecular Plant-Microbe Interactions* **9**, 748-757.

Rojo E, Titarenko E, Leon J, Berger S, Vancanneyt G, Sanchez-Serrano JJ. 1998. Reversible protein phosphorylation regulates jasmonic acid-dependent and -independent wound signal transduction pathways in *Arabidopsis thaliana. Plant Journal* **13**, 153-165.

Rossel JB, Wilson PB, Hussain D, Woo NS, Gordon MJ, Mewett OP, Howell KA,, Whelan J, Kazan K, Pogson BJ. 2007. Systemic and intracellular responses to photooxidative stress in Arabidopsis. *Plant Cell* **19**, 4091-4110.

Rossi M, Goggin FL, Milligan SB, Kaloshian I, Ullman DE, Williamson VM. 1998. The nematode resistance gene Mi of tomato confers resistance against the potato aphid. *Proceedings of the National Academy of Sciences USA* **95**, 9750-9754.

Ryalls JMW, Moore BD, Riegler M, Gherlenda AN, Johnson SN. 2015. Amino-acid mediated impacts of elevated carbon dioxide and simulated root herbivory on aphids are neutralized by increased air temperatures. *Journal of Experimental Botany* **66**, 613-623.

Segonzac C, Macho AP, Sanmartin M, Ntoukakis V, Sanchez-Serrano JJ, Zipfel C. 2014. Negative control of BAK1 by protein phosphatase 2A during plant innate immunity. *EMBO Journal* **33**, 2069-2079.

Sierla M, Rahikainen M, Salojarvi J, Kangasjarvi J, Kangasjarvi S. 2013. Apoplastic and chloroplastic redox signaling networks in plant stress responses. *Antioxidants and Redox Signaling* **18**, 2220-2239.

Singh V, Louis J, Ayre BG, Reese JC, Shah J. 2011. TREHALOSE PHOSPHATE SYNTHASE11-dependent trehalose metabolism promotes *Arabidopsis thaliana* defense against the phloem-feeding insect *Myzus persicae. Plant Journal* **67**, 94-104.

Smith CM, Boyko EV. 2007. The molecular bases of plant resistance and defense responses to aphid feeding: current status. *Entomologia Experimentalis et Applicata* **122**, 1-16.

Spoel SH, Koornneef A, Claessens SMC, *et al*. 2003. NPR1 modulates cross-talk between salicylate- and jasmonate-dependent defense pathways through a novel function in the cytosol. *Plant Cell* **15**, 760-770.

Staley JT, Stafford DB, Green ER, Leather SR, Rossiter JT, Poppy GM, Wright DJ. 2011. Plant nutrient supply determines competition between phytophagous insects. *Proceedings of the Royal Society B* **278**, 718-724.

Studham ME, MacIntosh GC. 2013. Multiple phytohormone signals control the transcriptional response to soybean aphid infestation in susceptible and resistant soybean plants. *Molecular Plant-Microbe Interactions* **26**, 116-129.

Subramanyam S, Sardesai N, Puthoff D, Meyer J, Nemacheck J, Gonzalo M, Williams CE. 2006. Expression of two wheat defense-response genes, *Hfr-1* and *Wci-1*, under biotic and abiotic stresses. *Plant Science* **170**, 90-103.

Sugio A, Dubreuil G, Giron D, Simon J-C. 2015. Plant-insect interactions under bacterial influence: ecological implications and underlying mechanisms. *Journal of Experimental Botany* **66**, 467-478.

Suzuki N, Koussevitzky S, Mittler R, Miller G. 2012. ROS and redox signalling in the response of plants to abiotic stress. *Plant, Cell and Environment* **35**, 259-270.

Szechynska-Hebda M, Kruk J, Gorecka M, Karpinska B, Karpinski S. 2010. Evidence for light wavelength-specific photoelectrophysiological signaling and memory of excess light episodes in Arabidopsis. *Plant Cell* **22**, 2201-2218.

Taj G, Agarwal P, Grant M, Kumar A. 2010. MAPK machinery in plants: recognition and response to different stresses through multiple signal transduction pathways. *Plant Signaling and Behavior* **5**, 1372-1380.

Tariq M, Rossiter JT, Wright DJ, Staley JT. 2013. Drought alters interactions between root and foliar herbivores. *Oecologia* **172**, 1095-1104.

Thangstad OP, Gilde B, Chadchawan S, Seem M, Husebye H, Bradley D, Bones AM. 2004. Cell specific, cross-species expression of myrosinases in *Brassica napus*, *Arabidopsis thaliana* and *Nicotiana tabacum. Plant Molecular Biology* **54**, 597-611.

Tjallingii WF. 2006. Salivary secretions by aphids interacting with proteins of phloem wound responses. *Journal of Experimental Botany* **57**, 739-745.

Ton J, Flors V, Mauch-Mani B. 2009. The multifaceted role of ABA in disease resistance. *Trends in Plant Science* **14**, 310-317.

Torres MA, Dangl JL, Jones JDG. 2002. Arabidopsis gp91(phox) homologues AtrbohD and AtrbohF are required for accumulation of reactive oxygen intermediates in the plant defense response. *Proceedings of the National Academy of Sciences USA* **99**, 517-522.

Trotta A, Wrzaczek M, Scharte J, *et al*. 2011. Regulatory subunit B'γ of protein phosphatase 2A prevents unnecessary defense reactions under low light in Arabidopsis. *Plant Physiology* **156**, 1464-1480.

Vandegehuchte ML, de la Pena E, Bonte D. 2010. Relative importance of biotic and abiotic soil components to plant growth and insect herbivore population dynamics. *PLoS ONE* **5**, e12937.

Van Eck L, Davidson RM, Wu SC, Zhao BYY, Botha AM, Leach JE, Lapitan NLV. 2014. The transcriptional network of WRKY53 in cereals links oxidative responses to biotic and abiotic stress inputs. *Functional and Integrative Genomics* **14**, 351-362.

Viger M, Hancock RD, Miglietta F, Taylor G. 2015. More plant growth but less plant defence? First global gene expression data for plants grown in soil amended with biochar. *Global Change Biology – Bioenergy* **7**, 658-672.

Villada ES, González EG, López-Sesé AI , Castiel AF , Gómez-Guillamón ML. 2009. Hypersensitive response to *Aphis gossypii* Glover in melon genotypes carrying the Vat gene. *Journal of Experimental Botany* **60**, 3269-3277.

de Vos M, Jander G. 2009. *Myzus persicae* (green peach aphid) salivary components induce defence responses in *Arabidopsis thaliana. Plant, Cell and Environment* **32**, 1548-1560.

de Vos M, van Oosten VR, van Poecke RM, *et al.* 2005. Signal signature and transcriptome changes of Arabidopsis during pathogen and insect attack. *Molecular Plant-Microbe Interactions* **18**, 923-937.

Wan J, Zhang X-C, Stacey G. 2008. Chitin signaling and plant disease resistance. *Plant Signaling and Behavior* **3**, 831-833.

Wang Q, Eneji AE, Kong X, Wang K, Dong H. 2015. Salt stress effects on secondary metabolites of cotton in relation to gene expression responsible for aphid development. *PLoS ONE* **10**, e0129541.

Whitfield AE, Falk BW, Rotenberg D. 2015. Insect vector-mediated transmission of plant viruses. *Virology* **479**, 278-289.

Will T, Kornemann SR, Furch ACU, Tjallingii WF, van Bel AJE. 2009. Aphid watery saliva counteracts sieve-tube occlusion: a universal phenomenon? *Journal of Experimental Biology* **212**, 3305-3312.

Wulff-Zottele C, Gatzke N, Kopka J, Orellana A, Hoefgen R, Fisahn J, Hesse H. 2010. Photosynthesis and metabolism interact during acclimation of Arabidopsis thaliana to high irradiance and sulphur depletion. *Plant, Cell and Environment* **33**, 1974-1988.

Xia XJ, Zhou YH, Shi K, Zhou J, Foyer CH, Yu JQ. 2015. Interplay between reactive oxygen species and hormones in the control of plant development and stress tolerance. *Journal of Experimental Botany* **66**, 2839-2856.

Xing Y, Jia W, Zhang J. 2008. AtMKK1 mediates ABA-induced CAT1 expression and H_2O_2 production via AtMPK6-coupled signaling in Arabidopsis. *Plant Journal* **54**, 440-451.

Yan J, Lipka AE, Schmelz EA, Buckler ES, Jander G. 2015. Accumulation of 5-hydroxynorvaline in maize (*Zea mays*) leaves is induced by insect feeding and abiotic stress. *Journal of Experimental Botany* **66**, 593-602.

Yu DQ, Liu YD, Fan BF, Klessig DF, Chen ZX. 1997. Is the high basal level of salicylic acid important for disease resistance in potato? *Plant Physiology* **115**, 343-349.

Zabala MT, Bennett M H, Truman WH, Grant MR. 2009. Antagonism between salicylic and abscisic acid reflects early host-pathogen conflict and moulds plant defence responses. *Plant Journal* **59**, 375-386.

Zavala JA, Ravetta DA. 2001. Allocation of photoassimilates to biomass, resin and carbohydrates in Grindelia chiloensis as affected by light intensity. *Field Crops Research* **69**, 143-149.

Zhang JY, Qu SC, Qiao YS, Zhang Z, Guo ZR. 2014. Overexpression of the *Malus hupehensis* MhNPR1 gene increased tolerance to salt and osmotic stress in transgenic tobacco. *Molecular Biology Reports* **41**, 1553-1561.

Zhang JY, Guo ZR, Qu SC, Zhang Z. 2012. Identification and molecular characterization of a class I chitinase gene (*Mhchit1*) from *Malus hupehensis. Plant Molecular Biology Reporter* **30**, 760-767.

Zhang L, Xiao SS, Li WQ, Feng W, Li J, Wu ZD, Gao XW, Liu FQ, Shao M. 2011. Overexpression of a Harpin-encoding gene hrf1 in rice enhances drought tolerance. *Journal of Experimental Botany* **62**, 4229-4238.

Zhao J, Williams CC, Last RL. 1998. Induction of Arabidopsis tryptophan pathway enzymes and camalexin by amino acid starvation, oxidative stress, and an abiotic elicitor. *Plant Cell* **10**, 359-370.

Zhao Y, Zhou J, Xing D. 2014. Phytochrome B-mediated activation of lipoxygenase modulates an excess red light-induced defence response in Arabidopsis. *Journal of Experimental Botany* **65**, 4907-4918.

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^- 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.

Plant cell wall oligosaccharides

Sheath proteins and peptides

Proteins and peptides of watery saliva origin

Proteins and peptides of symbiotic origin

Sheath saliva

Epidermis

Mesophyll

Companion cell

Sieve element