Annals of Botany **110**: 1503–1514, 2012
doi:10.1093/aob/mcs212, available online at www.aob.oxfordjournals.org

ANNALS OF BOTANY
Founded 1887

INVITED REVIEW

Defence on demand: mechanisms behind optimal defence patterns

Stefan Meldau[1,*], Matthias Erb[2] and Ian T. Baldwin[1]

[1]*Department of Molecular Ecology and* [2]*Root–Herbivore Interactions, Max-Planck-Institute for Chemical Ecology, Hans-Knöll-Str. 8, 07743, Jena, Germany*
** For correspondence. E-mail smeldau@ice.mpg.de*

Received: 6 July 2012 Returned for revision: 9 August 2012 Accepted: 22 August 2012 Published electronically: 28 September 2012

• *Background* The optimal defence hypothesis (ODH) predicts that tissues that contribute most to a plant's fitness and have the highest probability of being attacked will be the parts best defended against biotic threats, including herbivores. In general, young sink tissues and reproductive structures show stronger induced defence responses after attack from pathogens and herbivores and contain higher basal levels of specialized defensive metabolites than other plant parts. However, the underlying physiological mechanisms responsible for these developmentally regulated defence patterns remain unknown.
• *Scope* This review summarizes current knowledge about optimal defence patterns in above- and below-ground plant tissues, including information on basal and induced defence metabolite accumulation, defensive structures and their regulation by jasmonic acid (JA). Physiological regulations underlying developmental differences of tissues with contrasting defence patterns are highlighted, with a special focus on the role of classical plant growth hormones, including auxins, cytokinins, gibberellins and brassinosteroids, and their interactions with the JA pathway. By synthesizing recent findings about the dual roles of these growth hormones in plant development and defence responses, this review aims to provide a framework for new discoveries on the molecular basis of patterns predicted by the ODH.
• *Conclusions* Almost four decades after its formulation, we are just beginning to understand the underlying molecular mechanisms responsible for the patterns of defence allocation predicted by the ODH. A requirement for future advances will be to understand how developmental and defence processes are integrated.

Key words: Optimal defence hypothesis, growth, development, defence, herbivores, pathogens, jasmonic acid, auxin, gibberellins, cytokinins, brassinosteroids, plant–herbivore interactions.

INTRODUCTION

Plants have evolved sophisticated defence systems to cope with the multitude of harmful environmental conditions they face. Resistance strategies of plants against biotic threats such as those posed by herbivores and pathogens are very diverse, and include constitutive defences, which are already present in the absence of attack, as well as induced responses, which are activated only when cues that indicate the presence of an attacker are detected. Interestingly, most types of defence responses are not uniformly expressed across different tissues. Instead, the quality and quantity of various defence strategies are continuously adjusted by developmental and environmental cues. Such plasticity requires appropriate regulatory signalling systems.

Several hypotheses that invoke developmental, ecological and evolutionary constraints have been posed to describe the observed qualitative and quantitative differences in plant defences. One of the earliest and most influential is the optimal defence hypothesis (ODH). In 1974, McKey analysed the within-plant distribution of alkaloids, from which he concluded that 'the plant's limited supply of defensive compounds should be concentrated in those regions in which their presence would most increase the fitness of the plant'. Several assumptions underlie this statement: (*a*) plants use limited resources to produce defensive metabolites, hence defence metabolites are costly; (*b*) some parts of plants are more valuable than others and need to be better defended because (*c*) these tissues are comparably more attractive to biotic threats (Fig. 1). The main idea behind the ODH is therefore that tissue value and the probability of attack are the factors that determine the investments in defensive metabolites (Fig. 1). Although ODH has received considerable experimental support, we do not yet understand the mechanisms that mediate these within-plant distributions.

Direct defences (e.g. toxins or anti-digestive compounds; reviewed in Pichersky and Lewinsohn, 2011), indirect defences (e.g. volatiles, extrafloral nectars; reviewed in Dicke and Baldwin, 2010) or defensive structures (trichomes, thorns, resin ducts) were reported to display within-plant distributions consistent with the predictions of the ODH. The high degree of conservation of ODH patterns among diverse defence-related traits indicates that the regulatory mechanisms driving tissue-specific 'defence-on-demand' are likely to be adaptive for the plant. But how do plants mediate tissue-specific defence patterns predicted by the ODH? The simplest explanation is that developmental identity, which determines tissue value, and defence allocations share regulatory elements. In this way, the factors that modulate identity of a tissue would directly regulate the relative defensive investment. Plant signalling pathways mediating resistance against biotic stress, including phytohormonal regulation through

© The Author 2012. Published by Oxford University Press on behalf of the Annals of Botany Company. All rights reserved.
For Permissions, please email: journals.permissions@oup.com

source: https://doi.org/10.48350/70246 | downloaded: 19.4.2024

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

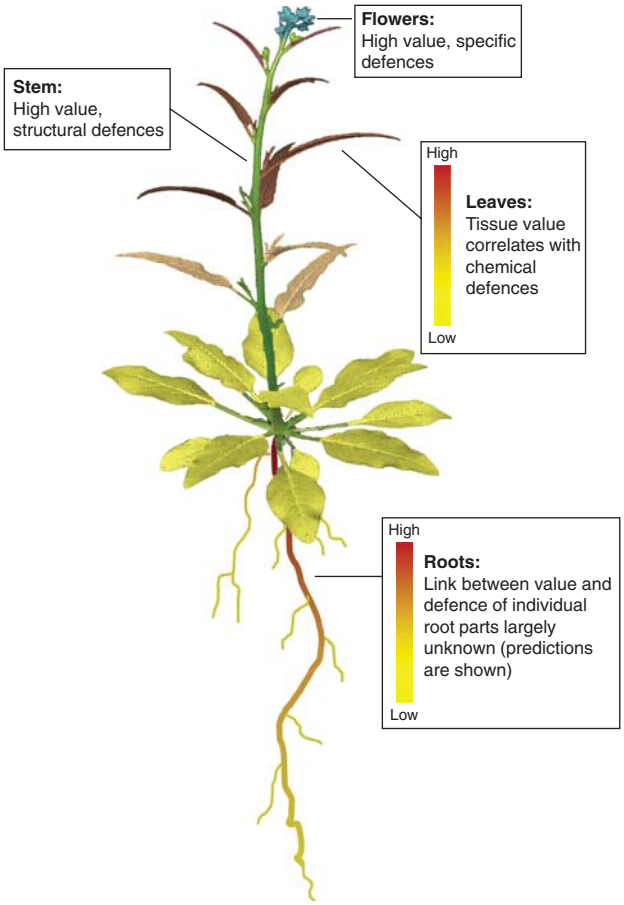

FIG. 1. Assumptions of the optimal defence hypothesis (ODH). The defensive status (represented as defence level, red area) of various plant tissues is not uniform (indicated as colour intensity gradient).Generally, young leaves are more strongly defended because they contribute more to a plant's future fitness than do old tissues. Stems are vital structures and are often protected through lignified cell walls and other structural barriers. Reproductive organs have a high value for the plant, but due to their unique ecological interaction with pollinators and seed dispersers, their defensive patterns change with the changing function of these tissues. Roots also interact specifically with their environment, including beneficial microbes, pathogens and root herbivores, and therefore possess a distinct chemical arsenal of secondary metabolites. Overall, it is predicted that the value of roots and, consequently, their defensive investment, should increase from the bottom to the top (van Dam, 2009). However, few studies have been conducted to test this hypothesis.

jasmonic acid (JA) and salicylic acid, have been studied intensively (reviewed in Howe and Jander, 2008; Walling, 2009). The role of classical growth hormones, such as auxins, cytokinins (CKs), brassinosteroids (BRs) and gibberellins (GAs) in regulating tissue differentiation and identity, organ development and source-sink strengths is also well described (reviewed in Durbak *et al.*, 2012). An emerging theme in plant defence research is the interaction or cross-talk among hormonal signalling pathways controlling plant development with the signalling units that regulate defence responses to pathogens and herbivores (reviewed in Erb *et al.*, 2012; Robert-Seilaniantz *et al.*, 2011). These new findings allow us now to evaluate the possible co-regulation of tissue value and within-plant distributions of defences through integration of developmental and defence-related signalling. In this review, we summarize literature about plant defence phenotypes that follow the patterns predicted by the ODH. We highlight recent advances in our understanding of the interaction among pathways regulating developmental processes with pathways that regulate defence responses. We then synthesize this knowledge and provide a set of new hypotheses about the regulation of developmentally driven defence responses.

OPTIMAL DEFENCE PATTERNS

Secondary metabolite concentrations are highest in young tissues

An overwhelming number of studies have found that constitutive levels of secondary compounds are higher in younger, developing leaves than older tissues. The growing list of secondary metabolites that exhibit this particular behaviour includes iridoid glycosides (Bowers *et al.*, 1992), phenolic compounds (Brunt *et al.*, 2006), glucosinolates (Brown *et al.*, 2003; Traw and Feeny, 2008), alkaloids (van Dam *et al.*, 1994), cyanide (Gleadow and Woodrow, 2000), furanocoumarins (Zangerl and Nitao, 1998) and volatile organic compounds (Radhika *et al.*, 2008). A recent study found a similar distribution in below ground tissues as well (Robert *et al.*, 2012). During our literature search, we found remarkably few studies that deviated from this general pattern (e.g. activities of defensive proteins in *A. thaliana*; Barto and Cipollini, 2005). As younger leaves and roots have also been found to be more important for plant fitness (Barto and Cipollini, 2005; Ohnmeiss and Baldwin, 2000), the higher defensive investment has been interpreted as being consistent with the predictions of the ODH (McCall and Fordyce, 2010). However, a careful assessment of the physiological basis behind the differences is necessary to understand whether this pattern involves active regulation. It has, for example, been argued that secondary metabolites may be diluted as leaves expand and cells elongate, which may be sufficient to reduce the concentration of secondary compounds without any change in biosynthesis. Accounting for such effects during sample preparation and chemical analysis is complicated by the fact that older tissues have more rigid cell walls that are enriched in cellulose and lignin, which may shift the mass balance from soluble to non-soluble metabolites in total tissue extracts. However, some studies report concentration differences that are very unlikely to stem from dilution effects or structural artefacts, as they are simply too large: van Dam and colleagues for instance found that young *Cynoglossum officinale* leaves contained 190 times more pyrrolizidine alkaloids than old leaves (van Dam *et al.*, 1994), and young leaves of *Brugmansia suaveolens* were shown to have 5 times higher concentrations of the toxic scopolamine at the fruit ripening stage (Alves *et al.*, 2007). Furthermore, a meta-analysis concluded that younger leaves were generally better defended independently of their state of expansion (McCall and Fordyce, 2010), and extracellular defensive traits like trichomes (Traw and Feeny, 2008), extrafloral nectar and VOCs (Radhika *et al.*, 2008) are most strongly expressed in young leaves as well, lending additional support to the hypothesis that plants actively invest more energy into defences of younger tissues. Arguably the most convincing evidence for the above idea would be to measure the activity

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

of the biosynthetic pathways in the different tissues rather than the final concentrations of defensive molecules. Unfortunately, this has rarely been attempted.

Young tissues are more responsive to herbivory

Young tissues not only contain the highest levels of constitutive secondary compounds, but are also more inducible than their older counterparts. Volatile organic compounds in *Phaseolus lunatus* are more strongly induced by JA in young than in old leaves (Radhika *et al.*, 2008). The same is true for extrafloral nectar in *Ricinus communis*, *P. lunatus* (Radhika *et al.*, 2008) and *Macaranga tanarius.* In maize, the expression of proteinase inhibitor genes in crown roots was shown to be more strongly inducible by JA than in the older primary roots (Robert *et al.*, 2012). Equally, nicotine is more strongly inducible by simulated herbivory in young tissues of *Nicotiana sylvestris* (Ohnmeiss and Baldwin, 2000). Remarkably, our literature search did not reveal any studies that go against this trend.

It should be noted that, while the ODH predicts more valuable tissues to be more strongly defended, its predictions regarding inducibility are less clear. Huijser and Schmid (2011), for instance, argue that the evolution of induced defences depends on the likelihood of attack: tissues that are frequently attacked should be constitutively defended, while tissues that only occasionally experience herbivory should be more likely to evolve induced defence mechanisms as a cost-saving strategy. As mentioned before, this adds a second dimension to the 'value' of a given tissue for the plant in the context of the ODH, as it becomes inversely proportional to the probability of attack multiplied by the fitness reduction the plant would experience from its loss. However, it can be argued that the evolution of inducibility may be favoured even if the likelihood of attack is high, as long as the realized fitness benefit in years of low herbivore pressure outweighs the fitness loss caused by the delayed onset of defence. Given that herbivore attack patterns are heterogeneous in space and time, it is unlikely for annuals to have a likelihood of attack close to one, and costly defences may therefore be inducible regardless of the tissue value.

Reproductive organs as a special case of defence allocation

Due to its very general assumptions, the ODH cannot only be used to predict ontogenetic patterns of defence within the same tissue type, but can also be used to explain defensive allocation from a whole-plant perspective. Reproductive organs such as flowers and developing seeds are unarguably the most valuable tissues of annual plants, and several studies have attempted to compare defence investment of vegetative and generative parts. Flowers of *B. suaveolens*, for example, were found to have scopolamine concentrations similar to young leaves, while the unripe fruits contained twice as much of the toxic alkaloid (Alves *et al.*, 2007). Similarly, aristolochic acids were found in 4-fold higher concentrations in the flowers of *Aristolochia californica* compared with stems and leaves (Fordyce, 2000). On the other hand, flowers and seeds of the creosotebush *Larrea tridentata* were found to contain significantly fewer phenolic compounds and less nordihydroguaiaretic acid than leaves (Hyder *et al.*, 2002). Although the last example can be interpreted as being consistent with the predictions of the ODH, as *L. tridentata* is a perennial bush that can flower several times while the other plants are annuals, it shows that flowers do not in all cases contain higher levels of secondary metabolites. A recent meta-analysis concluded that, overall, flowers have indeed higher concentrations of defensive chemicals than leaves (McCall and Fordyce, 2010). However, as the authors point out, this result should be interpreted with care, as the considered studies did only report total concentrations in flowers without separating petals and nectar, for example (McCall and Fordyce, 2010). Given the high degree of specialization of the different floral tissues (Barrett, 2010), their distinct genetic and metabolic programme (Wellmer and Riechmann, 2010) and their unique ecological interactions with pollinators and seed dispersers (Kessler and Baldwin, 2011), quantitative phytochemical comparisons may not necessarily yield meaningful results. For example, the interaction of flowers with pollinators involves a fine balance between toxicity and nutritional rewards of the floral nectar to maximize outcrossing success and deter nectar robbers (Kessler *et al.*, 2008, 2012), resulting in defensive patterns that are shaped by forces that are not directly related to the relative value of flowers compared with vegetative tissues.

DEVELOPMENTAL ASPECTS OF DEFENSIVE PATTERNS

The fitness value of different plant tissues is not only spatially heterogeneous, but has a distinct temporal component. A given tissue may be important for a plant at a specific stage in its development, but may become less important during later growth stages. Many grasses, for example, depend strongly on their primary root systems over the first weeks of development. As soon as adventitious roots start to form, these tissues take over most essential tasks of the root systems, and removal of the primary roots at this developmental stage no longer has negative consequences for plant growth (Robert *et al.*, 2012). The same holds true for older leaves of *Arabidopsis thaliana*, for example (Barto and Cipollini, 2005). Following the predictions of the ODH, the defensive investment of plants should reflect these changes in relative value. Indeed, an increasing number of studies document that the defensive patterns of plants follow a distinct developmental programme. Young maize plants, for example, contain high levels of the benzoxazinoid DIMBOA-Glc, which is methylated to HDMBOA-Glc upon insect infestation (Glauser *et al.*, 2011). As plants mature, DIMBOA-Glc levels drop and constitutive levels of HDMBOA-Glc increase (Cambier *et al.*, 2000), turning an induced pattern into a basal defence. Volatile organic compounds in maize and soybean are both more inducible in young seedlings than older plants (Köllner *et al.*, 2004; Rostas and Eggert, 2008). In this case, however, the induced defence response ceases without transitioning into constitutive expression. Here, it can be argued that the age-dependent transitions of inducibility reflects the fact that older plants have more mature leaves than young plants and, given the systemic nature of defence responses, that the observed effect may be equivalent to the differences observed between different

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

leaves of the same plant, as discussed above. However, a recent study clearly shows that the inducibility of *Nicotiana attenuata* is significantly reduced once the plant starts flowering, specifically, when it starts to elongate its corollas, an effect that can be reversed by removing the flowers (Diezel *et al.*, 2011). Therefore, any defensive allocation pattern has to be evaluated in the context of the plant's developmental status.

REGULATION OF DEFENCE METABOLITES: A DOMINANT ROLE FOR JASMONATES

Understanding the nature of signals important for the regulation of defensive metabolites in plants is crucial to elucidate mechanisms behind patterns predicted by the ODH. The biosynthesis of a vast majority of direct and indirect plant defence compounds is regulated by oxylipins, including JA and its isoleucine conjugate JA-Ile (De Geyter *et al.*, 2012). The JA-Ile isomer (+)-7-iso-JA-L-Ile is perceived by the F-box protein COI1 (CORONATINE-INSENSITIVE 1) which is part of a Skp/Cullin/F-box complex $SCF^{(CoI1)}$ that acts as an ubiquitin ligase (Xu *et al.*, 2002; Chini *et al.*, 2007; Paschold *et al.*, 2007). The binding of JA-Ile to $SCF^{(CoI1)}$ triggers the ubiquitination of JAZs (JASMONATE ZIM DOMAIN proteins), which are negative regulators of JA-induced transcriptional responses, and thereafter the degradation of JAZs through a 26S proteasome-mediated proteolytic pathway, and thus finally activates JA-mediated responses (Chini *et al.*, 2007; Thines *et al.*, 2007; Katsir *et al.*, 2008; Oh *et al.*, 2012). The JAZ proteins also interact with the transcriptional co-repressor TOPLESS (TPL), through the Novel Interactor of JAZ (NINJA) (Pauwels *et al.*, 2010). JAZ proteins are known to bind transcription factors, such as MYC2 in *A. thaliana* (Fig. 2A), which control transcription of a range of JA-inducible genes involved in secondary metabolite biosynthesis (reviewed in De Geyter *et al.*, 2012). JA-induced JAZ degradation releases these transcription factors and thereby activates biosynthetic pathways leading to secondary metabolite biosynthesis (Fig. 2A).

Given that the JA-signalling cascade was established early in the evolution of higher plants, it is likely that the regulation of the biosynthesis of a vast majority of secondary metabolites is controlled by JA and the increasing number of reports demonstrating this relationship is consistent with this assumption. The importance of JA and JA-Ile in the regulation of defensive metabolites is commonly demonstrated by either applying these hormones to plants or by using genetic approaches that impair JA/JA-Ile biosynthesis and perception. Comparing defence metabolite accumulation of plants with a genetically impaired JA pathway with those of wild-type plants makes it possible to identify basal and herbivory-induced metabolites. Since transformation techniques have not been established for many plant model systems, we can only report on a few examples of JA and COI1-dependent defence metabolites whose accumulation follows ODH-predicted patterns. In *N. attenuata*, JA- and COI1-regulated secondary metabolites that follow patterns predicted by the ODH include nicotine (Halitschke and Baldwin, 2003; Paschold *et al.*, 2007), hydroxygeranyllinalool diterpene glycosides (Jassbi *et al.*, 2008; Heiling *et al.*, 2010), TPI activity (Van Dam *et al.*, 2001; Zavala *et al.*, 2004) and an array of different phenolamides such as caffeoylputrescine (Kaur *et al.*, 2010; Onkokesung *et al.*, 2012). In flowering *N. attenuata* plants basal and JA-induced levels of hydroxygeranyllinalool diterpene glycosides, TPI activity and caffeoylputrescine show a gradient between young and old leavs with highest concentrations in young leaves (C. Brütting, unpubl. res.). Nicotine levels are more stable throughout the foliage of rosette and flowering-staged plants, their basal and induced levels increase over time (Baldwin and Ohnmeiss, 1993; Baldwin, 1999; C. Brütting, MPI Chemical Ecology, Germany, unpubl. res.) and unripe seed capsules accumulate the highest concentrations of nicotine in above-ground tissues of *N. attenuata* plants (Baldwin and Karb, 1995). Glucosinolates are abundant defensive metabolites in brassicaceous plants whose levels were reported to depend on JA signalling, tissue identity and plant ontogeny in several plant species including *A. thaliana* (Reichelt *et al.*, 2002; Mewis *et al.*, 2006) and *Brassica rapa*. Trichome density is also regulated by JA-signalling and positively correlated with tissue value in the Brassicaeae (Traw and Feeny, 2008; Yoshida *et al.*, 2009). Another model plant for which JA mutants are available is tomato (*Solanum lycopersicum*). Genetic analysis indicates that cultivated tomato possesses a plethora of inducible, JA-regulated defence metabolites that are involved in resistance to herbivores (Howe *et al.*, 1996; Thaler, 1999; Li *et al.*, 2002*a*). Some of these metabolites can be found in high concentrations in different types of glandular trichomes, including mono- and sesquiterpenes, glycoalkaloids, acyl sugars and polyphenolic compounds (Kang *et al.*, 2010). Similar to arabidopsis, the density of glandular trichomes in tomato is also regulated by JA (Boughton *et al.*, 2005; van Schie *et al.*, 2007). JA also regulates defence responses induced by herbivore attack in maize (Yan *et al.*, 2012), including benzoxazinoids and volatile organic compounds, both of which are under developmental control (Cambier *et al.*, 2000; Köllner *et al.*, 2004). Taken together, these data demonstrate that the levels of inducible metabolites, which follow ODH patterns, are also regulated by the JA pathway.

DEVELOPMENTAL REGULATION OF JA BIOSYNTHESIS AND PERCEPTION

If the JA pathway plays a major role in the co-ordination of defence metabolite accumulation, then the developmental control of the oxylipin pathway may enable plants to regulate patterns predicted by ODH. Some results from different plant species indicate such a relationship. For example, Ohnmeiss and Baldwin (2000) have shown a decrease in wound-induced JA in *N. sylvestris* during its ontogeny, which correlates with the levels of wound-induced nicotine accumulation. In *N. attenuata*, herbivory by the specialist lepidopteran herbivore *Manduca sexta* quickly amplifies the wound-induced JA levels 3- to 5-fold (Halitschke *et al.*, 2001). This response is mediated by the plants' perception of fatty acid–amino acid conjugates derived from *M. sexta* oral secretions (Halitschke and Baldwin, 2003). When flowering is initiated, this fatty acid–amino acid conjugate-induced JA burst declines rapidly, but can be recovered within 1 d by simply removing the plant's inflorescence (Diezel *et al.*, 2011), indicating that

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

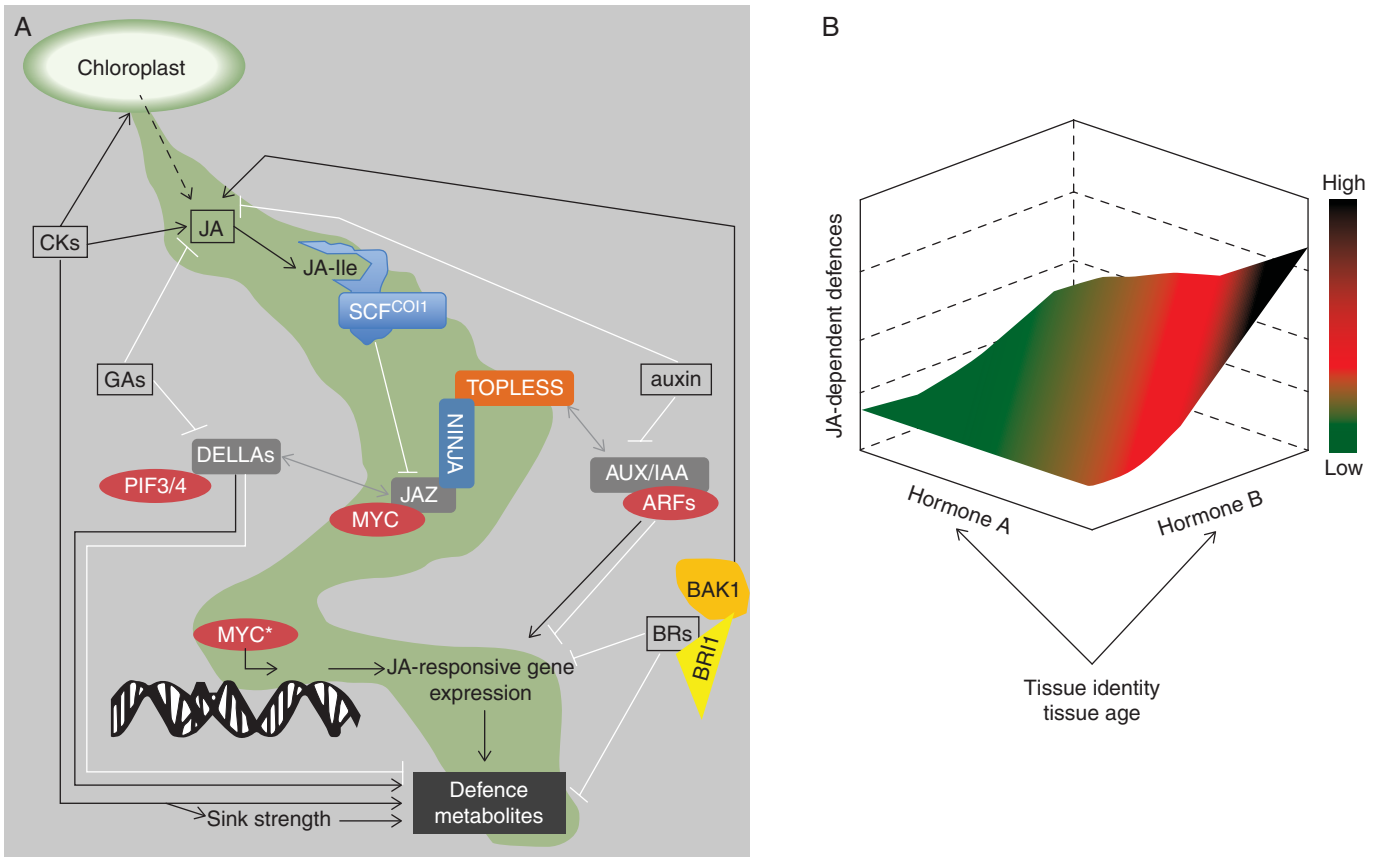

FIG. 2. (A) Hormonal cross-talk regulates JA-mediated defences. A conceptual, non-exhaustive overview is presented. The jasmonic acid (JA) pathway (green background) begins with JA biosynthesis in the chloroplast (black arrow with dotted line). The active JA–isoleucine (JA-Ile) conjugates bind to the SCFCOI1 complex, leading to the degradation of JAZ proteins and the release of MYC2-related transcription factors (MYC* = JAZ-free form), which activate JA-dependent gene expression thereby mediating biosynthesis of defensive metabolites. Developmentally controlled hormones [cytokinins (CKs), gibberellins (GAs), auxin and brassinosteroids (BRs)] can modulate the JA pathway, by controlling JA biosynthesis or JA signalling. CKs can positively influence chloroplast function and JA biosynthesis and JA-induced defence metabolites, GAs can inhibit JA levels and GA-induced degradation of DELLA proteins leading to activation of growth-related processes through PIF3/4. GA signalling can positively and negatively regulate JA-inducd defence responses. Auxin leads to degradation of AUX/IAA proteins which interact with TOPLESS to negatively regulate auxin-induced responses through ARFs. Auxin can inhibit JA biosynthesis and positively or negatively influence JA signalling. Brassinosteroids (BRs) are perceived by BRI1 and its co-receptor BAK, which is important for JA biosynthesis. BRs have been shown to negatively influence JA-induced defence responses. Positive control is indicated by unidirectional black arrows, protein–protein interactions by bidirectional grey arrows and negative regulation by white inhibitory arrows. (B) Developmental control of JA-mediated defences. Plant tissue identity and age differentially regulate the levels and signalling of growth-related hormones (such as cytokinins, auxin, gibberellins or brassinosteroids; demonstrated by Hormones A and B). The dynamic interactions between these hormone sectors orchestrate the jasmonic acid (JA)-dependent defence landscape (plotted here in 3-D with low and high defence status as indicated).

the transition from juvenile to reproductive growth tightly regulates herbivory-elicited JA-biosynthesis and JA-mediated defence responses in *N. attenuata* (van Dam *et al.*, 2001). The precise timing of the reduction in the JA burst with flower initiation, and its rapid recovery with flower removal, indicates that signalling rather than resource allocation processes orchestrate this response (Diezel *et al*, 2011). JA sensitivity, measured as JA-induced indirect defence accumulation, in *Phaseolus lunatus* (lima bean) and *Ricinus communis* (castor oil plant) is highly correlated with leaf position in 4-week-old plants; JA-induced extrafloral nectars and volatile emissions increase up to 10 times in the apical leaves, when compared with the basal stem leaves (Radhika *et al.*, 2008). These examples demonstrate that alterations in the defensive states of tissues can be correlated with their ability to mount JA levels in response to attack or with the tissue-specific regulation of JA sensitivity.

TO SERVE AND PROTECT: GROWTH HORMONES AND DEFENCE REGULATION

Our understanding of the underlying mechanisms mediating ontogenic regulation of JA biosynthesis and signalling is still very fragmentary. However, the recently discovered interactions between growth-related hormones and JA signalling provide hypotheses for ontogeny-driven defence pathway regulation. Several plant growth-related hormones including GAs, CKs, BAs and auxin have been shown to regulate JA biosynthesis or signalling. The complex interactions between these hormones and plant defence pathways were summarized in recent reviews (Robert-Seilaniantz *et al.*, 2011; Erb *et al.*,

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

2012). In the following section, we discuss whether the roles of CKs, auxin, GAs and BAs in plant growth and their interaction with the JA pathway can explain some aspects of the developmental regulation of defence processes. Other hormonal pathways, including ethylene, ABA and SA also play important regulatory roles in defence and development. We will not discuss further the role of these hormones in this review, but refer the interested reader to other excellent reviews about their role in plant growth and defence (Shinshi, 2008; Ton *et al.*, 2009; An and Mou, 2011; Robert-Seilaniantz *et al.*, 2011).

CKs. These play fundamental roles in plant growth, development and stress responses (Argueso *et al.*, 2009). CK levels are regulated by plant ontogeny: young developing tissues accumulate higher levels of CKs, whereas reduced CK levels promote leaf senescence (Ori *et al.*, 1999). Regulation of CK levels is critical for shoot apical meristem establishment and maintenance. Decreasing CK sensitivity conferred by the histidine kinase CK receptor mutant wooden leg (wol) impairs meristem development (Jasinski *et al.*, 2005) and triple mutants of three histidine kinase CK receptors have significantly smaller meristems (Higuchi *et al.*, 2004; Nishimura *et al.*, 2004). In line with these data, increased CK levels, either through external application or via expression of a bacterial CK biosynthesis gene, an isopentenyltransferase from *Agrobacterium tumefaciens*, is sufficient to partially rescue the meristem defects of the strong meristem-deficient mutants in arabidopsis (Yanai *et al.*, 2005). The *LONELY GUY* (*LOG*) gene, which encodes a phosphoribohydrolase that converts inactive forms of CK to active free-base forms, is expressed specifically in the distal regions of the shoot apical meristem and is required for meristem maintenance (Kurakawa *et al.*, 2007). CKs also play important roles in regulating root growth and development, such as limiting the size of the root apical meristem and the rate of root growth (Werner *et al.*, 2003; Dello Ioio *et al.*, 2007; Zhang *et al.*, 2011). Xylem sap CK levels can serve as a reliable reporter of vital root systems. Their levels rise with increasing soil nutrient supply, which also positively affects shoot branching (Mediene *et al.*, 2002). Several studies, using transgenic approaches, or by external applications of CKs, have showed that JA biosynthesis and JA-related defence metabolite accumulations are positively correlated with CK levels (Smigocki *et al.*, 1993; Sano *et al.*, 1996; Dervinis *et al.*, 2010). CKs enhance defence metabolite accumulation in commercial tobacco, although the role of JA in this process was not further investigated (Grosskinsky *et al.*, 2011). Mechanistically, CK signalling might directly enhance JA signalling, as has been shown for SA-mediated defences against pathogens in arabidopsis (Choi *et al.*, 2010; Argueso *et al.*, 2012; Naseem *et al.*, 2012). CKs may also support the biosynthesis of costly defences by regulating resource availability, as sink strength of valuable tissues can be regulated by CKs through increasing nitrogen and carbon resource allocation (Lara *et al.*, 2004; Ruffel *et al.*, 2011). Since CKs play a pivotal role as regulators of cell division (Haberlandt, 1913; Miller *et al.*, 1955), their contribution to optimal defence processes could be mediated through higher cell densities in rapidly growing tissues. One of the hallmarks of CK-regulated physiological processes is the control of chlorophyll contents, which might be a function of chloroplast density (Hewelt *et al.*, 2000; Jordi *et al.*, 2000; Kobayashi *et al.*, 2012). Changes in CK levels effect plastid gene expression, protein abundance and protein phosphorylation states, indicating that chloroplasts (and their precursors) are major targets of the CK pathway (Zubo *et al.*, 2008; Cortleven *et al.*, 2009; Cerny *et al.*, 2011; Kobayashi *et al.*, 2012). Pathological CK overproduction leads to changes in chloroplast ultrastructure (Polanska *et al.*, 2007). The dominant effect of CKs on plastid function and photosynthesis might be important for the regulation of plastid-mediated defence responses such as the biosynthesis of JA, which is initiated in the chloroplast (reviewed in Wasternack and Kombrink, 2010) and depends on chloroplast function (Mitra and Baldwin, 2008). Indeed, CK treatments in poplar lead to higher transcripts levels of JA-biosynthetic genes and increased JA levels and its precursors upon wounding (Dervinis *et al.*, 2010). Although direct experimental evidence for the role of CKs in ontogeny-driven defence metabolite accumulation is lacking, the literature presented here suggests a link (summarized in Fig. 2A). Using transgenic plants with tissue-specific alterations in CK levels, or signalling, will provide important tools to verify the relationships between CK levels and defence metabolite accumulation.

Auxin. This is one of the best-studied growth regulators of plants. Many aspects of plant development are regulated by auxin, including cell elongation and meristem activity. For example, an auxin gradient is essential for the establishment and maintenance of the stem cell niche and for proliferative cell division (Xu *et al.*, 2006; Petersson *et al.*, 2009). This auxin gradient is regulated by the differential expression and subcellular localizsation of auxin influx and efflux carriers (Swarup *et al.*, 2001; Blilou *et al.*, 2005; Blakeslee *et al.*, 2007; Grieneisen *et al.*, 2007). High auxin levels lead to the proteosome-mediated degradation of AUXIN/INDOLE-3-ACETIC ACID (Aux/IAA) proteins, thereby releasing the AUXIN RESPONSE FACTORS (ARFs) from their Aux/IAA-bound form, leading to transcriptional activation of auxin responses (Del Bianco and Kepinski, 2011). One of the ARFs, ARF5/MONOPTEROS (MP) is required for maintaining the heart of meristematic tissues, the quiescent centre (Vidaurre *et al.*, 2007). Another Aux/IAA protein, BODENLOS (BDL) is also degraded by auxin leading to activation of MP (Hamann *et al.*, 1999, 2002). BDL interacts with TOPLESS (TPL), a transcriptional co-repressor of genes involved in several hormone pathways (Long *et al.*, 2002, 2006; Szemenyei *et al.*, 2008) which is also required for BDL-mediated inhibition of MP activity.

How could auxin contribute to the emergence of JA-mediated optimal defence patterns? Auxin and JA signalling is connected through JAZ1 and MYC2, which are co-regulated by both hormones (Tiryaki and Staswick, 2002; Grunewald *et al.*, 2009). The signalling pathways of both hormones also share other regulatory proteins, including TPL (Szemenyei *et al.*, 2008). The EAR-motif protein Novel Interactor of JAZ (NINJA) connects TPL to the JAZ

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

complex, thereby mediating the repression of genes controlled by JAZ-bound transcription factors, such as MYC2. TPL also interacts with EAR-motif-containing AUX/IAA proteins, which are negative regulators of auxin-induced responses (Pauwels *et al.*, 2010). Positive correlations between auxin signalling and JA levels have been reported for arabidopsis flowers (Nagpal *et al.*, 2005); however, auxin treatments diminished wound induced JA and JA-mediated nicotine accumulations in leaves of *N. sylvestris* (Baldwin *et al.*, 1997) and JA-mediated lateral root formation in *A. thaliana*, suggesting that the interplay between auxin and JA is tissue and species specific. Thimann and Skoog had already shown in 1933 that initiation of auxillary shoot branching, induced by removing the inflorescence, could be inhibited by application of auxin to the decapitated apex (Thimann and Skoog, 1933). The shoot apex, and probably young leaves, are major sources of auxin, which is transported basipetally through the plant's polar transport stream and regulates apical dominance by inhibiting auxillary bud activity (Ljung *et al.*, 2001). McSteen and Lyser (2005) have suggested that auxin supply through the shoot apex can be generally considered as a reporter for the integrity of the primary apex and stresses, like herbivory, could strongly influence the provision of auxin. Since auxin affects JA and JA-induced responses in leaves, as described by Baldwin *et al.* (1997), changes in apical auxin supply could also be involved in the JA biosynthesis switch in leaves during juvenile to reproductive phase transition (Diezel *et al.*, 2011). Analysing auxin levels and responses during ontogenic transitions might provide important clues about the role of this hormone in shaping ODH patterns.

GAs. These are major determinants of plant architecture. Biosynthesis of GAs through the diterpenoid pathway from geranylgeranyl diphosphate is regulated in a complex manner (Yamaguchi, 2008). Perception of GAs is mediated by GIBBERELLIN INSENSITIVE DWARF 1 (GID1) (Ueguchi-Tanaka *et al.*, 2005), which enhances the degradation of DELLA proteins through an F-box protein SLEEPY1 (SLY1)-mediated ubiquitin-26S-proteasome pathway (Griffiths *et al.*, 2006; Ueguchi-Tanaka *et al.*, 2007). DELLA proteins are nuclear proteins acting as suppressors of GA-induced responses and thus control many aspects of development and stress resistance (Sun, 2010). One of the best-studied growth responses mediated by DELLA proteins originated from research on light-dependent growth regulation. PHYTOCHROME-INTERACTING FACTORs (PIFs) are transcription factors, which promote hypocotyl elongation of etiolated arabidopsis seedlings. Inhibition of hypocotyl elongation during de-etiolation is mediated through inhibition of GA accumulation and PIF degradation (Achard *et al.*, 2009). Direct binding of DELLA proteins to different PIF transcription factors leads to inhibition of PIF target gene expression and therefore hypocotyl elongation (Feng *et al.*, 2008; de Lucas *et al.*, 2008; Gallego-Bartolome *et al.*, 2010). DELLA stability is affected through regulation of GA homeostasis in various growth processes (reviewed in Sun, 2010). For example, GA signalling promotes root cell elongation and root apical meristem size by destabilizing DELLA proteins (reviewed in Galinha *et al.*, 2009). Intriguingly, DELLAs also bind to JAZ proteins, thereby bridging JA-mediated defence responses and plant growth. GA perception triggers reduced DELLA levels, which lead to inhibition of JA responses and JA biosynthesis (Navarro *et al.*, 2008; Cheng *et al.*, 2009). JA, in turn, delays GA-mediated DELLA protein degradation and JAZ9 inhibits the interaction of RGA (a DELLA protein) with PIF3; therefore JA signalling controls plant growth by interfering with the GA pathway (Yang *et al.*, 2012). In *N. attenuata*, transgenic lines, which hyper-accumulate JA and its related defence metabolites, have also reduced levels of GAs and growth (M. Heinrich *et al.*, unpubl. res.). In line with the data presented by Yang *et al.* (2012), growth rates can be recovered to WT levels by external application of GA3 (M. Heinrich *et al.*, unpubl. res.). Since GAs control both plant growth and defence responses, regulating GA levels and DELLA protein stability could be a mechanism by which plants orchestrate JA-mediated ODH patterns. However, the simple up-regulation of GAs in fitness-relevant tissues would restrict DELLA-mediated growth suppression, but, according to the present model of GA–JA crosstalk, would also reduce JA-mediated defence responses. These data indicate that simple linear relationships between GA and JA signalling are unlikely to explain ODH patterns in plants, although GAs might profoundly contribute to ontogenic defence responses through yet unknown pathways. In a recent report, Hong *et al.* (2012) demonstrated that DELLAs act in concert with the JA signalling pathway to promote floral emission of volatiles. In light of the paper by Navarro *et al.* (2008) these data indicate that different sets of JA-induced pathways can show contrasting regulation by GAs, indicating that GA–JA crosstalk is dependent on tissue identity and developmental stage (Hong *et al.*, 2012). Future experiments designed to analyse the effects of GAs on specific branches of the JA-induced defence metabolome throughout a plant's ontogeny might help to develop a coherent model about how these hormones shape plant growth and defence syndromes as predicted by the ODH.

BRs. These regulate a broad spectrum of plant growth and developmental processes, including plant architecture, vascular development, male fertility, flowering, senescence and photomorphogenesis (Gudesblat and Russinova, 2011). For example, BRs mediate leaf growth by positively regulating cell expansion and cell division (Nakaya *et al.*, 2002) and root growth, primarily through regulation of the root meristem size by cell cycle and stem cell niche control (Gonzalez-Garcia *et al.*, 2011; Hacham *et al.*, 2011). BRs are perceived by brassinosteroid insensitive 1 (BRI1), belonging to the family of a plasma membrane localized leucine rich-repeat (LRR)-receptor-like-kinases (reviewed in Kim and Wang, 2010). BAK1 (BRI1 associated kinase 1), another receptor-like kinase, was also reported to be required in the activation of BRI1 (Li *et al.*, 2002*b*; Nam and Li, 2002). BR-responsive gene expression is directly regulated by plant-specific transcription factors that include BES1, BZR1, and BES1/BZR1 homologues 1–4 in *A. thaliana* (He *et al.*, 2002; Yin *et al.*, 2002). Several studies indicate that BRs are involved in regulating defence pathways through the JA sector. JA responsiveness, as measured by trichome density, and defence metabolite accumulations in tomato (Campos *et al.*, 2009) as well as

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

JA-induced root growth inhibition in arabidopsis (Ren *et al.*, 2009) are counteracted by BRs. On the other hand, the expression of wounding and JA-responsive genes is enriched in microarray studies performed to identify BR signalling targets in arabidopsis and BRs enhance JA-induced anthocyanins in *A. thaliana* (Peng *et al.*, 2011). Silencing the co-receptor BAK1 diminished wounding and herbivory-induced JA biosynthesis and JA-induced TPI activity in *N. attenuata* (Yang *et al.*, 2011), providing a possible link between BR perception and tissue responsiveness to herbivore attack. These examples demonstrate that BR signalling regulates both growth and defence-mediated processes. Analysis of BR levels in tissues with contrasting defence phenotypes, in combination with tissue-specific regulation of BR metabolism and signalling will provide useful tools to gain insight into the role of BRs in regulation of optimal defence patterns. Taken together, these data show that CKs, auxin, GAs and BRs are potent regulators of the JA pathway and might therefore contribute to the developmental regulation of JA-dependent secondary metabolites (Fig. 2A).

PROFESSIONAL NETWORKS

The defence syndromes of different plant tissues are unlikely to be regulated by just a few plant hormones, but rather emerge through complex interactions among various hormonal pathways and other regulatory elements, such as small RNAs, involved in plant growth and defence (Robert-Seilaniantz *et al.*, 2011; Erb *et al.*, 2012). A number of studies have analysed the intricate relationships among different hormonal pathways during plant developmental processes (reviewed in Durbak *et al.*, 2012). Auxin derived from apical tissues, for example, is involved in the regulation of bud-outgrowth (see previous section). However, auxin itself does not enter the auxillary bud, but rather employs another pathway that regulates the activity of bud meristems (Hillman *et al.*, 1977; Morris, 1977; Booker *et al.*, 2003). Such 'second messengers' could be root-derived CKs, whose biosynthesis and transport is inhibited by auxin (Bangerth, 1994; Eklof *et al.*, 1997; Nordstrom *et al.*, 2004; McSteen and Leyser, 2005). In the absence of auxin-induced suppression, CKs could enter the buds and directly activate outgrowth (Cline, 1991). Both hormones interact through IAA3/SHY2, which is a negative regulator of auxin signalling that is transcriptionally regulated by CKs through the CK receptor AHK3 and the CK response regulator ARR1,12; a negative regulator of CK signalling (Taniguchi *et al.*, 2007; Dello Ioio *et al.*, 2008). CKs can also modulate auxin gradients through control of PIN protein levels (Stepanova and Alonso, 2011). Similarly, the interaction between developmentally regulated auxin and CK signalling could also mediate basal and induced ontogenic defence responses through their interactions with JA signalling. Although this has not yet been directly demonstrated for the JA pathway, recent modelling approaches combined with experimental analyses demonstrate a critical role for auxin–CK crosstalk in regulating the outcome of defence responses against pathogens in *A. thaliana* (Naseem *et al.*, 2012). The authors showed that resistance to pathogens is maximized by activation of the CK pathway with concomitant inhibition of auxin signalling. This model also predicts an important role for auxin–CK cross-talk in regulating levels of camalexin, the major phytoalexin metabolite of *A. thaliana*, which is involved in plant resistance to biotic stress (Naseem *et al.*, 2012). Using similar approaches to model interactions between growth-related hormones and the JA-dependent defence signalling might be able to provide important insights into the regulation of ontogenic-driven defence metabolite accumulations (Fig. 2B).

CONCLUDING REMARKS

Progress in molecular biology (functional genomics), analytical chemistry (metabolomics) and *in silico* methods (interactomics) is rapidly advancing our understanding of the mechanisms driving physiological processes behind ecologically motivated hypotheses, such as the ODH. The recently identified interactions among signalling pathways involved in plant growth, with defence signalling networks, provide a good starting point for the formulation of hypotheses on the regulation of ontogenically driven defense responses. Fundamental research on cross-talk among growth hormones and defence responses has mostly been performed with *A. thaliana* and often employs the measurements of only a few defence marker genes, whose roles in plant protection against biotic threats are not well established. Newly identified metabolites in *A. thaliana*, with proven defensive properties and whose biosynthesis is regulated by JA, such as the unusual amino acid *N*-δ-acetylornithine (Adio *et al.*, 2011) might provide useful markers for studying mechanisms behind ODH patterns in this plant. However, the number of plant species with fully annotated genomes is steadily increasing. Using other model plant systems with a diverse set of well-defined JA-mediated defence metabolites, whose genomes are accessible to genetic manipulations, will provide important resources to further analyse the contributions of CKs, auxin, GAs, BRs and other developmental pathways, to the within-plant distribution of defensive metabolites predicted by the ODH. However, single hormone pathways are unlikely to fully explain mechanisms behind ODH. Integrating datasets from multiple experiments with plants altered in specific hormone pathways, and using network analysis tools, will advance our understanding of how plants co-regulate tissue value and defensive strength.

ACKNOWLEDGEMENTS

We thank two anonymous reviewers for helpful comments on the manuscript. The work of S.M. is funded by Advanced Grant no. 293926 of the European Research Council to I.T.B. M.E. is supported by a Marie Curie Intra European Fellowship (grant no. 273107). I.T.B. is funded by the Max-Planck Society.

LITERATURE CITED

Achard P, Gusti A, Cheminant S, *et al*. 2009. Gibberellin signaling controls cell proliferation rate in Arabidopsis. *Current Biology* **19**: 1188–1193.

Adio AM, Casteel CL, De Vos M, *et al*. 2011. Biosynthesis and defensive function of Ndelta-acetylornithine, a jasmonate-induced Arabidopsis metabolite. *The Plant Cell* **23**: 3303–3318.

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

Alves MN, Sartoratto A, Trigo JR. 2007. Scopolamine in *Brugmansia suaveolens* (Solanaceae): defense, allocation, costs, and induced response. *Journal of Chemical Ecology* **33**: 297–309.

An CF, Mou ZL. 2011. Salicylic acid and its function in plant immunity. *Journal of Integrative Plant Biology* **53**: 412–428.

Argueso CT, Ferreira FJ, Kieber JJ. 2009. Environmental perception avenues: the interaction of cytokinin and environmental response pathways. *Plant, Cell & Environment* **32**: 1147–1160.

Argueso CT, Ferreira FJ, Epple P, *et al*. 2012. Two-component elements mediate interactions between cytokinin and salicylic acid in plant immunity. *PloS Genetics* **8**: e1002448. http://dx.doi.org/10.1371/journal.pgen.1002448.

Baldwin IT. 1999. Inducible nicotine production in native nicotiana as an example of adaptive phenotypic plasticity. *Journal of Chemical Ecology* **25**: 3–30

Baldwin IT, Karb MJ. 1995. Plasticity in allocation of nicotine to reproductive parts in *Nicotiana attenuata*. *Journal of Chemical Ecology* **21**: 807–809

Baldwin IT, Onmeiss TE. 1993. Alkaloidal responses to damage in Nicotiana native to North America. *Journal of Chemical Ecology* **19**: 1143–1153

Baldwin IT, Zhang ZP, Diab N, *et al*. 1997. Quantification, correlations and manipulations of wound-induced changes in jasmonic acid and nicotine in Nicotiana sylvestris. *Planta* **201**: 397–404.

Bangerth F. 1994. Response of cytokinin concentration in the xylem exudate of bean (*Phaseolus-vulgaris* L) plants to decapitation and auxin treatment, and relationship to apical dominance. *Planta* **194**: 439–442.

Barrett SCH. 2010. Understanding plant reproductive diversity. *Philosophical Transactions of the Royal Society. B: Biological Sciences* **365**: 99–109.

Barto EK, Cipollini D. 2005. Testing the optimal defense theory and the growth-differentiation balance hypothesis in *Arabidopsis thaliana*. *Oecologia* **146**: 169–178.

Blakeslee JJ, Bandyopadhyay A, Lee OR, *et al*. 2007. Interactions among PIN-FORMED and P-glycoprotein auxin transporters in Arabidopsis. *The Plant Cell* **19**: 131–147.

Blilou I, Xu J, Wildwater M, *et al*. 2005. The PIN auxin efflux facilitator network controls growth and patterning in Arabidopsis roots. *Nature* **433**: 39–44.

Booker J, Chatfield S, Leyser O. 2003. Auxin acts in xylem-associated or medullary cells to mediate apical dominance. *The Plant Cell* **15**: 495–507.

Boughton AJ, Hoover K, Felton GW. 2005. Methyl jasmonate application induces increased densities of glandular trichomes on tomato, *Lycopersicon esculentum*. *Journal of Chemical Ecology* **31**: 2211–2216.

Bowers MD, Collinge SK, Gamble SE, Schmitt J. 1992. Effcts of gnotype, habitat,and seasonal variation on iridoid glycoside content of *Plantago lanceolata* (Plantagiaceae) and the implications for insect herbivores. *Oecologia* **91**: 201–207.

Brown PD, Tokuhisa JG, Reichelt M, Gershenzon J. 2003. Variation of glucosinolate accumulation among different organs and developmental stages of *Arabidopsis thaliana*. *Phytochemistry* **62**: 471–481.

Brunt C, Read J, Sanson GD. 2006. Changes in resource concentration and defence during leaf development in a tough-leaved (*Nothofagus moorei*) and soft-leaved (*Toona ciliata*) species. *Oecologia* **148**: 583–592.

Cambier V, Hance T, de Hoffmann E. 2000. Variation of DIMBOA and related compounds content in relation to the age and plant organ in maize. *Phytochemistry* **53**: 223–229.

Campos ML, de Almeida M, Rossi ML, *et al*. 2009. Brassinosteroids interact negatively with jasmonates in the formation of anti-herbivory traits in tomato. *Journal of Experimental Botany* **60**: 4346–4360.

Cerny M, Dycka F, Bobalova J, Brzobohaty B. 2011. Early cytokinin response proteins and phosphoproteins of *Arabidopsis thaliana* identified by proteome and phosphoproteome profiling. *Journal of Experimental Botany* **62**: 921–937.

Cheng H, Song S, Xiao L, *et al*. 2009. Gibberellin acts through jasmonate to control the expression of *MYB21*, *MYB24*, and *MYB57* to promote stamen filament growth in *Arabidopsis*. *PloS Genetics* **5**: e1000440. http://dx.doi.org/10.1371/journal.pgen.1000440.

Chini A, Fonseca S, Fernandez G, *et al*. 2007. The JAZ family of repressors is the missing link in jasmonate signalling. *Nature* **448**: 666–671.

Choi J, Huh SU, Kojima M, Sakakibara H, Paek KH, Hwang I. 2010. The cytokinin-activated transcription factor ARR2 promotes plant immunity via TGA3/NPR1-dependent salicylic acid signaling in arabidopsis. *Developmental Cell* **19**: 284–295.

Cline MG. 1991. Apical dominance. *Botanical Review* **57**: 318–358.

Cortleven A, Remans T, Brenner WG, Valcke R. 2009. Selection of plastid- and nuclear-encoded reference genes to study the effect of altered endogenous cytokinin content on photosynthesis genes in *Nicotiana tabacum*. *Photosynthesis Research* **102**: 21–29.

van Dam NM. 2009. Belowground herbivory and plant defenses. *Annual Review of Ecology Evolution and Systematics* **40**: 373–391.

van Dam NM, Verpoorte R, van der Meijden E. 1994. Extreme differences in pyrrolizidine alkaloid levels between leaves of *Cynoglossum officinale*. *Phytochemistry* **37**: 1013–1016.

van Dam NM, Horn M, Mares M, Baldwin IT. 2001. Ontogeny constrains systemic protease inhibitor response in *Nicotiana attenuata*. *Journal of Chemical Ecology* **27**: 547–568.

De Geyter N, Gholami A, Goormachtig A, Goossens A. 2012. Transcriptional machineries in jasmonate-elicited plant secondary metabolism. *Trends in Plant Science* **17**: 1360–1385.

Del Bianco M, Kepinski S. 2011. Context, specificity, and self-organization in auxin response. *Cold Spring Harbor Perspectives in Biology* **3**: a001578. http://dx.doi.org/10.1101/cshperspect.a001578.

Dello Ioio R, Linhares FS, Scacchi E, *et al*. 2007. Cytokinins determine Arabidopsis root-meristem size by controlling cell differentiation. *Current Biology* **17**: 678–682.

Dello Ioio R, Nakamura K, Moubayidin L, *et al*. 2008. A genetic framework for the control of cell division and differentiation in the root meristem. *Science* **322**: 1380–1384.

Dervinis C, Frost CJ, Lawrence SD, Novak NG, Davis JM. 2010. Cytokinin primes plant responses to wounding and reduces insect performance. *Journal of Plant Growth Regulation* **29**: 289–296.

Dicke M, Baldwin IT. 2010. The evolutionary context for herbivore-induced plant volatiles: beyond the 'cry for help'. *Trends in Plant Science* **15**: 167–175.

Diezel C, Allmann S, Baldwin IT. 2011. Mechanisms of optimal defense patterns in *Nicotiana attenuata*: flowering attenuates herbivory-elicited ethylene and jasmonate signaling. *Journal of Integrative Plant Biology* **53**: 971–983.

Durbak A, Yao H, McSteen P. 2012. Hormone signaling in plant development. *Current Opinion in Plant Biology* **15**: 92–96.

Eklof S, Astot C, Blackwell J, Moritz T, Olsson O, Sandberg G. 1997. Auxin-cytokinin interactions in wild-type and transgenic tobacco. *Plant and Cell Physiology* **38**: 225–235.

Erb M, Meldau S, Howe GA. 2012. Role of phytohormones in insect-specific plant reactions. *Trends in Plant Science* **17**: 250–259.

Feng S, Martinez C, Gusmaroli G, *et al*. 2008. Coordinated regulation of *Arabidopsis thaliana* development by light and gibberellins. *Nature* **451**: 475–479.

Fordyce JA. 2000. A model without a mimic: aristolochic acids from the California pipevine swallowtail, *Battus philenor hirsuta*, and its host plant, *Aristolochia californica*. *Journal of Chemical Ecology* **26**: 2567–2578.

Galinha C, Bilsborough G, Tsiantis M. 2009. Hormonal input in plant meristems: a balancing act. *Seminars in Cell and Developmental Biology* **20**: 1149–1156.

Gallego-Bartolome J, Minguet EG, Marin JA, Prat S, Blazquez MA, Alabadi D. 2010. Transcriptional diversification and functional conservation between DELLA proteins in Arabidopsis. *Molecular Biology and Evolution* **27**: 1247–1256.

Glauser G, Marti G, Villard N, *et al*. 2011. Induction and detoxification of maize 1,4-benzoxazin-3-ones by insect herbivores. *The Plant Journal* **68**: 901–911.

Gleadow RM, Woodrow IE. 2000. Temporal and spatial variation in cyanogenic glycosides in *Eucalyptus cladocalyx*. *Tree Physiology* **20**: 591–598.

Gonzalez-Garcia MP, Vilarrasa-Blasi J, Zhiponova M, *et al*. 2011. Brassinosteroids control meristem size by promoting cell cycle progression in Arabidopsis roots. *Development* **138**: 849–859.

Grieneisen VA, Xu J, Maree AFM, Hogeweg P, Scheres B. 2007. Auxin transport is sufficient to generate a maximum and gradient guiding root growth. *Nature* **449**: 1008–1013.

Griffiths J, Murase K, Rieu I, *et al*. 2006. Genetic characterization and functional analysis of the GID1 gibberellin receptors in Arabidopsis. *The Plant Cell* **18**: 3399–3414.

Grosskinsky DK, Naseem M, Abdelmohsen UR, *et al*. 2011. Cytokinins mediate resistance against *Pseudomonas syringae* in tobacco through

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

increased antimicrobial phytoalexin synthesis independent of salicylic acid signaling. *Plant Physiology* **157**: 815–830.

Grunewald W, Vanholme B, Pauwels L, *et al*. 2009. Expression of the Arabidopsis jasmonate signalling repressor JAZ1/TIFY10A is stimulated by auxin. *EMBO Reports* **10**: 923–928.

Gudesblat GE, Russinova E. 2011. Plants grow on brassinosteroids. *Current Opinion in Plant Biology* **14**: 530–537.

Haberlandt G. 1913. The physiology of cellular division. *Sitzungsberichte der Koniglich Preussischen Akademie der Wissenschaften*: 318–345.

Hacham Y, Holland N, Butterfield C, *et al*. 2011. Brassinosteroid perception in the epidermis controls root meristem size. *Development* **138**: 839–848.

Halitschke R, Baldwin IT. 2003. Antisense LOX expression increases herbivore performance by decreasing defense responses and inhibiting growth-related transcriptional reorganization in *Nicotiana attenuata*. *The Plant Journal* **36**: 794–807.

Halitschke R, Schittko U, Pohnert G, Boland W, Baldwin IT. 2001. Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. III. Fatty acid-amino acid conjugates in herbivore oral secretions are necessary and sufficient for herbivore-specific plant responses. *Plant Physiology* **125**: 711–717.

Hamann T, Mayer U, Jurgens G. 1999. The auxin-insensitive bodenlos mutation affects primary root formation and apical–basal patterning in the Arabidopsis embryo. *Development* **126**: 1387–1395.

Hamann T, Benkova E, Baurle I, Kientz M, Jurgens G. 2002. The Arabidopsis BODENLOS gene encodes an auxin response protein inhibiting MONOPTEROS-mediated embryo patterning. *Genes & Development* **16**: 1610–1615.

He JX, Gendron JM, Yang YL, Li JM, Wang ZY. 2002. The GSK3-like kinase BIN2 phosphorylates and destabilizes BZR1, a positive regulator of the brassinosteroid signaling pathway in Arabidopsis. *Proceedings of the National Academy of Sciences of the USA* **99**: 10185–10190.

Heiling S, Schuman MC, Schoettner M, *et al*. 2010. Jasmonate and ppHsystemin regulate key malonylation steps in the biosynthesis of 17-hydroxygeranyllinalool diterpene glycosides, an abundant and effective direct defense against herbivores in *Nicotiana attenuata*. *The Plant Cell* **22**: 273–292.

Hewelt A, Prinsen E, Thomas M, Van Onckelen H, Meins F. 2000. Ectopic expression of maize knotted1 results in the cytokinin-autotrophic growth of cultured tobacco tissues. *Planta* **210**: 884–889.

Higuchi M, Mähönen AP, Tormakangas K, *et al*. 2004. Arabidopsis lacking all the three cytokinin-receptors exists with a small body. *Plant and Cell Physiology* **45**: pS216.

Hillman JR, Math VB, Medlow GC. 1977. Apical dominance and levels of indole acetic-acid in Phaseolus lateral buds. *Planta* **134**: 191–193.

Hong GJ, Xue XY, Mao YB, Wang LJ, Chen XY. 2012. Arabidopsis MYC2 interacts with DELLA proteins in regulating sesquiterpene synthase gene expression. *The Plant Cell* **24**: 2635–2648.

Howe GA, Jander G. 2008. Plant immunity to insect herbivores. *Annual Review of Plant Biology* **59**: 41–66.

Howe GA, Lightner J, Browse J, Ryan CA. 1996. An octadecanoid pathway mutant (JL5) of tomato is compromised in signaling for defense against insect attack. *The Plant Cell* **8**: 2067–2077.

Huijser P, Schmid M. 2011. The control of developmental phase transitions in plants. *Development* **138**: 4117–4129.

Hyder PW, Fredrickson EL, Estell RE, Tellez M, Gibbens RP. 2002. Distribution and concentration of total phenolics, condensed tannins, and nordihydroguaiaretic acid (NDGA) in creosotebush (*Larrea tridentata*). *Biochemical Systematics and Ecology* **30**: 905–912.

Jasinski S, Piazza P, Craft J, *et al*. 2005. KNOX action in Arabidopsis is mediated by coordinate regulation of cytokinin and gibberellin activities. *Current Biology* **15**: 1560–1565.

Jassbi AR, Gase K, Hettenhausen C, Schmidt A, Baldwin IT. 2008. Silencing geranylgeranyl diphosphate synthase in *Nicotiana attenuata* dramatically impairs resistance to tobacco hornworm. *Plant Physiology* **146**: 974–986.

Jordi W, Schapendonk A, Davelaar E, *et al*. 2000. Increased cytokinin levels in transgenic P-SAG12-IPT tobacco plants have large direct and indirect effects on leaf senescence, photosynthesis and N partitioning. *Plant, Cell & Environment* **23**: 279–289.

Kang JH, Liu GH, Shi F, Jones AD, Beaudry RM, Howe GA. 2010. The tomato odorless-2 mutant is defective in trichome-based production of diverse specialized metabolites and broad-spectrum resistance to insect herbivores. *Plant Physiology* **154**: 262–272.

Katsir L, Schilmiller AL, Staswick PE, He SY, Howe GA. 2008. COI1 is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatine. *Proceedings of the National Academy of Sciences of the USA* **105**: 7100–7105.

Kaur H, Heinzel N, Schottner M, Baldwin IT, Galis I. 2010. R2R3-NaMYB8 regulates the accumulation of phenylpropanoid-polyamine conjugates, which are essential for local and systemic defense against insect herbivores in *Nicotiana attenuata*. *Plant Physiology* **152**: 1731–1747.

Kessler D, Baldwin IT. 2011. Back to the past for pollination biology. *Current Opinion in Plant Biology* **14**: 429–434.

Kessler D, Gase K, Baldwin IT. 2008. Field experiments with transformed plants reveal the sense of floral scents. *Science* **321**: 1200–1202.

Kessler D, Bhattacharya S, Diezel C, *et al*. 2012. Unpredictability of nectar nicotine promotes outcrossing by hummingbirds in *Nicotiana attenuata*. *The Plant Journal* **71**: 529–538.

Kim TW, Wang ZY. 2010. Brassinosteroid signal transduction from receptor kinases to transcription factors. *Annual Review of Plant Biology* **61**: 681–704.

Kobayashi K, Baba S, Obayashi T, *et al*. 2012. Regulation of root greening by light and auxin/cytokinin signaling in Arabidopsis. *The Plant Cell* **24**: 1081–1095.

Köllner TG, Schnee C, Gershenzon J, Degenhardt J. 2004. The sesquiterpene hydrocarbons of maize (*Zea mays*) form five groups with distinct developmental and organ-specific distribution. *Phytochemistry* **65**: 1895–1902.

Kurakawa T, Ueda N, Maekawa M, *et al*. 2007. Direct control of shoot meristem activity by a cytokinin-activating enzyme. *Nature* **445**: 652–655.

Lara MEB, Garcia MCG, Fatima T, *et al*. 2004. Extracellular invertase is an essential component of cytokinin-mediated delay of senescence. *The Plant Cell* **16**: 1276–1287.

Li CY, Williams MM, Loh YT, Lee GI, Howe GA. 2002*a*. Resistance of cultivated tomato to cell content-feeding herbivores is regulated by the octadecanoid-signaling pathway. *Plant Physiology* **130**: 494–503.

Li J, Wen JQ, Lease KA, Doke JT, Tax FE, Walker JC. 2002*b*. BAK1, an Arabidopsis LRR receptor-like protein kinase, interacts with BRI1 and modulates brassinosteroid signaling. *Cell* **110**: 213–222.

Ljung K, Bhalerao RP, Sandberg G. 2001. Sites and homeostatic control of auxin biosynthesis in Arabidopsis during vegetative growth. *The Plant Journal* **28**: 465–474.

Long JA, Woody S, Poethig S, Meyerowitz EM, Barton K. 2002. Transformation of shoots into roots in Arabidopsis embryos mutant at the TOPLESS locus. *Development* **129**: 2797–2806.

Long JA, Ohno C, Smith ZR, Meyerowitz EM. 2006. TOPLESS regulates apical embryonic fate in Arabidopsis. *Science* **312**: 1520–1523.

de Lucas M, Daviere JM, Rodriguez-Falcon M, *et al*. 2008. A molecular framework for light and gibberellin control of cell elongation. *Nature* **451**: 480–484.

McCall AC, Fordyce JA. 2010. Can optimal defence theory be used to predict the distribution of plant chemical defences? *Journal of Ecology* **98**: 985–992.

McKey D. 1974. Adaptive patterns in alkaloid physiology. *American Naturalist* **108**: 305–320.

McSteen P, Leyser O. 2005. Shoot branching. *Annual Review of Plant Biology* **56**: 353–374.

Mediene S, Pages L, Jordan MO, Le Bot J, Adamowicz S. 2002. Influence of nitrogen availability on shoot development in young peach trees [*Prunus persica* (L.) Batsch]. *Trees – Structure and Function* **16**: 547–554.

Mewis I, Tokuhisa JG, Schultz JC, Appel HM, Ulrichs C, Gershenzon J. 2006. Gene expression and glucosinolate accumulation in *Arabidopsis thaliana* in response to generalist and specialist herbivores of different feeding guilds and the role of defense signaling pathways. *Phytochemistry* **67**: 2450–2462.

Miller CO, Skoog F, Vonsaltza MH, Strong FM. 1955. Kinetin, a cell division factor from deoxyribonucleic acid. *Journal of the American Chemical Society* **77**: 1392–1392.

Mitra S, Baldwin IT. 2008. Independently silencing two photosynthetic proteins in *Nicotiana attenuata* has different effects on herbivore resistance. *Plant Physiology* **148**: 1128–1138.

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

Morris DA. 1977. Transport of exogenous auxin in 2-branched dwarf pea-seedlings (*Pisum sativum*-L): some implications for polarity and apical dominance. *Planta* **136**: 91–96.

Nagpal P, Ellis CM, Weber H, *et al*. 2005. Auxin response factors ARF6 and ARF8 promote jasmonic acid production and flower maturation. *Development* **132**: 4107–4118.

Nakaya M, Tsukaya H, Murakami N, Kato M. 2002. Brassinosteroids control the proliferation of leaf cells of *Arabidopsis thaliana*. *Plant and Cell Physiology* **43**: 239–244.

Nam KH, Li JM. 2002. BRI1/BAK1, a receptor kinase pair mediating brassinosteroid signaling. *Cell* **110**: 203–212.

Naseem M, Philippi N, Hussain A, Wangorsch G, Ahmed N, Dandekar T. 2012. Integrated systems view on networking by hormones in Arabidopsis immunity reveals multiple crosstalk for cytokinin. *The Plant Cell* **24**:1793–1814.

Navarro L, Bari R, Achard P, *et al*. 2008. DELLAs control plant immune responses by modulating the balance of jasmonic acid and salicylic acid signaling. *Current Biology* **18**: 650–655.

Nishimura C, Ohashi Y, Sato S, Kato T, Tabata S, Ueguchi C. 2004. Histidine kinase homologs that act as cytokinin receptors possess overlapping functions in the regulation of shoot and root growth in Arabidopsis. *The Plant Cell* **16**: 1365–1377.

Nordstrom A, Tarkowski P, Tarkowska D, *et al*. 2004. Auxin regulation of cytokinin biosynthesis in *Arabidopsis thaliana*: a factor of potential importance for auxin-cytokinin-regulated development. *Proceedings of the National Academy of Sciences of the USA* **101**: 8039–8044.

Oh Y, Baldwin IT, Galis I. 2012. NaJAZh regulates a subset of defense responses against herbivores and spontaneous leaf necrosis in *Nicotiana attenuata* plants. *Plant Physiology* **159**: 769–788.

Ohnmeiss TE, Baldwin IT. 2000. Optimal Defense theory predicts the ontogeny of an induced nicotine defense. *Ecology* **81**: 1765–1783.

Onkokesung N, Gaquerel E, Kotkar H, Kaur H, Baldwin IT, Galis I. 2012. MYB8 controls inducible phenolamide levels by activating three novel hydroxycinnamoyl-Coenzyme A:Polyamine transferases in *Nicotiana attenuata*. *Plant Physiology* **158**: 389–407.

Ori N, Juarez MT, Jackson D, Yamaguchi J, Banowetz GM, Hake S. 1999. Leaf senescence is delayed in tobacco plants expressing the maize homeobox gene knotted1 under the control of a senescence-activated promoter. *The Plant Cell* **11**: 1073–1080.

Paschold A, Halitschke R, Baldwin IT. 2007. Co(i)-ordinating defenses: NaCOI1 mediates herbivore- induced resistance in *Nicotiana attenuata* and reveals the role of herbivore movement in avoiding defenses. *The Plant Journal* **51**: 79–91.

Pauwels L, Barbero GF, Geerinck J, *et al*. 2010. NINJA connects the co-repressor TOPLESS to jasmonate signalling. *Nature* **464**: 788–791.

Peng ZH, Han CY, Yuan LB, Zhang K, Huang HM, Ren CM. 2011. Brassinosteroid enhances jasmonate-induced anthocyanin accumulation in Arabidopsis seedlings. *Journal of Integrative Plant Biology* **53**: 632–640.

Petersson SV, Johansson AI, Kowalczyk M, *et al*. 2009. An auxin gradient and maximum in the Arabidopsis root apex shown by high-resolution cell-specific analysis of IAA distribution and synthesis. *The Plant Cell* **21**: 1659–1668.

Pichersky E, Lewinsohn E. 2011. Convergent evolution in plant specialized metabolism. *Annual Review of Plant Biology* **62**: 549–566.

Polanska L, Vicankova A, Novakova M, *et al*. 2007. Altered cytokinin metabolism affects cytokinin, auxin, and abscisic acid contents in leaves and chloroplasts, and chloroplast ultrastructure in transgenic tobacco. *Journal of Experimental Botany* **58**: 637–649.

Radhika V, Kost C, Bartram S, Heil M, Boland W. 2008. Testing the optimal defence hypothesis for two indirect defences: extrafloral nectar and volatile organic compounds. *Planta* **228**: 449–457.

Reichelt M, Brown PD, Schneider B, *et al*. 2002. Benzoic acid glucosinolate esters and other glucosinolates from *Arabidopsis thaliana*. *Phytochemistry* **59**: 663–671.

Ren CM, Han CY, Peng W, *et al*. 2009. A leaky mutation in DWARF4 reveals an antagonistic role of brassinosteroid in the inhibition of root growth by jasmonate in Arabidopsis. *Plant Physiology* **151**: 1412–1420.

Robert CA, Veyrat N, Glauser G, *et al*. 2012. A specialist root herbivore exploits defensive metabolites to locate nutritious tissues. *Ecology Letters* **15**: 55–64.

Robert-Seilaniantz A, Grant M, Jones JDG. 2011. Hormone crosstalk in plant disease and defense: more than just JASMONATE-SALICYLATE antagonism. *Annual Review of Phytopathology* **49**: 317–343.

Rostas M, Eggert K. 2008. Ontogenetic and spatio-temporal patterns of induced volatiles in *Glycine max* in the light of the optimal defence hypothesis. *Chemoecology* **18**: 29–38.

Ruffel S, Krouk G, Ristova D, Shasha D, Birnbaum KD, Coruzzi GM. 2011. Nitrogen economics of root foraging: transitive closure of the nitrate-cytokinin relay and distinct systemic signaling for N supply vs. demand. *Proceedings of the National Academy of Sciences of the USA* **108**: 18524–18529.

Sano H, Seo S, Koizumi N, Niki T, Iwamura H, Ohashi Y. 1996. Regulation by cytokinins of endogenous levels of jasmonic and salicylic acids in mechanically wounded tobacco plants. *Plant and Cell Physiology* **37**: 762–769.

van Schie CCN, Haring MA, Schuurink RC. 2007. Tomato linalool synthase is induced in trichomes by jasmonic acid. *Plant Molecular Biology* **64**: 251–263.

Shinshi H. 2008. Ethylene-regulated transcription and crosstalk with jasmonic acid. *Plant Science* **175**: 18–23.

Smigocki A, Neal JW, Mccanna I, Douglass L. 1993. Cytokinin-mediated insect resistance in Nicotiana plants transformed with the Ipt gene. *Plant Molecular Biology* **23**: 325–335.

Stepanova AN, Alonso JM. 2011. Bypassing transcription: a shortcut in cytokinin–auxin interactions. *Developmental Cell* **21**: 608–610.

Sun TP. 2010. Gibberellin-GID1-DELLA: a pivotal regulatory module for plant growth and development. *Plant Physiology* **154**: 567–570.

Swarup R, Friml J, Marchant A, *et al*. 2001. Localization of the auxin permease AUX1 suggests two functionally distinct hormone transport pathways operate in the Arabidopsis root apex. *Genes & Development* **15**: 2648–2653.

Szemenyei H, Hannon M, Long JA. 2008. TOPLESS mediates auxin-dependent transcriptional repression during Arabidopsis embryogenesis. *Science* **319**: 1384–1386.

Taniguchi M, Sasaki N, Tsuge T, Aoyama T, Oka A. 2007. ARR1 directly activates cytokinin response genes that encode proteins with diverse regulatory functions. *Plant and Cell Physiology* **48**: 263–277.

Thaler JS. 1999. Induced resistance in agricultural crops: effects of jasmonic acid on herbivory and yield in tomato plants. *Environmental Entomology* **28**: 30–37.

Thimann KV, Skoog F. 1933. Studies on the growth hormone of plants. III. The inhibiting action of the growth substance on bud development. *Proceedings of the National Academy of Sciences of the USA* **19**: 714–716.

Thines B, Katsir L, Melotto M, *et al*. 2007. JAZ repressor proteins are targets of the SCF(COI1) complex during jasmonate signalling. *Nature* **448**: 661–665.

Tiryaki I, Staswick PE. 2002. An Arabidopsis mutant defective in jasmonate response is allelic to the auxin-signaling mutant axr1. *Plant Physiology* **130**: 887–894.

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

Traw MB, Feeny P. 2008. Glucosinolates and trichomes track tissue value in two sympatric mustards. *Ecology* **89**: 763–772.

Ueguchi-Tanaka M, Ashikari M, Nakajima M, *et al*. 2005. GIBBERELLIN INSENSITIVE DWARF1 encodes a soluble receptor for gibberellin. *Nature* **437**: 693–698.

Ueguchi-Tanaka M, Nakajima M, Katoh E, *et al*. 2007. Molecular interactions of a soluble gibberellin receptor, GID1, with a rice DELLA protein, SLR1, and gibberellin. *The Plant Cell* **19**: 2140–2155.

Vidaurre DP, Ploense S, Krogan NT, Berleth T. 2007. AMP1 and MP antagonistically regulate embryo and meristem development in Arabidopsis. *Development* **134**: 2561–2567.

Walling LL. 2009. Adaptive defense responses to pathogens and insects. *Plant Innate Immunity* **51**: 551–612.

Wasternack C, Kombrink E. 2010. Jasmonates: structural requirements for lipid-derived signals active in plant stress responses and development. *ACS Chemical Biology* **5**: 63–77.

Wellmer F, Riechmann JL. 2010. Gene networks controlling the initiation of flower development. *Trends in Genetics* **26**: 519–527.

Werner T, Motyka V, Laucou V, Smets R, Van Onckelen H, Schmulling T. 2003. Cytokinin-deficient transgenic Arabidopsis plants show multiple developmental alterations indicating opposite functions of cytokinins in

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022

the regulation of shoot and root meristem activity. *The Plant Cell* **15**: 2532–2550.

Xu J, Hofhuis H, Heidstra R, Sauer M, Friml J, Scheres B. 2006. A molecular framework for plant regeneration. *Science* **311**: 385–388.

Xu LH, Liu FQ, Lechner E, *et al*. 2002. The SCFCOI1 ubiquitin-ligase complexes are required for jasmonate response in Arabidopsis. *The Plant Cell* **14**: 1919–1935.

Yamaguchi S. 2008. Gibberellin metabolism and its regulation. *Annual Review of Plant Biology* **59**: 225–251.

Yan YX, Christensen S, Isakeit T, *et al*. 2012. Disruption of OPR7 and OPR8 reveals the versatile functions of jasmonic acid in maize development and defense. *The Plant Cell* **24**: 1420–1436.

Yanai O, Shani E, Dolezal K, *et al*. 2005. Arabidopsis KNOXI proteins activate cytokinin biosynthesis. *Current Biology* **15**: 1566–1571.

Yang DH, Hettenhausen C, Baldwin IT, Wu J. 2011. BAK1 regulates the accumulation of jasmonic acid and the levels of trypsin proteinase inhibitors in *Nicotiana attenuata*'s responses to herbivory. *Journal of Experimental Botany* **62**: 641–652.

Yang DL, Yao J, Mei CS, *et al*. 2012. Plant hormone jasmonate prioritizes defense over growth by interfering with gibberellin signaling cascade. *Proceedings of the National Academy of Sciences of the USA* **109**: E1192–1200.

Yin YH, Wang ZY, Mora-Garcia S, *et al*. 2002. BES1 accumulates in the nucleus in response to brassinosteroids to regulate gene expression and promote stem elongation. *Cell* **109**: 181–191.

Yoshida Y, Sano R, Wada T, Takabayashi J, Okada K. 2009. Jasmonic acid control of GLABRA3 links inducible defense and trichome patterning in Arabidopsis. *Development* **136**: 1039–1048.

Zangerl AR, Nitao JK. 1998. Optimal defence, kin conflict and the distribution of furanocoumarins among offspring of wild parsnip. *Evolutionary Ecology* **12**: 443–457.

Zavala JA, Patankar AG, Gase K, Hui D, Baldwin IT. 2004. Manipulation of endogenous trypsin proteinase inhibitor production in *Nicotiana attenuata* demonstrates their function as antiherbivore defenses. *Plant Physiology* **134**: 1181–1190.

Zhang WJ, To JPC, Cheng CY, Schaller GE, Kieber JJ. 2011. Type-A response regulators are required for proper root apical meristem function through post-transcriptional regulation of PIN auxin efflux carriers. *The Plant Journal* **68**: 1–10.

Zubo YO, Yamburenko MV, Selivankina SY, *et al*. 2008. Cytokinin stimulates chloroplast transcription in detached barley leaves. *Plant Physiology* **148**: 1082–1093.

Downloaded from https://academic.oup.com/aob/article/110/8/1503/159755 by Universitätsbibliothek Bern user on 30 November 2022