New Phytologist

Review

Tansley review

Coordinated resource allocation to plant growth–defense tradeoffs

Author for correspondence:
Russell K. Monson
Email: russell.monson@colorado.edu

Received: *24 April 2021*
Accepted: *9 September 2021*

Russell K. Monson[1], Amy M. Trowbridge[2], Richard L. Lindroth[2] and Manuel T. Lerdau[3]

[1]Department of Ecology and Evolutionary Biology, University of Colorado, Boulder, CO 80309, USA; [2]Department of Entomology, University of Wisconsin-Madison, Madison, WI 53706, USA; [3]Department of Environmental Sciences, University of Virginia, Charlottesville, VA 22904, USA

Contents

Summary 1
I. Introduction 1
II. Growth–defense tradeoffs during the era of molecular discovery 2
III. Existing tradeoff hypotheses 3
IV. The coordinated resource allocation hypothesis 5
V. Cellular mechanisms of coordinated resource allocation 7
VI. Selection has shaped both the coordination process and the reserve safety margins that lead to phenotypic resilience 11
VII. Conclusions and future directions 12
Acknowledgements 13
References 13

New Phytologist (2021)
doi: 10.1111/nph.17773

Key words: constitutive, gibberellin, herbivory, induced, jasmonic acid, pathogen, phytochrome, salicylic acid.

Summary

Plant resource allocation patterns often reveal tradeoffs that favor growth (G) over defense (D), or vice versa. Ecologists most often explain G–D tradeoffs through principles of economic optimality, in which negative trait correlations are attributed to the reconciliation of fitness costs. Recently, researchers in molecular biology have developed 'big data' resources including multi-omic (e.g. transcriptomic, proteomic and metabolomic) studies that describe the cellular processes controlling gene expression in model species. In this synthesis, we bridge ecological theory with discoveries in multi-omics biology to better understand how selection has shaped the mechanisms of G–D tradeoffs. Multi-omic studies reveal strategically coordinated patterns in resource allocation that are enabled by phytohormone crosstalk and transcriptional signal cascades. Coordinated resource allocation justifies the framework of optimality theory, while providing mechanistic insight into the feedbacks and control hubs that calibrate G–D tradeoff commitments. We use the existing literature to describe the coordinated resource allocation hypothesis (CoRAH) that accounts for balanced cellular controls during the expression of G–D tradeoffs, while sustaining stored resource pools to buffer the impacts of future stresses. The integrative mechanisms of the CoRAH unify the supply- and demand-side perspectives of previous G–D tradeoff theories.

I. Introduction

For over 50 yr, researchers have relied on an economic analogy to describe resource-use and life-history attributes in theoretical treatments of plant form and function (Harper, 1967; Cohen, 1971; Mooney, 1972; Bloom *et al.*, 1985; Bazzaz *et al.*, 1987; Chapin *et al.*, 1990; Lerdau, 1992; Wright *et al.*, 2004; Michaletz *et al.*, 2016; Agrawal, 2020). Evolutionary optimization is a central tenet of the economic analogy (Parker & Maynard, 1990). Optimization is built on the concept that selection balances phenotypic traits according to cost–benefit criteria and produces resource-use patterns that maximize physiological responses to

stress and, ultimately, fitness (Schluter *et al.*, 1991; Deans *et al.*, 2020). Historically, models and theories employing economic optimization underlie some of our most enduring concepts concerning plant traits and adaptation. For example, Chapin (1980) used optimality concepts to explain the costs and benefits of trait syndromes associated with nutrient acquisition and resource-use efficiency. Mooney & Gulmon (1982) developed optimality arguments to explain trait correlations among leaf longevity, photosynthesis rate and defense investment. More recently, a variety of researchers have used economic concepts to justify trait correlations, or lack thereof, within the context of the global trait economics spectrum (Westoby *et al.*, 2002; Reich *et al.*, 2003; Wright *et al.*, 2004; Reich, 2014; Kruger *et al.*, 2020; Vleminckx *et al.*, 2021).

Correlated trait associations are attributed to multiple causes, including phenotypic plasticity, pleiotropy and niche specialization (Reznick, 1985; Cheverud, 1988; Futuyma & Moreno, 1988; Koricheva, 2002a). Negative trait associations, or tradeoffs, are most often explained as trait expression patterns that result from selection that reconciles competitive resource demands (e.g. Williams, 1966; Cohen, 1971; Reznick, 1985). However, other causes are possible, such as those resulting from ecological interactions and direct conflicts among trait functions (Stearns, 1977; Agrawal, 2020). One of the more frequently discussed trait tradeoffs is that for plant growth (G) vs defense (D) (Coley *et al.*, 1985; Bazzaz *et al.*, 1987; Herms & Mattson, 1992; Huot *et al.*, 2014; Schuman & Baldwin, 2016; Züst & Agrawal, 2017). Competing G–D demands are assumed to respond such that resource allocation to the process that least impacts fitness is reduced. This allows additional resources to flow to the process that most affects fitness, which results in a tradeoff. Growth–defense tradeoffs are influenced by genetic and environmental factors, as well as inherent physiological constraints such as internal resource recycling. Consequently, the causes of negatively associated allocational patterns and their impacts on fitness have been challenging to resolve (Reznick, 1985; Bergelson & Purrington, 1996; Koricheva, 2002a; Hahn & Maron, 2016; Ullmann-Zeunert *et al.*, 2016; Valim *et al.*, 2020). The pursuit of studies at the cellular scale improves our understanding of the controls over resource allocation patterns and clarifies the modes by which selection favors tradeoffs (Futuyma & Moreno, 1988; Schuman & Baldwin, 2016; Züst & Agrawal, 2017).

At the turn of the millennium, Coruzzi & Bush (2001) published studies revealing the prominent role of cellular signaling systems as homeostatic controls over trait expression and resource allocation. At about the same time, Kessler & Baldwin (2002) forecast the potential opportunities for knowledge of cellular-scale processes to further our understanding of plant defensive traits. Since those early reports, hundreds of gene-expression studies have been published that take advantage of genomic and data-analytic techniques. In the past two decades, researchers have produced a 'big data' revolution using multi-omic techniques (e.g. genomics, transcriptomics, proteomics and metabolomics). By employing these approaches, we can observe patterns of trait covariance at the scale of gene expression and thus mechanistically evaluate previous theories and models that were constructed at the organismal scale. For a variety of reasons, researchers have been slow to use these breakthroughs to bridge conceptual gaps across disciplinary scales. Most cellular studies concern a limited number of model species. This, along with complexity in the concepts and language of multi-omic approaches, has kept a majority of ecologists from applying cellular-scale discoveries to their own studies. Research within the cellular biology community, in turn, has focused on mechanistic nuance and has often lacked ecological realism such as context dependency.

In this synthesis, we consider recent breakthroughs in cellular biology to reassess the economic and optimization frameworks that underlie G–D tradeoff hypotheses. We ask the question: are the most frequently tested G–D tradeoff hypotheses, which were developed from organism-scale observations, supported by recent insights from studies on cellular processes? To focus specifically on this question, we have omitted coverage of several dimensions within the more general topic of plant defense theory, including the roles of ontogeny (Boege & Marquis, 2005), community diversity (Whitehead *et al.*, 2021), plant mating systems (Campbell *et al.*, 2014), competition (Viola *et al.*, 2010) and herbivore traits (Pacala & Crawley, 1992). Our starting point is a discussion of established G–D tradeoff hypotheses, which introduces the principal ecological and evolutionary perspectives at the organismic scale. From there, we move to discussions of cellular processes and initiate an analysis of the mechanisms capable of explaining selection for specific G and D traits. Using perspectives from multi-omic biology, we develop a new hypothesis – the coordinated resource allocation hypothesis (CoRAH). This hypothesis provides a means to connect previous tradeoff hypotheses based on economic optimization to mechanisms of cellular control. Our overall goal in presenting this synthesis is to illustrate the potential to use CoRAH, along with multi-trait discoveries at the cellular scale, to inform us about the underlying tenets of established ecological theory. As molecular and cellular biologists continue to produce multi-omic insights, a primary challenge will be to convert 'big data' into 'smart data', thus empowering a new era of cross-scale understanding and integration in ecology and plant physiology.

II. Growth–defense tradeoffs during the era of molecular discovery

> In general, tradeoffs cannot be assumed a priori since the tolerance function of individual phenotypes, which determines the cost or fitness function, is precisely what needs explanation. Empirical analysis of biochemical, physiological or morphological function is necessary to determine if a postulated tradeoff actually exists. Futuyma & Moreno (1988)

Conventional treatments of tradeoffs have emphasized trait–environment interactions at the organismic scale, or above (Agrawal, 2020). However, traits are the product of developmental processes that draw on specific types of organic substrates, providing the mass and energy for cellular and tissue construction. Selection that favors one form of a trait over another at the scale of organisms also favors differential patterns in the deployment of biochemical pathways and the expression of genes that control

pathway activities (Fig. 1a). In considering tradeoffs at the cellular scale it becomes possible to recognize more clearly threads of continuity from the regulatory processes that determine gene expression, through biochemical pathways, to the developmental processes that produce organismic traits.

Züst & Agrawal (2017) discussed two opposing forces that operate in sequence and result in negative trait tradeoffs through a process of stabilizing selection: one that initially favors increased resource investment in the traits that most improve fitness, relative those that least improve fitness; and one that progressively diminishes the benefit-to-cost difference between the two sets of traits. The latter force includes increased risks as a result of the de-emphasis of traits receiving less resource investment and the increased importance of ecological costs (*sensu* Strauss *et al.*, 2002). A complementary view is that the enhanced expression of one trait at the expense of another is a result of selection that has favored strategic coordination of resource allocation which, in turn, relies on cellular signaling and pathway feedbacks (Fig. 1b).

One can think of cellular regulation as an adaptive set of physiological traits, in and of themselves, although the experimental support for this notion is currently weak (e.g. Thaler *et al.*, 2012). Regulatory mechanisms allow plants to avoid internal resource limitations that unfavorably constrain responses to a variable environment (Schuman & Baldwin, 2016). Cellular regulatory systems are distinct from the anabolic and catabolic pathways that produce or consume trait substrates. Some of these perspectives were originally developed in the writings of Loomis (1932) and Herms & Mattson (1992), in which the classic dilemma for plants – to grow or defend – was presented as a fundamental tradeoff. With the advent of recent multi-omic discoveries we can now move even closer to the foundation of these interactions.

One issue that requires clarification when cell-to-organism scales are integrated is the accommodation of variable ways to gauge G. Researchers working at the cellular scale often focus on G through cell expansion or elongation, whereas ecologists focus on rates of biomass increase or C assimilation – growth metrics that are more closely associated with fitness. Although all modes of G will most likely scale in similar directions (increase or decrease) when faced with resource limitations, they will vary in their quantitative tradeoffs with D. Within a conceptual context, this is less of a problem than when attempting to model quantitative outcomes. However, it does complicate the language surrounding tradeoffs, particularly when using literature that spans cellular to organism scales.

III. Existing tradeoff hypotheses

1. The primary theories

Four general G–D tradeoff hypotheses have dominated our thinking over the past 35 yr (Stamp, 2003). Three of those hypotheses (the carbon-nutrient balance hypothesis, the resource availability hypothesis, and the growth-differentiation balance hypothesis) fall largely within the context of supply-side processes

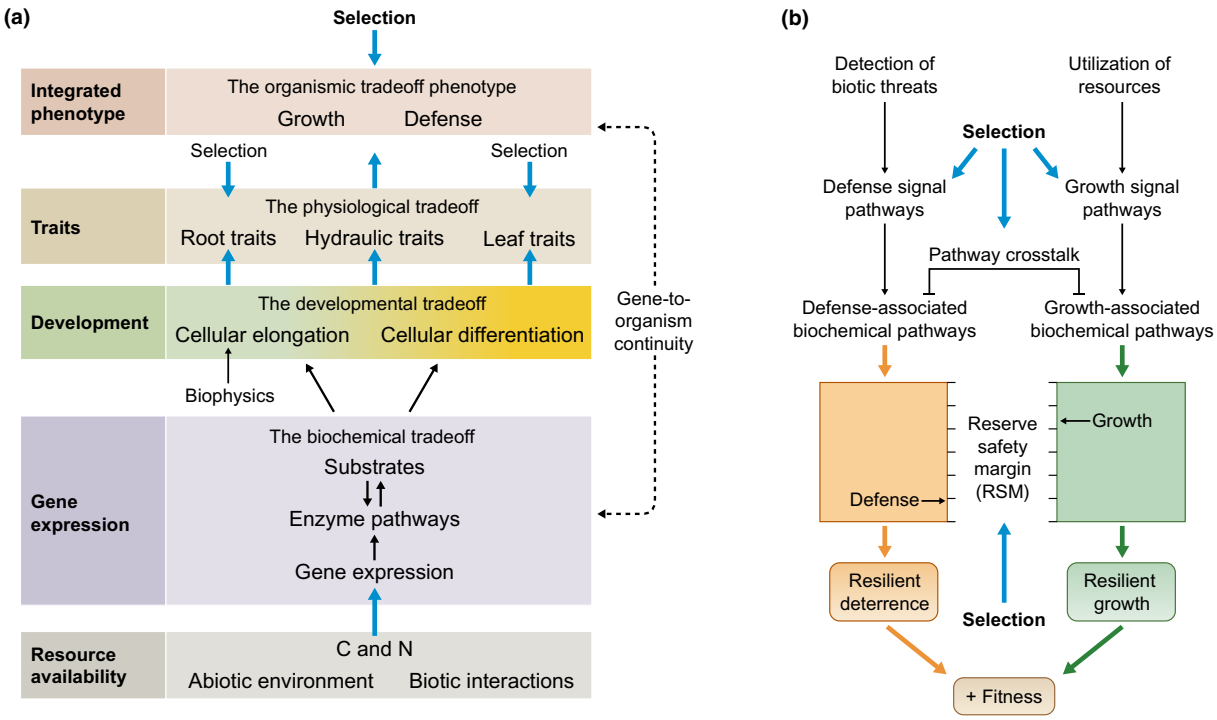

Fig. 1 (a) Scaled continuity in expression of the growth–defense tradeoff. (b) Selection has favored a coordinated form of resource allocation to growth or defense, dependent on phytohormone crosstalk among metabolic pathways and the maintenance of reserve safety margins (RSMs), both of which confer resilience to patterns of growth and defense in the face of environmental variability. Crosstalk signaling and associated feedbacks produce a strategic coordination in the expression of biochemical pathway fluxes that produce negative correlations among traits, and preserve RSMs for resilient phenotypic responses that deter biotic attacks, maximize growth and enhance fitness.

and one (the optimal defense hypothesis) falls within the context of demand-side processes.

The carbon-nutrient balance hypothesis (CNBH, see Table 1 for abbreviations), originally described in Bryant *et al.* (1983), used interspecific comparisons to present the case for differential allocation to G and D, but the mechanism that was proposed carried limited costs to fitness and was relevant only to intraplant patterns of resource allocation (Hamilton *et al.*, 2001; Koricheva, 2002b; Lerdau & Coley, 2002). In the condition of high light and low soil fertility, meristematic activity is limited by nutrients, causing a supply-side adjustment that transfers photosynthate in excess of that required for G, to D. This hypothesis epitomizes the resource 'overflow' concept, which in turn is embedded in the 'mass action' concept of metabolite flux. Thus, the CNBH is fundamentally a 'physiological' model driven by source-sink relations. The CNBH is operationally outside the economic framework of costs, benefits and tradeoffs, and is thus orthogonal to optimization modeling.

Table 1 The most frequently used abbreviations and accompanying terms.

Abbreviation	Term
ABA	Abscisic acid
A_{max}	Maximum net CO_2 assimilation rate (g CO_2 g^{-1} biomass d^{-1})
bHLH	basic helix–loop–helix (transcription factor family)
CNBH	Carbon-nutrient balance hypothesis
COI1	Coronatine Insensitive 1 (protein) (JA signaling)
CoRA(H)	Coordinated resource allocation (hypothesis)
DAMP	Damage-associated molecular pattern
DELLA	(aspartate-glutamate-leucine-leucine-alanine)-motif protein family (GA signaling)
ERF	ETS2 repressor factor (transcription factor family)
ET	Ethylene
ETI	Effector-triggered immunity
GDBH	Growth-differentiation balance hypothesis
GID1	Gibberellic acid-Insensitive Dwarf 1 (GA signal receptor protein)
HAMP	Herbivore-associated molecular pattern
JA	Jasmonic acid
JA-Ile	Jasmonoyl L-Isoleucine (active form of JA signal)
JAZ	Jasmonate ZIM (zinc finger) domain proteins (JA signaling)
MAMP	Microbial-associated molecular pattern
MAP	Mitogen-activated protein (kinase)
MYC	Myelocytomatosis (transcription factor family)
ODH	Optimal defense hypothesis
PAMP	Pathogen-associated molecular pattern
PCD	Programmed cell death
PhyB	Phytochrome B
PDF1.2a	Plant Defensin 1.2a (defense protein)
PIF	Phytochrome interacting factor (transcription factor)
PRR	Pattern recognition receptor
PTI	Pattern-triggered immunity
RAH	Resource availability hypothesis
RNAi	RNA interference (genetic transformation technology)
ROS	Reactive oxygen species
SA	Salicylic acid
SAR	Systemic acquired resistance
SAS	Shade avoidance syndrome
SCF^{COI}	SKP1-CUL1-F-box (E3 ligase family with COI F-box protein) (JA signaling)
SCF^{SLY1}	SKP1-CUL1-F-box (E3 ligase family with SLEEPY1 F-box protein) (GA signaling)
SnRK1	SNF1-Related Protein Kinase 1
TBF1	TL1-binding factor 1 (transcription factor)
TF	Transcription factor
TORC1	Target of rapamycin complex 1
Thi2.1	Thionin 2.1 (defense protein)
VSP2	Vegetative Storage Protein 2 (defense protein)

The resource allocation hypothesis (RAH), developed in Coley *et al.* (1985), starts from the CNBH premise of resource balance, but provides an explanation based on evolutionary optimization that brings the focus back to interspecific comparisons. According to the RAH, selection targets G within the 'acquisition' constraint of resource availability, and the G–D tradeoff within the 'allocation' constraint of resource partitioning. Thus, slower growth rates and higher levels of defense occur together in plants from resource-limited environments, compared with plants from resource-rich environments. The RAH exemplifies the so-called 'teeter-totter' model in which priorities for resource allocation to G or D, are coupled and mutually exclusive at any moment in time (Kliebenstein, 2016; Schuman & Baldwin, 2016). Van der Meijden *et al.* (1988) developed a slightly different perspective by focusing on G following defoliation; thus, uncoupling the allocation constraint in time. In this view, selection for rapid G *after* herbivory should provide phenotypic compensation for lower investment in D *before* herbivory. This results in the same phenotypic outcome predicted by the RAH – higher G with lower D – but without selection that reconciles simultaneous, competitive fitness costs. This temporally uncoupled mode of selection might also operate within the processes of coordinated resource allocation theories, and could, in theory, enhance the selective value of resource reserves (although see Section IV).

The growth-differentiation balance hypothesis (GDBH) presented in Herms & Mattson (1992), as an extension of Loomis (1932), starts from the assumption that photosynthetic production is not adequate to optimally provision both G and D, thus forcing the evolution of tradeoffs. The GDBH was originally developed from economic perspectives applied to plant physiology and metabolite partitioning. In one section of their paper (pp. 287–288), the authors discussed G–D tradeoffs as cases of optimization in coordinated source-sink activities. (This perspective is similar to the CoRAH model that we present later.) Even within this framework, however, the dominant tone of the GDBH is one of substrate-driven, supply-side control. Cellular mechanisms, especially those on the demand (sink) side of the economic analogy, were not well enough described to constrain the scope and form of cellular coordination mechanisms when Herms & Mattson (1992) originally presented their model. Within this context, our development of the CoRAH in this synthesis is a sequel to the original GDBH, but with updated capacity to integrate supply- and demand-side processes (*sensu* Lerdau *et al.*, 1994).

Another view of evolutionary processes within G and D resource allocation is offered by apparency models (e.g. Feeny, 1976), which are best exemplified by the optimal defense theory (ODH) (Rhoades, 1979). The underlying premises of the ODH are framed in demand-side processes (i.e. those associated with herbivore/pathogen selection pressures). Similar to the supply-side theories offered in the RAH and GDBH, the ODH is

ultimately grounded in evolutionary optimality. However, the ODH relies on the theory that biotic damage, not resource availability, is the primary factor determining evolved patterns of G and D resource partitioning. The ODH focuses more on the removal of biomass (by herbivory) than the loss of potential G as the primary fitness cost. In the years since the inception of the ODH, researchers have used optimality theory to forge connections between apparency and resource availability, providing a direct connection to G costs and G–D tradeoffs (de Jong, 1995; Yamamura & Tsuji, 1995).

Also, consistent with its emphasis on 'apparency', the ODH covers resource allocation to specific tissues, such as young leaves, flowers and seeds, as a result of their high importance to fitness (e.g. Ohnmeiss & Baldwin, 2000). These perspectives provide valuable insights into patterns of optimal resource allocation, and identify tradeoffs in D allocation across ontogenetic phases and among plant organs. While these patterns have clear relevance to how selection has shaped G–D tradeoffs, we currently lack insights from the cellular scale as to how ontogenetic allocation decisions are implemented. Lacking this context, we do not discuss these aspects further.

2. Quantitative description of the theories

Using quantitative relations, we can derive a more complete picture of the G–D tradeoff under conditions of resource limitation. Here, we focus on the quantitative form of the RAH. The realized growth of a plant (G_r, g d^{-1}) is described as:

$$G_r = \underset{\text{(Term 1)}}{G_m B(1 - kD^{\alpha})} - \underset{\text{(Term 2)}}{(H - mD^{\beta})} \qquad \text{Eqn 1}$$

where G_m is the maximum potential relative growth rate in the absence of herbivory (g g^{-1} d^{-1}), B is the biomass of the plant (g), D (g g^{-1}) is the biomass-equivalent investment in defense, k (g d^{-1}) and α are constants that relate a unit investment in D to a unit reduction in G_r, H(g d^{-1}) is potential herbivore consumption in the absence of D, and m (g d^{-1}) and β are constants that define the shape of the defense-deterrence curve (Coley *et al.*, 1985). In the original RAH, Eqn 1 was presented with currency units of g C. Here, we have changed the units to reflect a currency of g biomass. Term 1 represents the allocation component of the G–D tradeoff and term 2 represents an external cost to both G_r and D, discounted by the quality of the defense system. Term 2 operates without direct constraint from the allocation tradeoff. Term 1 can be simplified with the assumption that $\alpha = 1$, meaning that a tradeoff reduction of $G_m B$ is fully proportional to a tradeoff increase in D (de Jong, 1995). Eqn 1 is also conditioned by the assumption that term 2 is independent of plant size, reflecting a constant, rather than fractional, rate of daily biomass consumption. This assumption has been discussed by de Jong (1995), including suggested modifications to reflect fractional consumption.

From Eqn 1 it is clear that the principal tradeoff in the RAH is the allocation of biomass to G_r vs D. In essence, kD^{α} penalizes the capacity for $G_m B$ to support G_r as a result of the allocation of biomass equivalents to D. The penalty represents a cost without specification of mechanism. However, the basis for the function (i.e. $G_m B$) is clearly a supply-side term. In discussing the RAH, Coley *et al.* (1985) presented three supply-side causes for the evolution of resource-associated G–D tradeoffs – replacement costs (to G_r) for recovery during herbivory, replacement costs (to G_r) for lost biomass, and the relative cost–benefit margins of resource allocation to D vs G_r (defined by kD^{α}).

Within term 1 lies an implicit assumption that the absolute allocation of available resources to G_r and D is limited by the 'zero-sum' constraint – that is, allocation of a currency-normalized unit of resource to produce G_r will result in an equal deficit in allocation to D and vice versa (assuming that $\alpha = 1$; see Fig. 2a). The zero-sum constraint sets a boundary condition equal to the sum of all internal resources that limits the absolute values of G_r or D. The zero-sum constraint is underlain by certain assumptions that, if violated, would change optimal allocation ratios. For example, the theory is based on a binary assumption that resource use is classified as either supporting G_r or D. However, some past treatments have noted the multi-functional nature of certain resource investments (e.g. simultaneously benefiting G_r, D and abiotic stress tolerance; Bazzaz *et al.*, 1987; Kliebenstein, 2016; Monson *et al.*, 2021), which would change the quantitative relations of tradeoff margins. The zero-sum constraint also implies that there is no capacity to marshal new external resources without an equal investment of internal resources. The zero-sum constraint is required to justify the teeter-totter model.

Herms & Mattson (1992) revised the definition of G_m, from the earlier Coley *et al.* (1985) treatment, to separate 'evolutionary' and 'ecological' components. They developed a new term for potential growth rate (G_p). In the GDBH, G_m is reserved for the growth rate determined by long-term evolutionary constraints and is not affected by the challenges of competition, abiotic stress, and herbivory that occur within an organism's lifetime (Fig. 2b). In this context, G_p is bounded by the evolutionary limit of G_m, but is operationally determined by ecological conditions. Differences between G_m in the RAH and G_p in the GDBH will reflect, in part, trait cost–benefit relations shaped by past selection regimes. However, there are other local environmental drivers (e.g. competition and climate) that determine G_p; rendering empirical efforts to discern a G–D tradeoff imprecise.

IV. The coordinated resource allocation hypothesis

Using insights from multi-omic studies that determine G and D allocation patterns (e.g. Coolen *et al.*, 2016; Guo *et al.*, 2018a; Howe *et al.*, 2018; Ballaré & Austin, 2019; Aerts *et al.*, 2021), we propose a new hypothesis to complement the past supply- and demand-side hypotheses, referred to here as the CoRAH. We connect mechanistic insight at the cellular scale to the common tenets of evolutionary optimization that underlie the older hypotheses. A novel result that emerges from the CoRAH is the predicted (and observed) existence of stored resource reserves (established at a cost to both G and D) that provide a safety margin for responses to future biotic and abiotic stresses, while simultaneously balancing the G–D tradeoff. We refer to these stored resources as reserve safety margins (RSMs).

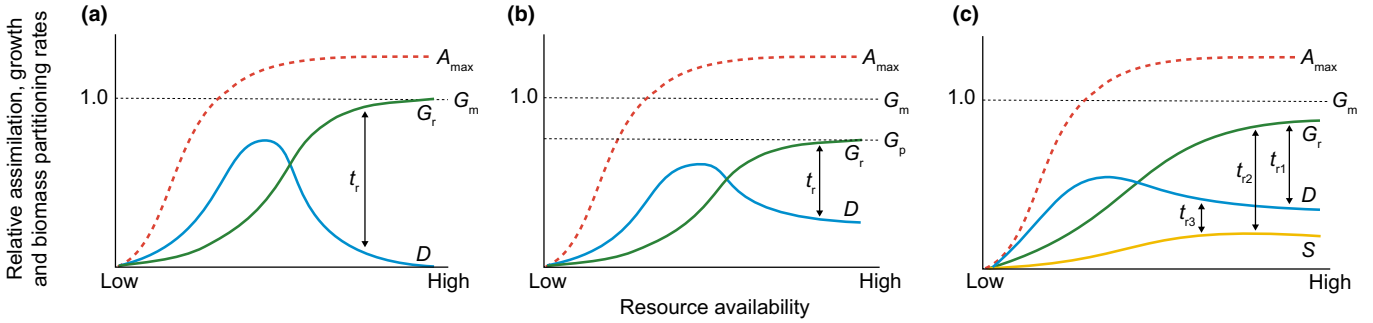

Fig. 2 Patterns of assimilation, growth and biomass allocation among growth (G) and defense (D) processes as a function of internal resource availability. In relating patterns to the resource availability hypothesis (RAH) we make an inherent assumption that $G_m = fA_{max}$, the maximum rate of net CO_2 assimilation. G_m is the maximum inherent relative growth rate in the absence of herbivory. Biomass allocation patterns (G_p, G_r, D and S) are expressed as fractional values relative to G_m (1.0). G_p is the potential growth rate; G_r is the realized growth rate; D are defense metabolites; S is the reserve storage; and t_r is the G–D tradeoff. (a) The RAH (after Herms & Mattson, 1992). (b) The growth-differentiation balance hypothesis (GDBH; after Herms & Mattson, 1992). (c) The coordinated resource allocation hypothesis (CoRAH). Three tradeoffs are presented in (c): the G_r–D tradeoff (t_{r1}), the G_r–S tradeoff (t_{r2}) and the D–S tradeoff (t_{r3}). In developing Eqn 2 in the text, we assume that S is the reserve safety margin (RSM).

Reserve safety margins provide a means for phenotypic resilience, a form of phenotypic plasticity that maintains functional homeostasis in the face of shifting environmental stresses (Tsuda *et al.*, 2009; Katagiri, 2018). Reserve safety margins can be approximated as a type of substrate reserve. Storage reserves are formed using resources that carry a direct fitness cost when diverted away from G and D (Millard, 1988; Chapin *et al.*, 1990). Accumulated reserves are formed when resource supply exceeds demand (e.g. resource overflow), and therefore incur no direct fitness cost. Reserve safety margins are analogous to stored reserves, although we use the term strictly to denote recovery from biotic stress. In theory, RSMs are critical to the adaptive recovery of plants from catastrophic herbivory. Babst *et al.* (2005) showed that jasmonic acid (JA) signaling is involved in the rapid transport of accumulated carbohydrates from leaves to stems and roots in poplar saplings – potentially providing longer-term RSMs at the whole-plant scale, which could be used during crown regrowth following defoliation. Studies using a broader range of species, however, have not consistently supported the use of stored reserves for post-defoliation regrowth (Strauss & Agrawal, 1999; van der Meijden *et al.*, 2000).

Smith & Stitt (2007) discussed numerous studies revealing the existence of regulatory mechanisms (referred to as 'acclimatory responses'), capable of calibrating carbon demand to match supply, and thus sustaining metabolic homeostasis. Although the original presentation of this concept was outside the context of adaptation to herbivory, it clearly established the role of metabolic coordination as a means of balancing carbohydrate supply and demand, and the necessity of sustaining stored reserves to buffer imbalances. Recent studies of JA signaling in *Arabidopsis* supported the acclimatory response hypothesis (Guo *et al.*, 2018b), showing that mutants lacking effective control over allocation to D also failed to sustain short-term carbohydrate reserves. Huang *et al.* (2018) showed that Norway spruce (*Picea abies*) trees grown at the CO_2 compensation point (i.e. no net CO_2 assimilation) downregulated growth and respiration, even to the point of root death as a result of C exhaustion, while sustaining constant amounts of aboveground carbohydrate reserves. Together, these studies reveal a growing recognition that the strategic regulation of resource reserves is a component of coordinated allocation responses and is probably an adaptation to bolster D during periods of extreme stress (Dietze *et al.*, 2014; Huang *et al.*, 2019). Within the framework of CoRAH, the formation of RSMs ensures that proper substrate forms and amounts are available when needed, and that they are depleted to minimal amounts when not. In this way, coordinated resource allocation provides a means of resource optimization and minimizes fitness costs in the face of G–D tradeoffs (see Fig. 1b).

We have developed a tradeoff term for the coordinated allocation to produce RSMs, which pose a direct cost to both G_r and D:

$$G_r = G_m C(1 - kD^{\alpha} - qS^{\gamma}) - (H - mD^{\beta}) \qquad \text{Eqn 2}$$
$$\text{(Term 1)} \qquad\qquad \text{(Term 2)}$$

where the term (qS^{γ}) is the cost–benefit balance of forming G_r-limited reserves. Here, S is the relative mass (g g^{-1}) of storage reserves, and q (g d^{-1}) and γ are constants that relate a unit investment in S to a unit reduction in G_r. Eqn 2 is a G–D tradeoff model that balances resource allocation among three competing functions – D, G_r and S. The relations presented in Eqn 2 broaden the set of allocation options and tradeoffs governed by the zero-sum constraint (Fig. 2c). Empirical tests of Eqn 2 are likely to become available in the near future as multi-omic approaches are applied to species with genetically conserved G–D tradeoffs. The measurement of dynamics in G_r, D and RSMs (as stored reserves) is currently being made routinely, although not always simultaneously, in various *Arabidopsis* lines (Campos *et al.*, 2016; Guo *et al.*, 2018a; Osella *et al.*, 2018; Major *et al.*, 2020; Mengarelli *et al.*, 2021). As these types of studies are directed toward quantitative, comparative tests of the mathematical relations in Eqn 2, we will be able to assess the validity of the zero-sum constraint on the overall equation, as well as the magnitudes of the specific tradeoffs (t_{r1}, t_{r2}, t_{r3}). These types of studies provide the explicit opportunity to falsify CoRAH, an essential feature for testing optimality models (Levins & Lewontin, 1985).

V. Cellular mechanisms of coordinated resource allocation

1. Coordinating G–D tradeoffs: the role of kinase signaling networks

Coordinated resource allocation implies the existence of interactive metabolic systems that register imbalances between resource supply and demand, activate changes in the flux ratios of biochemical pathways and optimize shifts in resource distribution. Primary control over supply-side coordination is applied through a family of kinase enzymes, known as target of rapamycin complexes (TORCs). Target of rapamycin complexes act as resource 'fuel gauges', sensing nutrient and energy availability and adjusting cellular growth to match (Fig. 3a). Although a few different forms of TORC are known, only TORC1 has been reported in plants (Dobrenel *et al.*, 2016). When cells have an abundance of sucrose, signaling through TORC1 enhances activity in anabolic pathways, while repressing catabolic pathways and minimizing intracellular autophagy (Sablowski & Dornelas, 2014). Recent studies in mammalian cells revealed a GTPase protein that binds directly to leucine and, depending on leucine concentrations, modifies TORC1 signaling to enhance (at high leucine) or inhibit (at low leucine) cellular growth (Chen *et al.*, 2021). In studies with *Arabidopsis*, targeted inhibition of TORC1 altered the expression of hundreds of genes, including those associated with signaling networks involving phytohormones (e.g. auxins, gibberellins and brassinosteroids; Dong *et al.*, 2015). Phytohormone networks are connected through crosstalk involving transcription factors (TFs) and protein–protein interactions that largely control cellular and plant growth rates (Fig. 3b). Thus, cellular metabolite status and growth processes in *Arabidopsis* are integrated through TORC1 activity. Together, these networks enable the expression of fast growth and low resource-use efficiency phenotypes at the acquisitive pole of the trait economic spectrum (Fig. 3d).

SNF1-Related Protein Kinase 1 (SnRK1) is a signal antagonist to TORC1. Expression of *SnRK1* genes is activated during periods of reduced resource availability (Wurzinger *et al.*, 2018). Thus, whereas TORC1 modifies cellular processes to increase use of abundant resources, SnRK1 reduces demand for dwindling resources. In the resource-depleted state, SnRK1 will suppress anabolic metabolism, reduce cellular growth rates and enhance intracellular autophagy (Emanuelle *et al.*, 2016; Liao & Bassham, 2020). SnRK1 also controls the distribution of carbon between growth and long-term starch storage (Tiessen *et al.*, 2003), thus regulating the formation of carbohydrate RSMs.

The interactions between TORC1 and SnRK1 are extended to include a direct role in regulating the G–D tradeoff (Margalha *et al.*,

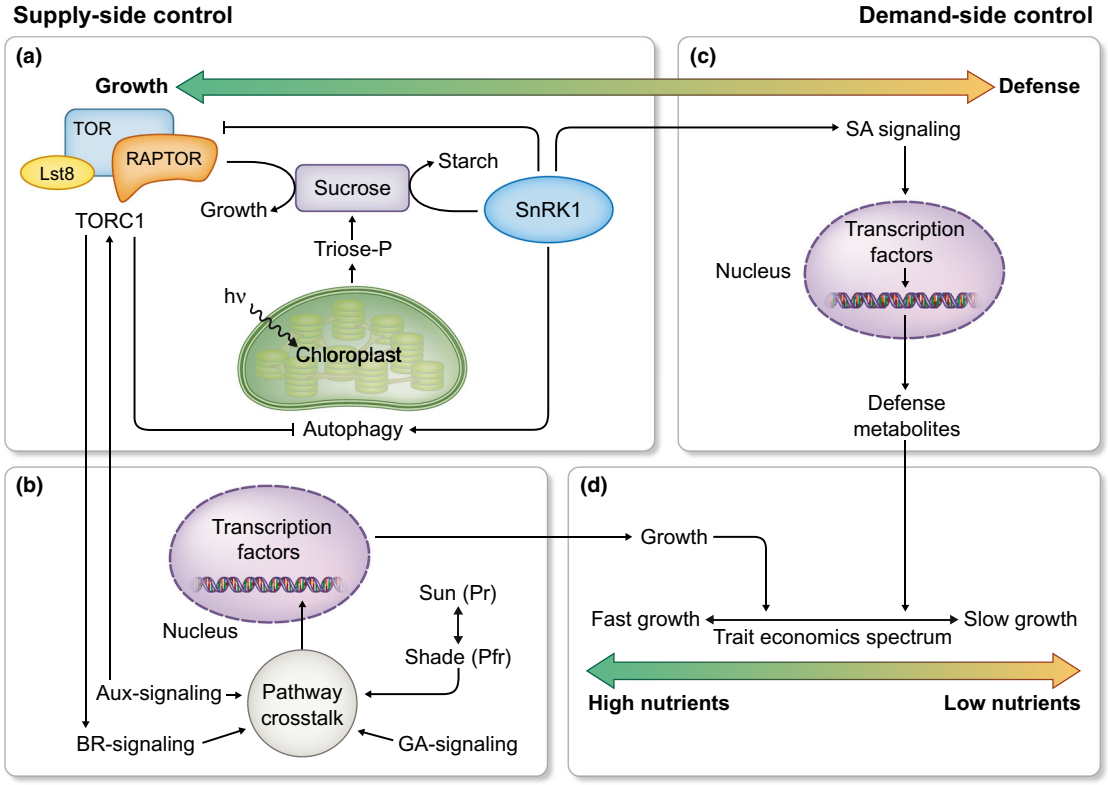

Fig. 3 Cellular processes involved with the regulation of trait expression associated with the growth–defense tradeoff and the fast–slow growth extremes of the trait economics spectrum. Much of the signaling is initiated in the sugar-sensing mechanisms of TORC1 and SnRK1 (a), which interact with phytohormone crosstalk (b, d). Connections between SnRK1 and salicylic acid (SA)-signaling link the availability of photosynthate to nutrient-use and secondary metabolite production (c). Pointed arrows indicate positive effects of one factor on another, whereas blunted arrows indicate negative effects. Aux, auxin; BR, brassinosteroid; GA, gibberellin; Lst8, Lethal with SEC13 protein 8; Pfr, phytochrome (far-red); Pr, phytochrome (red); SnRK1, SNF1 related protein kinase 1; TOR, target of rapamycin complex 1.

2019) (Fig. 3c). For example, rice transgenic lines with overexpressed *TORC1* exhibited enhanced growth rates, downregulated constitutive expression of salicylic acid (SA)-pathway genes, and increased frequencies of pathogenic infections (De Vleesschauwer *et al.*, 2018). Conversely, RNA interference lines with reduced TORC1 signaling exhibited upregulated expression of SA genes, greater resistance to pathogen infection, and reduced G. Increased expression of *SnRK1* in transgenic rice lines resulted in enhanced expression of SA- and JA-associated genes involved in D and increased resistance to pathogenic fungal infection (Filipe *et al.*, 2018). Growth rates in SnRK1-overexpression lines were reduced, including decreased rates of plant biomass accumulation, reduced tiller numbers and reduced leaf expansion rates, consistent with a central role in regulating the G–D tradeoff.

Over the past decade, researchers discovered a unique form of coordinated resource allocation that couples optimal use of nitrogen, a growth-limiting resource in many environments, to the expression of SA-associated D genes, particularly those involved in system acquired resistance (SAR). SAR is the form of whole-plant immunity associated with pathogenic infection. Although SAR is induced by an initial infection, it can persist for weeks to months, and includes priming of distal, uninfected parts of the plant (Conrath, 2006). Heterotic *Arabidopsis* hybrid lines with decreased allocation to SAR defense were deficient in one particularly important SA-responsive TF, called TBF1 (for TL1-binding transcription factor 1) (Groszmann *et al.*, 2015; Gonzalez-Bayon *et al.*, 2019). TBF1 directs the transcription of over 3000 SAR-associated genes (Pajerowska-Mukhtar *et al.*, 2012). While transcriptional activations of *TBF1* genes are under the influence of SA signaling, the amount of nuclear TBF1 protein is determined by translation. Two DNA open reading frames encoding the biosynthesis of multiple aromatic amino acids, particularly phenylalanine, occur immediately upstream of the *TBF1* mRNA translation-initiation codon (Xu *et al.*, 2017). Phenylalanine is the precursor to several defense metabolites produced in the phenylpropanoid pathway, including phenolic glycosides and tannins. Thus, as cellular pools of aromatic amino acids are depleted, replenishment leads to transcription and translation of both the open reading frame genes and *TBF1*. These coupled controls may turn out to represent a novel form of G–D trait integration, coupling low nitrogen resource availability and N-limited growth to constitutive expression of SA-controlled defense genes.

2. Sensory and signaling networks – the coordination of resource allocation and G–D tradeoffs associated with induced D responses

At the cellular scale, the induced response to biotic attack involves effective coordination between detection mechanisms and the upregulation of resource allocation to D traits. At the organism scale, induction provides a means to reduce the fitness costs of defensive resource investments (Karban & Myers, 1989). Induced D is regulated by different signaling pathways depending on the types of tissue injuries and attacking organisms (Glazebrook, 2005; Pieterse *et al.*, 2012). Biotrophic pathogens, which consume living tissue, most frequently induce a response through the SA pathway. Necrotrophic pathogens, such as pathogenic fungi, most frequently induce responses through the coupled JA/ethylene (ET) pathways.

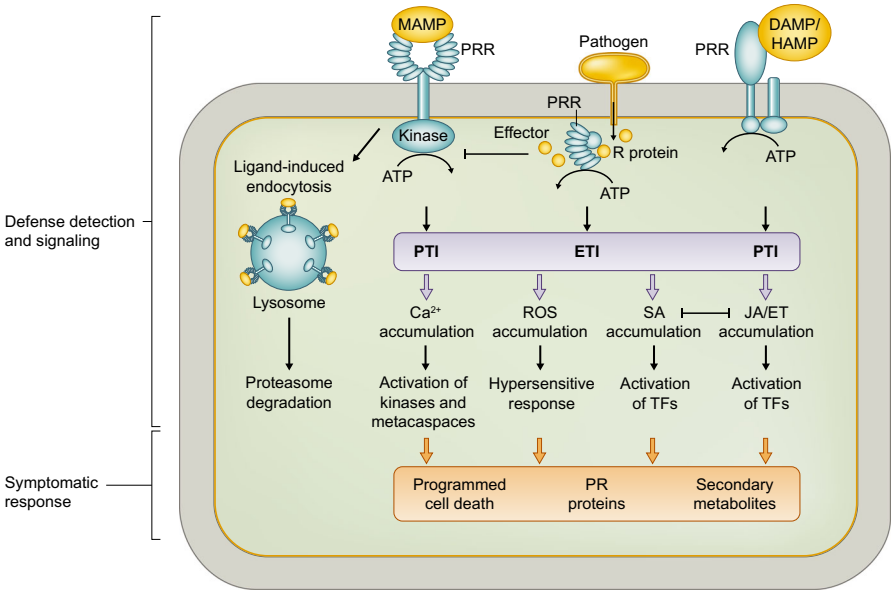

Fig. 4 An overview of the detection processes and symptomatic responses that form the lines of defense involved in biotic stress. Detection is initiated by pattern recognition receptor (PRR) proteins, either as membrane surface complexes or as cytosolic resistance (R) proteins. The first line of post-detection defense involves various signaling pathways associated with pattern-triggered immunity (PTI) and effector-triggered immunity (ETI), which lead to eventual activation of symptomatic defense mechanisms, including programmed cell death, the production of pathogenesis-resistance (PR) proteins, and activation of secondary metabolite synthesis. Pointed arrows indicate positive effects of one factor on another, whereas blunted lines indicate negative effects. PAMP, MAMP, DAMP and HAMP are pathogen-, microbial-, damage- or herbivore-associated molecular pattern, respectively; ET, ethylene; JA, jasmonic acid; ROS, reactive oxygen species; SA, salicylic acid.

Attacks by chewing insects are defended by coordinated interactions among the JA, ET and abscisic acid (ABA) pathways (Erb & Reymond, 2019). In all of these interactions, the response is initiated through binding between a host-cell pattern recognition receptor (PRR) and a conserved ligand that is unique to the attacking organism (Fig. 4). In infections involving biotrophic and necrotrophic pathogens, a microbial surface protein or polysaccharide serves as the complementary ligand, which is known as a pathogen- or microbial-associated molecular pattern (PAMP or MAMP, respectively). During an incipient infection, plant cells can be mechanically damaged, releasing molecular fragments that signal potential risks to neighboring cells. In this case, the detected ligand is known as a damage-associated molecular pattern (DAMP) (Heil & Land, 2014; Vega-Munoz *et al.*, 2020). A fourth type of molecular pattern has been identified as elicitors from feeding herbivores, often referred to as an herbivore-associated molecular pattern (HAMP) (Erb & Reymond, 2019). The most common type of HAMP occurs in the oral secretions of insects (e.g. Steinbrenner *et al.*, 2020).

Once a molecular pattern is recognized, the PRR initiates coordinated signal cascades involving mitogen-activated protein (MAP) kinases, intracellular Ca^{2+} redistribution and reactive oxygen species (ROS) (Vega-Munoz *et al.*, 2020; Sun & Zhang, 2021). The overall response is known as pattern-triggered immunity (PTI), which involves shifts in the expression of thousands of genes (Wang *et al.*, 2006; Birkenbihl *et al.*, 2017), and concomitant rapid (< 1 h) inhibition of further pathogenic colonization (Boller & Felix, 2009) (Fig. 4). The PTI provides a first line of defense, which is often referred to as basal immunity (Jones & Dangl, 2006).

Many host-adapted pathogens have evolved virulence factors, known as effectors, that are secreted into the host cell and resist PTI. As an example, *Pseudomonas syringae*, a biotrophic pathogen that attacks numerous plant species, has evolved a JA analog called coronatine, an effector that selectively alters patterns of JA signaling (Demianski *et al.*, 2012) and represses SA production (Cui *et al.*, 2005). The coronatine-induced rewiring of coordination pathways facilitates colonization and expanded infection. Pathogenic effectors are detected through a second group of cytoplasmic PRRs, also known as resistance (R) proteins. Detection of effectors by R proteins initiates a set of responses known as effector-triggered immunity (ETI) (Fig. 4). PTI and ETI share many similar downstream signaling components (Tsuda *et al.*, 2009; Aerts *et al.*, 2021). ETI signaling includes an acceleration in the hypersensitive response (HR) that produces a fast form of programmed cell death (PCD) at the site of infection (Bruggeman *et al.*, 2015; Radojicic *et al.*, 2018). The ETI responses interact with components of PTI, including the release of ROS and amplification of the HR (Dalio *et al.*, 2021). Necrotic tissue losses during ETI represent an acute cost to G. However, the overall effect is to slow infection during a crucial period of increased secondary metabolite synthesis, which compensates the short-term cost.

Necrotrophic pathogens and herbivores are detected through mechanisms that trigger JA/ET- or JA/ABA-associated signal cascades, while suppressing SA signaling (Pieterse *et al.*, 2012). The prioritization of one pathway over another is a result of differences in PRRs and the manner in which they interact with downstream signaling components (Zhang *et al.*, 2017). It has been commonly assumed that the antagonism between SA and JA signaling is adaptive, either for efficient use of resources or for beneficial tailoring to specific biotic agents, although there are uncertainties underlying these assumptions (Thaler *et al.*, 2012). The accumulation of JA following biotic attack begins in the chloroplast where signal precursors are produced from polyunsaturated fatty acids (Matsui, 2006). Jasmonic acid itself is produced through a series of oxidation reactions in the peroxisome. The control by JA over inducible plant defenses has two branches – one that responds to cues by necrotrophic fungi (the ERF branch) and one that responds to cues from insect herbivores (the MYC branch) (Fig. 5; Pieterse *et al.*, 2012). As part of the JA-dependent detection process, signaling kinases stimulate a burst of volatile ET (Li *et al.*, 2018), which functions along with JA and specific families of TFs to initiate transcription of necrophile-specific D genes. Herbivory leads to increases in tissue ABA, possibly because of local leaf water stress, which interacts with JA signaling to enhance the release of bound TFs that, in turn, initiate expression of herbivore-specific D genes (Vos *et al.*, 2013). Interactions among transcription regulators from the JA, ET and ABA pathways ultimately determine the alternative priorities of gene expression in shaping a defense against necrotrophic microbes or insect herbivores (Broekgaarden *et al.*, 2015; Erb & Reymond, 2019).

3. Crosstalk hubs: the cellular coordination of supply- and demand-side allocation pathways

Much of the known coordination associated with G–D tradeoffs is arbitrated by crosstalk among three regulatory hubs – DELLA, JAZ and phyB (Verhage *et al.*, 2010; Bemer *et al.*, 2017; Karasov *et al.*, 2017; Aerts *et al.*, 2021). Pathway crosstalk occurs through protein–protein interactions and phytohormone signaling that, in turn, influence the activities of TFs (Howe *et al.*, 2018; Fig. 6). All three hubs exert default control over pathway fluxes as negative (suppressive) transcriptional modifiers, which is common in the evolution of coordinated pathway expression (Casal *et al.*, 2004). Key signaling events reverse the default downregulated state of the hubs which, in turn, relax transcriptional suppression and activate targeted patterns of gene expression. The DELLA and phyB networks are especially active in calibrating supply-side control over trait expression concerning the acquisition of available resources (e.g. nutrients, water and light), whereas the JA-associated JAZ hub is specialized for calibrating demand-side induction of D. An integrative understanding of these three hubs provides the best opportunity yet to unify the supply- and demand-side mechanisms underlying the RAH, GDBH and ODH hypotheses.

DELLA proteins act as a constitutive 'brake' on cellular growth that must be deactivated by GA signaling before plants can respond to favorable environmental conditions. The mechanism by which DELLAs repress TF activity remains uncertain, but probably involves TF sequestration (Locascio *et al.*, 2013). DELLA deactivation occurs in the nucleus when GA molecules bind to the soluble receptor, gibberellic acid-insensitive dwarf 1 (GID1) protein, forming a tripartite complex consisting of GA, GID1 and DELLA (Xu *et al.*, 2014; Fig. 6). In *Arabidopsis*, the GA–

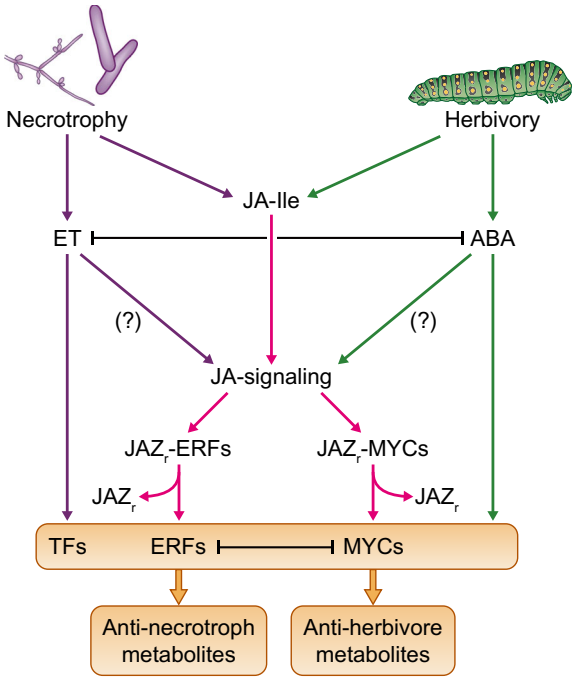

Fig. 5 The two branches of jasmonic acid/ethylene/abscisic acid (JA/ET/ABA)-associated responses to biotic attack by fungal necrotrophs and chewing-insect herbivores (based on Pieterse *et al*., 2012). Both pathways utilize JA signaling as a central control cascade but, through the collaboration of either ET or ABA, different sets of transcription factors (ERF TFs or MYC TFs) control the induction of gene expression. Increases in the active form of JA (JA-isoleucine (JA-Ile)) lead to decoupling between JAZ repressor protein complexes (JAZ_r) and their respective TFs, which enables the formation of DNA-associated transcription complexes and subsequent gene expression. Pointed arrows indicate positive effects of one factor on another, whereas blunted arrows indicate negative effects. Question marks indicate significant uncertainties that remain in our understanding of the interactive mechanisms involving JA, ET and ABA.

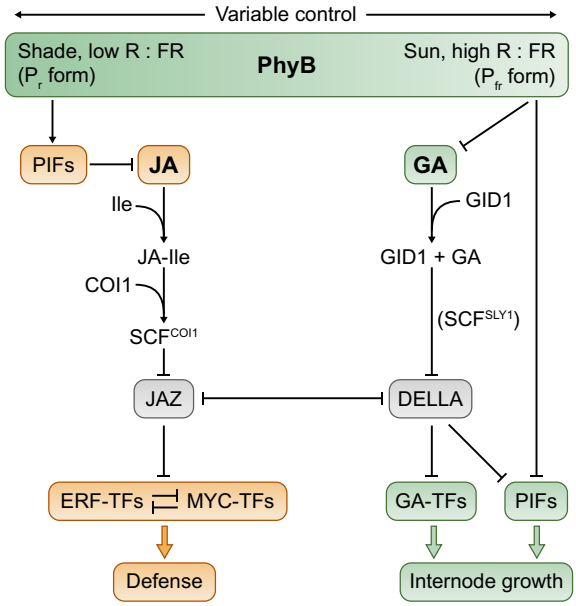

Fig. 6 Crosstalk patterns associated with the phytochrome-gibberellin-jasmonate growth–defense regulon. Phytchrome B (PhyB) shifts between two isoforms (P_{fr} in sunlight and P_r in shade), which interact with phytochrome-interacting transcription factors (PIFs) to activate growth or inactivate jasmonic acid (JA) promotion of defense. Binding between DELLA and JAZ transcription effectors participates in indirect control of the growth–defense tradeoff. Pointed arrows indicate positive effects of one factor on another, whereas blunted arrows indicate negative effects. Based in part on concepts presented in Campos *et al*. (2016). GA, gibberellic acid; R : FR, red : far-red.

GID1–DELLA complex leads to recruitment of the F-box protein complex SCF^{SLY1}, which includes ubiquitin ligase activity. Subsequent ubiquitination of DELLA leads to its degradation, thus freeing bound TFs and activating selective gene transcription.

One group of TFs controlled in part by DELLAs – and that enable crosstalk between the GA and phytochrome B (phyB) pathways – are the phytochrome interacting factors (PIFs; Rockwell *et al*., 2006). Phytochrome interacting factors activate the shade avoidance syndrome (SAS), a suite of traits that includes cellular elongation of shoot internodes, reduced branching and early flowering (Ballaré, 2014; Buti *et al*., 2020). phyB chromoproteins are reversibly toggled between two conformational isomers – the inactive red light (600–700 nm)-absorbing form (P_r) and the active far-red light (700–800 nm)-absorbing form (P_{fr}) (Quail, 2002). In shade-intolerant plants growing in sunny habitats, where the R : FR ratio is high, the P_{fr} (active) form is most abundant. P_{fr} suppresses the expression of SAS traits, in part through signaling that sustains the breakdown of PIFs (Lorrain *et al*., 2008) and in part through interactions with DELLAs (de Lucas *et al*., 2008). The close association of plants during density-dependent competition causes mutual shading and leads to subtle spectral shifts that decrease the local R : FR ratio (Ballaré *et al*., 1990). The spectral shifts, in turn, cause a change in the fraction of P_{fr} to P_r, and an accompanying increase in the stability and concentration of PIFs. Active PIFs, in turn, trigger an increase in GA synthesis, the breakdown of DELLAs, release of GA-associated TFs, and increased expression of SAS-associated genes. phyB-associated changes in G are opposed by responses in D (Ballaré, 2014; Campos *et al*., 2016). This form of the G–D tradeoff is not driven by resource competition, but rather is the result of coordinated regulation at the cellular scale (Campos *et al*., 2016; Ballaré & Austin, 2019; Fernández-Milmanda *et al*., 2020).

Similar to DELLAs, jasmonate ZIM (JAZ) proteins act as constitutive repressors that must be deactivated to enable JA-associated gene expression (Howe *et al*., 2018). JAZ proteins bind specific TFs, especially in the MYC family, and thus prevent them from promoting transcription. Following its production in the peroxisome, JA is converted to an active signal in the cytosol when it combines with isoleucine (Ile), forming jasmonoyl-L-isoleucine (JA-Ile). The receptor for JA-Ile is the nuclear protein, COI1, which associates with a larger complex known as Skp1-Cul1-F-box protein (SCF; Sheard *et al*., 2010). The binding of JA-Ile to the COI1/SCF complex (SCF^{COI}) induces an increased affinity towards JAZ proteins, which are ubiquitinated as a means to tag them for degradation. The degradation of JAZ proteins frees their bound TFs and enables JA-associated transcription.

Crosstalk between DELLAs and JAZs provides an indirect means of controlling the G–D tradeoff (Yang *et al*., 2012; Campos *et al*., 2016; Fig. 6). DELLAs bind to JAZs in a manner that

excludes their mutual interactions with TFs (Hou *et al*., 2010). Thus, in the presence of induced JA signaling and concomitant degradation of JAZ, the fraction of free DELLAs is increased, which in turn increases the likelihood of binding and suppressing GA-associated TFs. This interaction enforces a cellular G–D tradeoff – that is, increased expression of JA-associated defense causes decreased expression of GA-associated growth. Conversely, JAZs are freed as GA-dependent growth is stimulated, such as following the upregulated expression of genes associated with SAS traits, when DELLAs are degraded. This increases the capacity for sequestration of MYC TFs and decreases the expression of JA-dependent defense genes.

There is experimental evidence that the interactive DELLA, JAZ and PIF regulon provides plants with a well-coordinated capacity to calibrate resource distributions between G and D. As an example, Navarro *et al*. (2008) showed that a quadruple DELLA mutant, deficient in four of the five DELLAs in *Arabidopsis*, exhibited reduced expression of genes from the JA/ET-associated regulon, but increased expression of genes from the SA-associated regulon, compared with wild-type (WT) plants. As a result, mutant plants were more susceptible to infection by necrotrophic fungi, but less susceptible to infection by biotrophic bacteria. By contrast, plants with a GA-insensitive mutation, which stabilizes DELLAs, exhibited 100-fold increases in bacterial infection, but were more resistant to necrotrophic fungi. Recently, it was shown that interactions among the phytohormone signals that control the G–D tradeoff (GA-, JA-, and SA-dependent pathways) and responses to shade/sun availability (involving PIFs) are modified by the availability of MYC TFs that, in turn, respond to the composition of root bacterial community composition (Hou *et al*., 2021). Thus, the crosstalk among JAZs, DELLAs and PIFs, mitigated by MYC TFs, are likely to extend to whole-plant, root-to-shoot communication networks controlled by light and nutrient state changes.

VI. Selection has shaped both the coordination process and the reserve safety margins that lead to phenotypic resilience

Given the evolution of coordination systems as the mechanistic basis for observed G–D tradeoffs, a question arises as to whether selection has favored coordination itself, the RSMs that result from coordination, or an alternative. Recent studies of *Arabidopsis* mutants and genetic transformation lines have shown that the targets of selection in determining G–D tradeoffs include both the direct influences of coordination mechanisms and the indirect influences of RSMs (Table 2). Two related studies (Campos *et al*., 2016; Major *et al*., 2017) focused on a quintuple mutant plant (*jazQ*) deficient in five (out of 13) JAZ repressors. Recall that JAZ proteins function as transcription restraints on JA signaling, and thus prevent overexpression of D-associated genes. With several JAZ isoforms removed, leaves of *jazQ* exhibited a clear G–D tradeoff, showing higher constitutive allocations to D, greater resistance to herbivory and reduced G, compared with WT plants (Campos *et al*., 2016). When a mutation in *PHYB* was added to the *jazQ* mutant (producing *jazQ phyB*), the previously observed G restraints were relieved, without loss of increased D. The results confirm the role of regulatory interactions among JAZ and phyB in enforcing the allocational priorities to G and D – *ipso facto*, removal of the coordinated crosstalk of these components also removed the tradeoff. However, the results also contained a novel observation – that it is possible to uncouple the G–D tradeoff through modifications to the coordination systems. This observation provides promise that future crop species could be designed with

Table 2 Summary of growth–defense tradeoffs, resistance to herbivores/pathogens, and fitness metrics for jasmonic acid (JA) pathway *JAZ* and *FITNESS* gene mutants in *Arabidopsis thaliana*.

Study	Mutation	Growth	Defense	Herbivore effect/fitness metrics
Campos *et al*. (2016) Major *et al*. (2017)	*jaz* quintuple (5) (*jazQ*)	↓ Leaf area ↓ Root growth ↓ Petiole length ↓ Plant dry weight ↑ Flowering time	↑ Glucosinolates ↑ Anthocyanins ↑ Triterpenoid mRNA ↑ Phenylpropanoid mRNA	↓ Cabbage looper larval weight
Guo *et al*. (2018b)	*jaz* decuple (10) *jaz* undecuple (11) (*jazD*; *jazU*)	↓ Root growth ↓ Petiole length ↓ Leaf area ↓ Plant relative growth rate ↑ Flowering time	↑ Glucosinolates ↑ Anthocyanins ↑ ERF TFs	↓ Cabbage looper herbivory ↓ *Botrytis* fungal infection ↑ C starvation gene mRNA ↓ Seed mass
Major *et al*. (2020)	*jaz* decuple (10) (*jazD*)	↓ Root growth ↓ Leaf area ↓ Plant relative growth rate ↑ Flowering time	↑ Glucosinolates ↑ Anthocyanins ↑ Expression of defense genes (*VSP2*, *PDF1.2a*, and *Thi2.1*)	↓ Cabbage looper herbivory ↓ *Botrytis* fungal infection
Osella *et al*. (2018)	*fitness-1* single (1) *fitness-2* single (1)	No change in plant size	↑ Salicylic acid ↑ *PR1* mRNA	↓ *Pseudomonas syringae* infection ↑ Seed mass/plant
Mengarelli *et al*. (2021)	$FITNESS_{ox1}$	↓ Plant size	↓ Salicylic acid ↑ *jaz3* and *jaz10* mRNA	↑ *P. syringae* infection ↓ Seed mass/plant

Italicized lower-case gene designations indicate lack-of-function mutations (e.g. *jaz* and *fitness*), whereas the upper-case italicized gene designations refer to an overexpression gene construct (i.e. $FITNESS_{OX}$). ERF, ETS2 repressor factor (transcription factor family); TF, transcription factor.

enhanced growth and defense, and in a more basic context, it demonstrates that it is indeed the mechanisms of coordinated resource allocation that underlie effective enforcement of the G–D tradeoff in WT plants.

Guo *et al.* (2018a) analyzed G, D and fitness (seed yield) in a decuple (10 gene) JAZ mutant (*jazD*). With the loss of even more JAZ isoforms, *jazD* allocated more resources to D than to *jazQ*. Furthermore, *jazD* exhibited greater reductions in G and, in this case, reduced reproductive fitness. Perturbations to plant carbon balance were also observed, as leaf respiration rates increased, short-term starch and sucrose reserves were depleted, and the expression of marker genes associated with severe carbon limitation was increased. There was evidence that expression of at least one of the remaining three unmutated *JAZ* genes, *JAZ8*, was also increased (15-fold), demonstrating a remarkable capacity for expression redundancy in the JAZ family. When a *JAZ8* mutant gene was introduced to *jazD* plants, producing a homozygous undecuple (11 mutation) line (*jazU*), G was even further stunted and seed yield dropped to near zero. These studies revealed a crucial role for JAZ proteins in coordinating G–D tradeoffs, including maintenance of carbon reserves, sustained resiliency through gene redundancy and, ultimately, promotion of fitness.

Recent studies by Osella *et al.* (2018) and Mengarelli *et al.* (2021) have identified a gene, *FITNESS*, which exerts an even higher degree of prioritized control over the JAZ repressor system. Loss-of-function mutants (*fitness* 1 and 2) exhibited no change in G compared with WT lines, but with increased constitutive concentrations of SA, increased resistance to *P. syringae* infection, and increased seed mass per plant. The results demonstrated interactions between the FITNESS protein and JA signaling, but in a way that produced a positive covariance between G and D, not the negative association more typically shown for G–D traits.

Through all of these studies it is clear that there is significant potential for selection to favor differential trait expression within the G–D balance through adjustment in cellular coordination, even to the point of completely reversing the tradeoff. The mechanistic foundation for how selection could shape the G–D tradeoff is now well established, at least for the case of *Arabidopsis* lines. Now, the question arises as to whether selection has indeed worked through this process in natural populations and among a broader set of species.

VII. Conclusions and future directions

Recently reported multi-omic evidence for differential patterns of gene expression, combined with studies of cellular signaling networks and mutation analysis, have allowed researchers to describe the cellular mechanisms underlying G–D tradeoffs. Such perspectives provide process-based explanations for the theoretical framework that emerged from principles of economic optimization applied to the organismic scale over many decades, as well as a framework for unification of the supply- and demand-side processes of existing G–D tradeoff hypotheses. At the cellular scale, G–D tradeoffs are not the result of passive allocation responses to changing resource dynamics, but rather the product of active regulation through coordinated cascades of molecular signals and cross-linked patterns of pathway communication. Cellular coordination provides a means for phenotypic plasticity and appears to have been the target of selection for genetically determined G–D tradeoffs. While passive competitive resource allocation may have produced the metabolic stresses that impacted fitness in ancestral phenotypes, subsequent selection appears to have favored a strategic, coordinated form of allocation. The processes of cellular coordination uncovered in the recent multi-omic studies explain a robust form of physiological resilience to biotic stresses. This is the basis for our proposed framework within CoRAH.

There are clear caveats that accompany CoRAH, and that should be addressed in future work. In previous ecological studies, researchers have focused on taxonomic and habitat diversity as a means to reveal specific forms of the G–D tradeoff. The cellular processes of CoRAH, by contrast, have been revealed through detailed molecular studies of only a few model plant species. In order to truly bridge the gap between the ecological and cellular research communities, there is a need to establish context for CoRAH, both across species and ecological resource gradients. There are also questions about the degree to which patterns of coordinated resource allocation are fixed according to genotype. Aspen (*Populus tremuloides*) trees exhibit heritable G–D tradeoffs (Osier & Lindroth, 2006; Cope *et al.*, 2019), as well as conservation of diverse G–D genotypes within populations. The conserved G–D tradeoffs vary depending on resource availability (Osier & Lindroth, 2006) and the consequences of those tradeoffs in different environments can alter the genetic structure of aspen populations (Cope *et al.*, 2021). There are important genotype × environment interactions that have been studied at the organismic scale using traditional ecological and genetic approaches, and that need to be reconciled with higher-level controls over the expression of cellular coordination traits. This is especially true in determining the heritability of coordination mechanisms, including inherent mechanisms that connect phenotypic plasticity at the cellular and organismic scales, and their relationships to variability in G–D trait expression across gradients in resource availability, climate and biotic stress (e.g. competition and herbivory).

Even with these uncertainties, however, it is worth noting that the cellular mechanisms determining G–D tradeoffs are deeply represented among land-plant clades. Components of the JA- and SA-signaling pathways, with roles in coordination of biotic stress responses, are found in some of the most ancient plant lineages (Wang *et al.*, 2015; Monte *et al.*, 2019; Peñuelas *et al.*, 2019). Some coordination components, such as the TORC-SnRK signaling system, function in similar regulatory roles in all eukaryotes examined to date (van Dam *et al.*, 2011; Roustan *et al.*, 2016; Jamsheer *et al.*, 2019). Thus, while there are some gaps in our knowledge of the ecological context for coordinated resource allocation, we have a strong empirical basis to assume phylogenetic uniformity in the general form of coordination mechanisms.

Reaching beyond the cellular and organismic scales, an understanding of coordinated resource allocation could inform us on how to extend process-based connections from genes to ecosystems (Whitham *et al.*, 2006). In many ecosystems one or a few

'foundation species', with unique patterns of genetically determined trait associations, exert the dominant controls over system-level dynamics (Schweitzer *et al.*, 2008). Some foundation species (e.g. *Populus trichoma*, *Pinus taeda* and *P. abies*) also have fully sequenced genomes, enabling deeper multi-omic studies of molecular processes. One trait relevant to G–D tradeoffs, which could also be especially important to gene-to-ecosystem scaling, involves condensed tannin production in many tree species (Whitham *et al.*, 2006). The transfer of tannins to soils during leaf decomposition has been shown to have important ramifications for processes such as soil organic matter oxidation rates (Hättenschwiler *et al.*, 2011). Cellular coordination of constitutive tannin production within the integrated context of resource availability (e.g. Fig. 3), and concomitant indirect control over soil C and N mineralization rates, could provide the conceptual thread required to connect gene expression processes to ecosystem nutrient cycling.

In this synthesis, we have attempted to merge recent multi-omic discoveries with theories of evolutionary optimization as a means to better understand G–D allocational tradeoffs. A core principle underlying our synthesis is that past selection has favored a process of cellular coordination to enable effective responses to biotic attack, while retaining strategic resource reserves that provide a safety margin against future attacks. The CoRAH, using multi-omic data, provides the mechanistic underpinnings of optimization in G–D trait expression, and thus builds on previous hypotheses concerning trait tradeoffs. In essence, the tools of multi-omic discovery provide us with a biological 'Rosetta stone', capable of translating patterns in the gene-to-phenotype languages of trait associations, modes of selection and resource allocation. In the next few decades, as we progress in our understanding of multi-omic translation, we are likely to witness even further integration of theory and mechanism within the fields of cellular biology and ecology. This will forge a new form of mechanistic ecology with novel opportunities to develop closer ties to cell biology, genetics and biochemistry than have been possible in the past.

Acknowledgements

The authors acknowledge decades of past assistance and collaboration from our respective research groups and consistent support from the US National Science Foundation, the US Department of Agriculture and the US Department of Energy. AMT acknowledges support specifically from NSF-IOS grant 1755346, and MTL acknowledges support specifically from NSF-ATM 1837891. We are grateful for comments and a review by Professor Paul Stoy (University of Wisconsin-Madison). We acknowledge the insightful comments made by three anonymous reviewers, which greatly improved the original draft of the paper.

Author contributions

The concept for the paper was initially developed by RKM, with subsequent input from all authors. All authors contributed to writing the manuscript.

ORCID

Manuel T. Lerdau https://orcid.org/0000-0003-1864-0834
Richard L. Lindroth https://orcid.org/0000-0003-4587-7255
Russell K. Monson https://orcid.org/0000-0002-7671-4371
Amy M. Trowbridge https://orcid.org/0000-0001-8993-2530

Data availability

Original data were not generated for this synthetic analysis.

References

Aerts N, Mendes MP, van Wees SCM. 2021. Multiple levels of crosstalk in hormone networks regulating plant defense. *The Plant Journal* **105**: 489–504.

Agrawal AA. 2020. A scale-dependent framework for trade-offs, syndromes, and specialization in organismal biology. *Ecology* **101**: e02924.

Babst BA, Ferrieri RA, Gray DW, Lerdau M, Schlyer DJ, Schueller M, Thorpe MR, Orians CM. 2005. Jasmonic acid induces rapid changes in carbon transport and partitioning in *Populus*. *New Phytologist* **167**: 63–72.

Ballaré CL. 2014. Light regulation of plant defense. *Annual Review of Plant Biology* **65**: 335–363.

Ballaré CL, Austin AT. 2019. Recalculating growth and defense strategies under competition: key roles of photoreceptors and jasmonates. *Journal of Experimental Botany* **70**: 3425–3434.

Ballaré CL, Scopel AL, Sánchez RA. 1990. Far-red radiation reflected from adjacent leaves – an early signal of competition in plant canopies. *Science* **247**: 329–332.

Bazzaz FA, Chiariello NR, Coley PD, Pitelka LF. 1987. Allocating resources to reproduction and defense. *BioScience* **37**: 58–67.

Bemer M, van Dijk ADJ, Immink RGH, Angenent GC. 2017. Cross-family transcription factor interactions: an additional layer of gene regulation. *Trends in Plant Science* **22**: 66–80.

Bergelson J, Purrington CB. 1996. Surveying patterns in the cost of resistance in plants. *American Naturalist* **148**: 536558.

Birkenbihl RP, Liu S, Somssich IE. 2017. Transcriptional events defining plant immune responses. *Current Opinion in Plant Biology* **38**: 1–9.

Bloom AJ, Chapin FS, Mooney HA. 1985. Resource limitation in plants—an economic analogy. *Annual Review of Ecology and Systematics* **16**: 363–392.

Boege K, Marquis RJ. 2005. Facing herbivory as you grow up: the ontogeny of resistance in plants. *Trends in Ecology and Evolution* **20**: 441–448.

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.

Broekgaarden C, Caarls L, Vos IA, Pieterse CMJ, Van Wees SCM. 2015. Ethylene: traffic controller on hormonal crossroads to defense. *Plant Physiology* **169**: 2371–2379.

Bruggeman Q, Raynaud C, Benhamed M, Delarue M. 2015. To die or not to die? Lessons from lesion mimic mutants. *Frontiers in Plant Science* **6**: 24.

Bryant JP, Chapin FS III, Klein DR. 1983. Carbon nutrient balance of boreal plants in relation to vertebrate herbivory. *Oikos* **40**: 357–368.

Buti S, Pantazopoulou CK, van Gelderen K, Hoogers V, Reinen E, Pierik R. 2020. A gas-and-brake mechanism of bHLH proteins modulates shade avoidance. *Plant Physiology* **184**: 2137–2153.

Campbell SA, Halitschke R, Thaler JS, Kessler A. 2014. Plant mating systems affect adaptive plasticity in response to herbivory. *The Plant Journal* **78**: 481–490.

Campos ML, Yoshida Y, Major IT, Ferreira DD, Weraduwage SM, Froehlich JE, Johnson BF, Kramer DM, Jander G, Sharkey TD *et al.* 2016. Rewiring of jasmonate and phytochrome B signalling uncouples plant growth-defense tradeoffs. *Nature Communication* **7**: 12570.

Casal JJ, Fankhauser C, Coupland G, Blázquez MA. 2004. Signalling for developmental plasticity. *Trends in Plant Science* **9**: 309–314.

Chapin FS III. 1980. The mineral nutrition of wild plants. *Annual Review of Ecology and Systematics* **11**: 233–260.

Chapin FS, Schulze E-D, Mooney HA. 1990. The ecology and economics of storage in plants. *Annual Review of Ecology and Systematics* **21**: 423–447.

Chen J, Ou Y, Luo R, Wang J, Wang D, Guan JL, Li Y, Xia PX, Chen PR, Liu Y. 2021. SAR1B senses leucine levels to regulate mTORC1 signalling. *Nature* **596**: 281–284.

Cheverud JM. 1988. A comparison of genetic and phenotypic correlations. *Evolution* **42**: 958–968.

Cohen D. 1971. Maximizing final yield when growth is limited by time or by limiting resources. *Journal of Theoretical Biology* **33**: 299–307.

Coley PD, Bryant JP, Chapin FS III. 1985. Resource availability and plant antiherbivore defense. *Science* **230**: 895–899.

Conrath U. 2006. Systemic acquired resistance. *Plant Signaling and Behavior* **1**: 179–184.

Coolen S, Proietti S, Hickman R, Davila Olivas NH, Huang P-P, Van Verk MC, Van Pelt JA, Wittenberg AHJ, De Vos M, Prins M *et al.* 2016. Transcriptome dynamics of *Arabidopsis* during sequential biotic and abiotic stresses. *The Plant Journal* **86**: 249–267.

Cope OL, Keefover-Ring K, Kruger EL, Lindroth RL. 2021. Growth-defense trade-offs shape population genetic composition in an iconic forest tree species. *Proceedings of the National Academy of Sciences, USA* **118**: e2103162118.

Cope OL, Kruger EL, Rubert-Nason KF, Lindroth RL. 2019. Chemical defense over decadal scales: ontogenetic allocation trajectories and consequences for fitness in a foundation tree species. *Functional Ecology* **33**: 2105–2115.

Coruzzi G, Bush DR. 2001. Nitrogen and carbon nutrient and metabolite signaling in plants. *Plant Physiology* **125**: 61–64.

Cui J, Bahrami AK, Pringle EG, Hernandez-Guzman G, Bender CL, Pierce NE, Ausubel FM. 2005. *Pseudomonas syringae* manipulates systemic plant defenses against pathogens and herbivores. *Proceedings of the National Academy of Sciences, USA* **102**: 1791–1796.

Dalio RJD, Paschoal D, Arena GD, Magalhaes DM, Oliveira TS, Merfa MV, Maximo HJ, Machado MA. 2021. Hypersensitive response: from NLR pathogen recognition to cell death response. *Annals of Applied Biology* **178**: 268–280.

van Dam TJP, Zwartkruis FJT, Bos JL, Snel B. 2011. Evolution of the TOR pathway. *Journal of Molecular Evolution* **73**: 209–220.

De Vleesschauwer D, Filipe O, Hoffman G, Seifi HS, Haeck A, Canlas P, Van Bockhaven J, De Waele E, Demeestere K, Ronald P *et al.* 2018. Target of rapamycin signaling orchestrates growth-defense trade-offs in plants. *New Phytologist* **217**: 305–319.

Deans RM, Brodribb TJ, Busch FA, Farquhar GD. 2020. Optimization can provide the fundamental link between leaf photosynthesis, gas exchange and water relations. *Nature Plants* **6**: 1116–1125.

Demianski AJ, Chung KM, Kunkel BN. 2012. Analysis of Arabidopsis JAZ gene expression during *Pseudomonas syringae* pathogenesis. *Molecular Plant Pathology* **13**: 46–57.

Dietze MC, Sala A, Carbone MS, Czimczik CI, Mantooth JA, Richardson AD, Vargas R. 2014. Nonstructural carbon in woody plants. *Annual Review of Plant Biology* **65**: 667–687.

Dobrenel T, Caldana C, Hanson J, Robaglia C, Vincentz M, Veit B, Meyer C. 2016. TOR signaling and nutrient sensing. *Annual Review of Plant Biology* **67**: 261–285.

Dong P, Xiong FJ, Que YM, Wang K, Yu LH, Li ZG, Ren MZ. 2015. Expression profiling and functional analysis reveals that TOR is a key player in regulating photosynthesis and phytohormone signaling pathways in *Arabidopsis*. *Frontiers in Plant Science* **6**: 677.

Emanuelle S, Doblin MS, Stapleton DI, Bacic A, Gooley PR. 2016. Molecular insights into the enigmatic metabolic regulator, SnRK1. *Trends in Plant Science* **21**: 341–353.

Erb M, Reymond P. 2019. Molecular interactions between plants and insect herbivores. *Annual Review of Plant Biology* **70**: 527–557.

Feeny P. 1976. Plant apparency and chemical defense. In: Wallace JW, Mansell RL, eds. *Biochemical interaction between plants and insects. Recent advances in phytochemistry, vol. 10*. Boston, MA, USA: Springer, 1–40.

Fernández-Milmanda GL, Crocco CD, Reichelt M, Mazza CA, Köllner TG, Zhang T, Cargnel MD, Lichy MZ, Fiorucci A-S, Fankhauser C *et al.* 2020. A light-dependent molecular link between competition cues and defence responses in plants. *Nature Plants* **6**: 223–230.

Filipe O, De Vleesschauwer D, Haeck A, Demeestere K, Hofte M. 2018. The energy sensor OsSnRK1a confers broad-spectrum disease resistance in rice. *Scientific Reports* **8**: 3864.

Futuyma DJ, Moreno G. 1988. The evolution of ecological specialization. *Annual Review of Ecology and Systematics* **19**: 207–234.

Glazebrook J. 2005. Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. *Annual Review of Phytopathology* **43**: 205–227.

Gonzalez-Bayon R, Shen Y, Groszmann M, Zhu A, Wang A, Allu AD, Dennis ES, Peacock WJ, Greaves IK. 2019. Senescence and defense pathways contribute to heterosis. *Plant Physiology* **180**: 240–252.

Groszmann M, Gonzalez-Bayon R, Lyons RL, Greaves IK, Kazan K, Peacock WJ, Dennis ES. 2015. Hormone-regulated defense and stress response networks contribute to heterosis in *Arabidopsis* F1 hybrids. *Proceedings of the National Academy of Sciences, USA* **112**: 6397–6406.

Guo Q, Major IT, Howe GA. 2018a. Resolution of growth–defense conflict: mechanistic insights from jasmonate signaling. *Current Opinion Plant Biology* **44**: 72–81.

Guo Q, Yoshida Y, Major IT, Wang K, Sugimoto K, Kapali G, Havko NE, Benning C, Howe GA. 2018b. JAZ repressors of metabolic defense promote growth and reproductive fitness in *Arabidopsis*. *Proceedings of the National Academy of Sciences, USA* **115**: 10768–10777.

Hahn PG, Maron JL. 2016. A framework for predicting intraspecific variation in plant defense. *Trends in Ecology and Evolution* **31**: 646–656.

Hamilton JG, Zangerl AR, DeLucia EH, Berenbaum MR. 2001. The carbon-nutrient balance hypothesis: its rise and fall. *Ecology Letters* **4**: 86–95.

Harper JL. 1967. A Darwinian approach to plant ecology. *Journal of Ecology* **55**: 247–270.

Hättenschwiler S, Coq S, Barantal S, Handa IT. 2011. Leaf traits and decomposition in tropical rainforests: revisiting some commonly held views and towards a new hypothesis. *New Phytologist* **189**: 950–965.

Heil M, Land WG. 2014. Danger signals – damaged-self recognition across the tree of life. *Frontiers in Plant Science* **5**: 578.

Herms DA, Mattson WJ. 1992. The dilemma of plants—to grow or defend. *Quarterly Review of Biology* **67**: 283–335.

Hou S, Thiergart T, Vannier N, Mesny F, Ziegler J, Pickel B, Hacquard S. 2021. A microbiota–root–shoot circuit favours *Arabidopsis* growth over defence under suboptimal light. *Nature Plants* **7**: 1078–1092.

Hou XL, Lee LYC, Xia KF, Yen YY, Yu H. 2010. DELLAs modulate jasmonate signaling via competitive binding to JAZs. *Developmental Cell* **19**: 884–894.

Howe GA, Major IT, Koo AJ. 2018. Modularity in jasmonate signaling for multistress resilience. *Annual Review of Plant Biology* **69**: 387–415.

Huang J, Hammerbacher A, Weinhold A, Reichelt M, Gleixner G, Behrendt T, van Dam NM, Sala A, Gershenzon J, Trumbore S *et al.* 2018. Eyes on the future – evidence for trade-offs between growth, storage and defense in Norway spruce. *New Phytologist* **222**: 144–158.

Huang J, Kautz M, Trowbridge AM, Hammerbacher A, Raffa KF, Adams HD, Goodsman DW, Xu C, Meddens AJH, Kandasamy D *et al.* 2019. Tree defence and bark beetles in a drying world: carbon partitioning, functioning and modelling. *New Phytologist* **225**: 26–36.

Huot B, Yao J, Montgomery BL, He SY. 2014. Growth–defense trade-offs in plants: a balancing act to optimize fitness. *Molecular Plant* **7**: 1267–1287.

Jamsheer KM, Jindal S, Laxmi A. 2019. Evolution of TOR-SNRK1 dynamics in green plants and its integration with phytohrmone signaling networks. *Journal of Experimental Botany* **70**: 2239–2259.

Jones JDG, Dangl JL. 2006. The plant immune system. *Nature* **444**: 323–329.

de Jong TJ. 1995. Why fast-growing plants do not bother about defense. *Oikos* **74**: 545–548.

Karasov TL, Chae E, Herman JJ, Bergelson J. 2017. Mechanisms to mitigate the trade-off between growth and defense. *Plant Cell* **29**: 666–680.

Karban R, Myers JH. 1989. Induced plant responses to herbivory. *Annual Review of Ecology and Systematics* **20**: 331–348.

Katagiri F. 2018. Plant immune signaling from a network perspective. *Plant Science* **276**: 14–21.

Kessler A, Baldwin IT. 2002. Plant responses to insect herbivory: the emerging molecular analysis. *Annual Review of Plant Biology* **53**: 299–328.

Kliebenstein DJ. 2016. False idolatry of the mythical growth versus immunity trade-off in molecular systems plant pathology. *Physiological and Molecular Plant Pathology* **95**: 55–59.

Koricheva J. 2002a. Meta-analysis of sources of variation in fitness costs of plant antiherbivore defenses. *Ecology* **83**: 176–190.

Koricheva J. 2002b. The carbon-nutrient balance hypothesis is dead; long live the carbon-nutrient balance hypothesis? *Oikos* **98**: 537–539.

Kruger EL, Keefover-Ring K, Holeski LM, Lindroth RL. 2020. To compete or defend: linking functional trait variation with life-history tradeoffs in a foundation tree species. *Oecologia* **192**: 893–907.

Lerdau M. 1992. Future discounts and resource allocation in plants. *Functional Ecology* **6**: 371–375.

Lerdau M, Coley PD. 2002. Benefits of the carbon-nutrient balance hypothesis. *Oikos* **98**: 534–536.

Lerdau M, Litvak M, Monson R. 1994. Plant-chemical defense – monoterpenes and the growth-differentiation balance hypothesis. *Trends in Ecology and Evolution* **9**: 58–61.

Levins R, Lewontin R. 1985. *Chapter 2: Adaptation. The dialectal biologist.* Cambridge, UK: Harvard University Press, 303 pp.

Li S, Han XF, Yang LY, Deng XX, Wu HJ, Zhang MM, Liu YD, Zhang SQ, Xu J. 2018. Mitogen-activated protein kinases and calcium-dependent protein kinases are involved in wounding-induced ethylene biosynthesis in *Arabidopsis thaliana*. *Plant, Cell & Environment* **41**: 134–147.

Liao CY, Bassham DC. 2020. Combating stress: the interplay between hormone signaling and autophagy in plants. *Journal of Experimental Botany* **71**: 1723–1733.

Locascio A, Blázquez MA, Alabadí D. 2013. Genomic analysis of DELLA protein activity. *Plant and Cell Physiology* **54**: 1229–1237.

Loomis WE. 1932. Growth-differentiation balance vs. carbohydrate-nitrogen ratio. *Proceedings of the American Society of Horticultural Science* **29**: 240–245.

Lorrain S, Allen T, Duek PD, Whitelam GC, Fankhauser C. 2008. Phytochrome-mediated inhibition of shade avoidance involves degradation of growth-promoting bHLH transcription factors. *The Plant Journal* **53**: 312–323.

de Lucas M, Daviere JM, Rodriguez-Falcon M, Pontin M, Iglesias-Pedraz JM, Lorrain S, Fankhauser C, Blazquez MA, Titarenko E, Prat S. 2008. A molecular framework for light and gibberellin control of cell elongation. *Nature* **451**: 480–484.

Major IT, Guo Q, Zhai JL, Kapali G, Kramer DM, Howe GA. 2020. A phytochrome B independent pathway restricts growth at high levels of jasmonate defense. *Plant Physiology* **183**: 733–749.

Major IT, Yoshida Y, Campos ML, Kapali G, Xin XF, Sugimoto K, Ferreira DD, He SY, Howe GA. 2017. Regulation of growth-defense balance by the JASMONATE ZIM-DOMAIN (JAZ)-MYC transcriptional module. *New Phytologist* **215**: 1533–1547.

Margalha L, Confraria A, Baena-Gonzalez E. 2019. SnRK1 and TOR: modulating growth-defense trade-offs in plant stress responses. *Journal of Experimental Botany* **70**: 2261–2274.

Matsui K. 2006. Green leaf volatiles: hydroperoxide lyase pathway of oxylipin metabolism. *Current Opinion in Plant Biology* **9**: 274–280.

Mengarelli DA, Tewes LR, Balazadeh S, Zanor MI. 2021. FITNESS Acts as a negative regulator of immunity and influences the plant reproductive output after *Pseudomonas syringae* infection. *Frontiers in Plant Science* **12**: 606791.

Michaletz ST, Weiser MD, McDowell NG, Zhou JZ, Kaspari M, Helliker BR, Enquist BJ. 2016. The energetic and carbon economic origins of leaf thermoregulation. *Nature Plants* **2**: 16129.

Millard P. 1988. The accumulation and storage of nitrogen by herbaceous plants. *Plant, Cell & Environment* **11**: 1–8.

Monson RK, Weraduwage SM, Rosenkranz M, Schnitzler J-P, Sharkey TD. 2021. Leaf isoprene emission as a trait that mediates the growth-defense trade-off in the face of climate stress. *Oecologia*, in press. doi: 10.1007/s00442-020-04813-7.

Monte I, Franco-Zorilla JM, García-Casado G, Zamarreño AM, García-Mina JM, Nishihama R, Kohchi T, Solano R. 2019. A single JAZ repressor controls the jasmonate pathway in *Marchantia polymorpha*. *Molecular Plant* **12**: 185–198.

Mooney HA. 1972. The carbon balance of plants. *Annual Review of Ecology and Systematics* **3**: 315–346.

Mooney HA, Gulmon SL. 1982. Constraints on leaf structure and function in reference to herbivory. *BioScience* **32**: 198–206.

Navarro L, Bari R, Achard P, Lison P, Nemri A, Harberd NP, Jones JDG. 2008. DELLAs control plant immune responses by modulating the balance and salicylic acid signaling. *Current Biology* **18**: 650–655.

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

Osella AV, Mengarelli DA, Mateos J, Dong SC, Yanovsky MJ, Balazadeh S, Valle EM, Zanor MI. 2018. FITNESS, a CCT domain-containing protein, deregulates reactive oxygen species levels and leads to fine-tuning trade-offs between reproductive success and defence responses in *Arabidopsis*. *Plant, Cell & Environment* **41**: 2328–2341.

Osier TL, Lindroth RL. 2006. Genotype and environment determine allocation to and costs of resistance in quaking aspen. *Oecologia* **148**: 293–303.

Pacala SW, Crawley MJ. 1992. Herbivores and plant diversity. *American Naturalist* **140**: 243–260.

Pajerowska-Mukhtar KM, Wang W, Tada Y, Oka N, Tucker CL, Fonseca JP, Dong XN. 2012. The HSF-like transcription factor TBF1 is a major molecular switch for plant growth-to-defense transition. *Current Biology* **22**: 103–112.

Parker GA, Maynard SJ. 1990. Optimality theory in evolutionary biology. *Nature* **348**: 27–33.

Peñuelas M, Monte I, Schweizer F, Vallat A, Reymond P, Garcia-Casado G, Franco-Zorrilla JM, Solano R. 2019. Jasmonate-related MYC transcription factors are functionally conserved in *Marchantia polymorpha*. *Plant Cell* **31**: 2491–2509.

Pieterse CMJ, van der Does D, Zamioudis C, Leon-Reyes A, Van Wees SCM. 2012. Hormonal modulation of plant immunity. *Annual Review of Cell and Developmental Biology* **28**: 489–521.

Quail PH. 2002. Phytochrome photosensory signalling networks. *Nature Reviews Molecular Cell Biology* **3**: 85–93.

Radojicic A, Li X, Zhang YL. 2018. Salicylic acid: a double-edged sword for programed cell death in plants. *Frontiers in Plant Science* **9**: 1133.

Reich PB. 2014. The world-wide 'fast–slow' plant economics spectrum: a traits manifesto. *Journal of Ecology* **102**: 275–301.

Reich PB, Wright IJ, Cavender-Bares J, Craine JM, Oleksyn J, Westoby M, Walters MB. 2003. The evolution of plant functional variation: traits, spectra, and strategies. *International Journal of Plant Science* **164**: S143–S164.

Reznick D. 1985. Costs of reproduction: an evaluation of the empirical evidence. *Oikos* **44**: 257–267.

Rhoades DF. 1979. Evolution of plant chemical defense against herbivores. In: Rosenthal GA, Janzen DH, eds. *Herbivores: their interaction with secondary plant metabolites*. New York, NY, USA: Academic Press, 1–55.

Rockwell NC, Su YS, Lagarias JC. 2006. Phytochrome structure and signaling mechanisms. *Annual Review of Plant Biology* **57**: 837–858.

Roustan V, Jain A, Teige M, Ebersberger I, Weckwerth W. 2016. An evolutionary perspective of AMPK-TOR signaling in the three domains of life. *Journal of Experimental Botany* **67**: 3897–3907.

Sablowski R, Dornelas MC. 2014. Interplay between cell growth and cell cycle in plants. *Journal of Experimental Botany* **65**: 2703–2714.

Schluter D, Price TD, Rowe L. 1991. Conflicting selection pressures and life history trade-offs. *Proceedings of the Royal Society of London* **246**: 11–17.

Schuman MC, Baldwin IT. 2016. The layers of plant responses to insect herbivores. *Annual Review of Entomology* **61**: 373–394.

Schweitzer JA, Madritch MD, Bailey JK, LeRoy CJ, Fischer DG, Rehill BJ, Lindroth RL, Hagerman AE, Wooley SC, Hart SC *et al.* 2008. From genes to ecosystems: the genetic basis of condensed tannins and their role in nutrient regulation in a *Populus* model system. *Ecosystems* **11**: 1005–1020.

Sheard LB, Tan XU, Mao H, Withers J, Ben-Nissan G, Hinds TR, Kobayashi Y, Hsu F-F, Sharon M, Browse J *et al.* 2010. Jasmonate perception by inositol-phosphate-potentiated COI1-JAZ co-receptor. *Nature* **468**: 400–405.

Smith AM, Stitt M. 2007. Coordination of carbon supply and plant growth. *Plant, Cell & Environment* **30**: 1126–1149.

Stamp N. 2003. Out of the quagmire of plant defense hypotheses. *Quarterly Review of Biology* **78**: 23–55.

Stearns SC. 1977. The evolution of life-history traits: a critique of the theory and a review of the data. *Annual Review of Ecology and Systematics* **8**: 145–171.

Steinbrenner AD, Muñoz-Amatriaín M, Chaparro AF, Aguilar-Venegas JM, Lo S, Okuda S, Glauser G, Dongiovanni J, Shi DA, Hall M *et al.* 2020. A receptor-like protein mediates plant immune responses to herbivore-associated molecular patterns. *Proceedings of the National Academy of Sciences, USA* **117**: 31510–31518.

Strauss SY, Agrawal AA. 1999. The ecology and evolution of plant tolerance to herbivory. *Trends in Ecology and Evolution* **14**: 179–185.

Strauss SY, Rudgers JA, Lau JA, Irwin RE. 2002. Direct and ecological costs of resistance to herbivory. *Trends in Ecology and Evolution* **17**: 278–285.

Sun TJ, Zhang YL. 2021. Short- and long-distance signaling in plant defense. *The Plant Journal* **105**: 505–516.

Thaler JS, Humphrey PT, Whiteman NK. 2012. Evolution of jasmonate and salicylate signal crosstalk. *Trends in Plant Science* **17**: 260–270.

Tiessen A, Prescha K, Branscheid A, Palacios N, McKibbin R, Halford NG, Geigenberger P. 2003. Evidence that SNF1-related kinase and hexokinase are involved in separate sugar-signalling pathways modulating post-translational redox activation of ADP-glucose pyrophosphorylase in potato tubers. *The Plant Journal* **35**: 490–500.

Tsuda K, Sato M, Stoddard T, Glazebrook J, Katagiri F. 2009. Network properties of robust immunity in plants. *PLoS Genetics* **5**: e1000772.

Ullmann-Zeunert L, Stanton MA, Wielsch N, Bartram S, Hummert C, Svatos A, Baldwin IT, Groten K. 2016. Quantification of growth-defense trade-offs in a common currency: nitrogen required for phenolamide biosynthesis is not derived from ribulose-1,5-bisphosphate carboxylase/oxygenase turnover. *The Plant Journal* **75**: 417–429.

Valim H, Dalton H, Joo Y, McGale E, Halitschke R, Gaquerel E, Baldwin IT, Schuman MC. 2020. TOC1 in *Nicotiana attenuata* regulates efficient allocation of nitrogen to defense metabolites under herbivory stress. *New Phytologist* **228**: 1227–1242.

Van der Meijden E, De Boer NJ, Van der Veen-Van Wijk CAM. 2000. Pattern of storage and regrowth in ragwort. *Evolutionary Ecology* **14**: 439–455.

Van der Meijden E, Wijn M, Verkaar HJ. 1988. Defense and regrowth, alternative plant strategies in the struggle against herbivores. *Oikos* **51**: 355–363.

Vega-Munoz I, Duran-Flores D, Fernandez-Fernandez AD, Heyman J, Ritter A, Stael S. 2020. Breaking bad news: dynamic molecular mechanisms of wound response in plants. *Frontiers in Plant Science* **11**: 610445.

Verhage A, van Wees SCM, Pieterse CMJ. 2010. Plant immunity: it's the hormones talking, but what do they say? *Plant Physiology* **154**: 536–540.

Viola DV, Mordecai EA, Jaramillo AG, Sistla SA, Albertson LK, Gosnell JS, Cardinale BJ, Levine JM. 2010. Competition-defense tradeoffs and the maintenance of plant diversity. *Proceedings of the National Academy of Sciences, USA* **107**: 17217–17222.

Vleminckx J, Fortunel C, Valverde-Barrantes O, Paine CET, Engel J, Petronelli P, Dourdain AK, Guevara J, Beroujon S, Baraloto C. 2021. Resolving whole-plant economics from leaf, stem and root traits of 1467 Amazonian tree species. *Oikos* **130**: 1193–1208.

Vos IA, Verhage A, Schuurink RC, Watt LG, Pieterse CMJ, Van Wees SCM. 2013. Onset of herbivore-induced resistance in systemic tissue primed for jasmonate-dependent defenses is activated by abscisic acid. *Frontiers in Plant Science* **4**: 539.

Wang C, Liu Y, Li S-S, Han G-Z. 2015. Insights into the origin and evolution of the plant hormone signaling machinery. *Plant Physiology* **167**: 872–886.

Wang D, Amornsiripanitch N, Dong X. 2006. A genomic approach to identify regulatory nodes in the transcriptional network of systemic acquired resistance in plants. *PLoS Pathogens* **2**: 1042–1050.

Westoby M, Falster DS, Moles AT, Vesk PA, Wright IJ. 2002. Plant ecological strategies: some leading dimensions of variation between species. *Annual Review of Ecology and Systematics* **33**: 125–159.

Whitehead SR, Bass E, Corrigan A, Kessler A, Poveda K. 2021. Interaction diversity explains the maintenance of phytochemical diversity. *Ecology Letters* **24**: 1205–1214.

Whitham TG, Bailey JK, Schweitzer JA, Shuster SM, Bangert RK, LeRoy CJ, Lonsdorf EV, Allan GJ, DiFazio SP, Potts BM *et al.* 2006. A framework for community and ecosystem genetics: from genes to ecosystems. *Nature Reviews Genetics* **7**: 510–523.

Williams GC. 1966. Natural selection costs of reproduction and refinement of Lack's Principle. *American Naturalist* **100**: 687–690.

Wright IJ, Reich PB, Westoby M, Ackerly DD, Baruch Z, Bongers F, Cavender-Bares J, Chapin T, Cornelissen JHC, Diemer M *et al.* 2004. The worldwide leaf economics spectrum. *Nature* **428**: 821–827.

Wurzinger B, Nukarinen E, Nägele T, Weckwerth W, Teige M. 2018. The SnRK1 kinase as central mediator of energy signaling between different organelles. *Plant Physiology* **176**: 1085–1094.

Xu G, Yuan M, Ai C, Liu L, Zhuang E, Karapetyan S, Wang S, Dong X. 2017. uORF-mediated translation allows engineered plant disease resistance without fitness costs. *Nature* **545**: 491–494.

Xu H, Liu Q, Yao T, Fu X. 2014. Shedding light on integrative GA signaling. *Current Opinion in Plant Biology* **21**: 89–95.

Yamamura N, Tsuji N. 1995. Optimal strategy of plant antiherbivore defense – implications for apparency and resource-availability theories. *Ecological Research* **10**: 19–30.

Yang D-L, Yao J, Mei C-S, Tong X-H, Zeng L-J, Li Q, Xiao L-T, Sun T-P, Li J, Deng X-W *et al.* 2012. Plant hormone jasmonate prioritizes defense over growth by interfering with gibberellin signaling cascade. *Proceedings of the National Academy of Sciences, USA* **109**: 1192–1200.

Zhang L, Zhang F, Melotto M, Yao J, He SY. 2017. Jasmonate signaling and manipulation by pathogens and insects. *Journal of Experimental Botany* **68**: 1371–1385.

Züst T, Agrawal AA. 2017. Trade-offs between plant growth and defense: an emerging mechanistic synthesis. *Annual Review of Plant Biology* **68**: 513–534.