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False idolatry of the mythical growth versus immunity tradeoff in molecular systems plant pathology☆

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ABSTRACT

Systems studies of signal transduction pathways that modulate plant growth and immunity are rapidly identifying a large number of interactions within these pathways. These interactions are frequently presented as mechanisms allowing a plant to make proper decisions with regards to how to partition energy and resources in a proposed growth versus immunity tradeoff. This is a reinterpretation of the classical costs of resistance theory that has a long history in the ecology research community. While the ecology community is reinterpreting this theory, the reinterpretation has not been introduced into the molecular systems biology community that is studying the intersection of regulatory pathways. In this article, I describe evidence against a simple growth versus immunity tradeoff concept and propose an alternative wherein the intersection of these regulatory pathways is instead designed to coordinate these pathways, and not simply link them in mutual antagonism, to optimize fitness in complex environments where resistance/immunity and growth do not have simple linear relationships with fitness.

1. Introduction

Recent years have seen the re-introduction of ecological theories into studies focused on molecular plant pathology. This includes a burgeoning interest in the concept of costs of resistance and how this may drive an interplay between growth and immunity [1,2]. A cost of resistance is broadly defined as any negative effect that expressing a resistance trait has on plant fitness [2–5]. In plant pathology, this is being increasingly linked to an interest in tradeoffs between growth and immunity [6–14]. This is being driven by molecular studies that are finding interactions between regulatory genes involved in the control of growth and defense. These links are being found in related signaling pathways that interlink the brassinosteroid and effector triggered immunity pathways [7,11,12,15]. Similar links between development and plant defense signaling have been found for gibberellins, salicylic acid, auxin and jasmonic acid [16–22]. Interestingly, just as the molecular plant pathology community is beginning to investigate their systems using a cost of resistance framework, the ecology community is reassessing if there are costs of resistance and what this concept fundamentally means [3–5]. However, this reconsideration in the ecological community has not yet been translated into studies of molecular plant pathology. This article is intended to communicate misperceptions about costs of resistance that are permeating the molecular oriented literature and to provide alternate concepts of what is truly meant by the cost of immunity.

2. What do flux costs mean in a natural context?

The most common interpretation of resistance costs makes the implicit assumption that energy and elements are universally limiting for plant growth and/or fitness in the wild [13,14]. This is often pictorialized as a teeter-totter wherein the plant has to decide if any specific element or energy is placed into growth or defense. This simplistic representation also makes the implicit statement that there are no other options or avenues open to the plant in this process. This teeter-totter model can be classified as the idea that any flux of a nutrient into a defense process necessarily removes that nutrient from what otherwise would have been growth or biomass accumulation, i.e. a flux cost model (Fig. 1). However, the way the flux cost model is presented assumes that all nutrients are equally limiting which is an oversimplification of how nutrients limit plant fitness in the wild [23]. In natural ecosystems, growth is limited by imbalances in the availability of nutrients and

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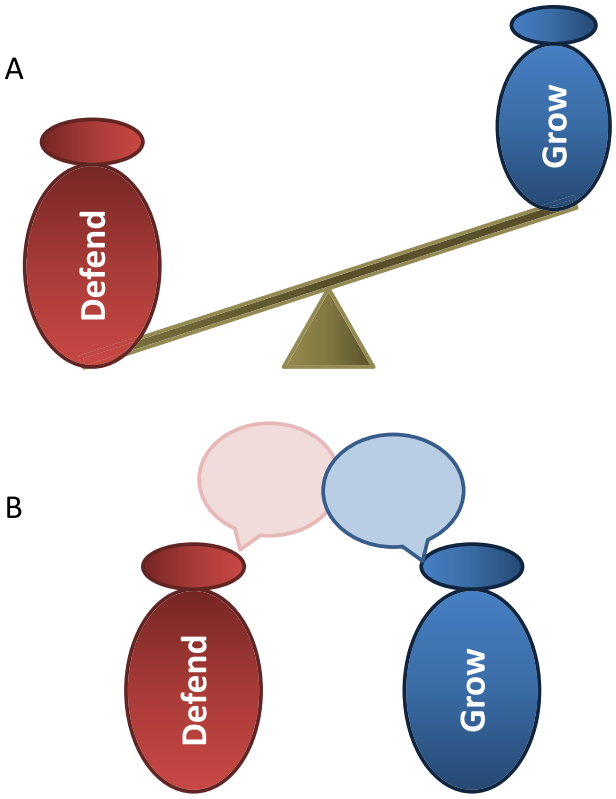

Fig. 1. Alternative models of growth and defense. A. Standard illustration of the growth vs immunity tradeoff where the two are in opposition. B. Alternative conceptualization whereby growth and defense are in a continual conversation to coordinate.

micronutrients rather than singular nutrient limitations. This means that potassium or phosphorus may be growth limiting in an environment where carbon and nitrogen may be freely available and non-limiting [23]. As such, it is the balance of nutrients that is more critical than any single nutrient when considering how nutrients may limit growth.

The observation that the plant is more responsive to the specific balance of nutrients and micronutrients raises the possibility that nutrient limitations on growth may be different than nutrient limitations on defense. Most plant defense mechanisms are based on proteins and metabolites that are predominantly Carbon and Nitrogen based with almost no associated Potassium and Phosphorus [24–26]. Thus, it is possible that in environments where Potassium, Phosphorus or other micronutrients are growth limiting that the plant has spare Carbon and Nitrogen available that cannot be used for growth. This spare Carbon and Nitrogen could then be used to create defenses at essentially no cost to growth. Thus, flux costs are not universal and instead are highly conditional based on the available nutrient and energy status of the plants environment. This suggests that the specific defense metabolism and mechanisms in a plant may be co-adapted to the plants typical nutrient profile to maximize the utilization of nutrients that are not rate limiting in that plants ecosystem niche. For example, a plant that typically grows in nitrogen limiting environments may shift from nitrogen-heavy defenses like alkaloids towards carboniferous defenses like terpenoids.

3. But constitutive defense mutants grow smaller?

A common argument in support of the idea that growth and defense must be in a tradeoff relationship is the observation that constitutive defense mutants are frequently small [27–32]. This argument posits that the elevated defense in these mutants is removing nutrients that would otherwise be utilized for growth. However, there is little evidence about the exact molecular reason for the diminutive growth in these mutants. A similar growth defect argument was proposed to explain why the *reduced epidermal fluorescence* (*ref*) mutants deficient in phenolic metabolism displayed growth defects [33–36]. It was posited that the deficiency in lignin production and structure created a simple growth defect due to altered lignin physics [33–36]. However, this was later found to be an incorrect interpretation as shown by a suppressor screen that identified mutants that alleviated the growth defects in the *ref* mutants [35,37–39]. These suppressors had wild-type growth rates while still containing the deficient lignin indicating that the growth effect was not caused by physical issues associated with the lignin deficit. Instead all of the suppressor mutations were in genes encoding components of the mediator transcriptional complex showing that the growth defect was caused by altered signal transduction and regulation of development and not by the physical or flux based costs of the *ref* mutant [35,37–39]. Thus, it is possible that mutants displaying constitutive defense responses may show a growth defect for regulatory or developmental reasons that have nothing to do with the associated flux cost of producing those defense responses. For example, constitutive expression of jasmonate mediated defenses didn't specifically alter growth but instead altered flowering time and other phenological processes suggesting that this constitutive jasmonate mutant displayed regulatory costs more than elemental costs [40]. Thus, more work needs to be done to understand why constitutive defense mutants display growth defects.

Modern biochemical genetics is providing a more explicit assessment of the flux costs associated with specific defenses by providing the ability to genetically delete individual defense genes or pathways and then assess any resulting growth effect. The simple growth vs immunity tradeoff model would suggest that the energy no longer used for defense in these mutants should be redirected from into growth. Analysis of genotypes altered in glucosinolate accumulation was unable to find a significant link with absolute growth [41–43]. Instead, it was only possible to see a small increase in relative growth rate in these mutants and only at early developmental stages and not in larger mature plants [41,42]. Importantly, this slight growth rate boost only benefited these plants when they are competing with other plant genotypes, i.e. a low glucosinolate plant would outgrow a high glucosinolate plant when they are in direct competition. Similar results had been found in the wild using Brassica [44–46]. Thus, if there is a direct growth benefit obtained by redirecting defenses to growth, these benefits appear to be specific to conditions when two genotypes are in direct competition. Thus, studying any growth vs immunity tradeoff hypothesis must involve competition studies.

4. What are other costs of immunity/resistance?

The experiment showing potentially the most overt cost of a resistance mechanism was a set of studies that linked the presence of an R gene to a fitness cost in the absence of the pathogen [47–49]. This study was originally controversial because the estimated flux cost, the energy required to produce a single R gene encoded protein that accumulates to low levels, did not agree with the measured 9% cost in fitness in plants expressing the functional R protein [48]. However, flux costs are not the only potential cost for a resistance mechanism. There are also ecological or opportunity costs wherein a resistance mechanism that may provide a fitness benefit by aiding the plants interaction with one organism