Journal of Chemical Ecology, Vol. 31, No. 10, October 2005 (©2005)
DOI: 10.1007/s10886-005-7099-7

HERBIVORES, VASCULAR PATHWAYS, AND SYSTEMIC INDUCTION: FACTS AND ARTIFACTS

COLIN ORIANS*

Department of Biology, Tufts University, Medford, MA 02155, USA

(Received March 22, 2004; revised December 1, 2004; accepted May 18, 2005)

Abstract—Over the past 10 years there has been tremendous growth in our understanding of molecular, chemical, and morphological induction of traits involved in the resistance of plants to herbivores. Although it is well established that the patterns of induction can be constrained by a plant's vascular architecture, studies often fail to account for these constraints. Failure to do so has the potential to severely underestimate both the patterns and extent of induction. Here I review (1) the evidence for vascular control of induced responses, (2) how interspecific variation in phyllotaxy influences spatial patterning of induction, (3) the factors, phloem transport and volatile production, that may break down vascular constraints and lead to more widespread induction, and (4) the experimental approaches that could be compromised when vascular architecture is not considered. I show that vascular constraints in systemic induction are commonplace, but vary among species. I suggest that when induction is more widespread than expected from patterns of phyllotaxy, differences in vascular connectivity and volatile production may be responsible. I argue that advances in the mechanisms of systemic induction, cross-talk between different signal transduction pathways, specificity of induction, costs and benefits of systemic induction, and the effects of induced changes on herbivores and their natural enemies require that experiments be designed to examine and/or control for vascular constraints in systemic induction.

Key Words—Systemic induction, vascular architecture, sectoriality, long-distance transport, source–sink dynamics, volatiles, experimental design.

INTRODUCTION

It is widely recognized that constitutive defenses limit the scale of the initial herbivore attack, whereas induced defenses determine patterns of subsequent

* To whom correspondence should be addressed. E-mail: Colin.Orians@Tufts.edu

attack (Karban and Myers, 1989; Haukioja, 1990; Jones et al., 1993; Karban and Baldwin, 1997). Induction has been documented in over 100 species of plants (Agrawal and Karban, 1999) and can be morphological, chemical, local, systemic, and highly specific (see Karban and Baldwin, 1997). For the purposes of this review, I define systemic induction as molecular, chemical, or morphological changes that occur in distant undamaged leaves. Recent advances have improved our understanding of plant responses to attack and the effects of these responses to herbivores. We now know that (1) damage results in long-distance transport of signal molecules that elicit changes in distant leaves (Zhang and Baldwin, 1997; Stratmann, 2003); (2) mechanical damage and damage by specific herbivores (and pathogens) generally elicit unique molecular, biochemical, and morphological responses (e.g., Baldwin, 1988; Thomma et al., 1998; Agrawal, 2000; Schittko et al., 2000; Walling, 2000; Voelckel and Baldwin, 2004); (3) induced responses can selectively affect the performance of herbivores and the behavior of natural enemies (Thaler, 1999a,b; Thaler et al., 1999, 2002; Agrawal, 2000; De Moraes et al., 2001; van Poecke et al., 2001); (4) there can be reproductive costs of induction (Baldwin et al., 1990, 1998; Agrawal and Karban, 1999; Heil and Baldwin, 2002; Zavala and Baldwin, 2004; Zavala et al., 2004); and (5) ontogeny sink and strength modify both the strength and pattern of induction (e.g., van Dam et al., 2001; Arnold and Schultz, 2002; Arnold et al., 2004).

We have made excellent progress in our understanding of the mechanisms, costs, and consequences of local and systemic induction. Unfortunately, the importance of spatial variation in systemic induction is often ignored but is critical to future progress. In this review, I focus on herbivore-induced induction within plants and argue that (1) vascular architecture, the pathway of signal movement to and away from the damaged leaf, is a key controller of systemic induction patterns; (2) when induction in some species is more widespread than expected, differences in phloem transport, release, and retrieval along the phloem pathway, and volatile production may allow for greater integration of the induced response; and (3) experimental designs that fail to account for spatial variation in induction are likely to underestimate or even fail to detect systemic induction. The points made apply to signal transduction following pathogen attack provided that the pathogen does not move systemically itself.

VASCULAR ARCHITECTURE

In this section, I first describe how vascular architecture determines patterns of systemic induction and then review inter- and intraspecific factors that control patterns of vascular architecture.

Vascular Architecture and Long-Distance Transport. Botanists have known for years that the transport of photosynthate, signal molecules, and hormones among leaves is controlled by vascular architecture (reviewed by Murray et al., 1982; Watson and Casper, 1984; Watson, 1986; Dickson and Isebrands, 1991; Sachs et al., 1993; Marshall, 1996). There is evidence for both phloem and xylem transport of signal molecules associated with chemical induction (Malone and Alarcon, 1995; Stratmann, 2003).

From a phloem perspective, the anatomy of sieve cells has evolved so that the path of least resistance is longitudinal, through the sieve plates (van Bel et al., 2002), and there is little symplastic transport between mature cells, and this limits movement among sieve cells (reviewed by Thorpe et al., 2005). The bulk of carbohydrate and signal transport is both longitudinal and toward sink leaves, but modified by vascular connectivity (Watson and Casper, 1984; Dickson and Isebrands, 1991; Preston, 1998; Arnold and Schultz, 2002). Connectivity is based on orthostichy—the phyllotactic arrangement of leaves that describes the distribution of vascular traces. Export from one leaf is greatest to orthostichous leaves because these leaves share primary vascular traces. Export to leaves in adjacent orthostichies is intermediate because these leaves have partial connectivity, and export is absent to leaves in opposite orthostichies because they lack vascular connectivity (Murray et al., 1982; Watson and Casper, 1984). There may be xylem transport of signal molecules as well (Malone and Alarcon, 1995). These authors suggest that upon leaf tissue damage, signal molecules can be drawn into the xylem and transported systemically. Since leaf-to-leaf connectivity is similar for xylem and phloem (see Orians et al., 2000; Zwieniecki et al., 2003; and reviewed by Orians et al., 2005), vascular constraints on signal transport are expected by either mechanism.

Whichever pathway of transport, xylem or phloem, within-plant variation in systemic induction is commonplace (Davis et al., 1991; Shulaev et al., 1995; Rhodes et al., 1999; Orians et al., 2000; Schittko and Baldwin, 2003). In cottonwood (*Populus trichocarpa* × *Populus deltoides*, Davis et al., 1991), tobacco (*Nicotiana* sp., Shulaev et al., 1995; Schittko and Baldwin, 2003), and tomato (*Lycopersicon esculentum*, Rhodes et al., 1999), leaves with direct vascular connections to the damaged leaf show greater defensive chemical induction than leaves without vascular connections. Systemic induction in tomato not only exhibits leaf-to-leaf variation, but within-leaf variation as well (Orians et al., 2000). Orians et al. (2000) found that proteinase inhibitor induction in tomato was greatest in leaflets that have direct vascular connections and lower in leaflets without direct connections. That tomato leaflets without direct connections exhibited induction suggest that signal transduction is not completely isolated to specific sectors (see Beyond Vascular Architecture for discussion of possible mechanisms).

Such chemical changes are ecologically relevant. Jones et al. (1993) demonstrated that differential induction alters leaf beetle performance on cottonwood (*Populus deltoides*). More recently, Viswanathan and Thaler (2004) found induced resistance in orthostichous leaves, but induced susceptibility in non-orthostichous leaves of *Solanum dulcamara* when damage was intense. This suggests that induction changes source–sink relationships (*sensu* Arnold and Schultz, 2002) and may result in greater allocation of resources toward resistance traits in the damaged sector and lowered allocation of resources toward resistance traits in other sectors.

Inter- and Intraspecific Variation in Phyllotaxy. Differences in vascular architecture among plants can alter patterns of systemic induction. Most angiosperms have a 2/5 phyllotaxy (Moore et al., 1998). A 2/5 phyllotaxy reflects the fact that every fifth leaf is vertically aligned after two spirals around the plant. In this arrangement, carbon exported from leaf 1 accumulates most in leaves 4 and 6, to a lesser extent in leaf 3, and minimally in leaves 2 and 5 (Dickson, 1991). However, some species have a 1/2 or 3/8 phyllotaxy (Moore et al., 1998). Moreover, since phyllotaxy may change through development—the phyllotactic arrangement of leaves in *Populus* changes from 2/5 to 3/8 to 5/13 with plant development (Larson, 1977)—it is essential to map the phyllotaxy with dyes (Orians et al., 2000) or isotopes (Larson, 1977) to determine the operative vascular connectivity.

BEYOND VASCULAR ARCHITECTURE

Vascular constraints on systemic induction are common; however, there is increasing evidence that induction in some species is less constrained (i.e., more integrated) (Shea and Watson, 1989; Mutikainen et al., 1996). Shea and Watson (1989) found that localized damage to fireweed, *Chamaenerion angustifolium*, despite having a 3/8 phyllotaxy, had little effect on fruit production within that orthostichy. They also report that ^{14}C flow was less restricted. More recently, Orians et al. (2000) and Kiefer and Slusarenko (2003) showed that leaf tissues lacking direct vascular connections are induced in tomato and *Arabidopsis*, respectively. What might lead to induction in other sectors? I suggest that two factors may facilitate greater integration: (1) symplastic and apoplastic pathways in long-distance phloem transport and (2) volatile release at the site of damage and subsequent induction within distant plant tissues.

Throughout development, there is extensive apoplastic exchange—release and retrieval—of phloem constituents along the phloem pathway (Minchin and Thorpe, 1984,1987; van Bel et al., 2002; van Bel, 2003; Thorpe et al., 2005). Such release and retrieval requires an extensive array of specialized transfer

cells and membrane-bound transporters (Pate and Dieter Jeschke, 1995; Fisher, 2000). Most importantly, from the perspective of integration, species differ both in type and in density of transporters (van Bel, 2003). Clearly, transport of damage-induced signal molecules could lead to more widespread induction. Further work is necessary to examine how interspecific variation in apoplastic exchange alters the degree of symplastic isolation and subsequent spatial patterning of systemic induction.

It appears that signals are most likely to reach young developing tissues in other sectors. There is little symplastic exchange among mature sieve elements, but early in tissue development there can be extensive symplastic transport due to high sieve element and plasmodesmal density (van Bel, 2003; Thorpe et al., 2005). This allows for greater movement between adjacent sieve elements in young developing tissues, and modification of microchannel size of the plasmodesmata can facilitate even greater exchange of carbohydrates, viruses, and perhaps signal molecules (Lucas, 1997; Ishiwatari et al., 1998; Ruiz-Medrano et al., 1999). Although many details remain unresolved, recent evidence suggests that modification of microchannel size is a highly regulated and active process (Ruiz-Medrano et al., 2004). Thus, symplastic transport, especially in combination with apoplastic exchange, could result in widespread induction in young developing tissues. Whether plants have evolved mechanisms to facilitate movement of signal molecules from one sector to another requires further study.

I suggest that volatile release from damaged tissues may also result in more widespread induction. In fact, volatiles may be the only mechanism to induce leaves on distant branches where vascular connections are lacking. It is well known that volatiles released from damaged tissues and can cause molecular and chemical changes in adjacent plants, and alter subsequent resistance (Farmer, 2001; Kessler and Baldwin, 2001). The implications of this work are great because they suggest that plants may eavesdrop on their neighbors and adjust their allocation to defenses accordingly. However, it appears that eavesdropping requires that plants be in close proximity (within 15 cm) (Karban et al., 1997). Air turbulence and movement serves to dramatically reduce the concentration of volatiles arriving at distant plants, and as a consequence, there is considerable debate concerning the importance of interplant communication under field conditions (Lerdau, 2002; Dicke et al., 2003). Given that volatile signals become increasingly dilute with distance, volatiles may be more important for intraplant communication. Such tissues are in close proximity and likely to have the appropriate receptors to respond but may be on different branches with no vascular connections (Watson, 1986). Although intraplant volatile communication has been hypothesized to occur (Farmer, 2001), no studies have examined the importance of intraplant volatile communication as a mechanism to bypass vascular constraints in systemic induction.

EXPERIMENTAL APPROACHES

Despite the potential for more widespread induction than would be predicted by vascular architecture alone, the presence of vascular constraints is common. Therefore, failure to account for vascular constraints in systemic induction can: (1) underestimate the strength of induction; (2) lead to erroneous conclusions about specificity of induction; and (3) hide important within-plant heterogeneity in leaf quality. In this section, I review the approaches researchers have taken in their experiments and highlight which of the approaches are likely to underestimate the strength of induction and lead to inappropriate conclusions. It is not my goal to provide an exhaustive literature review, but to use a few case studies to highlight how knowledge of vascular architecture might improve our understanding of systemic induction and ultimately lead to more carefully designed experiments.

To date there have been three approaches to studying induction and each differs in the importance of vascular architecture to the results (Table 1). (1) Some studies have released herbivores on young plants and then, later in development, measured induction of specific traits or induced resistance (hereafter termed "immunization"; Karban, 1986; Agrawal et al., 1999). (2) Other studies have isolated herbivores on specific leaves (or manually damaged the leaf) and then measured induction in undamaged leaves shortly thereafter (hereafter termed "localized damage"; Davis et al., 1991; Jones et al., 1993; Stout et al., 1996a,b). (3) More recently, researchers have sprayed plants with powerful elicitors such as jasmonic acid and salicylic acid (Cipollini and Redman, 1999; Thaler, 1999a,b; Thaler et al., 1999; Moran and Thompson, 2001; Redman et al., 2001; Cipollini, 2002). Most of the elicitor studies applied inducing agents to the entire plant and then measured whole-plant induction (or if applied to a specific leaf, induction was only measured in that same leaf; Cipollini and Redman, 1999). Because most experiments involving elicitors have effectively measured local induction, vascular architecture should have no effect on the outcome of experiments and will not be discussed further.

Immunization studies that give herbivores free access to plants early in development and then measure systemic induction later in development appear robust. For example, Karban (1986, 1987) showed that when young cotton, *Gossypium hirsutum*, seedlings are attacked by mites, the plants often show increased resistance to subsequent mite attack. Agrawal et al. (1999) obtained similar results with cucumber, *Cucumus sativus*. In these studies, cotyledons/leaves were damaged and the whole plant was assayed for subsequent resistance. Since the initial damage was systemic, we would not expect induction to vary among leaves (Table 1).

Problems may arise when initial damaged is localized to specific leaves and induced responses are measured in other parts of the plant. Induction could

TABLE 1. TYPICAL TREATMENTS IMPOSED IN INDUCTION STUDIES, THE TYPES OF RESPONSES MEASURED, AND THE IMPORTANCE OF VASCULAR ARCHITECTURE TO PATTERNS OF SYSTEMIC INDUCTION

Treatment	Response	Importance of vascular architecture	Possible consequences[a]
(1) Immunization e.g., cotyledon inoculation followed by removal of damaging agent	Subsequent leaves, e.g., first true leaves	Minimal	None (if initial damage is dispersed)
(2) Localized damage to specific leaf or leaves	Whole plant	→ Very	Underestimate
	Specific tissue	→ Very	Underestimate or failure to detect
(3) Application of chemical elicitors to whole plant	Whole plant	Not	None
	Specific tissue		(functionally equivalent to localized induction)

[a] Consequences of failure to control for vascular architecture range from none, to underestimation, to failure to detect induction.

be underestimated if damage is concentrated on specific leaves, but induced responses are examined at the whole-plant level or might be severely underestimated in studies that only assay specific leaves for induced responses when the previous damage was concentrated on other leaves (Table 1). As illustration, Agrawal (2000) placed a caterpillar of one of four herbivore species (*Plutella xylostella*, *Spodoptera exigua*, *Pieris rapae*, or *Trichoplusia ni*) on one leaf and then measured how these herbivores performed on the remainder of the damaged plant. He found that previous damage by *Plutella* (a specialist) and *Spodoptera* (a generalist) resulted in increased resistance to all species. In contrast, previous damage by *Pieris* only induced resistance to *Spodoptera* and *Pieris*, and damage by *Trichoplusia* (a generalist) failed to induce resistance to the other herbivores. Although these results clearly indicated specificity of induction, some of the differences in herbivore responses may reflect differences in feeding behavior. Perhaps, *Plutella*—the species that appeared to be least affected by previous feeding—tended to avoid feeding on leaves with direct vascular connections to the damaged leaf and, thus, avoided the most heavily induced leaves. The later explanation is intriguing and deserves further examination.

More recently, Traw and Dawson (2002) released herbivores on four-leafed *Brassica nigra* and then quantified differences in foliar trichome density and sinigrin concentration on specific leaves between damaged and undamaged plants. They found evidence for trichome induction, but the effect was often insignificant and varied among plants. Perhaps differences in initial damage,

which they report, but did not control for in their analysis, limited their ability to detect differences. Because they did not quantify the pattern of initial damage, they could have detected (1) no induction—if damaged was concentrated on a single leaf that did not have direct connections to the sampled leaf; (2) moderate induction—if damage was dispersed; or (3) high induction—if damage was concentrated on the leaf with direct vascular connections to the sampled leaf.

An extreme example of uneven damage exists in studies in which damage is applied to one leaf and induction is measured in other leaves. Since some leaves lack direct vascular connections and others have only partial connections, induction might be underestimated. For example, Stout et al. (1996a) used different leaflets from a leaf adjacent to the damaged leaf to examine how different enzymes are induced following damage. Since some of the leaflets had direct connections whereas others did not, it is possible that differences in induction reflect the experimental design, and not differences in the enzymes. If true, failure to detect induction could be an artifact of the experimental design. It would be better to measure induction in leaflets or leaves known to share direct vascular connections. Alternatively, researchers could distribute damage across several consecutive leaves to make sure all sectors were damaged (as done by Constabel et al., 2000).

CONCLUSION

Whether studying molecular mechanisms of systemic induction, cross-talk between different signal transduction pathways, specificity of induction, costs and benefits of systemic induction, or the effects of induced changes on herbivores and their natural enemies, *a priori* knowledge of vascular constraints on systemic induction will help researchers make robust conclusions. Care should be taken to control for (or to examine) the effects of vascular architecture on induction. Controlling for vascular architecture necessitates that initial damage is dispersed across all sectors of the plant. Examining its effects requires mapping the vascular connections and quantifying the amount of damage to specific leaves. Since within-plant heterogeneity in leaf quality may affect herbivore behavior or performance (Denno and McClure, 1983) and may even influence foraging patterns of natural enemies, we can even use vascular architecture to create and study the effects of heterogeneity on herbivores (reviewed by Orians and Jones, 2001). As we learn more about the mechanisms of within-plant communication, both by long-distance signal transport of dissolved substances within the vascular system and by volatiles, we will gain an even greater understanding of the importance of vascular architecture in systemic induction across species.

Acknowledgments—I thank Danush Viswanathan, Anurag Agrawal, Michael Thorpe, and two anonymous reviewers for comments on an early version of this manuscript and the National Science Foundation (DEB 9981568) and the Andrew Mellon Foundation for financial support.

REFERENCES

AGRAWAL, A. A. 2000. Specificity of induced resistance in wild radish: causes and consequences for two specialist and two generalist caterpillars. *Oikos* 89:493–500.

AGRAWAL, A. A. and KARBAN, R. 1999. Why induced defenses may be favored over constitutive strategies in plants, pp. 45–61, *in* R. Tollrian and C. D. Harvell (eds.). The Ecology and Evolution of Inducible Defenses. Princeton University Press, Princeton.

AGRAWAL, A. A., GORSKI, P. M., and TALLAMY, D. W. 1999. Polymorphism in plant defense against herbivory: Constitutive and induced resistance in *Cucumis sativus*. *J. Chem. Ecol.* 25:2285–2304.

ARNOLD, T. M. and SCHULTZ, J. C. 2002. Induced sink strength as a prerequisite for induced tannin biosynthesis in developing leaves of *Populus*. *Oecologia* 130:585–593.

ARNOLD, T. M., APPEL, H., PATEL, V., STOCUM, E., KAVALIER, A., and SCHULTZ, J. 2004. Carbohydrate translocation determines the phenolic content of *Populus* foliage: a test of the sink–source model of plant defense. *New Phytol.* 164:157–164.

BALDWIN, I. T. 1988. The alkaloidal responses of wild tobacco to real and simulated herbivory. *Oecologia* 77:378–381.

BALDWIN, I. T., SIMS, C. L., and KEAN, S. E. 1990. The reproductive consequences associated with inducible alkaloidal responses in wild tobacco. *Ecology* 71:252–262.

BALDWIN, I. T., GORHAM, D., SCHMELZ, E. A., LEWANDOWSKI, C. A., and LYNDS, G. Y. 1998. Allocation of nitrogen to an inducible defense and seed production in *Nicotiana attenuata*. *Oecologia* 115:541–552.

CIPOLLINI, D. 2002. Does competition magnify the fitness costs of induced responses in *Arabidopsis thaliana*? A manipulative approach *Oecologia* 131:514–520.

CIPOLLINI, D. F. and REDMAN, A. M. 1999. Age-dependent effects of jasmonic acid treatment and wind exposure on foliar oxidase activity and insect resistance in tomato. *J. Chem. Ecol.* 25:271–281.

CONSTABEL, C. P., YIP, L., PATTON, J. J., and CHRISTOPHER, M. E. 2000. Polyphenol oxidase from hybrid poplar. Cloning and expression in response to wounding and herbivory. *Plant Physiol.* 124:285–295.

DAVIS, M. A., GORDON, M. P., and SMIT, B. A. 1991. Assimilate movement dictates remote sites of wound-induced gene expression in poplar leaves. *Proc. Natl. Acad. Sci. USA* 88:2393–2396.

DE MORAES, C. M., MESCHER, M. C., and TUMLINSON, J. H. 2001. Caterpillar-induced nocturnal plant volatiles repel conspecific females. *Nature* 419:531–577.

DENNO, R. F. and MCCLURE, M. S. 1983. (eds.). Variable Plants and Herbivores in Natural and Managed Systems. Academic Press, New York.

DICKE, M., AGRAWAL, A. A., and BRUIN, J. 2003. Plants talk, but are they deaf? *Trends Plant Sci.* 8:403–405.

DICKSON, R. 1991. Carbon fixation and distribution in young *Populus* trees, pp. 51–85, *in* A. S. Raghavendra (ed.). Physiology of Trees. John Wiley and Sons, Inc., New York.

DICKSON, R. E. and ISEBRANDS, J. G. 1991. Leaves as regulators of stress response, pp. 3–34, *in* H. A. Mooney, W. E. Winner, and E. J. Pell (eds.). Response of Plants to Multiple Stresses. Academic Press Inc., San Diego.

FARMER, E. E. 2001. Surface-to-air signals. *Nature* 411:854–856.

FISHER, D. B. 2000. Long-distance transport, pp. 730–784, *in* B. B. Buchanan, W. Gruissem, and R. L. Jones (eds.). Biochemistry and Molecular Biology of Plants. American Society of Plant Physiologists, Rockville, MD.

HAUKIOJA, E. 1990. Induction of defenses in trees. *Annu. Rev. Entomol.* 36:25–42.

HEIL, M. and BALDWIN, I. T. 2002. Fitness costs of induced resistance: Emerging experimental support for a slippery concept. *Trends Plant Sci.* 7:61–67.

ISHIWATARI, Y., FUJIWARA, T., MCFARLAND, K. C., NEMOTO, K., HAYASHI, H., CHINO, M., and LUCAS, W. J. 1998. Rice phloem thioredoxin h has the capacity to mediate its own cell-to cell transport through plasmodesmata. *Planta* 205:12–22.

JONES, C. G., HOPPER, R. F., COLEMAN, J. S., and KRISCHIK, V. A. 1993. Control of systematically induced herbivore resistance by plant vascular architecture. *Oecologia* 93:452–456.

KARBAN, R. 1986. Induced resistance against spider mites in cotton: Field verification. *Entomol. Exp. Appl.* 42:239–242.

KARBAN, R. 1987. Environmental conditions affecting the strength of induced resistance against mites in cotton. *Oecologia* 73:414–419.

KARBAN, R. and BALDWIN, I. T. 1997. Induced Responses to Herbivory. University of Chicago Press, Chicago.

KARBAN, R. and MYERS, J. H. 1989. Induced plant responses to herbivory. *Annu. Rev. Ecol. Syst.* 20:331–348.

KARBAN, R., BALDWIN, I. T., BAXTER, K. J., LAUE, G., and FELTON, G. W. 1997. Communication between plants: induced resistance in wild tobacco plants following clipping of neighboring sagebrush. *Oecologia* 125:66–71.

KESSLER, A. and BALDWIN, I. T. 2001. Defensive function of herbivore-induced plant volatile emissions in nature. *Science* 291:2141–2144.

KIEFER, I. W. and SLUSARENKO, A. J. 2003. The pattern if systemic acquired resistance induction within the *Arabidopsis* rosette in relation to the pattern of translocation. *Plant Physiol.* 132:840–847.

LARSON, P. R. 1977. Phyllotactic transitions in the vascular system of *Populus deltoides* Bartr. as determined by ^{14}C labeling. *Planta* 134:241–249.

LERDAU, M. 2002. Plants talk—but can they listen? *Science* 298:361.

LUCAS, W. J. 1997. Application of microinjection techniques to plant nutrition. *Plant Soil* 196:175–189.

MALONE, M. and ALARCON, J.-J. 1995. Only xylem-borne factors can account for systemic wound signaling in the tomato plant. *Planta* 196:740–746.

MARSHALL, C. 1996. Sectoriality and physiological organisation in herbaceous plants: an overview. *Vegetatio* 127:9–16.

MINCHIN, P. E. H. and THORPE, M. R. 1984. Apoplastic phloem unloading in the stem of bean *Phaseolis-vulgaris*. *J. Exp. Bot.* 35:538–550.

MINCHIN, P. E. H. and THORPE, M. R. 1987. Measurement of unloading and reloading of photo-assimilate within the stem of bean. *J. Exp. Bot.* 38:211–220.

MOORE, R., CLARK, W. D., and VODOPICH, D. S. 1998. pp. 314–315, *in* Botany, 2nd edn. WCB/McGraw-Hill, Dubuque, IA.

MORAN, P. J. and THOMPSON, G. A. 2001. Molecular responses to aphid feeding in *Arabidopsis* in relation to plant defense pathways. *Plant Physiol.* 125:1074–1085.

MURRAY, B. J., MAUK, C., and NOODÉN, L. D. 1982. Restricted vascular pipelines (and orthostichies) in plants. *What's New Plant Physiol.* 13:33–36.

MUTIKAINEN, P., WALLS, M., and OVASKA, J. 1996. Herbivore-induced resistance in *Betula pendula*: the role of plant vascular architecture. *Oecologia* 108:723–727.

ORIANS, C. M. and JONES, C. G. 2001. Plants as resource mosaics: a functional model for predicting

patterns of within-plant resource heterogeneity to consumers based on vascular architecture and local environmental variability. *Oikos* 94:493–504.

ORIANS, C. M., POMERLEAU, J., and RICCO, R. 2000. Vascular architecture generates fine scale variation in the systemic induction of proteinase inhibitors in tomato. *J. Chem. Ecol.* 26:471–485.

ORIANS, C. M., BABST, B., and ZANNE, A. E. 2005. Vascular constraints and long-distance transport in dicots, *in* N. M. Holbrook and M. Zwieniecki (eds.). Vascular Transport in Plants. Elsevier/AP co-imprint, Oxford.

PATE, J. S. and DIETER JESCHKE, W. 1995. Role of stems in transport, storage, and circulation of ions and metabolites by the whole plant, pp. 177–204, *in* B. L. Gartner (ed.). Plant Stems: Physiology and Functional Morphology. Academic Press, San Diego.

PRESTON, K. A. 1998. The effects of developmental stage and source leaf position on integration and sectorial patterns of carbohydrate movement in an annual plant, *Perilla frutescens* (Lamiaceae). *Am. J. Bot.* 85:1695–1703.

REDMAN, A. M., CIPOLLINI, D., and SCHULTZ, J. C. 2001. Fitness costs of jasmonic acid-induced defense in tomato, *Lycopersicon esculentum*. *Oecologia* 126:380–385.

RHODES, J. D., THAIN, J. F., and WILDON, D. C. 1999. Evidence for physically distinct systemic signaling pathways in the wounded tomato plant. *Ann. Bot.* 84:109–116.

RUIZ-MEDRANO, R., XOCONOSTLE-CAZARES, B., and LUCAS, W. J. 1999. Phloem long-distance transport of CmNACP mRNA: implications for supracellular regulation in plants. *Development* 126:4405–4419.

RUIZ-MEDRANO, R., XOCONOSTLE-CAZARES, B., and KRAGLER, F. 2004. The plasmodesmatal transport pathway for homeotic proteins, silencing signals and viruses. *Curr. Opin. Plant Biol.* 7:641–650.

SACHS, T., NOVOPLANSKY, A., and COHEN, D. 1993. Plants as competing population of redundant organs. *Plant Cell Environ.* 16:765–770.

SCHITTKO, U. and BALDWIN, I. T. 2003. Constraints to herbivore-induced systemic responses: bidirectional signaling along orthostichies in *Nicotiana attenuata*. *J. Chem. Ecol.* 29:763–770.

SCHITTKO, U., PRESTON, C. A., and BALDWIN, I. T. 2000. Eating the evidence? *Manduca sexta* larvae cannot disrupt specific jasmonate induction in *Nicotiana attenuata* by rapid consumption *Planta* 21:343–346.

SHEA, M. M. and WATSON, M. A. 1989. Patterns of leaf and flower removal: their effect on fruit growth in *Chamaenerion angustifolium (fireweed)*. *Am. J. Bot.* 76:884–890.

SHULAEV, V., LEON, J., and RASKIN, I. 1995. Is salicylic acid a translocated signal of systemic acquired resistance in tobacco? *Plant Cell* 7:1691–1701.

STOUT, M. J., WORKMAN, K. V., and DUFFEY, S. S. 1996a. Identity, spatial distribution, and variability of induced chemical responses in tomato plants. *Entomol. Exp. Appl.* 79: 255–271.

STOUT, M. J., WORKMAN, K. V., WORKMAN, J. S., and DUFFEY, S. S. 1996b. Temporal and ontogenetic aspects of protein induction in foliage of the tomato, *Lycopersicon esculentum*. *Biochem. Syst. Ecol.* 24:1–16.

STRATMANN, J. W. 2003. Long-distance run in the wound response—jasmonic acid is pulling ahead. *Trends Plant Sci.* 8:247–250.

THALER, J. S. 1999. Induced resistance in agricultural crops: Effects of jasmonic acid on herbivory and yield in tomato plants. *Environ. Entomol.* 28:30–37.

THALER, J. S. 1999. Jasmonate-inducible plant defences cause increased parasitism of herbivores. *Nature* 399:686–688.

THALER, J. S., FIDANTSEF, A. L., DUFFEY, S. S., and BOSTOCK, R. M. 1999. Trade-offs in plant defense against pathogens and herbivores: A field demonstration of chemical elicitors of induced resistance. *J. Chem. Ecol.* 25:1597–1609.

THALER, J. S., KARBAN, R., ULLMAN, D. E., BOEGE, K., and BOSTOCK, R. M. 2002. Cross-talk between jasmonate and salicylate plant defense pathways: effects on several plant parasites. *Oecologia* 131:235–277.

THOMMA, B. P. H. J., EGGERMONT, K., PENNINCKX, I. A. M. A., MAUCH-MANI, B., VOGELSANG, R., CAMMUE, B. P. A., and BROEKAERT, W. F. 1998. Separate jasmonate-dependent and salicylate-dependent defense–response pathways in *Arabidopsis* are essential for resistance to distinct microbial pathogens. *Proc. Natl. Acad. Sci. USA* 95:15107–15111.

THORPE, M., MINCHIN, P., GOULD, N., and MCQUEEN, J. 2005. The stem apoplast: A potential communication channel in plant growth regulation, *in* N. M. Holbrook and M. Zwieniecki (eds.). Vascular Transport in Plants. Elsevier/AP co-imprint, Oxford.

TRAW, M. B. and DAWSON, T. E. 2002. Differential induction of trichomes by three herbivores of black mustard. *Oecologia* 131:526–532.

VAN BEL, A. J. E., EHLERS, K., and KNOBLAUCH, M. 2002. Sieve elements caught in the act. *Trends Plant Sci.* 7:126–132.

VAN BEL, A. J. E. 2003. Transport phloem: Low profile, high impact. *Plant Physiol.* 131:1509–1510.

VAN DAM, N. M., HORN, M., MARES, M., and BALDWIN, I. T. 2001. Ontogeny constrains systemic protease inhibitor response in *Nicotiana attenuata*. *J. Chem. Ecol.* 27:547–568.

VAN POECKE, R., POSTHUMUS, M. P., and DICKE, M. A. 2001. Herbivore-induced volatile production by *Arabidopsis thaliana* leads to attraction of the parasitoid *Cotesia rebucula*: chemical, behavioral, and gene-expression analysis. *J. Chem. Ecol.* 27:1911–1928.

VISWANATHAN, D. V. and THALER, J. S. 2004. Plant vascular architecture and within-plant spatial patterns in resource quality following herbivory. *J. Chem. Ecol.* 30:531–543.

VOELCKEL, C. and BALDWIN, I. T. 2004. Generalist and specialist lepidopteran larvae elicit different transcriptional responses in *Nicotiana attenuata*, which correlate with larval FAC profiles. *Ecol. Lett.* 7:70–775.

WALLING, L. L. 2000. The myriad plant responses to herbivores. *J. Plant Growth Regul.* 19:195–216.

WATSON, M. A. 1986. Integrated physiological units in plants. *TREE* 1:119–123.

WATSON, M. A. and CASPER, B. B. 1984. Morphogenetic constraints on patterns of carbon distribution in plants. *Annu. Rev. Ecol. Syst.* 15:233–258.

ZAVALA, J. A. and BALDWIN, I. T. 2004. Fitness benefits of trypsin proteinase inhibitor expression in *Nicotiana attenuata* are greater than their costs when plants are attacked. *BMC Ecol.* 4:1–15.

ZAVALA, J. A., PATANKAR, A., GASE, K., and BALDWIN, I. T. 2004. Constitutive and inducible trypsin proteinase inhibitor production incurs large fitness costs in *Nicotiana attenuata*. *Proc. Natl. Acad. Sci. USA* 101:1607–1612.

ZHANG, Z. P. and BALDWIN, I. T. 1997. Transport of [2-C-14]jasmonic acid from leaves to roots mimics wound-induced changes in endogenous jasmonic acid pools in *Nicotiana sylvestris*. *Planta* 203:436–441.

ZWIENIECKI, M. A., ORIANS, C. M., MELCHER, P. J., and HOLBROOK, N. M. 2003. Ionic control of lateral exchange of water between vascular bundles in tomato. *J. Exp. Bot.* 54:1399–1406.