Long-distance signalling in plant defence

Martin Heil1 and Jurriaan Ton2

Plants use inducible defence mechanisms to fend off harmful organisms. Resistance that is induced in response to local attack is often expressed systemically, that is, in organs that are not yet damaged. In the search for translocated defence signals, biochemical studies follow the physical movement of putative signals, and grafting experiments use mutants that are impaired in the production or perception of these signals. Long-distance signals can directly activate defence or can prime for the stronger and faster induction of defence. Historically, research has focused on the vascular transport of signalling metabolites, but volatiles can play a crucial role as well. We compare the advantages and constraints of vascular and airborne signals for the plant, and discuss how they can act in synergy to achieve optimised resistance in distal plant parts.

Dynamic and systemic plant defences
Plants possess chemical and physical defences to reduce damage by primary consumers: animals and microbes that use them as a food source. Because defence is costly, plants have evolved inducible defence mechanisms that are activated or amplified in response to attack. Particular signals can prime plant tissues, that is, prepare them for an augmented response to future attack without directly activating costly defence mechanisms. Herbivorous animals are highly mobile, however, and even pathogenic microbes can move within the plant. Hence, plants require additional countermeasures to halt the dispersal of the attacker and so prevent further damage. Systemically expressed induced resistance is a response that specifically aims to restrict the spread of the attacker: upon local attack, enhanced resistance spreads to distal organs that are not yet affected.

Systemically induced resistance can be achieved by two mechanisms. First, it can result from systemic transport of defensive metabolites; and second, it can result from de novo expression of resistance mechanisms that are activated by translocated signals from the stress-exposed tissues. Recent evidence supports a crucial role for both mechanisms. This review focuses on the search for these long-distance signals. We summarize phenotypic evidence traditionally studied translocation of signals through the vascular system, and outline the advantages and constraints of these two long-distance transport mechanisms. We discuss the possibility that airborne signals reach systemic tissues at concentrations that cause priming rather than the full induction of defence, and we propose a model that illustrates how vascular and airborne long-distance signals can act in synergy to induce a balanced resistance in distal plant parts.

Systemic induction of resistance at the phenotypic level
The first evidence for long-distance regulation of induced resistance came from the work by Frank Ross in the 1960s. In work published in 1961 [1], Ross demonstrated that inoculation of the lower leaves of tobacco (Nicotiana tabacum) plants with necrosis-inducing tobacco mosaic virus (TMV) induced a hypersensitive reaction that resulted in enhanced resistance to a second infection in the upper leaves [1]. This phenomenon was termed systemic acquired resistance (SAR). SAR was first detected 2–3 days after primary virus infection and reached a maximum after 7 days, demonstrating that plants require time to generate, transport and deliver the long-distance signal that induces resistance in the upper leaves. Ten years later, Terrence Green and Clarence Ryan [2] reported that tomato (Lycope rus esculentum) responds to insect feeding by producing defensive proteins. Proteinase inhibitors (PIs; i.e., compounds that reduce protein digestion by insects) were induced in both damaged and undamaged leaves. Supplying extracts from wounded leaves to excised, but otherwise undamaged, plants could also trigger the production of PIs, a clear hint of the existence of a mobile signal [3]. Finally, systemically transported signals are also involved when translocation of defensive compounds contributes to systemic resistance, for example, when nicotine is produced in the roots of tobacco (Nicotiana sylvestris) upon leaf damage [4].

SAR, PIs and toxic secondary compounds directly target the plants’ enemies and are called ‘direct defences’, but plants can also defend themselves by releasing volatile organic compounds or extrafloral nectar to attract natural enemies of the herbivores [5,6]. Although some volatile compounds have direct effects against pathogens and herbivores, this strategy is generally referred to as ‘indirect defence’. In many cases, induced volatile emission is a systemic response [7]. For instance, a water-soluble substance from insect-infested lima bean (Phaseolus lunatus)
elicited volatile emissions in undamaged plants [8], and feeding on roots can induce indirect defences in above-ground parts [9,10]. Thus, the activation of indirect defences also involves regulation by long-distance signals.

Long-distance signals in the SAR response
The initial discovery of SAR [1] initiated decades of research into its underlying mechanisms. In the 1970s, Leendert C. van Loon found that both locally and systemically induced resistance to pathogens are associated with the induction of pathogenesis-related (PR) proteins [11]. In tobacco, injection with salicylic acid (SA) elicited enhanced resistance to TMV, and application of SA triggered the accumulation of PR-proteins [12,13], leading to speculation that SA might function as the endogenous signal. In many plant species, SA undoubtedly plays an important role in both locally expressed basal resistance and SAR [14]. SAR is predominantly effective against pathogens that are sensitive to SA-dependent basal defence mechanisms [15], and so we can even state that SAR represents a systemic enhancement of SA-dependent basal resistance. Consequently, transportation of the elicitor through the whole plant seems an obvious explanation for the systemic nature of SAR.

How well does SA fit the criteria that define a transported long-distance signal (Box 1)? Intriguingly, SA levels increase in the petioles of pathogen-infected tobacco [16] and cucumber (Cucumis sativus) [17] leaves before the onset of SAR. Removal of pathogen-inoculated leaves before the SA levels increased in the petioles did not, however, impair the induction of SAR in upper leaves of cucumber [18]. Furthermore, SA-deficient tobacco rootstocks expressing SA hydroxylase were still capable of expressing SA hydroxylase were still capable of eliciting volatile emissions in undamaged plants [8], and feeding on roots can induce indirect defences in above-ground parts [9,10]. Thus, the activation of indirect defences also involves regulation by long-distance signals.

Box 1. What qualifies a factor as a transported signal?
Since first discovering systemic plant resistance in response to locally applied stress, scientists have searched for factors that serve as long-distance signals. The topic is still a matter of intensive discussion. Initially, many candidate molecules appeared promising, for example, because their exogenous application or over-expression triggered plant-wide defence, or because mutants in which they were not expressed failed to express resistance. To qualify fully as a long-distance signal, however, a factor must (i) induce a defensive response, (ii) be produced or released at the site of attack, (iii) be translocated from the attacked to the systemic tissue, and (iv) accumulate in the systemic tissue before resistance expression takes place. Several factors failed to meet these criteria, and comparisons among plant species have demonstrated that different signals might occur in different species, or that different signals orchestrate distinct defence responses depending on the nature of the attacking organism. Factors might be derived from the plant, the attacking agent, or both. For the majority of plant species, therefore, the search for the translocated signal(s) is not yet completed.

Long-distance signals in the wound response
Insect feeding inevitably leads to mechanical wounding, and mechanical damage can induce defence responses similar to those stimulated by insect feeding [27,28], at least when the spatio-temporal pattern of the mechanical damage resembles that caused by a consuming insect [29]. Although the insect’s mode of feeding and elicitors in the insect’s regurgitate clearly influence the nature and intensity of the response, the resulting changes in the plant’s physiology are commonly termed ‘wound response’ [30,31].

A classic example of a systemic wound response is PI synthesis in tomato [2]. Biochemical studies identified an 18-amino-acid peptide, systemin, that is released at wound sites upon feeding by chewing herbivores. This peptide is processed from the 200-amino-acid precursor prosystemin. 14C labelling studies showed that systemin is mobile within the plant [32]. Furthermore, tomato plants that overexpressed the prosystemin gene produced PIs constitutively [33]. Nevertheless, grafting experiments — particularly with spr1, a mutant that is defective in the perception of systemin [34] — have revealed that jasmonates, rather than systemin, are responsible for the systemic wound response (Box 3). For a systemic response to local damage, intact systemin signalling is required in the damaged tissue but not in the distal responding tissue. It seems, therefore, that systemin locally induces jasmonates, which are subsequently transported throughout the plant to trigger the systemic response [30,31].

The synthesis of jasmonic acid (JA) and its precursors and derivatives (collectively termed jasmonates) is a classical wound response, which starts with the liberation of linolenic acid from membranes followed by a multi-step conversion into JA by several enzymes [35]. Jasmonates induce a broad spectrum of defensive responses, such as the production of PIs, the release of volatiles, alkaloid production, trichome formation and the secretion of extrafloral nectar. Recently, it was discovered that jasmonates (most likely the JA-amino acid conjugate jasmonoyl–isoleucine) interact with the COI1 (CORONATIN-INSENSITIVE 1) unit of an E3 ubiquitin ligase complex termed SCFCOI1 (Skip/Cullin/F-box–COI1). This event promotes binding of the COI1-unit to JAZ (jasmonate ZIM-domain) proteins, thereby targeting the JAZ proteins for ubiquitination and rapid degradation. Because JAZ proteins are repressors of JA-inducible gene expression, their degradation allows transcription factors to stimulate JA-inducible gene expression [36–38].
Biosynthesis of JA is required at the site of damage, whereas its perception is required in the distal plant parts for a systemic induction of PIs [30]. This shows that jasmonates function as long-distance signals (Box 3). Interestingly, jasmonate signalling is also required for rhizobacteria-induced systemic resistance (ISR) in Arabidopsis, even though ISR is not marked by systemic induction of PIs [39]. Recently, Park and co-workers provided evidence that MeSA, rather than SA, functions as the critical mobile signal [24] (see Figure I).

Salicylic acid (SA) induces resistance to many pathogens [14,78] and accumulates locally in pathogen-infected tissues of different species such as Arabidopsis [79], tobacco [16], and cucumber [17]. SA bleeds out of petioles of pathogen-infected leaves [16,17] and labelling studies have confirmed systemic transport of SA from pathogen-infected leaves [80]. However, SAR can develop before SA levels rise in petioles of infected leaves [19], and accumulation and perception of SA are only critical in the systemic organs [20]. Recently, Park and co-workers provided evidence that MeSA, rather than SA, functions as the critical mobile signal [24] (see Figure I).

Figure I. The role of MeSA as a transported signal in SAR. (a) Grafting experiments with plants in which the SA-binding protein 2 (SABP2) gene is silenced demonstrated that a SABP2-silenced rootstock, which cannot convert MeSA into SA, was still capable of generating a long-distance signal to activate SAR in an upper wild-type scion. (b) Conversely, a SA methyltransferase 1 (SAMT1)-silenced rootstock, in which MeSA production is impaired, was not capable of generating this long-distance signal. (c) Furthermore, a recombinant SABP2 protein was constructed that is affected in SA-induced feedback inhibition of MeSA esterase activity. When this so-called A13L protein was expressed in SABP2-silenced rootstocks, no SAR signal was transported to the upper scion. Hence, SA-induced feedback inhibition of SABP2 is necessary to generate enough MeSA for long-distance transport to distal plant parts, where it is converted by SABP2 into active SA that triggers SAR. Together with the observation that exposure of lower plant parts to MeSA in gas-tight chambers can trigger SAR in non-exposed, upper leaves, these findings demonstrate that MeSA functions as the crucial long-distance signal in tobacco [24]. (d) Expression of SABP2, +, expression of SAR; −, no expression of SAR. (d) The interaction of SAMT1 with SABP2 (which is inhibited by free SA) regulates the synthesis of MeSA from SA in the infected tissue, and the release of SA from the transported MeSA in the systemic tissue. Although MeSA can be transported within the plant, it is possible that under natural circumstances a combination of airborne and vascular transport of MeSA is responsible for long-distance regulation of SAR [20]. (e) Structures of SA and methyl salicylate (MeSA).
Box 3. Jasmonates and long-distance signalling in the wound response

Hundreds of studies have demonstrated that exogenous application of JA induces defensive responses [5,35,81]. Concentrations of JA increase locally in response to tissue damage [4,28]. In tobacco, these locally increased JA levels are followed by enhanced synthesis of nicotine in the roots [4]. Labelling studies using tobacco confirmed that $^{14}$C-JA was transported from leaves to roots, and spatially distributed according to a strictly phloem-based transportation pathway [42]. Also the methyl ester of JA, MeJA, can be transported systematically.

However, $^{11}$C-imaging of MeJA in tobacco revealed distribution patterns of MeJA that could only be explained by transportation through both phloem and xylem accompanied by exchange between non-orthostichous vascular pathways [41]. The high vapour pressure of MeJA as compared to JA might account for the higher mobility of MeJA and opens the possibility of airborne defence signalling. Grafting experiments have confirmed that jasmonates function as crucial mobile signals in the wound response of tomato (see Figure I).

**Figure I.** The role of jasmonates in the systemic wound response. (a) The importance of JA perception in distal organs was demonstrated by the finding that wild-type scions of tomato that had been grafted onto damaged rootstocks of the jasmonate-insensitive 1 mutant (jai1; impaired in the COI1 protein) were still capable of expressing PIs. Conversely, jai1 scions grafted onto a damaged wild-type rootstock failed to activate the defence response. (b) That systemin is not the systematically transported signal becomes obvious from the observation that spr1 rootstocks, which are defective in systemin perception, cannot generate the transported signal, whereas spr1 scions can perceive the signal from wildtype rootstocks and express PIs. (c) Correspondingly, grafts with JA-biosynthetic mutants, such as the suppressor of prosystemin 2 (spr2) mutants [82] and the acyl-coA oxidase1 (acx1) mutant [83], revealed that intact JA synthesis in the rootstock, and not in the scion, is required for a functioning signal transmission [30], which can be amplified by active JA synthesis in the vascular bundles [31] +, expression of PIs; −, no expression of PIs. (d) GLVs (green leaf volatiles) have a double role as local and systemic products of the wound response and as elicitors of further systemic responses, which are mediated by vascular and airborne signalling. Starting from membrane-derived linolenic acid, JA is synthesized locally in the damaged tissue via the octadecanoid pathway, which is activated by systemin or other wounding-associated elicitors. JA can be converted to the more volatile MeJA, and both compounds have been reported to function as transported signals that induce systemic wound responses such as the synthesis of PIs, GLVs and other volatiles. During wound-induced synthesis of JA, GLVs are synthesized and subsequently released from the damaged tissue [63]. At relatively low concentrations, GLVs prime plants for enhanced induction of (Me)JA-dependent defences [57,61], thus they can synergize the systemic wound response to jasmonates. (e) Although structurally not related to JA and MeJA, GLVs such as (E)-2-hexenal, (Z)-3-hexenol and (Z)-3-hexenyl acetate can induce or prime wound-responsive genes and defence responses, pointing to a yet- unidentified mechanism of signal perception and transduction.
isms are behind the induction of systemic resistance in photosynthesising source leaves or in leaves that are non-orthostichous to the stimulated ones [51,52].

What means of transportation could exist if the signal is not only transmitted through the vascular system? JA and SA can be methylated and then become volatile. Furthermore, both MeJA and MeSA are potent inducers of defence responses [20,24,53,54]. It is possible, therefore, that MeSA and MeJA travel by both airborne transport and vascular transport to mediate long-distance induction of resistance. In fact, volatile-mediated induction of resistance in neighbouring plants is a well-documented phenomenon [55–57]. Undoubtedly, ‘long-distance signalling’ does not stop at a plant’s surface, and it has been suggested that volatile compounds also act as within-plant signals [44,58]. Indeed, studies using mechanically damaged sagebrush (Artemisia tridentata) revealed that airflow was necessary for systemic induction of resistance against herbivores, even among branches of the same individual [59]. Similarly, systemic induction of extrafloral nectar secretion by leaves of wild lima bean in response to beetle feeding occurred only when air was moving freely between leaves [57], and volatiles from herbivore-damaged leaves of poplar (Populus deltoides x nigra) augmented defence responses in adjacent leaves. Unfortunately, similar studies that explicitly demonstrate the involvement of volatiles in the within-plant regulation of SAR are lacking. This function is extremely likely, however, as demonstrated by Ingrid Kiefer and Alan Slusarenko’s observation that SAR induction in the Arabidopsis rosette extends beyond the route of assimilate movement along an orthostichy [51], and by the recent finding that MeSA functions as a crucial SAR signal in tobacco [24]. Further studies are required to determine how common the phenomenon of airborne within-plant signalling really is. It seems evident, however, that within-plant signalling by volatiles can synergize the response to vascular signalling molecules to regulate systemically expressed resistance (Figure 1).

Likely candidates in airborne long-distance signalling

Which volatiles are responsible for airborne signalling? Green-leaf volatiles (GLVs) have been associated with induced resistance in intact plants [60–62]. GLVs are C6-compounds that are rapidly released upon tissue damage [63]. Though mainly discussed in the context of insect feeding, GLVs are also active against pathogen infection [64], and this type of direct defence might, in fact, represent their original function [5]. Another airborne candidate is the gaseous hormone ethylene. Ethylene plays a modulating role in plant defensive reactions to pathogens [65]. For expression of SAR, ethylene perception is required locally in the pathogen-infected leaf [66], suggesting that ethylene does not function as a long-distance signal. Ethylene also augmented induced volatile production by maize upon exposure to the GLV (Z)-3-hexenol, but exposure to ethylene alone had no effect [67]. This indicates that ethylene increases the plant’s response to GLVs but is unlikely to serve as a primary signal. In addition to MeSA, MeJA, ethylene and GLVs, (Z)-jasmone can trigger defensive responses having undergone airborne transport. This herbivore-induced volatile activates different sets of genes than those induced by MeJA [68,69], however, suggesting a different mode of action. Correspondingly, (Z)-jasmone failed to induce extrafloral nectar secretion in lima bean, a typical JA-responsive trait that can be boosted by the GLV (Z)-3-hexenyl acetate [62].

Elucidating the mechanisms by which plants perceive volatile signals is a major challenge for future research. MeSA and MeJA can be reverted back into the active plant hormones jasmonic acid and salicylic acid, respectively. By contrast, little is known about the signalling response to GLVs. Details about the molecular perception of GLVs remain unknown. The structural diversity of resistance-inducing GLVs makes a specific receptor-protein-mediated mechanism of perception unlikely. It has been suggested that GLVs that have an α,β-unsaturated carbonyl group can trigger defence through their activity as reactive electrophile species [70], but other GLVs that have been reported to be biologically active lack this motif [62,67,71]. Changes in transmembrane potentials are involved in early signalling events in the cellular response to stress [43], and exposure to several GLVs did change membrane potentials in intact lima bean leaves (Massimo Maffei, pers. comm.). It is therefore tempting to speculate that the dissolving of volatiles in the membranes combined with interactions with membrane proteins, which are similar to the odorant-binding proteins of insects [72], leads to changes in transmembrane potentials and thereby induces gene activity [71].

Benefits of airborne and vascular signalling

Although volatiles have emerged as significant signals in induced resistance, it seems puzzling that plants use airborne regulation if they are already equipped with a sophisticated vascular transportation system. Allowing ‘eavesdropping’ by neighbouring plants might have ecological costs because neighbours usually compete for light, water and soil nutrients. However, airborne signalling has crucial advantages over vascular signalling.

First, airborne signalling overcomes restrictions resulting from the plant’s orthostichy [44,73]. Plant enemies often move from one leaf to the adjacent one, but directly adjacent leaves usually lack direct vascular connections. Vascular restrictions become even more problematic in large plants, such as trees, shrubs and vines, in which spatially neighbouring leaves often originate from separate branches. Furthermore, phloem fluxes are usually directed towards non-photosynthesizing tissues, whereas transport through xylem vessels follows the upwards transpiration flow. Even signals that are transported through both xylem and phloem hardly reach mature leaves that are older than the signal-producing leaf. By contrast, volatile-mediated induction of extrafloral nectar was observed in mature lima bean leaves that inserted below the induced leaves [57], and hence, airborne signals appear to have important functions in smaller plants too.

Second, volatile signal molecules can reach distal plant parts faster than compounds that are transported through vascular tissues. It is probably no coincidence that, of all classes of herbivore-induced volatiles, GLVs are most frequently described as active airborne signals. GLVs are in part released from pre-
Figure 1. Model of a two-step regulation system of systemic resistance by airborne and vascular long-distance signals. (a) Upon local attack by insects (left) or pathogens (right), plants rapidly emit airborne signals, such as green leaf volatiles, methyl jasmonate or methylsalicylic acid, which can sensitize distal plant parts for a second vascular signal, such as jasmonic acid or salicylic acid. (b) Kinetics of systemically expressed induced resistance. Rapid airborne signals (minutes – hours) trigger priming (priming phase – green symbols) and relatively little defence expression. These signals are followed by a second vascular signal (days – weeks; defence phase, red symbols) that boosts the induced defence expression. (c) Relationship between induced defence expression (defence amplitude) and dosage of airborne (green arrow) and vascular (red arrow) long-distance signals. Airborne signals can trigger induced defence at relatively high concentrations, but at lower concentrations, they prime for an enhanced defence induction by the vascular signal.

existing pools [63] and are generally synthesized from early precursors of JA extremely early during the wound response (Box 3). Fast signalling is an important advantage, as speed of defence activation is crucially important for the effectiveness of resistance to pathogens and insects.

The velocity and direction of within-plant signalling by volatiles depends on different biotic and abiotic factors, such as wind speed and direction, and on various plant characteristics, such as the presence of leaf hairs or a wax layer and probably many more factors. Further studies will be required to determine the extent by which locally
released volatiles affect systemic defence expression, and how the chemical properties of volatile signals affect the spatiotemporal pattern of systemic induction of resistance.

**Vascular and airborne signalling interact for optimal resistance expression**

The combination of internal and external pathways to achieve systemic defence activation provides plants with an interesting additional option for fine-tuning systemic resistance in yet-undamaged tissues. Relatively high concentrations of volatiles can trigger defences directly [74], but exposure to lower concentrations of volatiles, which fail to activate defences to their full extent, can still affect resistance. Such an alternative mode of action can be explained by sensitization of the plant’s defence arsenal [57,61,75]. This so-called priming for defence has been associated with various induced resistance phenomena [76]. Priming prepares the plant to respond more rapidly and/or effectively to subsequent attack, and it can be activated by much lower concentrations of the resistance-inducing signal than those required to induce active defences fully [69,77]. Hence, primed plants show no enhanced defence activity, but they respond much faster or more strongly to wounding or infection than unprimed plants. Although the priming and induction of direct defence constitute different mechanisms of protection, both result in a phenotypically similar resistance to pathogens and herbivores. Furthermore, some induced-resistance phenomena are based on a combination of both mechanisms. For instance, the induction of pathogen-induced SAR activates some defence-related genes, whereas other defensive genes become primed for augmented induction to subsequent pathogen attack [76].

Given the fact that volatiles diffuse in the air and are dispersed by eddy currents, it is plausible that resistance-inducing volatiles are often diluted to priming concentrations. In fact, self-priming by herbivore-induced volatiles has been described in the context of airborne within-plant signalling [57,73]. This suggests a two-step regulatory system in which airborne and vascular signals interact, probably to varying extents, to achieve an optimized orchestration of the systemic defence response (Figure 1). In this model, airborne signals prime distal tissues to respond more efficiently to vascular signals or direct attack. Such a two-step regulation provides additional fine-tuning of the systemic resistance response, while preventing defence reactions to false alarm signals. Self-priming by airborne signals prepares the systemic tissues for a rapid response, but full activation of costly defence mechanisms is allowed only after confirmation by the vascular long-distance signal (Figure 1).

**Conclusions and outlook**

JA and SA are crucial signals in the regulation of induced resistance to herbivores and pathogens, and have been shown to move in the vascular system. Their volatile derivatives, MeJA and MeSA, however, have been put forward as more likely candidates to mediate long-distance regulation of induced resistance. MeJA and MeSA, as well as GLVs, are released from damaged leaves and are mobile in the gas phase. The major benefit of a within-plant signalling by volatiles is that they overcome the spatial and temporal restrictions of the vascular system. Particularly at lower doses, airborne signals can prime for defence, thus preparing the distal organs for a more effective defence response rather than fully inducing them. Priming provides an additional dimension to the delicate balance between defence expression and competing processes, such as growth and reproduction.

Dosage-dependent effects also strongly reduce a putative ecological cost of external signalling: ‘eavesdropping’ by neighbours. Volatile-mediated signalling is most effective over relatively short distances, and the probability that the leaf nearest to an attacked one belongs to the same plant is relatively high. As a result, the chance that eavesdropping by competing plants becomes a quantitatively relevant problem remains low.

Plants combine vascular and airborne transport of the long-distance signals, providing a carefully balanced systemic defence reaction to local attack by herbivores and pathogens. Although both transportation pathways can co-exist within the same plant, their relative contribution to the overall response is likely to depend on the plant’s life history and anatomy, and on the nature of the attacking organism. Therefore, different plant species with different life styles will have to be considered if we are to gain a better understanding of the strategies by which plants optimize the induction of systemic resistance. The same holds true for the nature of locally and systemically functioning signals, which might differ amongst plant species.

**Acknowledgements**

We thank Christian Kost, Peter Bakker, Leendert C. (Kees) van Loon and three anonymous referees for valuable comments on earlier versions of this manuscript. Research activities of J.T. are supported by a BBBSRC Institute Career Path Fellowship (no. BB/E023959/1), M.H. gratefully acknowledges financial support from the German Research Foundation (DFG-grant He 31694–2) and from Consejo Nacional de Ciencia y Tecnología (CONACYT, México).

**References**


Li, C. et al. (2005) Role of β-oxidation in jasmonate biosynthesis and systemic wound signaling in tomato. Plant Cell 17, 971–986