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The ecological concept of costs of induced systemic resistance (ISR)

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Abstract

Plant defence is thought to provide benefits for the defended plants. Theoretical concepts must, therefore, explain why there is variation in defensive traits, which naively might be assumed to be present constitutively in fixed high amounts. Explanations are mainly based on the assumption of fitness costs. Investment in defence is thought to reduce the fitness of plants in enemy-free environments. Fitness costs often result from allocation costs, i.e. allocation of limited resources to defence, which then cannot be used for growth or other fitness-relevant processes. This theoretical concept can provide a useful tool for the interpretation of induced plant responses against pathogens, named induced systemic (or systemic acquired) resistance (ISR or SAR). Phenotypic plasticity, leading to induced responses, might have evolved mainly to reduce costs, since investment in defence is restricted to situations actually requiring defence. ISR can incur allocation costs and other, indirect costs, which ultimately may lead to fitness costs. Evolution of any defensive trait depends on both what a plant ideally 'should do' and what it actually 'is able to do'. Costs of defence constrain its expression. This might have important influences on the evolution of plant defensive traits, as well as on the exploitation of natural defences in agricultural crop protection.

Abbreviations: GDBH – growth differentiation balance hypothesis; JA – jasmonic acid; PR – pathogenesis related; SA – salicylic acid; SAR – systemic acquired resistance; ISR – induced systemic resistance.

Introduction

Plants have evolved different strategies to defend themselves against herbivores and pathogens. Many of these strategies involve the induction of defensive traits, which therefore appear in high amounts or intensities only when plants suffer attack (Karban and Baldwin, 1997; Agrawal et al., 1999b; Tollrian and Harvell, 1999). Most studies on induced plant responses against herbivores have so far been conducted in an ecological context (Karban and Baldwin, 1997; Baldwin and Preston, 1999; Paul et al., 2000). In contrast, work on induced responses against pathogens has concentrated on (i) the specificity of interactions (Jackson and Taylor, 1996; Somssich and Hahlbrock, 1998), (ii) the underlying signalling pathways (Ryals et al., 1994; Hunt et al., 1996; Hunt and Ryals, 1996; Schneider et al., 1996; van Loon, 1997), and (iii) how these findings might be used for agricultural purposes. Few studies have focused on ecological aspects of induced defences against pathogens (Heil, 1999; but see Hatcher, 1995). Some attempts have recently been made to integrate plant defence against herbivores and pathogens (Hatcher and Paul, 2000; Paul et al., 2000), but no unifying theory has been proposed for the ecological and evolutionary interpretation of both defensive strategies (Heil, 1999; 2000a).

In this review, I will argue that the ecological concept of costs, which is widely used in the context of anti-herbivore defence, can also provide a useful framework

to interpret findings on induced plant responses against pathogens. Studies on ISR that directly hint at relevant physiological costs will be reviewed along with evidence from studies in related fields such as, e.g., induced defence against herbivores, or direct defensive responses against pathogens. A much better understanding of the evolutionary and ecological constraints on induced responses is needed when these are to be used as effective methods in agricultural plant protection programmes.

Terminology

The debate during the 'First International Symposium on Induced Resistance to Plant Diseases' held in Corfu in May 2000 revealed that much confusion exists as consequence of the use of different terms such as induced systemic resistance (ISR), systemic induced resistance (SIR), systemic acquired resistance (SAR), induced plant immunity, induced defence against pathogens, and so on. These terms are used synonymously by some authors, while others use them to describe different phenomena.

I follow the definitions given by Karban and Baldwin (1997) for induced defence against herbivores in naming all morphological or biochemical changes in plants that occur after some kind of attack or elicitor treatment 'induced *responses*', while those induced responses that affect the performance of the attackers negatively are named 'induced *resistance*'. The term 'induced *defences*' is used exclusively to denote those responses which have positive consequences for the fitness of the responding plants. While many studies have demonstrated that systemically induced responses of plants to pathogen attack successfully restrict further infections by the inducing as well as other pathogens (Ryals et al., 1994; Hammerschmidt and Kuć, 1995; Hoffland et al., 1996; Jackson and Taylor, 1996; Schneider et al., 1996; Anfoka and Buchenauer, 1997; and literature cited therein), no study has demonstrated so far that this response has a positive effect on plant fitness in a natural environment. Therefore, to date it has to be named *resistance* in the sense of Karban and Baldwin (1997). This term matches the original one, 'systemic acquired resistance', which had been chosen by Ross (1961).

The term '*acquired*' seems to be the older one, and some arguments have been presented to favour this one over the term '*induced*' (van Loon, 1997). Yet, all studies on responses of plants against insects use the term '*induced*', and an initial induction by a challenging infection or elicitor treatment is required for this specific form of systemic resistance against pathogens, too. This review will try to demonstrate that one framework can be used for the interpretation of plant defence against both pathogens and herbivores. A unified terminology helps to point to the similarities in both responses and to encourage further integration of concepts. I therefore name the whole set of changes by which plants respond to an initial infection, or elicitor treatment, in becoming systemically resistant against pathogen attack *induced systemic resistance* (ISR), and use this term synonymously with the other widespread term which is used to describe the same phenomenon, *systemic acquired resistance* (SAR).

Evolutionary problems related to ISR and the concept of fitness costs

Induced systemic resistance in plants is a defensive mechanism that can be induced by a broad spectrum of pathogens, against many of which it is then effective (Hammerschmidt and Kuć, 1995). These main traits are already difficult to understand in an evolutionary or ecological context (Heil, 1999). What are the benefits of a systemically induced and unspecific resistance which relies on a 'challenging' infection? Why is ISR induced and not constitutive?

The concept of fitness costs has been used to explain why defensive traits that benefit plants are still subject to variation in natural populations, instead of being fixed genetically at a maximum level. This concept is also a useful tool to explain the occurrence of inducible defences, which are expressed only under certain circumstances. Stated simply, defence is assumed to incur costs to the plants, i.e., better defended plants are predicted to have a lower fitness as compared to less well defended plants, when both are compared under enemy-free conditions that prevent the defence from having any beneficial effects (Simms and Rausher, 1987; Simms and Fritz, 1990). Thus, defence should be established only when it is required. Although this explanation is widely used, surprisingly few studies have been conducted to measure fitness costs of any form of resistance, and only a restricted number of studies have demonstrated such costs (Bergelson and Purrington, 1996; but see Baldwin, 1998 and Agrawal et al., 1999a for the case of induced defence against herbivores, and Bergelson et al., 1996 for constitutive herbicide resistance). This has forced several authors

to propose alternative explanations as to why defence might be inducible (Agrawal and Karban, 1999).

Fitness costs may, but do not need to, be a consequence of allocation costs, which are addressed in most theoretical (e.g., Coley et al., 1985; Herms and Mattson, 1992) as well as empirical (for an overview, see e.g. Gershenzon, 1994) studies on costs of defences. Other consequences of defence, which might negatively affect plant fitness, can result from autotoxic properties of the defensive traits, or from negative influences on other organisms that affect plant fitness positively, i.e., mutualists. Moreover, even allocation costs translate to fitness costs only when the availability of resources is limited. Thus, they are not likely to appear under laboratory conditions, which are mostly characterised by optimised growing conditions and the absence of competition.

These problems may have prevented the allocation costs of ISR from becoming visible in most studies conducted so far, and only few studies have shown fitness costs of any form of pathogen resistance (but see Smedegaard-Petersen and Stolen, 1981; Smedegaard-Petersen and Tolstrup, 1985; Godard et al., 1999; Heil et al., 2000). However, many reported data, as well as theoretical considerations, are consistent with the assumption that ISR can incur relevant costs. This remains true for all three forms of costs to be discussed here, namely (i) allocation costs, (ii) costs resulting from autotoxicity of resistance traits, and (iii) costs resulting from negative influences on the plants' mutualists.

Allocation costs

All main steps of the signalling pathway, leading to ISR, require gene expression (Buell, 1999) and thus consume resources. This remains true for the initial steps characterised by the hypersensitive response (Baillieul et al., 1995; Kuć, 1995; Hunt and Ryals, 1996; Hammerschmidt and Nicholson, 1999), for the production of molecules such as salicylic acid (SA), which seem to be involved in signal transduction (Métraux et al., 1990; Mauch-Mani and Métraux, 1998; Hammerschmidt and Smith-Becker, 1999; Cameron, 2000), and for production of pathogenesis-related (PR) proteins (van Loon, 1997; van Loon and van Strien, 1999), phytoalexins (Kuć, 1995; Smith, 1996; Kuć, 1997; Hammerschmidt, 1999b; Hammerschmidt and Nicholson, 1999; van Loon and van Strien, 1999), and cell wall material (Nicholson and Hammerschmidt, 1992; Hammerschmidt and Nicholson, 1999), all of which form part of the plants' defensive responses. Consequently, Kuć (1995) has discussed whether stunted plants and lower productivity – that appear after application of elicitors of phytoalexin accumulation, or in plants which constitutively accumulate phytoalexins – may be caused by 'a marked diversion of energy and carbon precursors from vital processes'.

Autotoxicity

Some of the molecules involved in the signalling pathway (such as SA and the reactive oxygen species involved in the 'oxidative burst' which occurs at the beginning of the hypersensitive response, see Baker and Orlandi, 1995; Lamb and Dixon, 1997; Hammerschmidt and Nicholson, 1999) have already been demonstrated to have, or are likely to have, autotoxic effects (e.g., Rasmussen et al., 1991). Moreover, the hypersensitive response itself – which leads to controlled cell death at the site of infection – has at least some negative effects on the defending plant and thus presents a form of autotoxicity.

Effects on mutualists

No study has so far demonstrated convincingly negative effects of ISR on any plant mutualist, but the low specificity of this defence and the high number of mutualistic interactions with microorganisms make it very likely that some of these might be negatively affected by ISR. Most plants have evolved mutualistic interactions with root-colonising fungi and bacteria, and many grasses have mutualistic, endosymbiotic fungi (Clay, 1990). It is already known that several interactions between mutualists infecting roots (i.e., mycorrhizal fungi and root nodulating bacteria) and the plants' induced resistance against pathogens do occur. Most of these interactions are characterised by an increase in the plants' defensive systems at the onset of the mutualistic infection (see, e.g., Dumas-Gaudot et al., 1996; Van Wees et al., 1997; Cordier et al., 1998; van Loon et al., 1998; Pieterse and Van Loon, 1999; Ruiz-Lozano et al., 1999). For example, infecting fungi must overcome plant defensive responses which occur during the establishment of mycorrhiza (Kapulnik et al., 1996), and establishment of mycorrhiza can be delayed in plants which constitutively express some (but not other) PR proteins (Vierheilig et al., 1995). Moreover,

chemical induction of pathogen resistance in alfalfa (*Medicago sativa*) and faba beans (*Vicia faba*) can lead to a reduced size and number of root nodules (Martínez-Abarca et al., 1998; Heil, 2000b). As a consequence, ISR might incur indirect costs by negatively affecting the plants' mutualistic interactions.

These studies are very preliminary, and most plant mutualists can be expected to be adapted to cope with the natural defensive responses of their host plant. Further studies are needed to determine whether findings derived from studies with artificial induction of ISR (Martínez-Abarca et al., 1998; Heil, 2000b) have any relevance for naturally elicited ISR. Yet, negative effects on mutualistic microorganisms might be a serious problem in agricultural systems which rely on chemical induction of ISR.

Moreover, some pathogenesis-related proteins have been reported to be induced as a consequence of attack by herbivorous insects (Mayer et al., 1996; Inbar et al., 1998; 1999), and these PR-proteins (e.g., chitinases) seem to have negative effects on herbivores (Broadway et al., 1998; Inbar et al., 1998; 1999). It has, therefore, already been hypothesised that chitinases could have negative effects on mutualistic insects, too (Heil et al., 1999).

Applying the concept of costs to ISR

In the following section, the concept of costs is used to formulate predictions on how different traits of ISR should be expressed. Several of these predictions have already been made by Herms and Mattson (1992) in the growth differentiation balance-hypothesis (GDBH), which is formulated in the context of constitutive defence against herbivores.

The GDBH is based on the main assumption that there exists competition between metabolic pathways relevant for growth and pathways involved in 'differentiation', with the latter term comprising all processes which are commonly called 'secondary metabolism': 'The GDBH of plant defence is premised upon a physiological trade-off between growth and differentiation processes. The trade-off between growth and defence exists because secondary metabolism and structural reinforcement are physiologically constrained in dividing and enlarging cells, and because they divert resources from the production of new leaf area' (Herms and Mattson, 1992). This trade-off clearly constrains the ability of plants to defend themselves against pathogen attack.

Besides the main assumption of metabolic competition, several clear predictions on when and how defensive traits should be expressed can be drawn from this concept: (i) plants cannot produce unlimited amounts of defensive compounds; (ii) young, developing plant parts are hard to defend by 'expensive' forms of defence which consume limited resources; (iii) negative interactions should become more obvious when plants suffer from a shortage of limiting resources such as, nutrients; (iv) plants should avoid redundant defences; (v) the production of defensive compounds can cause fitness costs.

Metabolic competition

Support for a 'metabolic competition' between primary metabolism and ISR comes from studies on potato and on parsley cell cultures, where Rubisco levels (potato) and the expression of histone-encoding genes (parsley) were strongly reduced after pathogen infection or elicitor treatment (Longemann et al., 1995; Somssich and Hahlbrock, 1998). 'The metabolic significance of gene repression concomitant with gene activation during pathogen defence is probably associated with the downregulation of all disposable cellular activities' (Somssich and Hahlbrock, 1998).

Additional studies have been conducted on differential gene induction and repression in response to methyljasmonate, an important elicitor of wound- or herbivory-induced responses (for reviews, see Reinbothe et al., 1994; Creelman and Mullet, 1997). For example, Weidhase et al. (1987) reported the selective repression of several of those proteins which are present before jasmonate treatment. Reinbothe and co-workers have used barley to investigate responses of protein biosynthesis involved in production of, e.g., Rubisco and chlorophyll a/b binding proteins, to methyljasmonate treatment. Downregulation was found at the level of gene transcription (Reinbothe et al., 1994), in posttranscriptional transcript modifications (Reinbothe et al., 1993a), and in the stability of the transcripts (Reinbothe et al., 1993b). Finally, translation of mRNA encoding these proteins was also downregulated (Müller-Uri et al., 1988; Reinbothe et al., 1993b,c). Similar results have been presented in other studies (Wasternack et al., 1998).

Most of these results consider direct regulation processes rather than a simple metabolic 'competition' for limited resources. However, the question arises why biosynthesis involved in the production of vitally

important proteins is downregulated at all. An answer can be derived from the assumption of resource limitations, which have to be coped with by controlled shifts in metabolic resource flows from primary metabolism to defence. Corresponding to this interpretation and to that given by Somssich and Hahlbrock (1998) (see above), Weidhase et al. (1987) and Reinbothe et al. (1994) proposed that the amino acids released by the proteolytic degradation of photosynthetic proteins are re-utilised for synthesis of defence proteins. These effects can finally lead to chlorophyll loss and Rubisco degradation (Weidhase et al., 1987; Reinbothe et al., 1994) and therewith form important costs of methyljasmonate-induced defence, which are likely to occur in a comparable way in ISR.

Limited amounts of defence

Two main signalling pathways are involved in the induced plant responses against pathogens and herbivores. When plants are compromised in the total amounts of defence they can produce, negative interactions between these two responses are to be expected to occur, at least under limiting conditions. Several studies have already been conducted on reciprocal effects of induced responses against herbivores and pathogens. Some studies have provided hints on beneficial reciprocal influences. For example, herbivore-caused damage of tomato can increase resistance against the phytopathogen, *Pseudomonas syringae* (Stout et al., 1998b) and JA-inducible genes can be activated by pathogen attack (Schweizer et al., 1997). JA can induce phytoalexins and defence-related cell wall constituents (Nojiri et al., 1996; Creelman and Mullet, 1997; Wasternack and Parthier, 1997), and methyljasmonate can induce several PR-proteins (Xu et al., 1994). Treatment with both elicitors can protect tomato and potato against *Phytophtora infestans* (Cohen et al., 1993). Nicotine, which is induced as a response to herbivory (Baldwin, 1988a,b), can have negative effects on both herbivores and bacteria (Krischik et al., 1991). For overviews on reciprocally induced defences against pathogens and herbivores see Hatcher (1995) and Hatcher and Paul (2000).

However, most published data point to some kind of ‘signalling conflicts’ or ‘trade-offs’ (Bostock, 1999; Felton et al., 1999). The most thoroughly investigated system in this context is again tomato, *Lycopersicon esculentum*, for which both molecular and ecological studies have been conducted (for a review, see Thaler, 1999). These studies have demonstrated that chemical induction of ISR decreases the plants’ ability to express wound-inducible proteinase-inhibitors (Doares et al., 1995; Fidantsef et al., 1999). Correspondingly, treating leaves with a chemical elicitor of ISR increased their suitability for herbivorous caterpillars (Thaler et al., 1997; Stout et al., 1999). SA-treatment has been reported to inhibit wound- and JA-induced responses in the same plant (Stout et al., 1998c), and application of JA reduced the efficacy of chemical ISR elicitors (Thaler et al., 1997). Most probably, action of SA or related substances inhibits synthesis of JA (Penacortes et al., 1993).

There are striking theoretical problems in attempting to explain why an attack by one form of attacker has a predictive value for a reduced probability to face attacks by other organisms (Paul et al., 2000). The ‘signalling conflicts’ or ‘trade-offs’ between induced responses against pathogens and herbivores are, therefore, hard to explain when only the beneficial effects of the response are considered. However, evolution of any trait does not depend only on what a plant ideally ‘should do’, but also on factors constraining what it actually ‘is able to do’. The costs of defence provide an easy explanation for the negative interactions between both defensive pathways. Plants might simply be compromised in the total amounts of defensive compounds which can be produced during a limited time interval.

Defence and developmental stage

Few studies have investigated whether the ability of a plant, or part of a plant, to produce PR proteins or other ISR components depends on its age. The induction of PR-protein transcripts in tobacco leaf discs, floated on solutions of different sugars, depended strongly on the developmental stage of the leaves (Herbers et al., 1996), with older leaves being highly inducible, while the same concentration of sugars failed to elicit a clear response in leaves which had not fully unfolded. Herbers et al. (1996) used source–sink relationships for an interpretation and supposed that source leaves, which normally export sugars, suffer from a metabolic disturbance when facing external soluble sugars, while sink leaves are normally tailored to import external sugars, which therefore represent no metabolic disturbance. However, according to the hypothesis of Herms and Mattson (1992), young, still growing leaves may just be unable to produce PR-proteins simply because their whole metabolic apparatus is needed for the

biosynthesis of growth-relevant proteins. In contrast, other studies found that young, still growing leaves on intact plants are perfectly capable of producing PR proteins (van Loon, 1985). Further studies, focused directly on a possible age-dependency of production of ISR-relevant components, should be conducted to determine under which conditions ISR can be compromised in young, developing plant parts.

Reports on a further form of resistance against pathogens are in line with the hypothesis formulated above. Dependency of a resistance trait on leaf developmental stage which is comparable to that reported by Herbers et al. (1996) has been reported by Leisner et al. (1992; 1993) for resistance against long-distance movement of cauliflower mosaic virus in leaves of turnip and Arabidopsis. Correspondingly, the age-related resistance of Arabidopsis occurs only in older, pre-senescent plants (Cameron, 2000). Similar temporal patterns of resistance in plants, or parts of plants, against pathogens increasing with developmental stage have been reported for maize (Moose and Sisco, 1994). The expression of these constitutive forms of defence may have evolved mainly according to ecological and evolutionary demands, i.e., better defence of plants which have reached the flowering stage. However, the described patterns are clearly consistent with the predictions of Herms and Mattson (1992) and Coley et al. (1985) that young plants, which, under natural conditions, are forced to grow very fast to compete successful with other plants, may be strongly compromised in their ability to defend themselves against pathogens or herbivores.

Resource availability

The ability of a plant to defend itself against enemies should depend on resource availability (Coley et al., 1985), and allocation costs of defence should have stronger effects on growth under limiting conditions. Most studies on this topic have been conduced in the context of induced defence against herbivores. For example, nitrogen supply can have strong influences of several inducible and constitutive defences of tomato (Stout et al., 1998a), and higher costs of induced nicotine production were found in plants growing on soils with lower nitrogen content (Baldwin et al., 1998) or under competitive conditions (Baldwin and Hamilton, 2000). The effect of chemical induction of ISR on growth and seed set was studied in wheat plants which were cultivated in pots under competitive conditions and at different levels of nitrogen supply (Heil et al., 2000). Treating wheat plants with the chemical ISR elicitor BION® (benzo (1,2,3) thiadiazole-7-carbothioic acid S-methyl ester) under pathogen-free conditions resulted in a suppression of plant growth and a reduced yield, as compared to untreated plants. This suppression was most pronounced in plants suffering from a strong shortage of nitrogen (Heil et al., 2000). Comparable results have been obtained for wheat cultivated under agricultural field conditions by Stadnik and Buchenauer (1999), who reported that BION® treatment resulted in no positive effect on yield, most probably since the benefits of reduced infection rates were counterbalanced by the costs of ISR induction.

Redundant defences

Little is known about whether a plants' own chemical defence against pathogens is reduced in cases where its function is performed by an alternative mechanism. Chitinase activities in so-called myrmecophytic plants, which are well defended against herbivores and pathogenic fungi by the action of mutalistic ants, were lower than in related plants which have no ant-defenders (Heil et al., 1999; submitted), supporting the prediction that redundant defences are reduced to avoid superfluous costs. Corresponding evidence comes from studies focused on the chemical anti-herbivore defence of myrmecophytic plants (Rehr et al., 1973; Seigler and Ebinger, 1987).

Fitness costs

Studies on fitness costs of any resistance trait are scarce (but see Bergelson et al., 1996; Baldwin, 1998; Agrawal, 1999). However, as early as 1981, Smedegaard-Petersen and Stolen (1981) reported that a successful resistance response by barley against powdery mildew requires energy, and finally leads to a reduction in grain yield. It was later supposed that this energy requirement may be a reason for 'the limiting effect of disease resistance on yield' (Smedegaard-Petersen and Tolstrup, 1985). Negative effects on plant growth and flowering time of an overexpression of defence-related peroxidase in tobacco, and positive effects on the same fitness-relevant parameters of an underexpression of the same enzyme, have been reported by Lagrimini et al. (1997). Longemann et al. (1995) demonstrated reduced growth in parsley cell cultures that had been induced to express resistance

genes by infection or fungal elicitors: an effect that clearly would lead to reduced plant growth and fitness costs if it translates to the whole-plant level. Studies on chemically-induced ISR in wheat have shown a reduced seed set and, therewith, significant fitness costs of treatment with an ISR elicitor conducted under otherwise limiting conditions (Heil et al., 2000).

Conclusions and directions for future research

Most studies reported here have not been designed to test the hypothesis that resistance of plants against their enemies does cause costs. Nevertheless, they have revealed results which match predictions based on this hypothesis and thereby clearly corroborate it. Plants may evolve to reduce the costs of a distinct resistance trait (Bergelson and Purrington, 1996; Agrawal and Karban, 1999), and several factors that induce resistance may simultaneously have positive effects on the plants which counterbalance or even overweigh the costs of resistance. The latter seems to be the case when so-called plant-growth promoting bacteria induce pathogen resistance (van Loon et al., 1998).

The exact form of costs of resistance against pathogens, and whether they translate into evolutionary relevant fitness costs, does depend on both the type of defensive trait and the environmental conditions. These costs, which do not necessarily appear under all conditions, may nevertheless have strong influences on the evolution of defence, and thus on the plants' ability to express defence under certain circumstances.

This review strongly concentrated on the aspect of costs of ISR to demonstrate that allocation costs or other, indirect, forms of costs, that finally can translate to fitness costs of resistance, may have strong influences on the quality and quantity, and on the spatial and temporal patterns, of induced plant resistance against pathogens. This does not imply that other factors, which might influence the evolution of inducible defences (Agrawal and Karban, 1999), can be excluded from further debate. This is especially true for the argument that adaptation of pathogens or herbivores to a defensive trait is much more difficult when facing defences which are inducible and thus vary spatially and temporarily within populations and even within individual plants.

Even most arguments presented by Agrawal and Karban (1999) assume that plants cannot produce unlimited amounts of defence and thus are based on the theory that defence does cause costs. The value of the argument that only few studies have so far demonstrated costs is biased severely by the general psychological problem that aspects of costs, such as reduced growth and seed set, or suppressed gene activity, are widely regarded as 'negative' results and thus are less likely to be published. This especially holds for applied studies. While much work has been done on the induction of gene expression after challenging infections or elicitor treatment, fewer studies have been reported on potential gene suppression or down-regulation (but see examples reviewed by Somssich and Hahlbrock, 1998). Correspondingly, 'stunted' phenotypes resulting from transformation experiments which have been conducted to produce defence-overexpressing plants would provide important information on the costs of that particular defence. They are, however, often regarded as some kind of failed experiments. These observations are seldom quantified and even more seldom published (personal communications from several authors). The down-regulation of a gene is a result as good as its induction, and suppressive effects on plant growth are valuable results, too. Much more information on 'the other side of the coin', i.e., constraints that have influences on the expression of defence traits, would be important for both forms of research, basic research interested in the physiology, ecology and evolution of defence, and applied research devoted to the development of tools for agricultural plant protection.

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