Trends in
Plant Science

CellPress

Opinion

The Induced Resistance Lexicon: Do's and Don'ts

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To be protected from biological threats, plants have evolved an immune system comprising constitutive and inducible defenses. For example, upon perception of certain stimuli, plants can develop a conditioned state of enhanced defensive capacity against upcoming pathogens and pests, resulting in a phenotype called 'induced resistance' (IR). To tackle the confusing lexicon currently used in the IR field, we propose a widely applicable code of practice concerning the terminology and description of IR phenotypes using two main phenotypical aspects: local versus systemic resistance, and direct versus primed defense responses. Our general framework aims to improve uniformity and consistency in future scientific communication, which should help to avoid further misinterpretations and facilitate the accessibility and impact of this research field.

Highlights

Upon perception of certain stimuli, plants can develop a conditioned state of enhanced defensive capacity against upcoming pathogens and pests, resulting in a phenotype called 'induced resistance' (IR).

Scientific communication in the IR research domain is flawed with inconsistent use of various conceptualizations and terms.

Researchers working on non-model organisms and/or less-studied plant tissues – which often make use of distinct natural defense mechanisms – are struggling to choose the correct term for their observations.

Different biological and chemical IR stimuli tend to induce resistance through various pathways and hence terminology can and should not be linked to underlying mechanisms.

A History of Terminology Confusion

'**Induced resistance**' (**IR**; see Glossary) is a phenotype in which plants, once stimulated by certain pathogens, pests, beneficial microbes, chemical agents, physical wounding, or herbivory, exhibit lowered susceptibility for future **challenges** when compared with naïve control plants [1]. Apart from the observation of lowered susceptibility to pests and diseases, IR phenotypes can be confirmed at the level of defense responses, for instance, by augmented production of reactive oxygen species, enforced callose deposition, and altered epigenomes, transcriptomes, proteomes, metabolomes, etc.. These effects are similar to effects stimulated by pathogen-associated molecular patterns (PAMPs), damage-associated molecular patterns (DAMPs), or pathogen effectors.

Since the first recognition of IR phenotypes in the early 1900s [2–5], several terms and concepts have been introduced to describe IR. Terms introduced in the early days, such as 'plant immunization' [2], were continued to be used by some [6], while others introduced a variety of new terms. Sequeira [7] was among the first who raised the issue of a confusing terminology in this field. Certain terms were (re-)defined at the First International Symposium on Induced Resistance to Plant Diseases in 2000 [8]. Nevertheless, in 2006, Tuzun [9] published a manuscript entitled 'Terminology related to induced systemic resistance: Incorrect use of synonyms may lead to a scientific dilemma by misleading interpretation of results', illustrating that scientific communication concerning IR was still hampered by an inadequate terminology. More than a decade later, scientific research has led to a better molecular understanding of IR phenotypes and their underlying mechanisms. Unfortunately, this has not been accompanied by a uniform terminology and/or consistently used conceptualizations. Here, by tackling three points of confusion, we aim to disentangle the main concepts within the IR research field and to clarify the IR terminology in the light of recent findings.

Point 1 – A Clarification on Some IR Terms

Ross [10] introduced the term '**systemic acquired resistance**' (**SAR**) to refer to the reduced susceptibility to viruses in leaves of tobacco (*Nicotiana tabacum*) plants as a result of previous

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viral infections in distant leaves. However, in later studies by Ross and colleagues, several other terms were used to describe the same phenotype: 'systemic resistance' [11], 'systemic induced resistance' [12], and 'induced resistance' [13]. Nowadays, the term SAR is still being used and its definition generally encompasses the following elements: typically induced by a local inoculation with a necrotizing pathogen, predominantly mediated by the phytohormone salicylic acid (SA) and probably by *N*-hydroxypipecolic acid as well, and often associated with at least some accumulation of pathogenesis-related (PR) proteins [14,15]. In the 1990s, non-pathogenic plant growth-promoting rhizobacteria and fungi (PGPR and PGPF, respectively) were found to trigger similar IR phenotypes [16–19]. Pieterse *et al.* [20] demonstrated in the model plant arabidopsis (*Arabidopsis thaliana*) that this happened independently of SA and *PR* genes, but rather was based on the jasmonate (JA) and ethylene (ET) pathways. The term '**induced systemic resistance**' (**ISR**) was adopted to differentiate this IR phenomenon from SAR [21], although the two types of IR were considered as phenotypically similar [22]. Indeed, although the underlying defense mechanisms and spectrum of effectiveness may differ for specific SAR and ISR phenotypes, the observable defensive effects are often similar.

Over time, a dichotomy seems to be introduced with respect to the use of the terms SAR and ISR. Based on their predominant hormonal regulators in arabidopsis and the microorganisms initially found to induce these phenotypes, SAR and ISR are now often considered as fundamentally different IR phenotypes, despite earlier agreements for these terms to be used synonymously [8]. At the same time, ISR is also used as a general term to refer to systemic forms of IR, as initially agreed on [8]. Moreover, chemicals, non-proteinaceous amino acids, physical injury, volatile organic compounds, etc. have been found to elicit IR as well [1,23] but as non-biotic agents or actions, these stimuli do not seem to fit in the strict definitions of ISR and SAR.

To encompass all known IR phenomena in a uniform terminology, we encourage the scientific community to use 'induced resistance' (IR) as an umbrella term and '**IR stimulus**' as general reference for the evoking agent or action. ISR should be used when there is convincing evidence that upon local contact with an IR stimulus, endogenous signals are spread systemically to stimulate defense-related processes in essentially all plant parts. In the absence of such evidence, or when the stimulus itself is systemically distributed in the plant – as shown for exogenously applied synthetic β-aminobutyric acid (BABA) [24,25], and as currently being studied for migrating endophytes (V. Pastor, unpublished) – the more general term IR is recommended. Thus, although this is often done in contemporary literature, the term ISR should not be limited only to IR phenotypes triggered by PGPR or PGPF, while phytohormones other than JA and ET can be mediators of ISR. Because the term SAR has strictly been defined over the years (*vide supra*), we propose that these specific ISR phenotypes can still be referred to as 'SAR phenotypes'.

We make a plea to consider two main aspects of IR phenotypes when selecting an adequate terminology. The first is the predominant occurrence of **local resistance** or **systemic resistance** upon IR establishment. Correspondingly, this trait forms the first axis in our general framework to characterize IR phenotypes (Figure 1, Key Figure). Importantly, a strong local resistance does not exclude systemic effects. Indeed, as shown on the x-axis of Figure 1, we consider nearly all known IR phenotypes to be an outcome of both local and systemic resistance. Nevertheless, only when biologically relevant resistance is observed consistently throughout the entire plant, the terms ISR or SAR are appropriate.

Point 2 – IR: The Sum of Direct and Primed Defense Responses

Over the past decades, scientific progress revealed that the establishment of IR does not always depend on, and sometimes even is not associated with, a strong **direct defense response**

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Key Figure

A General Framework to Characterize Induced Resistance (IR) Phenotypes in Terms of Local/Systemic Resistance (x-Axis) and Direct/Primed Defense Responses (y-Axis)

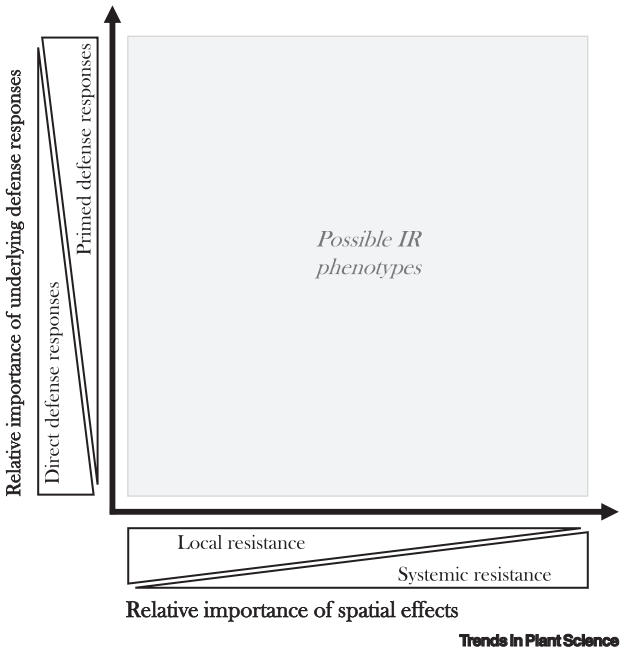

Figure 1. All IR phenotypes can be considered to be the result of both directly induced defense responses and primed defense responses, as well as of local resistance and systemic resistance. Importantly, the ratios of importance for these parameters can vary depending on many parameters such as the IR stimulus, its concentration or intensity, time point of analysis, plant under study, age of the plant, plant tissue under study, pathogen under study, analyzed read-out (see Figure 2).

upon application of an IR stimulus [1,26,27]. While some genes, enzymes, or pathways are not affected directly upon treatment with an IR stimulus, they can get activated earlier, stronger, and/or faster upon later challenges when compared with non-IR plants. Such an enhanced capacity to mobilize infection-induced cellular defense responses is referred to as '(defense) priming' [26]. Important to notice that **primed defense responses** do rely on various mechanisms that are activated directly upon IR stimulation, such as the accumulation of dormant signaling proteins, transcription factors and hormones, epigenetic alterations, and/or increased levels of receptors and coreceptors [1,14,28,29]. Nevertheless, the main outcome of defense priming is a boosted defense response that is only activated upon a later challenge. The relative contribution of direct and primed defense responses is the second main characteristic that we deem essential to describe IR phenotypes. Correspondingly, this facet forms the y-axis in the framework illustrated in Figure 1.

For a while, the primed defense responses were considered as the most relevant aspects of IR phenotypes [14,30], resulting in IR definitions being somehow adapted in various works [27,31–36]. Today it is clear that the IR phenotype is associated with both direct induction of

Glossary

Challenge: inoculation with a pathogen or pest after an IR phenotype has been established by a certain IR stimulus. If on purpose, it is typically to investigate the resulting level of resistance or the molecular effects on the affected defense response.
Direct defense responses: defense responses that are induced, locally or systemically, upon contact with the IR stimulus. Hence, these responses can be detected prior to any challenge.
Induced resistance (IR): reduced disease susceptibility of a plant in response to stimulation by a pathogen, insect herbivore or wounding, beneficial microbe, or chemical agent. IR is often effective against a broad spectrum of pests, pathogens, and sometimes even abiotic stresses.
Induced systemic resistance (ISR): type of IR that leads to resistance in plant tissues distant from those that were initially in contact with the stimulating biological or chemical agent. Should not be limited to IR phenotypes that are systemically triggered by plant growth-promoting rhizobacteria/fungi and are associated with JA/ET.
IR stimulus: any agent or action that leads to the establishment of an IR phenotype in the affected plant.
Local resistance: resistance observed in the plant tissue that was initially in contact to the IR stimulus.
Primed defense responses: defense responses that, because of defense priming, are activated earlier, stronger, and/or faster in IR plants upon subsequent challenge with a pathogen, pest, or insect herbivory, but not in the absence of an attacker. Although these defense response modulations are only observable upon later challenges, defense priming is associated with alterations which take place directly upon IR stimulation, and which make the plant to be primed for enhanced defense (e.g., epigenetic alterations, increased receptor presence, accumulation of dormant proteins and/or hormones, and other, currently unidentified, effects).
Systemic acquired resistance (SAR): specific type of ISR that classically, but not necessarily (i) leads to resistance in systemic tissues upon local infection by a necrotizing pathogen, (ii) is associated with at least some *PR* gene expression/PR protein accumulation, and (iii) is controlled mainly by the action

defense responses – which can be transient or long-lasting – and primed defense responses [1,29,37]. Nevertheless, some stimuli mainly work through direct activation of plant defense genes or metabolites, while others seem to work predominantly via defense priming. Undoubtedly, many other features are also relevant for the description and characterization of IR phenotypes, such as durability, plant species or cultivar, or mode-of-action. However, with respect to the selection of a proper terminology, the axes shown in Figure 1 are considered as the most relevant. As an illustration of how to use and interpret this graphical representation, some well-studied IR phenotypes have been characterized via this framework (Figure 2).

of SA and, probably, by *N*-hydroxypipecolic acid.
Systemic resistance: resistance observed in plant parts distant to those that were initially in contact with the IR stimulus, or even in the entire plant.

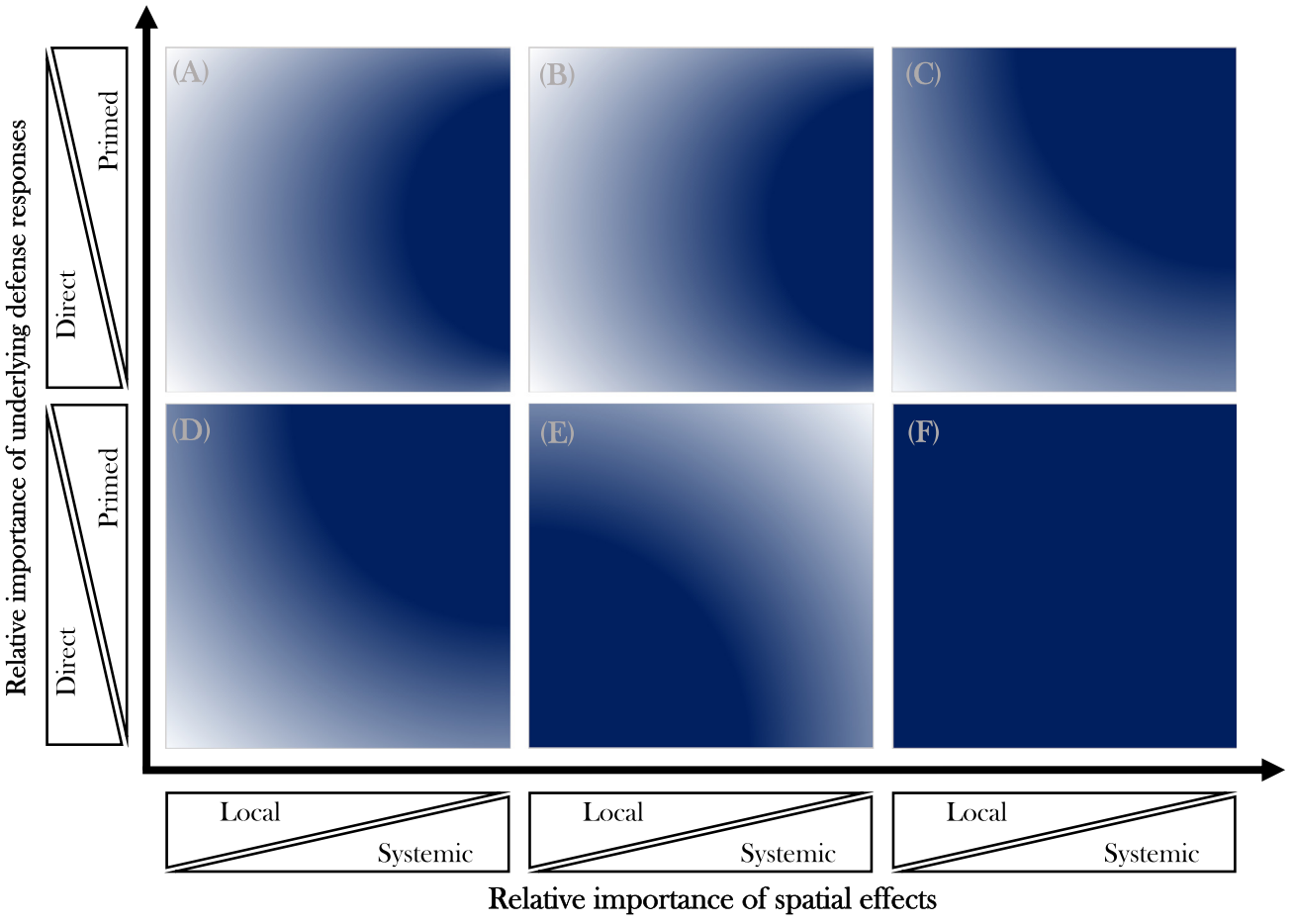

Figure 2. An Illustration of a Set of Well-studied Induced Resistance (IR) Phenotypes Using the Framework Presented in Figure 1. Color shading represents the relative importance of local versus systemic resistance, and direct versus primed defense responses for the indicated types of IR. (A) Pathogen-induced systemic acquired resistance (SAR) [15]: this phenotype is characterized – by definition – by a systemic form of IR and includes both direct and primed defense responses. (B) Ascorbate oxidase (AO)-induced systemic resistance (ISR) in rice (*Oryza sativa*) against *Meloidogyne graminicola* [62]: both primed and direct defense responses have been associated with this IR phenotype. Although panels (A) and (B) have a similar shading, AO-ISR is fundamentally different from SAR, as the stimulus is not a necrotizing pathogen, and the phenotype does not depend on salicylic acid but rather on jasmonic acid and ethylene [62]; (C) *Pseudomonas simiae* WCS417-ISR in arabidopsis [63]: for this IR phenotype nearly all observed defense responses have been shown to be primed and systemic; (D) and (E) β-aminobutyric acid (BABA)-IR upon application of low and high doses, respectively [27]: low BABA doses lead to systemically primed defense responses, while high BABA doses lead to directly activated responses mainly in the treated plant parts. These two panels clearly illustrate that for one specific IR stimulus, the underlying mechanisms can be different; (F) methyl-jasmonate (MeJA) IR [64–70]: via direct activation of defense responses [64–69] and via priming [68–70], MeJA has been described to stimulate plant resistance both systemically [64,66] and locally [64,65,69].

Point 3 – IR: What Is in a Name? And What Is Not?

To refer to chemicals that trigger IR, a plethora of terms has been introduced, mostly not accompanied by a clear definition, characterization, or thorough differentiation from others: 'resistance activator' [38], 'plant (defense) activator' [39–41], 'synthetic inducer of defense responses' [42], 'defense elicitor' [43], 'inducer of plant immunity' [44], '(plant) resistance inducer' [45,46], 'disease resistance compound' [47], 'elicitor' [48,49], 'inducer' [50], 'SAR inducer' [21], 'plant strengthener' [51,52], 'priming-inducing chemical' [31], 'priming agent' [53], etc. As these terms seem to be tacitly associated with specific underlying mechanisms – 'priming agents' may be thought of as mainly leading to primed defense responses, which can be conceived as opposed to the mode-of-actions of 'plant defense activators' – the concurrent use of these terms can be confusing to newcomers in the field and non-experts.

Apart from being confusing, a presumed association of these terms with specific underpinning effects can be considered as incorrect as well. Even for well-studied IR stimuli, the underlying mechanisms can differ depending on multiple parameters. Indeed, how IR is manifested relies on a multidependent and only partially characterized network [54,55]. Whether or not a specific IR phenotype is associated with direct and/or primed defense responses depends not only on the stimulus, but also on multiple experimental, environmental, and spatiotemporal parameters, as well as on the read-out in question (Box 1). Indeed, it has been shown that for BABA, the direct/and or primed activation of certain underlying mechanisms depends on the plant species under study [55], the applied BABA concentration [27], and the necrotrophic or biotrophic lifestyle of the pathogen that is battled [56,57], while indole-3-carboxylic acid (I3CA) has been shown to work differently depending on the age of the plant [58]. Here we propose to use the neutral term 'IR stimulus' to refer to any action or agent that induces IR.

Correspondingly, we recommend the use of a general 'stimulus-phenotype' terminology (e.g., 'BABA-IR', 'PGPR-ISR', 'chemical X-IR', '*Pseudomonas syringae*-SAR') that should not – and cannot – be associated with any underlying mechanism. Indeed, 'BABA-IR' or 'I3CA-IR' should merely be used to refer to a phenotype of enhanced defensive capacity, triggered upon treatment with BABA or I3CA. By not referring to underlying mechanisms, the hereby-presented terminology can be easily and correctly applicable for studies executed on non-model organisms or on less-studied tissues, which often employ distinct natural defense mechanisms. For the sake of clarity, the aforementioned terminology can optionally be extended by the pathogen for which an increased resistance is observed, as well as the plant host (e.g., 'BABA-IR against *Botrytis cinerea* in *Solanum lycopersicum*' or '*Pseudomonas syringae*-SAR against *Hyaloperonospora arabidopsidis* in arabidopsis'). However, adding a pathogen for which an increased resistance is observed is no necessity, as we consider IR

Box 1. Fitness Costs Associated with IR

An important consequence of the assumptions and conceptualizations described in this work lies in the evaluation of the cost-efficiency of IR stimuli. In some reports, predominant induction of direct plant defenses has been linked to evident fitness costs [27], whereas IR phenotypes mainly based on priming have been shown to be associated with lower fitness costs [27,59]. However, this is no ever-valid correlation as there are noticeable exceptions. For instance, the chemical compound diproline was identified as an IR stimulus based on direct induction of defense marker genes in rice (*Oryza sativa*) [60]. Nevertheless, diproline-IR was not associated with any obvious negative effects on rice growth or yield, even when plants were repetitively treated during their lifespan [60]. Indeed, a variety of parameters can influence the relative importance of primed and direct defense responses. Moreover, whether an intense and long-lasting induction of defense pathways, a mainly primed defense response, or an intermediate form is optimal depends on the cost–benefit balance in a given environment [29]. The model in Figure 1 in the main text is consistent with this notion: a specific localization on the y-axis in Figure 1 cannot be interpreted in terms of long-term physiological effects and/or allocation costs. Hereto, IR phenotypes should undergo relevant ecological assessments, as underlined by Martinez-Medina *et al.* [61].

also a proper term to refer to phenotypes in which defense mechanisms are enhanced, regardless of whether effective resistance against a specific pathogen has been experimentally confirmed.

Concluding Remarks

By discussing some potential pitfalls within the IR lexicon and clarifying a consensus point-of-view concerning the current terminology and conceptualizations, we hope to stimulate a more clear, consistent, and unambiguous scientific communication in this field. The aforementioned suggestions, in combination with the general framework presented in Figure 1, may contribute hereto as they can help to characterize and describe scientific observations in a more uniform manner. Although we encourage authors to use the hereby-presented terminology and conceptualization, we realize that because of novel findings (see Outstanding Questions) or alternative term usage in related scientific fields, specific terms or concepts might remain to be/become used differently. That is why, in general, we strongly encourage a well-evaluated terminology, provided with a thorough elaboration on the intended meaning for the various terms and concepts being used. We believe that our propositions can make the already existing IR vocabulary transparent and easily applicable for contemporary research, in which an expanding range of IR stimuli, plants, tissues, pathogens, and diseases is being studied. As novel introductions in this field can be facilitated by a consistent and widely supported lexicon, we are convinced that the general notion and appreciation for IR will expand, ultimately extending the reach and impact of the reported observations for plant sciences, agriculture, and beyond.

Outstanding Questions

How durable is IR in plants within and over generations?

Will IR be a sustainable and effective strategy for crop protection in future agriculture?

How can IR be integrated within other pest and disease management strategies?

What is the role of epigenetics in the regulation of IR?

How did IR develop in the evolutionary context of the plant immune system?

Are IR phenotypes that act through different molecular mechanisms (e.g., established by different stimuli) additive?

Declaration of Interests

The authors have no interests to declare.

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