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Title page

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The induced resistance lexicon: do's and don'ts

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Keywords

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Abstract

To be protected from biological threats, plants have evolved an immune system comprising constitutive and inducible defenses. Plants can, for example, upon perception of certain triggers, develop a conditioned state of enhanced defensive capacity against upcoming pathogens and pests, resulting in a phenotype called 'induced resistance' (IR). Although IR has been studied intensely over the last decades, scientific communication has been complicated by inconsistent use of various conceptualizations and terms. Here, we propose a widely applicable code of practice concerning the description of IR phenotypes. This code is based on a general framework and aims to improve uniformity and consistency in future communication. This should help to avoid further misinterpretations and facilitate the accessibility and impact of this research field.

Main text

A history of terminology confusion

'Induced resistance' (**IR**; see Glossary) is a phenotype in which plants, once triggered by certain pathogens, pests, beneficial microbes, chemical agents, physical wounding or herbivory, exhibit enhanced resistance against future **challenges** when compared to naïve control plants [1]. Indeed, IR phenotypes can be confirmed on the level of defense responses, for instance by augmented production of reactive oxygen species, enforced callose deposition, altered epigenomes, transcriptomes, proteomes or metabolomes, *et cetera*. Since the first recognition of IR phenotypes in the early 1900s [2–5], several terms and concepts have been introduced to describe IR. Sequeira (1983) was among the first who raised the issue of a confusing terminology in the field [6]. Certain terms were (re-)defined at the First International Symposium on Induced Resistance to Plant Diseases in 2000 [7]. Nevertheless, in 2006, Tuzun 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" [8], illustrating that scientific communication was still hampered by an inadequate terminology. More than a decade later, profound scientific research has led to a better molecular understanding of IR phenotypes and their underlying mechanisms. Unfortunately, this has not led to the end of the Babylonian confusion of tongues. With this article, we aim to disentangle several concepts within the IR research field by tackling four points of confusion and thus to clarify IR terminology in the light of recent findings.

Point 1) A clarification on some IR terms.

Ross (1961) introduced the term '**systemic acquired resistance**' (**SAR**) to refer to the reduced susceptibility to viruses in tobacco leaves, as a consequence of previous viral infections of distant leaves [9]. 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 the accumulation of pathogenesis-related (PR) proteins [10, 11]. In the 1990s, non-pathogenic plant growth-promoting

rhizobacteria and fungi (PGPR and PGPF, respectively) were found to trigger similar IR phenotypes [12–15]. Pieterse *et al.* (1996) demonstrated in the model plant *Arabidopsis thaliana* that this happened independently of SA and *PR* genes, but rather was based on the jasmonate (JA) and ethylene (ET) pathways [16]. The term '**induced systemic resistance'** (**ISR**) was adopted to differentiate this IR phenomenon from SAR [17], although the two types of IR were considered as phenotypically similar [18].

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 and triggering microorganisms, SAR and ISR are now often considered as fundamentally different IR phenotypes, despite earlier agreements for these terms to be used synonymously [7]. However, ISR has also been used to refer to a systemic form of IR, as initially agreed on [7]. Additionally, chemicals, non-proteinaceous amino acids, physical wounding, volatile organic compounds, *et cetera* have also been found to elicit IR [1, 19]. As non-biotic agents or actions, these triggers do not seem to fit in the strict definitions of ISR and SAR.

To encompass all possible IR phenomena in a uniform terminology, we encourage the scientific community to use 'induced resistance' as an umbrella term and '**(IR) trigger**' as general reference for the evoking element. ISR should be used when there is convincing evidence that upon local contact with an IR trigger, endogenous signals are spread systemically to stimulate defense-related processes in essentially all plant parts. In the absence of such evidence, or when the trigger itself is systemically distributed in the plant – as it was recently shown to be the case for exogenously applied synthetic β-aminobutyric acid (BABA) [20], and as currently being studied for migrating endophytes (V. Pastor, unpublished) – the more general term IR is recommended. Thus, although this is done often in contemporary literature, the term ISR should not be limited only to IR phenotypes that are triggered by PGPR or PGPF. Because the term SAR has strictly been defined over the years (see Glossary), we propose that these specific ISR phenotypes can still be referred to as 'SAR phenotypes'. However, despite the fact that chemical IR triggers have been referred to as 'SAR inducers' [17], we consider the general terms I(S)R more appropriate for phenotypes that are triggered by chemical compounds, as many chemicals rely on fundamentally different mode-of-actions when compared to pathogen-induced SAR.

Our proposed guidelines to discriminate IR, ISR and SAR should facilitate an initial selection for the most appropriate term to describe an observed IR phenotype. Hence, the spatial aspect (local - systemic) forms the first axis in our general framework to characterize IR phenotypes (Figure 1, Key Figure). Importantly, a relatively strong **local resistance** (i.e. a resistance observed in the tissue initially brought into contact with the IR trigger), does not exclude any systemic effects. Indeed, as shown on the X-axis of Figure 1, we consider nearly all IR phenotypes to be an outcome of both local and **systemic resistance**. Only when biologically relevant resistance is observed throughout the entire plant in a consistent manner, 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** upon application of a trigger [1, 21, 22]. While some genes, enzymes or pathways are not affected directly upon treatment with an IR trigger, it has been found that once IR is established in plants, they can get activated earlier, stronger and/or faster upon later challenges when compared to non-IR plants. Such an enhanced capacity to mobilize infection-induced cellular defense responses is referred to as '(defense) priming' [21]. Important to notice is that **primed defense responses** do rely on various mechanisms that are activated directly upon IR triggering, such as the accumulation of dormant signaling proteins, transcription factors and hormones, epigenetic alterations and/or increased levels of receptors [1, 10, 23, 24]. Nevertheless, the main outcome of defense priming is a boosted defense response which is only activated upon a later challenge.

For a while, the primed defense responses were considered as the most relevant effects for IR phenotypes [10, 25], with IR definitions being somehow adapted in various works [22, 26–31]. However, one should pay attention that the IR phenotype is associated with both direct induction of defense responses – which can be transient or long-lasting – and primed defense responses – which only become detectable after subsequent challenges [1, 24, 32]. While some triggers mainly work through direct activation of plant defense genes or metabolites, others seem to work predominantly via defense priming. As a second main parameter to describe IR phenotypes, the relative importance of direct and primed defense responses forms the Y-axis in the

framework illustrated in Figure 1. For the sake of clarity, some well-studied IR phenotypes were characterized using this framework in Figure 2.

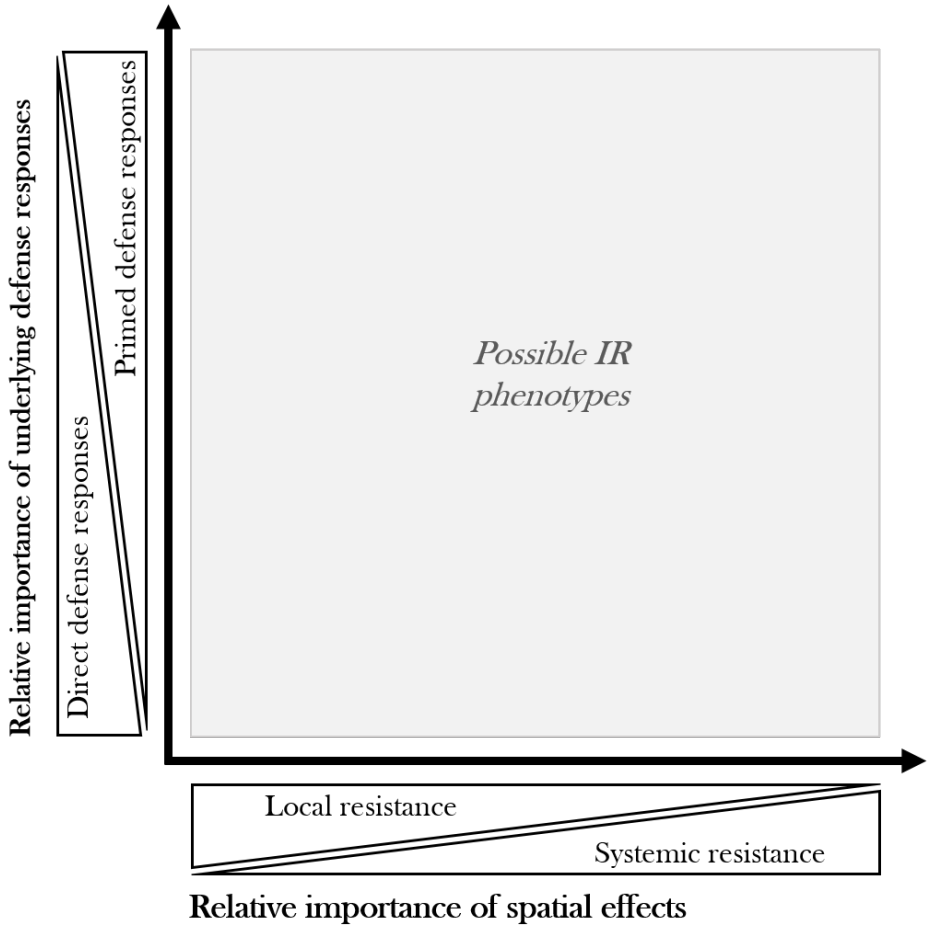

Figure 1 Key Figure. A general framework to characterize IR phenotypes in terms of local/systemic resistance (X-axis), and direct/primed defense responses (Y-axis). We consider all IR phenotypes to occupy the entire square region. Thus, all IR phenotypes can be considered to be the result of both directly 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 trigger, its concentration, time point of analysis, plant under study, age of the plant, plant tissue under study, pathogen under study, analyzed read-out, *et cetera* (see Figure 2). Following from this multi-dependency, it is obvious that specific IR triggers cannot be associated with specific and invariable underlying molecular mechanisms that result in the observed IR state.

Point 3) IR: what's in a name? And what's not?

To refer to chemicals which trigger IR, a plethora of terms has been introduced, mostly not accompanied by a clear definition, characterization or thorough differentiation from others: 'resistance activator' [33], 'plant (defense) activator' [34–36], 'synthetic inducer of defense

responses' [37], 'defense elicitor' [38], 'inducer of plant immunity' [39], '(plant) resistance inducer' [40, 41], 'disease resistance compound' [42], 'elicitor' [43, 44], 'inducer' [45], 'SAR inducer' [17], 'plant strengthener' [46, 47], 'priming-inducing chemical' [26], 'priming agent' [48], *et cetera*. As these terms tacitly might be 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 parallel use of these terms can be confusing to newcomers in the field. Even for well-studied IR triggers, the underlying mechanisms may differ based on multiple parameters, making separate IR subcategories undesirable (*vide infra*).

That is why we discourage the use of apparently meaningless – and potentially misleading – terms for (chemical) IR triggers, as well as the creation of additional IR subcategories in which IR triggers and/or phenotypes are considered to be strictly associated with specific underlying mechanisms. Indeed, how IR is manifested relies on a multi-dependent and only partially characterized network [49, 50]. Whether or not a specific case of IR establishment leads to a (detectable) alteration of a sector in this defense network, depends not only on the trigger, but on multiple experimental, environmental and spatio-temporal parameters, as well as on the read-out in question. For example, it has been shown that for BABA, the activation of certain underlying mechanisms depends on the plant species under study [50], the applied concentration [22] and the necrotrophic or biotrophic lifestyle of the pathogen that is battled [51, 52], while indole-3-carboxylic acid (I3CA) has been shown to work differently depending on the age of the treated plant [53]. Hence, we recommend a general "trigger-phenotype" terminology (e.g. 'BABA-IR', 'PGPR-ISR', 'chemical X-IR', '*Pseudomonas syringae*-SAR', *et cetera*) that should not 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, in which often distinct natural defense mechanisms are being observed.

For the sake of clarity, the above-mentioned terminology may be extended by adding the pathogen for which an increased resistance is observed, as well as the plant host: e.g. 'BABA-IR against *Botrytis cinerea* in tomato' 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 also a proper term to refer to phenotypes in which defense mechanisms are positively affected, regardless of effective resistance against a specific pathogen has been experimentally confirmed.

Point 4) An ecological assessment of fitness-related costs.

Predominant induction of direct plant defenses has been linked to more pronounced fitness costs [22], whereas IR phenotypes mainly based on priming are typically associated with lower fitness costs [22, 54]. However, this is not an ever-valid correlation as there are noticeable exceptions. For instance, the chemical compound diproline was identified as a potential IR trigger, just because it leads to a direct upregulation of specific defense marker genes in rice [55]. Nevertheless, diproline-IR in rice was not associated with any obvious negative effects on rice growth or yield, even when plants were repetitively treated during their entire lifespan [55]. With IR phenotypes being considered to be a result of direct and primed defense responses depending on many variables (see Points 2 and 3), a specific localization on the (Y-axis in the) graph in Figure 1, should not be interpreted in terms of long-term physiological effects and/or allocation costs. Nevertheless, IR phenotypes should be characterized profoundly through relevant ecological assessments, as described by Martinez-Medina *et al.* (2016) [56]. Indeed, 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 [24].

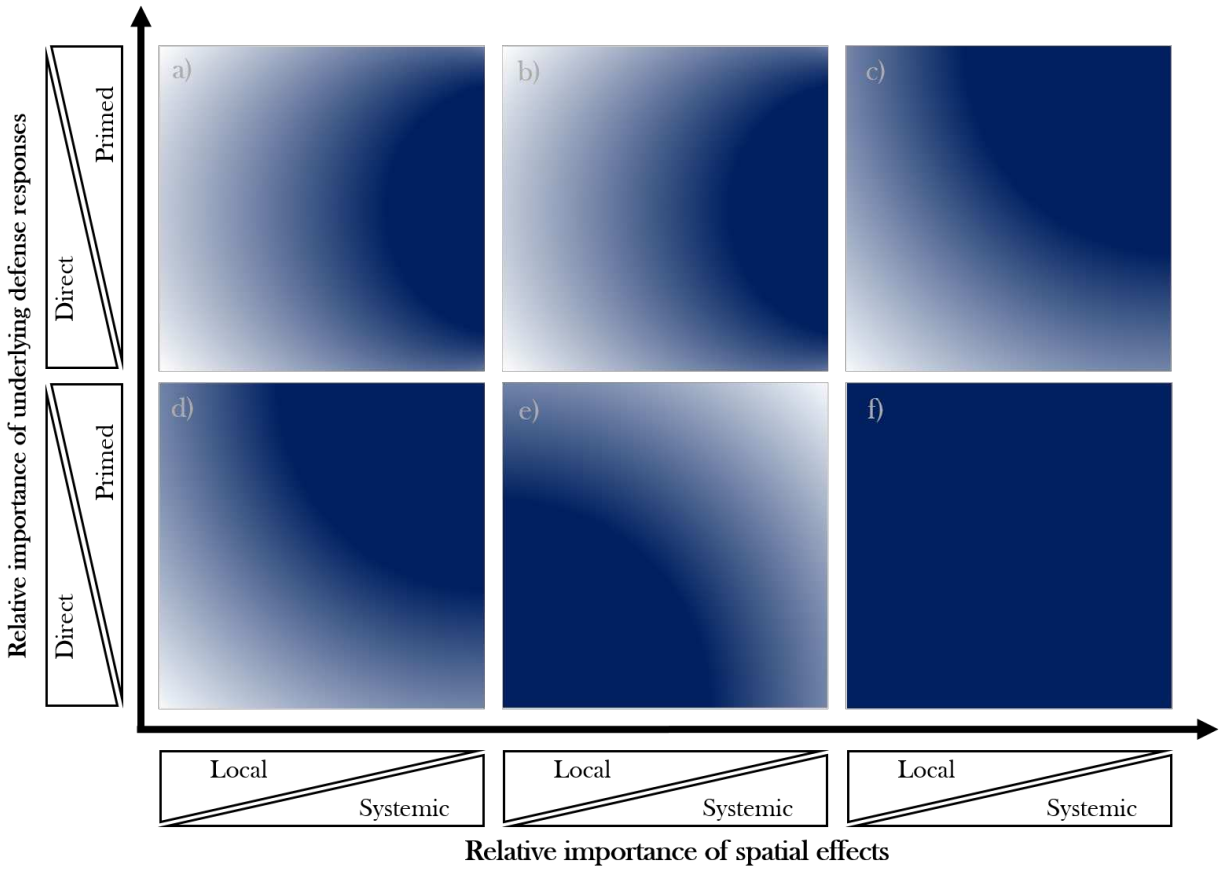

Figure 2. An illustration of a set of well-studied IR phenotypes using the framework presented in Figure 1. Color shading represents the relative importance of local versus systemic resistance, and direct versus primed defense responses for the indicated types of IR. a) pathogen-induced SAR [11]: this form is characterized – by definition – by a systemic form of IR, and consists of both direct and primed defense responses; b) ascorbate oxidase (AO)-ISR in rice against *Meloidogyne graminicola* [57]: both primed and direct defense responses have been identified for this IR phenotype. Although panel a and panel b have a similar shading, AO-ISR is fundamentally different from SAR, as the trigger is not a necrotizing pathogen, and the phenotype does not depend on salicylic acid but rather on jasmonic acid and ethylene; c) *Pseudomonas simiae* WCS417-ISR in *Arabidopsis* [C. M. J. Pieterse, unpublished]: for this IR phenotype nearly all observed defense responses have been shown to be primed and systemically; d) and e) BABA-IR upon application of low and high doses, respectively [22]: 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 trigger, the underlying mechanisms can be different; f) Methyl-jasmonate (MeJa)-IR [58-64]: via direct activation of defense responses [58-63] and via priming [62-64], MeJa has been described to activate plant resistance both systemically [58, 60] as locally [58, 59, 63].

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 abovementioned suggestions, in combination with the general framework presented in Figure 1, may contribute hereto as they can help to characterize and describe future observations in a more uniform manner. Although we encourage authors to use the hereby-presented terminology and conceptualization, we realize that because of novel future findings 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 transparently and easily applicable for contemporary research, in which an expanding range of IR triggers, plants, tissues and pathogens 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, also among non-specialists, ultimately extending the reach and impact of the reported observations for agriculture and beyond.

Glossary *(450 words max., now +/- 350)*

- **Challenge**: inoculation with a pathogen or pest after an IR phenotype has been established by a certain trigger. If on purpose, typically to investigate the resulting level of resistance or the molecular effects on the affected defense response.
- **Direct defense responses**: defense responses that are immediately induced, locally or systemically, upon contact with the IR trigger. Hence, these responses can be detected prior to any challenge.
- **Induced resistance (IR)**: enhanced disease resistance 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 distant plant tissues than those that were brought in contact with the triggering biological or chemical agent. Should not be limited only to IR phenotypes that are systemically triggered by plant growth-promoting rhizobacteria/fungi.
- **Local resistance**: resistance observed in the plant tissue that was brought into contact with the IR trigger.
- **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 the defense response modulations are only observable upon later challenges, priming is associated with metabolic alterations which take place directly upon IR triggering, and which make the plant to be primed for enhanced defense (e.g. epigenetic alterations, increased levels of receptor activity, 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 1) leads to resistance in systemic tissues upon local infection by a necrotizing pathogen, 2) is associated with *PR* gene expression/PR protein accumulation, and 3) is controlled mainly by the action of SA and, probably, by N-

hydroxypipecolic acid. Should not be used for IR phenotypes triggered by chemical compounds, as the more general terms IR or ISR are recommended hereto.

- **Systemic resistance**: resistance observed in plants parts distant to those that were brought into contact with the IR trigger, or even in the entire plant.
- **(IR) Trigger:** any agent or action that leads to the establishment of the IR phenotype in the affected plant.

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