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Review

Is modulating virus virulence by induced systemic resistance realistic?

Franco Faoro[a,b,*], Franco Gozzo[c]

[a] Department of Agricultural and Environmental Sciences, University of Milan, Via Celoria 2, 20133 Milano, Italy
[b] CNR, Institute for Sustainable Plant Protection, Strada delle Cacce 73, 10135 Turin, Italy
[c] Department of Food, Environmental and Nutritional Sciences, Section of Chemistry and Biomolecular Sciences, University of Milano, Via Celoria 2, 20133 Milano, Italy

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ABSTRACT

Induction of plant resistance, either achieved by chemicals (systemic acquired resistance, SAR) or by rhizobacteria (induced systemic resistance, ISR) is a possible and/or complementary alternative to manage virus infections in crops. SAR mechanisms operating against viruses are diverse, depending on the pathosystem, and may inhibit virus replication as well as cell-to-cell and long-distance movement. Inhibition is often mediated by salicylic acid with the involvement of alternative oxidase and reactive oxygen species. However, salicylate may also stimulate a separate downstream pathway, leading to the induction of an additional mechanism, based on RNA-dependent RNA polymerase 1-mediated RNA silencing. Thus, SAR and RNA silencing would closely cooperate in the defence against virus infection. Despite tremendous recent progress in the knowledge of SAR mechanisms, only a few compounds, including benzothiadiazole and chitosan have been shown to reduce the severity of systemic virus disease in controlled environment and, more modestly, in open field. Finally, ISR induction, has proved to be a promising strategy to control virus disease, particularly by seed bacterization with a mixture of plant growth-promoting rhizobacteria. However, the use of any of these treatments should be integrated with cultivation practices that reduce vector pressure by the use of insecticides, or by Bt crops.

Contents

* Corresponding author at: Department of Agricultural and Environmental Sciences, University of Milan, Via Celoria 2, 20133 Milano, Italy. Tel.: +39 0250316786; fax: +39 0250316781.
E-mail address: franco.faoro@unimi.it (F. Faoro).

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1. Introduction

Virus diseases account for about half of crop epidemics [1]. Their control is mainly based on prevention by using genetically resistant plants and by vector eradication, the latter implying high costs and heavy environmental impact. In fact, no effective antiviral compounds are available at present for field application [2]. Unfortunately, genetic resistance, either achieved by the conventional introduction of Mendelian genes [3] or by genetic engineering [4] can be overcome by viruses because of their genomic plasticity, and as it is very often based on gene-for-gene interaction [5], recently reviewed [2,6]. The possibility of inducing resistance in plants against viruses with chemicals or beneficial microorganisms deserves even more interest than that against bacteria and fungi, because there are alternative workable strategies for these organisms.

Conventionally, there are two forms of induced systemic resistance in plants. One is activated by numerous strains of plant-growth-promoting rhizobacteria (induced systemic resistance, ISR) and depends on hormones such as jasmonic acid and ethylene [7]. The best known systemic acquired resistance (SAR) is induced following a primary infection, particularly by pathogens inducing hypersensitive response [8], and, for practical purposes, can be mimicked by the use of chemicals [9,10]. In Arabidopsis biologically activated SAR involves the expression of a number of up-regulated genes, some of which are SA-independent while others are functionally associated with SA-depending defences and pathogenic-related (PR) proteins. Most of all up-regulated genes have been found to be up-regulated also by exogenous application of chemicals, such as benzothiadiazole [11]. Nevertheless, also in view of the relevance that diverse pathosystems may have on genes activation, the systemic resistance induced by exogenous chemical inducers cannot be necessarily authenticated as SAR on the basis of the mere expression of a handful of genes. An updated insight into SAR mechanisms, associated pathways, metabolites and epigenetic modifications has been highlighted in recent reviews [12–15].

Both ISR and SAR are a condition of alerted defence that provides long-lasting, broad spectrum resistance, which is effective against different pathogens, including viruses [15]. The exploitation of ISR against virus diseases has been less investigated [16–19]. There is conflicting evidence whether ISR is really effective against these pathogens [20].

In this review we pursue two main tasks:

(1) To expose evidence of the mechanisms responsible for preventing virus infections under controlled conditions. This task is mainly covered by the biochemical induction of SAR and the plant innate immune response involving RNA silencing, a predominant mode of basal plant defence against viruses [21].
(2) To ascertain the limits of defence against viral diseases in open field, induced by application of the so far available chemical inducers and biocontrol agents. In order to answer the question in the title, virus–host compatible interactions have been selected as the most difficult to control.

2. Systemic acquired resistance against plant viruses: an uncertain fight against many targets

2.1. Hypersensitive response-inducing viruses

The phenomenology of SAR was demonstrated for the first time in 1961 by using the pathosystem tobacco mosaic virus (TMV)-*Nicotiana tabacum* cv. Samsun NN [22]. The hypersensitive response (HR), following TMV inoculation, triggered systemic resistance to a subsequent challenge inoculation with the same virus, or other unrelated necrotic viruses (see a detailed historical account in [23]). It soon became evident that SAR was much more effective against viruses producing localized infection, i.e. following HR response, than against compatible viruses able to colonize the whole plant (Fig. 1) [24]. The reason of this apparently diverging behavior requires further insights. The diverse collection of PRs so far isolated do not appear to include antiviral agents, with the possible exception of PR-10, a 18 kDa ribonuclease from *Capsicum annum*, able to degrade TMV RNA [25]. Moreover, other events associated with SAR induction, such as cell wall fortification and phytoalexin synthesis, while effective against bacterial and fungal pathogens, do not prevent virus replication or spread [24]. Thus, the increase of endogenous salicylate would have to be the main defence response to inhibit virus replication, cell to cell and long distance movement of the viruses, which it does not always do [26–29]. It is not surprising that chemical-triggered SAR is only very effective against HR-inducing viruses: in such a case the plant is, to a certain extent, already "per se" resistant, being able to recognize a viral effector. Thus, a chemical treatment only primes the plant to respond more rapidly to the challenging inoculation, accelerating HR and, in turn, reducing cell-to-cell spreading of the virus and the number of cells involved in programmed cell death (PCD, Fig. 2) [27]. This, ultimately, results in the mitigation or lack of macroscopic symptoms. So, challenging a plant with an HR-inducing virus still remains one of the simplest methods to verify SAR establishment that can also be quantified by counting the number and measuring the size of necrotic local lesions produced on resistant and control leaf tissues.

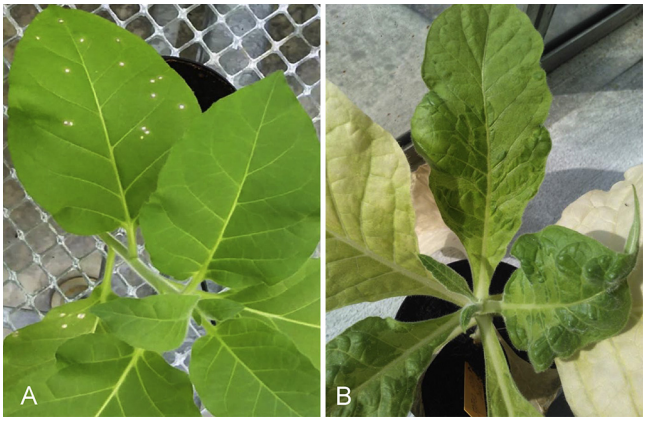

Fig. 1. Different type reactions of tobacco to viruses. (A) Tobacco Samsun NN that recognizes tobacco mosaic virus (TMV) triggering a hypersensitivity response (HR) that localizes the virus into necrotic lesions. This prevents systemic infection. (B) Compatible reaction of Tobacco White Burley that is not able to recognize TMV and is infected systemically, leading to plant death.

The cell-to-cell virus spreading and loading into phloem can also be delayed by callose deposition in plasmodesmata, either by enhancing it synthesis or by inhibiting its degradation through β-1,3-glucane synthase and β-1,3-glucanase, respectively [reviewed in 30,31], although viral proteins can counteract the activity of these enzymes by maintaining channels opened [32]. Callose deposition is mediated by abscisic acid (ABA) [33], which blocks salicylate-inducible defence responses [34], therefore ABA may be detrimental in limiting virus infection, unless callose deposition occurs in an early phase of the pathogenesis process, such as the case of HR-inducing viruses [35,36].

However, from an agronomical point of view the infection of HR-inducing viruses is often insignificant, as the related necroses actually represent an efficient form of resistance due to the recognition of a viral gene product by plant innate immune system [37,38]. The plant immune responses are classically formulated according to two lines of defence, interacting with pathogens following