

MiniReview

Silicon and plant disease resistance against pathogenic fungi

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Abstract

Silicon (Si) is a bioactive element associated with beneficial effects on mechanical and physiological properties of plants. Silicon alleviates abiotic and biotic stresses, and increases the resistance of plants to pathogenic fungi. Several studies have suggested that Si activates plant defense mechanisms, yet the exact nature of the interaction between the element and biochemical pathways leading to resistance remains unclear. Silicon possesses unique biochemical properties that may explain its bioactivity as a regulator of plant defense mechanisms. It can act as a modulator influencing the timing and extent of plant defense responses in a manner reminiscent of the role of secondary messengers in induced systemic resistance; it can also bind to hydroxyl groups of proteins strategically involved in signal transduction; or it can interfere with cationic co-factors of enzymes influencing pathogenesis-related events. Silicon may therefore interact with several key components of plant stress signaling systems leading to induced resistance.

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1. Introduction

Silicon (Si) has long been known to reduce the incidence of fungal diseases in a number of pathosystems. From the onset, it was proposed that deposition of amorphous silica in the leaf apoplast prevented penetration by pathogenic fungi. Although this mechanism may partly explain the prophylactic effects of Si, monomeric Si is also considered to be biologically active and to trigger a faster and more extensive deployment of plant natural defenses. This hypothesis was first proposed in the dicot system cucumber–powdery mildew [1] but is now believed to be generalized to both monocots and dicots. Consequently, it seems plausible that Si acts on general

mechanisms common to most plant species such as those leading to the expression of plant stress genes. In this review, different aspects of Si biochemistry are presented in the context of its possible interaction with plant defense activation. The objective is to bring forth potential alternatives to explore and explain the elusive role of Si in protecting plants against fungal diseases.

2. Silicon in biological systems

Silicon is the second most abundant element in the lithosphere (27.70%) and it is as important as phosphorus and magnesium (0.03%) in the biota [2]. Hydrated silica represents the second most abundant biogenic mineral after carbonate minerals [3]. Silicon is accumulated and metabolized by some prokaryotes [4], and Si

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compounds can stimulate the growth of a range of fungi [5]. It is well known that Si is essential for diatoms [6]. In mammals, Si is considered an essential trace element, required in bone, cartilage and connective tissue formation, enzymatic activities and other metabolic processes [7–9]. Silicon was suggested to act as a phosphoprotein effector in bone [10]. In mammals, Si is also reported to positively influence the immune system and to be required for lymphocyte proliferation [11]. The aqueous chemistry of Si is dominated by silicic acid at biological pH ranges [12]. Monosilicic acid can form stable complexes with organic hydroxy-containing molecules [13]. Biosilica also has been identified associated with various biomolecules including proteins and carbohydrates [14]. Hypervalent forms of silicon have been found to complex with a range of sugars and sugar derivatives [15,16]. Recently, Kinrade et al. [17] reported the first evidence of an organosilicon compound formed in vivo in the diatom *Navicula pelliculosa*. In diatoms, Si was suggested to affect phosphorylation of specific proteins required for the synthesis of DNA and specific mRNA [18,19].

3. Silicon in higher plants

The potential benefits of Si nutrition in plants have been extensively reviewed [20–22]. These include the enhancement of growth and yield, improvement of mechanical properties (stature, soil penetration by roots, exposure of leaves to light, resistance to lodging), reduction of transpiration and resistance to drought stress, resistance to salinity, resistance to metal toxicities, effects on enzyme activities and increased resistance to pathogens. While some of these properties are likely to derive from the deposition of amorphous silica ($\text{SiO}_2 \cdot n\text{H}_2\text{O}$), others should be considered as consequent to the bioactivity of monosilicic acid. Silicon is ubiquitous in monocotyledons and dicotyledons, in amounts equivalent or higher to those of phosphorus and magnesium [23]. Jones and Handreck [24] have divided plants into accumulators (10–15% dry weight) including wetland grasses, intermediate (1–3% dry weight) including dryland grasses, and non-accumulators (<1% dry weight) including dicots. Monosilicic acid is absorbed from the soil solution and it follows the transpiration stream. Where silicic acid is concentrated over a critical level (~ 100 ppm at biological pH), it polymerizes as phytoliths ($\text{SiO}_2 \cdot n\text{H}_2\text{O}$), which constitutes the bulk of a plant's Si content [24]. Silicon transporters have been characterized in a diatom [25]. The Si uptake system and Si transporters have also been characterized in rice [26]. Neumann and De Figueiredo [27], stating that Si is found in the plant cytoplasm and subcellular structures, proposed a mechanism of Si uptake, aside from that of membrane transporters, in which an endo-

cytotic process allows Si transport inside the cell. In plants, Si tends to polymerize in cell walls, cell lumen, intercellular spaces and in the subcuticular layer [28]. This process of opal formation is not occurring at random. Plant species differ with respect to the size and shape of phytoliths they accumulate. The nucleation and growth of these structures is under the control of specific proteins [29,30]. Other evidence supports the importance of organosilicon compounds in plants. In a selection of plants containing 0.74–3.59% Si, more than 50% of total Si was contained in the organic fraction versus polymerized and soluble forms. The organic Si fraction was found to bind proteins, phenolic compounds (lignin, condensed polyphenols), lipids and polysaccharides (cellulose, pectic substances) [31].

4. Silicon and disease resistance in plants

4.1. Mechanical role

The initial theory concerning the mode of action of Si in plant's prophylaxis involved a mechanical barrier against penetration. However, as early as 1965, this theory was put into doubt by Okuda and Takahashi [32], citing Yoshi's results [33] of non-correlation between Si treatment and leaf toughness as measured by a needle-puncture method: "From this result, it seemed that Si protected the rice plant against blast disease, but the increase in mechanical toughness of the plant tissue resulting from absorbed Si is not sufficient to explain the mechanism of protection". Nevertheless, this theory was maintained over the years. Carver et al. [34], upon the observation of Si accumulation in papillae consistent with findings from Kunoh and Ishizaki [35], stated that polymerized Si at attempted sites of penetration may provide an additional mean of resistance against penetration. Recently, Kim et al. [36] proposed the reinforcement of cell walls in rice as a mechanism for enhanced resistance provided by Si treatment. Yet, in this work, no evidence linked cell wall reinforcement with penetration failure by the fungus. It should be noted that the logical link proposed between Si deposition and pathogen resistance stems from the fact that Si has been reported in several pathosystems to accumulate at infection sites [37], a process also noted in *Arabidopsis* infected by powdery mildew (Fig. 1). This probably derives from a higher transpiration rate at sites where the cuticle is damaged rather than active transport in a defensive way. As a matter of fact, Chérif et al. [38] observed the accumulation of Si in needle-punctured leaf holes and showed the absence of such deposits when plants were grown under saturated humidity. Even though Si is effectively deposited at preferential sites of penetration, and is also continuously deposited at higher rates after penetration has occurred, the hypothesis of

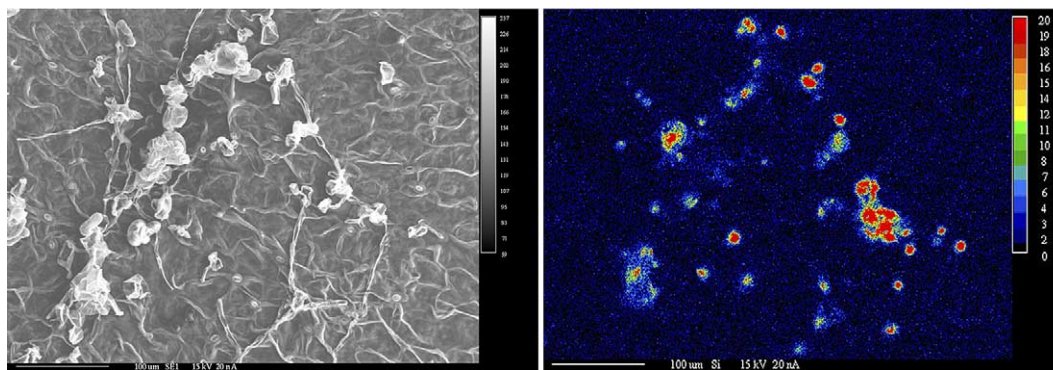


Fig. 1. Scanning electron (left) and X-ray (right) microanalysis showing that the accumulation of Si is coincident with *E. cichoracearum* presence on Si-treated *A. thaliana* leaves. The concentration of Si is indicated by color (see inset), where red represents the highest concentration of Si and black indicates no Si.

cell wall reinforcement by Si to explain enhanced resistance of plants against pathogenic fungi has been strongly contested in recent years.

4.2. Induced resistance

In the early 1990s, the first evidence disputing the role of Si as a mechanical barrier was reported in dicotyledonous models. Samuels et al. [39], using the cucumber–powdery mildew pathosystem, showed that within a short period of time after Si feeding was stopped, all prophylactic effects were lost. Thus, the interruption of Si feeding led to a loss of resistance even though opal had irreversibly accumulated and, according to the mechanical barrier hypothesis, should have slowed the pathogen development. Chérif et al. [40] contributed additional data contradicting the passive role of Si against fungi colonization by demonstrating that although Si failed to accumulate at infection sites under conditions of saturated humidity, Si-treated cucumber resisted more efficiently against *Pythium*. Chérif et al. [39,41] went on to propose that soluble Si activated defense mechanisms in cucumber against *Pythium* by showing enhanced activity of chitinases, peroxidases and polyphenoloxidases, and increased accumulation of phenolic compounds. In cucumber infected with powdery mildew, Fawe et al. [42] demonstrated the increased production of flavonoid phytoalexins in Si+ treated plants. Largely on the basis of experiments with cucumber, Fawe et al. [1] proposed that Si played an active role in reinforcing plant disease resistance by stimulating natural defense reactions. While this hypothesis became a paradigm in dicotyledons, its extension to monocotyledons, known to accumulate higher amounts of Si, remained invalidated. To address this question, different research groups carried out experiments with Si and monocot–pathogen interactions. In the wheat – *Blumeria graminis* f. sp. *tritici* (Bgt) system, histological and ultrastructural analyses revealed that epidermal cells of Si+ plants reacted to Bgt attack with specific defense

reactions including papilla formation, production of callose and release of electron-dense osmiophilic material identified by cytochemical labeling as glycosylated phenolics. These results suggested that Si mediated active localized cell defenses in wheat in the same way as observed in cucumber [43]. Furthermore, Datnoff's Group [44], working with rice blast in Florida, reported cytological evidence that Si-mediated resistance to *Magnaporthe grisea* in rice correlated with specific leaf cell reactions that interfered with the development of the fungus. The same group showed that in rice infected with *M. grisea*, Si was associated with higher accumulation of antimicrobial compounds at infection sites, including diterpenoid phytoalexins [45]. These recent results with Si and monocots bring not only further support to the theory that Si plays an active role in protecting plants against pathogens, but indicate that this role is not specific to dicots but rather generalized to the plant kingdom. However, the exact nature of the interaction of soluble Si with the plant's biochemical pathways leading to disease resistance remains unknown. In order to facilitate and harmonize the approaches to understand the role of Si in planta, Ghanmi et al. [46] proposed the use of the *Arabidopsis*–powdery mildew interaction, by showing that this model plant reacted to powdery mildew as other dicots and monocots did under Si treatment (Fig. 2).

5. How does Si activate plant defense reactions

5.1. Silicon mediated resistance

On the basis of their observations with cucumber, Fawe et al. [1] suggested a model to explain how Si would play a role in induced resistance. According to their model, Si bioactivity was compared to that of known activators/secondary messengers of systemic acquired resistance (SAR) whereby it would act as a modulator influencing the timing and extent of plant defense

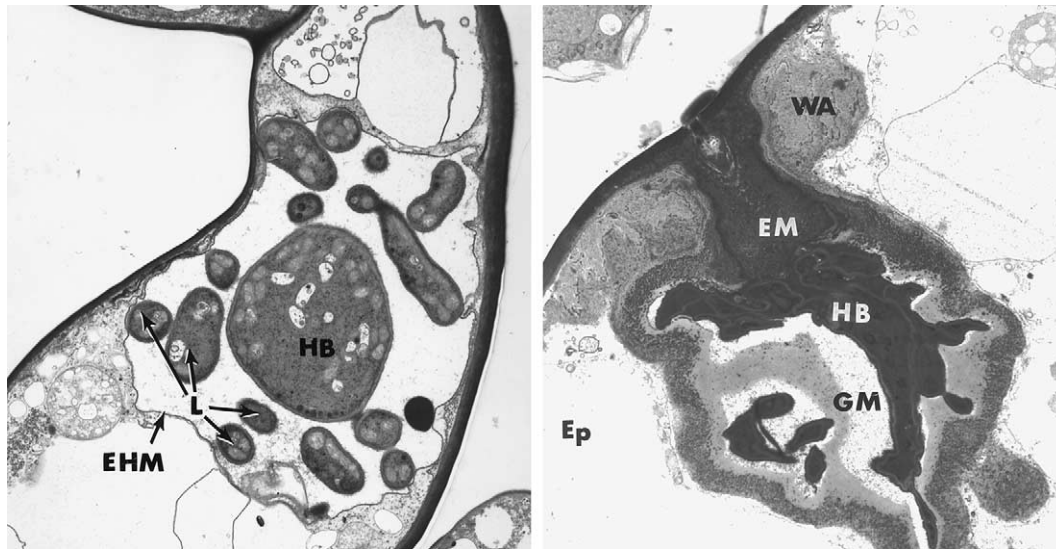


Fig. 2. Transmission electron micrographs of ultra-thin sections of *A. thaliana* leaves infected by *E. cichoracearum*. Silicon treatment (right) led to more efficient defense compared to control plants (left). EHM, extrahaustorial membrane; HB, haustorial body; Ep, epidermis; GM, granular material; WA, wall apposition; EM, electron-dense material; L, lobes. Adapted from Ghanmi et al. [46].

responses. Like secondary messengers, the effects of Si on secondary metabolism are significant only after elicitation; both Si and known activators are characterized by a saturable effect. A difference between known SAR activators and Si is the loss of activity when Si feeding is interrupted, because polymerization of Si leads to its inactivation as an inducer of resistance. These points of comparison prompted the authors to propose that Si acted as a signal in inducing defense responses.

5.2. Modulation of primary signal transduction

Silicic acid may modulate the activity of post-elicitation intracellular signaling systems. Hutcheson [47] has distinguished three classes of active defense mechanisms. The primary response occurs in cells infected by the pathogen, the secondary response is induced by elicitors and limited to cells adjacent to the initial infection site, and the systemic acquired response is transmitted hormonally to all tissues of the plant. Silicon is perhaps acting in the primary response, and the integration of enhanced signal transduction at the single cell level should result in increased levels of induced systemic resistance. Post-elicitation intracellular signaling leads to the expression of defense genes directing hypersensitive response, structural modifications of cell walls, stress hormones synthesis, antimicrobial compounds synthesis and PR proteins. As mentioned earlier, Si is involved in the processes leading, among other responses, to the accumulation of phytoalexins. The target of plant signaling upon pathogen elicitation is the cell nucleus, which receives information for de novo protein and antimicrobial compounds synthesis. Gene expression control through the phosphorylation of transcription

factors and their inhibitors is a major plant stress response. Signals leading to the expression of plant defense responses are transmitted to the nucleus through the activation of specific kinases/phosphatases cascades. This can be generalized to both endogenous [48–53] and exogenous [54] signaling events. Responses to biotic stresses are largely dependent on mitogen activated protein (MAP) kinases [55–58].

Protein kinases transmit information to the nucleus by the phosphorylation of hydroxyl group on amino acid residues. Silicon is known to bind to hydroxyl groups and may thus affect protein activity or conformation. The mode of action of Si in signal transduction may also derive from interactions with phosphorus. As early as 1906, Hall and Morrison [59] reported interactions between Si and phosphorus in barley. It is now considered that the internal improvement of P utilization and the broadening of P fertilization range provided by Si fertilization [60] derives from interactions with cationic metals such as Mn and Fe [61]. Metals play a structural role for many enzymes. Enzymatic dysfunctions may derive from the excess of essential metal species or the presence of toxic metal species [62]. Whether Si improves plant defenses indirectly by sequestering cationic metals, or directly by modulating protein activity involved in signal transduction remains to be investigated.

5.3. Silicon and induced systemic resistance

Silicon-fed plants will naturally translocate silicic acid throughout all tissues. Upon pathogen attack, the infected tissue will synthesize, among other defense reactions, antimicrobial compounds together with systemic

stress signals such as salicylic acid, jasmonic acid and ethylene. In a given cell, if Si indeed modulates the signaling events leading to the synthesis of antimicrobial compounds, it should also modulate the generation of systemic signals given that both processes depend on primary elicitation. Accordingly, silicic acid, without being itself a secondary messenger, could play a positive role in both local and systemic resistance.

6. Conclusion

Silicon is a bioactive element in various biological systems, but its mode of action in plants remains a matter of speculation. It has been shown to enhance the expression of natural defense mechanisms and the accumulation of phytoalexins in monocots and dicots. Because phytoalexins are highly specific in each plant species, it is more likely that Si acts on mechanisms shared by all plant species, such as those leading to the expression of plant stress genes (signaling cascades). Silicon, in the form of silicic acid, would act locally by inducing defense reactions in elicited cells and would also contribute to systemic resistance by enhancing the production of stress hormones. However, the exact mechanism by which Si modulates plant signaling remains unclear. From the gathered evidence, Si could act as a potentiator of plant defense responses or as an activator of strategic signaling proteins. Silicon may therefore interact with several key components of plant stress signaling systems ultimately leading to induced resistance against pathogenic fungi.

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