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The Role of Potassium Silicate in Quorum Quenching Against the Virulence of Ralstonia solanacearum, the Causal Agent of Bacterial Wilt in Tomato

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ABSTRACT: Bacterial wilt caused by *Ralstonia solanacearum* remains one of the most destructive constraints in tomato production worldwide. The pathogen's virulence is tightly regulated by quorum sensing (QS), which controls exopolysaccharide (EPS) biosynthesis, extracellular enzyme secretion, and biofilm formation. Targeting QS through quorum quenching (QQ) represents a promising anti-virulence strategy without imposing the selective pressure associated with conventional bactericides. This study investigated the dual role of potassium silicate as (i) a QS-interfering agent that modulates bacterial virulence traits and (ii) an inducer of host systemic resistance. Potassium silicate at 1 mM significantly reduced EPS production and biofilm formation, whereas 2 mM enhanced peroxidase activity in tomato plants. Disease severity was reduced during the early stages of infection in silica-treated plants. These findings indicate that potassium silicate attenuates bacterial wilt development through the integrated modulation of pathogen virulence and host defense responses. This study provides mechanistic insight into silicon-mediated plant protection and highlights potassium silicate as a sustainable strategy for bacterial wilt management.

KEYWORDS: Bacterial Wilt, Quorum Sensing, Quorum Quenching, Silicon-Mediated Resistance, Tomato Immunity

INTRODUCTION

Bacterial wilt is a devastating disease affecting a wide range of horticultural crops, including tomato (*Solanum lycopersicum* L.), and is caused by the soil-borne pathogen *Ralstonia solanacearum*. This pathogen is characterized by a broad host range, environmental persistence, and substantial genetic diversity, all of which complicate effective disease management. Bacterial wilt results in significant yield losses, often exceeding 50% under favorable environmental conditions (Huang et al., 2023; Youmbi et al., 2022). *Ralstonia solanacearum* infects plants through the roots and spreads into the vascular tissue, where it causes xylem occlusion and the characteristic wilt symptoms.

The mechanism by which *R. solanacearum* induces wilting is complex. After entering through the root system, the bacteria proliferate within the xylem vessels. As the population increases, the pathogen produces exopolysaccharides (EPS), which accumulate and obstruct xylem vessels. This blockage restricts the upward movement of water and nutrients, leading to water stress and subsequent wilting. In addition to EPS, the bacterium secretes extracellular enzymes and forms biofilms that facilitate colonization and persistence within plant tissues, further aggravating vascular dysfunction. The combined effects of impaired water transport, nutrient deprivation, and disrupted cellular hydration ultimately result in chlorosis, tissue collapse, and plant death if the disease is not effectively managed.

A defining characteristic of R. solanacearum pathogenicity is its quorum sensing (QS) regulatory system. Central to this system is the RasI/R signaling pathway, which synthesizes 3-hydroxy-palmitic acid methyl ester (3-OH PAME), a signaling molecule that coordinates the expression of virulence genes involved in EPS production, extracellular enzyme secretion, and biofilm formation (Jinli et al., 2022; Tsumori et al., 2022). EPS plays a critical role in vascular occlusion and symptom development, whereas biofilm formation enhances bacterial colonization and persistence within host tissues (Yang et al., 2021). Because QS regulates virulence rather than bacterial survival, its disruption—known as quorum quenching (QQ)—has emerged as a promising anti-virulence strategy that minimizes selective pressure associated with conventional bactericides (D'Aquila et al., 2024; Yi et al., 2021).

The economic impact of bacterial wilt is particularly severe in tomato cultivation. As a high-value horticultural commodity with increasing global demand, tomato production is highly vulnerable to R. solanacearum infection. The disease significantly reduces both yield and fruit quality. In many tomato-producing regions, including Indonesia, losses may exceed 50%, especially when infection occurs during critical growth stages. Furthermore, bacterial wilt reduces fruit marketability, resulting in substantial financial losses for farmers. The costs associated with disease control, particularly chemical pesticide application, further intensify

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the economic burden. Beyond direct production losses, bacterial wilt disrupts supply chains by increasing distribution costs and reducing product availability, potentially leading to market price fluctuations that ultimately affect consumers.

Conventional management strategies for bacterial wilt primarily rely on chemical pesticides and cultural practices such as crop rotation and soil sanitation. However, these approaches often fail to provide long-term control and may contribute to the emergence of resistant pathogen populations. Moreover, excessive reliance on chemical treatments raises environmental concerns and increases production costs. Consequently, the development of sustainable and environmentally sound alternatives has become increasingly important.

Potassium silicate has emerged as a promising alternative for bacterial wilt management due to its capacity to interfere with quorum sensing, a key regulatory mechanism controlling bacterial virulence. In R. solanacearum, QS governs EPS production, which is essential for vascular occlusion and symptom development. By disrupting QS-regulated processes, potassium silicate inhibits EPS synthesis and biofilm formation, thereby limiting bacterial colonization and persistence within host tissues.

In addition to its effects on the pathogen, potassium silicate enhances plant resistance to biotic stress. Silicon deposition in plant tissues strengthens cell walls, increasing structural rigidity and reducing pathogen penetration. Moreover, potassium silicate modulates defense-related hormonal pathways, particularly those associated with salicylic acid signaling, which is crucial for resistance against bacterial pathogens. Silicon treatment also promotes the accumulation of reactive oxygen species (ROS), which function as signaling molecules that activate downstream defense responses. Furthermore, potassium silicate has been reported to stimulate the production of phytoalexins and pathogenesis-related proteins, thereby reinforcing the plant's immune system.

Several studies have demonstrated that potassium silicate not only suppresses R. solanacearum virulence by interfering with QS-regulated processes but also enhances host innate immunity. By activating salicylic acid-mediated defense pathways in tomato, potassium silicate primes plants for stronger resistance against subsequent infections. This dual functionality—attenuating pathogen virulence while strengthening plant defense—highlights the potential of potassium silicate as an environmentally sustainable strategy for managing bacterial wilt in tomato production systems.

MATERIALS AND METHODS

Materials and Equipment

The equipment used in this study included disposable inoculating loops, L-spreaders, disposable Petri dishes, a furnace, an orbital shaker, an incubator, an autoclave, a centrifuge, a thermal cycler, a UV gel documentation system, an electrophoresis apparatus, disposable syringes, tubes, a spectrophotometer, 50 mL Falcon tubes, and other supporting laboratory instruments.

The materials used consisted of casamino acids, peptone, glucose, agar, 2,3,5-triphenyl tetrazolium chloride (TTC), KCl, 70% and 95% ethanol, sterile distilled water, 1× TAE buffer, TE buffer, phcA primers, 0.45 µm membrane filters, tomato seeds, and other supplementary reagents.

Preparation of *R. solanacearum*

The R. solanacearum RsGTO isolate was obtained from the laboratory collection and subcultured by streaking single bacterial colonies onto Casamino Acid Peptone Glucose Agar (CPGA) medium, followed by incubation at 28 °C for 24 h. The bacteria were subsequently propagated in liquid CPG medium under shaking conditions at 150 rpm for 24 h until reaching an optical density of OD600 = 0.1. Bacterial populations were determined using the spread plate method and calculated based on serial dilution.

Preparation of Potassium Silicate Solution

A 0.5 M stock solution of potassium silicate (K_2SiO_3; commercial brand "Kalsika") was prepared by dissolving 9.64 g of K_2SiO_3 in 100 mL of distilled water. The solution was filtered to remove impurities and allowed to stand overnight to ensure complete dissolution and stabilization prior to use. Working solutions of 1 mM and 2 mM were prepared by diluting the stock solution according to the dilution equation ($M_1V_1 = M_2V_2$), where M_1 and V_1 represent the concentration and volume of the stock solution, and M_2 and V_2 represent those of the working solution.

Preparation of Test Tomato Plants

Tomato seeds were surface-sterilized with 70% ethanol for 10 s and rinsed thoroughly with sterile distilled water. The seeds were germinated in a growth medium until seedlings reached 21 days of age. Seedlings were transplanted into pots containing a mixture

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of cocopeat and vermiculite when they reached approximately 8–10 cm in height and were free from visible pests and diseases. Potassium silicate treatment and bacterial inoculation were performed when plants had developed 8–10 true leaves.

Assay of Extracellular Polysaccharide (EPS) Production

A 24-h bacterial culture adjusted to OD600 = 0.1 was transferred into 50 mL Falcon tubes (3 mL per sample) and centrifuged. The supernatant was discarded, and the pellet was washed with 1 mM or 2 mM potassium silicate solution, followed by centrifugation at 5000 rpm for 10 min. The washing step was repeated, and the pellet was resuspended in the respective potassium silicate solution.

The resulting suspension was centrifuged, and the supernatant was filtered through a 0.45 µm membrane filter. A 3 mL aliquot was transferred into a sterile tube, and absolute ethanol was added at a ratio of 4:1 (ethanol:bacterial suspension). The mixture was incubated at –20 °C overnight, vortexed, and centrifuged again. The supernatant was discarded, and the pellet was resuspended in 10 µL of absolute ethanol. The suspension was transferred onto pre-weighed aluminum foil or microtubes, dried in an oven at 55 °C for 2 h, cooled, and weighed to determine EPS dry mass.

Assay of Endoglucanase (EGL) Production

The assay was conducted following the method described by Guerrero et al. (2015). Endoglucanase activity, which is responsible for cellulose degradation, was tested on solid carboxymethylcellulose (CMC) medium. A 1 µL aliquot of a 24-hour bacterial culture, previously washed with 1 mM and 2 mM potassium silicate, was inoculated onto the medium and incubated at 28 °C for 4 days. To visualize endoglucanase activity, 6 mL of Congo red solution (0.1%) was added to the medium and gently agitated on a shaker at 40 rpm for 15 minutes. The plates were then washed three times with 6 mL NaCl solution. If the clear zones were not sufficiently visible, the method described by Lijon et al. (2025) was applied, in which 5 mL of 5% acetic acid was added for 3 minutes to enhance the clarity of the hydrolysis zones (Huang et al., 2023).

Biofilm Formation Assay

Biofilm formation in R. solanacearum was quantified using the microplate assay. Briefly, a 24-hour culture of R. solanacearum was harvested by centrifugation and resuspended in CPG medium. The optical density of the suspension was adjusted to OD600 = 0.1. The culture was then washed with 1 mM and 2 mM potassium silicate solutions. A 500 µL aliquot was transferred into a microplate and incubated at 28 °C for 24, 48, and 72 hours. Following incubation, the wells were stained with 500 µL of crystal violet solution and washed three times with sterile distilled water. Absolute ethanol was then added to solubilize the dye, and biofilm biomass was quantified spectrophotometrically (Kumar et al., 2016).

Virulence Assay of *R. solanacearum* on Tomato Plants

The virulence assay was conducted on 21-day-old tomato plants that had previously received potassium silicate treatment and bacterial suspension. Inoculation was performed by applying 40 mL of R. solanacearum suspension to the plant roots. The bacterial suspension was adjusted to the desired concentration by measuring absorbance at 600 nm using a spectrophotometer. Disease progression was monitored daily for 14 days.

Peroxidase Assay in Tomato Plants

Peroxidase activity was analyzed in tomato leaf samples both before inoculation with R. solanacearum and after pathogen inoculation. The assay followed a modified method of (Shannon et al., 1966). Briefly, clean and dry test tubes were prepared, and 2100 µL of enzyme-free distilled water was added to each tube. Subsequently, 320 µL of phosphate buffer was added and mixed by pipetting, followed by the addition of 160 µL of hydrogen peroxide solution (0.5% H_2O_2) and 320 µL of 5% pyrogallol solution, each mixed by pipetting. Finally, 100 µL of enzyme extract obtained from tomato leaves was added and mixed by pipetting three times.

RESULT

Analysis of Virulence Factor Production

Based on Figure 1a, potassium silicate treatment resulted in a concentration-dependent reduction in EPS production by Ralstonia solanacearum. Both 1 mM and 2 mM treatments showed lower EPS levels compared to the untreated control, with the strongest suppression observed at 2 mM. This trend indicates progressive attenuation of a key virulence factor as silicate concentration increased.

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Endoglucanase (EGL) activity (Figure 1b) increased over time in all treatments, reflecting normal temporal enzyme expression. However, potassium silicate reduced the diameter of hydrolysis zones relative to the untreated control, particularly at 2 mM. At 24 h, EGL activity under the 2 mM treatment was not yet detectable, suggesting delayed enzyme expression. Although activity became visible at later time points, it remained lower than that of the control. The strongest suppression was observed at 72 h, indicating concentration-dependent inhibition of extracellular enzyme production rather than complete inhibition of bacterial growth.

Biofilm formation (Figure 1c) exhibited treatment-dependent temporal dynamics. The 1 mM potassium silicate treatment consistently reduced biofilm biomass across all observation days, suggesting stable interference with biofilm maturation. In contrast, the 2 mM treatment showed initial suppression followed by a marked increase at the final observation time, indicating a possible stress-induced compensatory response. Overall, these findings demonstrate that potassium silicate modulates biofilm development in a concentration- and time-dependent manner, with 1 mM providing more consistent anti-biofilm effects.

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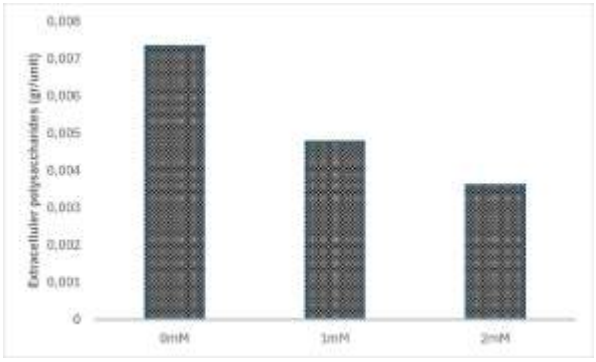
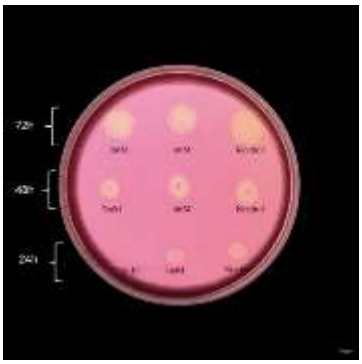
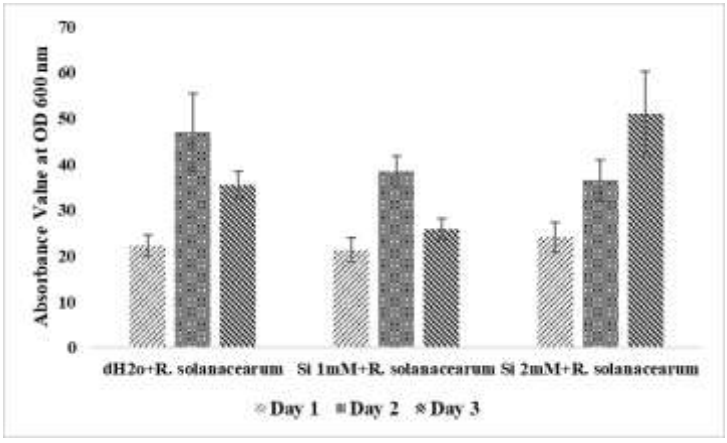


1a.



1b.



1c.

Figure 1. Effect of silica at concentrations of 1 mM and 2 mM on the production of EPS, EGL, and biofilm in *R. solanacearum*.

Disease Severity and Peroxidase Activity

Based on Figure 2a, disease severity progressively increased in all inoculated treatments over time. However, potassium silicate delayed disease progression compared to the untreated infected control. Although infection ultimately developed in all inoculated plants, silicon-treated plants exhibited lower final disease severity and a slower rate of symptom progression. These findings indicate that potassium silicate attenuates bacterial wilt development rather than completely preventing infection, suggesting a modulatory effect on host–pathogen interaction dynamics.

Peroxidase activity (Figure 2b) exhibited a time-dependent increase following pathogen inoculation. Plants treated with 1 mM potassium silicate showed the highest enzyme activity at the later stages of observation, exceeding that of infected controls. This

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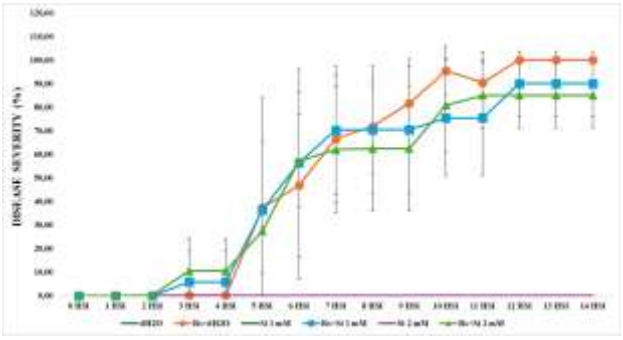
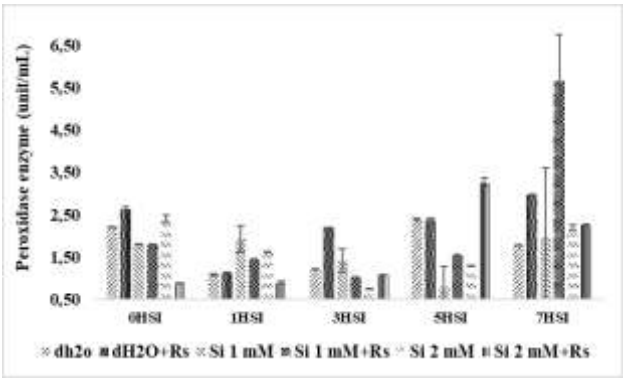
enhanced oxidative response suggests that potassium silicate acts as a defense-priming agent, promoting lignification and reactive oxygen species (ROS) detoxification mechanisms. In contrast, the 2 mM treatment did not induce a comparable increase in peroxidase activity, indicating the presence of an optimal concentration threshold for effective defense activation.

Visual assessment (Figure 2c) supported the quantitative disease severity data. Plants inoculated with *Ralstonia solanacearum* without silicon treatment exhibited pronounced wilting and structural collapse. In contrast, silicon-treated plants, particularly at 2 mM, showed delayed symptom development and maintained better structural integrity. These observations reinforce the protective role of potassium silicate in mitigating bacterial wilt progression through combined modulation of pathogen virulence and host defense responses.



2a.



2b.

2c.

Figure 2. Analysis of disease severity and peroxidase activity in tomato plants inoculated with *R. solanacearum* under silica treatments at concentrations of 1 mM and 2 mM.

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DISCUSSION

Despite increasing recognition of silicon as a beneficial element in enhancing plant resistance against biotic stresses, its direct role in modulating quorum sensing (QS)-associated virulence traits in *Ralstonia solanacearum* remains insufficiently understood. While previous studies have emphasized silicon's ability to structurally reinforce plant tissues and activate host immune signaling pathways (Jiang et al., 2019; Pereira, 2024; Wang et al., 2022), limited attention has been given to whether potassium silicate can simultaneously influence QS-regulated virulence traits—such as exopolysaccharide (EPS) production, extracellular enzyme secretion, and biofilm formation—while also priming host defense responses. This study bridges this gap by providing mechanistic insights into how potassium silicate influences both bacterial virulence and host defense in *Ralstonia solanacearum*–tomato interactions.

Our findings demonstrate that potassium silicate exerts a concentration-dependent effect on the virulence of *Ralstonia solanacearum*. One of the primary mechanisms through which the pathogen causes wilt in tomato plants is the production of EPS, which facilitates xylem occlusion and systemic colonization. The results of this study demonstrate that potassium silicate significantly reduces EPS production, suggesting that potassium silicate modulates the QS system governing EPS biosynthesis. The suppression of EPS accumulation likely contributes to delayed symptom onset, highlighting the role of potassium silicate in interfering with the pathogen's ability to establish effective infections. This result aligns with earlier findings by (Milling et al., 2011), who reported that EPS production is vital for the virulence of *Ralstonia solanacearum* and that its inhibition leads to a reduction in the severity of bacterial wilt.

Furthermore, the reduction in endoglucanase (EGL) activity observed in potassium silicate-treated plants further emphasizes the effect of potassium silicate on pathogen virulence. EGL, an extracellular enzyme responsible for degrading plant cell walls, is regulated by QS pathways. The attenuation of EGL activity under silicate treatment indicates that potassium silicate impedes the pathogen's ability to degrade host tissues and invade plant cells, thereby reducing the pathogen's overall invasive capacity. This observation supports the work of (Yi et al. (2021), who found that inhibition of EGL activity significantly reduced the pathogen's ability to invade and colonize plant tissues, reinforcing the notion that potassium silicate disrupts the regulatory systems involved in virulence factor production.

Biofilm formation, another key virulence trait regulated by QS, was also significantly reduced in the presence of potassium silicate, especially at the 1 mM concentration. Biofilms are critical for bacterial survival in the plant, providing protection from environmental stressors and contributing to persistent infection. The reduction in biofilm biomass observed with potassium silicate treatment suggests interference with the maturation of biofilms, likely by disrupting the coordination of QS-regulated gene expression required for biofilm development. Disruption of biofilm formation without compromising bacterial viability has been observed in other studies investigating quorum quenching mechanisms (D'Aquila et al., 2024). Interestingly, at the higher 2 mM concentration, there was a delayed enhancement in biofilm formation, suggesting an adaptive response by *Ralstonia solanacearum* under sub-lethal stress. This suggests that potassium silicate may induce compensatory regulatory changes in the pathogen, leading to altered bacterial behavior rather than direct inhibition of growth or viability. Spratt & Lane (2022) have reported similar adaptive responses in bacteria exposed to environmental stressors, emphasizing the dynamic and adaptable nature of bacterial biofilm regulation.

At the plant level, potassium silicate treatment resulted in slower disease progression and reduced disease severity compared to untreated controls. Although *Ralstonia solanacearum* infection was not entirely prevented, the reduced severity of symptoms in silicon-treated plants reflects enhanced plant resistance. Silicon has been well-documented for its role in strengthening plant cell walls through deposition, which limits pathogen penetration and improves structural integrity (Hawerroth et al., 2019). In this study, the reduction in vascular obstruction, likely due to lower EPS accumulation, was observed in silicon-treated plants, further confirming the role of potassium silicate in reinforcing the plant's physical barriers to pathogen invasion.

In addition to its structural role, potassium silicate also primes the plant's immune system. This is reflected in the significant increase in peroxidase activity observed in potassium silicate-treated plants, particularly at the 1 mM concentration. Peroxidases are key enzymes involved in lignification, reinforcing cell walls, and detoxifying reactive oxygen species (ROS) generated during infection. The enhanced peroxidase activity suggests that potassium silicate primes the plant's defense response, enabling a faster and more robust immune reaction upon pathogen challenge. This concept of defense priming is supported by the findings of Santos & Franco (2023), who suggested that priming mechanisms allow plants to respond more efficiently to subsequent pathogen attacks by enhancing their enzymatic activity. The stronger induction of peroxidase activity at the 1 mM concentration compared to the 2 mM concentration

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indicates that there is an optimal threshold for effective defense activation. At higher concentrations, excessive silicon may disrupt metabolic balance or interfere with key signaling pathways, reducing the plant's ability to activate defense mechanisms effectively.
Further supporting the concept that potassium silicate works by enhancing host defense mechanisms, several studies have shown that silicon can stimulate the production of other key antioxidant enzymes, such as superoxide dismutase (SOD) and catalase, which play significant roles in the plant's ability to detoxify ROS. These antioxidant enzymes mitigate oxidative stress generated during pathogen infection, contributing to the enhanced tolerance of plants to bacterial wilt. Moreover, silicon has been implicated in modulating plant hormone signaling pathways, including those associated with jasmonic acid (JA) and ethylene, which are critical for defense against necrotrophic pathogens like *Ralstonia solanacearum* (Verma et al., 2024). This highlights the multi-faceted role of silicon not only in reinforcing plant cell walls but also in orchestrating systemic immune responses that prepare plants for more effective defense.
Potassium silicate, therefore, functions as a dual-action agent in the host–pathogen interaction. It not only reduces *Ralstonia solanacearum* virulence by modulating QS-controlled processes such as EPS production, extracellular enzyme secretion, and biofilm formation, but also primes the plant's immune system, enhancing its ability to respond to subsequent pathogen challenges. Rather than acting as a direct antimicrobial agent, potassium silicate operates as a regulatory modulator of both bacterial communication systems and host defense pathways. This integrated mechanism, which combines anti-virulence and defense-priming effects, offers a more sustainable and environmentally friendly alternative to conventional bactericides, which often induce resistance and environmental harm. The use of potassium silicate presents a more eco-friendly and long-term solution for managing bacterial wilt in tomato crops, minimizing the environmental impact of chemical pesticides.

CONCLUSION

Potassium silicate effectively modulates both *Ralstonia solanacearum* virulence and tomato plant defense mechanisms. By interfering with quorum sensing–regulated virulence factors, including exopolysaccharide production, extracellular enzyme secretion, and biofilm formation, potassium silicate reduces pathogen colonization and disease severity. In addition, it primes plant defense responses by enhancing peroxidase activity and reinforcing structural barriers. These findings position potassium silicate as a promising and sustainable alternative to chemical pesticides, providing a dual mechanism for managing bacterial wilt while minimizing environmental impact. Further research is warranted to evaluate its long-term efficacy and broader applicability in agricultural systems.

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