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Silicon nonspecifically affects aggressiveness in *Fusarium* head blight pathogens

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Abstract

Silicon (Si) has been largely regarded to have no selection pressure on plant pathogens because it regulates plant defense system to reduce pathogen infection and development and does not directly affect pathogen itself; however, its effect seems to be specific in some pathogen-host plant interactions. In *Fusarium* head blight (FHB), associated with devastating agronomic effects on total yield and grain quality, which infects higher Si-absorbers and –accumulators wheat and barley plants; its effect on aggressiveness is unknown. To clarify the nature of reduction of FHB symptoms upon Si treatment at the earliest and latest development stages, we analyzed Si effect on nine aggressiveness components obtained under several experimental conditions. We used 16 fungal isolates of varying aggressiveness and eight bread wheat, durum wheat and barley cultivars with contrast susceptibility to disease. The positive effect of Si on enhancing host resistance against *Fusarium* infection in the young and adult host parts showed that the nine components evaluated in this study were negatively impacted by Si. Definitely, Si at 1.7 mM reduced equally FHB symptoms from the highest and least pathogenic isolates of the four tested *Fusarium* species regardless of botanical background for the used plant materials. This indicates for the first time that Si nonspecifically affects aggressiveness in FHB pathogens. Our results reveal that Si could be a valuable tool in integrated pathogen management by suppressing pathogen development on wheat and barley when affected by *Fusarium*. Most importantly, no hazard exists to emergence of Si-resistant pathogen populations upon Si applications on diverse FHB populations.

Keywords: *Fusarium* pathogens, pathogenicity, silicon application, wheat and barley resistance.

1. Introduction

Bread wheat (*Triticum aestivum*) is one of the most major grown crops worldwide. It is considered a main essential food for over 35% of the glob's population, supplying more than 20% of their daily calorie and protein requirements [1]. Durum wheat (*T. durum*) is principally planted under rainfed conditions in the Mediterranean region [2]. Its high protein content and typical vitreous kernel structure make it appropriate for several uses, involving the production of bulgur, pasta, couscous, flatbreads, and semolina [3]. Barley (*Hordeum vulgare*) is a vital dietary element of human beings and is also known as the poor man's crop as it necessities low input and has better adaptability to alkalinity, drought, salinity and marginal lands. It is planted over the spring season in almost all parts of the world with arid or semi-arid environments [4]. Unfortunately, *Triticum* and *Hordeum* can be invaded by several devastating diseases that decrease the harvest quantity and quality.

Fusarium head blight (FHB) is a largely known destructive fungal disease infecting the wheat and barley production over the world [5]. Head blight disease complex is attributable to at least 17 species with specific growth habitats which undergo principally under the *Fusarium* genus; *F. graminearum* and *F. culmorum* are reported to be the main aetiological causative agents in *Triticum* and *Hordeum* on a global scale [6]. FHB invasion is favored by warm and humid environmental conditions during flowering and

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early stages of kernel development [7]; *Fusarium* whitens wheat and barley heads, and the damaged heads can become infertile [8]. If grains are formed, they can seem discolored and shriveled [9]. FHB negatively decreases production and impairs kernel quality due especially to the accumulation of dangerous mycotoxins [10], particularly the trichothecene group including deoxynivalenol (DON), in harvested kernels [11]. These secondary metabolites with toxic effects on humans and animals make harvested wheat and barley kernels unacceptable for bakery, baking, malting and brewing industry. Consequently, *Fusarium* has become a food and feed safety topic [12].

Options for FHB control are limited [5, 13]. To date, breeding for FHB resistance is the most cost-efficient method to manage this disease [12]. The various resistance mechanisms in *Triticum* and *Hordeum* to *Fusarium* are quantitative in nature [5, 10]. Head blight resistance is highly complex and separated into many resistance types [6]. Aggressiveness is the most crucial fungal trait influencing FHB infection and stability of wheat and barley resistance [14]; it is also a crucial factor detecting the potential capacity of *Fusarium* isolates to cause FHB outbreaks [8, 10]. Some researches focusing on aggressiveness, as defined by the disease induced by a pathogenic isolate on a susceptible host in a non-race/isolate-specific interaction [15], of *Fusarium* complex have been addressed [16]. Nevertheless, breeding for head blight resistance has found to be complex due to strong aggressiveness/cultivar-by-environment interaction and complicated inheritance of resistance genes [13], leading to the absence of commercial *Triticum* and *Hordeum* cultivars with satisfactory genetic resistance [12]. Taken into account this confirmation, new techniques for promoting wheat/barley defense or preventing *Fusarium* pathogens necessity to be explored due to the fact that no single policy has yielded successful management and to avoid the negative influence of disease on crop productivity [8]. An increasingly popular policy is to augment the defense of cereals against pathogens. In this issue, mineral nutrients, in addition to their considerable function in plant metabolism, have a long history in modulating the impacts of plant pathogens on cultivated crops [17].

For instance, the feeding of silicon (Si) fertilization into plants has been suggested as useful alternative to traditional control techniques [18]. The requirement of Si for plant development and growth has not been proven yet because of its common availability [19]. However, Si has been well-reported to play an essential function to augment growth, development and production for a board array of field crops, especially under diverse biotic (i.e., pathogens and insect pests) and abiotic (i.e., metal toxicity, nutrient imbalance, logging, water deficit, water salinity, radiation damage, UV, and temperature extremes) constraints. Till date, the functions of Si in higher plants have remained a quandary [20]. Plants uptake silicic acid through the roots; subsequently translocate it to the shoots, where it is polymerized into silica [21]. Si could enhance noxious influences of biotic constraints [22], by strengthening plant's protective layer, mediating host plant resistance mechanisms, and up- and down-regulating certain genes and their defensive products [18]. In plants, the final Si content depends on several factors, involving plant species, plant age, the cultivar's ability to absorb and distribute Si around the plant, as well as the content of Si available in the soil [23]. Thus, plants are separated as low accumulators (<0.1% Si), intermediary accumulators (1%), and high accumulators (more than 5% Si on dry weight basis) regarding Si absorption [20]. For instance, *Triticum* and *Hordeum*, which are monocots, accumulate higher amounts of Si in their shoots [19].

Taken into consideration that wheat and barley are recognized as Si absorbers (their absorption capacity from the soil reported 50-150 kg Si/ha) and accumulators (accumulating Si in concentration up to 20 g/kg of dry weight) [18, 21], new confirmation proved that the Si up-taking and deposition could be explained by the active transport mechanisms inherent to the roots and the shoots [20]. In wheat and barley, Si amelioration is regarded to enhance resistance to several noxious fungal pathogens i.e., *Magnaporthe oryzae* causing blast; *Blumeria graminis* f. sp. *hordei* causing barley powdery mildew; *Zymoseptoria tritici* causing septoria leaf blotch; *Bipolaris sorokiniana* causing spot blotch; tan *Pyrenophora tritici-repentis* causing spot and *Blumeria graminis* f.

sp. *tritici* causing wheat powdery mildew [24, 25, 26]. Si applications ameliorated wheat and barley resistance against FHB under *in vitro*, controlled and field conditions [27, 28, 29]. Si has been largely regarded to have no selection pressure on plant pathogens because it regulates plant defense system to reduce pathogen infection and development [18] and does not directly affect pathogen itself [20]; however, its effect seems to be specific in some pathogen-host plant interactions [30, 31]. In FHB; its effect on aggressiveness is unknown. Si benefit is significant for sustainable cultivation of *Triticum* and *Hordeum* [28, 29], given the wide global existence of FHB species [12]. To our knowledge, this is the first study to clarify the nature of reduction of FHB symptoms upon Si treatment at the earliest and latest development stages. We thus analyzed Si effect on nine aggressiveness components obtained under several experimental conditions by using 16 fungal isolates of varying aggressiveness and eight bread wheat, durum wheat and barley cultivars with contrast susceptibility to disease.

2. Materials and method

2. 1. *Triticum* and *Hordeum*, FHB isolates, and inoculum preparation

A set of eight small-grain cereal cultivars of Syrian origin covering a wide genetic and resistant variability including six *T. aestivum* and *T. durum* cultivars and two *H. vulgare* cultivars: Arabi Abiad (AB) and Arabi Aswad (AS) was selected from earlier *in vitro*, growth chamber and field trials [32] to represent a range of quantitative resistance types to FHB invasion. Wheat and barley cultivars Bohoth10 (bread) and AS moderately resistant, Cham4, Douma4 (bread), and AB moderately susceptible, Cham7 and Cham9 (durum) susceptible to moderately susceptible, and Acsad65 (durum, susceptible) were employed.

Sixteen single-spore derived cultures of four *Fusarium* species causing head blight, i.e. (*F. culmorum* (5 isolates), *F. solani* (6 isolates), *F. verticillioides* (synonym *F. moniliforme*) (4 isolates), and *F. equiseti* (one isolate)) selected for their diverse aggressiveness levels (established on earlier several experimental data [32]) were employed. Through the 2015 growth season, *Fusarium* isolates were collected from naturally infected wheat heads over nine locations in Ghab Plain with a FHB history, one of the principal Syrian wheat production areas. The single spore cultures on Petri dishes with potato dextrose agar (PDA, HiMedia, HiMedia Laboratories) with 13 mg/l kanamycin sulphate ($C_{18}H_{38}N_4O_{15}S$) added after autoclaving at 121°C (Systec, 3870 EL), were classified morphologically by using the keys of Leslie and Summerell [33] to species level. The 16 *Fusarium* isolates causing head blight isolates were molecularly analyzed by using random amplified polymorphic DNA markers (Operon Technologies) [32]. FHB isolates were stored by freezing at-16°C or in sterile distilled water (SDW) at 4°C till use [34].

Fusarium inoculum utilized for inoculation for the *in vitro*, growth chamber and field experimentations was normally performed as following: FHB suspension stored in cold water or four to six agar plugs out of each stored single-spore culture kept by freezing were put over the surface of Petri dishes PDA and incubated in an incubator (JSPC, JS Research Inc.) at 22ºC under continuous darkness for 10 days to allow *Fusarium* sporulation and development. Following incubation, FHB isolates were covered with 10 ml of SDW and conidia were dislodged. FHB suspensions were filtered through 2 layers of sterile cheesecloth to remove the pieces of agar and mycelia and directly quantified with a Neubauer chamber under an optical microscope and diluted to a desirable concentration as inoculum sources.

2. 2. Aggressiveness evaluation under several experimental conditions

The estimation of aggressiveness of 16 *Fusarium* cultures infected all the tested eight *Triticum* and *Hordeum* cultivars was through the detection of latent period of detached leaf inoculation, area under disease progress curve of Petri-dish inoculation and coleoptile dwarfing of a coleoptile infection determined under *in vitro* conditions, disease incidence (DI) determined utilizing a head artificial inoculation, disease severity (DS) determined utilizing a floret artificial inoculation as well as area under disease progressive curve calculated on the basis of DI and DS under controlled conditions under growth chamber conditions, and DI and DS determined using a head artificial inoculation under field conditions over two growing seasons.

2. 3. Si treatment

Pure Si was employed to avoid the confounding impacts of other elements in some Si-based soil fertilizations [22]. Si source was a SiO_2 powder (Kieselsaure, Carl Roth GmbH + Co. KG), which is composed of 99% Si at a minimum content. Si at a concentration 1.7 mM reduced FHB symptoms following several applications in wheat and barley under *in vitro*, growth chamber and filed conditions [28, 29]. Liquid solution of Si (1.7 mM) was supplied to wheat and barley plants in the form of silicic acid [H_4SiO_4], which was prepared by dissolving the SiO_2 powder in demineralized water. Pathogenic reactions upon Si treatment of all cultivars infected with *Fusarium* fungi were previously evaluated according to methods described by Sakr and Kurdali [28, 29]. If Si at 1.7 mM reduced equally FHB symptoms from the highest and least pathogenic isolates of the four tested *Fusarium* species regardless of botanical background for the used plant materials, Si nonspecifically affects aggressiveness in *Fusarium* head blight pathogens.

2. 4. Experimental design

The experiments were conducted to precisely and correctly clarify the nature of reduction of FHB symptoms upon Si treatment at the earliest and latest development stages. For a given aggressiveness component, a 2 × 8 × 16 factorial experiment, consisting of two Si concentrations (0 and 1.7 mM, referred to as −Si and +Si plants thereafter), eight wheat and barley cultivars with different resistance levels, and 16 *Fusarium* isolates causing FHB with diverse aggressiveness, were arranged in a randomized design with three replications (Figure 1). The experiment was repeated twice.

2. 5. Statistical analyses

Prior to analysis of variance (ANOVA), the percentages were arcsine angular transformed to stabilize variances. Using the DSAASTAT add-in version 2011, data were subjected to factorial ANOVA and the averages of treatments compared by Fisher's least significant difference test at $P<0.05$. Comparison among FHB isolates infected the eight wheat and barley cultivars upon Si treatment were made by the contrast procedure.

2. Results and discussions

From the beginning, absorption of Si by the host is a requirement for enhancing harmful impacts of diverse biotic stresses involving devastating fungal diseases linked with Si feeding [18, 22]. Previous reports carried out on young and adult *Triticum* and *Hordeum* plants applied with Si enhanced the concept that 1.7 mM Si is requested for the efficient resistance to be manifested to FHB [28, 29]. Bearing in the mind that Si did not influence directly on the tested 16 FHB pathogens [19, 20]; we theoretically suggested that Si stops DON biosynthesis by enhancing wheat and barley defense mechanisms to *Fusarium* pathogenesis which include ameliorating activity of chitinase, peroxidase, phenylalanine ammonia-lyase and superoxide dismutase, enhancing the level of accumulation of derivatives of the hydrogen peroxide and phenylpropanoid pathway as well as the enhancing the level of inhibitory phenolic compounds [25]. However, no data are available if Si affects aggressiveness in FHB isolates varying in their symptoms specifically or nonspecifically. Because this issue is important regarding the hazard to emerge Si-resistant pathogen populations upon Si applications on diverse FHB populations [23], we therefore attempted to acquire novel observations about the behavior of aggressiveness of fungal isolates under Si treatments. For the first time, we showed non-specific effects of Si treatments on quantitative pathogenicity in four *Fusarium* species.

The quantitative pathogenicity, i.e., aggressiveness, of the analyzed 16 *Fusarium* cultures utilized in the present investigation is probably the consequence of timely expression of many genes, governing production of mycotoxins, hormones, specific metabolites as well as cell-wall-degrading enzymes that change the host's resistance response as shown earlier [12]. It is supposed that Si would play a greater function in resistance against the more aggressive isolate because of generally weak induced defenses against the isolate [35]. Values (% of control) of reductions of the nine aggressiveness comports of four FHB species treated with treatment of exogenous Si at 1.7 mM on bread wheat, durum wheat and barley plants are shown in Table 1.

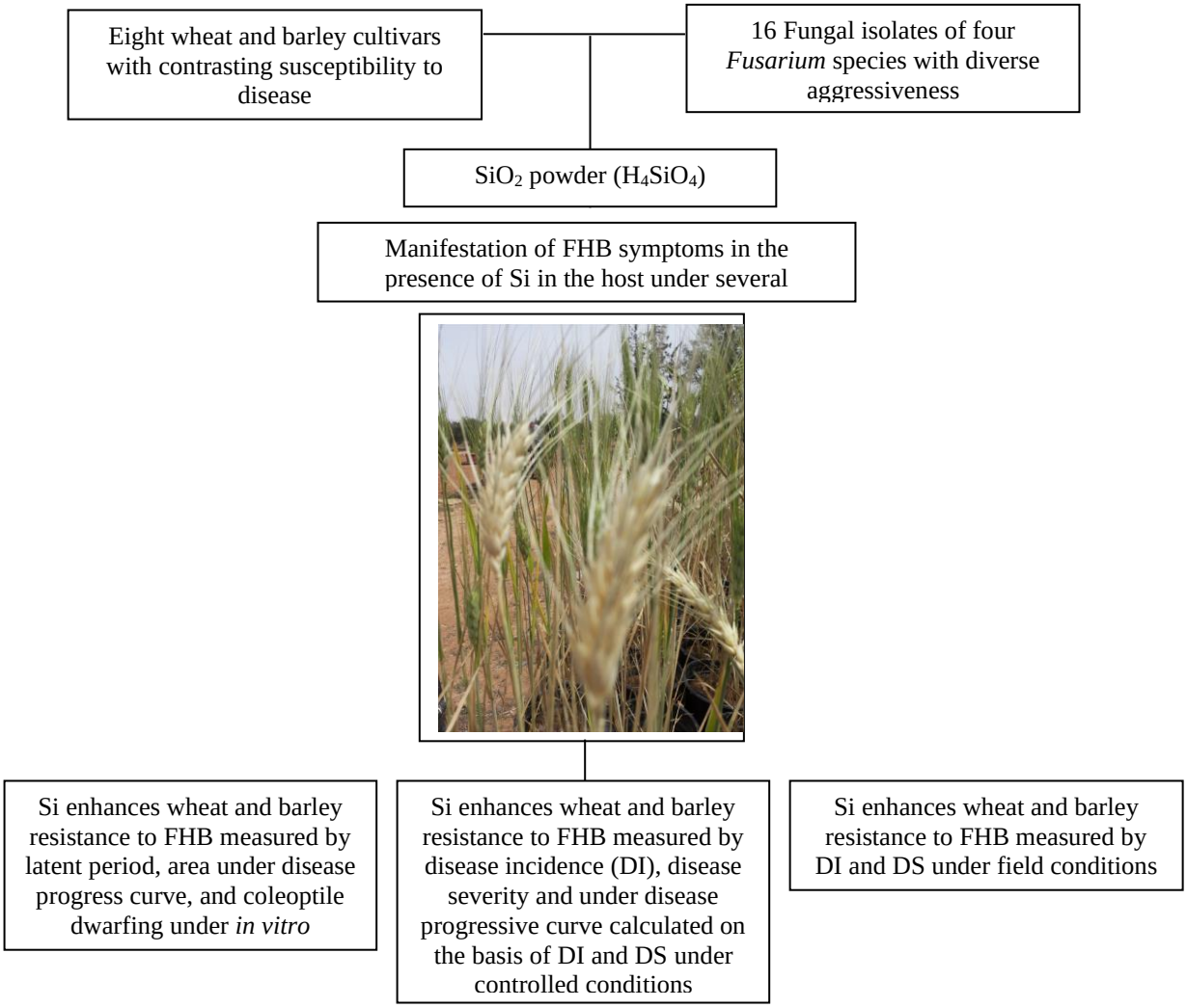

Figure 1. *A schema of the experimental design in the study*

Contrast analysis indicated that reductions in these nine components: i.e., latent period of detached leaf inoculation, area under disease progress curve of Petri-dish inoculation and coleoptile dwarfing of a coleoptile infection detected *in vitro*, disease incidence (DI) detected using a head artificial inoculation, disease severity (DS) detected using a floret artificial inoculation as well as area under disease progressive curve calculated on the basis of DI and DS under controlled conditions in a growth chamber, and DI and DS detected using a head artificial inoculation under field conditions over two growing seasons, were not significant among pathogen isolates with diverse pathogenicity ranging from highly to less pathogenic levels. Definitely, Si at 1.7 mM reduced equally FHB symptoms from the highest and least pathogenic isolates of the four tested *Fusarium* species regardless of botanical background for the used plant materials. In contrast to our findings, Si consistently delayed lesion development where *Oryza sativa* plants were infected with the aggressive *Xanthomonas oryzae* pv. *oryzae* isolate causing bacterial blight, but the impacts were less obvious where rice plants were infected with the less aggressive isolate [35]. Furthermore, the Si-impact seemed to be virus-specific, since this element decreased *Tobacco raingspot virus* symptoms formation and did not modify *Tobacco mosaic virus* damage in tobacco plants [31]. Such pathogen selectivity has also found for certain fungal infections [30]. Although Si impact appears to be isolate/species-specific, the application of Si fertilization has been promoted as a more promising option for the better control of plant diseases.

Table 1. Comparisons among reductions in latent period (LP) of detached leaf inoculation, area under disease progress curve (AUDPC) of Petri-dish inoculation and coleoptile dwarfing (CD) of a coleoptile infection detected in vitro, disease incidence (DI) detected using a head artificial inoculation, disease severity (DS) detected using a floret artificial inoculation as well as area under disease progressive curve calculated on the basis of DI and DS under controlled conditions (CC) in a growth chamber, and disease incidence and disease severity detected using a head artificial inoculation under field conditions (FC) over two growing seasons (% of control, inoculated with FHB pathogen and no addition of silicon) for a set of 16 fungal isolates for four Fusarium head blight species in eight wheat and barley cultivars

LP								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	14a	18a	16a	17a	15a	20a	17a	22a
F2 (*F. culmorum*)	13a	19a	16a	18a	14a	21a	18a	21a
F3 (*F. culmorum*)	14a	19a	16a	18a	14a	21a	19a	21a
F28 (*F. culmorum*)	13a	19a	15a	17a	15a	22a	17a	22a
F30 (*F. culmorum*)	14a	18a	15a	17a	14a	21a	17a	20a
F7 (*F. solani*)	14a	18a	15a	17a	15a	20a	17a	21a
F20 (*F. solani*)	15a	18a	16a	18a	14a	20a	18a	22a
F26 (*F. solani*)	15a	18a	16a	18a	15a	20a	18a	22a
F29 (*F. solani*)	14a	18a	16a	17a	14a	21a	18a	21a
F31 (*F. solani*)	14a	17a	16a	18a	14a	21a	18a	21a
F35 (*F. solani*)	14a	17a	17a	17a	14a	20a	17a	21a
F15 (*F. verticillioides*)	13a	18a	16a	18a	15a	20a	17a	22a
F16 (*F. verticillioides*)	13a	18a	16a	17a	15a	21a	17a	22a
F21 (*F. verticillioides*)	14a	18a	15a	17a	15a	21a	17a	21a
F27 (*F. verticillioides*)	15a	18a	15a	18a	15a	21a	18a	21a
F43 (*F. equiesti*)	15a	17a	16a	18a	14a	20a	18a	21a
AUDPC								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	13a	19a	15a	18a	16a	21a	18a	20a
F2 (*F. culmorum*)	14a	18a	17a	19a	16a	22a	19a	21a
F3 (*F. culmorum*)	14a	18a	16a	19a	15a	20a	19a	22a
F28 (*F. culmorum*)	14a	19a	15a	17a	15a	20a	17a	22a
F30 (*F. culmorum*)	13a	19a	15a	18a	14a	21a	19a	22a
F7 (*F. solani*)	13a	18a	16a	17a	14a	22a	17a	21a
F20 (*F. solani*)	15a	18a	16a	18a	14a	20a	17a	22a
F26 (*F. solani*)	15a	17a	15a	19a	17a	22a	18a	20a
F29 (*F. solani*)	13a	17a	15a	17a	14a	21a	17a	21a
F31 (*F. solani*)	13a	18a	17a	18a	15a	20a	18a	20a
F35 (*F. solani*)	14a	18a	16a	18a	15a	19a	18a	21a
F15 (*F. verticillioides*)	15a	17a	15a	18a	14a	21a	17a	21a
F16 (*F. verticillioides*)	13a	18a	16a	19a	15a	21a	19a	22a
F21 (*F. verticillioides*)	15a	17a	15a	17a	14a	21a	17a	22a
F27 (*F. verticillioides*)	14a	18a	17a	20a	16a	20a	19a	21a
F43 (*F. equiesti*)	14a	17a	16a	18a	14a	20a	19a	22a
CL								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	15a	19a	15a	18a	14a	19a	18a	22a
F2 (*F. culmorum*)	15a	18a	15a	18a	15a	20a	19a	21a
F3 (*F. culmorum*)	13a	20a	15a	19a	16a	20a	20a	21a
F28 (*F. culmorum*)	13a	20a	17a	17a	14a	20a	20a	22a
F30 (*F. culmorum*)	13a	19a	15a	18a	15a	20a	19a	20a
F7 (*F. solani*)	14a	19a	17a	17a	14a	21a	19a	21a
F20 (*F. solani*)	13a	19a	16a	17a	15a	21a	18a	22a

F26 (*F. solani*)	15a	19a	16a	18a	15a	21a	19a	22a
F29 (*F. solani*)	15a	17a	17a	18a	15a	20a	19a	21a
F31 (*F. solani*)	14a	18a	16a	18a	15a	20a	19a	21a
F35 (*F. solani*)	13a	19a	18a	18a	15a	21a	17a	21a
F15 (*F. verticillioides*)	13a	19a	16a	17a	14a	19a	19a	22a
F16 (*F. verticillioides*)	14a	18a	15a	17a	14a	20a	17a	22a
F21 (*F. verticillioides*)	15a	17a	14a	19a	16a	20a	19a	21a
F27 (*F. verticillioides*)	15a	18a	15a	18a	15a	19a	18a	21a
F43 (*F. equiesti*)	14a	18a	16a	18a	14a	20a	17a	21a
DI (CC)								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	15a	19a	16a	18a	14a	21a	17a	22a
F2 (*F. culmorum*)	14a	20a	15a	18a	14a	20a	18a	21a
F3 (*F. culmorum*)	13a	20a	16a	18a	13a	20a	19a	21a
F28 (*F. culmorum*)	14a	19a	16a	18a	15a	21a	17a	22a
F30 (*F. culmorum*)	14a	18a	17a	18a	15a	22a	17a	20a
F7 (*F. solani*)	15a	18a	15a	18a	15a	21a	17a	21a
F20 (*F. solani*)	15a	17a	17a	18a	14a	19a	18a	22a
F26 (*F. solani*)	15a	18a	16a	18a	15a	19a	18a	22a
F29 (*F. solani*)	15a	19a	17a	19a	15a	20a	18a	21a
F31 (*F. solani*)	14a	17a	16a	18a	14a	20a	18a	21a
F35 (*F. solani*)	16a	19a	17a	17a	14a	21a	17a	21a
F15 (*F. verticillioides*)	13a	17a	18a	19a	15a	20a	17a	22a
F16 (*F. verticillioides*)	14a	18a	15a	17a	15a	22a	17a	22a
F21 (*F. verticillioides*)	14a	17a	15a	19a	14a	21a	17a	21a
F27 (*F. verticillioides*)	16a	19a	14a	19a	15a	22a	18a	21a
F43 (*F. equiesti*)	15a	17a	16a	18a	16a	20a	18a	21a
DS (CC)								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	15a	19a	17a	18a	16a	22a	17a	22a
F2 (*F. culmorum*)	15a	20a	17a	18a	16a	22a	18a	21a
F3 (*F. culmorum*)	14a	18a	15a	19a	15a	22a	19a	21a
F28 (*F. culmorum*)	15a	19a	15a	17a	17a	22a	17a	22a
F30 (*F. culmorum*)	14a	18a	15a	16a	17a	22a	17a	20a
F7 (*F. solani*)	14a	17a	15a	19a	15a	19a	17a	21a
F20 (*F. solani*)	16a	18a	15a	18a	14a	20a	18a	22a
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F29 (*F. solani*)	13a	17a	16a	19a	16a	22a	18a	21a
F31 (*F. solani*)	14a	17a	17a	18a	14a	20a	18a	21a
F35 (*F. solani*)	14a	17a	17a	17a	14a	20a	17a	21a
F15 (*F. verticillioides*)	14a	19a	16a	17a	15a	21a	17a	22a
F16 (*F. verticillioides*)	13a	18a	17a	17a	17a	20a	17a	22a
F21 (*F. verticillioides*)	14a	20a	15a	18a	15a	21a	17a	21a
F27 (*F. verticillioides*)	15a	18a	16a	18a	16a	19a	18a	21a
F43 (*F. equiesti*)	15a	19a	16a	18a	14a	20a	18a	21a
AUDPC calculated on the basis of DI (CC)								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
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F2 (*F. culmorum*)	15a	19a	15a	18a	15a	20a	18a	21a
F3 (*F. culmorum*)	15a	20a	15a	17a	15a	21a	19a	21a
F28 (*F. culmorum*)	15a	19a	16a	17a	16a	21a	17a	22a
F30 (*F. culmorum*)	13a	19a	15a	18a	16a	22a	17a	20a
F7 (*F. solani*)	15a	18a	15a	17a	16a	22a	17a	21a

F20 (*F. solani*)	15a	19a	16a	18a	14a	21a	18a	22a
F26 (*F. solani*)	15a	18a	17a	19a	15a	22a	18a	22a
F29 (*F. solani*)	13a	17a	16a	17a	15a	22a	18a	21a
F31 (*F. solani*)	14a	17a	16a	17a	14a	20a	18a	21a
F35 (*F. solani*)	13a	17a	16a	16a	16a	21a	17a	21a
F15 (*F. verticillioides*)	13a	19a	16a	17a	15a	21a	17a	22a
F16 (*F. verticillioides*)	15a	18a	17a	17a	16a	22a	17a	22a
F21 (*F. verticillioides*)	14a	20a	15a	18a	15a	21a	17a	21a
F27 (*F. verticillioides*)	15a	18a	17a	19a	16a	21a	18a	21a
F43 (*F. equiesti*)	14a	17a	16a	18a	14a	20a	18a	21a
AUDPC calculated on the basis of DS (CC)								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	13a	19a	17a	18a	16a	21a	17a	22a
F2 (*F. culmorum*)	13a	19a	15a	18a	15a	22a	18a	21a
F3 (*F. culmorum*)	15a	18a	17a	19a	13a	22a	19a	21a
F28 (*F. culmorum*)	13a	19a	17a	17a	16a	22a	17a	22a
F30 (*F. culmorum*)	15a	17a	17a	17a	16a	22a	17a	20a
F7 (*F. solani*)	15a	19a	16a	18a	16a	20a	17a	21a
F20 (*F. solani*)	15a	18a	16a	17a	14a	20a	18a	22a
F26 (*F. solani*)	16a	19a	16a	17a	15a	20a	18a	22a
F29 (*F. solani*)	16a	18a	18a	17a	14a	22a	18a	21a
F31 (*F. solani*)	14a	18a	16a	18a	15a	22a	18a	21a
F35 (*F. solani*)	14a	19a	18a	19a	14a	22a	17a	21a
F15 (*F. verticillioides*)	16a	17a	16a	18a	15a	22a	17a	22a
F16 (*F. verticillioides*)	13a	17a	17a	17a	15a	22a	17a	22a
F21 (*F. verticillioides*)	14a	19a	15a	18a	15a	22a	17a	21a
F27 (*F. verticillioides*)	15a	17a	17a	18a	15a	22a	18a	21a
F43 (*F. equiesti*)	15a	19a	16a	18a	14a	22a	18a	21a
DI (FC)								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	13a	19a	17a	18a	14a	22a	17a	22a
F2 (*F. culmorum*)	13a	20a	17a	17a	14a	21a	18a	20a
F3 (*F. culmorum*)	13a	20a	16a	18a	15a	21a	19a	21a
F28 (*F. culmorum*)	14a	18a	17a	16a	15a	22a	17a	22a
F30 (*F. culmorum*)	14a	19a	15a	17a	14a	21a	17a	22a
F7 (*F. solani*)	15a	19a	17a	18a	15a	21a	17a	21a
F20 (*F. solani*)	15a	20a	16a	18a	16a	19a	18a	21a
F26 (*F. solani*)	15a	18a	16a	19a	15a	20a	19a	22a
F29 (*F. solani*)	14a	18a	15a	17a	16a	20a	17a	20a
F31 (*F. solani*)	14a	18a	16a	17a	14a	19a	17a	21a
F35 (*F. solani*)	15a	17a	15a	17a	15a	20a	18a	22a
F15 (*F. verticillioides*)	13a	19a	16a	19a	15a	20a	19a	22a
F16 (*F. verticillioides*)	15a	18a	17a	17a	16a	21a	17a	22a
F21 (*F. verticillioides*)	14a	19a	15a	19a	15a	19a	17a	21a
F27 (*F. verticillioides*)	14a	18a	17a	18a	16a	21a	17a	21a
F43 (*F. equiesti*)	15a	18a	16a	19a	14a	20a	18a	21a
DS (FC)								
Fungal isolates (identification)	**Acsad65**	**Cham4**	**Cham7**	**Douma4**	**Cham9**	**Bohoth10**	**Arabi Abiad**	**Arabi Aswad**
F1 (*F. culmorum*)	14a	18a	16a	17a	15a	20a	17a	22a
F2 (*F. culmorum*)	13a	19a	16a	18a	14a	21a	18a	22a
F3 (*F. culmorum*)	14a	19a	16a	18a	14a	21a	19a	20a
F28 (*F. culmorum*)	13a	19a	15a	17a	15a	22a	17a	21a
F30 (*F. culmorum*)	14a	18a	15a	17a	14a	21a	17a	21a

F7 (*F. solani*)	14a	18a	15a	17a	15a	20a	17a	21a
F20 (*F. solani*)	15a	18a	16a	18a	14a	20a	18a	22a
F26 (*F. solani*)	15a	18a	16a	18a	15a	20a	17a	19
F29 (*F. solani*)	14a	18a	16a	17a	14a	21a	18a	21a
F31 (*F. solani*)	14a	17a	16a	18a	14a	21a	19a	22a
F35 (*F. solani*)	14a	17a	17a	17a	14a	20a	17a	21a
F15 (*F. verticillioides*)	13a	18a	16a	18a	15a	20a	17a	22a
F16 (*F. verticillioides*)	13a	18a	16a	17a	15a	21a	17a	20a
F21 (*F. verticillioides*)	14a	18a	15a	17a	15a	21a	18a	22a
F27 (*F. verticillioides*)	15a	18a	15a	18a	15a	21a	18a	20a
F43 (*F. equiesti*)	15a	17a	16a	18a	14a	20a	18a	21a

Values are means of three replicates. According to the Fisher's LSD test, values for the same cultivar treated with root Si irrigation among the 16 FHB isolates followed by the same letter in same column are not significantly different at $P<0.05$. Pathogenic reactions upon Si of all cultivars infected with *Fusarium* fungi were previously evaluated according to methods described by Sakr and Kurdali [28, 29]. Values of reductions of the four indices measured under controlled conditions on Arabi Aswad and Arabi Abiad were presented previously by Sakr [35]

The positive impact of Si on augmenting host resistance against *Fusarium* development and fungal grwoth in the young and adult *Triticum* and *Hordeum* parts showed that the nine components evaluated in this study were negatively impacted by Si at 1.7 mM (Figure 2). Since Si is known to decrease the damage of fungal diseases in several crops [18, 19, 22], the score of this element in the young and adult wheat and barley parts was carefully equilibrated in all applications to express its potential capacity in the suppression of FHB disease. In field researches to detected the efficacy of Si for the prevention of plant diseases, Si level does not exceed 1.67 mM [23]. Recently, Si amendments to the soil as a fertilizer has been shown to decrease FHB DI and DS [28, 29, 36], particularly in combination with fungicide applications [27]. Several reports have shown the active participation of Si in mediating host resistance against fungal pathogens, involving *Blumeria graminis* causing powdery mildew [37, 38], *Bipolaris sorokiniana* causing spot blotch [39], *Drechslera tritici-repentis* causing tan spot [40], *F. culmorum, F. verticillioides, F. solani, F. equiseti* causing head blight [28, 29] and *Magnaporthe grisea* causing blast [41, 42]. A field report showed the impact of fertilization of soil with calcium silicate in decreasing FHB severity in wheat [36]. The impact of Si treatment to roots versus leaves in reducing FHB DI and DS was compared, and the data exhibited that neither application decreased DI nor DS during the initial infection stage. severity in wheat [36]. exhibited that neither application decreased DI nor DS during the initial infection stage. However, both characters were significantly decreased two weeks following initial infection, showing that successful decrease of *Fusarium* needs a minimum Si concentration to accumulate in the host tissues to modulate defense responses [28, 29].

An *in vitro* bio-experiment on *Triticum* and *Hordeum* showed that *Fusarium* pathogens can proliferate on the host surface, regardless of Si treatment, suggesting that Si may not act directly on *Fusarium* pathogens [28, 29]. Yobo *et al.* [43] tested the efficacy of treating potassium silicate under greenhouse conditions; however, their observations revealed no significant decrease in FHB severity in wheat. There is some evidence that the interaction of *Fusaruim* pathogens with Si treated plants ameliorates disease. Two novel reports present data that Si amendments were linked with FHB reduction in wheat and barley [28, 29]. However, it was also reported in these studies that Si treatments did not decrese hyphal growth, and that DON contamination in grains was more severe than in un-amended controls in susceptible cultivars [27, 28, 29, 36, 43]. For clarity, more work efforts are requisted to uncover the association of Si to FHB. Importantly, Si application at 1.7 mM reduced head blight damage in young and adult *Triticum* and *Hordeum* organs at the earliest and latest growth stages under *in vitro*, controlled and field conditions [28, 29], suggesting that Si equally augmented the expression of host resistance to FHB colonization and development in seedlings and adult wheat and barley plants.

Figure 2. Silicon applications at 1.7 mM enhance barley resistance to Fusarium head blight. FHB disease suppression on Arabi Abiad barley heads in response to silicon adding to soil at 21 days post inoculation under field conditions; (a) a non inoculated barley head treated with sterile distilled water, (b) a barley head inoculated with FHB pathogen using an artificial head inoculation assay and no silicon root application and (c) a barley head inoculated with FHB pathogen using an artificial head inoculation assay and multiple silicon root applications at 1.7 mM

It appears that Si modulates multiple similarly signaling pathways involved in head and seedlings [5, 11] reaction to *Fusarium* infection. Increasing confirmation also showed that Si plays a function in numerous key components in plant signaling systems [19, 20]. In contrast to our observations, no influence of soil Si on blast disease intensity in field plots at 34 days but found a significant linear decline in blast severity with augmenting Si concentration when the plots were re-sampled at 74 days [44]. Vu *et al.* [35] showed a marked plant age impact on the intensity of bacterial blight: disease lesions assessed at 32 days were largely unaffected by soil Si, but at 59 days, the impacts became increasingly typical. The more apparent impacts in older, field-grown plants are possibly due to a greater accumulation of Si in plant tissues as they age [35, 44].

3. Conclusion

Research on Si treatment for *Triticum* and *Hordeum* FHB disease decrease is its infancy; however, Si may provide an additional component for the control of FHB in wheat and barley, as it ameliorates plant defense reactions. For the first time, our current data indicate that Si nonspecifically affects aggressiveness in FHB pathogens. Our observations show that Si could be a valuable tool in integrated FHB management by suppressing pathogen development on wheat and barley when affected by *Fusarium*. Most importantly, no hazard exists to emergence of Si-resistant pathogen populations upon Si applications on diverse FHB populations. More physiological, cytological and biochemical analyses would be required to explore how Si can enhance small grain-cereal defenses to *Fusarium* invasion. All

of these data are promising outcomes for the application of Si as an effective and safe control method against FHB damage.

Compliance with Ethics Requirements. Author declares that he respects the journal's ethics requirements. Author declares that that he has no conflict of interest and all procedures involving human or animal subjects (if exist) respect the specific regulation and standards.

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