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Evidence of isolate-specific resistance in wheat and barley challenged with four *Fusarium* pathogens causing head blight

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Abstract

Quantitative resistance (QR) interacting with the aggressiveness of pathogens is found to be isolate-nonspecific and durable. However, minor isolate-specific reactions have been observed in various pathosystems rendering QR efficiency to be lost as pathogen isolates adapt to it. To test this hypothesis in Fusarium head blight (FHB) associated with devastating agronomic effects on cereals, we elucidated pathogenic interactions between eight cultivars with contrasting in QR resistance of durum wheat, bread wheat and barley and 16 fungal isolates with diverse aggressiveness to explore whether specific reactions exist between pathogen isolates and host that might be included in QR eroding. The reaction of cultivars to FHB species revealed as difference in the level of head blight damage caused by diverse isolates was evaluated by measuring nine aggressiveness components at the earliest and latest plant stages. Combined analysis of bio-experiments demonstrated that isolate × cultivar interactions were significant. QR stability in cultivars to FHB infection was fulfilled over several experimental conditions; however, FHB isolates showed a different ranking to all cultivars for the majority of the correlations testing the stability of aggressiveness. Our data suggest that QR of wheat and barley to *Fusarium* is possibly explained by minor isolate-specific (quantitative trait loci) QTLs and QTLs that confer resistance to a large number of fungal isolates as well. These findings confirm the minor-gene for minor-gene system which suggests specific reactions between *Fusarium* isolates and QTLs for resistance. To our best knowledge, this report has highlighted for the first time that among of the major risk components for the erosion of QR observed in *Triticum* and *Hordeum* to head blight is the specificity of the resistance.

Keywords: *Fusarium* pathogens, *Hordeum*, isolate specificity, resistance erosion, *Triticum*.

1.Introduction

Genetic resistance to plant pathogens is progressively being exhibited an important option to techniques of disease management relied on the application of chemicals [1, 2]. Commonly speaking, in several plant-pathogen interactions two resistance types cooperatively exist [3]. Isolate/race-specific resistance is obviously expressed qualitatively. This isolate/race specificity has been elucidated by postulating a gene for gene relationship that shows a near-complete control to disease [4]. Quantitative resistance (QR) interacting with aggressiveness (i.e., the capacity of a pathogen isolate to cause disease on a susceptible host) is dominated by one to many minor genes, the seeming quantitative trait loci (QTLs). It is originally found to isolate/race nonspecific and more stable than isolate/race-specific resistance [4]; thus it results in a decrease in pathogen damage rather than the lack of disease [5]. Van der Plank [6] hypothesized that the QR genes are equally efficient for all pathogen isolates. In fact, small isolte/race-specific impacts have been reported, for instance, in the polygenic QR of *Hordeum* to *Puccinia hordei*, i.e., leaf rust, considered to be of a race-nonspecific background [7]. Identical observations were found by Truong *et al.* [8], Chen *et al.* [9] and Le Clerc *et al.* [10] in pepper, mustard and carrot when recognizing isolate/race-specific QR resistance to *Phytophthora capsici*, *Plasmodiophora brassicae* and *Alternaria dauci*, respectively. These interactions can only be elucidated by postulating a minor-gene for minor-gene relationship according to Parlevliet and Zadoks [11], in an identical tendency as the major gene-for major gene

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relationships for the system recognized in qualitative resistance. The effectiveness of a QR depended on an isolate-specific QTL is predicted to reduce over time because resistant plants select pathogen isolates with a high level of aggressiveness [5]. These specific relationships may render quantitative resistances non-stable in an identical way than qualitative resistance [4]. Nevertheless, the outcomes of specific relationships on the durability of QR are still not well reported [1, 2].

Production of bread wheat (*Triticum aestivum*), durum wheat (*T. durum*) and barley (*Hordeum vulgare*) is directly menaced by *Fusarium* pathogens causing head blight (FHB), one of the most destructive and common fungal diseases of small-grain cereals [12]. It leads to considerable losses in crop production, quality and safety due to the risk of grain contamination by a board variation of mycotoxins [13]. FHB is caused by more than 17 *Fusarium* species of which *F. culmorum* and *F. graminearum* are of greatest existence and considered the strongest pathogenic pathogens [14]. The employment of quantitatively resistant wheat and barley cultivars is a fundamental component of a sustainable control policy of FHB [15]. FHB is under polygenic inheritance [12] that does not include major qualitative genes [14]. Several FHB-resistance QTLs were found in *T. aestivum*, while at most a restricted number of little influence QTL for head blight resistance have been recognized in *T. durum*. Relative to the main resistance QTL (*fhb1*, which participates to less than 40% of the resistance to head blight distinguished in *T aestivum*), impacts of *Fusarium* resistance QTL in *T. durum* are comparatively small (~10%) [16]. In *H. vulgare*, the number of distinguished QTLs changes in several studies, varying from only 1 QTL allele, to 2 QTLs, and up to 10 QTLs. Yet, limited sources of *Fusarium* resistance have been identified in *H. vulgare* and their QR level is modest [17].

The complicated interactions between cereal hosts and head blight pathogens is dynamic [12], and emergence of more pathogenic *Fusarium* isolates has been found in the fields [15]. The high genetic diversity for aggressiveness in FHB pathogens necessities coincident combination of many resistance genes to remain efficient in genotypes utilized over a large area [14, 16, 17]. In previous *in vitro* studies [18, 19], we showed that the QR reduced over time via the selection of isolates that were aggressive on both susceptible and resistant wheat and barley cultivars. In the above-cited mentioned reports, the question of the isolate-specificity of QR that could be included in eroding resistance was not elucidated. Hence, comprehension the complex relationships between QTLs genes and *Fusarium* pathogens could be an effective policy to manage FHB disease by breeding programs [12]. Recombinant inbred line (RIL) population has been used in many reports [7, 8, 9, 10] to explore to what extent QTLs participated to QR are isolate-specific in their impact. Such a perspective may throw light upon the presence of the minor-gene for minor-gene system presumed by Parlevliet and Zadoks [11] as a base for QR. However, RIL populations contributed to isolate-specific QR in wheat and barley challenged with *Fusarium* are not available till now. Thus, the most effective approach to follow potential specific relationships in pathogen populations seems to be via fungal inoculations and phenotypic head blight evaluations at the seedling and adult plant stages as described before by Krenz *et al.* [20] in wheat infected with *Mycosphaerella graminicola*.

The aim of this work was to elucidate specific reactions potentially included in the erosion of QR using *Fusarium* isolates with different aggressiveness and wheat and barley cultivars with contrasting in FHB resistance. We were asking whether these isolates are interacting with host cultivars in a similar or different way. In our study, significant isolate × host cultivar interactions were utilized as indications of specificity. When differential pathogenic responses will be observed, this would also indicate that the stability of aggressiveness is not achieved and isolate-specific resistance is fulfilled.

2. Materials and Methods

2. 1. Small-grain cereals, *Fusarium* isolates, and inoculum preparation

Eight small-grain cereal cultivars, i.e., two *H. vulgare* cultivars: Arabi Abiad (AB) and Arabi Aswad (AS) and three *T. aestivum* and three *T. durum* cultivars, of Syrian origin including with different genetic background and resistance levels to exhibit a gradual level of head blight resistance were used [21] as plant material. A wheat cultivar Acsad65 (durum) susceptible, and Cham7 and Cham9 (durum) susceptible to moderately susceptible, bread wheat and barley cultivars AB, Cham4 and Douma4 moderately susceptible, and AS and Bohoth10 (bread), moderately resistant,

based on previous FHB resistance assays by inoculation of detached leaves, seedlings, spikes and spikelets [21], were chosen due to their identical anthesis and maturity dates, and board usage in the market.

All *Fusarium* isolates, including six *F. solani*, five *F. culmorum*, four *F. verticillioides* (synonym *F. moniliforme*) isolates and one *F. equiseti* isolate, were sampled from naturally infected wheat grains. The 16 monosporic derived cultures of the field-background were chosen for their diverse aggressiveness established on earlier various experimental observations [21]. On Petri dishes with potato dextrose agar (PDA) with 13 mg/l kanamycin sulphate added after autoclaving, the isolates were morphologically identified with the aid of the Leslie and Summerell [22] manual on the bases of microscopic observations of the size and shape of micro- and macro-conidia, and were molecularly distinguished by Random amplification of polymorphic DNA markers [21]. These 16 *Fusarium* isolates were used in previous experiments conducted under *in vitro*, growth chamber and field trials to assess resistance levels of all wheat and barley cultivars [21], and resistance levels were correctly and precisely distinguished.

Single spore isolates were stored short term on PDA at 4°C and long term by freezing at-16°C or in sterile distilled water at 4°C [23], and fresh cultures were produced on PDA medium. After 14 days, conidia were collected in sterile distilled water (SDW). Then, the suspensions were filtered through two layers of sterile cheesecloth to remove agar and adhering mycelia. The spore concentration was adjusted under an optical microscope prior to use with the aid of a Neubauer chamber and diluted to appropriate concentrations as inoculum sources.

2.2. Measuring of aggressiveness components under several experimental conditions

In order to explore the isolate-specific resistance revealed as difference in the level of head blight damage caused by diverse isolates in host materials challenged with Fusaria, the reaction of eight *Triticum* and *Hordeum* cultivars to a set of 16 *Fusarium* isolates was evaluated by measuring nine aggressiveness components at the adult and seedling plant stages. Pathogenic responses of all cultivars infected with *Fusarium* fungi were previously evaluated according to techniques evaluated by Sakr [21]: area under disease progress curve of Petri-dish infection, latent period detached leaf infection, and coleoptile length reduction of a coleoptile inoculation determined *in vitro*, disease severity (DS) and disease incidence (DI) determined utilizing a detached head test in a growth chamber, and DI determined utilizing a spike artificial infection and DS determined utilizing a spikelet artificial infection in the growth chamber, DI and DS determined utilizing a spike artificial infection in the filed over three consecutive growing seasons. For testing the stability of aggressiveness used to determine the isolate-specific resistance, we correlated values of a given aggressiveness criterion among the eight wheat and barley cultivars giving a total of 28 possible comparisons. Thus, we provided 252 possible comparisons for the nine aggressiveness criteria over several experimental conditions. A significant correlation refers to a non-isolate specific resistance and non-significant correlation refers to an isolate-specific resistance. For a given aggressiveness criterion, the experiment system was subjected in a completely randomized block design with 3 replicates for each isolate and cultivar and the experiment was repeated twice.

2.3. Data analysis

The observed findings were arranged to analysis of variances (ANOVA) utilizing DSAASTAT add-in version 2011. Prior to statistical analysis to stabilize variances, the percentages were angular transformed. ANOVA involving the Fisher's LSD test at $P<0.05$ was utilized to determine the host cultivar × isolate interactions. The overall values per isolates were involved in the calculation of the sample correlation coefficients (Pearson r) at $P<0.05$.

3. Results and Discussion

QR is the basis of breeding for FHB disease resistance in small-grains host plants, i.e., wheat and barley [12], particularly to obtain durable resistance [15]. Taking a larger variation of cultivars and isolates in terms of QTLs and aggressiveness into account [16-17], we can therefore ask ourselves about possible isolate-race resistance impacts between wheat and barley cultivars and FHB isolates. Our earlier *in vitro* observations suggested that resistance of moderately resistant and susceptible wheat and barley cultivars eroded gradually to head blight pathogens [18, 19]. Alternations in the *Fusarium* populations may include either augmented pathogenicity generally or a modification in specificity with elevated pathogenicity on cultivars with a particular QR gene

[18, 19]. In this research, we showed pathogen-host specificity participated in possible erosion of QR.

It is widely accepted that resistant cereal cultivars present consistent resistance to nearly all FHB isolates globally [14]. In our investigation, combined analysis of bio-experiments demonstrated that isolate × cultivar interactions were significant (Table 1). These findings are in harmony with earlier reports that showed a significant interaction between wheat and barley and isolates of *Fusarium* pathogens [24]. In other pathogen-host interactions such as *Phoma*-sunflower, leaf blast-rice and *Septoria tritici* blotch-wheat [25, 26, 27], identical impacts have been observed. The above-mentioned results indicate that these hosts may possess diverse genes for resistance to the respective pathogen species. However, Miedaner *et al.* [28] found in their study about the resistance of four small-grain cereals (triticale, bread wheat, rye and durum wheat) challenged with FHB fungi that cereal × isolate and cultivar × isolate reactions were not significant.

Table 1. Analyses of variance for latent period (LP) of detached leaf inoculation, area under disease progress curve (AUDPC) of Petri-dish inoculation and coleoptile length reduction (CL) of a coleoptile infection detected in vitro, disease incidence (DI, Type I) and disease severity (DS, Type II) detected using a detached head test (DHT) under controlled conditions, and disease incidence (DI^{CC}, Type I) detected using a head artificial inoculation and disease severity (DS^{CC}, Type II) detected using a floret artificial inoculation under controlled conditions in a growth chamber, disease incidence (DI, Type I) and disease severity (DS, Type II) detected using a head artificial inoculation under field conditions (FC) over the three growing seasons (F-test values)

Source of variation	*df*	LP, AUDPC, CL, DI^{DHT}, Type I, DS^{DHT}, Type II, DI^{CC}, Type I, DS^{CC}, Type II, DI^{FC}, Type I, DS^{FC}, Type II
Cultivar (C)	7	++
Isolate (I)	15	++
C × I	105	++
Error	256	

++ – significant at 1% level; *df* – degree of freedom

Pathogenic reaction of all isolates on the eight wheat and barley cultivars was earlier analyzed and presented by Sakr (2023b).

Efficient use of appropriate QTL genes in QR breeding necessities QTL to be durably expressed over years and environments [2, 5]. Several reports in different host plants characterize QTLs for disease resistance that are durable over various environments, involving researches exploring QR to oilseed rape/blackleg, barley/leaf rust, bean/white mold, pea/Aphanomyces root rot, potato/late blight [29, 30, 31, 32] interactions. In the current research, QR stability in eight wheat and barley cultivars to FHB infection was fulfilled over several experimental conditions, i.e., seedling and adult plant stages, as well as across years. The reliability of this cultivar ranking was confirmed by the significant correlations among the resistance measured by the nine pathogenic criteria on host cultivars inoculated with a set of *Fusarium* isolates of four pathogen species (Table 2). In any case, the order of the cultivars was identical, in spite of the isolates inoculated. All wheat and barley cultivars infected with either high aggressive or low aggressive isolates exhibited a qualitative pattern. It then is probable that a major QTL gene with incomplete expression governs *Fusarium* resistance in wheat and barley as observed previously in wheat infected with *Mycosphaerella graminicola* [20]. It is accepted for QR that cultivar order relying on disease intensity is independent of the isolates used for assessing the resistance [6]. Our data agree with those found by Miedaner *et al.* [28] in which the cultivars showed an identical order as analyzed with FHB isolates with different aggressiveness levels. Another prominent example in FHB-wheat relationship, failure of QR in Sumai 3, released for 50 years ago, has not been observed, and it is still the best source globally for resistance to symptoms development in the head [12]. Le Clerc *et al.* [10] found that the host order was the similar upon inoculation with fungal isolates varying in their aggressiveness under several experimental conditions in the pathosystem of carrot and *Alternaria dauci*. Our data suggest that QR of wheat and barley to *Fusarium* is possibly elucidated by major QTLs that confer resistance to a large number of fungal isolates.

Table 2. Correlation coefficients between the resistance measured by latent period (LP) of detached leaf inoculation, area under disease progress curve (AUDPC) of Petri-dish inoculation and coleoptile length reduction (CL) of a coleoptile infection detected in vitro, disease incidence (DI, Type I) and disease severity (DS, Type II) detected using a detached head test (DHT) under controlled conditions, and disease incidence (DI^{CC}, Type I) detected using a head artificial inoculation and disease severity (DS^{CC}, Type II) detected using a floret artificial inoculation under controlled conditions in a growth chamber, disease incidence (DI, Type I) and disease severity (DS, Type II) detected using a head artificial inoculation under field conditions (FC) over the three growing seasons on eight wheat and barley cultivars of Syrian origin infected with a set of 16 fungal isolates of four *Fusarium* head blight species

Resistance component	AUDPC	LP	CL	DI^{DHT}, Type I	DS^{DHT}, Type II	DI^{CC}, Type I	DS^{CC}, Type II	DI^{FC}, Type I	DS^{FC}, Type II
AUDPC	1.000								
LP	-0.756*	1.000							
CL	-0.970***	0.742*	1.000						
DI^{DHT}, Type I	0.943***	-0.878**	-0.871**	1.000					
DS^{DHT}, Type II	0.991***	-0.731*	-0.950***	0.956***	1.000				
DI^{CC}, Type I	0.956***	-0.887**	-0.902**	0.988***	0.958***	1.000			
DS^{CC}, Type II	0.991***	-0.769*	-0.954***	0.971***	0.989***	0.976***	1.000		
DI^{FC}, Type I	0.826**	0.806*	-0.786*	0.799*	0.876**	0.811**	0.836**	1.000	
DS^{FC}, Type II	0.741*	0.765*	-0.740*	0.769*	0.785*	0.709*	0.730*	0.964***	1.000

(P<0.05)*, (P<0.01)**, (P<0.001)***.

Table 3. Correlation coefficients between aggressiveness components: latent period (LP) of detached leaf inoculation, area under disease progress curve (AUDPC) of Petri-dish inoculation and coleoptile length reduction (CL) of a coleoptile infection detected in vitro, disease incidence (DI) and disease severity (DS) detected using a detached head test (DHT) under controlled conditions, and disease incidence (DI^{CC}) detected using a head artificial inoculation and disease severity (DS^{CC}) detected using a floret artificial inoculation under controlled conditions in a growth chamber, disease incidence (DI) and disease severity (DS) detected using a head artificial inoculation under field conditions (FC) over the three growing seasons on eight wheat and barley cultivars of Syrian origin infected with a set of 16 fungal isolates of four Fusarium head blight species

LP								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.211ns	1.000						
Cham7	0.088ns	0.274ns	1.000					
Douma4	0.001ns	0.508*	0.302ns	1.000				
Cham9	0.159ns	0.404ns	0.384ns	0.438ns	1.000			
Bohoth10	0.099ns	0.224ns	-0.041ns	0.378ns	0.457ns	1.000		
Arabi Abiad	0.596*	-0.403ns	0.325ns	0.123ns	0.187ns	0.055ns	1.000	
Arabi Aswad	0.047ns	0.315ns	0.582*	0.490ns	0.350ns	0.325ns	0.575*	1.000
AUDPC								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1							
Cham4	-0.211ns	1.000						
Cham7	0.088ns	0.274ns	1.000					
Douma4	0.001ns	0.508*	0.302ns	1.000				
Cham9	0.159ns	0.404ns	0.384ns	0.438ns	1.000			
Bohoth10	0.099ns	0.224ns	-0.041ns	0.378ns	0.457ns	1.000		
Arabi Abiad	0.596*	-0.403ns	0.325ns	0.123ns	0.187ns	0.055ns	1.000	
Arabi Aswad	0.047ns	0.315ns	0.582*	0.490ns	0.350ns	0.325ns	0.575*	1.000
CL								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.175ns	1.000						

Table 3. *(continuation)*

Arabi Aswad	-0.188ns	0.137ns	0.085ns	0.522*	0.114ns	0.112ns	0.293ns	1.000
DI (DHT)								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.175ns	1.000						
Cham7	0.428ns	-0.021ns	1.000					
Douma4	-0.430ns	0.548*	0.022ns	1.000				
Cham9	-0.223ns	-0.116ns	0.112ns	0.098ns	1.000			
Bohoth10	-0.234ns	0.423ns	-0.017ns	0.428ns	0.246ns	1.000		
Arabi Abiad	0.610*	-0.379ns	0.273ns	-0.100ns	0.203ns	-0.242ns	1.000	
Arabi Aswad	-0.188ns	0.137ns	0.085ns	0.522*	0.114ns	0.112ns	0.293ns	1.000
DS (DHT)								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.175ns	1.000						
Cham7	0.428ns	-0.021ns	1.000					
Douma4	-0.430ns	0.548*	0.022ns	1.000				
Cham9	-0.223ns	-0.116ns	0.112ns	0.098ns	1.000			
Bohoth10	-0.234ns	0.423ns	-0.017ns	0.428ns	0.246ns	1.000		
Arabi Abiad	0.610*	-0.379ns	0.273ns	-0.100ns	0.203ns	-0.242ns	1.000	
Arabi Aswad	-0.188ns	0.137ns	0.085ns	0.522*	0.114ns	0.112ns	0.293ns	1.000
DI (CC)								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.175ns	1.000						
Cham7	0.428ns	-0.021ns	1.000					
Douma4	-0.430ns	0.548*	0.022ns	1.000				
Cham9	-0.223ns	-0.116ns	0.112ns	0.098ns	1.000			
Bohoth10	-0.234ns	0.423ns	-0.017ns	0.428ns	0.246ns	1.000		
Arabi Abiad	0.610*	-0.379ns	0.273ns	-0.100ns	0.203ns	-0.242ns	1.000	
Arabi Aswad	-0.188ns	0.137ns	0.085ns	0.522*	0.114ns	0.112ns	0.293ns	1.000
DS (CC)								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.461ns	1.000						
Cham7	0.076ns	0.339ns	1.000					
Douma4	0.562*	-0.312ns	0.134ns	1.000				
Cham9	0.509*	-0.312ns	0.134ns	0.335ns	1.000			
Bohoth10	-0.333ns	0.792***	0.028ns	-0.263ns	-0.263ns	1.000		
Arabi Abiad	0.759***	-0.365ns	0.025ns	0.619*	0.609*	-0.156ns	1.000	
Arabi Aswad	-0.276ns	0.476ns	0.227ns	-0.308ns	-0.308ns	0.637*	0.007ns	1.000
DI (FC)								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.461ns	1.000						
Cham7	0.076ns	0.339ns	1.000					
Douma4	0.562*	-0.312ns	0.134ns	1.000				
Cham9	0.552*	-0.301ns	0.204ns	0.331ns				
Bohoth10	-0.333ns	0.792***	0.028ns	-0.263ns	-0.263			
Arabi Abiad	0.759***	-0.365ns	0.025ns	0.619*	0.619*	-0.156	1.000	
Arabi Aswad	-0.276ns	0.476ns	0.227ns	-0.308ns	-0.308ns	0.637*	0.007ns	1.000
DS (FC)								
	Acsad65	Cham4	Cham7	Douma4	Cham9	Bohoth10	Arabi Abiad	Arabi Aswad
Acsad65	1.000							
Cham4	-0.461ns	1.000						
Cham7	0.076ns	0.339ns	1.000					
Douma4	0.562*	-0.312ns	0.134ns	1.000				
Cham9	0.501*	-0.406ns	0.251ns	0.154ns	1.000			
Bohoth10	-0.333ns	0.792***	0.028ns	-0.263ns	-0.263ns	1.000		
Arabi Abiad	0.759***	-0.365ns	0.025ns	0.619*	0.619ns	-0.156ns	1.000	
Arabi Aswad	-0.276ns	0.476ns	0.227ns	-0.308ns	-0.308ns	0.637*	0.007ns	1.000

(P<0.05)*, (P<0.01)**, (P<0.001)***, ns=not significant.

It is supposed that QR is usually governed by wide-spectrum resistance elements that are efficient versus a large number of isolates for one pathogen, and the order of the isolates depending on disease severity is independent of the cultivars employed for analyzing the aggressiveness [1]. In the FHB-cereal pathosytem, resistance to certain isolates of *Fusarium* species is proven to be a not isolate-specific in host cultivars [14]. The focus of this research, however, was on the order of sixteen FHB isolates of varying aggressiveness on eight wheat and barley cultivars. Our results showed that correlation values of the nine tested aggressiveness criteria among the host cultivars showed that less than 40 of the 252 possible comparisons were significantly correlated (Table 2). FHB isolates showed a different ranking to all cultivars for the majority of the correlations testing the stability of aggressiveness, indicating that isolate-specific resistance in *Triticum* and *Hordeum* infected with four *Fusarium* head blight pathogens is found. Nevertheless, our findings are not in harmony with those found by Miedaner *et al.* [28]; the correlations among isolates were all close to 1. The authors presumed that the utilized FHB isolates might have triggered the similar effectors independently from the cereal hosts and oppositely the point of invasion could be the identical in all cereal crops in spite of they might stimulate diverse QTLs/genes of the resistance. The present report shows that some QTLs for QR are isolate-specific and demonstrates plant stage-independent expression.

In the present investigation, the isolate specificity of the QTLs enhances the assumption that QR in the analyzed wheat and barley cultivars challenged with several FHB isolates with diverse aggressiveness may be established on a minor-gene for minor-gene relationship. The evidence analyzed here permits to the conclusion that isolate specificity of QR could participate in eroding resistance in wheat and barley [18, 19]. This focus on the necessity for better insight into the adaptive capacity of the fungi in order to predict the related stability of QR in cereal cultivars when exposed to diverse *Fusarium* populations. QR stability is probably to be reinforced by utilizing several QR sources that integrate with complementary resistance components [33], prioritizing sources of QR that decrease isolate-specific effects [34] and pathogen diversity for aggressiveness traits [35]. Some reports have previously characterized isolate-specific resistance components, proposing that differential relationship between isolate and plant may be exist in QR [7, 8, 9, 10]. Parlevliet [36] documented more than 40 years ago small but significant isolate-cultivar relationships in quantitatively resistant *H. vulgare* cultivars. This led him to suppose the 'minor-gene for minor-gene' postulate elucidate the nature of QR [11]. In fact, the above-mentioned examples together with the current findings demonstrate that minor-gene for-minor-gene reactions do exist in pathogen-plant interactions.

4. Conclusion

Regarding isolate-specific and isolate-nonspecific QTLs determined for QR to a set of 16 *Fusarium* isolates of four fungal species, it is proposed that both nonspecific and specific genes link with QR to FHB isolates. Adaptation to head blight resistance and specificity are more probably to exist when genetic variability is being preserved at a very high level. These data assure the minor-gene for minor-gene system proposing specific responses between *Fusarium* isolates and QTLs for QR. To our best knowledge, the current research has highlighted for the first time that among of the major risk factors for the erosion of QR observed in *Triticum* and *Hordeum* to FHB is the specificity of the resistance. The pyramiding of isolate-specific together with isolate-nonspecific QTLs could enhance the level of resistance to a board range of FHB isolates. Knowledge about the nature of QR will undoubtedly participate in the development of a policy for the control of *Fusarium* resistance durability.

Compliance with Ethics Requirements. Authors declare that they respect the journal's ethics requirements. Authors declare that that they have no conflict of interest and all procedures involving human or animal subjects (if exist) respect the specific regulation and standards.

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