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ADAPTATION OF FUSARIUM SPECIES CAUSING HEAD BLIGHT TO QUANTITATIVE RESISTANCE IN WHEAT: FIELD EVIDENCE FOR INCREASED AGGRESSIVENESS IN A NEW PATHOGEN POPULATION

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ABSTRACT

The use of quantitatively resistant wheat cultivars is an essential component of a sustainable management strategy of Fusarium head blight (FHB), caused by several *Fusarium* species. However, little information is available on the variation of aggressiveness of the newly emerging FHB collection compared to old one. It is therefore important to determine to what extent FHB populations can be selected for increased aggressiveness by wheat cultivars with several levels of quantitative resistance. To this end, FHB populations were sampled in 2005 (old population) and in 2015 (new population) from one of the major Syrian wheat production regions, chosen as a location where head blight occurs regularly. New and old FHB isolates were characterized for aggressiveness by single-floret inoculation under controlled conditions on eight durum and bread wheat cultivars of contrasting susceptibility to FHB, and molecularly distinguished using DNA markers. Results showed the new population caused a higher disease severity (ranging from 55% to 67%) than the old population. Thus, their aggressiveness increased between early and late samplings, suggesting that wheat plants cultivated over 10 years selected for increased aggressiveness during epidemics. Our comparative population genetic analyses with analyzed markers showed that the new population had more polymorphic loci compared with the old one. The information obtained in this study indicated that FHB populations adapt to prevailing wheat cultivars, irrespective of their resistance levels, and can therefore overcome polygenic, quantitative resistance. Adaptation to wheat resulting in increased pathogen aggressiveness that was not specific may render quantitative resistance nondurable if not properly managed.

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INTRODUCTION

The use of plant genetic resistance in agro-systems offers an interesting alternative to disease control methods based on the use of chemicals (Mundt, 2014; Sakr, 2023), but it also has a considerable effect on the evolutionary pathogen dynamics (McDonald and Linde, 2002) rendering resistance lacks durability due to the rapid pathogen evolution (Simmonds, 1991; Dangl and Jones, 2001). In the phytopathology literature, two extreme resistance categories for the resistance have historically been recognized (Parlevliet, 2002). Qualitative resistance faced with the qualitative component of pathogenicity, i.e., virulence, is based on a gene-for-gene relationship, and it confers a near-complete protection to disease (Dangl and Jones, 2001). The mechanisms of the rapid adaptation of the pathogen frequently resulting in the breakdown of major resistance genes have already been studied in details

(Mundt, 2014). Quantitative resistance associated with the presence of quantitative trait loci, QTLs, does not confer absolute protection (Simmonds, 1991), but is considered to be either be specific to some pathogen isolates or be effective against all known isolates of the pathogen (Cowger and Brown, 2019; Sakr, 2023). Over several generations, quantitative resistance can select for isolates with an increased rate of quantitative component of pathogenicity, i.e., aggressiveness which is dependent on the host, the pathogen and the interactions between them (McDonald and Linde, 2002), leading to erosion of the resistance (Mundt, 2014). Quantitative host resistance is assumed to be more durable than qualitative resistance, since it exerts less selective pressure on the pathogen (Simmonds, 1991; Dangl and Jones, 2001). However, QTLs overcome by a slow pathogen adaptation is poorly understood by contrast to qualitative resistance broken by a swift process in pathogens (Parlevliet, 2002), which make them difficult to predict their durability (Mundt, 2014). Alternations in the pathogen population may include either augmented aggressiveness in general or a variation in specificity with higher aggressiveness on cultivars with a particular quantitative resistance gene (Cowger and Brown, 2019; Sakr, 2023).

Fusarium head blight (FHB) is one of the most destructive diseases of bread wheat (*Triticum aestivum*) and durum wheat (*T. durum*) and other small-grain cereals (i.e., barley, oat, rye and triticale) on a global scale (Parry *et al.*, 1995). FHB can lead to severe yield and grain losses, decreasing grain weight as well as varying protein accumulation (Dweba *et al.*, 2017). In addition, FHB is able to biosynthesize and accumulate dangerous mycotoxins in harvested grain, secondary metabolites with toxic impacts on humans and animals (Fernando *et al.*, 2021). Several species in the genus *Fusarium* can cause FHB in wheat but *F. graminearum* and *F. culmorum* are generally considered the two most important and aggressive FHB causal agents worldwide. Other species, such as *F. equiseti*, *F. acuminatum*, *F. sporotrichioides* and *F. poae* notoriously show lower aggressiveness on wheat heads by causing reduced symptoms, and *F. avenaceum* has a modest aggressiveness (Bottalico and Perrone, 2002; Xue *et al.*, 2004; Xue *et al.*, 2019). Experiments about population alternations of FHB pathogens have showed the diffusion of highly aggressive *Fusarium* isolates (Fernando *et al.*, 2021).

FHB recently re-emerged as an important disease in the main producing areas, such as North America, Europe, and China, increasing the need for effective and durable control strategies (Dweba *et al.*, 2017). The use of quantitatively resistant wheat cultivars is an essential component of a sustainable management strategy of FHB (Xu and Nicholson, 2009). FHB is under polygenic inheritance that does not involve major gene and is modulated by the strong cultivar-by-environment interaction (Fernando *et al.*, 2021). Two main types of resistance to FHB have been identified: Type I, resistance to initial infection and Type II, resistance to the movement and spread of the fungi within the spike (Xu and Nicholson, 2009). Considerable QTLs for FHB resistance were reported in bread wheat, while only a limited number of little impact QTLs for FHB resistance have been recognized in durum wheat (Xu and Nicholson, 2009; Dweba *et al.*, 2017).

Since FHB species are highly variable pathogens, especially for aggressiveness (Bottalico and Perrone, 2002; Xue *et al.*, 2004; Xue *et al.*, 2019), it is therefore important to determine to what extent FHB populations can be selected for increased aggressiveness by wheat cultivars with several levels of quantitative resistance. To date, only one field study (Puri and Zhong, 2010) showed that newly emerging collection of *F. graminearum* isolates (since 2000) were significantly more aggressive than old pathogen isolates (before 2000); this difference in aggressiveness could vary with wheat cultivars released. Puri and Zhong (2010) suggested investigating whether FHB wheat resistance plays any role in aggressive evolution of new *F. graminearum* population.

For FHB-wheat researches in Syria, earlier studies have reported a great variation of aggressiveness in various *Fusarium* species collected regularly in 2005 to 2015 from farmer's fields at the Ghab Plain (Alazem, 2007; Sakr, 2019b, 2019a). It is one of the major Syrian agricultural production regions consisting of durum and bread wheat cultivars grown over several years and displayed varying levels of resistance to FHB disease agents (Alazem, 2007; Sakr, 2019b, 2019a). Recently, Sakr (2022a, 2022b) explored how the deployment of quantitative wheat and barley resistance affects changes of the aggressiveness under *in vitro* conditions which leaded to potential resistance erosion. These findings provide the first direct evidence that FHB pathogens evolve rapidly to adapt by increasing aggressiveness to

wheat and barley, indicating a risk of directional selection and possible erosion of FHB resistance Sakr (2022a, 2022b). However, no field evidence is available if quantitative resistance in wheat cultivated in Ghab Plain can select for isolates with higher aggressiveness.

The complex interactions between FHB pathogens and wheat host involve several genetic factors from both partners, the outcomes being quantitative resistances by cereals and aggressiveness (Dweba *et al.*, 2017; Fernando *et al.*, 2021). This interaction is dynamic, and emergence of more aggressive FHB populations has been observed in the fields (Puri and Zhong, 2010) and under *in vitro* conditions (Sakr, 2022a). It suggests adaptation followed by a spread of some strains in their environment, including adaptation to FHB resistant breeds and rendering resistance lacks durability which leads to resistance erosion (Puri and Zhong, 2010; Sakr, 2022a). However, little information is available on the variation of aggressiveness of the newly emerging FHB collection compared to old one. The hypothesis of this study is to test that the FHB population newly sampled from different quantitatively resistant wheat cultivars in Ghab Plain in 2015 vary in disease severity compared with the old FHB population collected in 2005. Thus, the aim of the present study was to determine whether populations of FHB were more aggressive at the end, 2015, than at the start, 2005, of epidemics. We characterized new and old FHB isolates by single-floret inoculation under controlled conditions on eight durum and bread wheat cultivars with different levels of FHB resistance, amplified fragment length polymorphism (AFLP), and random amplified polymorphic DNA (RAPD) analyses.

MATERIALS AND METHODS

Isolate Collection and Isolation

A total of 48 single-spore derived isolates subordinated to diverse *Fusarium* species, including 32 isolates collected during spring over the 2005 growing season of 9 FHB pathogens, old population (Alazem, 2007), i.e., *F. moniliforme* (synonym *F. verticillioides*), *F. culmorum*, *F. proliferatum*, *F. equiseti*, *F. sambucinum*, *F. solani*, *F. semitectum*, *F. avenaceum* and *F. compactum*, and 16 isolates sampled during spring over the 2015 growing season of 4 FHB pathogens, new population, i.e., *F. culmorum*, *F. solani*, *F. moniliforme* and *F. equiseti* from different localities of Ghab Plain, chosen as a location where head blight occurs regularly (Alazem, 2007; Sakr, 2019b, 2019a). These fungal isolates were isolated from symptomatic durum and bread wheat spikes collected from farmers' fields planted with wheats of contrasting susceptibility to FHB. These 48 isolates were identified as members of *Fusarium* genus based on standard morphological traits, including conidium and microconidiophore morphology, along with the cultural characteristics on potato dextrose agar (PDA) with 13 mg/l kanamycin sulphate added after autoclaving, such as pigmentation, colony morphology, density of aerial and medial mycelium and sporulation shape and type as described by the key of Nelson *et al.* (1983) for the old population and Leslie and Summerell (2006) for the new one. Single-spore 16 fungal cultures were stored in sterile distilled water at 4°C and kept by freezing at-16°C until needed (Sakr, 2020).

Aggressiveness Tests

A growth chamber experiment was conducted to test the aggressiveness of 48 isolates of diverse *Fusarium* species (32 isolates of old population analyzed previously by Alazem (2007) and 16 isolates of new collection tested herein in this study) on 8 durum and bread wheat cultivars with different levels of FHB resistance, including in decreasing order of susceptibility to FHB infection: cvs. Hourani (durum, susceptible), Cham5, Cham3, Bohoth5 and Douma3 (durum, susceptible to moderately susceptible), Cham4, Cham6 (bread, moderately susceptible) and Bohoth6 (bread, moderately resistant) as ranked from single-floret inoculation in a growth chamber (Alazem, 2007).

A randomized complete design was used with three replications. For each wheat cultivar, 15-cm pots filled with 2 kg of air-dried, sieved (2 mm) pasteurized soil per replication were planted with 8 surface-sterilized wheat seeds/pot. Three pots per cultivar were left un-inoculated and served as controls. The plants were grown at 20°C with 16 h of light until anthesis. Following emergence, plants were thinned to 5 seedlings. Irrigation was done (300 ml/pot) once a week until infection and plants were fertilized (0.173 g/pot) to avoid nitrogen deficit by providing urea at two dates: thinning and tillering. Macroconidia suspensions of the isolates were prepared by gently scraping the surface of 10-day-old cultures multiplied at 22ºC under continuous darkness to allow mycelial growth and sporulation on PDA after flooding with 10 ml of sterile water. Then, macroconidia were filtered through two layers of sterile cheesecloth to remove mycelia. The concentration of the

suspensions was adjusted to 5 × 10^4 macroconidia/ml with a hemacytometer. The central floret of individual spikes at anthesis was inoculated with 10 µl of the inoculum using a micropipette. The entire inoculated heads were covered with a polythene bag for 48 h to maintain high humidity. Control plants were sprayed with sterile distilled water. The experiment was repeated twice.

Disease rating was done at 21 days after inoculation at the soft dough stage. Disease severity, DS (% symptomatic spikelets/spike) was evaluated based on the number of spikelets infected on a nine grade scale according to Xue *et al.,* (2004), where 1 < 5%, 2 = 5–17%, 3 = 18–30%, 4 = 31–43%, 5 = 44–56%,6 = 57–69%, 7 = 70–82%, 8 = 83–95% and 9 > 95% of the spikelets with FHB symptoms. The change of aggressiveness of the new population and the old one was measured by comparing DS of the 16 *Fusarium* isolates of the new population to the 32 *Fusarium* isolates of the old one for each tested wheat cultivar.

Molecular Analyses

AFLP analysis was performed for the 32 isolates of the old collection according to Vos *et al.* (1995) and presented by Alazem (2007). However, RAPD analysis was conducted on the 16 isolates of the new collection as described by Williams *et al.* (1990) and presented by Sakr and Shoaib (2021).

DNA isolation

Single spore cultures of old and new FHB isolates were grown on PDA-Petri dishes in dark at 25°C for 10 days. Mycelium was harvested and DNA was extracted according to standard protocol (Leach *et al.*, 1986) and re-suspended in TE buffer (10 mM Tris-HCl, pH 8.0; 1 mM EDTA). Genomic DNA of a high quality and with concentrations ranging from 100 - 500 ng/µl was obtained from all *Fusarium* isolates. Quality and concentrations of *Fusarium* DNA was checked by agarose gel electrophoresis.

AFLP analysis

The AFLP protocol was carried out as reported by Vos *et al.* (1995). The pre-amplification reaction was performed using E+A and M+C primers (*Eco*RI and *Mse*I primers contain one selective nucleotide at the 3 end to reduce the number of amplified fragments).

RAPD analysis

According to Williams *et al.* (1990), amplification reactions were carried out in a final volume of 25 µl containing 1X PCR buffer, 1U Taq polymerase, 0.2 mM dNTPs, 2.0 mM MgCl2, 0.35 µM of each primer and 100 ng genomic DNA per µl of reaction mixture.

Molecular Data Analysis

Molecular (AFLP, RAPD) markers were visually scored as 1 for the presence and 0 for the absence of a band. The genetic similarity between pairs was estimated according to Nei and Li (1979). A dendrogram based on similarity coefficients was generated using the Unweighted Pair Grouping Method with Arithmetic mean (UPGMA) of the STATISTICA 6 computer package (Fitch and Margoliash, 1967). The experiments were repeated twice for each isolate to confirm repeatability, and the monomorphic bands were removed from the analysis.

Statistical Analyses

The arcsine and log transformed experimental data were subjected to analysis of variances (ANOVA) using DSAASTAT add-in version 2011. The differences between old collection and new collection were compared using Fisher's least significant difference test at the probability level 0.05%.

RESULTS AND DISCUSSION

Comprehension how pathogens evolve according to pressures employed by their plant hosts is crucial for the deducing of policies directed at the durable management of resistant cultivars (McDonald and Linde, 2002; Mundt, 2014; Cowger and Brown, 2019; Sakr, 2023). For the first time, field evidence highlighted that that the introduction of wheat quantitative resistance can lead to an aggressiveness shift in FHB populations. FHB pathogens are evolving means to overcome host resistance genes, enabled by its mixed reproduction system, encompassing both sexual and asexual reproductive states, and allowing for genetic recombination and the propagation of clones (Xu and Nicholson, 2009; Dweba *et al.*, 2017). In the current research, we tried to solve the question of the extent and conditions in which adaptation to quantitative resistance can occur. From an evolutionary standpoint, answering this question demonstrated that *Fusarium* species causing head blight in the current study show general adaptation (i.e., adapt to the prevailing resource).

FHB species are pathogens of heightened concern for the wheat and barley industries due to their high aggressiveness and ability to cause severe disease under epidemic conditions (Fernando *et al.*, 2021). Results in Table 1 showing that single-floret inoculation used to determine the aggressiveness of the two populations to

eight durum and bread wheat cultivars of contrasting susceptibility to FHB that the new population caused a higher DS (ranging from 55% to 67%) with the probability level 0.05% than the old population. Also, the ranking of eight tested wheat cultivars unchanged mostly when facing the two FHB populations, referring to a non-isolate nature of quantitative wheat resistance to FHB pathogens (Xu and Nicholson, 2009). Across the evaluation of susceptibility of the analyzed wheat cultivars, Bohoth6 (bread) showed the lowest DS and Hourani (durum) showed the highest DS. As expected, the level of susceptibility to FHB infection decreases from durum wheat to bread wheat (Dweba *et al.*, 2017). In fact, adaptation to durum and bread wheats of contrasting susceptibility to FHB cultivated in the surveyed region for long period had resulted in increased pathogen aggressiveness that was not specific because new FHB isolates had higher aggressiveness on the eight tested wheat cultivars varying in quantitative resistance. Our results are in accordance with those reported by Bjor and Mulelid (1991) in the *Phytophthora infestans*-potato pathosystem.

Table 1. Disease severity (DS) (%) on each of the eight durum and bread wheat cultivars with different levels of FHB resistance averaged based on old and new Fusarium head blight (FHB) populations collected at 2005 and 2015, respectively from Ghab Plain.

Wheat cultivar	DS (%)	
	Old population	New population
Hourani	38b	75a (61%)[z]
Cham5	29b	65a (55%)
Cham3	24b	59a (58%)
Bohoth5	19b	50a (62%)
Douma3	18b	45a (60%)
Cham4	16b	41a (61%)
Cham6	13b	40a (67%)
Bohoth6	11b	31a (64%)

DS was measured for the 32 FHB isolates of the old population and presented by Alazem (2007). DS was measured herein for the 16 FHB isolates of the new population.

According to the Fisher's LSD test, means followed by the same letter within a linage are not significantly different at the probability level 0.05%. The change of aggressiveness of new population and old one was measured by comparing DS of the 16 *Fusarium* isolates of the new population to the 32 *Fusarium* isolates of the old one for each tested wheat cultivar. In the current study, the 16 FHB isolates of new population were reanalyzed for DS on Cham4 and Cham6; however, pathogenic reaction for all isolates on these two cultivars was analyzed previously and presented by Sakr (2019b, 2019a).

We observed that aggressiveness of the new population sampled in 2015 and the old one sampled in 2005 increased between the two dates of sampling in a 10-years interval. The data from aggressiveness measurements in both old and new FHB populations are consistent with the hypothesis that new population is adapted to the locally dominant resources (in this case, cultivars), irrespective of the resistance characteristics of these cultivars. A similar result was observed in the populations of *Blumeria graminis tritici* (Villaréal and Lannou, 2000), *P. infestans* (Andrivon *et al.*, 2007) and *F. moniliforme*, *F. oxysporum* and *F. solani* (Abdelmagid *et al.*, 2016). This variation in aggressiveness over the course of the epidemic shows that a selection for the isolates most aggressive exists. On a long-term basis, this selection could affect the durability of quantitative resistance, and might explain the progressive decrease in the resistance of quantitatively wheat resistant cultivars. *Fusarium* species causing head blight species are evolving tools to defeat quantitative wheat resistance genes, enabled by its mixed reproduction system, including both sexual and asexual reproductive states, and allowing for genetic recombination and the propagation of clones (McDonald and Linde, 2002; Fernando *et al.*, 2021). Two proposed hypotheses could explain this decreased durability. First, FHB pathogens had the combination of a larger number of mutations in their genome to overcome polygenic

resistance. Second, selection pressure exerted on FHB pathogens was higher and distributed among limited genes, which augmented the risk of emergence of aggressive variants from the pathogen populations.

The genome of head blight pathogens shows low levels of repetitive elements due to the targeting mechanisms associated with repeat induced point mutation (Cuomo *et al.*, 2007), thereby promoting rapid adaptation to selection pressures (Fernando *et al.*, 2021). Although results deduced from the AFLP for the old population (Alazem, 2007) and RADP for the new one (Sakr and Shoaib, 2021) (Figure 1) revealed a high genetic variation within diverse *Fusarium* species, our comparative population genetic analyses with analyzed markers showed that the new population had more polymorphic loci compared with the old one.

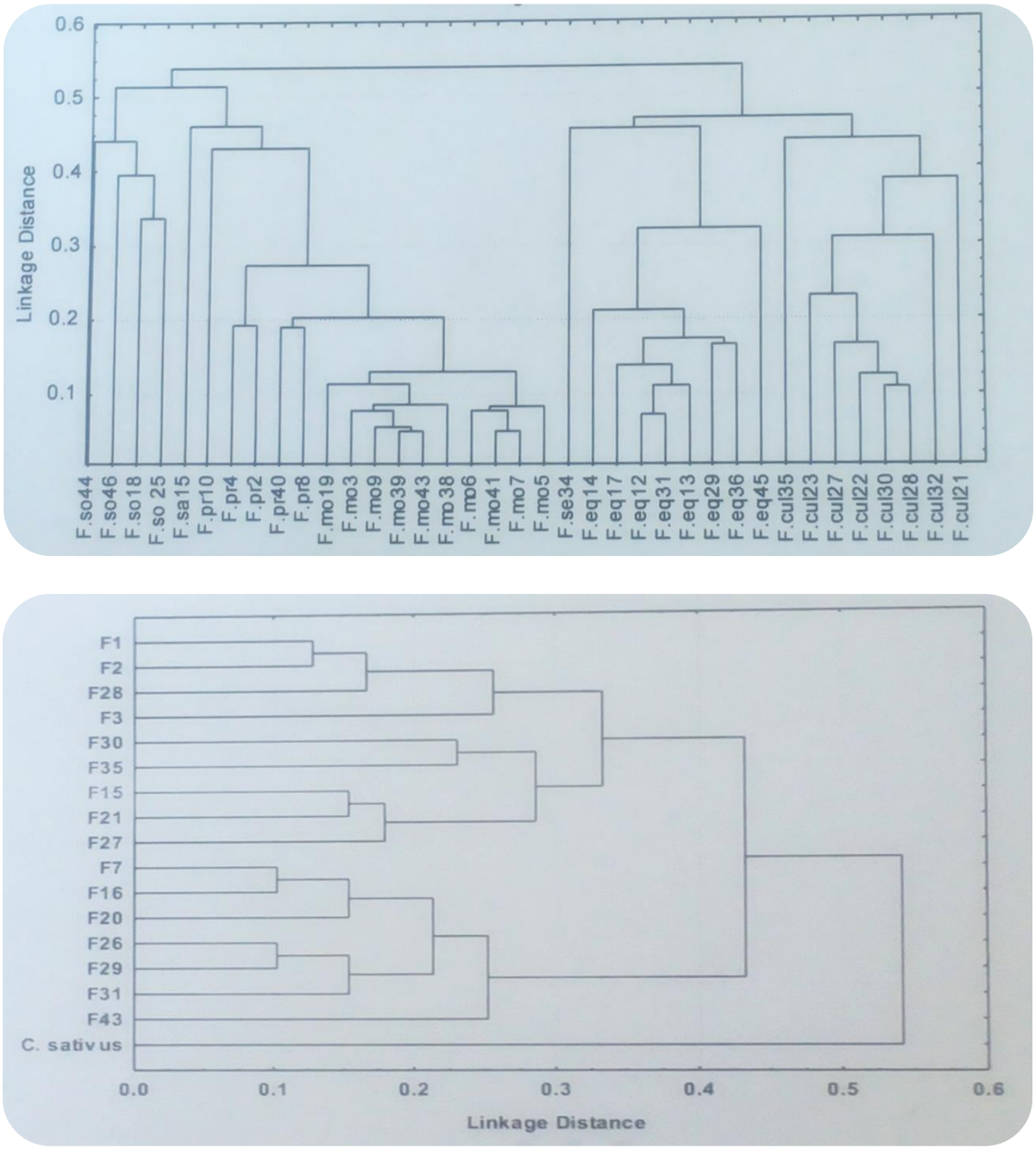

Figure 1. Unweighted pair grouping method with arithmetic mean generated for the amplified fragment length polymorphism (AFLP) (A) and random amplified polymorphic DNA (RAPD) (B) showing genetic relationships among *Fusarium* species causing head blight in old and new FHB populations collected in 2005 and 2015, respectively from Ghab Plain.

AFLP analysis was performed for the 32 FHB isolates of the old population and presented by Alazem (2007). RAPD analysis was conducted on the 16 FHB isolates of the new population and presented by Sakr and Shoaib (2021). The dendrogram was based on the genetic distances calculated according to Nei and Li (1979).

More molecular marker bands are linked with the existence of more rerto-transposon insertion sites and subsequently with higher evolutionary position, therefore, species displaying more molecular marker bands will be more advanced evolutionary (Williams *et al.*, 1990; Vos *et al.*, 1995). Further, a significant genetic differentiation was detected between the two populations in which the UPGMA dendrogram generated for the AFLP data distinguished the isolates of the tested nine FHB species, Figure 1, A (Alazem, 2007); however, RADP data distinguished the isolates of two *Fusarium* pathogens out of four tested pathogens, Figure 1, B (Sakr and Shoaib, 2021).

These results suggest that the new population is different from the old one and might have emerged more recently. *Fusarium* genome studies have demonstrated the existence of specialized pathogenicity chromosomes with evidence of horizontal acquisition (Ma *et al.*, 2010). Our data are comparable with those found by Puri and Zhong (2010) for newly and prevalent populations of *F. graminearum* and reported by Abdelmagid *et al.* (2016) for virulent and less virulent isolates of *F. moniliforme*, *F. oxysporum* and *F. solani*.

While fungal pathogens can defeat plant resistance barriers because of aggressiveness shifts due to mutation, immigration or sexual recombination (McDonald and Linde, 2002; Sakr, 2023), such variation in aggressiveness observed herein can be ascribed to mutation and/or sexual recombination. A new mutation was identified and functionally validated in the gene *FgVe1*, coding for a velvet protein known to be involved in pathogenicity and secondary metabolism production in several fungi (Laurent *et al.*, 2021). The high-gene flow suggests potential to create pathogen populations that can rapidly adapt to management strategies like fungicide applications and resistant cultivars (Talas and McDonald, 2015). The potential role of immigration contributing to changes in aggressiveness exceeded since all analyzed isolates came from a single and restricted geographic area, i.e., Ghab Plain region, which had exhibited the first presence of the FHB disease in Syrian wheat fields (Alazem, 2007). In the genome of *F. oxysporum*, transposons produce spontaneous mutations causing changes in aggressiveness (Wang *et al.*, 2008). Furthermore, the genome of *F. graminearum* displays small levels of repetitive elements owing to the targeting mechanisms combined with repeat induced point mutation (Guo *et al.*, 2008), thereby inducing rapid adaptation to selection pressures. On the other hand, increases in the aggressiveness of adapted *Plasmopara viticola* (downy mildew) isolates resulted from sexual propagation of the pathogen on partially grapevine resistant cultivars (Delmas *et al.*, 2016).

CONCLUSION

The information obtained in this study indicated that FHB populations adapt to prevailing wheat cultivars, irrespective of their resistance levels, and can therefore overcome polygenic, quantitative resistance. Indeed, we demonstrated that host plant resistance did not guarantee high resistance to FHB since resistance broke down due to aggressiveness shifts of *Fusarium* species. Adaptation to wheat resulting in increased pathogen aggressiveness that was not specific may render quantitative resistance nondurable if not properly managed. Therefore, pyramiding of several QTL*s* with high impact in one wheat cultivar may extend durability. Also, quantitative resistance integrated with other management practices, i.e., disease forecasting, direct chemical fungicide treatment, bacterial antagonists as bio-control agents and crop rotation, will be the most promising and effective management strategy for FHB control.

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STATEMENTS AND DECLARATIONS

Authors declare are no financial or non-financial conflicts of interest.

AUTHORS CONTRIBUTIONS

Nachaat Sakr designed and completed the research solely.

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