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Tissue-Specific Responses of Cereals to Two *Fusarium* Diseases and Effects of Plant Height and Drought Stress on Their Susceptibility

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Abstract: Multiple species of *Fusarium* can infect wheat and barley plants at various stages of development. *Fusarium* head blight (FHB) refers to the infection of spikes and developing kernels by these pathogens, and crown rot (FCR) infers to infection of the root, crown, and basal stem by *Fusarium* pathogens. Interestingly, most of the host genes conferring resistance to these two diseases are different in both wheat and barley, and plants' susceptibility to these two diseases are oppositely affected by both plant height and reduced water availability. Available results do not support the hypothesis that reduced height genes have different effects on biotrophic and necrotrophic diseases. Rather, differences in temperature and humidity in microenvironments surrounding the infected tissues and the difference in the physical barriers originating from the difference in cell density seem to be important factors affecting the development of these two diseases. The fact that genes conferring resistance to Type I and Type II of FHB are different indicates that it could be feasible to identify and exploit genes showing resistance at the three distinct stages of FCR infection for breeding varieties with further enhanced resistance. The strong association between FCR severity and drought stress suggests that it should be possible to exploit some of the genes underlying drought tolerance in improving resistance to FCR.

Keywords: DELLA; drought stress; FCR; FHB; plant height; *Rht* genes

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1. Introduction

Multiple species of *Fusarium* can infect wheat and barley plants at various stages of their development. The term FHB (also known as scab) refers to *Fusarium* infection of spikes and developing kernels by these pathogens. When the root tissue, crown and basal stem are infected by *Fusarium* pathogens, the disease is named as FCR (Figure 1). Although field surveys suggest that the dominant *Fusarium* isolates associated with FHB and FCR can be different, many *Fusarium* species associated with FHB can also be isolated from FCR-infected plants [1,2]. Additionally, all *Fusarium* pathogens that cause FHB are also responsible for FCR under laboratory environments [3].

FHB is among the most intensively investigated diseases in cereals. The abundance of inoculum and weather conditions, mainly moisture and temperature, during and after anthesis determines the severity of FHB. The symptoms of FHB can become visible shortly after anthesis under environments of high humidity and temperature which are highly beneficial for initiation and propagation of FHB infection. Diseased spikelets exhibit premature bleaching, and the bleaching of spikelets can progress throughout large numbers of entire spikes. Often, the infected grains shrink and wrinkle with shrivelled appearance with colour ranging from pink to light brown. FHB is one of the most serious diseases

affecting wheat production worldwide [4,5]. The associated costs in the USA in the last decade alone exceeded US$ 2 billion [5]. In addition to grain yield loss, FHB-infected kernels can produce mycotoxins, especially deoxynivalenol (DON), which poses risks to animal and human health [3]. 'Field resistance' to FHB can be divided into two major components, resistance to initial infection (Type I) and resistance to disease spread within infected spikes (Type II) [6]. Type I resistance is evaluated by spraying spore suspension over flowering spikes and counting the diseased spikelets. Type II resistance is evaluated by delivering conidia into a single floret of a spike and counting the infected spikelets after a given time. Clearly, accurate assessment of Type I resistance needs to be carried out before the onset of Type II symptoms. However, many FHB studies have been conducted by using spray inoculation and evaluating a combination of both Type I and Type II resistance [7–12]. It is well known that when compared to that of Type I, the development of Type II disease is less prone to environmental influence [7,13]. Plant height, heading date, spike length, density of spikelets, and anther retention are among characteristics affecting FHB infection in wheat [14,15].

Figure 1. Symptoms of *Fusarium* crown rot in the field: brown necrotic lesions on tiller bases (**left**), seedling death (**middle**) and whiteheads (**right**).

Symptoms associated with FCR include seedling death, brown necrotic lesions on the coleoptile, tiller base, roots and subcrown internode, and the rotting of root, crown, and stem tissues (Figure 1). Stem base browning is widely used to measure FCR severity [16,17]. Whiteheads are the most pronounced visual impact of FCR in wheat and they often occur when plants are drought-stressed after anthesis. Whiteheads can be completely devoid of grain or possess shrivelled grains, resulting in an increase in screenings. Consequently, crop yield and hence crop value (via yield reduction and often quality downgrade) can be significantly reduced [3]. There is a significant difference in FCR sensitivity among barley ($2n = 2x = 14$, genome HH), bread ($2n = 6x = 42$, genomes AABBDD), and durum wheat ($2n = 4x = 28$, genome BBAA), with durum wheat being the most susceptible [17]. Interestingly, whiteheads are rare in barley crops [18]. The reasons for the difference in whitehead production between these crop species remain unknown.

FCR is a chronic disease in cereal production in semi-arid regions worldwide [3]. Based on figures from the last available surveys conducted in Australia more than a decade ago, FCR causes an estimated annual yield loss in wheat and barley of about A$97 million [19,20]. The incidence and severity of FCR have been exacerbated in recent years by an increase in the intensity of cereal production for economic reasons combined with the wide adoption of reduced tillage [3]. Data from recent research at Merredin and Wongan Hills in Western Australia show that high levels of FCR can cause average yield losses of 19% in bread

wheats and 18% in barley [21]; even higher yield losses occur elsewhere—e.g., up to 35.0% in the USA [22], 43.0% in Turkey [23], and 45.0% in Iran [24]. In recent years, the incidence and severity of FCR have progressively increased in major wheat-producing areas and FCR became a major threat to wheat production in China [25]. FCR infection can also produce high concentrations of mycotoxin in infected plants under experimental conditions, but levels of mycotoxins in harvested grains of FCR-infected plants are generally very low and do not pose a serious threat to further processing or consumption [26].

Significant progress has been made in genetically improving resistance to the latter in recent years. Several loci conferring FCR resistance have been detected in both wheat and barley [18]. Some of these were detected from wild relatives or landraces [16,27,28] while others from elite varieties [12,29]. Effects of these loci have been validated by analysing near isogenic lines (NILs) generated for each of them [30–34], and molecular markers tightly linked to several of these loci have been obtained by assessing NIL-derived populations [31,35–37]. Effects of gene pyramiding in further enhancing FCR have been demonstrated for both barley [38] and wheat [39].

2. Different Genes Control FHB and FCR Resistance

Considering that FHB and FCR are basically two different diseases caused by the same *Fusarium* pathogens infecting different organs, it seems logical to expect that they must share many features. Indeed, all resistance genes or loci reported so far for both FHB [40] and FCR [18] are not pathogen species-specific. In other words, a gene or locus giving resistance to one *Fusarium* species would give resistance to other *Fusarium* species as well. Supporting this notion is a report that the gene *Fhb7* is resistant to both FHB and FCR [41]. However, *Fhb7* seems to be an exception. Other known sources of FHB resistance, including both Sumai 3 and Wangshuibai, do not seem to provide resistance to FCR [42]. Enhanced resistance to FHB was easily detected in all genotypes containing the best-known resistance gene *Fhb1* [9], but the same materials did not show any improved resistance to FCR [42].

It may not be a total surprise that most of the genes conferring resistance to FHB do not show any effect on FCR and *vice versa*. Available results suggest that genes conferring resistance to Type I and Type II of FHB are also different [8]. Similarly, different genes are responsible for FCR resistance at different developmental stages of wheat and barley [43]. Based on the analysis of pathogen quantity in infected plants, FCR infection and spread show a typical characteristic of three distinct phases [44]. It would be of interest to find out whether different sources of resistance or different resistance genes behave differently at each of these different phases of infection and whether these different features of resistance and tolerance could be exploited to develop varieties with further enhanced FCR resistance [18].

3. FHB and FCR Occur Differently under Drought Stress Conditions

The developments of FHB and FCR show opposite patterns in drought-affected plants. Severe yield losses from FHB are driven by a combination of warm and wet weather coinciding with crop anthesis [5]. On the other hand, severe FCR damage occurs mainly in crops affected by drought stress, especially late in the growing seasons [22,45,46]. Results from a laboratory-based studies showed that drought stress prolongs the initial infection phase but enhances the proliferation and spread of *Fusarium* pathogens after the initial infection phase in FCR development [47].

Reasons for the difference in susceptibility of plants to these two *Fusarium* diseases under drought conditions are unknown. One of the most obvious possibilities that may contribute to the difference is the conditions in times of infection. Infection of FCR can occur once seeds start to germinate, a time when the temperature is low in all regions of cereal production worldwide. FHB infection occurs following anthesis when the temperature is high and water availability is usually low [40]. However, it is not clear how the difference in temperature during disease infection would interact with other factors and results in the different patterns of development of these two different diseases under conditions of

drought stress. Nevertheless, the strong association between FCR resistance and drought tolerance suggests that some genes conferring drought tolerance might be exploited for enhancing FCR resistance.

4. The Difference in Plant Height Is Associated with Differential Responses to FHB and FCR

The associations between plant height and susceptibility to FHB and FCR are also opposite in both wheat and barley. Taller plants tend to be more tolerant to FHB but more susceptible to FCR. Several possible explanations for this phenomenon were offered. As the principal inoculum for FHB infection under field conditions are *Fusarium* ascospores that are found in debris on soil surface, one of the explanations is that spikes of shorter plants are physically located closer to the source of infection [48,49]. However, this possibility cannot explain why shorter plants growing in glasshouses (where a large quantity of *Fusarium* pathogens is unlikely to be found on the floor) are also more susceptible to FHB infection. A second possible reason explaining why shorter plants tend to be more susceptible to FHB is that genes reducing plant height could be positioned in a proximity of those conferring a susceptibility to the disease [50,51]. This possibility is based on findings showing that several QTLs conferring FHB resistances have been co-located with reduced-height (*Rht*) loci [10,11,50,52–54]. However, a study based on the use of NILs targeting individual *Rht* genes showed that the dwarf isolines were invariably more susceptible to FHB infection [55]. While it is possible that one or two of the *Rht* genes may be causally linked to genes conferring susceptibility to this disease, it is difficult to extrapolate this to all *Rht* genes. Another possibility is that, compared with those of tall plants, spikes of shorter plants are exposed to microenvironments with higher humidity [48]. However, direct experimental studies have not revealed the difference in the moisture between spikes from tall and short plants [48]. Opposite results, however, were obtained in experiments with NILs for several different *Rht* genes by studying response to FHB infection by separating Type I and Type II resistance [55]. At their natural height, dwarf isoline was always more susceptible to Type I infection for each of the NIL pairs assessed. However, when the dwarf isolines were physically raised so that their heads were at the same height as those of their respective tall lines, difference in Type II resistance to FHB between the tall and dwarf isolines disappeared for each of the NIL pairs. These results showed beyond doubt that moisture levels in the microenvironments exposed by spikes are responsible for the observed differences between tall and dwarf genotypes, although the difference in air moisture between the two microenvironments may not always be detectable with available equipment. It was also found in this study that the effects of drought stress on Type II resistance (spread of pathogens within infected plants) are marginal [55].

When compared with those on FHB, studies on FCR are limited. However, the association between plant height and development of FCR infection was revealed in a study in which 12 pairs of NILs for six different *Rht* genes located on different chromosomes in wheat were examined. When compared with their taller counterparts, dwarf isolines showed better resistance to FCR [56,57]. Results from histological analyses and real-time quantitative PCR on two pairs of NILs for a *Rht* gene in barley showed that *F. pseudograminearum* hyphae were detected earlier and proliferated more rapidly during the time-course of FCR development in the tall isolines at each of the time points assessed during FCR development. As the cell density of the dwarf isolines are also significantly higher than those of the tall ones, it was speculated that the increased cell density associated with dwarf genes could act as a physical barrier to the spread of FCR in cereals [58].

Interestingly, the association between plant height and disease resistance have also been investigated by targeting reduced-height genes. Following the advent of the 'Green revolution' *Rht-B1b* and *Rht-D1b*' genes were introduced into modern cereal varieties that encode mutant forms of DELLA proteins that consist of transcription factors characterized by a short stretch of amino acids (D-E-L-L-A) in their N-terminal regions, which are highly conserved among different plant species. These proteins repress GA (gibberellic acid)-

responsive growth [59]. In addition to their effects on plant development, DELLA proteins are also believed to affect biotrophic and necrotrophic diseases. Through their influence on the balance between salicylic acid and jasmonic acid pathways, DELLA genes likely increased susceptibility to necrotrophs but increased resistance to biotrophs. The hypothesis that DELLA genes may have different effects on pathogens with contrasting lifestyles was initially proposed in a study on *Arabidopsis* [60]. A similar claim was then made in wheat and barley, where FHB was assessed as one of the necrotrophic diseases [61]. However, some *Rht* genes do not confer dwarfism by encoding mutant forms of DELLA proteins. For example, *Rht8* confers dwarfism by reducing sensitivity to brassinosteroids [62]. Like those conferred by all other *Rht* genes, shorter plants caused by *Rht8* also showed worse resistance to FHB in wheat [55]. Similarly, the *Rht* gene *uzu*, another non-GA-responsive semi-dwarf gene, is also linked to increased resistance to FCR [63]. Clearly, these results contradict the hypothesis that DELLA genes increase susceptibility to necrotrophs but increase resistance to biotrophs by balancing between salicylic acid and jasmonic acid pathways [61,62].

The question then is why cell density does not slow down the spread of FHB, as shorter plants tend to be more susceptible to this disease? One of the possible answers to this question is the relative importance of micro-environments vs physical barriers. The higher humidity and temperature of shorter plants play a larger role when compared with the higher cell density in the shorter plants for FHB, but the latter may have a larger effect than the former on FCR resistance.

With the understanding that QTL mapping based on analysis of segregating populations provides only limited resolution [64], the interaction between plant height and FCR severity means that we need to be very cautious when dealing with resistance loci that locate closely with those for plant height. The FCR locus located on chromosome arm 3HL in barley [65] and the one on 4B in wheat [66] are two of such examples. The values of these resistance loci need to be properly assessed before further efforts on incorporating them into breeding programs should be made.

5. Conclusions

Although FHB and FCR can be caused by the same pathogens, available results show that most of the reported host genes conferring resistance to these two diseases are different. The susceptibility of plants to these two diseases are also oppositely affected by either plant height or drought stress. The hypothesis that DELLA genes affect biotic and abiotic stresses differently by balancing the salicylic acid and jasmonic acid signal pathways does not stand and needs to be examined further. The moisture levels in the microenvironments to which infected tissue are exposed and the difference in physical barriers resulting from differential cell density seem to be two additional factors affecting resistance to these two diseases. Available results indicate that different genes conferring FCR resistance may exist for each of the three distinctive phases during the disease development, and that some genes conferring drought tolerance can be effective in generating breeding lines with enhancing resistance to FCR.

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