QTL ANALYSIS FOR FUSARIUM HEAD BLIGHT RESISTANCE IN TUNISIAN-DERIVED DURUM WHEAT POPULATIONS

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The Supervisory Committee certifies that this ***disquisition*** complies with North Dakota State University's regulations and meets the accepted standards for the degree of

DOCTOR OF PHILOSOPHY

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ABSTRACT

Durum (2n=4x=28; AABB) wheat is the grain of choice for the production of high-quality pasta products. *Fusarium* spp. are causal pathogens for Fusarium Head Blight (FHB). Limited host resistance to this disease exists among adapted durum cultivars. The use of Tunisian-derived durum lines for integration of FHB resistance in cultivars was evaluated. The genetic characterization of FHB resistance was evaluated, and markers associated with FHB resistance are presented in two populations. Two backcross inbred line (BIL) populations derived from cross between a resistant durum genotype 'Tunisian 108' and susceptible durum wheat cultivars 'Ben' and 'Lebsock' were screened to identify QTL for FHB resistance. Analysis of variance showed significant effect of genotypes on FHB severity and incidence despite high level of interaction between environment and genotypes. A total of 329 and 331 DArT and microsatellite markers covered a distance of 1887.6 and 1748 cM in two populations respectively. Composite interval mapping using two linkage maps and the phenotypic data revealed 11 different FHB resistance QTL on seven different chromosomes (1A, 1B, 2B, 3B, 5A, 5B, and 7B) in Tunisian/Ben derived population and 15 different FHB resistant QTL on seven different chromosomes (1A, 1B, 3A, 3B, 4A, 5A, and 6B) in population derived from cross between Tunisian/Lebsock. At least two novel QTL were identified on chromosome 2B (*Qfhb.ndsu-2B*) 4A (*Qfhs.ndsu-4A*) in Tunisian/Ben//Ben and Tunisian/Lebsock//Lebsock population respectively. Location of the two FHB resistance QTL on chromosome 1B and two QTL on 5A were identical in both populations. Owing to cumulative effects of resistance QTL, high level of transgressive segregation was observed in both populations. Our finding revealed

an alternative tetraploid FHB resistance source from Tunisian genomic background that can be utilized with associated markers for wheat germplasm enhancement.

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CHAPTER I. GENERAL INTRODUCTION

Durum wheat [*T. turgidum* (L.) var. *durum*, 2n = 4x = 28, genomes AABB] is an important cereal crop in the United States, used in pasta and noodle production. Sixty-eight percent of U.S. durum production occurs in North Dakota http://www.gipsa.usda.gov/fgis/educout/commgallery/gr_du.html). For more than two decades, North Dakota durum wheat cultivation has been heavily impacted by *Fusarium* Head Blight (FHB) disease. Favorable conditions for FHB caused epidemic outbreaks that resulted in production losses of $2.7 billion in wheat and barley (*Hordeum vulgare* L.) from 1998 through 2000 in the United States (Nganje et al. 2002), and totaling $6.2 billion from 1993 through 2001 in the northern Great Plains (Nganje et al. 2004).

Fusarium head blight is commonly referred to as "wheat scab"; this descriptive nomination harkens back to the first documented FHB-outbreak, which occurred in England in 1884 (Stack 2003). In the same approximate decade, Scab outbreaks were reported in the Americas (Gilbert et al. 2000; McMullen 2003), Asia (Bai et al. 2003; Bhat et al. 1989), Australia (Burgess et al. 1987), Europe (Parryet al. 1995; Xu et al. 2008), and South Africa (Kriel and Pretorius 2008; Scott et al. 1988).

FHB is caused primarily by the fungus *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schw.) Petch]. The impact of disease on wheat is highest in warm and humid environmental conditions (Stack and McMullen 1985; Gilbert and Tekauz 2000). Under such conditions, durum wheat is highly susceptible to the pathogen during anthesis and early kernel development (Gilbert and Tekauz 2000). FHB can cause bleached spikes, spikelet sterility, poor seed filling, low weight and tombstone seeds (Wang 1996). Furthermore, grain marketing value

is drastically affected by scabby wheat because of mycotoxin contamination from deoxynivalenol (DON), diminished milling quality and diminished baking quality (Gilbert and Tekauz 2000; Bechtel et al. 1985). *Fusarium*-contaminated grain is the main source of mycotoxins in the food chain, which can cause Alimentary Toxic Aleukia (ATA), a potentially fatal condition in humans (Foroud and Edues 2009).

The development of host genetic resistance is the preferred strategy to reduce FHB effects, Since cultural management practices and chemical controls are considered impractical or ineffective (Bai and Shaner 1994). Therefore, identification and incorporation of FHB resistance genes has been a point of particular emphasis by plant breeders, for the purpose of improving host plant resistance.

Resistance to FHB exhibits the characteristics of a quantitative trait (Schroeder and Christensen 1963; Buerstmayr et al. 2012). Genetic studies of quantitative trait loci (QTL) require the development of population structures such as recombinant inbred lines (RIL), near isogenic lines (NIL), double haploid lines, or backcross derived lines, and the consequent genotypic and phenotypic characterization of experimental populations. There are five types of physiological resistance for FHB in the host; namely, resistance to (i) disease penetration (Type I), (ii) floral spread (Type II), (iii) *Fusarium* Damaged Kernel (FDK) (Type III), (iv) yield reduction (Type IV) and (v) mycotoxin accumulation (Type V) (Schroeder and Christensen 1963; Mesterhazy 1995).

The accumulation of QTL associated with broad FHB resistance indices is essential to cultivar development. Identifying DNA markers tightly associated with FHB resistance will speed up the introgression of resistance QTL into the desirable cultivars (Bai and Shaner 1994).

Investigations have shown positive association between different resistance indices. Hence, selection for one type of resistance likely enables selection for other types (Lemmens et al. 2005).

Problems associated with collecting reliable FHB resistance data (Campbell and Lipps 1998) and the polygenic nature of FHB resistance (Buerstmayr et al. 2009; Rudd et al. 2001) have focused efforts of research groups toward identifying new sources of resistance and associated molecular markers for FHB resistance genes.

Strong genotype by environment interaction is anticipated in FHB disease outcomes, and quantitative inheritance is inherently complicated (Buerstmayr et al. 2009; Rudd et al. 2001). Undesirable environmental effects can be indirectly managed by using highly-resistant genotypes. Genotypes with moderate resistance can reduce the impact of FHB, although this resistance may not be stable under high disease pressure (Snijders and Perkowski 1990; Mesterhazy 1995; Buerstmayr et al. 1996; Gosman et al. 2007; Buerstmayr et al. 2002). Historically, FHB resistance sources have been limited to hexaploid cultivars such as 'Sumai3' and 'Wangshuibai' (Mardi et al. 2005) and to a limited extent, 'Frontana' from Brazil (Mardi et al. 2006).

Hexaploid Chinese germplasm and derivatives have been studied extensively using QTL mapping (Bai et al. 1999; Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002; Zhou et al. 2002; Bourdoncle and Ohm 2003; Shen et al. 2003). Over the past two decades, many major and minor QTL associated with FHB resistance have been identified on almost all wheat chromosomes (Anderson et al. 2001; Buerstmayr et al. 2009; Lin et al. 2006; Liu and Anderson 2003; Ma et al. 2006; Waldron et al. 1999). *Fhb1* on chromosome 3BS is a QTL with

a stable major effect on FHB Type II (Anderson 2007) and Type V resistance (Lemmens et al. 2005) across different genetic backgrounds; hence, it has been used in breeding programs worldwide. Other resistance QTL reported in wheat includes a QTL on chromosome 5A (Chen et al. 2006; Lin et al. 2006), *Fhb2* on chromosome 6B (Anderson et al. 2001; Cuthbert et al. 2007) *Fhb4* on chromosome 4B (Lin et al. 2006; Xue et al. 2010), and *Fhb3* reported in wheat- *Leymus racemosus* integration lines (Qi et al. 2008). Additionally, QTL on chromosomes 2D (Somers et al. 2003) and 5A (Chen et al. 2006) have been reported to contribute towards Type V resistance. However to date, only *Fhb1* has shown a stable major effect on Type II or V resistance; all other QTL have either only minor effects or unstable effects in different hexaploid backgrounds.

Lack of resistance sources in tetraploid durum wheat has shifted efforts toward utilization of wild emmer, alien species (Oliver et al. 2005; Oliver et al. 2007) and exotic lines (Elias et al. 2005). Utilization of limited host resistance in wheat imposes a strong selective pressure for virulent pathogen strains (Gervais et al. 2003). Consequently, exploration for alternative sources of FHB resistance has become a necessary endeavor. Recent investigation at North Dakota State University revealed five prominent Tunisian lines with moderate levels of Type II resistance to FHB and generally acceptable agronomic traits (Elias et al. 2005; Huhn et al. 2012). The same lines were utilized as donor parents for introgression of resistance factors in North Dakota cultivars. The objectives of this study were to identify genomic regions significantly associated with FHB resistance and incorporate Tunisian FHB resistance QTL into the genomic background of locally adapted North Dakota durum wheat cultivars.

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CHAPTER II. LITERATURE REVIEW

Wheat

Wheat is considered one of the 'three strategic cereal crops' with over 600 million tons harvested annually (http://faostat.fao.org/). European union, China, India and the United States of America are top four wheat producers in the world respectively. It is unrivalled in its range of diversity, cultural value and range of cultivation. Wheat can be planted from 67˚N in Scandinavia and Russia to 45˚S in Argentina, including elevated regions in the tropics and sub-tropics (Feldman et al.1995). Wheat is the universal ancient cereal (Zohary and Hopf 2000) and the world's foremost crop plant (Feldman et al. 1995; Gustafson et al. 2009).

Poaceae (grasses) family evolved 50–70 million years ago (Mya) (Kellogg, 2001; Huang et al. 2002), and wheat, barley and oats which are member of Pooideae sub-family diverged around 20 Mya (Inda et al. 2008). Non-scientific selections by early farmers separated the desirable landraces from their wild relatives, leading to wide domestication. Two essential selection events for domestication were (i) the loss of spike shattering at maturity, caused by recessive alleles at the Br (brittle rachis) locus (Nalam et al. 2006), and (ii) change from hulled kernels to free-threshing naked kernels, due to a dominant mutation at the Q locus, which modified the effects of recessive mutations at the Tg (tenacious glume) locus (Jantasuriyarat et al. 2004; Simons et al. 2006; Dubcovsky and Dvorak 2007).

It is now generally accepted that the A genomes of tetraploid and hexaploid wheat originated from A genomes of wild and cultivated einkorn (*Triticum urartu*, genome A^uA^u) (Dvorak et al. 1993). The B genome of tetraploid and hexaploid wheat is likely derived from

Aegilops speltoides (genome SS) (Dvorak et al. 1993; Peng et al. 2011). Wild emmer wheat (*T. dicoccoides*, 2n = 4x = 28, genome A^uA^uBB) emerged 300,000–500,000 years BP (before present) (Huang et al. 2002; Dvorak and Akhunov 2005). The earliest evidences show that, hunter-gatherers started collecting cereals 19,000 BP from Ohalo II which was a permanent site of epipaleolithic on the southwestern shore of the Sea of Galilee, Israel (Feldman and Kislev 2007). The first cultivation of wheat occurred in south-eastern part of Turkey about 10,000 years ago as part of the 'Neolithic Revolution', which was a cultural transition among early humankind from hunting and gathering of food to a systematic culture of plants and animals. The earliest cultivated forms of wheat were diploid einkorn and tetraploid emmer wheat (Dubcovsky and Dvorak 2007). Further conscious selection gradually resulted in cultivated emmer (*T. dicoccum*, 2n = 4x = 28, genome A^uA^uBB). Spontaneous hybridization between cultivate emmer and another goat grass (*Ae. tauschii*, 2n = 2x = 14, genome DD) created an early spelt (*T. spelta*, 2n = 6x = 42, genome A^uA^uBBDD) around 9,000 BP (Kislev 1980; Dvorak et al. 1998; Matsuoka and Nasuda 2004). Divergence levels of A and B genomes in hexaploid wheat are drastically high in comparison to genomes of their diploid progenitors. While nominal divergence of D genome in hexaploid wheat indicate the D genome hybridization was the latest and relatively recent speciation event occurred 9,000 years ago (Feldman and Millet 2001). There are no report for wild type hexaploid wheat in exception of *T. tibetanum* which has been discovered as a weed in wheat and barley fields (Dubcovsky and Dvorak 2007; Haudry et al. 2007; Fu and Somers 2009).

Durum Wheat

Durum wheat (*Triticum turgidum* L. var. *durum*) is a monocotyledonous allotetraploid (Genome AABB, 2n = 4x = 28) plant of the *Gramineae* family, *Triticeae* tribe and belongs to the genus *Triticum* which is the second most important species in this genus, next to common wheat

(*T. aestivum* L.). The starting point of evolutionary process in durum wheat was precisely delineated in the previous section.

Durum is a mid-tall to semi-dwarf stature annual grass with flat leaf blades and a terminal floral spike consisting of perfect flowers (Bozzini et al. 1998). The primary root system consists of seminal and adventitious roots which change to a permanent root system. The stem is cylindrical, erect, usually hollow (some are solid), and subdivided into internodes or solid stems (Clarke et al. 2002). Auxiliary buds at the basal nodes are the origin of culms (tillers), which in normal growing conditions may be a total of three culms in addition to the main shoot. The main stem and each culm produce a terminal inflorescence (Bozzini 1988). As with other grasses, durum wheat leaves have a leaf sheath (the basal portion) which covers the terminal portion of the stem, and is linear with parallel veins and an acute apex. A thin and transparent membrane (ligule), with two small lateral appendices (auricles) is characteristic of durum stems.

The structure of durum wheat inflorescence is a spike composed of a rachis with spikelets separated by short internodes (Bozzini 1988). Each spikelet consists of two glumes (bracts) enclosing two to five florets, all located on a rachilla. Each floret is enclosed between a lemma and palea. Each perfect flower contains three stamens with bilocular anthers and a pistil with two styles and feathery stigmas. Each floret has the potential to produce a one-seeded fruit called a caryopsis. The caryopsis is generally termed a kernel, or seed. Each seed contains a large endosperm and a flattened embryo located at the apex of the seed and close to the base of the floret. Ideal growing conditions for durum wheat are a relatively dry climate, with warm days and cool nights during the growing season (Mediterranean and temperate climates). Seed germination starts at 2°C, but the optimal germination temperature is 15°C (Bozzini 1988).

Durum wheat has mostly spring growth habit; however, there are lines with winter growth habit in the southern regions of USA (Donmez et al. 2000).

Durum wheat grain is the source for semolina, which is used in pasta products, couscous and bulgur in North Africa, and traditional breads in Morocco. Durum (Latin word for 'hard') has the hardest kernel among wheat, with high protein content and high gluten strength, large size, and amber color. The unique yellowish endosperm in durum gives pasta its golden color. Strong gluten and non-sticky dough characteristics have made durum the ideal grain for pasta production (Clarke et al. 2006).

Centers of Origin of Durum Wheat

To date, two wild diploid wheat species characterized as *T. boeoticum* (A^bA^b) and *T. urartu* (A^uA^u). It is highly believed that *T. boeoticum* is the ancestor of einkorn wheat (*T. monococcum*) which is unrelated with cultivated tetraploid and hexaploid wheat (Gandilian 1972; Johnson 1975; Johnson and Dhaliwal 1976; Dorofeev et al. 1979; Nesbitt and Samuel 1996; Perrino et al. 1996; Heun et al. 1997; Dvorak et al. 1998; Kilian et al. 2007; Ozkan et al. 2010). Although *T. urartu* had no role in wheat domestication, it played a critical role in wheat evolution and donated the A^u genome to all tetraploid and hexaploid wheat (Dvorak et al. 1993; Zohary and Hopf 2000).

Wheat domestication occurred mainly in the wild tetraploid wheat. There are two wild tetraploid wheat species with similar morphology and different genomic structure known as *T. dicoccoides* and *T. araraticum* (Zohary and Hopf 2000). Wild emmer (*T. dicoccoides*) naturally grows across the Fertile Crescent and Rosh pinna, eastern Galilee (Peng et al. 2011).

The domesticated form of *T. dicoccoides* is known as *T. dicoccum* (emmer, A^uA^uBB), which is believed to be domesticated in southeast Turkey (Ozkan et al. 2002, 2005; Mori et al. 2003; Luo et al. 2007). Geographical location of domestication for tetraploid wheat was reconsidered by Ozkan et al. (2005) and Luo et al. (2007). Phylogenetic analysis revealed two different races of *T. dicoccoides*: the western race which is the native of Israel, Syria, Lebanon and Jordan; and the central-eastern race, which has been frequently sampled in Turkey and rarely in Iraq and Iran. Ozkan et al. (2002) Mori et al. (2003) and Luo et al. (2007) reported the central-eastern race as the only progenitor of the domesticated germplasm at a single site in eastern Turkey. However, Peng et al. (2011) strongly support the model of multiple-site independent domestication of wild emmer wheat across the Levant (the western part of the Fertile Crescent). According to the model of multiple site independent domestication, the genes for non-brittleness were incorporated into many wild emmer genotypes through frequent spontaneous hybridizations, followed by human selection. Consequently, emmer wheat evolved to different polymorphic tetraploid species rather than a single genotype (Feldman and Kislev 2007). Several cultivated tetraploid A^uA^uBB wheat were derived later from the domesticated emmer: *T. carthlicum* (Persian wheat), *T. polonicum* (Polish wheat), *T. ispahanicum*, *T. turanicum* (Khurasan wheat), and *T. turgidum* (English or pollard wheat) (Peng et al. 2011). However, *T. durum* (free-threshing naked wheat) originated from *T. dicoccum* somewhat later (Damania 1998) and possibly independently (Salamini et al. 2002; Ozkan et al. 2005).

Breeding Aspects of Durum Wheat

Repeated cycles of inbreeding and double haploid technology are two main strategies in durum breeding program specifically in North America (Knox et al. 2000). Simultaneous improvement of agronomic performance, disease resistance and grain quality traits are the main

breeding objectives in durum breeding programs. The most important agronomic traits for durum include grain yield, drought tolerance, disease and insect resistance, straw strength, resistance to shattering, and harvest ability. Important physical grain quality traits are seed weight, seed size, and percentage of hard vitreous kernels. The most important processing quality traits include protein concentration, yellow pigment concentration, grain cadmium concentration, gluten strength and semolina milling properties. Incorporating and maintaining stable resistance to the seed borne diseases, wheat rusts, leaf-spotting diseases, and *Fusarium* head blight have been major objectives in numerous durum breeding programs.

Fusarium Head Blight

History of FHB

The disease name "wheat scab" harks back to the first documented FHB-outbreak in England in 1884 (Stack 2003). Approximately at the same decade, scab outbreaks have been reported in the Americas (Gilbert et al. 2000; McMullen 2003), Asia (Bai et al. 2003; Bhat et al. 1989), Australia (Burgess et al. 1987), Europe (Parry et al. 1995; Xu et al. 2008), and South Africa (Kriel and Pretorius 2008; Scott et al. 1988).

All the evidence show that FHB epidemic started in 1917 and caused tremendous grain losses exceeded to 288,000 metric tons (MT), and later the grain losses increased to 2.18 million MT in 1919. Large yield losses were also recorded during the period between 1928 and 1937. The FHB impact continuously increased to 2.72 and 4.78 MT in 1982 and 1993 in North Dakota, South Dakota, Minnesota, and Manitoba, but during the period of 1998 to 2000 the grain loss decreased to 1.3 MT in the United States (McMullen et al. 1997; Stack 2003). Although the FHB outbreak was not as tremendous as previous years during 1998 to 2000, it still caused $ 2.7

billion loss in wheat and barley (*Hordeum vulgare* L.) in the United States (Nganje et al. 2002). The evidence showed that, scab caused $6.2 billion loss only in northern Great Plains from 1993 through 2001 (Nganje et al. 2004).

To date seventeen different *Fusarium* species have been isolated from infected wheat and barley (Gale 2003). However, the most virulent type was *Fusarium graminearum* schwabe [telomorph= *Gibberella zea* (schwein) Petch] worldwide (Bai and Shaner 2004). *Fusarium. graminearum* is a homothallic fungus and the most prominent causal agent of FHB in the U. S., Canada, and Europe (McMullen et al. 1997). Other related species such as *F. culmorum* (Smith) Sacc., *F. avenaceum* (Fries) Sacc., *F. moniliforme* Sheldon., *F. oxysporum* Schlect., *F. poae* (Peck) might also be causal involved in head blight complex. Several or all of these pathogens can be found simultaneously in wheat spikes (Wiese 1987). This fungus has impressive survivability on living and dead residue of a wide range of hosts (Shaner 2003). Likely, *Fusarium* ascospores are released from the soil surface, and airflow disperses them under favorable environmental conditions. Hence, plant debris and residue are the most important primary sources of inoculum (Sutton 1982). Plant infection initiates when airborne spores invade the head at anthesis. Under favorable environmental conditions, infection spreads throughout the developing caryopsis, floral bracts, and rachis (Bai and Shaner 1994).

Poor quality is a consequence of *Fusarium* invasion to the wheat kernel, which is a consequence of the destruction of starch granules, cell walls, and storage proteins in endosperm (Bechtel et al. 1985; Meyer et al. 1986; Nightingale et al. 1999). As a result, a *Fusarium* epidemic can be a serious problem for seed production. In heavily affected kernels, the embryos may be infected and seed germination is reduced (Bechtel et al. 1985). Root rot in small grains,

including durum, hexaploid wheat (*T. aestivum*), oat (*Avena sativa),* barley (*Hordeum vulgare*), and rye (*Secale cereale* L.) are other symptoms of this complicated pathogen (McMullen et al. 1997).

End use quality, as relates to human and animal consumption, is also drastically affected by *Fusarium* infection. The most notorious mycotoxins of *Fusarium culmorum* and *Fusarium graminearum* are of the trichothecene class: deoxynivalenol (DON, vomitoxin), acetyl-deoxynivalenol (the isomers 3-ADON and 15-ADON) and nivalenol (Niv) (Marasas et al. 1984; Marasas 2001). DON produced with head blight in wheat can also have physiological effects in other parts of the plant. Growth of wheat coleoptile is strongly inhibited at 10^{-3}M concentration of DON and 3-ADON. However, wheat seedling growth is completely inhibited at a concentration of 10^{-4}M of DON in infected seed. Thus, selection for low head blight ratings will indirectly select for low DON contamination (Snijders 2004).

Although accumulation of DON is the most frequent outcome of FHB, the toxicity of other isomers such as 3-ADON and 15-ADON is about twice that of DON (Mirocha et al. 1989) and NIV toxicity is10 times greater than that of DON (Joffe 1986). *Fusarium*-contaminated grain is the main source of mycotoxins in the food chain, and it can cause Alimentary Toxic Aleukia (ATA), a potentially fatal condition in humans (Foroud and Edues 2009). Trichothecene poisoning in humans may occur because of dietary intake of DON; the symptoms are characterized by: skin irritation, food refusal, vomiting, diarrhea, hemorrhages, neural disturbance, miscarriage and death (Joffe 1986; Kuiper-Goodman 1985). Chronic ingestion of small amounts of trichothecenes may suppress the human immune system and predispose the human body to other infectious diseases (Kuiper-Goodman 1985; Miller and Atkinson 1987).

Feeding animals contaminated grain leads to poor performance, health problems, and eventually economic losses. Only 0.3mg kg^{-1} of DON in swine diets result in decreased feed consumption and weight gain (Trenholm et al. 1984). Young poultry such as chickens and turkeys can tolerate diets that contain DON up to at least 5 mg kg^{-1} from wheat (Hamilton et al. 1985).

Source of Inoculum and Life Cycle of Fusarium

Fusarium spp. can survive saprophytically on plant debris (Parry et al. 1994). *Fusarium graminearum* can maintain its viability as mycelium, ascospores, macroconidia and chlamydospores. Owing to wide range of graminaceous hosts, FHB epidemic may originate from inoculum associated with host residues (Sutton 1982). Thus, continuous wheat cropping or using corn and wheat in rotation can significantly increase the possibility of FHB outbreak (Pirgozliev et al. 2003). It is also believed that, broad leafs weeds are another source of inoculum and overwintering for *Fusarium* (Jenkinson and Parry 1994)

Perithecia (sexual fruiting structures) form on host residue and release ascospores after maturing. Natural infection occurs when air-born ascospores land on spikelets during anthesis, where they germinate and enter the tissues using natural openings in the lemma, palea and glume, or through the anther (Ritieni and Edwards 2008; Trail 2009; Parry et al. 1995). After penetrating the plant, fungus can initially grow in intercellular space, and then start colonizing intracellular and entire tissue (Bushnell et al. 2003; Trail 2009).

At infection onset, the fungus grows intercellularly and asymptomatically (Bushnell et al. 2003; Guenther and Trail 2005; Jansen et al. 2005), spreading through the xylem and pith. Necrosis begins as the fungus grows rapidly and colonizes the tissue. Water soaking is initial symptom at this stage which particularly occurs in chlorenchyma tissue. Following water

soaking, colonized tissue becomes bleached which is typical hallmark for head blight of wheat. Almost immediately after floret infection, the fungus starts expressing genes for DON biosynthesis (Jansen et al. 2005). The sexual proliferation for *F. graminearum* starts with dikaryotic phase (binucleate phase) which occurs when two genetically distinct nuclei remain paired as new cells form. An extended dikaryotic phase is a specific hallmark of the phylum Ascomycota (to which *F. graminearum* belongs). Homothallism is another specificity of this fungus which is due to the presence of genes associated with both mating types (*Mat1-1 and Mat1-2*) in the haploid genome (Yun et al. 2000). Consequently, this fungus does not need a sexually distinct partner to regenerate sexual spores (ascospores), and as a result, the two nuclei of the binucleate could be genetically identical. *Fusarium graminearum* can complete its life cycle in association with its host. Similar to majority of fungi, *Fusarium graminearum* is haploid for most of its life cycle and sexual proliferation begins with the formation of hyphae with binucleate cells. The binucleate cells develop to groups of small coiled cells, which are the fruiting body initials (Trail and Common 2000). The initials develop uninterrupted into flask-shaped perithecia which contain tubular sacs. The tubular sacs named asci contain ascospores, which are the products of meiosis. Asci move up to the mouth of the perithecium and discharge their ascospores into the air. The entire life cycle takes about two weeks, in the laboratory, however asci maturing ascospores releasing occur in the last 4 day of life cycle (Bowden and Leslie 1999; Trail et al. 2002). Perithecium initials regenerate in association with the plant's stomatas and silica cells in infected plants, and together with the binucleate hyphae form the overwintering structures (Guenther and Trail 2005). Perithecia are ephemeral and airborne ascospores are the primary inoculum. Therefore, the disease is considered to be a monocyclic, and elimination of the sexual stage results in substantial disease reduction in field trials

(Desjardin et al. 2006). In warm and humid environmental condition, copious numbers of conidia (*i.e.*, asexual spores) are produced on the surface of infected plants or on crop residue. The fusiform conidia are produced in slimy masses on sporodochia (cushion-shaped hyphal structures) and they are spread by rain-splash (Deacon 2006). Although the relative contribution of conidia versus ascospore to disease outbreak is still unknown, the short-distance required for dispersal of conidia is well documented (Shaner 2003).

Mechanisms of Fusarium Head Blight Resistance

The FHB resistance is both dominant and quantitative. Neither gene-for-gene resistance interaction, nor immunity to the disease has been reported so far in wheat (Buerstmayr et al. 2009). Stability of resistance is dependent on environmental factors at the time of infection and aggressiveness factors associated with the invading Fusarium strain, although resistance has been shown to be stable in genotypes with very high levels of resistance (Mesterhazy 1995; Miedaner et al. 2001).

There are five types of physiological resistance for FHB in the host listed in the literature, namely resistance to (i) disease penetration (Type I), (ii) floral spread (Type II), (iii) Fusarium Damaged Kernel (FDK; Type III), (iv) yield reduction (Type IV) and (v) mycotoxin accumulation (Type V) (Schroeder and Christensen 1963; Mesterhazy 1995). Boutigny et al. (2008) defined two other type of resistance: Type V-1 resistance refers to the host's ability to chemically modify trichothecenes, resulting in toxin degradation or detoxification; Type V-2 resistance refers to the host's ability to inhibit trichothecenes. Investigations have shown positive association between different components of resistance index, hence selection for one type of resistance would indirectly enable selection for other types (Lemmens et al. 2005).

Measuring mycotoxin accumulation is expensive and Type I resistance is difficult to assess; therefore, screening genotypes for Type II resistance is easier for assessing FHB infection (Buerstmayr et al. 2009). Interestingly, delayed hyphal colonization of the vascular bundles in the rachis is observed in Type II resistant genotypes (Kang and Buchenauer 2000); that is, disease spread is inhibited. Although tricothecenes do not appear to play a role in initial infection of kernels or fruit coat (Bai et al. 2001; Jansen et al. 2005), trichothecenes are present and necessary for disease spread (Proctor et al. 1995; Eudes et al. 2001; Bai et al. 2001; Langevin et al. 2004). Consequently, Type II resistance might be an effective disease index for selecting genotypes with lower amount of mycotoxin accumulation (Type V resistance).

Resistance Sources

To date, FHB-resistance sources have been identified in hexaploid wheat germplasm from China, Japan, Korea and Brazil (Buerstmayr et al. 2009). During the past fifteen years, incorporation of resistance into the best available cultivars has been a major focus of breeding programs. Undesirable agronomic traits and low adaptation to various environments are inherent problems of resistance sources (Buerstmayr et al. 2009; Ghavami et al. 2011).

FHB epidemics have long been a major problem in some wheat growing regions of Asia (Liu 1985). Thus, breeding for FHB resistance was an early priority for Asian wheat breeding programs. ‘Sumai 3’, ‘Ning 7840’, ‘Ning 8331’, ‘Wangshuibai’, ‘Chokwang’ are cultivars which have resistance, and have been utilized for breeding and genetic studies of Fusarium resistance worldwide. Although these cultivars and lines have some resistance QTL in common, different QTL also have been reported (Buerstmayr et al. 2009). Frontana is another moderate resistant hexaploid wheat which has been used for genetic studies and breeding (Mardi et al.

2006). Winter wheat cultivars including 'Sincron' (Ittu et al. 2000), 'Renan' (Gervais et al. 2003), and 'Arina' (Paillard et al. 2004) also have been used widely in FHB resistance analysis. Wheat-related species also can be alternative sources for FHB resistance (Mentewab et al. 2000). *Thinopyrum ponticum* (syn. *Lophopyrum ponticum*) is a wild relative that was used for chromosome substitution and translocation, with enhanced response to FHB (Shen et al. 2004). Several alien species, including *Elymus humidas*, *Elymus racemifar*, *Roegneriakamoji* and *Leymus racemosus* have been used as donors of FHB resistance genes. However, there is no evidence for mapping the involved QTL in these species (Ban 1997, Chen et al. 2005; Oliver et al. 2005).

Limited sources of variation for FHB resistance in *T. durum* necessitate the use of wild relatives like *T. dicoccum* and *T. dicoccoides* as alternative sources for resistance genes (Buerstmayr et al. 2003b; Oliver et al. 2007). Ban and Watanabe (2001) reported that chromosome 3A of *T. dicoccoides* accession FA-15-3 (syn. Israel A) is involved in FHB resistance. Otto et al. (2002) screened recombinant inbred chromosome lines (RICL) and identified a major type II resistance QTL located on chromosome3A. Somers et al. (2006) identified a resistant line carrying an effective QTL in a population by screening a mapping population derived from the cross of the *T. durum* cultivar 'Strongfield' with the *T. carthlicum* cultivar 'Blackbird'. Elias et al. (2005) screened 7,500 durum accessions and identified five lines for Tunisian germplasm with moderate Type II resistance. Huhn et al. (2012) confirmed that Tunisian line numbers 7, 18, 34, 36 and 108 possess novel Type II resistance. Ghavami et al. (2011) screened populations derived from crosses between adapted cultivars and Tunisian lines 7, 18, 34 and 36 and identified lines exhibiting transgressive segregation for Type II FHB resistance.

Trait Assessment

Plant Materials

The main goal in trait introgression is to identify near isogenic lines (NIL) with a desired trait(s) of a donor line in a specific genetic background (Eshed and Zamir 1995). In most genetic mapping studies, a population is developed that segregates for the trait in question. The population is derived from a cross between two genotypes that exhibit substantial phenotypic difference for the trait studied. Recombinant inbred lines (RILs), fully homozygous doubled haploid (DH) populations, or populations derived from backcrosses are the most common population types in mapping projects. Backcross-derived populations are advantageous in cases where the resistance donor is an exotic or wild line and the recipient line is an adapted genotype (Tanksley and Nelson 1996).

Phenotyping

The most critical aspect of phenotypic evaluation for Fusarium resistance is accurate data assessment, due to highly variable genotype by environmental interactions. Disease severity depends on resistance factors in the plant, aggressiveness of the fungus and environmental conditions (Fuentes et al. 2005). To decrease the environmental effects on disease assessment, uniform inoculum pressure in field and greenhouse is critical (Dill-Macky 2003). The pathogen species or pathogen strains utilized for inoculation also have drastic effect on precise disease assessment. Although various species may be involved in disease incidence, *Fusarium graminearum*, *Fusarium culmorum* and *Fusarium avenaceum* have been detected as the dominant species in FHB occurrence (Parry et al. 1995; Dill-Macky 2003).

Despite high genetic variability in *Fusarium spp*. (Bowden and Leslie 1999), resistance has shown inherent non-specific qualities for the most prevalent species like *Fusarium culmorum* and *Fusarium graminearum*. To date, no biological races with specific host–pathogen interaction have been detected, while aggressiveness of well-known strains (isolates) is widely different (Snijders and Van Eeuwijk 1991; Van Eeuwijk et al. 1995; Mesterhazy et al.1999). Inoculation methods could be another important factor in precise disease quantification. Spraying conidia, grain-spawn method (scattering *Fusarium*-infected corn or barley grains), sowing infected maize stubble on the soil and single spikelet inoculation are the usual methods used to mimic natural infection conditions (Buerustmayer et al. 2009).

Disease assessments must be in accordance with the type of resistance under investigation. To date, five types of resistance to FHB are used to quantify and characterize infection. These types are as previously presented under 'Mechanisms of Fusarium Head Blight resistance.

Genotyping

Using genetic markers in plant breeding were first reported about ninety years ago (Crouch and Ortiz 2004). However, after innovation of DNA markers technology in 1980s, the efficiency of plant breeding has been dramatically enhanced (Jonah et al. 2011). Construction of genetic maps using restriction fragment length polymorphism (RFLP) opened a new era for genotypic characterization of living things (Botstein et al. 1980). After the invention of polymerase chain reaction (PCR) technology (Mullis and Faloona 1987), a large number of approaches were developed for marker deployment. The most important criteria for developing ideal makers are: (1) level of polymorphism, (2) abundance (3) adequate resolution for genetic

differences (4) locus specificity (5) amount of linkage to distinct phenotypes, (6) simplicity, and (7) technical requirements and cost. The choice of marker to utilize depends upon numerous considerations, including the species under investigation, the objectives, equipment and resources available to the researcher (Agarwal et al. 2008). The goal of genotypic characterization in population genetics is to define the genetic variability present in the given experimental population. Generally, this means determining the relative genetic contributions of the parental lines to the progenies. Molecular markers provide the means to determine genetic differences among lines in a population at various loci. The segregation of markers within a population presents the possibility of determining the relative genetic distance between loci. There are numerous types of molecular markers available for genetic characterization (Agarwal et al. 2008). The most used molecular markers for genetic studies are: restriction fragment length polymorphism (RFLP; Botstein et al. 1980), random amplified polymorphic DNA (RAPD; Williams et al. 1990), amplified fragment length polymorphism (AFLP; Vos et al. 1995), microsatellite or short tandem repeats or simple sequences repeats (SSR; Tautz and Renz1984; Akkaya et al. 1992), single nucleotide polymorphism (SNPs) with high throughput SNP detection systems (Shen et al. 2005, Rostoks et al. 2006), and array-based medium to high throughput markers like DArT (diversity array technology; Akbari et al. 2006).

Molecular Mapping of Quantitative Loci

Many traits of agronomic importance such as FHB resistance are regulated by multiple genes. Such traits known as quantitative traits show a continuous range of variation in a population. Establishment of large collections of molecular/genetic markers, which could be used to construct detailed genetic maps of species have been a key development in the field of complex trait analysis. The detailed maps then could provide the foundation for modern-day

QTL mapping methodologies (Edwards et al. 1987; Korolet al. 1998). QTL mapping was proposed by Sax (1923) for the first time and elaborated by Thoday (1961). The basic concept of QTL mapping is to test the association of genomic and chromosomal regions with the quantitative traits of interest (Mohan et al. 1997; Young 1996). The amount of co-segregation of a molecular marker with a QTL depends on the distance between them (Collard et al. 2005; Young 1996). There are several factors that may affect accuracy of QTL mapping. A high-density map can potentially provide more power to detect authentic QTL. QTL with minor effect may not be detectable especially when heritability of the trait is low (Hyne et al. 1995). Discrimination of two QTL which are very close on the same chromosome might be impossible (Kearsey and Farquhar 1998; Young 1996). Since QTL mapping starts with a mapping population, the parents for the population should exhibit significant contrast for desired trait(s) (Liu 1998; Collard et al. 2005). For high resolution QTL mapping usually a large population is required (Collard et al. 2005). However, populations consisted of 70 to 250 lines has been used in most preliminary mapping studies (Mohan et al. 1997).

Some of the routine techniques are single-marker analysis (SMA; Edwards et al. 1987; Luo and Kearsey 1989), interval mapping (Lander and Botstein 1989), composite interval mapping (CIM; Zeng 1993; Zeng 1994; Jansen 1993; Jansen and Stam 1994) and multiple traits mapping (Jiang and Zeng 1995; Korol et al. 1998), or multiple interval mapping (MIM; Tanksley 1993) which provide statistical analyses of the associations between phenotypes and genotypes. The main purpose is to dissect the specific regions of a genome that affect complex traits.

The simplest method for QTL mapping is analysis of variance (ANOVA, sometimes called “marker regression”) at the marker loci (Soller et al. 1976). This method has the capacity

of inclusion of more covariates, such as treatment and environment. ANOVA approach for QTL mapping has three major weaknesses: lack of separate estimates of QTL location and QTL effect, individuals with missing genotypic data have to be excluded from analysis, and low power for QTL detection for widely spaced markers (Broman 2001).

Single marker analysis (SMA) or single locus regression method makes use of a genetic map of the typed markers, and similar to analysis of variance, assumes the presence of a single QTL. Each location in the map is assessed as the location of the putative QTL once at a time. Interval mapping has several advantages over analysis of variance at the marker loci. This analysis can depict a curve which indicates the evidence for QTL location. It provides the information about QTL positions and QTL effects. Interval mapping is applicable even when the marker-genotype data is incomplete (Lincoln and Lander 1992). SIM analysis requires higher level of statistical software and higher amount of time for computational process in comparison to analysis of variance.

There are several advantages to multiple QTL mapping, especially when the QTL are located on separate chromosomes. In this way, the model maintains the presence of a QTL, then reduces the residual variation, and eventually provides greater power to detect other QTL. The multiple QTL mapping can separate the linked QTL, and identifies the interaction (epistasis) between them (Mackay 1996, 2001).

The simplest multiple QTL method is forward selection in interval mapping which can be used even for missing marker-genotype data. Composite interval mapping (CIM) is a highly utilized approach widely applied in practice (Jansen 1993; Zeng 1994). To reduce the residual variation, a subset of marker loci needs to be selected as covariate in this method. The subset of

marker loci serves as proxies for other QTL and increase the mapping resolution. Selecting the appropriate marker loci is always a matter of concern in CIM, and this problem has not been resolved (Broman 2001). An interesting development is multiple interval mapping (MIM), which practically is an extension of interval mapping and analysis of variances (Kao et al. 1999; Zeng et al. 1999). This method covers all the above mentioned advantages, but it cannot be the final solution for all of the QTL mapping problems.

Short Review of FHB Resistance QTL Mapping

QTL for FHB resistance have been found on almost all wheat chromosomes (Buerstmayr et al. 2009). The story of molecular mapping for FHB resistance QTL almost started with Waldron et al. (1999) investigation, when they identified five genomic regions with significant association with FHB resistance using RFLPs. Using interval analysis on recombinant inbred lines derived from Somai3 (resistance)×Stoa (moderately susceptible), they identified three QTL on chromosome 3BS of Sumai3 and two QTL on 2AL of Stoa. These two group of assigned QTL (*QFhs.ndsu-3B* and *QFhs.ndsu-2A*) then were validated by other research groups (Anderson et al. 2001; Buerstmayr et al. 2002), and even Cuthbert et al. (2006) confirmed that, the *Fhb* 1 on chromosome 3BS is the most effective FHB resistance factor ever identified. It is commonly believed that, the *Fhb* 1 major resistance QTL has been donated from a Taiwan parent to sumai3 (Bai et al. 2003; Shen et al. 2003). Bai et al. (1999) screened 133 recombinant inbred lines derived by single seed descent from Ning 7840×Clark, and they detected a FHB QTL with major effect using AFLP. With 300 primer combinations and two contrasting bulked DNA, they detected 11 polymorphic loci, associated with resistance and used these markers for linkage map construction with unknown chromosomal location.

Combining RFLP, AFLP, and SSR markers, Anderson et al. (2001) mapped two new QTL on chromosome 3AL and 6AS using a recombinant inbred line population derived from ND2603 (sumai3×wheaton)×Butte 86. They screened all the DNA markers which were associated with resistance on the Sumai3×Stoa population and also reported the major effect of the QTL located on 3BS and 6BS incorporated from Sumai3. Although Bai et al. (2003) believed that, the 3BS QTL may not be the same as *Fhb* 1 in different resistant cultivar, Mardi et al. (2005) and Yu et al. (2008) strongly verified the same 3BS-QTL in other resistant cultivar "Wangshuibai".

Buerstmayr et al. (2003a) mapped two major QTLs on chromosomes 3B (*Qfhs.ndsu-3BS*) and 5A (*Qfhs.ifa-5A*) that explained 40 to 48% of phenotypic variation for visual resistance to FHB severity (Type II resistance), using DH lines derived from the cross between CM-82036 (resistant) and Remus (susceptible). The *Qfhs.ndsu-3BS* appeared to be associated mainly with resistance to fungal spread (Type II), and *Qfhs.ifa-5A* primarily with resistance to fungal penetration (Type I). Somers et al. (2003) characterized alternate sources for FHB resistance using 91 double haploid lines derived from cross between Wuham and Maringa. Using 328 polymorphic loci, they reported two novel QTL located on chromosome 4B and 3BS proximal to centromere and confirmed two other QTL on chromosome 2DL and 3BS. The application of QTL mapping was interestingly depicted in their investigation, because all lines with 2DL, 3BS FHB resistance alleles showed 32% of pathogen infection, and the line with 4B, 3BS alleles showed 27% of infection.

Stack et al. (1999) screened a series of Langdon-*T. dicoccoides* [LDN(DIC)] chromosome substitutions for FHB resistance and identified LDN(DIC-3A) substitution as

resistance to FHB type II infection. Otto et al. (2002) studied detailed map of chromosome 3A using a population consisted of recombinant inbred chromosome lines (RICLs) derived from *T. dicoccoides* LDN (DIC-3A) substitution and Langdon-16. They identified a major Type II resistance FHB QTL on long arm of chromosome 3A which could explain 37% of phenotypic variation.

Oliver et al. (2008) evaluated 375 accessions of five subspecies to identify new sources of resistance. In this applied investigation, Persian wheat [*T. turgidum* subsp. carthlicum (Nevski) Á. Löve and D. Löve], cultivated emmer wheat [*T. turgidum* subsp. dicoccum (Schrank ex Schübler) Thell.], Polish wheat [*T. turgidum* subsp. Polonicum (L.) Thell.], Oriental wheat [*T. turgidum* sub sp. *turanicum* (Jakubz.) Á. Löve and D. Löve], and Poulard wheat (*T. turgidum* L. sub sp. *turgidum*) were used in over three greenhouse seasons and two field screening. Preliminary evaluation showed that 16 *T. turgidum* subsp. carthlicum and 4 *T. turgidum* subsp. dicoccum accessions consistently exhibited resistance or moderate resistance to FHB.

Ghavami et al. (2011) identified a major Type II FHB resistance QTL on chromosome 5B and several minor QTL in an association mapping study using four different BIL populations derived from Tunisian Durum wheat.

Project Justification

Annual worldwide grain production of durum wheat (*Triticum turgidum* L. var. *durum*) is estimated at 27.5 million metric tons. But FHB causes tremendous changes in this amount. North Dakota, as the largest durum producer in USA (over 80% of the acreage of durum

farmlands), suffered over three billion dollars of total loss because of FHB epidemic in the period of 1993-1997 (Nganje et al. 2004).

As cultural and management practices, the most effective fungicide treatments can only reduce damage from FHB by about 50% (McMullen et al. 1997), identification of resistance sources in wheat germplasm and pyramiding resistance factors into the most productive cultivars is still the most cost-effective method for disease control. Lack of sources with complete immunity to FHB caused considerable explorations for resistant and moderately resistant genotypes in wheat breeding programs (Waldron et al. 1999). Unfortunately, neither seedling tests nor *in-vitro* screening methods for FHB resistance have been established or validated to date (Buerstmayr et al. 2002). Therefore, visual selection for FHB resistance is still the most applicable screening method which can be combined with marker assisted selection to accelerate resistance breeding process (Stack et al. 2002).

Apart from a few exceptions, the actual function of FHB resistance genes is not known and there is no evidence for large effect FHB resistance gene (QTL) cloning to date (Buerstmayr et al. 2012). Despite considerable number of reports for FHB QTL mapping in hexaploid wheat (Waldron et al. 1999; Bai et al. 1999), there are a few FHB resistance QTL mapping for tetraploid wheat. There are approximately 52 peer-reviewed studies reporting QTL for FHB resistance in wheat, from which, 46 were identified in hexaploid wheat, 4 in tetraploid and 2 in related species.

Incorporation of FHB resistance from common wheat to durum has met with limited success (Ghavami et al. 2011; Burestmayr et al. 2012). Thus, the overall levels of FHB resistance available for exploitation in durum wheat breeding programs are low. Incorporation

of FHB resistance into regional breeding material has been the essential wheat breeding strategy. Thus, so called 'exotic' resistance sources (*e.g.* Asian spring wheat) or moderately effective 'native' resistance have been used to improve tetraploid wheat germplasm.

The durum wheat breeding program at North Dakota State University (NDSU) screened approximately 6000 durum wheat accessions, but led in no achievement in first attempt (Elias et al. 2005). In further investigation NDSU durum wheat breeding program screened 1500 accessions from the International Center for Agricultural Research in the Dry Areas (ICARDA), and eventually selected five Tunisian lines with a moderate level of Type II resistance (Elias et al. 2005). The same Tunisian sources as "exotic lines" with promising level of resistance to FHB Type II were used to reinforce locally adapted cultivars of North Dakota.

Huhn et al. (2012) reported that the Tunisian lines number 7, 18, 34, 36 and 108 may possess novel Type II resistance QTL. Ghavami et al. (2011) revealed that, the genetic backgrounds of Tunisian derived lines are different and even they can rival with the best resistant hexaploid wheat (sumai 3) with 10 to 25% of infection rate owing to transgressive segregation. In order to characterize FHB resistance QTL in Tunisian germplasm, two back cross derived recombinant inbred lines population were screened in this investigation.

Objectives

This study emphasizes the importance of developing highly resistant cultivars through introgression of resistance from moderate resistance sources. The objectives of current study were to identify genomic regions significantly associated with FHB resistance and incorporate Tunisian FHB resistance QTL into the genomic background of locally adapted durum wheat

cultivars of North Dakota. Thus, FHB epidemic have been simulated in the field and greenhouse to scrutinize two segregating population (i.) to develop a molecular linkage map, (ii.) to identify genomic regions significantly associated with FHB resistance, (iii.) to determine molecular markers linked to FHB resistance (iv.) to introduce promising new line(s) with FHB resistance that can be used as sources for further durum wheat breeding programs.

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CHAPTER III. DETECTION OF NOVEL QTL FOR FUSARIUM HEAD BLIGHT RESISTANCE IN A TUNISIAN-DERIVED DURUM WHEAT POPULATION

Abstract

Controlling Fusarium head blight (FHB) damage has been a challenge for durum wheat (*Triticum turgidum*) producers in the US for more than two decades. Tremendous economic losses due to decrement in grain yield and quality are attributed to this disease. Thus, transfer and pyramiding of FHB-resistance loci into locally adapted cultivars is critical. In this study, a backcross inbred line population (BC_1F_7) of 171 individuals derived from a cross between a resistant durum genotype 'Tunisian 108' and a susceptible durum cultivar 'Ben' was used to identify regions associated with FHB resistance. The population, along with checks, was evaluated for FHB Type II resistance in two greenhouse and two field experiments. Analysis of variance showed significant effect of genotypes on FHB severity and incidence as well as environment and G×E interaction. Approximately 8% of the lines in field nursery and 25% of the lines in the greenhouse screening were consistently more resistant than the Tunisian parent. A framework linkage map of 329 markers was developed representing 239 unique loci and covering a total distance of 1887.6 cM. Composite interval mapping revealed 11 different QTL on seven different chromosomes. A novel region on chromosome 2B was identified (*Qfhb.ndsu-2B*) which provides resistance to multiple FHB components including severity, incidence, mycotoxin production and frequency of damaged kernels. Introgression of this segment can be beneficial to the development of FHB-resistant durum cultivars. A region identified on chromosome 5A in this study has been identified in other hexaploid and tetraploid material indicating a possible evolutionary significance.

Introduction

Fusarium head blight (FHB), caused primarily by the fungus *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schw.) Petch], is a devastating disease of both common wheat (*T. aestivum* L., 2n = 6x = 42, AABBDD) and durum wheat (*T. turgidum* L. var. *durum* Desf., 2n = 4x = 28, AABB) worldwide (Chen et al. 2007). The impact of this disease on wheat is exceptionally high under warm and humid conditions (Stack and McMullen 1985; Gilbert and Tekauz 2000). Favorable conditions for *Fusarium* infection from 1997-2000 caused an epidemic in the US resulting in $ 2.7 billion loss in wheat and barley (*Hordeum vulgare* L.) (Nganje et al. 2002), and $ 6.2 billion in the Northern Great Plains from 1993 through 2001 (Nganje et al. 2004).

Fusarium head blight is commonly referred to as "wheat scab"; a descriptive nomination that harkens back to the first documented FHB-outbreak, which occurred in England in 1884 (Stack 2003). In the same approximate decade, FHB outbreaks were reported in North America (Gilbert et al. 2000; McMullen 2003), Asia (Bai et al. 2003; Bhat et al. 1989), Australia (Burgess et al. 1987), Europe (Parry et al. 1995; Xu et al. 2008), and South Africa (Kriel and Pretorius 2008; Scott et al. 1988). FHB can cause bleached spikes, spikelet sterility, poor seed filling, low weight and tombstone seeds (Wang 1996). Furthermore, grain marketing value is drastically affected by scabby wheat because of mycotoxin contamination from deoxynivalenol (DON), diminished milling quality and baking quality (Gilbert and Tekauz 2000; Bechtel et al. 1985). Fusarium-contaminated grain is the main source of mycotoxins in the food chain which can cause Alimentary Toxic Aleukia, a potentially fatal condition in humans (Foroud and Edues 2009).

Development of host genetic resistance is the preferred strategy to reduce FHB effects, since cultural management practices and chemical controls are considered impractical or ineffective (Bai and Shaner 1994). Therefore, identification and incorporation of FHB resistance genes has been a point of particular emphasis for plant breeders, for the purpose of improving host plant resistance. Problems associated with collecting reliable FHB resistance data (Campbell and Lipps 1998) and the polygenic nature of this plant-pathogen interaction (Buerstmayr et al. 2009; Rudd et al. 2001) have driven researchers to identify tightly linked molecular markers to assist breeding programs (Bai and Shaner 1994). As FHB resistance generally exhibits the characteristics of a quantitative trait identification of linked markers require the development of populations such as recombinant inbred lines (RIL), near isogenic lines (NIL), double haploid lines, or backcross derived lines, genotypic and phenotypic screening and analysis.

There are five types of physiological host resistance for FHB listed in the literature; namely, resistance to (1) disease penetration (Type I), (2) floral spread (Type II), (3) Fusarium Damaged Kernel (FDK; Type III), (4) yield reduction (Type IV) and (5) mycotoxin accumulation (Type V) (Schroeder and Christensen 1963; Mesterhazy 1995). Investigations have shown positive associations between different types of resistance. Hence, selection for one resistance type may indirectly enable selection for another type (Lemmens et al. 2005). As type I resistance is difficult to assess and measurement of mycotoxin accumulation assays are expensive, most researchers have focused on Type II resistance as the preferred index for disease assessment following FHB infection (Buerstmayr et al. 2009).

Historically, FHB resistance sources have been limited to hexaploid cultivars such as 'Sumai 3', 'Wangshuibai' (Mardi et al. 2005) and, to a lesser extent, 'Frontana' from Brazil (Mardi et al.2006). Despite successful introduction of stable FHB resistance quantitative trait loci (QTL) into hexaploid wheat, poor yield and low grain quality were frequent early obstacles in developing FHB resistance (Buerstmayr et al. 2002). In recent decades notable high-quality cultivars with available FHB-resistance factors have been developed (Buerstmayr et al. 2012). However, introgression of FHB resistance sources from bread wheat to tetraploid wheat has been nominal (Buerstmayr et al. 2009; Ghavami et al. 2011). As well, utilization of limited host resistance imposes a strong selective pressure for virulent pathogen strains (Gervais et al.2003). Consequently, exploration for alternative sources of FHB resistance remains a necessary endeavor.

Hexaploid Chinese germplasm and derivatives have been studied extensively using quantitative trait loci (QTL) mapping (Bai et al. 1999; Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002; Zhou et al. 2002; Bourdoncle and Ohm 2003; Shen et al.2003). However, genetic studies of FHB resistance in tetraploid wheat are limited; due, in large measure, to a dearth of resistant resources. Otto et al. (2002) reported the first FHB resistance QTL on 3A (*Qfhs.ndsu-3AS*), and Kumar et al. (2007) identified another QTL on 7A (*Qfhs.fcu-7AL*), each from different sources of *T. dicoccoides*. The low contribution of FHB resistance QTL identified in durum wheat also is problematic. The largest QTL effect reported in durum was on chromosome 2BL, and explained only 26% of the phenotypic variance in the study (Somers et al. 2006). Thus, the overall amount of FHB resistance available for utilization in durum wheat is low.

The introgression of resistance components from common wheat into durum wheat has also been practically limited, due to different ploidy levels and the existence of suppressor genes (Ghavami et al. 2011). Suppressor genes that mask resistance QTL or enhance pathogen proliferation are anticipated to exist in tetraploid genomic background. A suppressor gene located on chromosome 5B in wheat exemplifies the situation where one gene can increase pathogen susceptibility, such as susceptibility to *Mycosphaerella graminicola* (Arraiano et al. 2007). Garvin et al. (2009) theorized the epistatic influence of suppressor genes on chromosome 2A that overcome FHB resistance in durum wheat. Significant additive gene action in FHB resistance (Snijders 1990) and other complications in host resistance breeding also need to be considered.

The lack of resistance sources in tetraploid durum wheat has shifted efforts toward utilization of wild emmer, alien species (Oliver et al. 2005; Oliver et al. 2007) and exotic lines (Ghavami et al. 2011). Recent investigation at North Dakota State University revealed five prominent Tunisian durum lines with moderate level of type II resistance to FHB and generally acceptable agronomic traits (Elias et al. 2005; Ghavami et al. 2011; Huhn et al. 2012). The objectives of the current study were: (i.) to identify genomic regions significantly associated with FHB resistance in a Tunisian durum line, (ii.) to construct a genetic linkage map, (iii.) to identify the most resistant line(s) for further breeding practices.

Material and Methods

Development of Backcross Inbred Line (BIL) Population

The present study used a BIL population developed from a cross of two durum wheat genotypes: 'Ben' and Tunisian 108. The recurrent parent 'Ben' is a medium height, high-yielding commercial spring durum wheat cultivar with nominal resistance to FHB (Elias and Miller 1998). The donor parent is Tunisian durum wheat genotype Tunisian 108, with moderate resistance to FHB. The F_1 plants from the cross of Ben and Tunisian 108 were backcrossed once to Ben and each line was then selfed for seven generations by single-seed descent method, resulting in a population of BILs. A total of 171 BC_1F_7 BILs along with their parental genotypes (Tunisian 108 and Ben) and fourteen checks were evaluated for FHB resistance in this study. The checks included four hexaploid resistant genotypes 'Sumai 3'(PI 481542), 'Wangshubai', 'Glenn'(Mergoum et al. 2006), and ND2710 (Frohberg et al. 2004); nine moderate resistant tetraploid genotypes 'Divide' (Elias and Manthey, 2007), 'Maier' (Elias and Miller 2000), 'Lebsock' (Elias et al. 2001), Tunisian 7, Tunisian 18, Tunisian 34, Tunisian 36, D91103, D95097; and one susceptible genotype, D87450. D91103, D95097, and D87450 are durum experimental lines developed by the durum wheat breeding and genetics program at North Dakota State University.

Phenotypic Evaluation

Phenotypic evaluation was performed in two field trials and two greenhouse experiments during 2010 and 2011. The field trials were conducted in Prosper, North Dakota and the data for resistance to initial infection (Type I) and disease spread (Type II) was recorded during both

years, and for deoxynivalenol (DON), 3-acetate deoxynivalenol (3ADON), 15-acetate deoxynivalenol (15ADON), nivalenol accumulation (Niv) and *Fusarium*-damaged kernel (FDK) assessment during 2011. Lines were planted in randomized complete block design (RCBD) with two replicates, were irrigated with water misting system 1 minute in 1 hour intervals, and were inoculated using corn kernel inoculation method in both field scab nursery experiments (Stack 1989). The inoculum was a mixture of three different isolates of *F. graminearum* (R010, R1267, and R1322) to maximize the infection likelihood and to simulate natural environmental conditions. Disease was assessed as the percentage (0 to 100%) of visually infected spikelets per whole spikes 21 days after anthesis (Gosman et al. 2005). The greenhouse trial included the population and check plants using RCBD with two replications for type II resistance (severity) in the USDA-ARS greenhouse, Fargo, North Dakota, during 2010 to 2011. Briefly: three seeds were planted in 10 inches clay pots and an average of 4 to 6 spikes per line in each of the two replicates were inoculated with a 10 μl droplet containing a mixture of conidia from three isolates (50,000 conidia/ml). Greenhouse inoculation was performed at beginning of anthesis. Inoculum was injected directly into the second or third single spikelet near the bottom of the spike, following procedures described by Stack (1989). Each inoculated spike was covered by misted plastic bag for 72 hours. Disease spread was scored according to visual symptoms and based on number of infected spikelets per spike 21 days after inoculation (Stack and McMullen 1985).

Statistical Analysis of Phenotypic Data

Analysis of variance for disease incidence, severity, FDK, DON, 3ADON, 15ADON and nivalenol were conducted using Proc GLM of SAS software version 9.1 (SAS Institute, Cary,

NC). Combined analysis was fulfilled after testing the homogeneity of error for different locations and years with the same software. Using Proc Corr command (SAS 9.1), Pearson's correlation between disease incidence, severity, FDK, DON, 3ADON, 15ADON and nivalenol contents were calculated. Broad-sense heritability (h^2) was estimated using following formula:

$$h^2 = \sigma^2_g/(\sigma^2_g+\sigma^2_{gy}/y+\sigma_e{}^2/ry)$$

Where:

σ^2_g = Genotypic variance

σ^2_{gy} = Genotype × year interaction variance

σ^2_e = Residual error variance

r = Replications

y = Years

Genotypic Analysis

DNA from the BIL population lines, checks, and parental lines was extracted according to conditions prescribed by Triticarte Pty. Ltd. (http://www.triticarte.com.au/). A total of 171 BILs, two parental genotypes and nine checks were genotyped using Diversity Array Technology (DArT) assay, performed by Triticarte Pty. Ltd. (Canberra, Australia; http://www.triticarte.com.au), as described by Akbari et al. (2006). An array of 2300 DArT markers, distributed across the entire durum wheat genome, was used to screen all lines.

In addition, 305 microsatellite markers were tested for polymorphism on the parental lines using standard polymerase chain reaction (PCR) conditions. These markers were selected from previously published wheat maps (Roder et al. 1998; Somers et al. 2004; Song et al. 2005) and consisted primarily of GWM (Gatersleben Wheat Microsatellites; Roder et al. 1995; Roder et al. 1998; Ganal and Roder 2007), WMC (Wheat Microsatellite Consortium; Gupta et al. 2002), Dupont Company (DuPw; Eujayl et al. 2002), CFA, CFD (INRA Clermont-Ferrand; Sourdille et al. 2004; Guyomarc'h et al. 2002), and BARC (Beltsville Agriculture Research Center; Song et al. 2005). PCR conditions were as originally described (Roder et al. 1998; Somers et al. 2004; Song et al. 2005). The PCR products were separated on 8% non-denaturing polyacrylamide gels and visualized using ethidium bromide.

Genetic Map Construction and QTL Analysis

The genotypic marker data for all the polymorphic DArT and SSR on 171 BILs was used to construct the linkage maps using MAPMAKER v. 2.0 (Lander et al. 1989). Briefly: A set of DART markers were selected and used as anchors based on available genetic maps (Mantovani et al. 2008; Peleg et al. 2008; Ghavami et al. 2011; Huang et al. 2012; Kumar et al. 2013) to assign remaining markers to individual chromosomes. The main criteria for anchor marker selection were that they have been mapped only to one specific chromosome in earlier studies (Mantovani et al. 2008; Peleg et al. 2008; Ghavami et al. 2011; Huang et al. 2012; Kumar et al. 2013), and in total, cover as much of each chromosome as possible. A minimum LOD score of 3.0 and maximum distance of 30 cM was used to assign markers to a particular group or chromosome. Map distances were calculated from recombination fractions using the Kosambi

mapping function (Kosambi 1943). Linkage maps were established using MapChart (Voorrips 2002).

QTL analysis was carried out by composite interval mapping (CIM) using QTL Cartographer V2.5 (Wang et al. 2006). To detect authentic QTL, we used model 6 with forward and backward step-wise regression, five markers as cofactors to control genetic background, and a 10 cM genome-wide scan window. A minimum LOD score of 2.0 was used for determining the presence of a putative QTL. Confidence intervals (CI) were obtained using positions ±1 LOD away from the peak and the QTL with overlapping CIs were considered as same. Permutation test using 500 iterations also was performed to determine threshold LOD scores for each trait in in each environment with 5% error rate. QTL above threshold LOD scores were considered definitive QTL.

Results

Phenotypic Analysis

There were significant differences among genotypes including the parents for disease severity resistance when evaluated in the field and the greenhouse (Table 3.1). There were significant differences among genotypes for all traits except 3ADON (Table 3.1). The low genetic variability for accumulation of the toxin in our experimental population could be the reason for lack of difference among genotypes for this trait. Correlations were computed for years and disease evaluations, tested for homogeneity, and pooled when deemed appropriate. There was no correlation between the disease severity (disease spread) in the greenhouse screening and field trial for 2010; however, a low but significant correlation was observed

between the greenhouse and the field experiments in 2011 (0.18, P<0.01). Significant positive correlation was found between the years for field (r=0.26, P< 0.0005) and greenhouse (r=0.16, P<0.04).

Since FDK, DON, 3ADON, and 15ADON were evaluated only at one location, the correlations between these factors were calculated only for 2011. Positive significant correlations were observed between disease severity in field and FDK (r =0.51, p<0.0001), FDK and DON (r =0.30, P<0.0001), and DON -3ADON, -15ADON -NIV, and 3ADON and 15ADON (Table 2). However, disease severity in field was not correlated with DON components (Table 3.2). Broad sense heritability estimates for resistance to disease severity in filed 0.23, disease severity in greenhouse 0.44, FDK 0.87, DON 0.43, 3ADON 0.9, 15ADON 0.47 and Niv 0.58 were calculated. Heritability estimates for FDK, DON 3ADON, 15ADON, and Niv are upward biased for they were calculated based on one environment and genotype by environment interaction could not be taken into account.

Transgressive segregation for resistance to FHB severity and incidence was observed within the population (Figure 3.1). Approximately 8% of the lines performed better than the resistant parent in the field for incidence and 25-30% of lines evaluated in the greenhouse for severity**.** Wide range of variation and transgressive segregation was observed for FDK within the population (Figure 3.2).

Genetic Map

Seventy one out of 305 microsatellite markers tested detected polymorphism between the two parental genotypes Tunisian 108 and Ben. DArT analysis identified an additional 310 polymorphic markers. These 381 markers (71 SSRs and 310 DArTs) were used to genotype the

mapping population comprised of 171 BILs. However, 30 highly distorted DArT markers were excluded. Therefore a total of 351 markers were used for genetic map construction. At a minimum LOD of 3 a total of 329 markers (267 DArT s and 62 SSRs) were mapped to 239 unique loci located on 13 chromosomes in exception of chromosomes 4B (Figure 3.3). The number of markers mapped on individual chromosomes varied from seven on 3A to 66 on 3B. The 329 mapped markers, representing 239 unique loci, covered a total genetic distance of 1887.6 cM, with an average distance of 7.89 cM between any two loci. The total genetic distance for B genome chromosomes was 1116.8 cM, and 770.8cM for A genome chromosomes. Individual chromosome lengths varied from 16.9 cM for 3A to 217.3 cM for 5B. Comparative investigation showed 211 out of 329 DArT and SSR mapped markers on Tunisian108/Beb//Ben (64.13%) were consistent with recently available bread and durum wheat maps (Marone et al. 2012; Mantovani et al. 2008; Akbari et al. 2006; Semagn et al. 2006; Crossa et al. 2007).

Distribution of Markers among Chromosomes and Genomes

The seven homologous groups of the tetraploid wheat genome varied in the number of markers, map length, and marker density. Total marker number and density was highest in homeologous group 3 (total 73 loci, with 3.20 cM per marker). Homeologous group 2 had the lowest marker number and density (total 27 loci, 10.26 cM per marker). Total map length was longest (381.9 cM) in group 1, whereas group 4 had the shortest map length (110.5 cM). Marker density also differed on the two genomes, with 182 (55.32%) markers mapped to the B genome and 149 (45.29%) to the A genome. Although DArT analysis revealed 11 polymorphic markers for chromosome 4B, they were excluded from mapping process due to unexpected distortion.

Lack of polymorphic microsatellite loci on chromosome 4B was another reason this portion of the genome was not mapped.

QTL Analysis

QTL analysis for FHB resistance in this population identified a total of eleven different regions significantly associated with the trait. These QTL were located on seven different chromosomes (1A, 1B, 2B, 3B, 5A, 5B, and 7B), with one QTL each on chromosome 1A, 2B and 3B, while two QTL each on chromosomes 1B, 5A, 5B and 7B. The majority of the FHB effective regions (8 out of 11) mapped to a B-genome chromosome, while only three regions mapped to A-genome chromosomes. These 17 QTL identified in 11 different regions include five regions significantly associated with Type II resistance in field trails, four with Type II resistance in greenhouse experiments, four with Type III (FDK) and four with Type V resistance (two with DON and two with 15ADON). The QTL located on chromosome 2B (*Qfhb.ndsu-2B*) was associated with resistance to type II, III and V FHB resistance. Another QTL located on chromosome 3B (*Qfhb.ndsu-3B*) was associated with Type II and Type V resistance. The remaining nine QTL were significantly associated with only one of the resistance types. The phenotypic variation controlled by these QTL ranged from 4.81% (*Qfhb.ndsu-7B.1*) to 23.74% (*Qfhb.ndsu-5A.1*). The positive alleles for increased resistance at six loci (including the QTL on chromosome 2B and 3B) were contributed by the moderately resistant Tunisian parent (Tun108), while positive alleles for increased resistance at five loci were contributed by the susceptible parent Ben (Table 3).

Discussion

For several decades, exploitation of new sources of resistance to FHB in bread wheat has been the most effective approach to breeding for FHB resistance. Despite well-known resources of FHB resistance in hexaploid wheat, sources with the same amount of resistance have not been found in tetraploid wheat. Incorporation of resistance sources from wild tetraploid and common wheat to durum were also encountered with limited achievement (Garvin et al. 2009; Kumar et al. 2007). The North Dakota State University (NDSU) durum wheat breeding program screened approximately 7,000 durum wheat accessions collected from all around the world without identifying potential resistance sources (Elias et al. 2005). In further investigation, NDSU durum wheat breeding program screened 1,500 accessions from the International Center for Agricultural Research in the Dry Areas (ICARDA), which identified five Tunisian lines (Tunisian 7, 18, 34, 36 and 108) with moderate levels of Type II resistance (Elias et al. 2005; Huhn et al. 2012). The five Tunisian lines were used for introgression of FHB resistance into locally adapted cultivars. Huhn et al. (2012) reported that the Tunisian 7, 18, 34, 36 and 108 could possess novel QTL for FBH resistance. Ghavami et al. (2011) revealed that the genetic backgrounds of Tunisian lines are distinctly different from adapted cultivars. Further, some derived lines from these sources could rival the best resistant hexaploid wheat (e.g., Sumai 3) lines with FHB severity of 10 to 25%, possibly due to transgressive segregation. The same phenomena was observed in the current investigation where approximately 8% of lines in the field and 25-30% lines in the greenhouse showed lower levels of disease incidence and severity than the resistant parent. Transgressive segregation for FHB resistance has been observed in durum wheat lines carrying QTL from both parents of the cross (Somers et al. 2006). Ghavami et al. (2011) also observed the same phenomenon in a Tun34/Lebsock//Lebsock population, and they reported different

QTL for FHB severity from both parents and noted the possibility of having a 2A suppressor QTL (gene) in modern durum cultivars. The suppressor factor(s) may mask the resistant QTL and may be lacking in some lines showing transgressive segregation in the positive direction.

In two field screenings, most of the lines with low incidence showed low level of DON accumulation and FDK. Hence, selection for one type of resistance would indirectly enable selection for other types of resistance (Lemmens et al. 2005). Also, a few lines were identified with medium to high level of resistance, low FDK and DON accumulation. These lines provided resistance comparable with that shown in resistant hexaploid genotypes (Sumai 3 and Wangshubai). The significant positive correlations among incidence and FDK as well as FDK and DON content observed in this study also were confirmed in an earlier report (Malla 2005). Positive high correlation ($r > 0.7$) was observed between DON, 3ADON and 15A DON (Table 3.2). This suggests that selection for one trait may indirectly lead to gains in the other traits. The range for DON was quite wide (2.4 to114.4 ppm) compared to 15ADON, 3ADON and Niv (0.2-3.2, 0.2-1.5 and 0.3-1.9 ppm, respectively. The genetic variation and heritability (h^2=44%) for DON also was comparable to 15ADON (47%) and Niv (59%). Therefore, measuring DON is a good indicator of pathogen toxicity for the isolates used in this study. It was reported that 93% of the pathogen isolates collected in North Dakota before 2008 produce DON and mainly 15A DON (Puri et al. 2010). Thus, having no variation for 3ADON in this population with the isolates used is expected. We also recommend adding the most aggressive new 3ADON isolates to the FHB screening mixtures in the future.

Despite significant genotype by environment interactions, heritability for resistance to FHB severity in field (44%) and greenhouse (23%) were enough to justify their selection in a

breeding program. FDK is a better indicator of FHB damage, because of its high heritability (h^2=88%) and high positive correlation to disease severity in field and DON accumulation (Table 2). The significant positive correlations between FDK with disease severity and DON already have been confirmed by other studies (Bonin and Kolb 2009; Malla et al. 2010; Paul et al. 2005). Lines showing high levels of resistance have been tested in two additional seasons with multiple replications, and show consistent FHB resistance (unpublished results). We believe field FHB screening should be based on a mixture of Type I and Type II evaluations. Since both disease penetration (Type I) and disease spread through spike (Type II) are affected by environmental conditions, combination of the two indices and selection for both could decrease the environmental variance and increase the chances of selecting for more resistant lines. FHB field screening provides a better opportunity to select for FHB resistance, but reduces the heritability and the genetic gain in each round of selection.

Several QTL for different FHB-related traits were identified on chromosomes 1A, 1B, 2B, 3B, 5A, 5B and 7B. But as the genotype by environment effect was significant for all QTL, most QTL were inconsistent over different years except for the QTL on chromosome 2B and 3B (Table 3.3). Some of the QTL found in this study were identified previously on different chromosomes of durum wheat (Ruan et al. 2012). Interestingly, the majority of the QTL regions identified here were on B-genome chromosomes.

The DON accumulation QTL identified on chromosome 1A by this study, and two Type II FHB resistance QTL reported by Semagn et al. (2006) and Jiang *et al.* (2007a, b) seem to be in similar region. The two microsatellite markers linked to the two Type II QTL (*barc213* and *barc148*, respectively) were tested on our experimental population but were monomorphic;

however, DArT markers closely linked to DON QTL in this study (*wPt0767*) were mapped with above mentioned microsatellite markers in a close linkage group by Marone et al. (2012). Taken together these data suggest the QTL for DON accumulation and Type II resistance on 1A maybe the same or represent tightly linked loci.

A QTL was identified on the long arm of Chromosome 1B using combined field 2010 and 2011 data. The QTL was not observed in either of the individual field year environments, despite the homogeneity of the 2010 and 2011 field data. The QTL was derived from the recurrent parent 'Ben', with r^2=16.3. The QTL was located to the middle of the long arm of Chromosome 1B (Akbari et al. 2006, Marone et al. 2011). Several other researchers also reported FHB resistance factors on chromosome 1B, at varied locations (Shen et al. 2003, Semagn et al. 2007, Li et al. 2012). The relationship of the QTL we identified to those factors cannot be established, due to their indeterminate placement.

The novel QTL *Qfhb.ndsu-2B* with multiple components of resistance showed significant effect in field and greenhouse screening, with r^2 =6%. *Qfhb.ndsu-2BL* spans a 19.2 cM marker interval. The marker (*gwm71*) linked to this interval has been mapped to the short arm of chromosome 2B in various mapping studies (Roder et al. 1998, Marone et al. 2012). To date, a number of FHB QTL have been reported on chromosome 2B (Buerstmayr et al. 2009); some of which were localized to the short arm of chromosome 2B (Somers et al. 2006, Gervais et al. 2003). These QTL appear located on a different region of chromosome 2B than is presented here.

The QTL found on chromosome 3B in this investigation is not detected as the same location as *Qfhs.ndsu-3BS* from *T. aestivum* (Waldron et al. 1999) or *T.turgidum* var. durum

(Tun34) reported recently (Ghavami et al. 2011). The chromosome 3B QTL identified for Type I, II and V resistance is apparently different from *Fhb1*-QTL reported by Waldron et al. (1999) and Anderson *et al.* (2001). The interval for this QTL (*Xbarc229* and *XwPt9766*) located on long arm of chromosome 3B (Xu et al. 2008; Marone et al. 2012). To date there are at least three reports for 3BL-FHB Type II resistance QTL, however the associated microsatellite loci with these three QTL (*Xgwm247*, *Xgwm131b* and *Xgwm285*) do not correspond to the same region of the 3BL-QTL identified in this investigation. Although there are numerous reports that confirm the important role of the short arm of 3B to various types of *Fusarium* resistance, only Type II resistance has been reported for the long arm of this chromosome (Bourdoncle et al. 2003; Paillard et al. 2004; Liu et al. 2007). Thus, the FHB-3BL QTL reported in this investigation may be a novel resistance factor, with effect on multiple indices of FHB resistance.

A QTL with large effect on severity and a QTL with small effect on FDK adjacent to marker locus *Xbarc141* on chromosome 5A in this study were identified. These QTL are apparently in the same region as the QTL with linked loci *Xgwm293* and *Xgwm156* reported by Buerstmayr et al. (2003a,b). Marone et al. (2012) mapped these two later markers in a 37.9 cM region containing *Xbarc141*. Yang et al. (2005) also reported a QTL for incidence with minor effect at the same chromosomal region including *Xgwm293*. Chen et al. (2006) validated a major QTL at the same chromosomal region between *Xbarc117* and *Xbarc187*. There are numerous reports for a QTL on chromosome 5A with minor effects on disease incidence and severity associated with different markers (Jiang et al. 2007a,b; Lin et al. 2006; Steiner et al. 2004; Gervais et al. 2003; Shen et al. 2003; Paillard et al. 2004). Interestingly, the location of the QTL with large effect on chromosome 5A identified for severity and FDK is proximal to QTL regions detected in *T. aestivum* and *T. macha* (Buerstmayr et al. 2002; Buerstmayr et al. 2011).

Buerstmayer et al. (2003a, b) proposed that the *Qfhs.ifa-5A* contributes to both Type I and to a lesser extent, Type II resistance. In contrast, we have observed contribution (r^2=19%) toward severity measured in the greenhouse screening (Type II), but we were unable to identify any QTL for incidence measured in the field screening. The low heritability of Type II resistance in the field screening (0.23) could be explained by the masking effect of QTL contributing to disease incidence. Another explanation for diminished effect could be that the genetic background of material used in the present study differs from that used by Buerstmayer et al. (2003a, b). Identifying a QTL for FDK in almost the same area and existence of a significant correlation (0.51) between FDK and incidence suggests this QTL may have contribution toward incidence as well.

Comparing the flanking DArT markers of the two QTL regions for FHB Type II resistance mapped on chromosome 7B in this study with the cumulative maps provided by Marone et al. (2012) and Mantovani et al. (2008) indicated that, they are located on proximal and distal regions of 7BS. The resistance QTL located on distal part of 7BS, was derived from the recurrent parent 'Ben'. This FHB QTL has been identified in moderately resistant cultivars 'Dream' (Schmolke et al. 2005; Haberle et al. 2007) and 'Cansas' (Klahr et al. 2007). In all studies, this FHB resistance QTL was linked to Xgwm46; however, the marker for this locus was not polymorphic in the population studied here. The type I FHB resistance QTL located on the proximal region of 7BS has not been reported in any other investigation. Further analysis indicates that the type II FHB resistance QTL identified by Ghavami et al. (2011) in Tunisian background and Type I FHB resistance QTL identified by Gilsinger et al. (2005) derived from cultivar 'Goldfield' are both located on long arm of 7B chromosome.

Conclusions

The ultimate goal of a breeding program for FHB resistance is to reduce the disease symptom and mycotoxin accumulation in the seeds. In this study, we could detect promising resistance line with low level of infection and mycotoxin contamination. The novel *Qfhb.ndsu-2B* durum wheat FHB QTL found in this study shows utility for conferring resistance to multiple FHB resistance components, including severity (measured in the greenhouse), DON and FDK (measured in the field). There also was a QTL identified for disease severity in field in close proximity to *Qfhb.ndsu-2B*. Therefore, using marker assisted selection for introgression of this chromosome segment may be beneficial for developing FHB resistance in durum wheat populations. The 5A QTL found in this study locates very close to *Qfhs.ifa-5A* in spring wheat, and also locates close to *Qfhb.usw-5A*, found recently in durum wheat indicate that, this FHB resistance QTL is from a common ancestor and predates the wheat and durum speciation event(s).

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Table 3.1. Disease severity and toxin levels in Tunisian108/Ben//Ben BC_1F_7 population across the experiments

	Disease value (%)					Toxin value (ppm)			
	F2010[a]	F2011[a]	GH2010[b]	GH2011[b]	FDK[c]	DON[d]	3ADON[e]	15ADON[f]	Niv[g]
Population Mean	71	69	40	69	66.71	34.12	0.43	0.80	0.36
Std Dev[h]	11	21	22	20	21.29	19.75	0.23	0.46	0.22
Minimum	30	15	07	25	10	2.40	0.20	0.2	0.30
Maximum	93	95	100	95	100	114.40	1.50	3.20	1.90
Ben	80	82	81	75	85	17.5	0.40	0.40	0.40
Tunisian 108	50	54	46	50	60	25.0	0.40	0.40	0.5
LSD1 (0.05)	21	21	21	21	14.76	25.35	0.37	0.56	0.22
LSD2 (0.05)	29	29	29	29	20.60	35.70	0.53	0.79	0.30

[a]. Field experiment
[b]. Greenhouse experiment
[c]. Fusarium damaged kernel
[d]. Deoxynivalenol
[e]. 3-acetate deoxynivalenol
[f]. 15-acetate deoxynivalenol
[g]. Nivalenol
[h]. Standard deviation
LSD1 compare BIL population mean to parents; LSD2 compare two BILs

Table 3.2. Correlation coefficient between different FHB components; Field experiment, 2011

Traits	Dis.Inc[a]	FDK[b]	DON[c]	3ADON[d]	15ADON[e]	Niv[f]
Dis.Sev[a]		0.51	0.12	0.07	0.13	0.17
		<.0001	0.1082	0.3646	0.0914	0.0289
FDK[b]			0.30	0.14	0.16	0.04
			<.0001	0.0649	0.0425	0.5626
DON[c]				0.77	0.83	0.29
				<.0001	<.0001	<.0.0001
3ADON[d]					0.71	0.27
					<.0001	0.0004
15ADON[e]						0.20
						0.0089
Niv[f]						

[a]. Disease severity
[b]. Fusarium damaged kernel
[c]. Deoxynivalenol
[d]. 3-acetate deoxynivalenol
[e] 15-acetate deoxynivalenol
[f]. Nivalenol

Table 3.3. Putative QTL for FHB severity, FDK and toxic components detected in Tunasian108/Ben//Ben BC_1F_7 BIL mapping population

Trait	Env.	QTL	Marker interval	Positions (cM)	LOD	Additive effect	R^2 (%)
FHB Severity	GH 2010	*Qfhb.ndsu-2B*	*Xwmc96-Xbarc353*	74.11	2.893	0.692	6.1
FHB Severity	GH 2010	*Qfhb.ndsu-3B*	*Xwpt0384-Xbarc229*	167.91	4.44	0.8003	10.8
FHB Severity	GH2010	*Qfhb.ndsu-5A*	*Xbarc2187-Xbarc141*	86.21	6.881	1.1718	23.7
FHB Severity	GH 2011	*Qfhb.ndsu-5B*	*Xwpt5928-Xwpt5604*	127.91	2.00	-0.497	5.1
FHB Severity	Avg across GH	*Qfhb.ndsu-2B*	*Xwmc96-Xbarc353*	76.11	2.523	0.5552	7.0
FHB Severity	Avg across GH	*Qfhb.ndsu-3B*	*Xwpt0384-Xbarc229*	163.91	3.217	0.5389	7.8
FHB Severity	Avg across GH	*Qfhb.ndsu-5A*	*Xbarc2187-Xbarc141*	88.21	5.539	0.9963	19.1
FHB Severity	Field 2011	*Qfhb.ndsu-2B*	*Xgwm71-Xbarc297*	61.81	2.127	0.6187	5.8
FHB Severity	Field 2011	*Qfhb.ndsu-3B*	*Xwpt0384-Xbarc229*	142.61	3.518	0.7141	8.5
FHB Severity	Field 2011	*Qfhb.ndsu-7B*	*Xwpt7975-Xwpt5846*	3.91	2.154	-0.4957	4.8
FHB Severity	Field 2011	*Qfhb.ndsu-7B*	*Xgpw1054-Xwpt0884*	47.91	4.342	0.7856	9.7
FHB Severity	Avg across field	*Qfhb.ndsu-1B*	*Xwpt1818-Xwpt5061*	156.81	3.06	-0.746	16.3
FHB Severity	Avg across field	*Qfhb.ndsu-7B*	*Xwpt7975-Xwpt5846*	3.91	4.182	-0.4348	9.5
DON	Field 2011	*Qfhb.ndsu-1A*	*Xwpt7784-Xwpt6853*	146.81	2.063	-4.5926	4.8
DON	Field 2011	*Qfhb.ndsu-2B*	*Xbarc297-Xwmc96*	70.41	2.887	7.7066	10.0
15ADON	Field 2011	*Qfhb.ndsu-2B*	*Xgwm71-Xbarc297*	70.41	2.155	0.1393	6.1
15ADON	Field 2011	*Qfhb.ndsu-3B*	*Xwpt0384-Xbarc229*	161.91	2.337	0.1285	5.4
FDK	Field 2011	*Qfhb.ndsu-1B*	*Xgwm264-Xwpt3451*	81.31	5.124	7.8064	11.7
FDK	Field 2011	*Qfhb.ndsu-2B*	*Xwmc96-Xbarc353*	78.11	2.273	6.4989	6.1
FDK	Field 2011	*Qfhb.ndsu-5A*	*Xbarc141-Xwpt4248*	101.01	3.305	-6.6074	7.1
FDK	Field 2011	*Qfhb.ndsu-5B*	*Xwpt6902-Xwpt5514*	22.01	2.084	6.5267	7.8

[a]. Deoxynivalenol

[b]. 15-acetate deoxynivalenol

[c]. Fusaruim Damaged Kernel

[d]. Environments

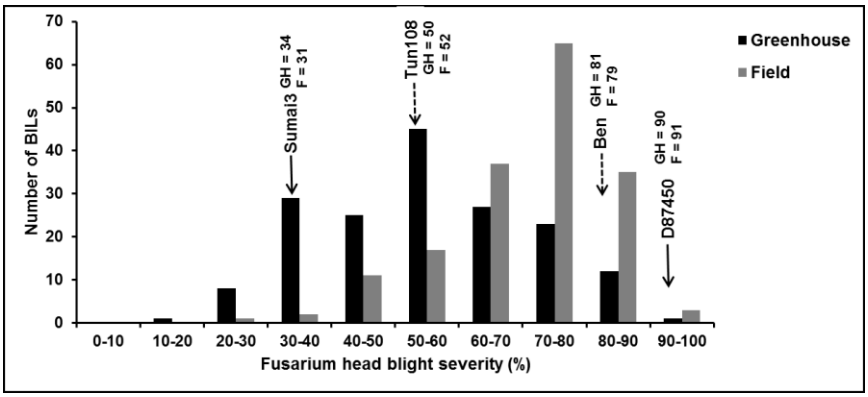

Figure 3.1. Frequency distribution of FHB severity and incidence means among BIL genotypes in Tun108/Ben//Ben population in two greenhouse and two field experiments during 2010 to 2011. The average of FHB infection for the parents and the resistant (Sumai3) and susceptible (D87450) checks are indicated by arrows

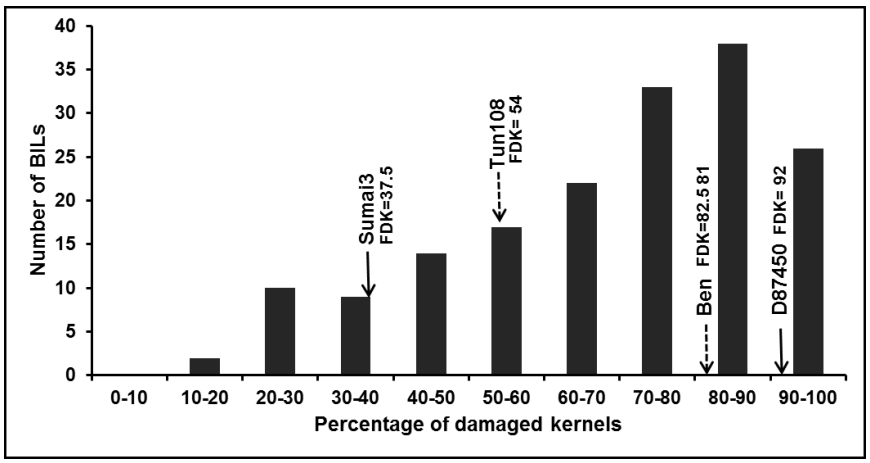

Figure 3.2. Frequency of Fusarium damaged kernels (FDK) means among genotypes in Tun108/Ben//Ben population in 2011 field experiments. The average of FDK for the parents, the resistant (Sumai3) and susceptible (D87450) checks are indicated by arrows

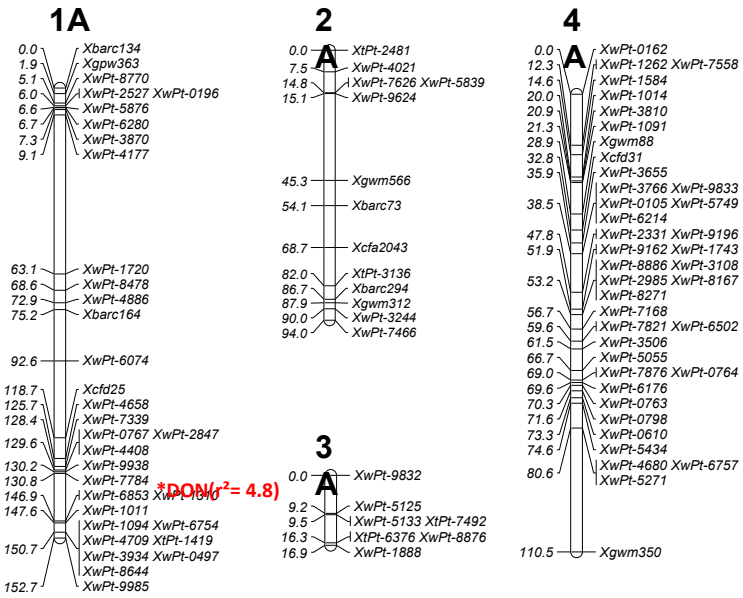

Figure 3.3. Genetic linkage mape of 329 mapped markes and putative QTL for Type II, III and V identified in BC_1F_7 BIL population derived from cross between Tunisian108/Ben//Ben. (F=Field; GH=greenhouse)

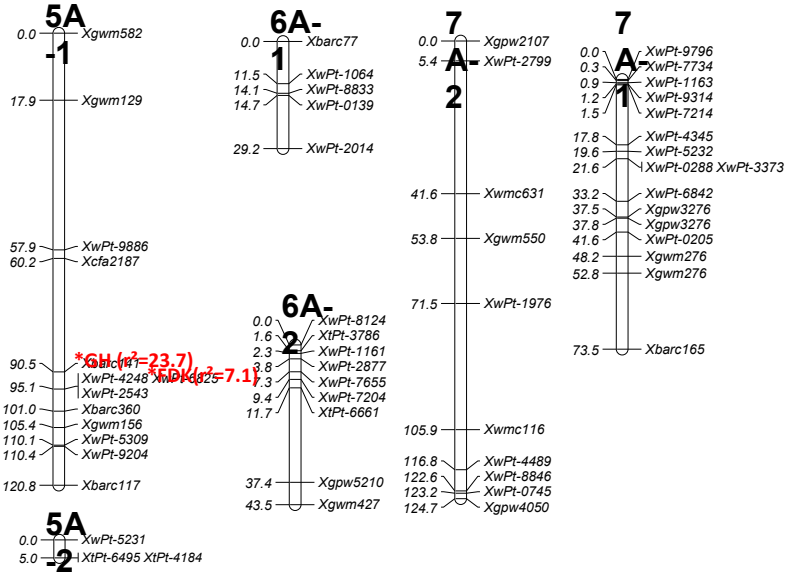

Figure 3.3. Genetic linkage mape of 329 mapped markes and putative QTL for Type II, III and V identified in BC_1F_7 BIL population derived from cross between Tunisian108/Ben//Ben (continued). (F=Field; GH=greenhouse)

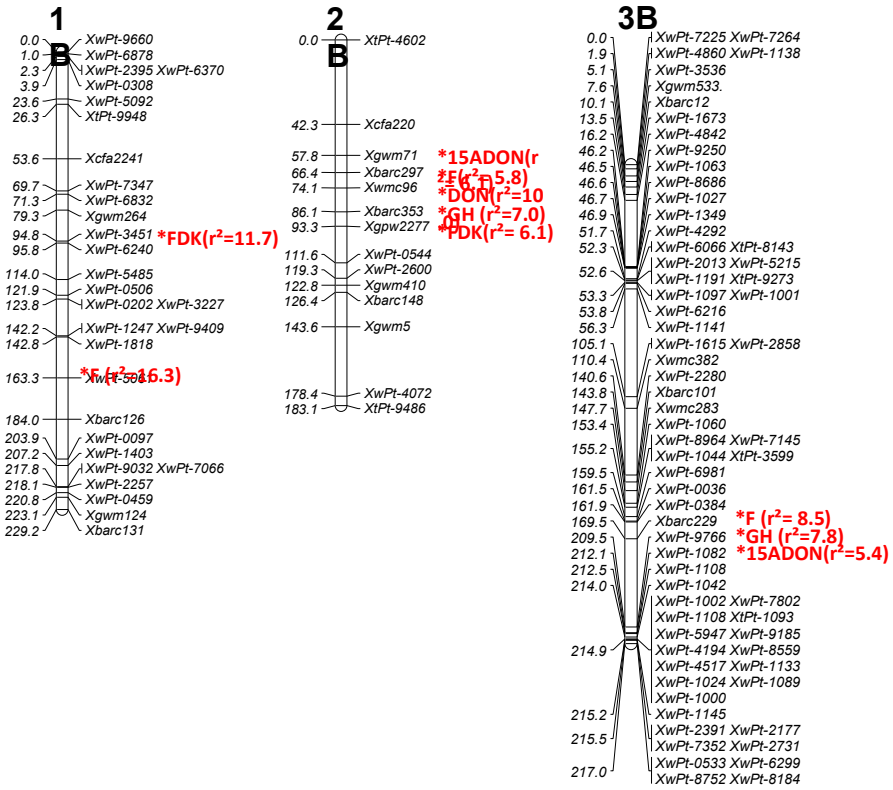

Figure 3.3. Genetic linkage mape of 329 mapped markes and putative QTL for Type II, III and V identified in BC_1F_7 BIL population derived from cross between Tunisian108/Ben//Ben (continued). (F=Field; GH=greenhouse)

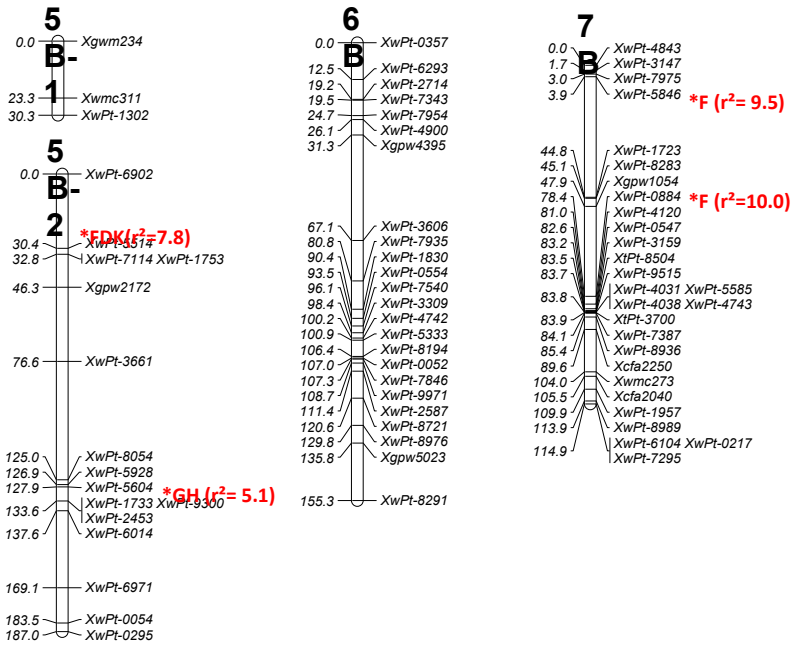

Figure 3.3. Genetic linkage mape of 329 mapped markes and putative QTL for Type II, III and V identified in BC_1F_7 BIL population derived from cross between Tunisian108/Ben//Ben (continued). (F=Field; GH=greenhouse)

CHAPTER IV. IDENTIFICATION OF QTL FOR FUSARIUM HEAD BLIGH RESISTANCE IN A TUNISIAN DURUM WHEAT POPULATION

Abstract

Fusarium head blight (FHB) of wheat, caused primarily by *Fusarium graminearum* Schwabe [telomorph *Gibberella zea* (Schwein)], is a fungal disease that causes tremendous economic losses due to decrement in grain yield and quality. Direct and secondary economic losses, food safety concerns, and lack of effective resistance sources in durum wheat (*Triticum turgidum* L. var. Durum Desf.) continue to motivate FHB resistance research. A backcross inbred lines (BIL) population (BC_1F_7) of 172 individuals derived from a cross between an exotic resistant durum genotype 'Tunisian 108' and an adapted durum wheat cultivar 'Lebsock' was used to identify quantitative trait loci (QTL) for FHB resistance. FHB resistance was evaluated in two green houses and two field experiments. Analysis of variance showed significant genotypic effect on FHB severity and incidence. Transgressive segregation for FHB resistance was observed. A framework linkage map of 331 molecular markers was developed. The 331 markers represent 243 unique loci and cover a total distance of 1,748 cM between all marker loci. Composite interval mapping revealed 15 different QTL on seven different chromosomes (1A, 1B, 3A, 3B, 4A, 5A, and 6B). At least one novel QTL (*Qfhb.ndsu-4A*) on chromosome 4A was identified in this investigation which confers resistance to FHB severity. Our investigation confirms the breeding value of Tunisian lines for incorporation of resistance factors into adapted cultivars.

Introduction

Fusarium head blight is a devastating fungal disease of small grain cereals caused by different Fusarium genera in temperate regions worldwide. Despite a wide range of Fusarium species and strains, there is no report of a biological race with specific host-pathogen interaction. *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schw.) Petch] is the common causal pathogen for both bread wheat (*T. aestivum* L., 2n = 6x = 42, AABBDD) and durum wheat (*T. turgidum* L. var. durum, 2n = 4x = 28, AABB) worldwide (Chen et al. 2007).

FHB leads to severe loss in grain yield and quality owing to contamination with toxic fungal metabolites such as deoxynivalenol or nivalenol (Gilbert and Tekauz 2000; Stack, 2003). *Fusarium*-contaminated grain is the main source of mycotoxins in the food chain (Foroud and Edues 2009), making the infected grain unsuitable for consumption as food or feed (Bai et al. 2001; Gilbert and Tekauz 2000; Bechtel et al. 1985). FHB epidemics caused an estimated $6.2 billion economic loss in the US Northern Great Plains alone from 1993-2001 (Nganje et al. 2004).

Five types of physiological resistance to FHB are the commonly presented in the literature; namely, resistance to (1) disease penetration or initial infection (Type I), (2) floral spread or fungal spread within the spike (Type II), (3) Fusarium Damaged Kernel (FDK; Type III), (4) yield reduction (Type IV) and (5) mycotoxin accumulation (Type V) (Schroeder and Christensen 1963; Mesterhazy, 1995). Owing to positive correlation between different resistance components, selection for one type of resistance may facilitate enhancing other types of FHB resistance in breeding programs (Lemmens et al. 2005).

Since cultural management practice and chemical controls are largely impractical or ineffective (Bai and Shaner 1994), exploration for new genetic resistance sources and their incorporation into adapted cultivars is the most efficacious strategy for reducing FHB damages (Buerstmayr et al. 2012). Whereas resistance to FHB is quantitatively inherited, phenotypic evaluation is complicated by significant genotype by environment interactions (Miedaneret al. 2001*)*.

The genetics of FHB resistance has been extensively studied in hexaploid germplasm and derivatives using quantitative trait loci (QTL) mapping (Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2009; Li et al. 2011; Liu et al. 2012). As a result of these studies, numerous QTL located on all 21 chromosomes of wheat have been identified for different resistance types in bread wheat (for review, see Buerstmayr et al. 2009; Cativelli et al. 2013), although the resistance sources are mainly limited to cultivars such as 'Sumai3', 'Wangshuibai' (Mardi et al. 2005), and in some limited extent, 'Frontana', from Brazil (Mardi et al. 2006). In comparison to hexaploid wheat, genetic studies of FHB resistance in tetraploid wheat are few, as are resistance resources (Ghavami et al. 2011; Buerstmayr et al. 2012; Huhn et al. 2012). Limited resistance in tetraploid germplasm may be due to active susceptibility factors or suppressor genes (Ban and Watanabe 2001; Kishii et al. 2005). This hypothesis was reinforced when Garvin et al. (2009) reported the first QTL with FHB susceptibility effect and presumed epistatic influence on resistance QTL located on chromosome 2A of *T. dicoccoides* line Israel A. A like example of negative epistatic resistance gene effect was reported by Arraiano et al. (2007), whereby a suppressor gene located on wheat chromosome 5B in wheat increased susceptibility to *Mycosphaerella graminicola*.

Attempts to incorporate resistance from common wheat into durum wheat have had limited success, likely due to different ploidy levels and the existence of suppressor genes (Gilbert et al. 2000; Oliver et al. 2007; Ghavami et al. 2011), lack of D genome (Fakhfakh et al. 2011) and a narrower genetic base compared to bread wheat (Oliver et al. 2008). Lack of resistance sources has shifted investigations toward wild relatives of durum wheat. Prospective resistance sources have included wild emmer wheat, *T. dicoccoides* (Buerstmayr et al. 2003; Miller et al. 1998; Oliver et al. 2007), cultivated emmer wheat, *T. dicoccum*, and Persian durum wheat, *T. carthlicum* (Oliver et al. 2008); however, only a few moderately FHB-resistant accessions have been identified. Otto et al. (2002) reported the first FHB resistance QTL on 3A chromosome (*Qfhs.ndsu-3AS*), derived from *T. dicoccoides* accession Israel A and linked to microsatellite locus *Xgwm2* (Chen et al. 2007). This QTL was further confirmed by Gladysz et al. (2007) in a BC_1-RIL population derived from cross between *T. durum* cultivar 'Helidur' (recurrent parent) and *T. dicoccoides* (donor parent), where it showed a larger effect. This study also identified two FHB resistance QTL from the susceptible parent (Helidur) on chromosomes 2B and 4A. Kumar et al. (2007) identified a QTL on 7A (*Qfhs.fcu-7AL*) from *T. dicoccoides* accession PI478742. Somers et al. (2006) identified two type II resistance QTL on the long arm of chromosome 2B and the short arm of chromosome 6B using a double haploid population derived from a cross between *T. durum* cultivar 'Strongfield' and *T. carthlicum* cultivar 'Blackbird'. The low contribution for FHB resistance from identified QTL in durum wheat background is a major obstacle to breeding FHB resistance. The QTL with the largest effect was detected for Type II resistance in durum wheat on chromosome 4B, and explained only 18% of the phenotypic variance (Buerstmayr et al. 2012).

For several decades, developing moderately resistant cultivars with lower disease symptom and mycotoxin accumulation have been a top priority in wheat breeding programs. Recent efforts to deploy new FHB resistance sources and enrich the locally adapted wheat germplasm have intensified (Elias, personal communication). In an effort to identify new sources of FHB resistance in durum wheat, approximately7,500 durum wheat accessions from across the world were screened for at North Dakota State University (NDSU), and five Tunisian lines (Tun7, Tun18, Tun34, Tun36 and Tun108) with moderate level of Type II FHB resistance were identified (Elias et al. 2005; Huhn et al. 2012).

Ghavami et al. (2011) used backcross inbred lines (BIL) developed by crossing four Tunisian tetraploid sources of resistance (Tun7, Tun18, Tun34, Tun36) with four different high yielding locally adopted durum cultivars (Ben,' 'Maier,' 'Lebsock,' and 'Mountrail) to identify several markers associated with FHB resistance. This study also showed that Tunisian-derived lines can provide FHB resistance comparable to best resistant hexaploid wheat (Sumai-3) sources.

The identification of new sources of resistance is always desirable in any breeding program to achieve higher FHB resistance levels through gene pyramiding. Similarly, it also is important to understand the genetics of resistance in order to utilize these sources more effectively using modern biotechnological tools like molecular markers. In this direction, we used to Tunisian tetraploid lines identified as FHB resistant sources (Elias et al. 2005; Huhn et al. 2012) to develop several mapping population using susceptible or moderately resistant locally adopted cultivars to understand the genetics of FHB resistance in these lines.

In the present study, a BIL population was developed from a cross of 'Tunisian 108' and 'Lebsock'. Tunisian 108 is an exotic Tunisian durum wheat genotype with moderate resistance to FHB (Huhn et al. 2012), while Lebsock is a high-yielding commercial cultivar with nominal FHB susceptible (Elias et al. 2001). The objectives of the current study were (i.) to develop a molecular linkage map, (ii.) to identify genomic regions significantly associated with FHB resistance, (iii.) to determine molecular markers linked to FHB resistance (iv.) to introduce promising new line(s) with FHB resistance that can be used as sources for further durum wheat breeding programs.

Material and Methods

Plant Materials

A BIL durum wheat population was developed from a cross between 'Lebsock' and 'Tunisian 108' (Elias et al. 2005; Huhn et al. 2012). 'Lebsock' is a high-yielding commercial cultivar with nominal FHB resistance, and was used as the recurrent parent. Tunisian 108 is an exotic Tunisian durum wheat genotype with moderate resistance to FHB (Ghavami et al. 2011; Huhn et al. 2012) and was used as the donor parent. The F_1 plants were backcrossed to 'Lebsock' and each line was then selfed seven generations. Individual lines were advanced by single-seed descent in each generation. A total of 172 BC_1F_7 lines along with parental genotypes (Tunisian 108 and Lebsock) and fourteen checks were evaluated for FHB resistance. The checks included four resistant hexaploid genotypes: 'Sumai3', 'Wangshubai', 'Glenn' (Mergoum et al. 2006), and ND2710; nine moderate resistant tetraploid genotypes: 'Divide' (Elias and Manthey 2007), 'Maier' (Elias and Miller 2000), 'Ben' (Elias and Miller 1998), Tunisian 7, Tunisian 18,

Tunisian 34, Tunisian 36 (Huhn et al. 2012), D91103, and D95097; and one susceptible genotype, D87450.

Phenotypic Evaluation

Phenotypic evaluation was performed in two field trials and two greenhouse experiments in 2010 and 2011. Field trials were conducted in Prosper, North Dakota, genotypes were evaluated for resistance to initial infection or disease penetration and spread (considered Type I and II resistance) in 2010 and 2011. Deoxynivalenol (DON), 3-acetate deoxynivalenol (3ADON), 15-acetate deoxynivalenol (15ADON), nivalenol accumulation and *Fusarium*-damaged kernel (FDK) were assessed only in 2011. Twelve seeds were planted as hill plots in a field mist-irrigated FHB nursery using a randomized complete block design with two replicates. Field inoculation was conducted using the corn kernel inoculation method (Stack 1989). A mixture of three different isolates of *Fusarium graminearum* (R010, R1267, and R1322) was used to maximize the likelihood of infection. Inoculum spreading was initiated at anthesis, and was repeated three times hence at 10 day intervals. Phenotypic evaluation for FHB resistance was performed based on percentage (0 to 100%) of visual symptom on spikelets per whole spikes or percentage of infected spikelets (PIS) in each replicate, 28 days after anthesis (Gosman et al. 2005).

Greenhouse screening was conducted on the population, parental lines and checks for disease spread (Type II resistance) using randomized complete block design (RCBD) with two replicates in the USDA-ARS greenhouse, Fargo, North Dakota, during 2010 and 2011. Briefly, three seeds from each line were planted for each replicate in clay pots and arranged in RCBD with two replicates. Four to six spikes in each replicate were inoculated with a 10 µl droplet of

the isolate mixture in suspension (50,000 conidia/ml). Inoculum was injected directly into the second or third spikelet near the bottom of the spike as described by Stack (1989). Inoculated spikes were covered by a misted plastic bag for 72 hours and greenhouse temperature was adjusted to 22 to 27˚C. Disease spread was scored according to visual symptoms based on number of infected spikelets per spike 21 days after inoculation (Stack and McMullen 1985).

Statistical Analysis

Analysis of variance for single and combined years for disease incidence (Type I) and severity (Type II were conducted using Proc GLM of SAS software version 9.2 (SAS Institute, Cary, NC). While only single analysis of variance were conducted for FDK, DON, 3ADON, 15ADON and nivalenol. Using Proc Corr command (SAS 9.2), Pearson's correlation between disease incidence, severity, FDK, DON, 3ADON, 15ADON and nivalenol contents were calculated. Broad-sense heritability (h^2) was estimated using following formula:

$$h^2 = \sigma^2_g/(\sigma^2_g+\sigma^2_{gy}/y+\sigma^2_e/ry)$$

Where:

σ^2g = Genotypic variance

σ^2gy = Genotype × year interaction variance

σ^2e = Residual error variance

r = Replications

y = Years

Genotypic Analysis

DNA was extracted from the BIL population, parental lines, and checks following the method as described by Triticarte Pty. Ltd. (http://www.triticarte.com.au/content/DNApreparation. html). A total of 172 BILs, two parental lines and 14 checks were genotyped using Diversity Array technology (DArT) assay by Triticarte Pty. Ltd. (Canberra, Australia; http://www.triticarte.com.au), as described by Akbari et al. (2006). An array of 2,300 DArT markers distributed across the entire durum wheat genome was used to screen all lines.

Additionally, 305 microsatellite markers were tested for polymorphism between parental lines using standard polymerase chain reaction (PCR). These markers consisted of Gatersleben Wheat Microsatellites (GWM; Roder et al. 1995, 1998; Ganal and Roder 2007), Wheat Microsatellite Consortium (WMC; Gupta et al. 2002), DuPont Company (DuPw; Eujayl et al., 2002), INRA Clermont-Ferrand (CFA and CFD; Sourdille et al. 2004; Guyomarc'h et al. 2002), and Beltsville Agriculture Research Center (BARC; Song et al. 2005). PCR conditions were as originally described (Röder et al. 1998; Somers et al. 2004; Song et al. 2005). PCR products were separated on an 8% non-denaturing polyacrylamide gel and stained with ethidium bromide for scoring. Markers identified as polymorphic between parental lines were screened on the BIL population.

Genetic Map Construction and QTL Analysis

Linkage maps were constructed by MAPMAKER, v2.0 (Lander et al., 1987) using genotypic data on polymorphic DArT and SSR markers. A set of DArT markers were selected based on available genetic maps (Mantovani et al. 2008; Peleg et al. 2008; Ghavami et al. 2011;

Huang et al. 2012; Kumar et al. 2013) and used as anchors to assign remaining markers to individual chromosomes. Markers were assigned to individual linkage groups at a minimum LOD score of 3 and maximum distance of 30 cM. Map distances were calculated using Kosambi mapping function (Kosambi 1944). Linkage maps were drawn using MapChart (Voorrips 2002).

QTL analysis was performed using composite interval mapping (CIM) method by QTL Cartographer V2.5 (Wang et al. 2012). In QTL Cartographer, model 6 with forward and backward step-wise regression was used with five markers as cofactors and a 10 cM genome-wide scan window to control genetic background. A minimum LOD score of 2.0 was used for determining the presence of a putative QTL. Confidence intervals (CI) were obtained using positions ±1 LOD away from the QTL peak, and QTL with overlapping CIs were considered as same. Permutation test using 500 iterations also was performed to determine threshold LOD scores for each trait in each environment with 5% error rate. QTL above threshold LOD scores were considered definitive QTL.

Results

Phenotypic Analysis

FHB reactions were significantly different for check plants and the entire population in greenhouse and field screening across the experiment. There were significant differences among BILs genotypes ($p<0.0001$) for Type II FHB resistance across the experiments (Table 4.1). Significant genotype by environment interaction (G×E; $p<0.0001$) was observed in the field trials. Correlations were computed for years and disease evaluations, tested for homogeneity, and pooled when deemed appropriate. Significant correlation was observed for resistance

disease severity ($r=0.27$, $p<0.0005$) between the two field trials as well as for the two greenhouse experiments ($r=0.23$, $p<0.025$). However, there was no significant correlation between field and greenhouse disease assessments.

Significant ($p<0.0001$) differences for FDK, DON, 3ADON, 15ADON in the BIL population. However, no significant difference among BILs was observed for nivalenol. Variances of disease severity in field and greenhouse experiments were homogeneous for years, and there was a positive correlation between years. Thus, the data were pooled across years for Pearson's correlation analysis. Analyses of measured FDK, DON, 3ADON, 15ADON and nivalenol was based on the 2011 field experiment. There was positive significant correlations between FDK and mycotoxin components (DON, 3ADON, 15ADON), with the exception of nivalenol (Table 4.2). Although significant correlation was observed between disease severity in field and FDK ($r=0.53$, $p<0.0001$), no significant correlation was observed between disease severity in field and mycotoxin components. Positive significant correlation between DON and 3ADON ($r=0.56$, $p<0.0001$), DON and 15ADON ($r=0.76$, $p<0.0001$), 3ADON and 15ADON ($r=0.64$, $p<0.0001$) was observed, while no significant correlation between DON components and nivalenol was observed. Based on ANOVA, the broad sense heritability estimated 0.37, 0.40, 0.75, 0.75, 0.78 and 0.75, for disease severity in greenhouse and field experiments, FDK, DON, 3ADON and 15ADON respectively.

There was a wide range of variation for disease incidence, disease severity, FDK, and mycotoxin compounds with the exception of nivalenol in the BIL population (Table 4.1). Although the mean value of the population for disease incidence (0.72) and disease severity

(0.47) were skewed toward susceptibility across the experiments, these levels were lower than the mean disease values for the resistance donor parent.

Transgressive segregation for resistance to FHB severity and incidence was clearly observed within the population (Figure 4.1). Approximately 2% of the BILs lines in the field assays and 16% of them in greenhouse screening were consistently better than the resistant parent across years. However, four lines were identified with the lowest present of infected spikelets, FDK and mycotoxine components across experiments.

DArT and SSR Analysis

A total of 29 (out of 303) SSR markers detected polymorphism between parental genotypes 'Tunisian 108' and 'Lebsock'. These 29 SSR markers, along with 344 polymorphic DArT markers, were used to genotype the 172 BC_1F_7 lines. Thirty-six DArT markers showed highly distorted segregation ratios and were excluded from the analysis. The remaining 337 markers were used for genetic map construction. At a minimum of 3 LOD score, a total of 331 markers were mapped to 243 unique loci in 14 linkage groups, belonging to all 14 chromosomes (Fig 3). The number of mapped markers on individual chromosomes varied from three markers on chromosome 4B to as many as 59 markers on chromosome 3B. These 331 mapped markers representing 243 loci covered a total genetic distance of 1,748 cM with an average distance of 7.2 between any two adjacent marker loci. The length of individual chromosomes ranged between 14.4 cM for chromosome 4B to 270.5 cM for chromosome 5A. The total genetic distance of the A genome chromosomes was 1,031.1 cM, and 716.9 cM for the B genome chromosomes.

Genomic Distribution of Mapped Markers

The seven homeologous groups of the tetraploid wheat genome varied in the number of markers, map length, and marker densities. Homeologous group 3 had the largest number of markers, with 74 mapped loci. Homeologous group 6 had the highest marker density, with 3.96 cM average distance between two adjacent marker loci. In contrast, homeologous group 5 had the lowest number of the mapped markers and the lowest marker density (35 loci; 8.02 cM/marker). Total map length was longest in homeologous group 3, at 326.9 cM, whereas group 6 had the shortest map length at 150.6 cM. Marker density also was different for the two genomes, with 160 (43.33%) markers mapped to the A genome and 149 (52.67%) to the B genome. Chromosome 4B was not mapped in this investigation, for two primary reasons: i.) some polymorphic chromosome 4B DArT markers were excluded from the mapping process due to distorted segregation ratios, and ii.) a dearth of polymorphic chromosome 4BSSR markers.

QTL Mapping Analysis

Composite interval mapping in the Tunisian108/Lebsock//Lebsock BIL population revealed a total of 15 QTL for different types of FHB resistance (Table 3). These QTL were mapped to seven different chromosomes (1A, 1B, 3A, 3B, 4A, 5A, and 6B). Six QTL were identified for disease incidence (Type I resistance); four were mapped to B genome chromosomes and two were localized on A genome chromosomes. In contrast, four out of six QTL identified for disease severity were mapped to A genome chromosomes and two were mapped to B genome chromosomes. For DON components, three QTL were identified; two of which were mapped to chromosome 3B, and one to chromosome 3A. Due to low variation for nivalenol accumulation, the two minor QTL identified for this trait were excluded. Of the 15

FHB QTL identified, 10 loci originated from the moderately resistant Tunisian parent, while five loci were from the recurrent parent, Lebsock (Table 3).

Discussion

The mean of disease severity in field (72.5%) was high and was skewed toward susceptibility across the two years field experiments. However, the disease severity mean (47%) was moderately low in the two greenhouse screenings (Table 1). Low disease severity likely is due to type II resistance factors in 'Lebsock', previously reported by Ghavami et al. (2011). The effect of nivalenol accumulation was not significant owing to low trait variation.

Transgressive segregation for FHB resistance has been reported in durum wheat lines carrying resistant QTL from both parents (Somers *et al*., 2006). A high level of transgressive segregation in a Tunisian-derived population (Tunisian34/Lebsock//Lebsock) was reported by Ghavami et al. (2011). In the present investigation, a few lines showed all types of resistance across the experiments, with lower symptom levels in comparison to the moderately resistant parent 'Tunisian 108'. Resistance in some BILs rivaled that shown in Chinese resistant lines 'Sumai3' and 'Wangshuibai'. Considerable differences were observed between the QTL positions identified in this investigation and previously identified QTL in Tunisian-derived populations (Ghavami et al. 2011). Two reasons might cause these discrepancies: i.) wide genetic diversity in Tunisian background for FHB resistance, and ii.) existence of suppressor QTL (gene) in modern durum cultivars, which can mask the resistant QTL and may have been removed in some of the lines showing positive transgressive segregation. This hypothesis (ii.) was presented and discussed in the introduction.

Despite the wide range of variation exhibited for four types of disease resistance measured in this investigation, correlation between disease incidence intensity and DON component accumulation was not significant. QTL analysis did not detect any QTL for FDK, despite wide variation for this trait in the population. FDK assessment showed significant correlation with disease incidence in one comparison, and DON component accumulation in another comparison (Table 2). Thus, FDK could be an efficacious index for FHB phenotypic selection. The significant positive correlations between incidence and FDK as well as FDK and DON content observed in this study were reported earlier (Malla 2005). Hence, selection for one type of resistance indirectly enables selection for other types of resistance (Lemmens et al. 2005).

In this study, the heritability for FHB Type II resistance in field (0.40) and greenhouse (0.37) presents utility for resistance selection in a breeding program. The heritability of FDK was high (0.75) and had a high positive correlation to incidence and DON component accumulation (Table 2.). Positive significant correlations were assessed between FDK and DON and disease incidence, despite significant genotype by environment interaction. The positive significant correlations between FDK with disease severity, incidence and DON were established in earlier studies (Bonin and Kolb 2009; Malla et al. 2010; Paul et al. 2005).

To date, reports of FHB resistance QTL on the short arm of chromosome 1A are very limited. Flanking DArT markers for the Type I FHB QTL (*wPt5876-wPt6280*) and the linked marker to Type II FHB QTL (*wPt4177-wPt3870*) were mapped previously on the short arm of chromosome 1A by Marone et al. (2012). A possible homolog for the QTL we identified might be the Type II and Type V minor resistance QTL reported by Jiang et al. (2007a, b). Semagn et

al. (2007) identified a major QTL for Type II and V FHB resistance QTL mapped on distal part of the long arm of chromosome 1A, which is different than the QTL we identified. Schemolke et al. (2008) identified a minor FHB QTL for Type I resistance on chromosome 1A (arm undesignated) linked to amplified fragment length polymorphism (AFLP) markers. Thus, the Type I FHB QTL flanked by *wPt4886- wPt3698* on the short arm of chromosome 1A can be considered novel.

The marker interval of two identified QTL on chromosome 1B (wPt0506-wPt1818) were mapped previously on the middle of the long arm of this chromosome (Marone et al. 2011; Akbari et al. 2006). There is only one clear report for identifying FHB QTL on the distal end of the long arm of chromosome 1B (Semagn et al. 2007). Shen et al. (2003) identified a Type II FHB resistance QTL on proximal region of the short arm of chromosome 1B (Marone et al. 2011). Li et al. (2012) identified a Type II FHB resistance QTL, flanked by Xbarc207 and Xbarc181on the proximal region of the long arm of chromosome 1B. Thus, owing to the lack of clear reports for FHB QTL detection on proximal region of the long arm of chromosome 1B, the FHB resistance QTL on the long arm of chromosome 1B in this study might be considered a novel resistance factor in tetraploid wheat.

There are numerous reports for FHB resistance QTL on chromosome 3A. While we detected Type II and V resistance QTL on chromosome 3A, the QTL were not consistent across experiments. The marker linked to the Type V resistance QTL (*wPt 2938*) was previously mapped by Akbari et al. (2006) on distal end of the short arm of chromosome 3A. Bourdoncle and Ohm (2003) reported a Type II resistance QTL on the short arm of chromosome 3A linked to *Xgwm5* which is tightly linked to *wPt2938* (Crossa et al., 2007). Otto et al. (2002) and Chen

et al. (2007) both reported Type II resistance QTL originated from *T. dicoccoides* line FA-15-3. Both QTL mapped by Crossa et al. (2007) were linked to *Xgwm2* locus on short arm of chromosome 3A, which is very close to the marker interval identified for Type V FHB resistance QTL in this investigation. Yang et al. (2005) also reported a type I resistance QTL tightly linked to *Xwmc428*, which is very close to the chromosomal region mentioned by Crossa et al. (2007). The marker (wPt1888) mapped closest to one of the Type II resistance QTL in the distal region of the long arm of chromosome 3A was identified previously by others (Akbari et al. 2006; Crossa et al. 2007; Marone et al. 2012). This QTL can be consider similar to the Type II resistance QTL on the long arm of chromosome 3A, linked to *Xwmc264* and *Xgwm155* loci,identified by Paillard et al. (2004).

Flanking markers of three FHB resistance QTL on chromosome 3B have not been reported previously in other mapping studies. Thus, comparing the QTL identified by these markers with previously identified resistance factors is not possible. The flanking markers of 3B FHB resistance QTL (*XwPt0036* and *XwPt384*) likely are located on the distal region of the long arm of chromosome 3B (Crossa et al. 2007; Marone et al. 2012). To date, a few FHB resistance QTL on the long arm of chromosome 3B have been reported in different mapping studies. Bourdoncle and Ohm (2003) identified QTL for resistance to FHB incidence and spread linked to *Xgwm247* locus on the long arm of chromosome 3B of hexaploid wheat. While two other mapping studies established *XwPt0384* and *Xgwm247* loci in two different linkage groups on the long arm of chromosome 3B (Crossa et al. 2007; Marone et al. 2012). Paillard et al. (2004) also reported a consistent QTL for resistance to severity on long arm of chromosome 3B; however, data suggests that this QTL is located on proximal region of the long arm of chromosome 3B.

Reports involving wheat chromosome 4A with FHB resistance are limited. To date, only one FHB resistance factor has been reported for each arm of chromosome 4A (Buerstmayr et al. 2009). Steed et al. (2005) reported the first FHB QTL located on the short arm of chromosome 4A for Type I resistance, originated from *Triticum aestivum* L. subsp. *macha*. The FHB QTL on chromosome 4A identified in this investigation could be novel, at least for tetraploid wheat. A microsatellite marker (*wmc96*) tightly linked to this QTL was previously mapped to the short arm of chromosome 4A by Somers et al. (2004). Lin et al. (2006) reported a FHB QTL linked to *wmc96* marker, but it was mapped to chromosome 5A.

The two FHB resistance QTL mapped on 5A apparently are the same QTL previously identified by other research groups. The microsatellite marker (*gwm 165*) linked to the chromosome 5A QTL for resistance to severity is the same marker reported by Buerstmayr et al. (2003a,b). The other 5A-QTL for FHB resistance, linked to *gwm291*, was reported by Paillard et al. (2004) and was linked to *gwm 165*. Further investigation revealed that the chromosomal region for 6B-FHB resistance QTL is the same as previously reported by Yang et al. (2005) for Type I, II and III FHB resistance.

Contribution of FHB resistance factors from an adapted parent is not an uncommon phenomenon in FHB resistance studies (Waldron et al. 1999; Shen et al. 2003; Mardi et al. 2006; Ghavami et al. 2011). In this investigation, the adapted parent ('Lebsock') conferred several resistance factors identified in the mapping population: Four Type II resistance QTL, located on chromosomes 1A, 3A and 3B; and one Type V resistance QTL located on chromosome 3A. Ghavami et al. (2011) also reported a Type II resistance QTL incorporated from 'Lebsock'

located on the long arm of chromosome 5B. This finding confirms that Lebsock may possess genes for FHB resistance, as Ghavami et al. (2011) emphasized.

In a previous FHB QTL mapping study (unpublished), we screened a similar BIL population derived from cross between 'Tunisian108' and 'Ben'. Although the donor parent was the same in these two studies, only four resistant QTL were identified in common. The effects of the common QTL are different in their respective populations, owing to high genotype by environmental interactions and unknown epistatic effects. The two resistance QTL identified on chromosome 1B (tightly linked to *XwPt1818* and *XwPt3451* loci) in this report are identical to the major FHB QTL in our previous investigation. Locations of two minor FHB resistance QTL on chromosome 5A (*XwPt4248* and *XtPt418*) are identical to Type II and V FHB resistance QTL presented in our previous study.

Conclusions

Tunisian 108, reported here, represents a useful resource of FHB resistance. The results of this study, in combination with previous work (unpublished), suggest that considerable potential also exists to exploit latent or minor genetic factors from recurrent (susceptible) parent lines in conventional resistant by susceptible FHB breeding-designed crosses. Likely, such minor factors may have strong varietal difference, and experimental limitations may make their elucidation difficult. However, the utility of such FHB resistance factors presents opportunity to exploit transgressive segregants in resistant line development. The minor FHB QTL mapped on 4A chromosome in this investigation may be a new resistance factor, useful toward developing comprehensive FHB resistance. The other mapped QTL presented here are consistent with

previously reported resistance factors. These observations show that the ancient QTL for FHB resistance preserved in *T. aestivum* are relatively consistent in *T. turgidum*.

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Table 4.1. Disease severity and toxin levels in Tunisian108/Lebsock//Lebsock BC_1F_7 population across the experiments

	Disease values (%)					Toxin value (ppm)			
	F2010[a]	F2011[a]	GH2010[b]	GH2011[b]	FDK[c]	DON[d]	3ADON[e]	15ADON[f]	Niv[g]
Population mean	67	78	34	60	78	39.18	0.63	0.72	0.50
Std Dev[h]	10	14	21	21	16	15.86	0.27	0.28	0.01
Minimum	35	10	5	18	15	5.80	0.50	0.50	0.50
Maximum	90	95	95	95	100	98.80	3.30	2.20	0.60
Lebsock	84	90	84	80	86	31.40	0.60	0.60	0.60
Tunisian 108	48	52	54	50	60	25.00	0.40	0.40	0.50
LSD1 (0.05)	18	18	16	16	14.80	14.08	0.20	0.25	0.02
LSD2 (0.05)	26	26	23	23	20.87	19.85	0.28	0.36	0.03

[a]. Field experiment
[b]. Greenhouse experiment
[c]. Fusarium damaged kernel
[d]. Deoxynivalenol
[e]. 3-acetate deoxynivalenol
[f]. 15-acetate deoxynivalenol
[g]. Nivalenol
[h]. Standard deviation
LSD1 compares BIL population mean to parents; LSD2 compares two BILs

Table 4.2. Correlation coefficient assessment for different FHB resistance components measured in a BC_1F_7 population derived from cross between Tunisian108 and Lebsock in 2011 field experiment

Traits	Sev[a]	FDK[b]	DON[c]	3ADON[d]	15ADON[e]	Niv[f]
Sev[a]		0.52	0.03	0.01	0.01	0.01
		<0.0001	0.6499	0.8570	0.8428	0.0992
FDK[b]			0.20	0.08	0.12	0.14
			0.0102	0.2734	o.1257	0.0642
DON[c]				0.56	0.76	0.01
				<0.0001	<0.0001	0.9295
3ADON[d]					0.64	0.03
					<0.0001	0.7208
15ADON[e]						0.10
						0.1833
Niv[f]						

[a]. Disease severity
[b]. Fusarium damaged kernel
[c]. Deoxynivalenol
[d]. 3-acetate deoxynivalenol
[e]. 15-acetate deoxynivalenol
[f]. Nivalenol

Table 4.3. Putative QTL for disease severity, and mycotoxic components detected in Tunasian108/Lebsock//Lebsock BC_1F_7 mapping population

Disease components	Experiments	Chromosome	Position (cM)	Flanking markers	Marker Interval	LOD Score.	Additive	R2 (%)
Severity	F2010d	1B	36.9	wPt0506-wPt5485	10	2.4	2.7	5.4
	F2010	1A	12.5	wPt5876-wPt6280	3	2.4	3.7	5.1
	F2010	1A	35.0	wPt4886-wPt3698	7	3.6	4.0	9.3
	F2010	6B	84.3	rPt1040-wPt8976	25	2.3	2.9	5.5
	F2011	3B	130.7	wPt0384-wPt6981	23	3.9	-4.8	9.3
	F2011	4A	33.7	wmc96-wPt0054	3	3.1	3.8	6.3
	F2011	5A	193.1	wPt2357-gwm291	16	2.1	4.0	4.3
	GH2010e	1A	18.2	wPt3870-wPt4886	6	3.0	-5.5	8.5
	GH2010	3B	3.5	wPt0250-wPt10965	3	2.1	-5.8	4.7
	GH2010	5A	76.5	wPt5309-gwm156	7	2.6	7.8	9.9
	GH2011	1B	53.9	wPt5485-wPt1818	11	2.1	5.5	5.4
	GH2011	3A	98.9	tPt7492-wPt0819	6	2.6	9.0	7.3
	GH2011	3A	165.8	wPt8876-wPt2562	11	2.7	-7.2	6.9
DON[a]	F2011	3B	27.1	wPt10687-wPt11451	12	2.2	4.6	5.1
3-ADON[b]	F2011	3B	38.6	wPt10325-tPt9948	16	2.4	0.1	6.5
15-ADON[c]	F2011	3A	21.3	wPt2938-tPt1143	2	2.9	-0.1	6.9

[a]. 3-acetate deoxynivalenol
[b]. 15-acetate deoxynivalenol
[c]. Nivalenol
[d]. Field experiment
[e]. Greenhouse experiment

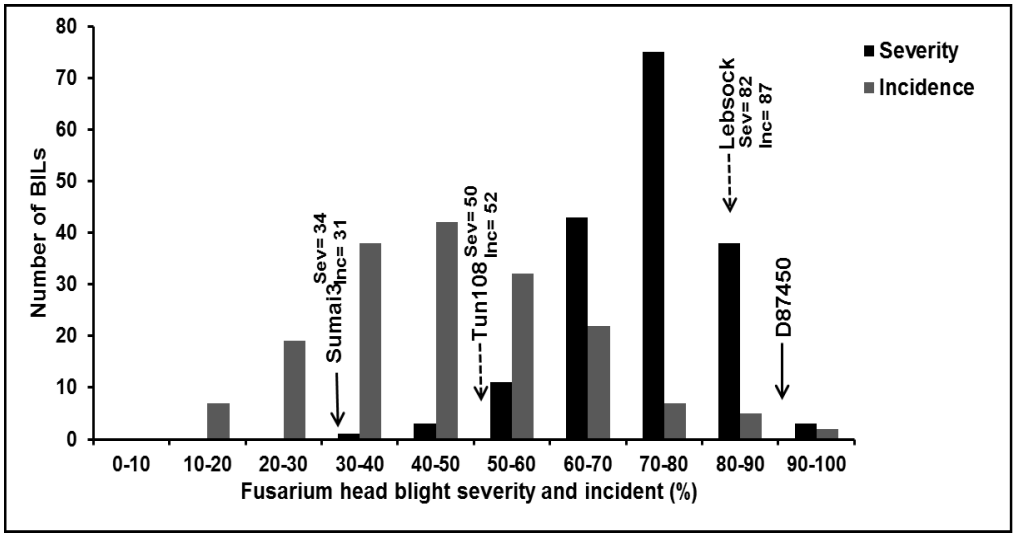

Figure 4.1. Frequency distribution of FHB severity and incidence means of genotypes in a Tun108/Lebsock//Lebsock population in two greenhouse and two field experiments during 2010 and 2011. The average of FHB infection for the parents, the resistant (Sumai3) and susceptible (D87450) checks are indicated by arrows

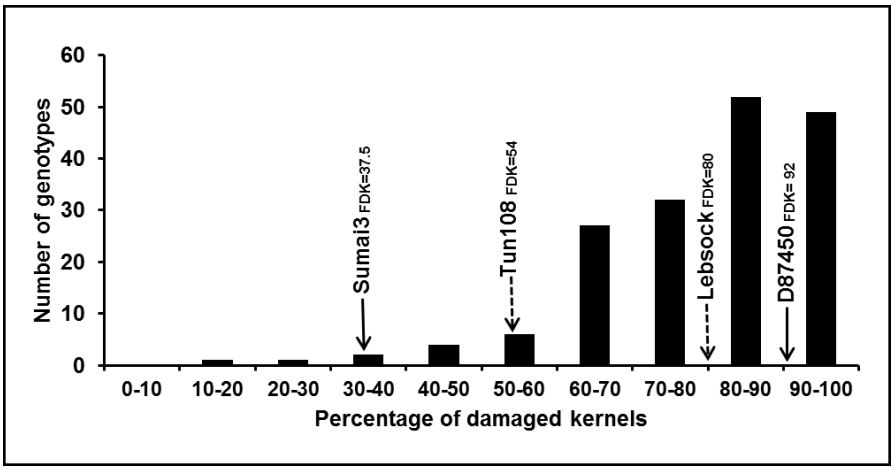

Figure 4.2. Frequency of Fusarium damaged kernels (FDK) means of genotypes in Tun108/Lebsock//Lebsock population in 2011 field experiments. The average of FDK for the parents, the resistant (Sumai3) and susceptible (D87450) checks are indicated by arrows

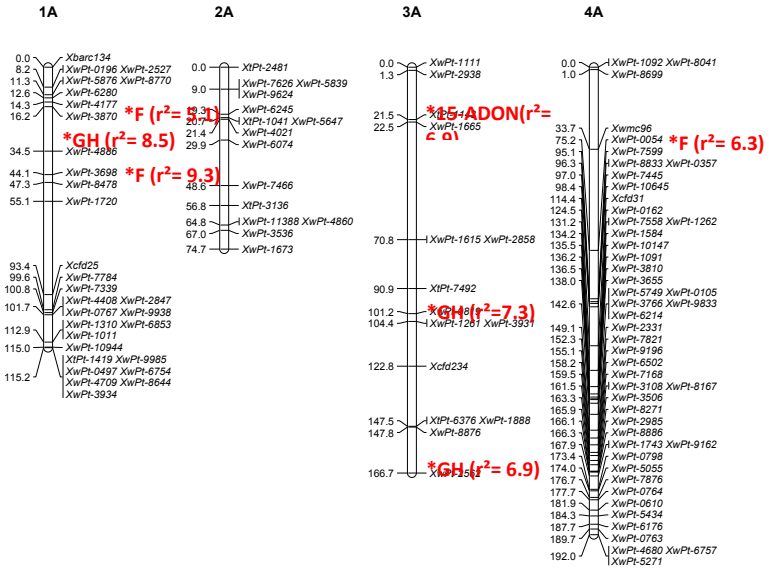

Figure 4.3. Genetic linkage map of 331 mapped markers and putative QTL for BC_1F_7 population derived from a cross between Tunisian108 and 'Lebsock (F= Field; GH= Greanhouse)'

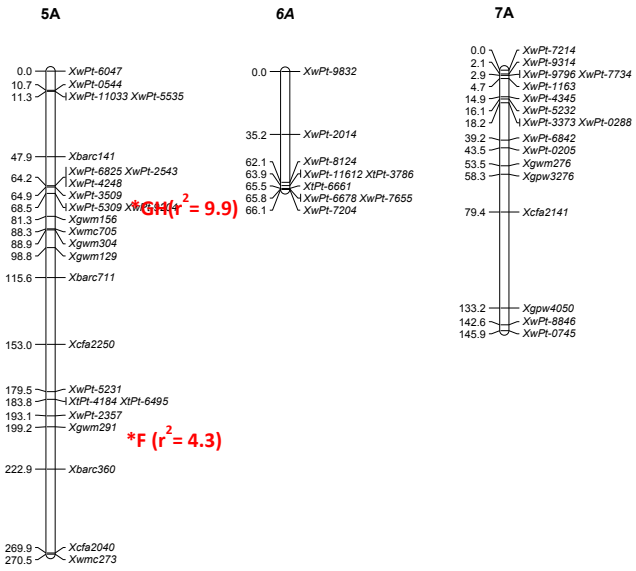

Figure 4.3. Genetic linkage map of 331 mapped markers and putative QTL for BC_1F_7 population derived from a cross between Tunisian108 and 'Lebsock (continued). (F= Field; GH= Greanhouse)'

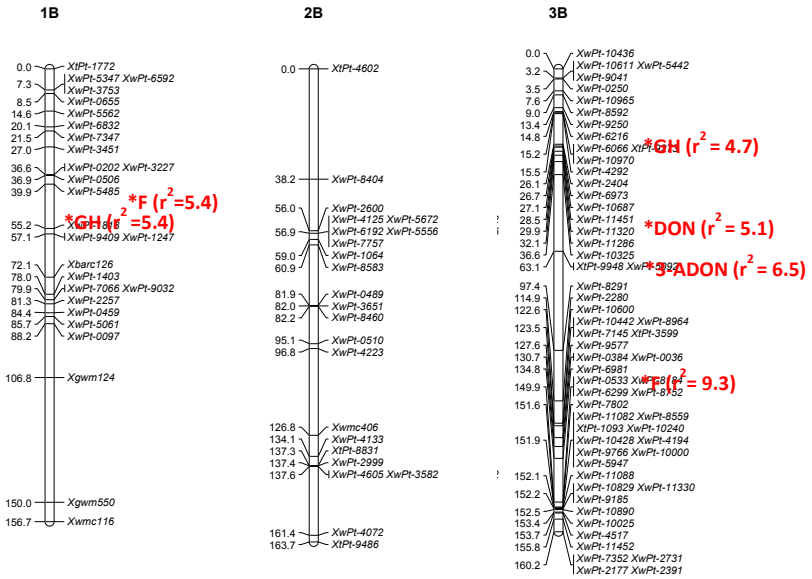

Figure 4.3. Genetic linkage map of 331 mapped markers and putative QTL for BC_1F_7 population derived from a cross between Tunisian108 and 'Lebsock (continued). (F= Field; GH= Greanhouse)'

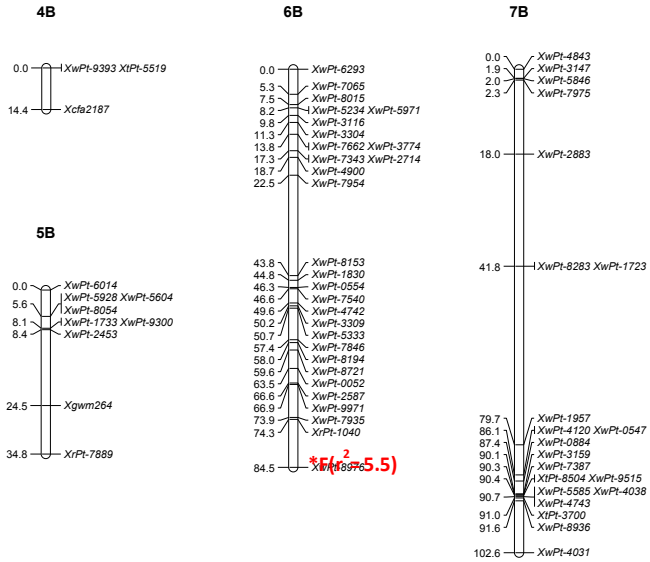

Figure 4.3. Genetic linkage map of 331 mapped markers and putative QTL for BC_1F_7 population derived from a cross between Tunisian108 and 'Lebsock (continued). (F= Field; GH= Greanhouse)'

CHAPTER V. FINAL CONCLUSIONS

Sporadic well-characterized sources of FHB resistance with poor agronomic and quality traits is the most important obstacle for resistance breeding (Comeau et al. 2010). Combination of multiple genes action in FHB resistance sources is another issue for resistance breeding process (Miedaner and Korzun 2012). The challenge is even more stunning as there is little demonstrated variation of FHB resistance in the tetraploid germplasm pool (Somers et al. 2006). It is strongly believed that, some suppressor gene(s) or QTL may neutralize the effective FHB resistance factors in tetraploid genome (Ban and Watanabe 2001; Kishii et al. 2005; Garvin et al. 2009; Ghavami et al. 2011). Another hypothetical point of view insists on role of D genome in resistance. Accordingly, D genome of hexaploid wheat encodes resistance-inducing factors that are missing in tetraploid wheat (Fakhfakh et al. 2011). North Dakota State University (NDSU) durum wheat breeding program made tremendous strives to find new sources of FHB resistance in recent decades (Elias et al. 2005; Huhn et al. 2012; Ghavami et al. 2011). In this investigation we strived to maximize QTL detection by utilizing two BILs population with common donor parent,Tunisisan108/Ben//Ben (BIL_1) and Tunisian108/Lebsock//Lebsock (BIL_2). The current study revealed at least 6 novel FHB resistance QTL which have not been reported by previous investigations. Most of the remaining 26 FHB resistance QTL identified in this study have been characterized previously in hexaploid sources.

In BIL_1 population, the Type V resistance QTL identified on 1A chromosome might be the same QTL which has been characterized in CJ9306 source (Jiang et al. 2007ab) and NK93604 source (Semagn et al. 2007). The Type III FHB resistance QTL mapped on 1B chromosome of BIL_1 has been identified previously in Fundulea201R source (Shen et al. 2003)

and Arina source (Semagn et al. 2007) both of them as Type II FHB resistance QTL. Fundulea201R is a Romanian line with accumulated FHB resistance from various sources other than Chinese lines, and Arina is a winter wheat variety with moderate (Paillard et al. 2003), to high (Miedaner et al. 2001) resistance to FHB. The two resistance QTL for disease severity in field and greenhouse which have been identified on 1B chromosome in BIL_1 (tightly linked to *XwPt1818* and *XwPt3451* loci) are identical to the FHB resistance QTL in BIL_2. High level of genotype by environmental interaction is one of the reasons for different effects of these identical QTL in two populations.

The consistence FHB resistance QTL located on 2BS chromosome in BIL_1 Population might be a novel QTL. Somers et al. (2006) reported a Type II resistance QTL located on 2BS which incorporated by susceptible parent (*T. durum* cv. Strongfield) and Burestmayr et al. (2011) identified a Type II resistance QTL on 2BS chromosome as wall which incorporated from FHB resistant Transcaucasian hexaploid landrace(*T. macha*.). Further consideration of the maps provided by Crossa et al. (2007), Marone et al. (2012) revealed that, the markers interval for 2BS-FHB QTL which has been previously identified in *T. durum* cv. 'Strongfield' source, is located in different location of 2B chromosome. Although the FHB resistance QTL identified in this investigation is located approximately close to the same region which Buerstmayr et al. (2011) reported, however the QTL we have reported here is for different type of resistance.

The QTL with different effects detected for different environments, localized on chromosome 3B of BIL_1 were apparently different from *Fhb1*-QTL reported by Waldron et al. (1999) and Anderson et al. (2001). The marker interval for this QTL (*barc229* and *wPt9766*) located on long arm of chromosome 3B (Xu *et al.* 2008; Marone *et al.* 2012). To date, there are

two reports for 3BL-FHB QTL (Type II), however the associated microsatellite loci with these two QTL (*Xgwm247* and *Xgwm131*) do not correspond to the same region of the 3BL-QTL identified in this investigation. Bourdoncle and Ohm (2003) identified type II FHB resistance QTL on 3BL, but the markers interval of this QTL were mapped on a very distal part of the chromosome (Crossa et al. 2007). The source of resistance allele was a resistant hexaploid wheat 'Huapei 57-2' (developed in China) in their investigation. Paillard et al. (2004) identified a Type II FHB resistance QTL incorporated from the Arina resistance sources in proximal region of 3BL chromosome. The flanking markers linked to the 3BL FHB QTL (*gwm131* and *cfa2143*) have been frequently mapped on long arm of 3B chromosome very close to centromere (Roder et al. 1998; Somers et al. 2004; Paillard et al. 2003). Thus, the FHB-3BL QTL reported in this investigation might be a novel resistance factor, with different type of FHB resistance. The FHB resistance QTL for disease severity for field trial in BIL_2 and type II and V resistance QTL identified in BIL_1 have been mapped on 3B chromosome tightly linked to *XwPt0036* and *XwPt0384* loci in two experimental populations.

All of the evidences show that, 4A chromosome in wheat is not actively involved in FHB resistance (Buerstmayr *et al.* 2009). To date only one FHB resistance factor reported for each arm of this chromosome (Paillard *et al.* 2004; Steed *et al.* 2005; Buerstmayr *et al.* 2009). Steed et al. (2005) reported the first and apparently the last FHB Type I resistance QTL located on very distal region of chromosome 4AS incorporated from a double haploid population (HsTm4ADH) originated from *Triticum aestivum* L. subsp. *Macha.* The FHB QTL identified on chromosome 4A in this investigation is tightly linked to microsatellite marker *wmc96*. This marker mapped for the first time by Somers et al. (2004) in very proximal region on 4AS chromosome. Thus, the 4AS FHB QTL could be a novel resistance factor which we identified. A consistent and

apparently novel FHB resistance QTL which has been detected on 2B chromosome in BIL_1 population also is tightly linked to the *Xwmc96* loci. This marker might be tightly linked to FHB resistance factor because at least there is one report for FHB QTL linked to *wmc96* marker which has been mapped on chromosome 5A by Lin et al. (2006). This is not surprising, because this locus has been mapped frequently on different chromosome such as 5A, 5D, 3A, 6A and 7A (Somers et al. 2004; Paillard et al. 2003).

The major Type II resistance QTL and two Type III resistance QTL localized on chromosome 5A were consistence in both experimental populations. These QTL have been frequently detected by different research group and, there are numerous reports for QTL on chromosome 5A with minor effects on disease incidence and severity associated with different markers (Jiang et al. 2007a,b; Lin et al. 2006; Steiner et al. 2004; Gervais et al. 2003; Shen et al. 2003; Paillard et al. 2004). The major Type II FHB resistance QTL linked to the *Xbarc141* locus on chromosome 5A of BIL_1 might be the same QTL which was reported by Zhang et al (2012). The Type III 5A-FHB resistance QTL identified in BIL_1 is located in the same FHB resistace effective region which has been detected in *T. aestivum* and *T. macha* (Buerstmayr et al. 2002; Buerstmayr et al. 2011). The two minor FHB Type III resistance QTL identified on 5A chromosome in BIL_1 Population, are linked to the same markers (*XwPt4246* and *XtPt418*) which are associated with two identified QTL in BIL_2 population. Interestingly, the Type II FHB resistance QTL on 5A chromosome of BIL_2 linked to the *gwm165* is the same QTL which Buerstmayr et al. (2003 a,b) have already reported. The other 5A-QTL for FHB resistance to severity in BIL_2 population, which is linked to *gwm291*, has been reported by Paillard et al. (2004) exactly linked with the same marker. Thus, FHB resistance factors on 5A chromosome in resistance sources have been properly conserved.

Although Ghavami et al. (2011) used the Tunisian lines as resistance sources, they reported different FHB effective chromosomal regions in comparison to this investigation. The reason of this discrepancy might be because of source of resistance allele. The source of 5B FHB QTL was 'Lebsock' (susceptible parent) in their study, while the incorporating resistance allele was from 'Ben' (susceptible parent) in this investigation. The other type III FHB QTL identified in BIL_1 is only linked with some DArT markers which are not mapped on the same chromosome in available maps. This is a common problem of DArT markers that even Liu et al. (2012) were recently encountered with.

Comparing the flanking DArT marker of two FHB resistance QTL mapped on 7B in BIL_1 with the cumulative maps provided by Marone et al. (2012) and Mantovani et al. (2008) revealed that, they are located on proximal and distal regions of chromosome 7BS. The Resistance QTL located on distal part of 7BS, was incorporated by susceptible parent (Ben). This FHB QTL has been identified in moderately resistant cultivars 'Dream' (Schmolke et al. 2005; Haberle et al. 2007) and 'Cansas' (Klahr et al. 2007) sources, linked to *gwm46* microsatellite marker. However this marker was not polymorphic in this investigation. The FHB resistance QTL located on the proximal region of 7BS has not been reported in any investigation. Further study revealed that, the Type II FHB resistance QTL identified by Ghavami et al. (2011) in Tunisian back ground and Type I FHB resistance QTL identified by Gilsinger et al. (2005) incorporated by cultivar 'Goldfield' are located on long arm of 7B chromosome. The parents and in some cases entire population were tested by DArT marker developed by Ghavami et al. (2011), however the result was not applicable for mapping owing to lack of polymorphism. The donor parent that they used was a different Tunisian line (Tunisian 34). Thus, Tunisian resistance sources might comprise various type of FHB resistance QTL with different associated loci.

Reducing mycotoxin contamination in the seeds is the ultimate goal of a breeding program for FHB resistance (Buerstmayr et al. 2009). Since the correlation of FDK and DON accumulation was high in this study, thus, FDK was used for selecting transgressively segregated resistance lines in both populations. We strongly suggest Fusarium damaged kernel (FDK) assessment as the best phenotypic evaluation criterion for the entire FHB resistance breeding program. In summary, Tunisian lines can be amongst a few tetraploid resistance sources have been ever identified.

he major and consistent FHB resistance QTL which have been identified in Tunisian genomic background can be used for further resistant breeding program, however the inconsistent minor QTL need more validation before incorporating to highly adapted commercial cultivar.

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