REVIEW

Cereal Resistance to Fusarium Head Blight and Possibilities of its Improvement through Breeding

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Abstract: The aim of this review is to summarize recent information on Fusarium head blight (FHB) in small grain cereals, especially in wheat and barley. Basic information on FHB epidemiology, types of resistance and plant resistance mechanisms is included. Standard methods for the evaluation of the individual types of FHB resistance and the extent of infection are briefly described. Special attention is paid to the sources of FHB resistance of different origin and possibility of their exploitation in cereal breeding. Unfortunately, a high level of FHB resistance was detected in non-adapted germplasm or distant relatives, which is a serious impediment to breeding progress in this field. The present state of breeding for FHB resistance in wheat, barley, rye, triticale and oats was analyzed. It was shown that large-scale QTL detections provide new opportunities for increasing the resistance; however, multi-step phenotypic selection still remains to be the most effective tool. Pedigree analyses indicated that the latest progress reached in this field was obtained through the cumulation of resistance genes coming from heterogeneous sources with different response to FHB.

Keywords: FHB; breeding; sources of resistance; quantitative trait loci; marker-assisted selection; wheat; barley; rye; triticale; oats

Fusarium head blight (FHB), also called scab, is a disease of small grain cereals, especially wheat and barley, but also oats, rye and triticale, caused by anamorph forms (= asexual forms of *Ascomycetes* fungi propagating via asexual spores called conidia) of several fungi species – predominantly *Fusarium graminearum* Schwabe, *Fusarium culmorum* (W.G. Smith)Sacc., *Fusarium avenaceum* (Fr.) Sacc. (teleomorph *Gibberella avenacea*); *Fusarium poae* and *Microdochium nivale* (snow mould). The most worldwide-spread sources of FHB are the fungi *Fusarium graminearum* (teleomorph *Gibberella zeae* /Schwein./ Petch) and *Fusarium culmorum* (no teleomorph has been found to date). Two distinct groups of *F. graminearum* strains with different sexual behaviour were described in Australia (Burgess *et al.* 1975) and in the U.S.A. (Cook 1981). Group 1 isolates are heterothallic and produce perithecia only rarely in nature, while Group 2 populations are homothallic and produce perithecia more often. Apart from FHB, *F. culmorum* and *F. graminearum* also cause other serious diseases of small grain cereals, *e.g.* foot rot and crown rot of wheat, foot rot of rye, and others.

FHB poses a potential threat to small grain cereals grown especially in habitats with wet climatic conditions (more than 92% of relative humidity) occurring from the stage of flowering to the soft-

dough stage of kernel development. Besides water availability, also temperature, aeration and light are crucial climatic factors influencing the production and dispersal of inocula, growth, competition, mycotoxin production and pathogenicity. *Fusarium* species were found to differ in their climatic distribution and in the optimum climatic conditions required for their persistence, but most species produce inocula, grow best, and are more pathogenic to cereal heads at warm temperatures and under humid conditions (Doohan *et al.* 2003).

Since the control of FHB spread by fungicides, crop rotation and deep ploughing need not always be an efficient means of crop protection, breeding of FHB resistant cultivars (lines) represents the main approach how to eliminate yield losses and contamination of grain by mycotoxins caused by this disease.

The problem of FHB in small grain cereals has been reviewed by Parry *et al.* (1995), Miedaner (1997), Bai and Shaner (2004) and others. The situation in the European wheat gene pool was described precisely by Mesterházy (2003). Buerstmayr *et al.* (2009) have recently summarized the problems of FHB in wheat and published a list of all FHB resistance QTL (quantitative trait loci) detected in the wheat gene pool so far. Basic information on pathogen biology including the description of its life cycle, production of trichothecene mycotoxins and its population biology is given in the review by Goswami and Kistler (2004).

In this review, we would like to focus on the sources of FHB resistance and on progress in the breeding of FHB resistant cultivars of small grain cereals with respect to the Central European region.

Manifestation of the disease and its spreading

The first record of FHB or scab comes from Smith (1884), who reported the occurrence of this disease in England and attributed it to the fungus *Fusisporium culmorum*.

The symptoms of the disease are quite similar in various small grain cereals: the first symptoms appear as small patches of discoloration at the base of the glume of a floret in a spikelet. As the infection progresses, the patches of discoloration may spread to the rachis and invade the whole spike. The infection is visible as patches which first have a bleached (light) appearance, later they become orange, salmon pink, red or tan or they can even change the infected seeds into hard, dark brown objects (called 'tomb stones'). Recently, Voigt *et al.* (2005) have found out that secreted lipase encoded by the *FGL1* gene of *Fusarium graminearum* acts as a virulence factor in the process of wheat spike infection.

The major potential risk for both humans and animals caused by the infection is the production of mycotoxins, either oestrogenic zearalenone (ZEA or ZON), which is mainly found in infected maize (*Zea mays*) (Yoshizawa 1991), or non-macrocyclic trichothecenes. According to their chemical nature, the trichothecenes produced by *F. culmorum* and *F. graminearum* are divided into two types: type A toxins (HT-2 toxin, T-2 toxin) and type B toxins including the most abundantly produced mycotoxins in small grain cereals, especially deoxynivalenol (DON), its derivatives 3-acetyl-DON and 15-acetyl-DON, and nivalenol (NIV) and 4-acetyl-NIV called also fusarenone X (FX). Most *Fusarium* strains can simultaneously produce multiple types of mycotoxins, *e.g.* DON and ZEA. In general, both DON and NIV chemotypes were reported in Africa, Asia and Europe, while only the DON chemotype was found in the USA (Miller *et al.* 1999; Lee *et al.* 2002). Our latest studies performed in the territory of the Czech Republic (Sumíková *et al.* 2008) showed very rare occurrence of the NIV chemotype and high prevalence of the DON chemotype. DON differs from NIV only in the absence of a hydroxyl group at C-4. However, this difference may have important consequences for the fitness of the producing organism as it alters the bioactivity and toxicity of trichothecenes. For example, the toxicity of NIV is several times higher than that of DON (Alexander *et al.* 2000). The molecular basis of NIV or DON-producing abilities of the isolates lies in the functionality of the *Tri13* gene coding for a cytochrome P-450 enzyme which is functional in NIV-producing isolates, while non-functional in DON-producing strains (Brown *et al.* 2002; Lee *et al.* 2002).

The infection is spread through both the asexual spores (macroconidia) and the sexual ascospores by wind, rain-splash dispersal or by arthropod vectors such as mites (*Siteroptes graminum*), clover leaf weevil (*Hypera punctata*), grasshoppers (*Melanoplus bivittatus*), picnic beetles (*Glischrochilus quarisignatus*), and even the common housefly (*Musca domestica*). Crop residues on the soil surface also represent an important reservoir of

pathogens, containing ascospores, macroconidia and hyphal fragments which can serve as inoculum. The fungus spreads by direct penetration of glumes, palea and rachilla. The fungus infects other spikelets internally through vascular bundles of the rachilla and rachis in susceptible wheat. The infection of plants by other diseases (*e.g.* BYDV) can significantly contribute to the spread of FHB (*e.g.* Liu & Buchenauer 2005). Ascospores, macroconidia (asexual spores), chlamydospores and hyphal fragments in the soil surface debris are the main sources of inoculum.

Types and mechanisms of FHB resistance

FHB resistance is polygenic and its expression is highly influenced by the environment. It becomes obvious that resistance against FHB in small grain cereals is determined quantitatively by several quantitative trait loci (QTL). It is, however, important that many studies showed common and durable resistance to different *Fusarium* spp. causing FHB (Stack *et al.* 1997; Hollins *et al.* 2003; Mesterházy *et al.* 2005). Though the isolates of different *Fusarium* spp. differed largely in quantitative aggressiveness, they did not show any evident qualitative differences in virulence (Mesterházy *et al.* 2005; Akinsanmi *et al.* 2006; Šíp *et al.* 2008). Since the pathogenic specialization can be classified as low, wheat breeding for general *Fusarium* resistance may be possible.

Five types of resistance to FHB have been distinguished in common wheat; the first two have already been classified by Schroeder and Christensen (1963) as resistance to the initial infection (Type I resistance) and resistance to subsequent spread (Type II resistance). Later, resistance to mycotoxin accumulation, indicated by mycotoxin concentrations, uncorrelated with Type I or Type II resistance was recognized (Miller & Arnison 1986). The following components (types) of head blight resistance were distinguished by Mesterházy (1995, 2002): I. Resistance to invasion, II. Resistance to spreading, III. Resistance to kernel infection, IV. Tolerance, V. Resistance to toxin accumulation, VI. Resistance to late blighting, and VII. Resistance to head death above infection site. Boutigny *et al.* (2008) have proposed to divide Type V resistance into two sub-types: Type V-1 resistance, which lies in the promotion of mycotoxin degradation or detoxification, and Type V-2 resistance, which means the prevention of mycotoxin biosynthesis.

Several mechanisms of FHB resistance have evolved in cereals. Mechanisms of resistance to mycotoxin accumulation (Type V resistance) lie in the production of enzymes which are aimed at the detoxification and degradation of mycotoxins. These enzymes include members from the family of pathogenesis-related (PR) proteins like PR-2 (glucanases), PR-3 (chitinases), PR-4 (chitinase types I, II) and PR-5 (thaumatin-like proteins) proteins. The expression of enzymes like peroxidases, lipoxygenase and other ROS scavenging enzymes is also enhanced. It was found out by Li *et al.* (2001) that the FHB-resistant cultivar Sumai 3, one of the most worldwide used source of FHB resistance in wheat breeding programmes, accumulates higher levels of chitinases and β-1,3-glucanases than its susceptible mutant. A wide range of secondary metabolites with pro-oxidant properties like phenolic compounds, carotenoids and linoleic acid hydroperoxides are synthesized in order to act as modulators of mycotoxin biosynthesis. Recently, Bernardo *et al.* (2007) compared changes in gene expression initiated by the infection in resistant cultivar Ning 7840 and susceptible cultivar Clark and found the enhanced expression of defence-related genes (precursor of chitinase II, genes encoding PR proteins, P450, three genes with unknown function) in Ning 7840 relative to Clark during early stages of fungal stress. A detailed review on cereal mechanisms of degradation of *Fusarium* mycotoxins has recently been published by Boutigny *et al.* (2008).

It is also becoming evident that FHB resistance (predominantly Type I and II resistance, *i.e.* FHB initial infection and subsequent spread) is associated with alleles determining plant morphological and physiological characteristics, especially plant height, heading date (Parry *et al.* 1995), inflorescence architecture and traits associated with flowering such as narrow flower opening (Gilsinger *et al.* 2005) or anther extrusion (Taylor 2004). The FHB resistance associated with plant physiological and morphological characteristics is also called passive resistance (Mesterházy 1995). It can be generally summed up that plants with loose spike branching (loose heads) exhibit better resistance to FHB spread than plants with dense heads; however, the grain yield of these cultivars is much lower than of those with dense heads, naturally. Recently, it was found by Zhu

et al. (1999) in doubled haploid (DH) lines from a cross between the two-rowed genotypes of barley, Gobernadora and CMB643, that all but two QTL for resistance to FHB coincide with the QTL for inflorescence architecture and plant height. The authors hypothesized that the architecture of the inflorescence might affect the spread of FHB in barley; however, they concluded that they did not find out whether the coincidence of these two types of QTL was due to linkage or pleiotropy and they concluded that further research was needed in this area. In wheat, several FHB resistance QTLs overlap with QTL for plant height, weight of infected spikelets or awns when examining crosses between unrelated cultivars which differ in these morphological traits (*e.g.* Gervais *et al.* 2003; Srinivasachary *et al.* 2008a). Quite a lot of papers have also been published which examine the effect of allelic constitution at *Rht-B1* locus (also called *Rht1*) and *Rht-D1* locus (also called *Rht2*; loci determining plant height located on 4BS and 4DS, respectively) on FHB resistance in wheat. Srinivasachary *et al.* (2008b) have shown on a mapping population between Spark (a cultivar relatively resistant to FHB carrying *Rht-D1a* allele) and Rialto (FHB susceptible cultivar carrying *Rht-D1b* allele) that the level of FHB resistance correlates with the allele type at *Rht-D1* dwarfing locus, *i.e.* the presence of mutant *Rht-D1b* semi-dwarfing allele, which is considered beneficial for agronomic purposes, results in shorter plants with decreased FHB type I resistance when compared to normal *Rht-D1a* allele. Similar results were obtained by Buerstmayr *et al.* (2008) from the evaluation of FHB resistance in recombinant lines derived from the crosses Arina/Forno, Arina/Riband, Dream/Lynx, G16-92/Hussar, Renan/Récital, SVP-72017/Capo and Capo/Sumai 3 under natural conditions at different locations in Europe. However, Voss *et al.* (2008) showed on a set of three segregating populations of winter wheat that the correlation between the presence of *Rht-D1b* allele and FHB resistance is not absolute, *i.e.* lines carrying *Rht-1Db* allele with a relatively high level of FHB resistance can be obtained. Similarly, Holzapfel *et al.* (2008) showed on three sets of RILs between European winter wheats that the *Rht-D1* genomic region is the major source of variation in FHB resistance in the European wheat gene pool. Analogously to Voss *et al.* (2008), they also obtained semi-dwarf lines with acceptable levels of FHB resistance due to the effects of other minor QTLs. *Rht-B1b* allele at *Rht-B1* locus seems to have a less negative effect, especially on Type II resistance (Srinivasachary *et al.* 2009). At both *Rht-B1b* and *Rht-D1b* loci, DELLA proteins, which are negative regulators of gibberellin (GA) signalling, are located. Since it is becoming evident that *Fusarium* species exploit GA signalling pathways during plant infection, it seems very probable that the negative effect of *Rht-D1* semi-dwarfing alleles (and orthologous *Rht-B1 ones)* on FHB resistance is due to pleiotropy rather than to linkage drag (Holzapfel *et al.* 2008; Nicholson *et al.* 2008). Thus, other semi-dwarfing genes are becoming important in breeding programmes. Recently, Perovic *et al.* (2008) have shown that the introgression of another dwarfing gene, *Rht8*, can lead to the construction of high-yielding semi-dwarf lines with a sufficiently high level of FHB resistance. The authors explained their success by a physiologically different mode of action of the *Rht8* gene in comparison with widely used *Rht-B1b* and *Rht-D1b* dwarfing genes (*Rht8* is a GA-sensitive gene tightly linked to the *Ppd-D1a* allele conferring photoperiod insensitivity, while *Rht-B1b* and *Rht-D1b* alleles are GA-insensitive).

Another adverse characteristic that is usually associated with the introgression of Sumai 3-derived FHB resistance QTL is kernel shattering. Sumai 3 is susceptible to shattering. Zhang and Mergoum (2007) studied the relationship between FHB resistance QTL and the QTL determining kernel shattering in a population of RILs between Sumai 3 and FHB susceptible cultivar Stoa and found out that there would exist a tight linkage between three major FHB resistance QTL and kernel shattering QTL. Another association of FHB resistance (or moderate tolerance) QTL and some physiological characteristics was shown in a study of Gilsinger *et al.* (2005), who found an association and an overlap between FHB resistance QTL and flower opening in a population of RILs between Goldfield (a narrow opening wheat cultivar) and Patterson (a wide opening wheat cultivar).

Generally, it can be stated that the introgression of FHB resistance QTL into elite high-yielding cultivars often brings about a potential risk of worsening of important morphological and physiological characteristics. Therefore, detailed mapping of the FHB resistance QTL as well as the overlapping QTL determining important agronomical characteristics is necessary in order to make significant progress in FHB resistance breeding.

Methods for evaluation of FHB resistance in small grain cereals (wheat)

It is very important to find a reliable method to assess variety resistance to FHB. Several artificial inoculation methods have been reported (Bekele 1985; Liu 1985). Specific inoculation procedures to evaluate fungal spread in the ear (Type II resistance) involve the introduction of inoculum into the central spikelet of an ear at early anthesis (Schroeder & Christensen 1963; Bai & Shaner 1996). Inoculation methods include a hypodermic syringe, a micropipette or a small tuft of cotton soaked in inoculum. These techniques have proved to be reliable (Wang & Miller 1988), but are labour-intensive, and important resistance mechanisms contributing to field resistance might not be detected. Spray inoculation is used for the assessment of both Type I and Type II resistance. It seems that Type I resistance at present cannot be directly measured, only as a difference between point and spraying inoculation. For breeding, the spraying methodology is more suitable as it considers a much wider genetic background than the point inoculation does (Mesterházy *et al.* 2008). To minimize year/location effects on results, it often appears necessary to support the disease development by irrigation of plots.

Management practices such as preceding crop and tillage system play an important role in natural infection of FHB. The use of maize as preceding crop and reduced tillage system with the application of maize plant debris for resistance evaluation was described by Buerstmayr *et al.* (1999). The evaluation of a response to natural (the preceding crop maize and reduced tillage system) and artificial *Fusarium* spp. infection is used for the classification of resistance to FHB in wheat varieties included in the Official Trials in the Czech Republic. Though DON contents after natural and artificial infection were significantly interrelated, the obtained results mainly indicated the importance of simultaneous evaluation of FHB resistance under both conditions. An advantage of the evaluation of variety response under conditions of natural infection can lie in a better correspondence with conditions in agricultural practice, but also data obtained after artificial inoculation are evidently needed to differentiate better between varieties in resistance to FHB (Chrpová *et al.* 2008).

The resistance of studied wheat accessions could be described by disease score on head, % of Fusarium damaged grains (FDG), reduction of kernel weight per head and DON content (Wiśniewska *et al.* 2004). Another criterion of the expression of resistance to FHB is area under the disease progress curve – AUDPC (Shanner & Finney 1977). Lemmens *et al.* (2004) found out that good correlations between AUDPC and DON content are present at low to intermediate disease levels. Both FDG and reduction in thousand grain weight reflecting the kernel infection can be considered valuable traits (Šíp *et al*. 2002), but FDG is more frequently used as an indicator of FHB resistance (Arseniuk *et al*. 1999; Ittu *et al.* 2000; Wiśniewska *et al.* 2002; Lemmens *et al.* 2003; Mesterházy *et al.* 2005). A new method for the evaluation of FHB infection with respect to the plant phenological stage called REML (a statistical method using a mixed model) was recently published by Emrich *et al.* (2008) in order to avoid an unintended selection for late-heading genotypes when evaluating FHB infection in wheat.

Pre-screening for specific partial disease resistance components against FHB using rapid *in vitro* techniques and morphological characteristics may be of use in achieving favourable combinations of resistance components at a greater frequency in FHB resistance breeding. In the detached leaf assay, the partial disease resistance components such as incubation period (period from inoculation to the first appearance of symptoms), latent period (period from inoculation to sporulation) and lesion length are measured (Diamond & Cooke 1999; Browne & Cooke 2004; Browne *et al.* 2006). Significant relationships were found between the FHB and seedling blight resistance *in vitro*, but such relationships were generally highly dependent on the cultivar, and therefore the *in vitro* test is likely the best means for measuring components of FHB resistance and/or genotype-specific resistance components (Brennan *et al.* 2007).

Sources of natural resistance to FHB

Bread wheat (*Triticum aestivum* L.)

Asia: Wheats with the highest levels of FHB resistance come from the Far East region. Apart from Sumai 3, a Chinese spring wheat cultivar used as a source of FHB Type II resistance globally (*e.g.* del Blanco *et al.* 2003; Ittu *et al.* 2008), other cultivars with relatively high FHB resistance

come from this region, *e.g.* Chinese cultivars Ning 7840, Ning 894037 (Shen *et al.* 2003a; Zhou *et al.* 2003), Wangshuibai (Zhang *et al.* 2004; Jia *et al.* 2005; Mardi *et al.* 2005; Lin *et al.* 2004, 2006; Ma *et al*. 2006), Wuhan, Japanese cultivars Nobeokabozu and Nyu Bai (Nyubai) or Korean cultivar Chokwang (Yang *et al.* 2005). Sumai 3 is a highly FHB resistant Chinese spring wheat cultivar which was derived from a cross between Italian wheat Funo and Chinese landrace line Taiwan Xiaomai Wheat. Three major QTL for FHB resistance located on 3BS, 6BS and 5AL have been described in the crosses using the lines derived from Sumai 3 (Waldron *et al.* 1999; Anderson *et al.* 2001; Buerstmayr *et al.* 2002, 2003; Yang *et al.* 2003). The analysis of SSR markers in the studies of Shen *et al.* (2003a), Yang *et al.* (2006) and Yu *et al.* (2006) showed that the major QTL on 3BS, 6BS and 5AL originate from Taiwan Xiaomai Wheat. The QTL on 3BS, 6BS, 5AL and 2DS in Sumai 3 are currently the most precisely characterised FHB resistance QTL in wheat. Cuthbert *et al.* (2006) precisely mapped the QTL on 3BS as a single Mendelian factor with a bimodal type of inheritance and named it *Fhb1* gene. The *Fhb1* gene is predominantly responsible for Type II resistance and is flanked by SSR markers *Xgwm533* and *Xgwm493*. However, Lemmens *et al.* (2008) showed that in the wheat line CM-82036, the *Fhb1* gene confers detoxification of DON and NIV. Since the authors proposed different mechanisms of detoxification for DON and NIV, they suggested that *Fhb1* may be a cluster of at least two resistance genes rather than a single gene. Nevertheless, they concluded that further investigations were necessary to resolve the nature of *Fhb1*. Cuthbert *et al.* (2007) also finely mapped 6BS QTL conferring field resistance as a Mendelian factor named *Fhb2* and flanked by SSR markers *gwm133* and *gwm644*. The QTL on 2DS conferring Type III resistance was recently characterised by Handa *et al.* (2008), who exploited collinearity between wheat and rice genomes and proposed that the QTL on 2DS can encode the wheat homologue of rice multidrug resistance-associated protein (MRP). The FHB QTL region on 2DS flanks the reduced height gene *Rht8* which might influence the initial infection of FHB under field conditions. Handa *et al.* (2008) thus showed a possible strategy how to identify candidate genes underlying some FHB resistance QTL in wheat.

Detailed description of new sources and results of mapping QTL for FHB resistance in the Asian wheat germplasm was provided by Yu (2007).

America: From the South American wheat gene pool, Brazilian spring wheat cultivars Frontana and Encruzilhada are widely used as sources of predominantly Type I FHB resistance QTL (Steiner *et al.* 2004). Relatively high disease incidence (quite low Type II resistance) and high percentage of spikes showing partial sterility (low Type VII resistance) were, however, detected in Frontana after single floret inoculations (Šíp *et al.* 2003). In the North American region, the soft red winter wheat cultivar Ernie released in 1994 by the University of Missouri Agricultural Experiment Station (McKendry *et al.* 1995) represents a promising source of FHB resistance QTL other than Sumai 3 (Liu *et al.* 2005, 2007).

Europe: Genetic diversity of FHB resistance in European winter wheat was recently evaluated by Zwart *et al.* (2008a), who concluded that this study would assist in the selection of parental lines in order to increase the efficiency of breeding efforts for FHB resistance.

There is an ample evidence available that some older winter wheat cultivars, *e.g.* Kooperatorka, Praag 8 and Bizel, possess relatively high levels of FHB resistance and they may serve as alternative sources of FHB resistance non-derived from Sumai 3 and other Asian wheats (Snijders 1990; Šíp & Stuchlíková 1997; Badea *et al.* 2008). However, due to a low agronomic value these tall materials have not been widely exploited in the European wheat breeding programmes until now. Martynov and Dobrotvorskaya (2006) screened 149 Russian and Ukrainian winter wheat cultivars for FHB resistance and found a moderate level of FHB resistance in the cultivars Hostianum 237, Odesskaya 16 and their derivatives. As already mentioned above, the *Rht-D1* locus for semi-dwarf growth represents a major source of variation in the level of FHB resistance in the Western European winter wheat gene pool (Holzapfel *et al.* 2008).

Promising sources of moderate resistance to FHB (better adapted to the conditions that are prevalent in Europe) may be the Swiss cultivar Arina (Paillard *et al.* 2004), German cultivars Cansas, Petrus and Dream (Klahr *et al.* 2007; Schmolke *et al.* 2005), French cultivars Renan (Gervais *et al.* 2003) and Apache (Holzapfel *et al.* 2008), Dutch cultivar Romanus (Badea *et*

al. 2008) and Romanian cultivar Fundulea F201R (Ittu *et al.* 2002; Shen *et al.* 2003b). Using the cultivars Centenaire, Tulsa, Tuareg, Elegant and Arina as sources of moderate type II FHB resistance, specific hybrid combinations were identified with this resistance type at the level equal to or more resistant than the cultivar Arina (Zwart *et al.* (2008b). Badea *et al.* (2008) designated as resistant to FHB also the German cultivars Hermann, Sobi, Sokrates and Toras, Swiss cutivar Runal and Hungarian cultivar M2234. Gosman *et al.* (2007) investigated FHB resistance in 50 cultivars from the U.K. National List of winter wheat cultivars approved for sale and they found out that only three cultivars (Soissons /*Rht-B1b*, *Rht-D1a*/, Spark /*Rht-D1a*/ and Vector) exhibited a higher level of FHB resistance than the FHB-susceptible cultivar Wizard. A moderate level of resistance to FHB was also found in the currently grown Czech cultivars Simila and Alana (Chrpová *et al.* 2007), Sakura (Horčička *et al.* 2007) (Figure 1) and Bakfis, which possesses above all Type V FHB resistance (Laml & Pánek 2008). These cultivars are promising sources of FHB resistance with adaptation to European conditions.

The potential sources of FHB resistance adapted to European conditions were traced back using pedigree analysis (Figure 2). It is obvious from this analysis that it is very difficult to identify the most promising crossing types. For example the cultivars Estica, Hana, Virtue and Caribo can be considered as parents occurring more frequently in pedigrees of moderately resistant germplasm. While Estica and Hana can be considered moderately or medium resistant to FHB (Hana only to the accumulation of mycotoxins in grain) according to available literature (Váňová *et al.* 2001; Šíp *et al.* 2002), semi-dwarf Virtue and tall Caribo do not evidently possess a desirable resistance level (Bandurska *et al.* 1994; Hilton *et al.* 1999). Out of the five registered cultivars tested for resistance to FHB that are descendants of Estica, four cultivars (Simila, Sakura, Romanus and Centenaire) can be considered moderately resistant while Drifter has to be regarded as susceptible. The cultivar Hana was found to be a parent of four moderately resistant Czech varieties (Alana, Alka, Samanta and Sakura), but also of susceptible Saskia and Vlasta and medium resistant Brea and Sulamit. As documented by Schmolke *et al.* (2008), the Swiss cultivar Arina was incorporated in the pedigree of the resistant line G16-92, which was used for the development of resistant German advanced breeding lines like HUS 690 and HUS 692 (L. Hartl, personal communication). Many crosses with Arina were also performed in the Czech Republic, but without evident success (L. Bobková, personal communication). The non-adapted sources of high resistance (*e.g.* Sumai 3) have not yet been successfully used for introducing FHB resistance into adapted, high-yielding European cultivars, although there is a long-time evidence of this source of resistance. It can be derived from this analysis that the recent progress reached in the field of FHB resistance was obtained by combining effective genes using the heterogeneous germplasm with variable response to FHB for crossing. Zwart *et al.* (2008b) analyzed the inheritance of type II

Figure 1. Moderate resistance to FHB in the modern Czech winter wheat cultivar Sakura (on the left) following spraying inoculation with the mixture of *Fusarium graminearum* isolates; on the right: response to FHB in the susceptible Darwin

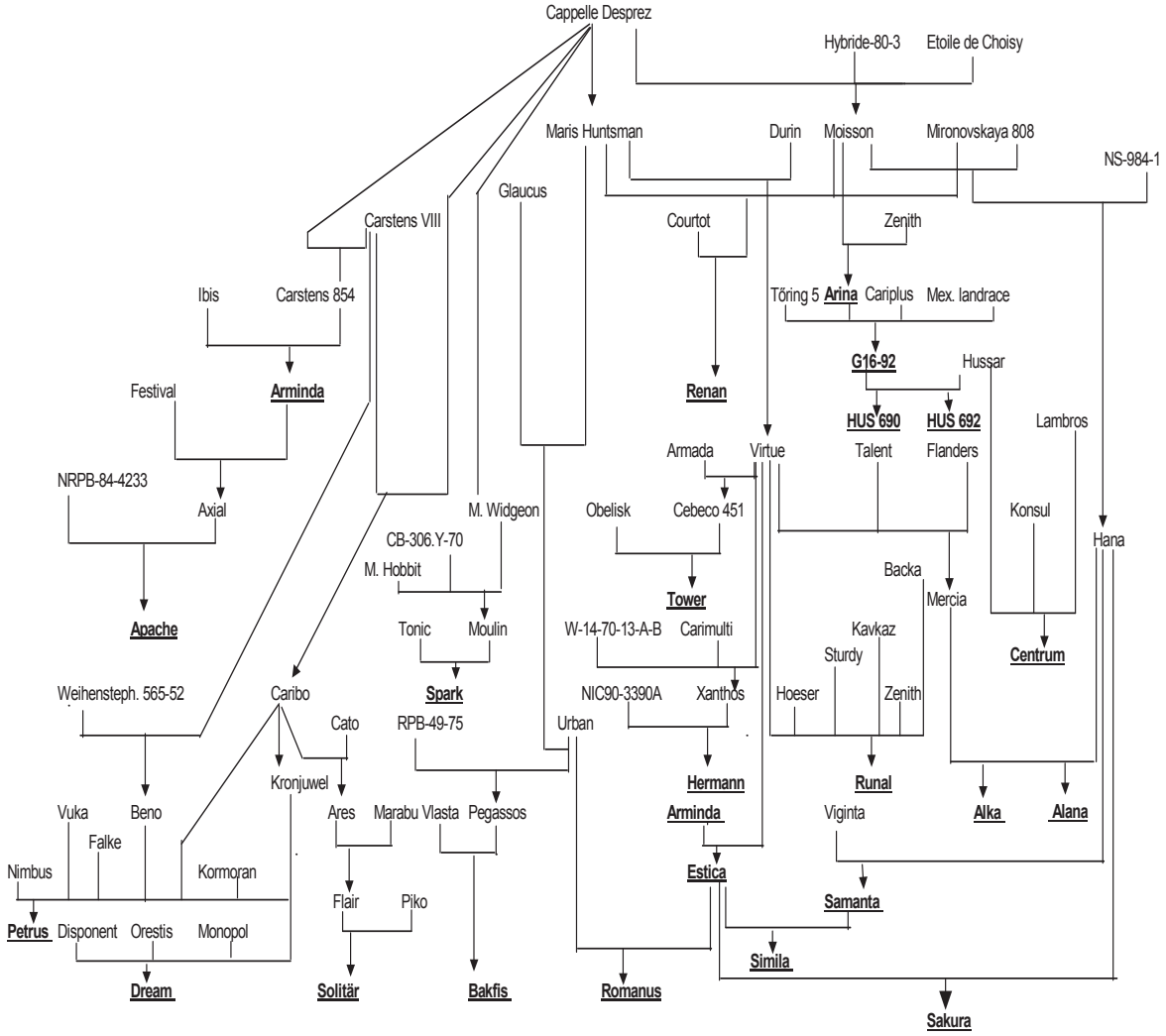

Figure 2. Pedigrees of European winter wheat varieties showing moderate resistance to Fusarium head blight (in bold letters, underlined)

FHB resistance in selected sources of moderate resistance and they showed the importance of both general and specific combining ability effects in the interaction with environment. Specific hybrid combinations of Western European wheat cultivars identified in the F_1 generation exhibited FHB resistance at a level equal to or even higher than in the Arina cultivar.

Sources of FHB resistance which do not come from cultivated wheat

A possible source of FHB resistance for cultivated wheat varieties belonging to both the hexaploid wheat (*Triticum aestivum*) and tetraploid wheat (*T. turgidum* L. ssp. *durum*) is represented by their tetraploid relative wild emmer – *T. turgidum* ssp. *dicoccoides* (Körn. ex Aschers. et Graebn.) Thell. Otto *et al.* (2002) investigated FHB resistance in Langdon – *T. dicoccoides* chromosome substitution lines and identified a single major QTL on 3AS, *Qfhs. ndsu-3AS*, associated with an SSR marker *Xgwm2*. Later, Chen *et al.* (2007) proved that this locus was not homoeologous to the 3BS resistance locus *Qfhs. ndsu-3BS* of Sumai 3 and proposed new markers, *Xfcp401* and *Xfcp397.2*, flanking this locus. A Type I FHB resistance QTL was found on 4AS in a DH population derived from *T. aestivum* ssp. *macha* (Dekapr. et Menabde) by Steed *et al.* (2005). This QTL co-segregated with SSR marker *Xgwm165*.

Wheatgrass (*Lophopyrum* = *Thinopyrum* sp.) is another source of FHB resistance that may be exploited in wheat breeding programmes. Shen and Ohm (2006) identified a QTL for FHB resistance on 7EL of the genome of *Lophopyrum elongatum* and

derived chromosomal substitution lines combining chromosome 7E with the genetic background of Ning 7840 carrying the 3BS FHB resistance locus. The researchers reported that the 7E QTL derived from *Lophopyrum* had a greater effect on the disease resistance than the 3BS locus in their experiment. Later, Shen and Ohm (2007) used a set of chromosomal translocation lines between *Thinopyrum ponticum* and the wheat cultivar Thatcher and identified FHB resistance QTL (*Qfhs.pur-7EL*) on 7EL in a $7el_2$ line which was flanked by codominant markers *XBE445653 and Xcfa2240*, which may be useful for gene pyramiding techniques.

Very recently, the mammoth wildrye – *Leymus racemosus* (Lam.) Tzvelev – has turned to contain a new source of FHB resistance which can possibly be utilized in the breeding programmes aimed at the improvement of FHB resistance in cultivated wheat. Qi *et al.* (2008) characterised a series of wheat-*Leymus* chromosomal translocation lines and described a new resistance gene, *Fhb3*, located at 7Lr#1S (short arm of chromosome 7 originating from *Leymus*).

QTL for FHB resistance in common wheat

Buerstmayr *et al.* (2009) have summarized the results from 52 studies dealing with detection of FHB resistance QTL in different wheat sources; 46 studies were carried out with hexaploid wheats, four with tetraploid wheats (secondary gene pool for common wheat) and two with wheat-related species (*Lophopyrum*, *Thinopyrum*) (tertiary gene pool for common wheat). QTL detections are available for the following resistance sources:

Sources of Asian origin: Sumai 3, Stoa, Ning 7840, ND2603, CM-82036, Ning 894037, Alondra, Huapei 57-2, Nyu Bai, Patterson, Wuhan 1, DH-181, AC Foremost, W14, CS-SM3-7ADS, CJ 9306, Gamenya, Wangshuibai, 5DL, Chokwang, Seri82.

Sources of American origin: Ernie, Frontana.

Sources of European origin (winter wheats with a moderate level of FHB resistance): Arina, Cansas, Dream, Forno, F201R, Goldfield, Hussar, Lynx, Patterson, Remus, Renan, Sincron, Spark, G16-92, NK93604.

Barley (*Hordeum vulgare* L.)

In barley, Type I resistance is important (resistance to initial infection). Type II resistance (spread of infection via vascular bundles in rachilla and rachis within the spike) is less important in barley than in wheat (Zhu *et al.* 1999).

Two-rowed barley Gobernadora (Zhu *et al.* 1999) and six-rowed, nonmalting barley Chevron, CI 4192 (a landrace from China) and Svanhals (a landrace from Sweden) exhibited low levels of FHB (Steffenson 1999) and belong to the most widely used FHB resistant cultivars. Chevron was first introduced in Switzerland in 1914. It is resistant to FHB and kernel discoloration (KD), but its agronomical use is limited due to poor agronomic characteristics, namely weak straw, tall plants, late heading, thin kernels and low yield. Therefore, great effort has been exerted to derive more agronomically valuable lines (materials) from Chevron (Rasmusson *et al.* 1999).

De la Pena *et al.* (1999) identified QTL for resistance to FHB, DON accumulation and kernel discoloration (KD) in $F_{4:7}$ breeding lines derived from Chevron × M69 cross (M69 is an elite breeding line susceptible to FHB). Steffenson and Smith (2006) reported that the Chevron alleles at the QTL on chromosome 2H reduced FHB by 43% and the alleles at the 6H region by 22%, while increasing grain protein by 14 g/kg. These authors also mentioned that marker-assisted selection (MAS) was then generally used to select lines homozygous for the resistant marker allele in the F_2 generation prior to single seed descent (SSD).

Hori *et al.* (2005) studied the nature of FHB resistance in a set of 125 RILs between Russia 6 (a two-rowed resistant cultivar of Russian origin) and H.E.S. 4 (a six-rowed susceptible cultivar). They identified three FHB resistance QTL, two of them located on 2HL and the third on 5HS. One of the QTL on 2HL was coincident with the *vrs1* locus, which determines the inflorescence row type, and the other QTL on 2HL was positioned in the vicinity of the cleistogamy locus (*cly1* or *Cly2*), which determines inflorescence opening. Thus, in accordance with the results published by Zhu *et al.* (1999), it becomes evident that the FHB resistance QTL are often associated with QTL determining inflorescence characteristics in barley. Whether this association is due to linkage or pleiotropy, remains to be elucidated. Recently, Sato *et al.* (2008) showed on a set of RILs between Harbin two-row and other five two-rowed cultivars that no FHB resistance QTL was associated with the *vrs1* locus, but all RIL populations contained FHB resistance QTL at *cly1*/*Cly2* locus (cleistogamy locus) on 2HL.

Buerstmayr *et al.* (2004) evaluated the level of FHB resistance in a set of 143 barley lines containing 88 European spring barley lines and cultivars, 33 accessions from the genebank at IPK Gatersleben and 22 lines of North American origin. The lines with the lowest FHB severity were two-rowed landraces CIho 4196 and PI 566203. Apart from the Swiss six-rowed cultivar Chevron, other European spring barley cultivars with moderate resistance (although with 2–3 times higher AUDPC values than the most resistant lines) were identified, *e.g.* Hellana, Pixel, Secura, Thuringia. One of the relatively highly resistant cultivars was also an old hulless line HOR 1867 which is the old Czech cultivar Boehmische Nackte. The researchers found a significant negative correlation ($r = -0.55$) between plant height and FHB resistance. Similarly, Šíp *et al.* (2004) found a high level of resistance to an aggressive isolate of *Fusarium culmorum* in Chevron and CI 4196 as important sources of FHB resistance in the barley germplasm and moderate resistance in the cultivars Jersey, Olbram and Scarlett when examining FHB resistance and DON content in spring barley cultivars registered in the Czech Republic.

Most FHB resistant barley cultivars exhibit poor agronomical characteristics, have poor malting quality and are two-rowed (Zhu *et al.* 1999).

Fusarium **infection in oats** (*Avena sativa* L.)

Oats is generally less susceptible to *Fusarium* infection than wheat or barley due to the panicle structure of its inflorescence resulting in single spikelet infections and slower infection spread. Several *Fusarium* species infect oats – besides *F. culmorum* and *F. graminearum*, the species *F. avenaceum* (predominant in Poland), *F. langsethiae* (common in Scandinavia; Torp & Nirenberg 2004), *F. poae* and *F. sporotrichioides* (major sources of *Fusarium* infection in Canada) appear in regions where oats is the staple cereal crop. Infections caused by *F. culmorum* and *F. graminearum* are prevalent in wet (humid) years, while infections by *F. langsethiae* predominate in dry seasons. *F. avenaceum* produces predominantly moniliformin (MON) and *F. langsethiae* and *F. sporotrichioides* produce highly toxic type A trichothecene T-2 toxin and its deacetylated form HT-2 toxin. Fusarium Head Blight (FHB) is often difficult to assess in oats. The most reliable approach to the evaluation of *Fusarium* infection in oats is scoring of infected seeds after harvest (expressed mostly as percentage of infected seeds). This is usually assessed by a "freeze and blot" method (Limonard 1966). One hundred seeds are imbibed for 24 h on a wet filter paper in a transparent plastic box and then frozen to kill the germ. After the freezing treatment, boxes with seeds are placed under the source of UV-light and a fraction of infected seeds is calculated after 10–12 days of UV-treatment. Alternatively, the evaluation of mycotoxin content (especially DON) in kernels is carried out. These two data do not often correlate. The visible symptoms of *Fusarium* infection in oats may include brownish, necrotic seeds or pink mycelium on glumes which is visible only at high air humidity.

There exists a considerable genetic variation in oat susceptibility to *Fusarium* infection and mycotoxin accumulation among various cultivars (sources). Generally, early cultivars and hulless (naked) cultivars are less susceptible to infection. Cultivars Bessin and Lena seem to be highly susceptible to *F. graminearum* infection during the germination stage (Bjørnstad & Skinnes 2008). Among Canadian oats, naked oat cultivar Boudrias and hulled cultivar Leggett reveal a relatively high tolerance to *Fusarium* infections (Tekauz *et al.* 2008). A promising source of *Fusarium* resistance in the oat gene pool may be represented by lines (materials) derived from *Avena sterilis*. In 2001, a set of progeny lines from direct or back-crosses to *Avena sterilis* (accessions originating in Eritrea or Israel or unknown) from Dr. K.J. Frey's programme at Iowa State University were inoculated in the field with *F. culmorum*. Single-seed descent (SSD) lines were developed from crosses with the cultivars Hurdal (Norwegian, early maturing) and Belinda (Swedish, late maturing). They were tested (spray inoculations with *F. culmorum*) in 2007 and repeatedly in 2008.

Fusarium **infection in rye** (*Secale cereale* L.) **and triticale** (*Triticosecale*)

Rye and triticale are generally less susceptible to FHB than wheat. Cultivated rye and triticale varieties also exhibit a narrower range of FHB resistance than wheat cultivars do (see *e.g.* Miedaner *et al.* 2001). Miedaner and Perkowski (1996) and Miedaner *et al.* (2001) found twice lower amounts of DON in winter rye varieties than in winter wheats under field conditions. Miedaner *et al.* (2001) reported that under the same infection conditions (an experiment carried out in a

growth chamber under controlled conditions) rye accumulated nine times lower DON amounts than wheat and triticale had five times lower DON levels than wheat after inoculation with the same *Fusarium culmorum* isolate. The inheritance of FHB resistance in rye and triticale is determined mainly by additive genetic effects. According to field tests carried out by Miedaner *et al.* (2001), the winter rye variety Amando exhibited a relatively high level of FHB resistance.

Triticale is generally less susceptible to infections caused by *Fusarium culmorum* and *Fusarium graminearum* than wheat and barley. The predominant *Fusarium* species found on triticale is *Fusarium avenaceum* (Chełkowski *et al.* 2000), the main mycotoxin product of which is moniliformin (MON). In triticale as well as in wheat and barley there exist differences in susceptibility to Fusarium infections among various cultivars. Ittu and Ittu (2008) found a negative correlation between AUDPC and relative kernel weight expressed as % of the control in ten triticale varieties grown in Romania after an artificial inoculation with *F. culmorum* and *F. graminearum* isolates. They concluded that the evaluation of these parameters might be beneficial in selection of triticale genotypes with improved resistance to FHB (*e.g.* the cultivars Gorun, Lotru, Plai and Stil of the ten Romanian cultivars tested in this study).

Miedaner *et al.* (2004) determined a positive correlation between DON content and percentage of *Fusarium* damaged kernels (FDK) rating in a set of 55 triticale genotypes after an artificial inoculation by *F. culmorum* isolate in a test consisting of six location-by-year combinations. In repeated tests, the triticale variety Lasko showed a relatively high level of FHB resistance (Oettler & Wahle 2001). Oettler *et al.* (2004) showed that additive genetic effects are of major importance in the inheritance of FHB resistance in triticale.

Characteristics of approaches usable in breeding for FHB resistance

Recently, much progress has been made in the breeding of small grain cereals, especially wheat and barley, for resistance to FHB. In wheat and barley, the search for sources of FHB resistance has led to detection of several QTL conferring increased resistance to FHB. In wheat, however, the QTL with a strong effect on FHB resistance were detected in exotic materials of Asian origin such as Sumai 3, Wangshuibai, *etc.*, which are genetically distant from the European wheat gene pool. Several QTL affecting FHB resistance moderately have also been detected and characterized in European wheat cultivars. However, the effect of QTL from the European wheat gene pool is largely dependent on a given genetic background in a breeding population. Moreover, the introduction of FHB resistance QTL originating from an exotic source into an elite breeding material is often associated with the introgression of several other QTL which negatively affect agronomically important plant characteristics such as plant height, inflorescence architecture, heading date and final yield. It is often unresolved whether this coincidence of FHB resistance and adverse agronomical characteristics is a consequence of pleiotropy (= two different phenotypic effects of one locus) or linkage drag (= the presence of two different tightly linked loci with different effects). This problem – pleiotropy or linkage drag – should be resolved by fine mapping of FHB resistance QTL and their association with tightly linked molecular markers.

Rapid progress in techniques of molecular biology has led to the construction of more convenient (and cheaper) SSR and STS markers than previously used RFLP and AFLP markers. The association of FHB resistance QTL with molecular markers could be employed in marker-assisted selection (MAS), *i.e.* selection of those genotypes that carry a given molecular marker which is tightly linked to the resistance QTL and thus probably also carry this desirable QTL. Since the inheritance of FHB resistance evidently seems to be predominantly governed by additive effects, the stacking of non-homoeologous QTL from different sources in one breeding population (so called QTL pyramiding) could lead to the construction of plant material carrying different QTL and exhibiting improved FHB resistance when compared to the original QTL sources (*e.g.* Wilde *et al.* 2007, 2008).

A list of FHB resistance QTL identified in the wheat gene pool so far has recently been published in Buerstmayr *et al.* (2009). Currently, researchers are trying to distinguish homoeologous QTL of different origin from non-homoeologous ones. One approach how to resolve this problem is meta-QTL (MQTL) analysis. Löffler *et al.* (2009) analyzed FHB resistance QTL in populations derived from Sumai 3, Wangshuibai and Arina and they described 19 MQTL (QTL shared by different genetic sources) located on 12 chro-

mosomes on all three genomes (six, ten and three MQTL on A, B and D genomes, respectively). Some MQTL coincided with the previously identified FHB resistance QTL (*e.g.* regions on chromosomes 3BS, 5A and 6BS), some were newly identified. It is important that resolving the genetic architecture of FHB resistance by QTL meta-analysis may enable the breeders to choose parents not comprising the same resistance loci or QTL intervals and exploit appropriate MQTL together with their respective markers in MAS.

In barley, major QTL for FHB resistance have been co-localized to the loci determining row type (*Vrs* locus determining six-row or two-row type of barley inflorescence) and flowering type (*Cly* locus determining open – chasmogamous – or closed – cleistogamous – type of flowering) on 2HL. In addition to these major QTL, other QTL with minor effects have been detected on 5H, 6H, *etc.*

Based on several studies (reviewed for wheat in Buerstmayr *et al.* 2009), it should be noted that even the major QTL are responsible only for a minor part of phenotypic variation in FHB resistance. Thus, it appears to be necessary in breeding programmes to combine genotypic selection (selection of genotypes containing FHB resistance QTL via MAS) with phenotypic selection of genotypes revealing a sufficient level of FHB resistance in field tests (Wilde *et al.* 2007; Löffler *et al.* 2009). It is evident that the latest improvement in FHB resistance was reached through the combination of favourable not yet identified genes via phenotypic selection. Cultivars with a higher resistance level were the results of breeding programmes targeted on FHB resistance such as F201 R (Ittu *et al.* 1997) or the resistance of these cultivars was detected during the breeding process. It was shown by Wilde *et al.* (2007) that both phenotypic and marker-assisted selection (MAS) are efficient tools in practical breeding and have a potential to reduce FHB symptoms and grain DON content. The large range of variation for FHB resistance was detected within the marker-based variant and selection gain was substantially enhanced when additional phenotypic selection was accomplished in three steps. It is, however, evident that the combination of MAS and phenotypic selection can be highly expensive, particularly when recurrent backcrossing is needed with the use of non-adapted cultivars as resistance sources. Phenotypic selection (evaluation of plant response to Fusarium infection in field conditions) will probably play also a decisive role in the near future, which can be deduced from current breeding results. However, it is highly disadvantageous that the cultivar response to FHB is strongly influenced by environmental conditions. Moreover, another impediment for the breeders is represented by the multi-component quantitative expression of this trait, which often prevents detection of similar results on reaction to FHB in different experiments. Therefore, the importance of using reliable screening methods (described earlier in this study) should be emphasized. It should also be highlighted that these methods could be more fruitful when applied on a multi-environment and multi-replication basis. Seed shortage, complicated assessment of the resistance level and necessity to combine FHB resistance with other desirable agronomic traits, like yield capacity, quality and adaptation, are the main obstacles to screening for FHB resistance since very early hybrid generations. As demonstrated in detail *e.g.* by Ittu *et al.* (2002) for wheat and Steffenson and Smith (2006) for barley, the field evaluation of FHB resistance does not usually start earlier than in F_5.

Transgenosis is another approach how to improve the plant genetic background for higher FHB resistance. However, the practical use of transgenic techniques in breeding programmes is limited by the EU strict legislation. Nevertheless, a few studies on the improvement of FHB resistance via transgenosis have already been published, *e.g.* Balconi *et al.* (2007), who introduced maize antifungal gene *b-32* under CaMV 35S promoter into the wheat cultivar Veery and obtained transgenic plants with enhanced Type II and Type III resistance and reduced level of kernel shattering. Other work published by Chen *et al.* already in 1999 reported enhanced resistance in the wheat cultivar Bobwhite expressing a thaumatin-like protein from rice.

Conclusions for resistance breeding

– It is evident from this study that sources of high FHB resistance predominantly come from a germplasm not adapted to European conditions or from distant wheat relatives, which is a serious impediment to breeding progress. For cultivation in the Central European region, only moderately resistant cultivars are available at present. These

cultivars (breeding lines) were usually obtained after multi-step phenotypic selection through the combination of favourable not yet identified genes derived from heterogeneous sources differing in response to FHB.

– Owing to the existence of different resistance components and quantitative expression of this trait, it is advantageous to perform simultaneous selection on a multi-environment and multi-replication basis both in conditions of artificial infection and in conditions of natural infection under high disease pressure (after maize as a preceding crop and reduced tillage).

– Seed shortage, multi-component quantitative nature, high affection of the disease level by interactions with the environment and necessity to combine FHB resistance with other desirable agronomic traits prevent breeders from screening for this trait in the early hybrid generations on an individual plant basis. On the plant basis, indirect selection for morphological or physiological characters associated with FHB resistance (like greater plant height, GA sensitivity, earliness, narrow flower opening, loose spike branching) can be performed, but it is unlikely that this selection will be successful in general.

– Clear recommendations for breeding practice how to select prospective hybrid combinations are difficult to postulate. However, some studies indicate that the analysis for a resistance level in early hybrid generations (F_1 or F_2) could be advantageous from these aspects (Zwart *et al.* 2008b). The selection of resistant parents having QTL on different chromosomes is now feasible in wheat due to the use of a QTL meta-analysis approach.

– Even though much progress in the development of FHB moderately resistant cultivars has been achieved until now mainly with the use of conventional breeding methods (after crossing of adapted materials), new opportunities for the utilization of genetically distant sources possessing high FHB resistance may be given by the identification of QTL or genes underlying this resistance. It was shown that the marker-based introgression of resistance QTL in traditional breeding materials may result in a substantial increase in resistance within the shortest possible time. However, this expectation appears true only when the donor-QTL effects are large enough (like on chromosomes 3BS or 5A in wheat) to suppress several adverse effects of a given genetic background.

– To exploit the full range of quantitative variation for FHB resistance, phenotypic selection in the field should follow marker-based selection to bail out genetic variance that is caused by the other resistance genes undetected in QTL mapping studies. Though laborious, time-consuming and restricted by the environmentally dependent symptom expression, phenotypic selection still remains to be the most effective tool and provides an opportunity to select for other highly economically important traits (like plant height, resistance to leaf diseases, yield components, *etc.*), which is especially valuable when introgressing non-adapted germplasm.

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