

Review

Breeding wheat and rye for resistance to *Fusarium* diseases

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Abstract

Fusarium culmorum and *F. graminearum* Groups 1 and 2 cause seedling blight, crown rot, foot rot and head blight in wheat and rye that may affect grain yield and quality for baking and feeding. This review starts with an analysis of *Fusarium* populations with regard to their genetic variation for aggressiveness, mycotoxin production, and isolate-by-host genotype interaction. To assess resistance in the different host growth stages, quantitative inoculation and disease assessment techniques are necessary. Based on estimated population parameters, breeding strategies are reviewed to improve *Fusarium* resistance in wheat and rye. Epidemiological and toxicological aspects of *Fusarium* resistance that are important for resistance breeding are discussed.

F. culmorum and *F. graminearum* display large genetic variation for aggressiveness in isolate collections and in naturally occurring populations. The production of mycotoxins, especially deoxynivalenol and its derivatives, is a common trait in these populations. Significant isolate-by-host genotype interactions were not found across environments in wheat and rye.

Artificial infections in the field are indispensable for improving *Fusarium* crown rot, foot rot and head blight resistance in wheat and rye. For a reliable disease assessment of large populations, disease severity ratings were found to be the most convenient. The differentiation of host resistance is greatly influenced by an array of nongenetic factors (macro-environment, microclimate, host growth stage, host organ) that show significant interactions with host genotype. Selection for environmentally stable resistance has to be performed in several environments under a maximum array of different infection levels. Selection in early growth stages or on one plant organ does not in most cases allow prediction of resistance in adult-plant stages or another plant organ.

Significant genetic variation for resistance exists for all *Fusarium*-incited diseases in breeding populations of wheat and rye. The pathosystems studied displayed a prevalence of additive gene action with no consistent specific combining ability effects and thus rapid progress can be expected from recurrent selection. In wheat, intensive testing of parental genotypes allows good prediction of the mean head blight resistance after crossing. Subsequent selection during selfing generations enables the use of transgression towards resistance. In hybrid breeding of winter rye, the close correlation between foot rot resistance of inbred lines and their GCA effects implies that selection based on the lines *per se* should be highly effective. This is not valid for *F. culmorum* head blight of winter rye caused by a greater susceptibility of the inbred lines compared to their crosses.

For both foot rot and head blight resistance, a high correlation between the resistance to *F. graminearum* and *F. culmorum* was found in wheat and rye. Mycotoxin accumulation occurs to a great extent in naturally and artificially infected plant stands. The correlation between resistance traits and mycotoxin contents are medium and highly dependent on the environment. Further experiments are needed to clarify whether greater resistance will lead to a correlated reduction of the mycotoxin content of the grains under natural infection.

Key words: aggressiveness — crown rot — disease assessment — foot rot — *Fusarium* spec. — head blight — inoculation techniques — mycotoxins — quantitative-genetic parameter — resistance breeding — seedling blight — selection — wheat

Fusarium species are economically important pathogens in most agricultural crops. They occur on all vegetative and reproductive organs of plants, causing wilts, rots or blights. Moreover, they have been isolated from soils of every continent except Antarctica (Windels 1992). In small-grain cereals, about 20 *Fusarium* species have been regularly associated with disease symptoms (Duben and Fehrmann 1979a, Gerlach and Nirenberg 1982). *F. culmorum* (W.G. Smith) Sacc., *F. graminearum* Schwabe (teleomorph: *Gibberella zeae* (Schw.) Petch), and *F. avenaceum* (Corda ex Fries) Sacc. (teleomorph: *G. avenacea* Cook) were most frequently isolated (Cook 1968, Duben and Fehrmann 1979a, Mesterházy 1995a). This review will focus on *F. culmorum* and *F. graminearum* because *F. avenaceum* isolates were generally found to be less aggressive in small-grain cereals (Colhoun et al. 1968, Mesterházy 1978, Duben and Fehrmann 1979b, Diehl 1984, Stack and McMullen 1985, Wilcoxson et al. 1988).

F. culmorum and *F. graminearum* are 'generalists', infecting all cereal species including wheat (*Triticum aestivum* L.) and rye (*Secale cereale* L.), and a large number of nongramineous hosts (Gerlach and Nirenberg 1982). *F. culmorum* is preferentially found in maritime regions, while *F. graminearum* dominates in more continental areas (Cook 1981a, Mesterházy 1995a, Parry et al. 1995). Their relative prevalence seems to be mainly determined by weather conditions. The kind of disease they cause is predominantly a function of inoculum, plant growth stage and environment (Cook 1981a).

Seedling blight caused by *F. culmorum* and *F. graminearum* Group 2 is mainly seed-borne in the humid climates and leads to a reduced number of plants and secondary attack by pests (e.g. *Oscinella frit*) due to thinned stands and delayed plant development. Seedling blight caused by *F. graminearum* Group 1 results, in contrast, from soil-borne infection and occurs mostly in dry soils (Burgess et al. 1981).

At the stem base of small-grain cereals, three epidemiologically distinct *Fusarium*-incited diseases may occur: (1) brown foot rot caused by *F. culmorum* and *F. graminearum* Group 2 in areas with high soil moisture and humidity (Cook 1981a); (2) crown rot caused by *F. graminearum* Group 1 under dry weather and soil conditions, especially in the North-western USA (Cook 1968), eastern Australia (Burgess et al. 1981),

South Africa (Marasas et al. 1988); and (3) common root rot caused by a complex attack of *F. culmorum*, *F. graminearum* Group 2, and *Bipolaris sorokiniana* in the Great Plains of the USA and the Prairie Provinces of Canada (Windels and Holen 1989). The attack of brown foot rot starts from above-ground inoculum (Cook 1981a), with the fungus penetrating the successive layers of the leaf sheaths during the growth period and finally reaching the stem. The lowest internodes, but not the crowns or roots, show necrotic lesions and may develop a soft rot that cannot be seen before flowering (Fehrmann 1988). Brown foot rot is often the result of a complex attack of *F. culmorum*, *F. avenaceum*, *Pseudocercospora herpotrichoides* and *Microdochium nivale* (Duben and Fehrmann 1979a, Miedaner et al. 1993b). Brown foot rot causes yield losses due to a reduced capacity of the stem for the movement of water and nutrients and an increased risk of lodging. Additionally, baking and feeding quality are impaired by lodged wheat and rye crops. Crown rot is caused by below-ground inoculum entering the plants around emerging roots and crowns (Cook 1981a). The infection remains latent unless the plant is subjected to heavy water stress reaching plant water potentials between -32 and -35 bars or lower (Cook 1981b). The characteristic symptoms are scattered, bleached, and dead plants among unaffected plants in fields exposed to water stress. Crown rot causes premature ripening and thus a reduction of kernel number and/or kernel weight (Burgess et al. 1981). In the more humid climate of Central Europe such heavy water stress on wheat-grown soils and the combined crown rot is unlikely to occur (Jenkins et al. 1988) but in dry wheat-growing areas crown rot is the most destructive stem disease (Burgess et al. 1981, Cook 1981a, Wildermuth and McNamara 1994).

Fusarium species are usually not primary pathogens of healthy upper leaf blades. By means of lesions caused through powdery mildew (Mathis et al. 1986) and aphids or through mechanical wounds (Diehl 1984), they frequently enter leaves. Head blight is caused by ascospore or macroconidia infection in periods of high humidity ($> 92-94\%$ relative humidity, Cook 1981a) and temperatures above 15°C (Parry et al. 1995). Infections may occur at any time from head emergence to maturity, but disease severity is greatest when inoculum is present in the flowering period of both wheat and rye (Anderson 1948, Diehl 1984, Mielke 1988, Gang 1997). Symptoms and disease development have been extensively reviewed (Cook 1981a, Sutton 1982, Teich 1989, Parry et al. 1995). Head blight epidemics result in severe yield loss by destruction of the embryo and/or reduction of kernel weight, poor milling and baking quality of wheat (Meyer et al. 1986, Pomeranz et al. 1990), reduced germination rate and seedling vigour in the following crop (Manka 1989), and also contamination of kernels with mycotoxins. *F. culmorum* and *F. graminearum* are both capable of producing trichothecene type A toxins (HT-2 toxin, T-2 toxin), type B toxins (mainly deoxynivalenol, 3-acetyldeoxynivalenol, 15-acetyldeoxynivalenol, nivalenol, fusarenone-X, calonectrin), and zearalenone in epidemics in wheat, barley, triticale, and rye (Marasas et al. 1984, Chelkowski 1989, Perkowski et al. 1995). Several mycotoxins may occur simultaneously in different composition and amounts. They are hazardous to animal and human health (Friend and Trenholm 1988, Pomeranz et al. 1990, Snijders 1990a).

For natural infections, yield losses caused by the various *Fusarium* diseases were reported to range from 7% to 17% for seedling blight (Greaney et al. 1938, Duben 1978), from 10% to 30% for foot rot (Duben 1978, Meyer 1985), from 0% to

17% for crown rot (Dodman and Wildermuth 1987), and from 30% to 70% for head blight (Martin and Johnston 1982). With artificial inoculation, much greater losses can occur with the various diseases (e.g. Purss 1966, Diehl 1984, Miedaner and Walther 1987, Chelkowski 1989, Snijders 1990f, Miedaner et al. 1993a). Fungicide treatment and agricultural management practices only reduce the damage but they cannot prevent yield and quality losses (Mielke 1988, Teich 1989, Milus and Parsons 1994). Thus, the development of cultivars with appropriate disease resistance is the most effective means of controlling *Fusarium* diseases.

The state of knowledge of *F. graminearum* and *F. culmorum* has been thoroughly reviewed with regard to symptomatology and epidemiology (Burgess et al. 1981, Cook 1981a, Sutton 1982, Jenkins et al. 1988, Teich 1989, Miller 1994, Parry et al. 1995), and toxicology (Marasas et al. 1984, Chelkowski 1989, Pomeranz et al. 1990). This review therefore concentrates on the genetic analysis of *F. culmorum* and *F. graminearum* populations, the genetics of host resistance, and breeding aspects in wheat and rye.

Genetic analysis of *Fusarium* populations

F. graminearum and *F. culmorum* are genetically closely related species. Comparisons of esterase patterns, serology, and estimation of relatedness from large subunit rRNA sequences of the two species have shown little or no difference between them (Szecsi et al. 1976, Hornok 1980, Guadet et al. 1989, Partridge 1991). DNA profiling by RAPDs (randomly amplified polymorphic DNA) detected characteristic differential banding patterns between the two species (Schilling et al. 1996).

Asexual and sexual variation

F. graminearum produces macroconidia and the perfect stage *G. zeae* can be found abundantly as blue-black perithecia developing on the affected host tissue early in the growing season (Sutton 1982, Jenkins et al. 1988). Sexual recombination generally results in increased genetic variation (Burdon 1993) that should allow *F. graminearum* populations to adapt rapidly to changing environmental conditions or, possibly, to host resistance genes (see Host specialization and isolate-by-host genotype interaction). Indeed, studies on vegetative compatibility groups (VCGs) of a natural *F. graminearum* population revealed great diversity of VCGs on the same wheat head, indicating that sexual recombination exists in the field (Bowden and Leslie 1994). Two distinct populations of *F. graminearum*, designated as Groups 1 and 2, were detected in Australia (Burgess et al. 1975) and the USA (Cook 1981a) with different sexual behaviour. Group 1 isolates are heterothallic and do not produce perithecia on carnation leaf agar (CLA) and only very rarely in nature, whereas the Group 2 population is homothallic and readily forms perithecia on CLA and in nature (Francis and Burgess 1977). Most recently, *F. graminearum* Group 1 has also been reported from northern Africa, Italy, and California (Burgess et al. 1996).

F. culmorum is an imperfect fungus with asexually-formed macroconidia. No sexual stage is known to date. However, somatic recombination through heterokaryosis (Puhalla 1981) could compensate for the lack of frequent sexual recombination. Moreover, parasexuality has been demonstrated in other *Fusarium* species (Puhalla 1981). At the population level, additional sources of pathogen variability such as mutation,

genetic drift, gene flow, and selection have to be considered (McDonald et al. 1989).

For more than half a century, a problem commonly discussed in *Fusarium* species is their high morphological variability in culture (Leonian 1929, Oswald 1949, Jones 1954, Burgess et al. 1994). A freshly isolated culture may remain stable for years or may gradually change its appearance during subculturing or may produce suddenly sectors of growth different from the wild type (Puhalla 1981). These effects could be caused by the *Fusarium* species, the genotype of the isolate, the cultural medium or other nongenetic factors. Similarly, physiological changes, especially a reduced aggressiveness on host plants, may occur frequently in agar cultures (Oswald 1949). To get reproducible results across years, variability in *Fusarium* stock cultures should be reduced by long-term storage methods such as maintenance on sterile soil (Miller 1945), in liquefied nitrogen or as lyophilized cultures (Burgess et al. 1994).

Aggressiveness and genetic structure of *Fusarium* populations

The most important character of *Fusarium* populations with regard to resistance selection is their aggressiveness, i.e. their capacity to cause disease in pathosystems with race-nonspecific resistance (Vanderplank 1984). Variation for aggressiveness was previously shown for a small number of individual isolates of *F. culmorum* and *F. graminearum* in greenhouse experiments (Johnston and Greaney 1942, Mesterházy 1981, 1984) and in the field (Snijders and van Eeuwijk 1991, van Eeuwijk et al. 1995). For *F. culmorum* causing head blight of winter rye, significant genotypic variation for aggressiveness was found in field experiments among 42 isolates collected from nine European countries and Australia (Miedaner et al. 1996a). Despite different mean disease ratings across five environments (location-year combinations), the correlations between environments were close ($r = 0.69-0.77$, $P = 0.01$). The broad-sense heritability was high ($h^2 = 0.92$) and the frequency distribution of aggressiveness was clearly of a quantitative nature. These findings indicate that a substantial amount of phenotypic variation for aggressiveness is caused by genetic effects and that aggressiveness is inherited as a complex trait.

The broad genetic variation for aggressiveness reported for *F. culmorum* and *F. graminearum* isolates (Mesterházy 1984, Miedaner et al. 1996a) might be overestimated owing to the widely differing geographic origins and host sources of the isolates collected. However, the analysis of 21, 38 and 54 isolates of *F. graminearum* and *F. culmorum* populations collected from three naturally-infected fields in south Germany, also revealed significant genotypic variation for aggressiveness within each field (Miedaner and Schilling 1996). On average, 60% of the total variation for aggressiveness determined in *F. graminearum* and *F. culmorum* collections from different countries were to be found within one field population.

Molecular marker techniques developed recently for *F. culmorum* and *F. graminearum* populations (Ouellet and Seifert 1993, Nicholson et al. 1993, Schilling 1996) allow a detailed analysis of the variation within and between populations. For one of the above-mentioned *F. graminearum* populations, the high diversity in aggressiveness within one field could be confirmed by a polymerase chain reaction (PCR)-based molecular analysis of 70 hierarchically sampled isolates. Among these, 53 unique genotypes could be distinguished by 37 polymorphic RAPD markers (Schilling 1996). Genotypic diversity according to Stoddart and Taylor (1988) reached 63% of the maximum

possible value (G_{max}). Similarly high levels of 77% and 62% of G_{max} were determined for single-field populations of *Cryphonectria parasitica* (Milgroom et al. 1992) and *Mycosphaerella graminicola* (= *Septoria tritici*) (Boeger et al. 1993). In contrast, lower levels were reported for *Stagonospora* (= *Septoria*) *nodorum* ($G_{max} = 34\%$ and 20% , McDonald et al. 1994), *Rhynchosporium secalis* ($G_{max} = 21\%$, McDermott et al. 1989), and *Pyrenophora teres* ($G_{max} = 15\%$, Peever and Milgroom 1994), as revealed by various types of molecular markers. Obviously, these estimates are highly dependent on restrictions of technique and the individual population. For instance, Boeger et al. (1993) found the reported G_{max} of 62% in one Oregon population of *Mycosphaerella graminicola*, but in a Californian population of the same pathogen they estimated a G_{max} of 14%. Therefore, a geographically wider sample of *F. graminearum* populations should be investigated in future. Calculating genetic similarities for RAPDs between the isolates of the *F. graminearum* field population and a world-wide collection of 25 isolates of *F. graminearum* revealed close relations among them (Schilling 1996). This single field population, therefore, represents *F. graminearum* genotypes present over a wide geographical area. Moreover, no spatial structuring within the field population could be found. The variation detected at any of the nine sampling sites accounted for 85% of the total variance observed within the whole field (Schilling 1996). Thus, genetic variation among *F. graminearum* isolates occurs on an extremely small spatial scale. This was confirmed by VCG analyses within a Kansas field population of *F. graminearum* (Bowden and Leslie 1994). Among a total of 26 isolates, 19 different VCGs were identified.

Mycotoxin production

F. graminearum produces a variety of mycotoxins, namely non-macrocytic trichothecenes and the oestrogenic zearalenone (ZEA). Among these, deoxynivalenol (DON) and its derivatives 3-acetyl DON and 15-acetyl DON, ZEA and, in some parts of the world, also nivalenol (NIV) are most often encountered in wheat (Tanaka et al. 1988, Mirocha et al. 1989, Scott 1990). The co-occurrence of several of these mycotoxins has often been reported (Müller and Schwadorf 1993, Mirocha et al. 1994). The frequency of mycotoxin-producing isolates in natural populations seems to be high. Of 114 isolates of *F. graminearum* collected from soil or cereals on a world-wide basis, 95% and 89% were capable of producing DON and ZEA *in vitro*, respectively (Mirocha et al. 1989). The production of 15-acetyl DON is described as characteristic for North American and Mexican isolates, whereas *F. graminearum* isolates collected outside these areas form mainly 3-acetyl DON (Mirocha et al. 1989, Miller et al. 1991). However, other patterns can also be found in smaller percentages. Some isolates of *F. graminearum* also produce NIV, but they were found only rarely in the USA (Miller et al. 1991). They are more common in Japan (Ichinoe et al. 1983, Miller et al. 1991), but have also been detected in Hungary, Poland, and Italy. Although DON and NIV are chemically related, DON producers do not produce NIV and vice versa (Ichinoe et al. 1983). ZEA can be found in small-grain cereals, the appearance of large amounts appears to be associated predominantly with corn (Yoshizawa 1991). Out of 2403 samples of small-grain cereals analysed world-wide, about 20% were found to contain ZEA (Yoshizawa 1991). Group 1 and Group 2 isolates of *F. graminearum* produce similar mycotoxins with minor differences in the relative amounts of each (Blaney and Dodman 1988).

F. culmorum generally forms the same mycotoxins as *F. graminearum* (Marasas et al. 1984, Snijders 1990a, Atanassov et al. 1994). Its ability to produce NIV has only recently been detected (Atanassov et al. 1994, Mirocha et al. 1994, Gang 1997). In an experiment with 42 isolates of *F. culmorum* sampled from nine European countries and Australia and inoculated on winter rye heads, 34 isolates formed DON with a mean of 39 mg/kg across two locations. Seven other isolates produced NIV in concentrations ranging from 18 to 30 mg/kg (Gang 1997). Thus, mycotoxin production also seems to be a common feature among *F. culmorum* isolates.

For both *F. graminearum* and *F. culmorum*, the mycotoxins produced and the amount of mycotoxin production depends highly on the isolate investigated (Marasas et al. 1984, Miller et al. 1991, Atanassov et al. 1994). From the DON production of *F. culmorum* isolates determined under culture conditions *in vitro*, no prediction of the quantity produced with the same isolates in field inoculations was possible ($r = 0.25$, Gang 1997).

It has been suggested, that DON may contribute to the pathogenicity of *F. graminearum* and *F. culmorum*. It inhibits protein synthesis (Miller 1989) and growth of wheat coleoptile tissue and seedlings (Bruins et al. 1993). However, it appears that DON production may not be required for pathogenicity, i.e. the ability to cause disease, because *F. graminearum* isolates that were unable to produce DON and 3-acetyl DON *in vitro* were pathogenic on wheat, rye, and triticale seedlings (Manka et al. 1985). In agreement with this, from protoplast fusions between a pathogenic DON producer and a nonpathogenic DON nonproducer, *F. graminearum* isolates were regenerated that were DON nonproducers but highly pathogenic on plants and vice versa (Adams and Hart 1989). Proctor et al. (1995) described a trichothecene-deficient *F. graminearum* isolate, generated by gene disruption, that was still capable of causing disease symptoms. Thus, DON has no relation to pathogenicity, but might contribute to aggressiveness, i.e. the extent of fungal colonization within host tissue in the early stages of pathogenesis (Snijders and Krechting 1992).

Host specialization and isolate-by-host genotype interaction

Host specialization in both *F. culmorum* and *F. graminearum* is low. In a *F. culmorum* population of 42 isolates that originated from eight gramineous host species and soil habitats, all isolates were capable of infecting winter rye heads (Miedaner et al. 1996a). In *F. graminearum*, the two established subgroups (Group 1 and 2) showed different pathogenic behaviour on wheat and maize (Francis and Burgess 1977). For *F. graminearum* isolates of Group 2 no specialization among various hosts could be established (Cook 1981a).

Knowledge of the significance of specific interactions between isolates and host genotypes is crucial for resistance testing and the prediction of durability of resistance. A significant interaction would suggest a vertical type of resistance with the possibility for the pathogen population to adapt rapidly to resistance genes of the host (Vanderplank 1984). In consequence, the ranking order of host genotypes according to their resistance would depend on which isolate of the pathogen is used for testing. In several experiments such interactions have been reported previously for *F. graminearum* and *F. culmorum* isolates in head blight of wheat (Mesterházy 1978, 1983, 1987, Snijders and van Eeuwijk 1991). However, interaction patterns were not stable over experiments and years, and host genotype ranking was only slightly influenced by the isolates. In these

studies, scaling effects most probably biased the estimate of isolate-by-host genotype interaction because the aggressiveness of the tested isolates differed highly (Snijders and van Eeuwijk 1991). A collaborative study across five European countries with 25 winter wheat genotypes and a range of isolates indicated no isolate-specific resistance (van Eeuwijk et al. 1995). No significant ($P > 0.1$) isolate-by-host genotype interaction was found in a head-blight test with seven *F. culmorum* isolates and seven winter rye inbred lines across five environments (Miedaner et al. 1996b). While the isolates were selected for similar aggressiveness to avoid the scaling effects mentioned, the rye lines showed a significantly different susceptibility.

In conclusion, host resistance to *F. culmorum* or *F. graminearum* is not influenced by the fungal isolate. The use of only one aggressive isolate might, therefore, suffice for resistance screening. However, natural field populations of *F. graminearum* and *F. culmorum* showed high genetic diversity for aggressiveness (Miedaner and Schilling 1996). The use of a mixture of several isolates with known aggressiveness of the individual isolates might improve environmental stability of artificial inoculation. Mesterházy (1988) proposed the use of several individual isolates with different levels of aggressiveness in a factorial manner for resistance testing.

Summarizing these data, it is evident that *F. culmorum* and *F. graminearum* populations show a great range of morphological variation, a large genetic diversity for aggressiveness, mycotoxin production, and molecular markers, a low level of pathogenic specialization, and no distinct isolate-by-host genotype interaction.

Inoculation techniques and assessment of resistance

In general, efficient selection for quantitative resistance requires a test with a reproducible inoculation method, high repeatability, and quantitative disease assessment. If the test is conducted under controlled or semi-controlled environmental conditions, the results must correspond to field data (see Association of resistance with host growth stage and host organ). Since a resistance test should also be transferable to practical breeding programmes, it has to be simple in handling, as cheap as possible, rapid, and applicable to large plant populations.

Seedling resistance

A variety of tests under controlled environmental conditions have been developed for screening seedling resistance to *F. culmorum* and *F. graminearum* Group 2. The inoculation methods are principally as follows: direct inoculation of seed or seedlings (Colhoun 1970, Mesterházy 1978, Duben and Fehrmann 1979b, Miedaner 1986), spreading inoculum on to the substrate (Mielke 1988, Höxter et al. 1992), inoculation of a special layer within the substrate (Baltzer 1930, Statler and Darlington 1972), inoculation of the whole substrate (Mishra and Braun 1976, Piglionica 1977, Mesterházy 1978, Miedaner 1988), and spraying plants with spore suspensions (Mishra 1971). With most of these inoculation methods, high disease severity and significant genotypic differentiation was achievable (Mishra 1971, Miedaner 1986). Since *F. culmorum* and *F. graminearum* are most destructive to wheat plants at higher temperatures and low soil water content (Colhoun et al. 1968, Cook 1981b), some authors prefer resistance testing under such stress conditions. Mesterházy (1978) established a seedling test in Petri dishes at 25°C and Piglionica (1977) incubated inoculated

durum wheat seedlings under severe drought. However, methods combining *Fusarium* inoculation with abiotic stress factors mix different plant responses that are genetically unrelated. This should provoke genotype-by-stress factor interaction, thus reducing selection efficiency for *Fusarium* resistance alleles. Unless selection is warranted for special stress situations, such as for dryland crown rot, testing of resistance should be performed under good growing conditions for both host and pathogen. Field tests of *Fusarium* seedling blight can be performed by dressing the seed with a spore suspension (Greaney et al. 1938).

For assessing *Fusarium* seedling resistance in the greenhouse, a number of methods have been used including counting the number of surviving tillers per plant or number of surviving plants, disease severity ratings, measuring fresh and dry matter of whole plants, or roots and shoots separately, or length of roots and shoots. All these traits may detect genotypic differences. It should, however, be noted that counting the number of surviving tillers or plants is a qualitative measure (dead/alive) superimposed on a quantitative trait and should, therefore, not be used.

Crown rot resistance

Natural infections of wheat by *F. graminearum* Group 1 occur mainly under low soil water potentials (Cook 1981b, Liddell and Burgess 1988). This, and the attack from below-ground inoculum, has to be simulated for successful greenhouse tests (Liddell and Burgess 1988). Additionally, to avoid seedling death the seed should not be in direct contact with the inoculum. In a test published by Wildermuth and McNamara (1994) plants are grown in steam-air treated soil with 37.5% water capacity (-0.1 bar) inoculated with colonized wheat-barley grain placed in a thin layer between the seed and the soil surface. The coleoptiles will be infected when they grow through this inoculum layer. After 22 days at 25°C, the extent of necrosis in the first three leaf sheaths is rated separately for each leaf sheath on a 0-4 scale and the values are added up. Significant genotypic differences could be detected among wheat genotypes with this test.

In the field, crown rot symptoms are artificially most accurately produced by sowing seed dusted with benomyl into furrows along which *F. graminearum*-colonized grain is distributed (Dodman and Wildermuth 1987). With a special planting machine, inoculum can be placed mechanically around the seed so that it does not have direct contact with the seed (McNamara and Wildermuth 1996). This method most probably resembles natural infections, because inoculum of *F. graminearum* Group 1 in the field is mainly located in infested residues from previous crops (Wearing and Burgess 1977). The test was found to result in more reliable and higher disease severities than the seed inoculation method proposed by Purss (1966). The most reliable measure for disease assessment was by rating the proportion of discoloured tillers at maturity (Dodman and Wildermuth 1987).

Foot rot resistance

Two of the methods mentioned for seedling infection tests, spraying spore suspensions and spreading inoculum on to the substrate, are also capable of inoculating plants at later growth stages in the greenhouse. Höxter et al. (1992) mixed colonized, crushed wheat grain medium with vermiculite (10:90, v/v) and maintained a high humidity at the basal parts of plants with filter paper soaked with water. In this system, infections at the

basal parts of winter rye became visible within four weeks at temperatures of 18/16°C (day/night).

For artificially inducing brown foot rot in the field, the spread of cereal grain colonized by *Fusarium* spp. directly on to the plants during the period from third-leaf to mid-tillering stage has been used successfully (Bockmann 1962, Mielke 1988). The inoculum is prepared in glass flasks on water-soaked whole oat or wheat kernels where the fungus is incubated for 4-6 weeks. Afterwards, the inoculum is dried and can be stored in the refrigerator (at about 2°C) until needed. By crushing the inoculum in a mill (sieve size 0.5-1 mm), a better distribution on to the plants can be obtained (Höxter et al. 1992) and the depletion of inoculum by rodents and birds should be hindered. Alternatively, conidia suspensions can be sprayed on to the crop at early growth stages (Mielke 1988). In winter rye, both methods resulted in similar mean disease ratings, but the application of conidia suspensions revealed a better genotypic differentiation in a 1-year experiment (Höxter et al. 1992).

Assessment of foot rot resistance in the field is hindered by multiple infections of the stems by different fungi (*Fusarium* spp., *Pseudocercospora herpotrichoides*, *Microdochium nivale*). The composition of the pathogen population depends greatly on years and locations (Duben and Fehrmann 1979a, Wegener and Wolf 1995). The differentiation of pathogens solely by disease symptoms proved to be unsuitable, because only early infections with *P. herpotrichoides* cause typical lesions ('eyespot'). Inoculation with *Fusarium* spp. enhances the frequency of that pathogen, although *P. herpotrichoides* may occur simultaneously in considerable amounts (Miedaner et al. 1993b). Similarly, infections of the same stems by several species were observed at high frequencies in wheat and rye (Duben and Fehrmann 1979a, Miedaner et al. 1993b). Therefore, the effects of different foot rot pathogens on host genotypes cannot be separated by visual disease assessment in field experiments. A combined analysis across environments merely reflects the host resistance to the whole pathogen complex.

Disease severity of adult plants grown in the field is mainly assessed between milk and full-ripening stage, or after harvest, by visually scoring 50-100 randomly chosen stems according to their lesion size and/or lesion severity (Bockmann 1963, Mielke 1988). The optimal date for sampling is the milk-ripening stage when uninfected stems are still green. In later stages, foot rot pathogens show extensive saprophytic growth within the stems and this is only weakly correlated to host resistance at the milk-ripening stage for *F. culmorum* (Miedaner et al. 1995d) and *P. herpotrichoides* (Lind 1992). Optimal sample size for winter rye, estimated on the basis of six environments and three replications, was 10-25 stems per micro-plot (0.6-1.0 m²) for genetically homogeneous material (inbred lines, single-cross hybrids, Miedaner et al. 1995a). The sample size is highly dependent on the amount of genetic variance within entries and for heterogeneous rye materials, therefore, greater sample sizes could be necessary. Resistance evaluation by rating lesion severity, estimates lesion size and the amount of weakening of the stems in one score (Miedaner et al. 1995a). This takes into account that the severity of foot infections depends less on the total area affected (lesion size) than on the depth of penetration of the lesions at the stem (Fitt et al. 1988).

Alternatively, resistance can be estimated by measuring *Fusarium* protein in rye by a recently developed indirect enzyme-linked immunosorbent assay (ELISA, Beyer et al. 1993). The ELISA detects protein of all *Fusarium* species tested but shows no significant cross-reaction with other foot-rot pathogens and

enables a quantitative, reproducible differentiation among genotypes with different levels of fungal infection (Beyer 1995). The correlation between ELISA absorbances and lesion severity ratings greatly depended on plant material and test environments. In pot experiments in the greenhouse, where the composition of the inoculum can be controlled, a close association between both traits was generally found ($r \approx 0.8$, Beyer 1995). In different field experiments in rye, however, correlations were inconsistent despite significant genotypic variance for both traits. When lesions were mainly caused by *Fusarium* species, as indicated by the percentage of reisolation on agar, coefficients of correlation ranged between 0.40 and 0.83 (Beyer 1995). In two field trials across two years where *P. herpotrichoides* was also present in considerable proportions (15–67%), the correlations between lesion severity rating and ELISA absorbances ranged from –0.21 to 0.57, being significant in one out of the four experiments only (T. Miedaner, unpublished data). Most probably, necrotic lesions caused by *P. herpotrichoides* were included in the lesion severity rating but not in the ELISA because of its specificity to *Fusarium* species. This is especially a problem when late infections with *P. herpotrichoides* occur and the characteristic eyespot symptoms do not show up as frequently. The correlation could also be biased by the colonization of the stem by nonpathogenic *Fusarium* species detected by the ELISA but not resulting in lesions. However, this factor should have been of less importance in these experiments, because all samples were taken at milk-ripening when the stems were still green. In addition, tolerance mechanisms might lead to medium or even nonsignificant correlations between lesion severity rating and the amount of fungal mycelium assessed by ELISA. Such tolerances were detected in a rye inbred population of 50 lines (Beyer 1995). With about 10% of the host genotypes, the same ELISA absorbances represented significantly different lesion severity ratings and vice versa leading to a medium correlation between both traits ($r = 0.51$, $P = 0.01$) despite the absence of *P. herpotrichoides* in this experiment (Beyer 1995). In most experiments mentioned above, genotypic variance of lesion severity rating was considerably greater than that of ELISA absorbance. This might also affect the correlation between both traits.

Other methods of determining fungal biomass within host tissues developed for various plant pathogens, such as ergosterol content (Seitz et al. 1979) and protein-specific staining methods (Wolf and Fric 1981) have not been widely used for resistance testing with *Fusarium*. They are expensive and laborious and, thus, applicable only to small plant populations. Recently-developed methods based on molecular techniques, such as species-specific PCR-based markers (Schilling et al. 1996) are not yet capable of quantifying fungal growth within the host.

Summarizing these results, ELISA is an important tool for diagnostic and epidemiological analyses in disease complexes and in diseases where infections remain latent during a substantial period of host growth. For the evaluation of quantitative resistance to *Fusarium* spp., however, lesion severity rating seems to gain higher selection responses. Moreover, the species-nonspecific lesion severity rating better reflects the total pathogenic situation on locations than individual ELISA tests. Even when the resistances to different foot rot fungi should not be genetically correlated, selection by lesion severity rating across several environments with different fungal populations will most probably yield genotypes with complex foot rot resistances.

The rating of individual stems for lesion severity cannot be replaced by indirect characters such as rating of lodging or straw stability. No substantial correlations between these traits and lesion severity rating were found for *P. herpotrichoides* in wheat (Allan and Roberts 1991) and various foot rot pathogens in rye (Ludwig 1992). Ratings of lodging and straw stiffness are often confounded with effects of stem architecture, plant height, ear weight, or environmental factors, such as stand density, wind velocity and rainfall. Because foot rot resistance is only a part of the whole lodging complex, different components might be selected in different environments when rating lodging or straw stiffness alone. This will considerably reduce selection efficiency for foot rot resistance and might have been one reason for low selection efficiency in practical breeding programmes until recently.

Head blight resistance

Fusarium head infection can occur under natural conditions from heading to harvest when temperature and moisture are favourable (Parry et al. 1995). The use of natural epidemics for testing *Fusarium* head blight resistance is only effective in environments where the disease occurs regularly. In other areas, *Fusarium* head blight can easily be induced artificially by spraying conidia suspensions of 250 000–2 000 000 spores/ml during flowering on to the heads of wheat and rye (Mesterházy 1978, Mielke 1988, Miedaner et al. 1993a). Conidia can be produced in mass either on colonized wheat grain inoculum (Bockmann 1962) or suspension cultures ventilated by sterilized air (Mesterházy 1978). In areas with low humidity during inoculation, sprayed heads can be covered by polyethylene bags for 24–48 h (Mesterházy 1988). A mist irrigation device was used successfully to enhance infection frequency and disease severity (Snijders and Perkowski 1990, Miedaner et al. 1993a). Most authors agree that mid-anthesis is the most susceptible developmental stage with the greatest reduction of grain weight in wheat (Anderson 1948, Diehl 1984, Mielke 1988) and rye (Baltzer 1930, Gang 1997). Alternatively, inoculation with *F. graminearum* was performed by scattering colonized wheat kernels bearing perithecia in the field in spring (Zhou 1985) or by dripping spore suspensions into single spikelets and observing the rapidity of spread of disease (Schroeder and Christensen 1963, Zhang and Pan 1982). The last technique allows the separation of different resistance components, because the pathogen spread within the head (type 2 resistance, see Components of resistance) can be analysed without considering the resistance to initial penetration.

To assess head blight resistance in inoculated field experiments, visual scoring of the symptoms on a whole-plot basis and determination of yield or yield components relative to the non-inoculated treatment are usually performed in wheat and rye. The symptoms of *F. culmorum* and *F. graminearum* induced by artificial head infection during mid-anthesis are clearly visible as prematurely bleached spikelets. Symptom development starts 5–20 days after inoculation and progresses subsequently until the end of the milk-ripening stage (Miedaner et al. 1995b, Parry et al. 1995). Afterwards, the epidemic can no longer be followed visually because the heads turn yellow. The length of the incubation period, the slope of the disease progress curve and the terminal disease rating are dependent on weather conditions and are, therefore, highly variable among environments (Parry et al. 1995, Gang 1997). Thus, the date of optimal genotypic differentiation in resistance evaluation trials may differ

greatly between environments and several ratings should be collected during the epidemic. Most authors assess the proportion of bleached spikelets per plot on a 1–4 (Mesterházy 1978) or 1–9 scale (Diehl 1984, Mielke 1988). Alternatively, head blight rating as the product of the percentage of number of heads infected and the proportion of bleached spikelets per infected head have been calculated to improve genotypic differentiation (Snijders and Perkowski 1990). For the same reason, Miedaner et al. (1993a) proposed the use of an average head blight rating, which is the arithmetic mean of all individual disease ratings with significant genotypic variance. The area under the disease progress curve (AUDPC) was also proposed for disease assessment (Bai et al. 1993). However, because *Fusarium* head blight is a monocyclic disease, the AUDPC is highly correlated with a single rating date (Snijders 1990c). Snijders and Krechting (1992) reported a good correlation between visual disease assessment and ergosterol analysis among 22 wheat genotypes inoculated with *F. culmorum* in a 1-year experiment.

Grain weight per head, 1000-grain weight and number of kernels relative to the non-inoculated treatment can be used to assess yield reduction caused by *Fusarium* infection. Since severe *Fusarium* infections reduce both 1000-grain weight and the number of kernels, grain weight per head was found to provide the best genotypic differentiation in wheat and rye (Miedaner and Walther 1987, Miedaner et al. 1993a, van Eeuwijk et al. 1995). Reduction in kernel number occurs mostly at high infection levels and/or with highly susceptible genotypes. Thus, genetic variance across different levels of infection is smaller for this yield component than for relative 1000-grain weight or relative grain weight (Miedaner and Walther 1987, Snijders 1990f, Miedaner et al. 1993a). Correlations between head blight rating and relative grain weight ranged from –0.47 to –0.96 in wheat (Mesterházy 1987, Miedaner and Walther 1987, Snijders 1990f, van Eeuwijk et al. 1995) and from –0.43 to –0.81 in rye (Miedaner et al. 1993a, 1995b,c), depending in the latter crop on the heterogeneity and heterozygosity of the materials tested. These correlations indicate that head blight rating will result mostly in a similar ranking of genotypes as the determination of relative grain weight. In most cases, the latter trait exhibited higher error variances and/or lower heritability estimates than head blight rating (Snijders 1990f, Miedaner et al. 1993a, 1995c, Gang 1997). This may be caused by a nonuniform plant density in plots (Mesterházy 1983) or a generally higher genotype-by-environment interaction variance of yield components. For evaluating relative grain weight or its components twice the number of plots (noninoculated versus inoculated) are required than is necessary for head blight rating. Hence, rating *Fusarium* head blight according to visual symptoms is the most sensitive and reliable resistance trait, especially if large host populations have to be screened (Snijders 1990f, Miedaner et al. 1993a, Mesterházy 1995a). In wheat, rating of the harvested kernels for visible *Fusarium* symptoms, such as shrivelling or discoloration, has also been found useful for resistance selection (Mesterházy 1995a).

Reproducibility of head blight infections across environments and differentiation of host genotypes greatly depend on nongenetic effects (Table 1). The host resistance of winter rye is highly influenced by genotype-by-environment interactions as previously reported in wheat (Mesterházy 1987, 1995a, Snijders and van Eeuwijk 1991). The genotypic differentiation was found to be influenced significantly by the head-developmental stage (Table 1). When spore suspensions were sprayed at mid-heading, heading, and mid-anthesis stages, and 4 days after mid-

anthesis on to winter rye heads, coefficients of phenotypic correlation among these various stages ranged from 0.13 to 0.56 with no significance ($P > 0.1$) in all instances (Gang 1997). To compensate this interaction in artificial inoculation experiments, all genotypes should be inoculated at exactly the same host growth stage, preferably mid-anthesis.

The effect of the microclimate was investigated in the field by (1) using a mist irrigation device during incubation period, (2) bagging the heads for 24–48 h, and (3) using no additional device. Treatment 1 primarily enhances humidity, whereas treatment 2 improves both humidity and temperature. The effect of these treatments was highly significant in all individual years leading to an interaction with host genotype that accounted for 27–45% of host genotypic variance in winter rye (Table 1). Phenotypic correlations among the three treatments ranged from 0.76 to 0.80 ($P = 0.01$). Thus, host genotype differentiation was influenced less by microclimate than by the head-developmental stage. Similarly, Mesterházy (1988) reported only slight effects of the duration of bagging (24 h versus 48 h) on the ranking of wheat genotypes. In the field, however, humidity and temperature can be manipulated only to a limited extent. The weather conditions varying from year to year had the highest impact on host differentiation as indicated by the significant host genotype-by-year interaction variance that accounted for 76% of the host genotypic variance in the microclimate experiment (Gang 1997). The effects of humidity and temperature on host differentiation could be better analysed in air-conditioned greenhouse cabins (Table 1). Twelve inbred lines were inoculated with or without covering with polyethylene foil for 48 h after inoculation and incubated until head blight rating under cool (18/16°C day/night) or warm conditions (24/20°C day/night). Humidity in the first 48 h after inoculation had the greatest impact on host differentiation (Table 1), although temperature also showed a significant interaction with the genotypes.

In the field, each genotype should be inoculated at its respective flowering time. The inoculation dates vary, therefore, with possibly different weather conditions at each date. The actual effect of this factor depends greatly on the stability of the weather conditions during flowering and thus varies from year to year in humid climates. This may additionally enhance host genotype-by-environment interaction, especially when testing genotypes that show a great variation for flowering date, e.g. F_2 populations, exotic breeding stocks.

Association of resistance with host growth stage and host organ

Diseases caused by *F. culmorum* and *F. graminearum* may occur in all growth stages of cereals and on all plant organs. For resistance breeding, it is of crucial importance to know if the resistances of various host growth stages and organs are associated. In this case, resistance breeding would be greatly simplified by a correlated selection gain between the respective plant growth stages or plant organs. Moreover, the resistance level of breeding populations could then be predicted in early growth stages where the test systems are cheaper, and could be done in an off-season programme in greenhouse or growth room pot experiments. Therefore, various attempts have been made to estimate the correlation between resistance in young-plant stages and several *Fusarium* diseases in adult-plant stages (Table 2). All reports revealed significant genetic variation in the growth stages tested. However, seedling resistance to *F. cul-*

Table 1: Interaction between host genotype (H) and various nongenetic factors (F) measured as relative variance component estimate (σ^2 , relative to variance component estimate of host genotype (= 100)) and heritability (h^2 , entry-mean basis) for head blight rating in different winter rye experiments inoculated with *Fusarium culmorum* (for description of factors see text)

Environment	Factors analysed (F)	Factors	Number of		σ_H^2	$\sigma_{H \times F}^2$	h^2
			Environments	Host genotypes (H)			
Field	Environment	—	5	7	100	47	0.91
	Head development stage	4	4	12	100	28	0.86
	Microclimate	3	3	20	100	27–45 ¹	0.73
Greenhouse	Humidity	2	1	12	100	102	—
	Temperature	2	1	12	100	30	—

All estimates of variance components were significant at probability level $P = 0.05$

¹Range of $\sigma_{H \times F}^2$ across 3 environments

Table 2: Correlation studies between young-plant resistance assessed in the greenhouse and different adult-plant resistances for *Fusarium graminearum* Groups (Gr.) 1 and 2 or *F. culmorum* in wheat

Correlation of young-plant infection to	<i>Fusarium</i> species	Number of entries	Correlation	Reference
Crown rot	<i>F. graminearum</i> Gr. 1	10	None ¹	Purss (1966)
	<i>F. graminearum</i> Gr. 1	8	— ²	Klein et al. (1985)
	<i>F. graminearum</i> Gr. 1	9	None ¹	van Wyk et al. (1988)
	<i>F. graminearum</i> Gr. 1	28	0.78	Wildermuth and McNamara (1994)
Foot rot	<i>F. culmorum</i>	192	None ¹	Piglionica (1977)
Head blight	<i>F. culmorum</i>	49	0.05	Miedaner (1986)
	<i>F. graminearum</i> Gr. 2	218	0.22–0.48	Mesterházy (1987)
	<i>F. graminearum</i> Gr. 2	10	None ¹	Woodward and Wilcoxson (1991)

¹No coefficient of correlation was given in the respective paper and obviously no correlation was found judging by the reported disease severities of the genotypes tested for the respective growth stages

²The resistance of six out of eight cultivars agreed with the field test

morum or *F. graminearum* Group 2 assessed in wheat under controlled environmental conditions was found to be only weakly or not correlated at all with adult-plant resistance (Table 2). For *F. graminearum* Group 1 the reports are contradictory. Purss (1966) and van Wyk et al. (1988) found no obvious correlation. Klein et al. (1985) reported an association of seedling to adult-plant resistance for six out of eight cultivars tested; the other two cultivars showing different ranking in both growth stages. Wildermuth and McNamara (1994) found a considerably greater correlation when testing 28 genotypes with a newly developed method. In all studies, the effect of different test environments (greenhouse versus field) is confounded with different growth stages (seedling versus adult-plant stages) and/or plant organs (shoot, leaf versus root, crown, foot, head). Moreover, the seedling tests are often biased by infection with seed-borne *Fusarium* species that cannot be fully controlled by seed disinfection (Piglionica 1977).

To estimate the correlations between *Fusarium* resistances in different host growth stages unbiased by the factors mentioned, several approaches have been used in winter rye. Höxter et al. (1992) compared the resistances at first leaf, second/third leaf, start of tillering, advanced tillering, and jointing stage in a greenhouse experiment with 10 inbred lines by artificial inoculation of basal plant parts with *F. culmorum*. Despite significant genotypic variation within each growth stage, the correlation coefficients averaged 0.38. Associations were closer among some early growth stages ($r = 0.61$ – 0.71). Correlation with field data was significant only when the plants in the greenhouse were inoculated at the jointing stage. To test whether *Fusarium*

resistances are also growth-stage specific in the field, Miedaner et al. (1995d) inoculated 11 winter rye inbred lines in autumn and early spring with *F. culmorum*. Disease progress within the basal parts was followed at seven growth stages (third leaf, mid tillering, jointing, heading, flowering, milk ripening and full ripening) during 2 years by ELISA. Significant genotypic coefficients of correlation between growth stages were achieved only among the adult-plant stages heading, flowering and milk ripening. No correlation was found between young-plant and adult-plant stages.

A highly specific host reaction was detected when resistances to foot rot and head blight were evaluated in the adult-plant stage in the field. No correlation was found between the resistances of both host organs among 186 full-sib families (Miedaner et al. 1995c) and 20 inbred lines for disease ratings (Miedaner et al. 1997). For the inbred lines, the content of *Fusarium*-specific proteins within the host tissue was also determined by ELISA. This did not improve the correlation between both host organs, although foot rot ratings correlated significantly with ELISA absorbances (Miedaner et al. 1997). These findings illustrate that interactions between the inoculated *Fusarium* isolates and naturally occurring foot-rot pathogens were not responsible for the lack of correlation with head blight resistance. They are in agreement with greenhouse data in wheat, where also no correlation between *F. culmorum* foot rot and head blight was found among 12 genotypes (Snijders 1990b).

Summarizing these data, most studies indicated strong interactions between host genotypes and plant growth stages and host genotypes and plant organs, respectively. An exception

might be crown rot resistance in wheat where a young-plant test seems to correlate moderately with adult-plant resistance in the field. It is evident that in all other diseases the resistance of wheat and rye to *Fusarium* spp. is most probably caused by different resistance mechanisms in different growth stages and plant organs of the host. As a consequence, selection for adult-plant resistance cannot be done in early growth stages with the tests reported, the resistance of each plant organ has to be selected separately.

Components of resistance

The mechanisms of resistance to *Fusarium* diseases are complex and yet not fully understood. Most probably, an array of factors is responsible for reduced susceptibility, including morphological, physiological and biochemical effects, expressing passive or active defence reactions by the host. Apparently, the pathogen itself and any one of these host resistance factors are greatly influenced by environmental conditions, thus further complicating the analysis of resistance.

Thorough investigations on the physiological causes of resistance to *Fusarium* brown foot rot are not known to the author. For crown rot resistance in wheat, it has been shown recently that crown depth may be one factor of resistance (Wildermuth et al. 1996). Between crown depth of noninoculated seedlings and relative crown rot susceptibility a correlation coefficient of 0.62 was found among 22 cultivars. Cook (1981a) reported that wheat genotypes with deeper roots have a greater diffusive resistance to vapour loss from the leaf or more efficient water use. This results in less damage to crown rot in the Pacific North-west of the USA, because they have a higher tolerance to drought.

Different mechanisms were detected that may contribute to resistance to *P. herpotrichoides* infections of wheat, such as special hypodermis structures, thickening and lignification of subepidermal and intervascular parenchyma cell walls (Murray and Bruehl 1983), induced thickening of epidermal cell walls in leaf sheaths of seedlings, lignification of parenchyma cell walls in stem tissue attacked, and localized appositions of cell wall material containing lignin (= lignitubers, Murray and Ye 1986). Murray and Bruehl (1983) reported that early lignification of the invaded cells enables less susceptible host genotypes to limit lesion development. Although, increased lignification can be induced by pathogen attack (Sherwood and Vance 1980, Lamb et al. 1989), it does not totally prevent the spread of the pathogen within infected tissue (Murray and Bruehl 1983) but might lead to the observed partial level of resistance. Induced lignin formation normally has only a low specificity for the attacking pathogen species (Ride 1980) and may, therefore, play a role in resistance to *Fusarium* also. The good agreement of foot rot resistance of winter rye inbred lines in different environments with highly differing proportions of *Fusarium* spp., *P. herpotrichoides* and *M. nivale* infections (Miedaner et al. 1995a) indicate that pathogen-nonspecific mechanisms could be involved in foot rot resistance in rye.

For *Fusarium* head blight of wheat, two components of resistance have already been described by Schroeder and Christensen (1963): (1) resistance against penetration, and (2) resistance against pathogen spread within the host tissue. Additionally, resistance against effects of DON and/or accumulation of DON (Miller and Arnison 1986, Snijders and Perkowski 1990), and resistance to seed infection (Mesterházy 1995a) have been reported. Resistance to penetration and to pathogen spread are

not genetically related and both resistance components greatly depend on host growth stage (Schroeder and Christensen 1963). Resistance to DON effects can result from either degradation of this mycotoxin by the host, increased membrane stability of the resistant host genotypes, and/or modified host receptor sites (Miller 1989). The first effect has been experimentally demonstrated for the resistant variety 'Frontana' in suspension cultures (Miller and Arnison 1986). The great range of DON:ergosterol ratios found in genotype screenings for head blight resistance is an indirect indication that such mechanisms might also be working in other genotypes (Miller et al. 1985). According to Snijders and Krechting (1992) DON is transported from the infection site on the glumes to the young kernel before the pathogen invades the seed. Resistance to seed infection was found among wheat genotypes that revealed the same disease severity, measured as relative grain weight, but significantly different seed-infection rates as shown by agar-plating techniques across environments (Mesterházy 1995a). Thus, resistance to seed infection can also be described as a special case of tolerance. Generally, tolerance is proposed when different resistance traits are not reacting similarly in some of the genotypes (Mesterházy 1995a). For example, five out of 25 genotypes differed significantly in relative grain weight in the field, although their head blight ratings were similar over 5 years (Mesterházy 1995a). The underlying mechanisms are not yet understood.

In addition to physiological resistance components, morphological characters of the host are also reported to affect resistance expression in natural epidemics. Mesterházy (1995a) provided experimental evidence that dwarfing (< 70 cm) and awning of wheat genotypes generally favour disease severity. Dwarf genotypes provide a shorter distance between soil and the lowest leaf and between flag leaf and spike. If *Fusarium* inoculum is splash-dispersed from the soil or the stem base, as suggested by Jenkinson and Parry (1994), the heads of dwarf entries should be exposed to a greater amount of primary inoculum than genotypes with normal straw length. Awning genotypes might accumulate more airborne spores due to a larger head surface and might preserve humidity from dew and rain over a longer period of time (Mesterházy 1995a). Within dwarf and awning genotypes, however, significant genotypic variation for *F. graminearum* head blight was found (Mesterházy 1995a), suggesting that the breeding of moderately-resistant dwarf or awning lines should be possible. Compact, erect heads were reported to be more susceptible than less dense, nodding heads (Mesterházy 1989). In artificial inoculation tests all these morphological characters showed no relationship to susceptibility (Mesterházy 1995a). In contrast to these reports, Bruehl (1967) and Love and Seitz (1987) could not identify any morphological characters that correlate with susceptibility in USA wheat material. Other morphological characters influencing head blight resistance might be related to flowering habit, e.g. length of flowering period, anther morphology, position and density of florets and length of time the flowers remain open (Schroeder and Christensen 1963). The presence of anthers after pollination was thought to enhance the initial attack by *Fusarium* spp. (Takegami 1957, Strange and Smith 1971). In agreement with this, choline and betaine compounds found within the anthers were shown to stimulate the growth of *F. graminearum* *in vitro* (Strange et al. 1978). Newer results indicate, however, that this nutritional effect may not be simply correlated with susceptibility, because anther extracts from the highly resistant

Japanese wheat cultivar 'Nobeoka Bozu' stimulated mycelium growth of *F. graminearum* significantly (Nkongolo et al. 1993).

Genetics of resistance and breeding strategies

The general aim of a breeding programme is to achieve a maximum selection gain for the character to be improved. The response of selection (R) will be given by $R = ih^2\sigma_p$, where i is the intensity of selection, h^2 the heritability, and σ_p the phenotypic standard deviation (Falconer 1989). The formula implies that efficiency of selection rises with high selection intensity, high intensity of testing (numbers of replications, environments, plot size), reduction of the experimental error (reproducible test system, favourable management practices, appropriate experimental design and optimal allocation), high genetic variance of the host populations, and small genotype-by-environment interaction variances (Sprague and Eberhard 1977). Estimates of the above-mentioned genetic parameters are required for developing appropriate breeding schemes with a high selection gain. It should, however, be noted that these parameters also depend on the populations, number of environments and number of replications used for testing (Falconer 1989).

Crown rot of wheat

McKnight and Hart (1966) and Purss (1966) were the first to report quantitative differences of resistance to crown rot. Among about 30 cultivars they found no source of complete resistance, but two cultivars ('Gala', 'Mengavi') with a reasonable level of field resistance. Several more were reported after screening some 400 additional cultivars, including 'Gluyas Early' and 'Mexico 234' (Wildermuth and Purss 1971, Wildermuth and McNamara 1994). The more resistant cultivars consistently showed a lower yield reduction, a lower proportion of discoloured tillers at the stem base and a lower number of white heads (Dodman et al. 1985).

The genetic basis of resistance was examined in diallel crosses between four resistant and four susceptible cultivars (R. L. Dodman and G. B. Wildermuth, unpublished, cited after Burgess et al. 1981). The data showed that several genes were responsible, with crown rot resistance being recessive. In a cross between the moderately resistant cultivar 'Gala' and a highly susceptible cultivar, transgression for resistance has been observed in the progenies (Dodman et al. 1985), indicating an additive oligogenic or polygenic inheritance of crown rot resistance. At Queensland, a backcross programme is managed for introgressing crown rot resistance into modern wheat varieties (G. B. Wildermuth, personal communication).

Foot rot of wheat

In contrast to crown rot, brown foot rot is caused by above-ground inoculum infection during the whole growth period, when the humidity in cereal stands is high enough (Cook 1981a). In addition to *F. culmorum* and *P. herpotrichoides*, *F. graminearum* Group 2 isolates were reported to cause such diseases of aerial plant parts (Burgess et al. 1981).

Genetic analysis of *Fusarium* foot rot resistance in wheat is scarce, most likely because resistance to strawbreaker, caused by *P. herpotrichoides*, has been given more attention by scientists and breeders in the humid climates. Indeed, *Fusarium* infections of the stem bases do not cause severe lodging in most years. In a study over 8 years with varying numbers of wheat cultivars ($n = 45-151$), a mean foot rot rating of 3.3 and a mean lodging score of 3.6 (1 = no lodging, 9 = full lodging) was

detected by artificial field infections (Mielke 1988). However, necrotic spots caused by *Fusarium* spp. were frequently observed in surveys of naturally infected wheat stems by re-isolation on agar (Duben and Fehrmann 1979a, Jenkins et al. 1988, Wegener and Wolf 1995). The role of *Fusarium* spp. as primary or secondary pathogens in this disease complex is still unclear.

Genetic variation for resistance to brown foot rot caused by *F. culmorum* was reported from a 2-year field study with 39 winter wheat varieties (Mielke 1988). Foot rot ratings ranged from 2.5 to 5.5 on a 1-9 scale in a continuous distribution.

Head blight of wheat

Large quantitative variation for head blight resistance in winter wheat was found for both *F. culmorum* and *F. graminearum* Group 2. Hanson et al. (1950) summarized the results of former USA evaluation trials across thousands of entries and reported that all genotypes became infected, i.e. no source of complete resistance was found. Moreover, with only few exceptions, most genotypes proved to be susceptible. Durum wheats were generally more susceptible than bread wheats and no resistance source for durum wheat was found. Similar conclusions were drawn from more recent tests (Walther 1976, Mesterházy 1987, 1989, Miedaner and Walther 1987, Mielke 1988, Tomasovic 1989, Snijders 1990f, Saur 1991, Bai and Shaner 1994). Distinct resistance sources for *F. graminearum*-incited head blight were reported from three origins: winter wheats from Eastern Europe, spring wheats from Japan and China (e.g. 'Nobeoka Bozu Komugi', 'Sumai 3', 'Ning' selections), and from Brazil (e.g. 'Frontana', 'Encruzilhada') (Schroeder and Christensen 1963, Mesterházy 1987). Snijders (1990f) confirmed resistance of some of these wheat materials for infections to *F. culmorum* and identified additional accessions from these gene pools.

Despite a high genotypic variance, genotype-by-environment interaction plays a major role in the wheat/*Fusarium* head blight pathosystem (Mesterházy 1987, 1989, 1995a). Therefore, correlations of host resistance to *F. graminearum* between years may vary considerably. In experiments over 6 years, Mesterházy (1995a) reported correlation coefficients between each of two years ranging from 0.19 to 0.81 for head blight rating and from 0.32 to 0.67 for relative grain weight. The stability of resistance expression over environments greatly depended on the resistance level of the genotypes studied. Highly resistant material in general showed less variation across environments than medium susceptible genotypes (Mesterházy 1995a). These data indicate, that tests across several environments are necessary to rank genotypes properly for their resistance to *Fusarium* head blight.

In quantitative-genetic experiments, a preponderance of additive variance for resistance to *F. graminearum* and *F. culmorum* has been found (Gu 1983, Bai et al. 1993, and Snijders 1990c,d, respectively). Accordingly, the mean head blight resistance of F_2 populations can be predicted by the resistance of the parental lines (Snijders 1990d,e). The only exceptions were found in progenies from crosses where one awned genotype was involved (Snijders 1990d). Within the nonadditive components of genetic variation, dominance was found most often but dominance expressed as heterosis for resistance was significant in some F_1 crosses only (Hanson et al. 1950, Tomasovic 1989, Snijders 1990c). The occurrence of epistatic effects of the additive-by-additive component was reported for only a minority of crosses (Snijders 1990d, Bai et al. 1993).

An estimate of the number of effective factors by generation

mean analysis was first published by Nakagawa (1955); he found three genes that controlled scab resistance. In more recent surveys, Bai and Xhiao (1989) and Bai et al. (1993) reported one to three genes responsible for resistance to *F. graminearum* head blight in Chinese materials. Snijders (1990d) found, on the basis of 45 crosses, that the number of effective factors varied from one to six for *F. culmorum* head blight. It should be noted that, from the theoretical point of view, all these estimates are only a rough indication of the true number of genes responsible for *Fusarium* head blight resistance. At least three assumptions cannot be fulfilled in the experiments: (1) equal gene action, (2) one parent supplies only positive, the other only negative alleles, and (3) equal degrees of dominance (Wright 1968). Moreover, in any one cross only a limited sample of genes contributes to segregation and, therefore, the real number of genes will most likely be underestimated (Geiger and Heun 1989). High genotype-by-environment interaction will also affect the estimation of gene number when the experiments are not conducted in several environments.

Monosomic analyses showed resistance genes to be assigned to two to five different wheat chromosomes (Yu 1982, Yu 1990). 'Sumai 3', one of the most important Chinese sources of resistance, for instance, had resistance genes on chromosomes 2A, 5A, 1B, 6D, and 7D (Yu 1982). In the future, analysis by molecular markers should enable the number of genes and their chromosomal location to be estimated more precisely.

Practical breeding comprises three phases: procuring initial variation, choosing varietal parents and testing experimental varieties (Schnell 1982). For initial variation, the results indicate that a large genotypic variance for head blight resistance exists within adapted breeding populations (Mielke 1988). The number of highly resistant accessions, however, is usually small (Mielke 1988, Bai and Shaner 1994). Therefore, foreign germplasm from Eastern Europe, China, and Brazil has been proposed as sources for resistance (Snijders 1990f, Mesterházy 1995a). Recently, selections from crosses of a Chinese ('Sumai 3') and a Japanese ('Nobeoka Bozu') resistance source with European wheats have been shown to yield highly resistant progenies (Mesterházy et al. 1994). However, such crosses introducing nonadapted genotypes require high input of time and labour for selecting progenies showing acceptable combinations of head blight resistance and other agronomic characters. Alternatively, the use of quantitative variation by recurrent selection (RS) should also be efficient due to the high amount of additive gene action found in this pathosystem. This was shown experimentally by Jiang et al. (1994) who reported a significant selection gain from three cycles (C) of RS for *F. graminearum* head blight resistance among Chinese populations. Starting with a highly susceptible population (C₀) that showed on average 50.8% of diseased spikelets, they were able to reduce this level by 9–10% per cycle. After the third cycle, the improved population had 27.7% diseased spikelets. For comparison, 'Sumai' 3, tested as a highly resistant check in the same experiment, had 15.7% infection. A second experiment resulted in a similar high selection gain. For *F. culmorum*, Snijders (1990e) found an average selection response of 3.7% for head blight rating for a single selection step in 25 F₂ families. In both studies, selection was directed solely towards *Fusarium* resistance and so, in practical breeding programmes where other agronomic traits are selected in parallel, the selection gain would be smaller. However, the rapid progress from these selection experiments supports the hypothesis that only a low to medium number of

effective factors for head blight resistance was segregating in the populations tested (Geiger and Heun 1989).

If populations improved by RS are used for line development, the resistance of the resulting lines should be superior compared with lines derived from unselected populations. In breeding programmes without introgressing exotic resistance sources or applying RS, potential crossing partners should at least be tested for *Fusarium* resistance across several environments. The use of crossing partners with at least moderate resistance levels might result in positive transgression for higher resistance during line development (Snijders 1990e).

The second phase for creating resistant varieties is the selection among segregating generations. A first selection in the F₂ generation uses the maximum of genetic variance and saves time and space in subsequent generations. However, in the F₂ generation, only a selection between single plants is possible which results in low heritabilities of head blight rating. Snijders (1990d) reported an average broad-sense heritability of 0.39 ranging from 0.05 to 0.89 among 23 F₂ families. The high probability of disease escape in single-plant selection (Snijders 1990e), the occurrence of competition effects between heterozygous single plants, the high variation in flowering date combined with an important genotype-by-head-developmental stage interaction will further restrict selection efficiency in the F₂ generation. Selection in the F₂ might nevertheless be beneficial when applied as negative selection of the 30–50% most susceptible entries.

In practical breeding programmes with special emphasis on head blight resistance a first selection is usually made in the F₃ or F₄ generation, which allows assessment on microplots. In breeding programmes with lower priority on *Fusarium* resistance, selection might be postponed until the F₅ or F₆ generation when the first tests across locations are possible. In the first selection step, head blight and/or kernel rating for disease symptoms could be used because of the high selection intensity feasible. In a later step, relative grain weight might also be analysed.

Foot rot of rye

Similarly to wheat, foot rot of rye is mostly present as a disease complex with several *Fusarium* species and *P. herpotrichoides* as predominant pathogens (Miedaner et al. 1993b). Depending on the environment, additional infections by *M. nivale* can frequently be found (Miedaner et al. 1993b).

Early studies of Baltzer (1930) and a more recent survey by Bojarczuk and Bojarczuk (1985) detected considerable genetic variation for *Fusarium* foot rot resistance among open-pollinated population varieties. Among full-sib families of the 'Petkus' population variety 'Halo' and also among various hybrid rye materials, significant genotypic variance was detected (Table 3). None of the genotypes tested displayed complete resistance. The relative importance of genotypic variance was greatest in inbred lines and considerably lower in single-cross hybrids or full-sib families. Compared with inbred lines, error variances in full-sib families were 4.5 times higher and, as a consequence, heritability estimates were low. This illustrates the influence of the genetic structure on evaluating resistance in the obligate outcrossing rye. Overall, genotype-by-environment interaction was important. Correlations among environments were high for 16 inbred lines (Miedaner et al. 1995a) but generally low for 186 full-sib families (Miedaner et al. 1995c), although testing intensity was comparable. Because the importance of genotype-by-environment interaction was similar for both materials

Table 3: Relative importance of genotype (σ_g^2), genotype-by-environment (σ_{ge}^2) and error (σ^2) variance components and heritabilities (h^2 , entry-mean basis) in different winter rye materials evaluated for foot rot and *Fusarium* head blight resistances

Plant material	Entries	Number of Environments	Variance components (%)			h^2	Reference
			σ_g^2	σ_{ge}^2	σ^2		
Foot rot resistance							
Full-sib families	186	5	23	19	59	0.54	Miedaner et al. (1995c)
Single-cross hybrids	42	6	47	30	23	0.82	T. Miedaner unpublished data
Inbred lines	16	6	65	23	13	0.92	Miedaner et al. (1995a)
Head blight resistance							
S_2^2 lines ¹	44	2	26	53	21	0.45	Miedaner et al. (1995b)
Three-way hybrids	88	2	29	31	40	0.53	Miedaner et al. (1995b)
Full-sib families	186	3	43	11	46	0.69	Miedaner et al. (1995c)
Single-cross hybrids	40	2	61	22	18	0.75	Miedaner and Geiger (1996)
Inbred lines	16	2	70	25	5	0.85	Miedaner et al. (1993a)

All estimates of genotype and genotype-by-environment interaction variance components were significant at probability level $P = 0.01$

¹ S_2^2 lines produced by selfing S_1 plants followed by one open pollination on isolation plots

(Table 3), this is most probably caused by differing error variances in individual environments masking the genotypic variances. To test this hypothesis, the correlation between each pair of environments was corrected for the experimental error on the respective environments by a formula described elsewhere (Miedaner et al. 1995c). These error-corrected correlations were considerably higher than the phenotypic correlations for the full-sib families. This illustrates that among heterogeneous entries a large degree of genotypic covariance between the environments might be masked by the high proportion of error variance on total variance. The influence of error variance is much smaller in homogeneous rye materials (Table 3).

For hybrid rye breeding, the partitioning of genetic variance into general and specific combining ability (GCA and SCA, respectively) is of utmost importance. Differences between 15 diallel and 11 factorial single crosses were caused mainly by GCA effects, with SCA being negligible (Miedaner et al. 1995a). The genotypic correlations between the resistance of the lines *per se* and their respective GCA effects were tight ($r \approx 0.9$, $P = 0.01$), also suggesting predominantly additive gene action.

The amount of genetic variance available in adapted source populations is crucial for improving foot rot resistance. In all the studies cited above, the absolute amount of genotypic variance was low within the 'Petkus' and 'Carsten' gene pools. Even among inbred entries, that should theoretically display the highest possible genotypic variance, this parameter resembled only 0.6 rating classes on the 1–9 scale. Therefore, it might be worthwhile to search for rye materials with greater genotypic variance. A strong preponderance of additive gene action and high heritability estimates offer good prospects for progress from recurrent selection (RS). RS can be performed with a full-sib family scheme (Geiger 1988) or on the basis of inbred generations (Wilde 1987). Full-sibs are produced by crossing pairs of vegetatively cloned single plants in the first year. In the following year, the testing of small plots will allow selection for *Fusarium* resistance at two to three locations. Subsequently, positively selected full-sibs are recombined in a factorial scheme. With this procedure, selection efficiency for foot rot resistance in breeding open-pollinated varieties should be improved.

Because self-incompatible populations are still used as base populations for inbred line development in hybrid rye breeding, RS within them will also result in a greater average resistance of the inbred lines derived from such improved populations (Sprague and Eberhardt 1977). The expected response to selec-

tion should be, however, lower in non-inbred compared with inbred materials because of the smaller genotypic variation and the lower heritability seen among full-sibs. On the other hand, the full-sib family scheme needs only 2 years per cycle, whereas selecting foot rot resistance in S_1 or S_2 generation will require 3 years or 5 years per cycle, respectively.

In hybrid breeding, a good agreement between the resistance of lines and their crosses allows selection among lines *per se*, i.e. without prior test-crossing. The decision in which generation selection for foot rot resistance should be started depends on the heritability of the trait under selection and the amount of labour necessary for its assessment (see formula above). Ludwig (1992) estimated heritabilities for lesion severity rating in the S_1 generation on one-row microplots ranging from 0.33 (one environment, one replicate) to 0.66 (three environments, two replicates). To reach this level of heritability at least 15–20 stems per plot should be individually scored. Because such a great amount of labour is required in the short period between the milk-ripening stage and planting, selection intensity will be reduced or costs for extra labour will increase in early, segregating generations where large populations have to be screened. Considering that in the S_1 generation highly heritable traits such as shortness, early maturity, lodging resistance, 1000-grain weight, and sprouting resistance could be assessed with a high selection gain, it seems advisable to postpone selection for quantitative foot rot resistance to the S_2 generation. Since no heterosis for resistance was found (Miedaner et al. 1995a), selection is necessary in both the seed-parent and the pollinator gene pool to achieve sufficiently resistant hybrids. Artificial inoculation is recommended to support a sufficiently high level of disease severity that allows genotypic differentiation in each of the environments tested.

Head blight of rye

For head blight resistance, significant quantitative variation was found in all material tested (Table 3). Heterogeneous entries such as S_2^2 lines (i.e. S_2 lines multiplied by one generation of open pollination in isolation plots), threeway-cross hybrids, and full-sib families lead to medium heritability estimates and to high genotype-by-environment or error variances. In contrast, homogeneous single-cross hybrids and inbred lines showed much greater genetic variances and higher heritabilities. Head blight rating was continuously distributed in all material. The relative importance of genotypic variance available in current

breeding populations seems to be higher for head blight than for foot rot resistance (Table 3).

In a study with 40 factorial single-cross interpool hybrids, genetic variation was mainly caused by GCA effects, indicating a prevalence of additive gene action (Miedaner and Geiger 1996). Although SCA variances were significant in 15–20% of all cross combinations in individual years, these nonadditive effects were inconsistent over years. The authors speculate that different inoculation dates even among cross combinations with one parent in common, which were necessary for inoculating each genotype exactly at its respective flowering time, might have contributed to these effects.

No significant line-by-tester interaction was found when 44 S_2^2 lines of the 'Carsten' gene pool were crossed to two genetically different testers of the opposite gene pool (Miedaner et al. 1995b) and evaluated for head blight rating and relative grain weight. This confirms a high amount of additive gene action. The correlation between the lines and their test-crosses, however, was not significant across 2 years for both resistance traits. In contrast, at the heterozygous level the correlation between both test-cross series was significant ($r = 0.58$, $P = 0.01$). This result might be explained by the high inbreeding depression found for *F. culmorum*-head blight resistance in this experiment. Moreover, among the S_2 population tested, the inbreeding coefficient should have varied considerably owing to the propagation of the inbred lines by open pollination in isolated plots and sampling effects of the S_1 gametes. All these factors related to inbreeding cannot be detected among the non-inbred test-cross progenies and will, therefore, prevent a correlation between the two genetic groups. As a consequence, the *Fusarium* resistance of hybrid rye materials has to be tested on the respective heterozygous level.

In practical breeding programmes, selection intensity for head blight resistance will be high, because inoculations are easy to handle even in large populations and disease assessment by head blight rating is inexpensive and powerful (see Inoculation techniques and assessment of resistance). Because inbred lines are more susceptible to *Fusarium* head blight than heterozygous material and seed infection reduces seed quality in hybrid rye production considerably, selection must be performed on both, the line and test-cross level. Selection for resistance among lines could be started in the S_1 generation due to the high selection intensity possible. On the heterozygous level, the test-crosses produced for selection of combining ability in yield can be used to select for head blight resistance. Usually, test-crossing starts on the B_1 (= backcross 1) level in the seed-parent gene pool and with S_2 lines in the pollinator gene pool (Geiger 1985). One generation later, selection for combining ability is repeated with greater testing intensity (more testers, more locations). Because the seed production of test-cross progenies is performed on isolated plots, enough seed will be available for testing head blight resistance simultaneously. Such a multi-step selection scheme would also meet with the requirements for testing in as many environments as possible due to the highly important genotype-by-environment interaction. Considering the significant correlations between two test-cross series for *F. culmorum* head blight resistance on the heterozygous level (Miedaner et al. 1995b), crossing to only a few tester genotypes will suffice. As in wheat, only head blight rating should be used for disease assessment in early generations. The rating of kernel discoloration and shrivelling as a measure of disease severity, as previously suggested in wheat is more difficult in rye (Perkowski et al. 1995) because of

its greater variability in seed shape and colour. In later inbred generations and/or among test-cross progenies relative grain weight might additionally be measured in a noninoculated and an inoculated treatment block.

Epidemiological and toxicological aspects of resistance to *Fusarium graminearum* and *F. culmorum*

Correlation between resistances to *Fusarium graminearum* and *F. culmorum*

Several attempts have been made to investigate the correlation between resistances to *F. graminearum* Group 2 and *F. culmorum* in different host growth stages. For young-plant and foot-rot resistance in winter rye, Höxter et al. (1992) tested 10 inbred lines for their resistance to both species in five growth stages from first leaf to the jointing stage in the greenhouse and found correlations ranging from 0.74 to 0.87 ($P = 0.01$) between the resistance to both *Fusarium* species. Similarly, close correlations were found between resistance to *F. graminearum* and *F. culmorum* foot rot at the milk-ripening stage in plastic houses among different rye materials ($r = 0.71$ and $r = 0.85$, Höxter et al. 1992 and Ludwig 1992, respectively).

For head blight resistance in winter wheat, the coefficient of phenotypic correlation between resistance to *F. culmorum* and *F. graminearum* was 0.90 ($P = 0.01$) when tested over 5 years (Mesterházy 1987, 1988). Scott and Benedikz (1986) inoculated eight winter wheat cultivars with *F. culmorum*, *F. graminearum*, *F. avenaceum*, *F. (= Microdochium) nivale*, and *F. poae* and found a similar genotype ranking for all *Fusarium* species. Similar results were achieved with inoculation of 25 wheat genotypes with either *F. culmorum*, *F. graminearum*, or *M. nivale* in a study across 18 environments (van Eeuwijk et al. 1995). In winter rye, a tight association between resistance to *F. culmorum* and *F. graminearum* head blight ($r = 0.96$, $P = 0.01$) was also reported (Miedaner et al. 1993a). Thus, the genetic basis of resistance to diseases caused by either *F. culmorum* or *F. graminearum* Group 2, and possibly by other *Fusarium* species, is most likely to be the same in all wheat and rye material tested.

Resistance to mycotoxin accumulation

Host genotypes suffering *Fusarium* head blight might accumulate several mycotoxins in their grains (see Mycotoxin Production earlier). Most commonly, DON and its metabolites were found on a world-wide basis (Snijders 1990a). In naturally infected grain, mean DON concentrations of wheat samples collected arbitrarily ranged from 0.03 mg/kg to 1.78 mg/kg with maximum values between 0.14 mg/kg and 8.53 mg/kg (Snijders 1990a). In a 5-year analysis of wheat in South-west Germany, Müller and Schwadorf (1993) found a mean DON content of 1.6 mg/kg, ranging from 0.004 mg/kg to 20.5 mg/kg. For rye, information about the natural occurrence of mycotoxins is scarce and restricted to small sample sizes. Amounts of 0.01–1.05 mg/kg DON, 0.02–0.04 mg/kg 3-acetyl DON, 0.01–0.04 mg/kg nivalenol and 0.01–0.05 mg/kg zearalenone are documented (Tanaka et al. 1988, Chelkowski 1989, Gareis et al. 1989, Scott 1990, Yoshizawa 1991).

In artificial *Fusarium* inoculations much higher DON concentrations have been reported in wheat (Mesterházy and Bartók 1993, Trissler 1993). Among 27 winter rye hybrids, DON contents ranging from 6 mg/kg to 27 mg/kg across 2 years were found (Miedaner and Perkowski 1996). Generally, DON concentrations appear to be lower in rye than in wheat or triticale when tested in the same experiment with comparable disease

Table 4: Range of correlation coefficients between various resistance traits and deoxynivalenol (DON) content across environments in wheat and rye

<i>Fusarium</i> species	Host species	Number of Genotypes	Number of Environments	Correlation between DON content and			Reference
				Head blight rating	Relative grain weight ¹	Other resistance traits	
<i>F. culmorum</i>	Wheat	54	3	0.43–0.62	–0.40 to –0.56	0.18–0.60 ²	Trissler (1993)
	Rye	27	2	0.01–0.47	–0.27 to –0.69	—	Miedaner and Perkowski (1996)
<i>F. graminearum</i>	Wheat	25	2	—	0.16 ³	0.74 ^{3,4}	Teich et al. (1987)
	Wheat	18	2	—	—	0.59–0.77 ⁵	Love and Seitz (1987)
	Wheat	54	3	0.57–0.86	–0.48 to –0.82	0.33–0.57 ²	Trissler (1993)
	Wheat	32	2	0.50–0.53	–0.55 to –0.62	0.70–0.76 ⁵	Mesterházy and Bartók (1993)

¹ Grain weight of inoculated relative to noninoculated plots

² Relative 1000-grain weight

³ No range across environments given

⁴ Percent of diseased heads per plot

⁵ Seed infection rate

severities (Miller et al. 1985, Kiecana et al. 1987). However, in both studies highly susceptible rye genotypes were not included. In a collaborative analysis of six wheat, six triticale and 12 rye genotypes, rye and triticale accumulated a comparable amount of DON, but wheat showed three times higher DON contents across two locations although mean disease severity in wheat was somewhat lower than in rye (Reinbrecht et al. 1996).

Because DON was also detected in healthy looking wheat (0.2–5.9 mg/kg) and rye (0.1–0.8 mg/kg) kernels (Perkowski et al. 1990, and 1995, respectively), and keeping in mind that mycotoxin analyses are expensive, an association between resistance traits and mycotoxin accumulation would greatly enhance progress in selection of less toxin-accumulating genotypes. Most studies found a low to medium correlation between resistance traits and DON content in inoculation experiments (Table 4). No tendency could be found for head blight rating, relative grain weight or 1000-grain weight to be better correlated with DON content. The disease incidence (% diseased heads per plot) and the seed infection rate appear to result in somewhat higher correlations to DON content. In most studies, the environment (location–year combination) strongly influenced the correlation. Only Mesterházy and Bartók (1993) reported environmentally stable correlations across 2 years.

Possible causes for only moderate correlations between resistance traits and DON accumulation might be rooted in the disease epidemiology. In years when disease is severe, or in highly susceptible genotypes, kernel number per head will be low due to loss of severely shrivelled grains in threshing or their early abortion in the head. In such cases, mycotoxin content of grain samples may be underestimated. In addition, when infections occur early, *F. culmorum* can invade the primary bundles in the head. The head sections above the infection site turn white owing to water and nutrient depletion without being colonized. Thus, mycotoxin contamination is unlikely to occur in those parts of the head. Moreover, symptom expression or yield reduction and mycelial growth and mycotoxin production might be influenced, at least partly, by different environmental conditions. This hypothesis is supported by the moderate correlation between head blight rating and ergosterol content of infected heads (Trissler 1993, Miedaner and Perkowski 1996). Moreover, the correlation between a resistance trait and mycotoxin content depends on the fungal isolate used, as reported for three and six individual *Fusarium* spp. isolates, respectively, by Snijders and Perkowski (1990) and Atanassov et al. (1994).

In general, highly resistant wheat genotypes such as ‘Sumai

3’, ‘Wuhan 37E-OY-OFC’, ‘Wuhan 10B-OY-OFC’ from China, ‘Nobeoka Bozu’ from Japan and material developed from these sources showed very low DON contents even under favourable epidemic conditions (Mirocha et al. 1994, Mesterházy 1995b, Mesterházy, personal communication). However, the level of resistance in most breeders’ materials is lower. In particular, moderate susceptible genotypes may show considerable deviation from the mean regression of resistance traits on DON content (Teich et al. 1987, Mesterházy et al. 1994).

Durability of *Fusarium* resistance

The success of resistant varieties in practice depends on the durability of the improved resistance. However, experimental results on this aspect are very limited for the wheat, rye/*Fusarium* pathosystems. Durability of resistance depends on pathogen variation, mechanisms and inheritance of resistance and agricultural management practices (Parlevliet 1993). Variation in natural *Fusarium* populations tends to be large, as revealed from morphological, physiological and marker data (Bowden and Leslie 1994, Miedaner and Schilling 1996, Schilling 1996). However, the occurrence of specific adaptation of certain *F. culmorum* or *F. graminearum* isolates to a host cultivar appears to be unlikely because (1) a low degree of pathogenic specialization was reported, (2) both *Fusarium* species are good saprophytes in soil habitats, (3) no consistent isolate-by-host genotype specificity was found in wheat and rye, and (4) host resistance was shown to be inherited quantitatively with no single genotype being completely resistant. Selection pressure on a *Fusarium* population, therefore, should be small. Even if a high level of quantitative resistance might cause a change in the composition of *Fusarium* populations in future, erosion of resistance would most likely be stepwise and slow, making adequate resistance available for acceptable periods of time. These theoretical considerations are supported by some experimental results of Mesterházy (1995a) who reported head blight resistance to be stable for one highly resistant genotype when tested across 16 environments. However, the ultimate test of durability of resistance is to grow the resistant variety over a longer period of time on a great acreage in areas where the disease occurs regularly (Johnson 1993).

Conclusions and further research needs

Substantial progress has been made in understanding the genetic basis of resistance to *F. graminearum* and *F. culmorum* in

small-grain cereals. Host resistance and pathogen aggressiveness were shown to be expressed on a continuous scale in the pathosystems analysed. Quantitative resistances are usually caused by the simultaneous segregation of several to many genes and diverse non-genetic factors (Geiger and Heun 1989). A low to medium number of effective factors was estimated for the control of *F. culmorum* and *F. graminearum* head blight in winter wheat by quantitative-genetic approaches. Despite significant genetic variation in wheat and rye populations for all *Fusarium* diseases assessed, progress from selection is restricted due to the high specificity of host resistance to macro-environment, microclimate, host growth stage, and host organ. Important interactions of the last two factors with host genotype are the main reasons that explain why indirect selection in the young-plant stage is not feasible for the prediction of resistance in adult-plant stages. Likewise, selection techniques using deoxynivalenol (DON) as selecting agent for populations of seedlings, coleoptile segments, callus derived from different sources, or isolated embryos did not show any correlation with head blight resistance (Bruins et al. 1993, Mesterházy et al. 1994).

For foot rot resistance, the interactions of the different pathogen taxa found within the same host tissue are not understood. *Fusarium* spp., *P. herpotrichoides*, and *M. nivale* are frequently isolated from the same necrotic lesions, however, the sequence of their attack and their significance for lesion severity may alter substantially among different environments. Thus, the relationship between host resistances to these different foot-rot-inducing fungi is of special interest for the breeder. Despite some attempts to investigate these interactions, no conclusive results have been found to date (Jenkins et al. 1988, Beyer 1995).

Taking all experimental data together, resistance breeding for *Fusarium* diseases is not limited by the lack of genetic variability but by the limited selection response. Thus, mapping resistance gene complexes by DNA markers should provide a solution to this problem. Analysis of quantitative trait loci (QTL) could be used to determine the number of genes involved, to assess gene action and interaction, to investigate the correlation between resistance and other agronomic traits, and finally to study the interactions with plant organs, plant growth stages and environments at the individual QTL level (Geiger et al. 1995). Molecular markers could be used to transfer important QTLs from exotic germplasm to adapted breeding material (marker-assisted backcrossing), or to select within progenies of crosses between susceptible and resistant genotypes (marker-assisted selection). However, the precision of mapping QTLs for *Fusarium* resistances will greatly depend on the heritability of resistance assessment, the number of effective factors, the distribution of QTLs across the genome (linkage) and the occurrence of nongenetic factors (van Ooijen 1992). In particular the great importance of host genotype-by-environment interaction and the association of host resistance with plant growth stage require a high experimental input (large number of environments, different inoculation treatments) for properly estimating the genotypic values needed for a precise QTL mapping of *Fusarium* resistances.

Doubled-haploid (DH) techniques might offer a further approach to enhance selection efficiency. DH lines derived from F_1 crosses are completely homozygous. They allow selection for *Fusarium* resistance in multi-environmental tests with a maximal genetic variance between homogeneous entries and, therefore, a precise estimation of the genotypic value. Because this is not possible for selection in segregating generations, DH

techniques would offer a perfect solution to select for quantitative resistant genotypes. A fast recurrent selection (RS) scheme could be achieved that would be especially advantageous for inbreeding crops (Foroughi-Wehr and Wenzel 1990). Genetically, an RS scheme based on the DH technique would be most advantageous when the resistance is mainly governed by recessive genes not closely linked to undesired agronomic traits, because the probability of recombination between closely linked genes is lower in DH steps than by subsequent selfing (Becker 1993). The inheritance studies showed a low importance of dominance for most of the pathosystems reported. A linkage to agronomically undesired traits most probably occurs when the resistance genes are being introgressed from exotic germplasm. Then, the occurrence of undesired linkages should be either experimentally tested or the first cycle(s) of RS should be done by single-seed descent. A maximal selection gain would be achievable if DH techniques could be combined with efficient marker-assisted selection. A reliable selection for *Fusarium* resistances would then be possible directly with DNA from the regenerated single plants. Although the DH technique can be used successfully in wheat, this is not yet feasible for winter rye owing to the low regeneration rate in adapted breeding material (Flehinghaus-Roux et al. 1995).

Another subject of interest for the breeder is whether reduced susceptibility of the host genotypes to *Fusarium* head blight will necessarily result in a correlated reduction of the mycotoxin content in the grains. This depends on the correlation between resistance traits and mycotoxin contents which were found to be medium for DON only. Moreover, the number and relative importance of different mycotoxins should also be considered in future studies. Although DON is reported to be the most prevalent mycotoxin in *F. culmorum* and *F. graminearum* infections of small-grain cereals, seven out of 42 *F. culmorum* isolates tested were capable of producing high levels of nivalenol on a susceptible winter rye genotype (Gang 1997). In addition, *F. graminearum* isolates can secrete high amounts of zearalenone (Marasas et al. 1984). Considering the great importance of mycotoxin contamination for animal and human health, the interactions between *Fusarium* isolates, host genotypes, mycotoxin accumulation and environment should be analysed in more detail.

The most serious lack of knowledge in resistance genetics of *Fusarium* diseases concerns the causes of host resistance and pathogen aggressiveness. For all *Fusarium* diseases in small-grain cereals, less susceptible host genotypes can be identified, but little is known about the molecular or physiological basis of the resistance mechanisms. The relative contribution of pre-infection mechanical barriers or postinfection host defences is also unknown. Similarly, the role of the mycotoxins in pathogenesis is still not clear. Does a highly aggressive isolate cause more disease because it produces more toxin, or does it produce more toxin because it causes more disease (Yoder 1981)? This could only be answered if the kinetics of mycotoxin production during the very early processes of pathogenesis are monitored by highly sensitive assays. In addition, the existence and possible role of other factors that may be responsible for aggressiveness, such as cell-wall degrading enzymes, hormones, or specific metabolites altering a host's resistance reaction, are not known. All these questions do not substantially impede selection efficiency for *Fusarium* resistance but their answers would greatly contribute to our understanding of the fascinating cereal-*Fusarium* interaction and may offer new approaches for resistance breeding.

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References

- Adams, G. C., and L. P. Hart, 1989: The role of deoxynivalenol and 15-acetyldeoxynivalenol in pathogenesis of *Gibberella zeae*, as elucidated through protoplast fusions between toxigenic and nontoxigenic strains. *Phytopathology* **79**, 404—408.
- Allan, R. E., and D. E. Roberts, 1991: Inheritance of reaction to strawbreaker foot rot in two wheat populations. *Crop Sci.* **31**, 943—947.
- Anderson, A. L., 1948: The development of *Gibberella zeae* head blight of wheat. *Phytopathology* **38**, 595—611.
- Atanassov, Z., C. Nakamura, N. Mori, C. Kaneda, H. Kato, Y. Jin, T. Yoshizawa, and K. Murai, 1994: Mycotoxin production and pathogenicity of *Fusarium* species and wheat resistance to *Fusarium* head blight. *Can. J. Bot.* **72**, 161—167.
- Bai, G., and G. Shaner, 1994: Scab of wheat: Prospects for control. *Plant Dis.* **78**, 760—766.
- Bai, G. H., and Q. P. Xiao, 1989: Genetic analysis on resistance to scab in six wheat cultivars. *Acta Agric. Shanghai* **5**, 17—23 (cited after Bai and Shaner 1994).
- , G. Shaner, and H. Ohm, 1993: Inheritance of resistance to *Fusarium graminearum* in eight wheat cultivars. *Phytopathology* **83**, 1414 (Abstr.).
- Baltzer, U., 1930: Untersuchungen über die Anfälligkeit des Roggens für Fusariosen. *Phytopath. Z.* **2**, 377—441.
- Becker, H., 1993: Pflanzenzüchtung. E. Ulmer, Stuttgart.
- Beyer, W., 1995: Genetische und epidemiologische Untersuchungen zum Fußkrankheitskomplex bei Roggen (*Secale cereale* L.) mittels ELISA (Enzyme-linked immunosorbent assay). Ulrich E. Grauer, Stuttgart.
- , H. Höxter, T. Miedaner, E. Sander, and H. H. Geiger, 1993: Indirect ELISA for quantitative assessment of *Fusarium* spp. in rye. *Z. PflKrankh. PflSchutz* **100**, 278—284.
- Blaney, B. J., and R. L. Dodman, 1988: Production of the mycotoxins zearalenone, 4-deoxynivalenol and nivalenol by isolates of *Fusarium graminearum* Groups 1 and 2 from cereals in Queensland. *Aust. J. Agric. Res.* **39**, 21—29.
- Bockmann, H., 1962: Künstliche Freilandinfektionen mit den Erregern der Fuß- und Ährenkrankheiten des Weizens. I. Vorbereitung und Durchführung der Feldinfektionen sowie deren Neben- und Nachwirkungen. *Nachrichtenbl. Dt. Pflschutzdienst (Braunschweig)* **14**, 153—156.
- , 1963: Künstliche Freilandinfektionen mit den Erregern der Fuß- und Ährenkrankheiten des Weizens. II. Die Infektionswirkung und ihre Beurteilung nach dem Schadbild. *Nachrichtenbl. Dt. Pflschutzdienst (Braunschweig)* **15**, 33—37.
- Boeger, J. M., R. S. Chen, and B. A. McDonald, 1993: Gene flow between geographic populations of *Mycosphaerella graminicola* (anamorph *Septoria tritici*) detected with restriction fragment length polymorphism markers. *Phytopathology* **83**, 1148—1154.
- Bojarczuk, M., and J. Bojarczuk, 1985: Breeding of rye resistant to fungous diseases. *Eucarpia Meet. Cereal Section on Rye*, Svalöv, Sweden, Part II, 351—364.
- Bowden, J. L., and J. F. Leslie, 1994: Diversity of *Gibberella zeae* at small spatial scales. *Phytopathology* **84**, 1140 (Abstr.).
- Bruehl, G., 1967: *Fusarium* head blight or scab. In: L. P. Reitz and K. S. Queensbury (eds), *Wheat and Wheat Improvement*, 388—390. American Society of Agronomy, Madison, WI.
- Bruins, M. B. M., I. Karsai, J. Schepers, and C. H. A. Snijders, 1993: Phytotoxicity of deoxynivalenol to wheat tissue with regard to *in vitro* selection for *Fusarium* head blight resistance. *Plant Science* **94**, 195—206.
- Burdon, J. J., 1993: The structure of pathogen populations on natural plant communities. *Annu. Rev. Phytopathol.* **31**, 305—323.
- Burgess, L. W., A. H. Wearing, and T. A. Toussoun, 1975: Surveys of *Fusaria* associated with crown rot of wheat in eastern Australia. *Australian J. Agric. Res.* **26**, 791—799.
- , R. L. Dodman, W. Pont, and P. Mayers., 1981: *Fusarium* diseases of wheat, maize, and grain sorghum in Eastern Australia. In: P. E. Nelson, T. A. Toussoun, and R. J. Cook (eds), *Fusarium—Diseases, Biology, and Taxonomy*, 64—76. Pennsylvania State University Press, University Park, PA.
- , B. A. Summerell, S. Bullock, K. P. Gott, and D. Backhouse, 1994: *Laboratory Manual for Fusarium Research*, 3rd edn. University of Sydney, Sydney.
- , —, D. Backhouse, F. Benyon, and J. Levic, 1996: Biodiversity and population studies in *Fusarium*. *Sydowia* **48**, 1—11.
- Chelkowski, J., 1989: Formation of mycotoxins produced by *Fusaria* in heads of wheat, triticale, and rye. In: J. Chelkowski (ed.), *Fusarium—Mycotoxins, Taxonomy, and Pathogenicity*, 63—84. Elsevier, Amsterdam.
- Colhoun, J., 1970: Epidemiology of seed-borne *Fusarium* diseases of cereals. *Ann. Acad. Sci. Fenn. A. IV Biologica* **168**, 31—36.
- , G. S. Taylor, and R. Tomlinson, 1968: *Fusarium* diseases of cereals. II. Infection of seedlings by *F. culmorum* and *F. avenaceum* in relation to environmental factors. *Trans. Br. Mycol. Soc.* **51**, 397—404.
- Cook, R. J., 1968: *Fusarium* root and foot rot of cereals in the Pacific Northwest. *Phytopathology* **58**, 127—131.
- , 1981a: *Fusarium* diseases of wheat and other small grains in North America. In: P. E. Nelson, T. A. Toussoun, and R. J. Cook (eds), *Fusarium—Diseases, Biology, and Taxonomy*, 39—52. Pennsylvania State University Press, University Park, PA.
- , 1981b: Water relations in the biology of *Fusarium*. In: P. E. Nelson, T. A. Toussoun, and R. J. Cook (eds), *Fusarium—Diseases, Biology, and Taxonomy*, 236—244. Pennsylvania State University Press, University Park, PA.
- Diehl, T., 1984: Weizenfusariosen—Zur Symptomentwicklung und Schadensanalyse bei Blatt- und Ährenbefall. PhD Thesis, Univ. Göttingen, Germany.
- Dodman, R. L., and G. B. Wildermuth, 1987: Inoculation methods for assessing resistance in wheat to crown rot caused by *Fusarium graminearum* Group 1. *Austr. J. Agric. Res.* **38**, 473—486.
- , G. B. Wildermuth, T. A. Klein, and F. W. Ellison, 1985: Field resistance of wheat cultivars to crown rot (*Fusarium graminearum* Group 1). In: C. A. Parker, A. D. Rovira, K. J. Moore, P. T. W. Wong, and J. F. Kollmorgen (eds), *Ecology and Management of Soilborne Plant Pathogens*, 167—168. American Phytopathol. Soc., St Paul, MN.
- Duben, J., 1978: Untersuchungen zum Fußkrankheitskomplex an Winterweizen unter besonderer Berücksichtigung von Arten der Gattung *Fusarium* Lk. PhD Thesis, Univ. Göttingen, Germany.
- , and H. Fehrmann, 1979a: Vorkommen und Pathogenität von *Fusarium*-Arten an Winterweizen in der Bundesrepublik Deutschland. I. Artenspektrum und jahreszeitliche Sukzession an der Halmbasis. *Z. PflKrankh. PflSchutz* **86**, 638—652.
- , and —, 1979b: Vorkommen und Pathogenität von *Fusarium*-Arten an Winterweizen in der Bundesrepublik Deutschland. II. Vergleich der Pathogenität als Erreger von Keimlings-, Halmbasis- und Ährenkrankheiten. *Z. PflKrankh. PflSchutz* **86**, 705—728.
- van Eeuwijk, F. A., A. Mesterházy, C. I. Kling, P. Ruckenbauer, L. Saur, H. Bürstmayr, M. Lemmens, L. C. P. Keizer, N. Maurin, and C. H. A. Snijders, 1995: Assessing non-specificity of resistance in wheat to head blight caused by inoculation with European strains of

- Fusarium culmorum*, *F. graminearum* and *F. nivale* using a multiplicative model for interaction. *Theor. Appl. Genet.* **90**, 221—228.
- Falconer, D. S., 1989: Introduction to Quantitative Genetics, 3rd edn. Longman, London/New York.
- Fehrmann, H., 1988: *Fusarium culmorum*. In: I. M. Smith, J. Dunez, D. H. Phillips, R. A. Lelliot, and S. A. Archer (eds), European Handbook of Plant Diseases, 287—289. Blackwell Scientific, Oxford.
- Fitt, B. D. L., A. Goulds, and R. W. Polley, 1988: Eyespot (*Pseudocercospora herpotrichoides*) epidemiology in relation to prediction of disease severity and yield loss in winter wheat—a review. *Plant Pathol.* **37**, 311—328.
- Flehinghaus-Roux, T., S. Deimling, and H. H. Geiger, 1995: Anther-culture ability in *Secale cereale* L. *Plant Breeding* **114**, 259—261.
- Foroughi-Wehr, B., and G. Wenzel, 1990: Recurrent selection alternating with haploid steps—a rapid breeding procedure for combining agronomic traits in inbreeders. *Theor. Appl. Genet.* **80**, 564—568.
- Francis, R. G., and L. W. Burgess, 1977: Characteristics of two populations of *Fusarium roseum* 'Graminearum' in eastern Australia. *Trans. Br. Mycol. Soc.* **68**, 421—427.
- Friend, D. W., and H. L. Trenholm, 1988: Mycotoxins in pig nutrition—review article. *Pig News Inf.* **9**, 395—401.
- Gang, G., 1997: Genetische, umweltbedingte und physiologische Einflüsse auf die Resistenz bzw. Aggressivität im Pathosystem Roggen/*Fusarium culmorum* (W.G.Sm.) Sacc. PhD Thesis, Univ. Hohenheim, Stuttgart, Germany.
- Gareis, M., I. Bauer, C. Enders, and B. Gedek, 1989: Contamination of cereals and food with *Fusarium* mycotoxins in European countries. In: J. Chelkowski (ed.), *Fusarium—Mycotoxins, Taxonomy, and Pathogenicity*, 441—472. Elsevier, Amsterdam.
- Geiger, H. H., 1985: Hybrid breeding in rye. Eucarpia Meet. Cereal Section on Rye, Svalöv, Sweden, Part I, 237—265.
- , 1988: Züchtung. In: W. Seibel and W. Steller (eds), Roggen, 25—44. Behr's Verlag, Hamburg.
- , and M. Heun, 1989: Genetics of quantitative resistance to fungal diseases. *Annu. Rev. Phytopathol.* **27**, 317—341.
- , T. Miedaner, and H. G. Welz, 1995: Genetic analysis of quantitative disease resistance. Proc. XIII Int. Plant Protection Congr. 2—7 July 1995, The Hague, The Netherlands. *Eur. J. Plant Pathol.* (Special issue), 805 (Abstr.).
- Gerlach, W., and H. Nirenberg, 1982: The genus *Fusarium*—a pictorial atlas. *Mitt. Biol. Bundesanst. Land-/Forstw. Berlin-Dahlem* **209**, 1—406.
- Greaney, F. J., J. E. Machacek, and C. L. Johnston, 1938: Varietal resistance of wheat and oats to root rot caused by *Fusarium culmorum* and *Helminthosporium sativum*. *Sci. Agr.* **18**, 500—523.
- Gu, J., 1983: A study on the genetics of resistance to wheat scab. *Sci. Agric. Sinica* **6**, 61—64.
- Guadet, J., J. Julien, J. F. Lafay, and Y. Brygoo, 1989: Phylogeny of some *Fusarium* species as determined by large subunit rRNA sequence comparison. *Mol. Biol. Evol.* **6**, 227—242.
- Hanson, E. W., E. R. Ausemus, and E. C. Stakman, 1950: Varietal resistance of spring wheats to fusarial head blights. *Phytopathology* **40**, 902—914.
- Hornok, L., 1980: Serotaxonomy of *Fusarium* species of the sections Gibbosum and Discolor. *Trans. Brit. Mycol. Soc.* **74**, 73—78.
- Höxter, H., G. Sieber, T. Miedaner, and H. H. Geiger, 1992: Test systems for evaluating quantitative resistance to *Fusarium* foot rot in inbred lines of winter rye. *Plant Breeding* **108**, 274—282.
- Ichinoe, M., H. Kurata, Y. Sugiura, and Y. Ueno, 1983: Chemotaxonomy of *Gibberella zeae* with special reference to production of trichothecenes and zearalenone. *Appl. Environ. Microbiol.* **46**, 1364—1369.
- Jenkins, J. E. E., W. S. Clark, and A. E. Buckle, 1988: *Fusarium* Diseases of Cereals. Home Grown Cereals Authority Review No. 4. Home Grown Cereals Authority, London, UK.
- Jenkinson, P., and D. W. Parry, 1994: Splash dispersal of conidia of *Fusarium culmorum* and *Fusarium avenaceum*. *Mycol. Res.* **98**, 506—510.
- Jiang, G., Z. Wu, and D. Huang, 1994: Effects of recurrent selection for resistance to scab (*Giberella zeae*) in wheat. *Euphytica* **72**, 107—113.
- Johnston, C. L., and F. J. Greaney, 1942: Studies on the pathogenicity of *Fusarium* species associated with root rot of wheat. *Phytopathology* **32**, 670—684.
- Johnson, R., 1993: Durability of disease resistance in crops: Some closing remarks about the topic and the Symposium. In: Th. Jacobs, and J. E. Parlevliet (eds), *Durability of Disease Resistance*, 283—300. Kluwer, Wageningen.
- Jones, D. R. H., 1954: Variation in culture of *Fusarium culmorum*. *Nature* **173**, 37—38.
- Kiecana, I., J. Perkowski, J. Chelkowski, and A. Visconti, 1987: Trichothecene mycotoxins in kernels and head fusariosis susceptibility in winter triticale. Proc. European Seminar on *Fusarium—Mycotoxins, Taxonomy, Pathogenicity*; 8–10 Sept., 1987, Warsaw, Poland. *Mycotoxin Res.* (Special Issue), 53—56.
- Klein, T. A., C. M. Liddell, L. W. Burgess, and F. W. Ellison, 1985: Glasshouse test for tolerance of wheat to crown rot caused by *Fusarium graminearum* Group 1. In: C. A. Parker, A. D. Rovira, K. J. Moore, P. T. W. Wong, and J. F. Kollmorgen (eds), *Ecology and Management of Soilborne Plant Pathogens*, 172—173. American Phytopathol. Soc., St Paul, MN.
- Lamb, C. J., M. A. Lawton, M. Dron, and R. A. Dixon, 1989: Signals and transduction mechanisms for activation of plant defenses against microbial attack. *Cell* **56**, 215—224.
- Leonian, L. H., 1929: Studies on the variability and dissociations in the genus *Fusarium*. *Phytopathology* **19**, 753—867.
- Liddell, C. M., and L. W. Burgess, 1988: Wax partitioned soil columns to study the influence of soil moisture potential on the infection of wheat by *Fusarium graminearum* Group 1. *Phytopathology* **78**, 185—189.
- Lind, V., 1992: Measuring the severity of eyespot disease induced by *Pseudocercospora herpotrichoides* (Fron) Deighton in wheat cultivars at different growth stages. *Plant Breeding* **108**, 202—209.
- Love, G. R., and L. M. Seitz, 1987: Effects of location and cultivar on *Fusarium* head blight in wheat from Kansas in 1982–83. *Cereal Chem.* **64**, 124—128.
- Ludwig, W., 1992: Quantitativ-genetische und methodische Untersuchungen zur Züchtung auf Fußkrankheitsresistenz bei Roggen (*Secale cereale* L.). Ulrich E. Grauer, Stuttgart.
- Manka, M., 1989: Fusaria as pathogens of cereal seedlings. In: J. Chelkowski (ed.): *Fusarium—Mycotoxins, Taxonomy and Pathogenicity*, 329—355. Elsevier, Amsterdam.
- , A. Visconti, J. Chelkowski, and A. Bottalico, 1985: Pathogenicity of *Fusarium* isolates from wheat, rye, and triticale towards seedlings and their ability to produce trichothecenes and zearalenone. *Phytopathol. Z.* **113**, 24—29.
- Marasas, W. F. O., P. E. Nelson, and T. A. Toussoun, 1984: *Toxigenic Fusarium Species*. Pennsylvania State University Press, University Park, London.
- , W. G. J. Voigt, S. C. Lamprecht, and P. S. van Wyk, 1988: Crown rot and head blight of wheat caused by *Fusarium graminearum* Groups 1 and 2 in the Southern Cape Province. *Phytophylactica* **20**, 385—389.
- Martin, R. A., and H. W. Johnston, 1982: Effects and control of *Fusarium* diseases of cereal grains in the Atlantic Provinces. *Can. J. Plant Pathol.* **4**, 210—216.
- Mathis, A., H. R. Forrer, and C. Gessler, 1986: Powdery mildew pustules supporting *Fusarium culmorum* infection of wheat leaves. *Plant Disease* **70**, 53—54.
- McDermott, J. M., B. A. McDonald, R. W. Allard, and R. K. Webster, 1989: Genetic variability for pathogenicity, isozyme, ribosomal DNA and colony color variants in populations of *Rhynchosporium secalis*. *Genetics* **122**, 561—565.
- McDonald, B. A., J. M. McDermott, S. B. Goodwin, and R. W. Allard, 1989: The population biology of host–pathogen interactions. *Annu. Rev. Phytopathol.* **27**, 77—94.
- , J. Miles, R. Nelson, and R. E. Pettway, 1994: Genetic variability

- in nuclear DNA in field populations of *Stagonospora nodorum*. *Phytopathology* **84**, 250—255.
- McKnight, T., and J. Hart, 1966: Some field observations on crown rot disease of wheat caused by *Fusarium graminearum*. *Qld. J. Agric. Anim. Sci.* **23**, 373—378.
- McNamara, R. B., and G. B. Wildermuth, 1996: A modified tyne to inoculate wheat with *Fusarium graminearum* Group 1. Proc. 8th Assembly Wheat Breeding Soc. Australia, 29 Sept.—4 Oct., 1996, The Australian National Univ., Canberra, 146—147.
- Mesterházy, A., 1978: Comparative analysis of artificial inoculation methods with *Fusarium* spp. on winter wheat varieties. *Phytopath. Z.* **93**, 12—25.
- , 1981: The role of aggressiveness of *Fusarium graminearum* isolates in inoculation tests on wheat in seedling stage. *Acta Phytopath. Sci. Acad. Hung.* **16**, 281—292.
- , 1983: Breeding wheat for resistance to *Fusarium graminearum* and *Fusarium culmorum*. *Z. Pflanzzüchtg.* **91**, 295—311.
- , 1984: A laboratory method to predict pathogenicity of *Fusarium graminearum* in field and resistance of wheat to scab. *Acta Phytopath. Acad. Sci. Hung.* **19**, 205—218.
- , 1987: Selection of head blight resistant wheats through improved seedling resistance. *Plant Breeding* **98**, 25—36.
- , 1988: Expression of resistance of wheat to *Fusarium culmorum* and *Fusarium graminearum* under various experimental conditions. *J. Phytopathology* **123**, 304—310.
- , 1989: Progress in breeding of wheat and corn genotypes not susceptible to infection by Fusaria. In: J. Chelkowski (ed.), *Fusarium—Mycotoxins, Taxonomy, and Pathogenicity*, 357—386. Elsevier, Amsterdam.
- , 1995a: Types and components of resistance to *Fusarium* head blight of wheat. *Plant Breeding* **114**, 377—386.
- , 1995b: Creating highly resistant winter wheats to *Fusarium* scab. Proc. Int. Sem. *Fusarium—Mycotoxins, Taxonomy, and Pathogenicity*, May 9—13, 1995, Martina Franca, Italy, 118—119.
- , and T. Bartók, 1993: Resistance and pathogenicity influencing toxin (DON) contamination of wheat varieties following *Fusarium* infection. *Hod. Rosl. Aklim. Nasien* **37**, 9—15.
- , —, and F. Sagi, 1994: New results in selecting scab resistant wheats and its impact on toxin contamination. *Genet. Polonica* **35B**, 47—62.
- Meyer, D., D. Weipert, and H. Mielke, 1986: Beeinflussung der Qualität von Weizen durch den Befall mit *Fusarium culmorum*. *Getreide, Mehl und Brot* **40**, 35—39.
- Meyer, J., 1985: Breeding for Resistance. *Eucarpia Meet. Cereal Section on Rye*, Svalöv, Sweden, Part II, 335—349.
- Miedaner, T., 1986: Entwicklung von Methoden zur Bestimmung der *Fusarium*-Resistenz in frühen Wachstumsstadien des Weizens. PhD Thesis, Univ. Hohenheim, Germany.
- , 1988: The development of a host-pathogen system for evaluating *Fusarium* resistance in early growth stages of wheat. *J. Phytopathol.* **121**, 150—158.
- , and H. H. Geiger, 1996: Estimates of combining ability for resistance of winter rye to *Fusarium culmorum* head blight. *Euphytica* **89**, 339—344.
- , and J. Perkowski, 1996: Correlations among *Fusarium culmorum* head blight resistance, fungal colonization, and mycotoxin contents in winter rye. *Plant Breeding* **115**, 347—351.
- , and A. G. Schilling, 1996: Genetic variation of aggressiveness in individual field populations of *Fusarium graminearum* and *F. culmorum* tested on winter rye. *Europ. J. Plant Pathol.* **102**, 823—830.
- , and H. Walther, 1987: Ermittlung der *Fusarium*-Resistenz im Ährenstadium von Weizen. *Z. PflKrankh. PflSchutz* **94**, 337—347.
- , D. C. Borchardt, and H. H. Geiger, 1993a: Genetic analysis of inbred lines and their crosses for resistance to head blight (*Fusarium culmorum*, *F. graminearum*) in winter rye. *Euphytica* **65**, 123—133.
- , W. F. Ludwig, H. Höxter, W. Beyer, and H. H. Geiger, 1993b: Analysis of quantitative resistance to *Fusarium* foot rot in winter rye. *Hod. Rosl. Aklim. Nasien* **37**, 51—59.
- , —, and H. H. Geiger, 1995a: Inheritance of foot rot resistance in winter rye. *Crop Sci.* **35**, 388—393.
- , D. E. Ziegler, and H. H. Geiger, 1995b: Variation and covariation for quantitative resistance to head blight (*Fusarium culmorum*) in two testcross series of S₂ lines in winter rye. *Plant Breeding* **114**, 155—159.
- , F. J. Fromme, and H. H. Geiger, 1995c: Genetic variation for foot rot and *Fusarium* head blight resistance among full-sib families of a self-incompatible winter rye (*Secale cereale* L.) population. *Theor. Appl. Genet.* **91**, 862—868.
- , W. Beyer, and H. H. Geiger, 1995d: Growth stage specific resistance of winter rye to *Microdochium nivale* and *Fusarium* spp. in the field assessed by immunological methods. *Phytopathology* **85**, 416—421.
- , G. Gang, and H. H. Geiger, 1996a: Quantitative-genetic basis of aggressiveness of 42 isolates of *Fusarium culmorum* for winter rye head blight. *Plant Dis.* **80**, 500—504.
- , G. Gang, and H. H. Geiger, 1996b: Genetics of resistance and aggressiveness in the winter rye/*Fusarium culmorum* head blight pathosystem. *Vortr. Pflanzzücht.* **35**, 165—174.
- , —, C. Reinbrecht, and H. H. Geiger, 1997: Lack of association between *Fusarium* foot rot and head blight resistance in winter rye. *Crop Sci.* **37**, in press.
- Mielke, H., 1988: Untersuchungen über *Fusarium culmorum* (W. G. Sm) Sacc. als Fuss- und Ährenkrankheitserreger beim Weizen. *Mitt. Biol. Bundesanstalt Land-/Forstw. Berlin-Dahlem* **238**, 1—101.
- Milgroom, M. G., S. E. Lipari, and W. A. Powell, 1992: DNA fingerprinting and analysis of population structure in the chestnut blight fungus, *Cryphonectria parasitica*. *Genetics* **131**, 297—306.
- Miller, J. D., 1989: Effects of *Fusarium graminearum* metabolites on wheat cells. In: A. Graniti, R. D. Durbin, and A. Ballio (eds), *Phytotoxins and Plant Pathogenesis*. NATO ASI Ser. H Cell Biol. **27**, 449—452.
- , 1994: Epidemiology of *Fusarium* ear diseases of cereals. In: J. D. Miller, and H. L. Trenholm (eds), *Mycotoxins in Grain—Compounds other than Aflatoxin*, 19—36. Eagen Press, St Paul, MN.
- , and P. G. Arnison, 1986: Degradation of deoxynivalenol by suspension cultures of the *Fusarium* head blight resistant cultivar Frontana. *Can. J. Plant Pathol.* **8**, 147—150.
- , J. C. Young, and D. R. Sampson, 1985: Deoxynivalenol and *Fusarium* head blight resistance in spring cereals. *Phytopath. Z.* **113**, 359—367.
- , R. Greenhalgh, Y. Z. Wang, and M. Lu, 1991: Trichothecene chemotypes of three *Fusarium* species. *Mycologia* **83**, 121—130.
- Miller, J. J., 1945: Studies on the *Fusarium* of muskmelon wilt. I. Pathogenic and cultural studies with particular reference to the cause and nature of variation in the causal organism. *Canad. J. Res. C* **23**, 16—43.
- Milus, E. A., and C. E. Parsons, 1994: Evaluation of foliar fungicides for controlling *Fusarium* head blight of wheat. *Plant Dis.* **78**, 697—699.
- Mirocha, C. J., H. K. Abbas, C. E. Windels, and W. Xie, 1989: Variation in deoxynivalenol, 15-acetyldeoxynivalenol, 3-acetyldeoxynivalenol, and zearalenone production by *Fusarium graminearum* isolates. *Appl. Environ. Microbiol.* **55**, 1315—1316.
- , W. Xie, Y. Xu, R. D. Wilcoxson, R. P. Woodward, R. H. Etebarian, and G. Behele, 1994: Production of trichothecene mycotoxins by *Fusarium graminearum* and *Fusarium culmorum* on barley and wheat. *Mycopathologia* **128**, 19—23.
- Mishra, C. B. P., 1971: Untersuchungen über Fusskrankheitserreger der Gattung *Fusarium* Lk. an Weizen. PhD Thesis, Univ. of Halle, Germany.
- , and L. Braun, 1976: Der Einfluss des Inokulums auf den *Fusarium*-Befall des Weizens. *Arch. Phytopath. u. Pflanzenschutz* **12**, 323—327.
- Müller, H. M., and K. Schwadorf, 1993: A survey of the natural occurrence of *Fusarium* toxins in wheat grown in a southwestern area of Germany. *Mycopathologia* **121**, 115—121.
- Murray, T. D., and G. W. Bruehl, 1983: Role of the hypodermis and

- secondary cell wall thickening in basal stem internodes in resistance to strawbreaker foot rot in winter wheat. *Phytopathology* **73**, 261—268.
- , and H. Ye, 1986: Papilla formation and hypersensitivity at penetration sites and resistance to *Pseudocercospora herpotrichoides* in winter wheat. *Phytopathology* **76**, 737—744.
- Nakagawa, M., 1955: Studies on ear-scab resistance of wheat plants. 2. Genetical factors affecting the inheritance on ear-scab disease of wheat plants. *Japan. J. Breeding* **5**, 15—22.
- Nicholson, P., P. Jenkinson, H. N. Rezanoor, and D. W. Parry, 1993: Restriction fragment length polymorphism analysis of variation in *Fusarium* species causing ear blight of cereals. *Plant Pathol.* **42**, 905—914.
- Nkongolo, K. K., D. Dostaler, and L. Couture, 1993: Effet de la bétaine, de la choline et d'extraits d'anthères du blé, sur la croissance du *Fusarium graminearum*. *Rev. Can. Phytopathol.* **15**, 81—84.
- van Ooijen, J. W., 1992: Accuracy of mapping quantitative trait loci in autogamous species. *Theor. Appl. Genet.* **84**, 803—811.
- Oswald, J. W., 1949: Cultural variation, taxonomy and pathogenicity of *Fusarium* species associated with cereal root rots. *Phytopathology* **39**, 359—376.
- Ouellet, T., and K. A. Seifert, 1993: Genetic characterization of *Fusarium graminearum* strains using RAPD and PCR amplification. *Phytopathology* **83**, 1003—1007.
- Parlevliet, J. E., 1993: What is durable resistance, a general outline. In: Th. Jacobs, and J. E. Parlevliet (eds), *Durability of Disease Resistance*, 23—40. Kluwer, Dordrecht.
- Parry, D. W., P. Jenkinson, and L. McLeod, 1995: *Fusarium* ear blight (scab) in small grain cereals — a review. *Plant Pathol.* **44**, 207—238.
- Partridge, J. E., 1991: Specific gene products of *Fusarium*: Analysis of ribosomal proteins. *Phytopathology* **81**, 1055—1058.
- Peever, T. L., and M. G. Milgroom, 1994: Genetic structure of *Pyrenophora teres* populations determined with random amplified polymorphic DNA markers. *Can. J. Bot.* **72**, 915—923.
- Perkowski, J., J. Chelkowski, and W. Wakulinski, 1990: Mycotoxins in cereal grain. Part 13. Deoxynivalenol and 3-acetyl-deoxynivalenol in wheat kernels and chaff with head fusariosis symptoms. *Die Nahrung* **34**, 325—328.
- , T. Miedaner, H. H. Geiger, H. M. Müller, and J. Chelkowski, 1995: Occurrence of deoxynivalenol (DON), 3-acetyl-DON, zearalenone, and ergosterol in winter rye inoculated with *Fusarium culmorum*. *Cereal Chem.* **72**, 205—209.
- Piglionica, V., 1977: Resistance of durum wheat cultivars to *Fusarium culmorum* and the difficulty of bringing greenhouse data into agreement with field results. In: *Induced Mutations against Plant Diseases*, 409—427. Int. Atomic Energy Agency (IAEA), Vienna.
- Pomeranz, Y., D. B. Bechtel, D. B. Sauer, and L. M. Seitz, 1990: *Fusarium* headblight (scab) in cereal grains. *Adv. Cereal Sci. Technol.* **10**, 373—433.
- Proctor, R. H., T. M. Hohn, and S. P. McCormick, 1995: Reduced virulence of *Gibberella zeae* caused by disruption of a trichothecene toxin biosynthetic gene. *Mol. Plant-Microbe Interact.* **8**, 593—601.
- Puhalla, J., 1981: Genetic considerations of the genus *Fusarium*. In: P. E. Nelson, T. A. Toussoun, and R. J. Cook (eds), *Fusarium — Diseases, Biology, and Taxonomy*, 291—305. The Pennsylvania State University Press, University Park, PA.
- Purss, G. S., 1966: Studies of varietal resistance to crown rot of wheat caused by *Fusarium graminearum* Schwabe. *Qld. J. Agric. Anim. Sci.* **23**, 475—498.
- Reinbrecht, C., T. Miedaner, M. Schollenberger, U. Lauber, and H. H. Geiger, 1996: Comparison of mycotoxin accumulation in grains of rye, triticale, and wheat inoculated with *Fusarium culmorum*. *Vortr. Pflanzenzüchtg.* **35**, 196—197.
- Ride, J. P., 1980: The effect of induced lignification on the resistance of wheat cell walls to fungal degradation. *Physiol. Plant Pathol.* **16**, 187—196.
- Saur, L., 1991: Sources of resistance to head blight caused by *Fusarium culmorum* in bread wheat and related species. *Agronomie* **11**, 535—541.
- Schilling, A. G., 1996: Characterization and differentiation of the cereal pathogens *Fusarium culmorum* and *F. graminearum* by PCR-based molecular markers. PhD Thesis, Univ. Hohenheim, Germany.
- , E. M. Möller, and H. H. Geiger, 1996: Polymerase chain reaction-based assays for species-specific detection of *Fusarium culmorum*, *F. graminearum* and *F. avenaceum*. *Phytopathology* **86**, 515—522.
- Schnell, F. W., 1982: A synoptic study of the methods and categories of plant breeding. *Z. Pflanzenzüchtg.* **89**, 1—18.
- Schroeder, H. W., and J. J. Christensen, 1963: Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. *Phytopathology* **53**, 831—838.
- Scott, P. M., 1990: Trichothecenes in grains. *Cereal Foods World* **35**, 661—666.
- Scott, P. R., and P. W. Benedikz, 1986: *Septoria* and *Fusarium* ear blight. *Ann. Rep. Plant Breed. Inst.* 1985, p. 100, Cambridge, UK (Cited after Parry et al. 1995).
- Seitz, L. M., D. B. Sauer, R. Burroughs, H. E. Mohr, and J. D. Hubbard, 1979: Ergosterol as a measure of fungal growth. *Phytopathology* **69**, 1202—1203.
- Sherwood, R. T., and C. P. Vance, 1980: Resistance to fungal penetration in Gramineae. *Phytopathology* **70**, 273—279.
- Snijders, C. H. A., 1990a: *Fusarium* head blight and mycotoxin contamination of wheat, a review. *Neth. J. Pl. Pathol.* **96**, 187—198.
- , 1990b: Systemic fungal growth of *Fusarium culmorum* in stems of winter wheat. *J. Phytopathology* **129**, 133—140.
- , 1990c: Diallel analysis of resistance to head blight caused by *Fusarium culmorum* in winter wheat. *Euphytica* **50**, 1—9.
- , 1990d: The inheritance of resistance to head blight caused by *Fusarium culmorum* in winter wheat. *Euphytica* **50**, 11—18.
- , 1990e: Response to selection in F₂ generations of winter wheat for resistance to head blight caused by *Fusarium culmorum*. *Euphytica* **50**, 163—169.
- , 1990f: Genetic variation for resistance to *Fusarium* head blight in bread wheat. *Euphytica* **50**, 171—179.
- , and F. A. van Eeuwijk, 1991: Genotype-strain interactions for resistance to *Fusarium* head blight caused by *Fusarium culmorum* in winter wheat. *Theor. Appl. Genet.* **81**, 239—244.
- , and C. F. Krechting, 1992: Inhibition of deoxynivalenol translocation and fungal colonization in *Fusarium* head blight resistant wheat. *Can. J. Bot.* **70**, 1570—1576.
- , and J. Perkowski, 1990: Effects of head blight caused by *Fusarium culmorum* on toxin content and weight of wheat kernels. *Phytopathology* **80**, 566—570.
- Sprague, G. F., and S. A. Eberhart, 1977: Corn Breeding. In: C. F. Sprague (ed.), *Corn and Corn Improvement*, 305—362. American Soc. of Agronomy, Madison, WI.
- Stack, R. W., and M. P. McMullen, 1985: Head blighting potential of *Fusarium* species associated with spring wheat heads. *Can. J. Plant Pathol.* **7**, 79—82.
- Statler, G. D., and L. C. Darlington, 1972: Resistance of hard red spring wheat and durum wheat to seedling blight and crown rots. *Plant Dis. Rep.* **56**, 788—791.
- Strange, R. N., and H. Smith, 1971: A fungal stimulant in anthers which predisposes wheat to attack by *Fusarium graminearum*. *Physiol. Pl. Pathol.* **1**, 141—150.
- , A. Deramo, and H. Smith, 1978: Virulence enhancement of *Fusarium graminearum* by choline and betaine and of *Botrytis cinerea* by other constituents of wheat germ. *Trans. Br. Mycol. Soc.* **70**, 201—207.
- Stoddart, J. A., and J. F. Taylor, 1988: Genotypic diversity: Estimation and prediction in samples. *Genetics* **118**, 705—711.
- Sutton, J. C., 1982: Epidemiology of wheat blight and maize ear rot caused by *Fusarium graminearum*. *Can. J. Pl. Pathol.* **4**, 195—209.
- Szecs, A., F. Szentkiralyi, and C. Koves-Pechy, 1976: Comparison of esterase patterns of *Fusarium culmorum* and *Fusarium graminearum*. *Acta Phytopathol. Acad. Sci. Hung.* **11**, 183—203.
- Takegami, S., 1957: On the relation between the existence of wheat anthers and the infection of *Gibberella zeae* (Schw.) Petch. *Proc.*

- Crop. Sci. Soc. Japan **26**, 31—32 (Cited after Rev. Appl. Mycol. **37**, 155 (Abstr.), 1958).
- Tanaka, T., A. Hasegawa, S. Yamamoto, U.-S. Lee, Y. Sugiura, and Y. Ueno, 1988: Worldwide contamination of cereals by the *Fusarium* mycotoxins nivalenol, deoxynivalenol and zearalenone. 1. Survey of 19 countries. J. Agric. Food Chem. **36**, 980—983.
- Teich, A. H., 1989: Epidemiology of wheat (*Triticum aestivum* L.) scab caused by *Fusarium* spp. In: J. Chelkowski (ed.), *Fusarium—Mycotoxins, Taxonomy, and Pathogenicity*, 269—282. Elsevier, Amsterdam.
- , L. Shugar, and A. Smid, 1987: Soft white winter wheat cultivar field resistance to scab and deoxynivalenol accumulation. Cer. Res. Communication **15**, 109—114.
- Tomasovic, S., 1989: Wheat breeding for resistance to *Fusarium* diseases, especially to *Fusarium graminearum* Schw. Wheat Inf. Serv. **69**, 13—17.
- Trissler, R., 1993: Untersuchungen zu Parametern der Fusarienresistenz von Winterweizensorten. PhD Thesis, Univ. Hohenheim, Stuttgart, Germany.
- Vanderplank, J. E., 1984: Disease Resistance in Plants, 2nd edn. Academic Press, London.
- Walther, H., 1976: Prüfung von Weizen auf Befall mit den Ährenkrankheiten *Septoria nodorum* und *Fusarium culmorum* durch künstliche Infektion im Feldversuch. Tag.-Ber. Akad. Landwirtsch.-Wiss. DDR **143**, 427—436.
- Wearing, A. H., and L. W. Burgess, 1977: Distribution of *Fusarium roseum* 'Graminearum' Group 1 and its mode of survival in eastern Australian wheat belt soils. Trans. Br. Mycol. Soc. **69**, 429—442.
- Wegener, M., and G. A. Wolf, 1995: Halmbasiskrankheit auch durch Fusarien. Pflanzenschutz-Praxis, Heft **1**, 27—29.
- Wilcoxson, R. D., T. Kommedahl, E. A. Ozmon, and C. E. Windels, 1988: Occurrence of *Fusarium* species in scabby wheat from Minnesota and their pathogenicity to wheat. Phytopathology **78**, 586—589.
- Wilde, P., 1987: Schätzung von Populationsparametern und Untersuchungen zur Effizienz verschiedener Verfahren der Rekurrenten Selektion bei Winterroggen. PhD Thesis, Univ. Hohenheim, Stuttgart, Germany.
- Wildermuth, G. B., and R. B. McNamara, 1994: Testing wheat seedlings for resistance to crown rot caused by *Fusarium graminearum* Group 1. Plant Dis. **78**, 949—953.
- , —, and J. S. Quick, 1996: Crown depth and susceptibility to crown rot in wheat, Proc. 8th Assembly Wheat Breeding Soc. Australia, 29 Sept.—4 Oct., 1996, The Australian National Univ., Canberra, Australia, 194—197.
- , and G. S. Purss, 1971: Further sources of field resistance to crown rot of wheat caused by *Fusarium graminearum*. Aust. J. Exp. Agric. Anim. Husb. **11**, 455—459.
- Windels, C. E., 1992: *Fusarium*. In: L. L. Singleton, J. D. Mihail, and C. M. Rush (eds), Methods for Research on Soilborn Phytopathogenic Fungi, 115—128. APS Press, St Paul, MN.
- , and C. Holen, 1989: Association of *Bipolaris sorokiniana*, *Fusarium graminearum* Group 2 and *F. culmorum* on spring wheat differing in severity of common root rot. Plant Dis. **73**, 953—956.
- Wolf, G., and F. Fric, 1981: A rapid staining method for *Erysiphe graminis* f. sp. *hordei* in and on whole barley leaves with a protein-specific dye. Phytopathology **71**, 596—598.
- Woodward, R. P., and R. D. Wilcoxson, 1991: Comparison of greenhouse seedling blight and field scab induced by *Fusarium graminearum* in spring wheats. Phytopathology **81**, 1146 (Abstr.).
- Wright, S., 1968: Genetic and biometric foundations. Evaluation and the genetics of populations. A treatise in three volumes, Vol. I: Genetic and Biometric Foundations. The Univ. of Chicago Press, Chicago.
- van Wyk, P. S., G. D. C. Pauer, J. P. Rheeder, O. Los, and W. F. O. Marasas, 1988: Reaction of different wheat cultivars to crown rot caused by *Fusarium graminearum* Group 1. Phytophylactica **20**, 69—72.
- Yoder, O. C., 1981: Genetic analysis as a tool for determining the significance of host-specific toxin and other factors of disease. In: R. C. Staples, and G. H. Toenniessen (eds), Plant Disease Control, 3—11. Wiley and Sons, New York.
- Yoshizawa, T., 1991: Natural occurrence of mycotoxins in small grain cereals (wheat, barley, rye, oats, sorghum, millet, rice). In: J. E. Smith, and R. S. Henderson (eds), Mycotoxins and Animal Foods, 301—324. CRC Press, Boca Raton, FL.
- Yu, Y. J., 1982: Monosomic analysis for scab resistance and yield components in the wheat cultivar Soo-moo-3. Cer. Res. Commun. **10**, 185—190 (Cited after Bai and Shaner 1994).
- , 1990: Genetic analysis for scab resistance in four wheat cultivars PHJZM, HHDTB, CYHM, and YGFZ. In: L. H. Zhu (ed.), Advances in Researches on Inheritance to Diseases in Major Crops, 197—205. Jiangsu Sci-Tech. Publ. House, Nanjing, (Cited after Bai and Shaner 1994).
- Zhang, L. Q., and X. P. Pan, 1982: A study on resistance to colonization of *Gibberella zeae* in wheat varieties. J. S. China Agric. Coll. **3**, 21—29.
- Zhou, C. F., 1985: Production constraints and research priorities in the southern winter wheat region of China. In: Wheat for More Tropical Environments, 72—77. CIMMYT, Mexico DF.

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