The amount of genetic variation for resistance to foot rot caused by *Pseudocercosporella herpotrichoides*, *Fusarium* spp., and *Microdochium nivale* and for resistance to head blight caused by *Fusarium culmorum* are important parameters when estimating selection gain from recurrent selection in winter rye. One-hundred and eighty-six full-sib families of the self-incompatible population variety Halo, representing the Petkus gene pool, were tested for foot-rot resistance at five German location-year combinations (environments) and for head-blight resistance in three environments with artificial inoculation in all but one environment. Foot-rot rating was based on 25 stems per plot scored individually on a 1–9 scale. Head-blight resistance was plotwise scored on a 1–9 scale and, additionally, grain-weight per spike was measured relative to the non-inoculated control plots. Significant estimates of genotypic variance and medium-sized heritabilities ($h^2 = 0.51–0.69$) were observed in the combined analyses for all resistance traits. In four out of five environments, the amount of genetic variance was substantially smaller for foot-rot than for head-blight rating. Considerable environmental effects and significant genotype-environment interactions were found for both foot-rot and head-blight resistance. Coefficients of error-corrected correlation among environments were considerably closer than phenotypic correlations. No significant association was found between the resistances to both diseases ($r = -0.20$ to 0.17). In conclusion, intra-population improvement by recurrent selection should lead to substantial higher foot-rot and head-blight resistances due to significant quantitative genetic variation within Halo. Selection should be carried out in several environments. Lack of correlation between foot-rot and head-blight resistance requires separate infection tests for improving both resistances.

**Key words** Quantitative resistance · *Pseudocercosporella herpotrichoides* · *Microdochium nivale* · *Fusarium culmorum* · Population parameter

**Introduction**

*Fusarium* species are widespread soil-borne pathogens infecting winter rye (*Secale cereale* L.) at all host growth stages. In Germany, foot rot in wheat is mainly caused by *Pseudocercosporella herpotrichoides* (Fron) Deighton, and *Fusarium* species, especially *F. culmorum* (W.G.Sm.) Sacc., *F. graminearum* Schwabe, and *F. avenaceum* (Fries) Sacc., and *Microdochium nivale* (Fries) Samuels & Hallet (Duben and Fehrmann 1979). In winter rye, at least two of these species have been found in most locations simultaneously and could frequently be isolated from the same stems or even from the same necrotic lesions (Miedaner et al. 1993a). Resistance selection should, therefore, be directed towards the whole pathogen complex. Yield losses by foot rot are due to restricted nutrient and water transport and increased lodging. They are reported to amount to up to 45% depending on environment and the fungal species involved (Meyer 1985; Frauenstein 1987). Resistance is inherited quantitatively in hybrid rye material (Höxter et al. 1992; Miedaner et al. 1995a) and population varieties (Bojarczuk and Bojarczuk 1985), with no genotype being completely resistant or susceptible. This indicates that several genes control resistance (Geiger and Heun 1989). Similar results were obtained with head blight resulting from *F. culmorum* in winter rye (Miedaner et al. 1993b, 1995b). The most common *Fusarium* species causing head blight are *F. culmorum* and *F. graminearum*. Epidemic infections lead to considerable yield losses (Miedaner et al. 1993b),
increased seedling and foot-rot infection of the following crop (Duben and Fehrman 1980), and grain contamination with mycotoxins (Chelkowski 1989; Perkowski et al. 1995).

In hybrid rye breeding, population varieties are still important sources of variability for inbred-line development (Geiger 1985). For this purpose, they are either crossed directly with self-fertile material or improved previously by recurrent selection (Geiger 1988). Increasing the frequency of resistance genes in a breeding population also enhances the chance to select superior genotypes from it (Sprague and Eberhart 1977). To date, no data are available on the variation of resistances to foot rot and head blight within self-incompatible rye populations. Thus, the aim of the presented study was to (1) estimate the relative importance of genotypic variance and genotype-environment interaction variance, and (2) calculate the heritability for resistance to foot rot and head blight from multi-location tests of full-sib families from the widely grown population variety Halo.

**Materials and methods**

**Plant material**

Single plants from the self-incompatible, open-pollinated winter rye cultivar Halo were vegetatively cloned and two single plants with 10–20 clones each were crossed pairwise between isolation walls to obtain full-sib families. Halo represents the ‘Petkus’ gene pool, one of the major heterotic groups currently used in hybrid rye breeding (Geiger 1985). For all experiments, two sets of 93 full-sibs each, randomly chosen from a larger population, were used. Full-sib families were randomly assigned to the two sets.

**Foot-rot resistance tests**

Foot-rot resistance was tested in 1990 at Bergen near Celle (BER) and Klausheide near Münster (KLA) in northern Germany. In 1991, BER, KLA, and additionally the experimental station Oberer Lindenhof near Reutlingen (OLI) in southern Germany, were used. Each full-sib family was grown in one-row microplots of 1 m length with 0.42 m spacing between plots at a seed density of 320 kernels m⁻². The experiments were conducted utilizing two separate but adjacent 10 × 10-lattice designs with two replicates including seven standard varieties. Recommended cultural practices were used throughout the growing season at each location.

Genotypes were artificially inoculated by spreading dry, crushed wheat-grain material colonized with either P. herpotrichoides var. acutiformis (BER90, BER91), an equidosal mixture of F. culmorum and F. graminearum (KLA91), or with M. nivale var. nivale (OLI91). At KLA, genotypes were exposed to natural soil-borne infection. Source and preparation of inoculum, and inoculation procedure were exactly the same as previously described (Miedaner et al. 1995a). At late-milk ripening, 25 randomly chosen stems per plot were harvested. Stems were thoroughly washed, the outer leaf sheaths removed, and the lower 10 cm of the stem base visually rated for foot rot on a 1–9 scale as follows: 1 = no lesion visible, 2 = lesion of pinpoint size, 3 = lesion covering less than one-fourth the circumference of the stem, 4 = lesion covering one-fourth to half the circumference of the stem, 5 = lesion covering about half to three-fourths the circumference of the stem, 6 = lesion covering more than three-fourths the circumference of the stem, 7 = lesion girdling the stem, no softening of the tissue, 8 = lesion girdling the stem, moderate softening of the tissue, 9 = stem fully necrotic and softened. This scale describes both necrotization of the stems and softening of the tissue.

To evaluate disease incidence of the individual foot-rot fungi, necrotic lesions from 100 diseased stems per replicate and environment were cut out, surface-disinfected and incubated on agar as recently described by Miedaner et al. (1995a). The resulting colonies were identified according to their conidial morphology (Nirenberg 1981).

**Head-blight resistance tests**

The experiments with head-blight inoculation were grown in 1990 at Stuttgart-Hohenheim (HOH) and in 1991 at HOH and BER. At HOH, two-row microplots of 1 m length with 0.21 m spacing between rows and 0.42 m spacing to the neighboring plot were used. At BER, genotypes were grown in one-row microplots of 1.5 m length with 0.42 m spacing between plots. Each of the two sets was planted in two adjacent treatment blocks: non-inoculated vs inoculated. Plots within both treatment blocks were arranged according to a 10 × 10-lattice design including seven standard varieties. Recommended cultural practices were used throughout the growing season at each location.

Inoculum production and inoculation were performed as previously described (Miedaner et al. 1995b). Each genotype was artificially inoculated at its respective flowering time, and routinely for a second time 3 days later, with a mixture of four F. culmorum isolates. The inoculum consisted of a spore suspension with 0.5 × 10⁶ spores per ml and was applied at a rate of 30 ml per row. At HOH, the field was sprinkled with a mist irrigation device in the morning after every inoculation from 7:00 to 12:00 h every 30 min for a period of 2-min each to ensure a high relative humidity. At BER91, no irrigation water was applied. Disease severity was rated four times at HOH and two times at BER on a 1–9 scale (1 = no symptoms to 9 = fully diseased, Mielke 1988). Two resistance traits – head-blight rating, averaged over all ratings with significant genotypic differentiation, and grain weight per spike relative to the non-inoculated plots – were assessed (Miedaner et al. 1995b).

**Statistical analyses**

Analyses of variance were based on single-plot data. The two sets can be considered as independent material replicates because the 93 full-sibs were randomly assigned to the sets. Analyses of variance across the five location-year combinations (environments) were based on single-plot-adjusted entry means (Cochran and Cox 1957). For each environment, trait values and their residuals were normally distributed. Repeatability and broad-sense heritability estimates were based on entry means across replications (in individual environments) and across environments respectively (Wricke and Weber 1986; Falconer 1989). Coefficients of phenotypic correlation (r_p) between all pairs of environments were calculated using the established procedure (Falconer 1989). To exclude the error effects that mask the genotypic covariance between the two environments, coefficients of phenotypic correlation were corrected according to the following formula:

\[
    r_{pc} = \frac{cov(g_1, g_2) + cov(ge_1, ge_2)}{\sqrt{\sigma^2 g_1 + \sigma^2 ge_1} \sqrt{\sigma^2 g_2 + \sigma^2 ge_2}}
\]

with r_{pc} = coefficient of error-corrected correlation, cov = covariance of the two environments 1 and 2, with the subscripts g or ge meaning genotypic or genotype-environment covariance; \( \sigma^2_g \), \( \sigma^2_{ge} \) = genotypic or genotype-environment interaction variance for environment 1 or 2, respectively. Any deviation of the error-corrected correlation from a value of 1 is caused by genotype-environment interaction variance under the assumption that cov(ge_1, ge_2) is zero or at least considerably smaller than the respective variances. The standard errors of the error-corrected correlation coefficients were computed according to Mode and Robinson (1959). The same procedure was used to calculate error-corrected correlation coefficients between foot-rot and head-blight resistances for all pairs of environments. To demonstrate the theoretical frequency distributions of the genotypic values for the individual environments (see