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Parameter estimates for ergot susceptibility in rye based on harvest ware from artificially inoculated trials and from farmers' fields

Introduction

Rye (*Secale cereale* L.) is a major staple crop in the EU grown on 2.6 million hectares (FAOSTAT 2013). 80% of this acreage is grown in Germany, Poland, and Austria. Ergot, caused by *Claviceps purpurea* (Fr.) Tul., is a severe disease in rye. The fungus replaces the caryopses by a dark mycelial mass known as a sclerotium (Mielke 2000). The disease causes severe economic damage due to the contamination of grain with ergot sclerotia. They contain up to 30 ergot alkaloids with extremely deleterious effects on the central nervous systems of mammals including humans.

EU threshold levels are at the moment 0.1% of sclerotia or sclerotia fragments in grain for animal nutrition and 0.05% for human consumption. Effective cleaning on the basis of photocells is feasible for commercial grain production but creates extra costs. .

Conidia of *C. purpurea* infect only young, usually unfertilized ovaries of grasses and cereals and mimic pollen tube growth (Oeser et al. 2002). They are most successful when the ovaries are unfertilized. Therefore, disease appears mainly in sites/years with cold, rainy weather periods during flowering resulting in a lack of viable pollen and delayed pollination.

The main objectives of this study are to (i) estimate the accuracy (heritability) of trials with natural and artificial ergot affection, (ii) estimate the genetic correlation between ergot affection using inoculated and non-inoculated trials.

Material

Ergot affection data (% ergot weight) from

(i) inoculated trials by the Bundessortenamt (BSA) between 2009 and 2014

(ii) a survey based on farmers harvest ware samples (“Besondere Ernteermittlung”) assessed by the Max Rubner-Institut (MRI) 2011-2014

(iii) Inoculated and non-inoculated field experiments from the EU pilot trials 2012-2013

- Note that the above data are highly unbalanced
- Permission to use and publish these data needs to be requested from BSA (D. Rentel) and MRI (A. Hüsken)
- Note that MRI data are not earlier available than 2 to 3 years after the third year of evaluation of this variety in official trials (BSA), i.e. there is a time lag between the ergot data from the BSA and the survey data on ergot affection in commercial rye harvest lots by the MRI

Methods

- Estimate variance components from data produced in naturally (MRI) and artificially infected (BSA) environments and from the EU pilot trial, respectively.
- Quantify relative size of variance components due to genotypes (G) or groups of cultivars (e.g. population, fully and partially restored hybrids) and environments (E) in relation to genotype x environment (GxE) interaction variances. Depending on the model used E can be further subdivided into ears (Y) or locations(L)
- Estimate phenotypic and genotypic correlations between naturally artificially infected environments
- Draft recommendations how to allocate trial capacities to environments such as years, locations and replications for an efficient and accurate ergot resistance assessment.

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