

# Quantitative characterisation of malting barleys for consistency in grain protein concentration

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## Abstract

Partitioning of the sum of squares due to genotype-environment interactions showed that imperfect correlation was the main cause for inconsistency in grain protein from one environment to the other. Components attributable to specific genotypes were derived using data collected between 1995 and 1999 in South Australia. The estimates showed a high degree of repeatability. Franklin exhibited the largest consecutive sum of squares due to imperfect correlations.

## Key words

Genotype-environment interaction, heterogeneity of variance, imperfect correlation.

## Introduction

Genotype-environment interactions have been of paramount importance in breeding low-protein barleys (8; 2) because of the financial consequence to producers and end-users. Although the phenomenon has been largely considered biometrical, its biological basis is well-established (6). Breeding for reduced variability is therefore feasible, but unlike mean trait performance, phenotypic plasticity is a difficult trait measure and select for directly.

A concept suggested about half a century ago by Falconer (5) has been extensively utilised by evolutionary biologists in analysis of genotype-environment interactions. The method is based on partitioning of the interaction sum of squares associated with a given genotype into two components, one due to heterogeneity of variance and the other due to imperfect correlation. Partitioning of the interaction sum of squares by this method is desirable because the first component measures the change in genetic variance component across environments while the second component, which measures the extent to which performance across environments is correlated, is a coefficient of stability (7). Herein, we evaluated the potential usefulness of the method in characterising malting barley genotypes for ability to produce consistently low grain protein concentration.

## Materials and Methods

Grain protein data were obtained from regional evaluation trials carried out between 1995 and 1999 in South Australia. Genotypes and locations varied in each year of the trials and the data were highly unbalanced. To ensure valid comparison of the estimates for a given genotype, genotype x location interaction was the focus, using a GL matrix of 8 genotypes and 17. Data analysis was performed on a yearly basis. For each year, the interaction sum of squares associated with any given genotype was partitioned into two components according to the descriptions of Muir et al. (7) based on method 2 as:

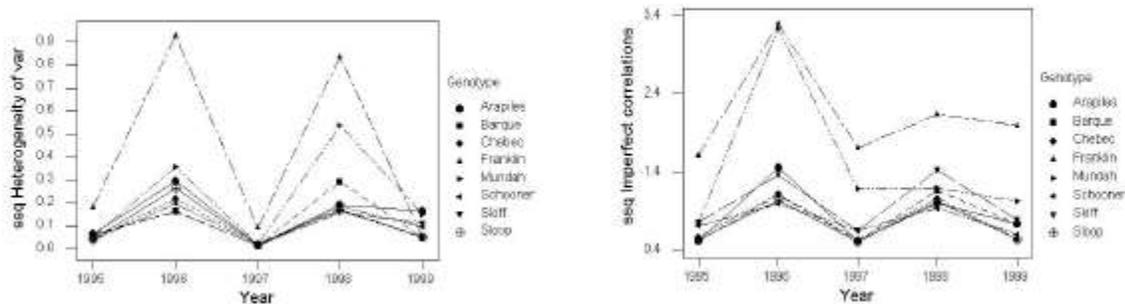
$$SS(GE)_i = \sum_{i=1}^g SS(HV)_{ii'} + \sum_{i=1}^g SS(IC)_{ii'}$$

Where  $SS(HV)_{ii'} = n(S_i - S_{i'})^2/g$  measured the degree to which heterogeneity of variance accounts for GE interaction, and  $SS(IC)_{ii'} = 2n(1-r_{ii'}) S_i S_{i'}/g$ , measured the extent to which performance in one environment fails to predict performance in the other. All calculations were performed with a program written in GENSTAT and based on the descriptions of Muir et al. (7).

## Results

Partitioning of the GEI into the two components for each genotype is presented in Fig.1. The results showed a high degree of persistence in characterising a genotype's contribution of GEI. In general, the component due to imperfect correlation was substantially larger than that due to the heterogeneity of variance, which showed that interaction effects were mostly due to imperfect correlations.

For consistency of performance, it is desired that a genotype's contribution to GEI should be largely due to heterogeneity of variance, and none of the cultivars evaluated was outstanding in this regard. Franklin had the largest sum of squares due to GEI, followed by Mundah. Franklin also had the largest sum of squares due to heterogeneity of variance, and an equally large sum of squares due to imperfect correlation. This makes the cultivar undesirable in breeding for stability, as the lack of correlation is the component that can potentially complicate selection.



**Figure 1. Interaction plot of components of GEI assignable to eight malting barley genotypes, calculated from grain protein concentration across 17 locations.**

## Discussion

Genotype-environment interaction may arise for two reasons, one being the difference in responses of the same set of genes to different environments, and the other being the expression of different sets of genes in different environments (5, 9; 3). If the same set of genes are expressed in different environments, then the differences in responses may be regarded as due to heterogeneity of genetic or error variances (or both) across environments. If, on the other hand, different sets of genes are expressed, then there exists GE interaction that cannot be accounted for simply by differences in genetic variance between environments. It is important to distinguish between these two components because the lack of genetic correlation among environments is the component that intrinsically complicates selection (1, 10).

Although this concept has been widely applied to analyses of genotype-environment interactions at the trait level (4, 10; Chapman et al. 2000), such trait-based analysis is not very useful in plant breeding since recommendations among genotypes cannot be made. The analysis presented in this paper showed a high degree of repeatability of estimates for genotypes, which has not been demonstrated for other measures of stability.

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## References

1. Basford, K.E. and Cooper M. 1998. *Aust. J. Agric. Res.* **49**, 153-174.
2. Bertholdsson, N.O. 1998. *Eur. J. Agron.* **9**, 213-222.
3. Cockerham, C.C. 1963. In: *Statistical genetics and plant breeding.* (Ed. W. D. Hanson and H. F. Robinson) NAS-NRC, Washington, D.C., pp 53-94.
4. Cooper, M. and DeLacy I.H. 1994. *Theor. Appl. Genet.* **88**, 561-572.
5. Falconer, D.S. 1952. *American Natur.* **86**, 293-298.
6. Schlichting, C.D. and Pigliucci, M. 1998. *Phenotypic evolution: a reaction norm perspective.* Sinauer Asso, Inc, Sunderland Massachusetts, pp 387.
7. Muir, W., Nyquist, W.E. and Xu, S. 1992. *Theor. Appl. Genet.* **84**, 193-200.
8. Piper, T.E. and Rasmusson, D.C. 1984. *Crop Sci.* **24**, 853-854.
9. Robertson, A. 1959. *Biometrics* **15**, 469-485.
10. Wu, R. and Stettler, R.F., 1997. *Heredity* **78**, 124-134.