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Some vocabulary and grammar for the analysis of multi-environment trials, as applied to the analysis of FPB and PPB trials

F.A. van Eeuwijk[1], M. Cooper[2,4], I.H. DeLacy[2], S. Ceccarelli[3] & S. Grando[3]
[1]*Dpt. Plant Sciences, Wageningen University, Lab. of Plant Breeding, P.O. Box 386, Wageningen, The Netherlands;* [2]*School of Land and Food Sciences, The University of Queensland, Brisbane, Qld 4072, Australia;* [3]*International Center for Agricultural Research in the Dry Areas, P.O. Box 5466, Aleppo, Syria;* [4]*Pioneer Hi-Bred International Inc., 7300 NW 62nd Avenue, P.O. Box 1004, Johnston, Iowa, 50131-1004, USA*

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Summary

For the improvement of genetic material suitable for on farm use under low-input conditions, participatory and formal plant breeding strategies are frequently presented as competing options. A common frame of reference to phrase mechanisms and purposes related to breeding strategies will facilitate clearer descriptions of similarities and differences between participatory plant breeding and formal plant breeding. In this paper an attempt is made to develop such a common framework by means of a statistically inspired language that acknowledges the importance of both on farm trials and research centre trials as sources of information for on farm genetic improvement. Key concepts are the genetic correlation between environments, and the heterogeneity of phenotypic and genetic variance over environments. Classic selection response theory is taken as the starting point for the comparison of selection trials (on farm and research centre) with respect to the expected genetic improvement in a target environment (low-input farms). The variance-covariance parameters that form the input for selection response comparisons traditionally come from a mixed model fit to multi-environment trial data. In this paper we propose a recently developed class of mixed models, namely multiplicative mixed models, also called factor-analytic models, for modelling genetic variances and covariances (correlations). Mixed multiplicative models allow genetic variances and covariances to be dependent on quantitative descriptors of the environment, and confer a high flexibility in the choice of variance-covariance structure, without requiring the estimation of a prohibitively high number of parameters. As a result detailed considerations regarding selection response comparisons are facilitated. The statistical machinery involved is illustrated on an example data set consisting of barley trials from the International Center for Agricultural Research in the Dry Areas (ICARDA). Analysis of the example data showed that participatory plant breeding and formal plant breeding are better interpreted as providing complementary rather than competing information.

Abbreviations: CR – correlated response; DR – direct response; FPB – Formal plant breeding; PPB – participatory plant breeding

Introduction

Disputes continue about the advantages of Participatory Plant Breeding (PPB) over Formal Plant Breeding (FPB) methods (see Eyzaguirre & Iwanga, 1995), where PPB methods have been advocated in place of FPB methods for complex target environments (Joshi & Witcombe, 1995; Ceccarelli, 1996). In this paper we will refer to PPB as those methods where farmers practice selection on their own fields (also defined as decentralized-participatory breeding), as opposed to

FPB where decisions on selection are centrally taken by a breeder without consulting farmers. A prerequisite for the comparison of the efficiency of selection by both approaches is a common language in which the distinguished approaches can be phrased. A natural candidate for such a language is provided by the selection response framework, as acknowledged by the contribution of Atlin et al. (2001), elsewhere in this volume. Within this framework we compare the response due to selection in the environment of interest itself to a correlated response due to selection in another environment. The environment for which progress is wanted is called the target environment. The environment where selection takes place is called the selection environment. In selection response theory we compare the response for the situation where target and selection environment are the same, the direct response situation, with the situation in which target and selection environment differ, the correlated (indirect) response situation. The direct response to selection in the target environment is given by $DR_T = i_T\ h_T\ V_{G_T}^{1/2}$, with i_T the selection intensity, h_T the square root out of the heritability and $V_{G_T}^{1/2}$ the genetic standard deviation, all in the target environment. The correlated response in the target environment, from selection in the selection environment, is $CR_T = i_S\ h_S\ r_{G_{ST}}\ V_{G_T}^{1/2}$, with i_S, h_S and $V_{G_T}^{1/2}$ selection intensity, square root heritability and genetic standard deviation, where the subscript S refers to the selection environment and T to the target environment. The quantity $r_{G_{ST}}$ stands for the genetic correlation between selection environment and target environment (Falconer, 1981).

A criterion for decisions on the profitability of selection in another environment than the target is the ratio CR_T/DR_T. When we assume the selection intensity to be the same for selection and target environment we obtain $\frac{CR_T}{DR_T} = \frac{h_S r_{G_{ST}}}{h_T}$. Thus when the product of genetic correlation and square root heritability in the selection environment is larger than the square root heritability in the target environment, it is attractive to perform selection in another environment than the target. Indirect, correlated selection is preferred when the heritability in the selection environment is considerably higher than the heritability in the target environment, while the genetic correlation is still substantial. Lack of perfect correlation should be compensated for by higher heritability to make correlated selection work. Intuitively, one would say that genetic correlations can be made high by choosing the conditions in the selection environment to mimic those in the target environment. We would argue that this is a core principle that should be used for the effective design of breeding trials, regardless of whether they are considered to be components of PPB or FPB. Heritabilities might be increased by reducing plot errors through improved trial management or by increasing replication (Atlin et al., 2001). FPB, as selection at a research centre for an on farm target environment, becomes an interesting option whenever the loss of genetic correlation due to selection in another environment than the target environment is compensated by an increase in heritability.

The class of statistical models that is implied by the selection response framework is that of mixed (analysis of variance) models. In comparison to the standard, fixed analysis of variance models, mixed models are characterized by extra distributional assumptions for some of the effects. When dealing with responses to selection, a common choice is to interpret the genotypic main effects and the genotype by environment interaction effects as normally distributed random variables with mean zero and variance V_G and V_{GE}, respectively, although other choices are often more appropriate for the variance-covariance structure, as will be discussed in the next section.

Estimates for the *CR/DR*-ratio depend on the structure of the mixed model for the response in target and selection environments. The choice of mixed model is critical for the kind of argument that can be developed on the feasibility of FPB in relation to PPB. Many applied researchers are not fully conscious of the choices available for the variance-covariance structure of models and the implications of these choices, simply because they accept the defaults of the statistical package they are using. These defaults are rarely appropriate for multi-environment trial (MET) data. For example, the default structure for the genetic variances and covariances in most packages is best known in the statistical literature under the name of 'compound symmetry' (Neter et al., 1996), where the alternative name of 'uniform correlation' (Cullis et al., 1998; Smith, 1999) probably better expresses its most distinctive characteristic. This structure is based on the assumption that the genetic correlation and genetic covariance between any pair of environments will be the same and that the genetic variance is constant over environments. So, when a MET contains a group of stress environments and a group of optimal environments, analysis by a mixed model with a uniform correlation model for the genetic variance-covariance structure produces a model in which the

genetic correlation between pairs of optimal environments is equal to the genetic correlation between pairs of stressful environments, and the same genetic correlation is supposed to be valid for pairs of stress and optimal environments. Furthermore, the genetic variance is supposed to be independent of the presence of stress conditions. It will be obvious that a uniform correlation model can only be adequate within a reasonably homogeneous group of environments.

PPB has been advocated as a preferred strategy over FPB for heterogeneous target environments where there will exist neither a perfect correlation among environments nor a uniform genetic correlation (Ceccarelli, 1996). For a fair comparison of PPB and FPB the implementation of more advanced mixed models is a prerequisite. Alternatives for the uniform correlation model allow genetic correlations to differ between pairs of environments, while genetic variances are made dependent on the environment. As a consequence the behaviour of the *CR/DR* ratio for pairs of environments can change in an unrestricted way when going from one pair of environments to another pair of environments. In this paper we will describe some more advanced mixed models for genotype by environment data, with special attention for 'unstructured' variance-covariance models and mixed multiplicative models, also called factor-analytic models. To set the stage, first two well known elementary models will be summarized; the two-way additive mixed model and the uniform correlation model. The scope of application of these simpler models is limited, as they imply severe restrictions on the type of data. More flexible mixed models will subsequently be presented.

The meaning of models and their parameters is best understood from application to intrinsically interesting data. We will use barley data from the International Center for Agricultural Research in the Dry Areas (ICARDA) for illustration. In the target environment system of Syrian dry lands, the timing of the incidence and severity of drought are major environmental variables influencing adaptation and the yield of barley varieties. The emphasis in our treatment will be on the interpretation of the model parameters, i.e., genetic correlations between environments and genetic variances within environments, while details on estimation and inference can be found elsewhere (Gilmour et al., 1995; Gogel et al., 1995; Oman, 1991; Piepho, 1997; Piepho et al., 1998; SAS, 1997; Smith, 1999). For didactical reasons we have chosen to present the mixed models in the context of models for two-way genotype by environment tables, where the environmental dimension is not further developed in terms of a factorial location × year structure. Furthermore, for the same reasons we have ignored the relatively innocent complication of block or spatial structure at the lowest level of variation.

Basic mixed models with variances and covariances independent of environments

In a classical approach towards modelling phenotypic responses in METs a choice is made between two types of models, the difference being the absence or presence of terms for genotype by environment interaction. When the objective of the modelling exercise is the obtainment of information on correlated and direct responses to selection, a simple base line model to start with is the mixed additive model with genotypic main effects taken random and environmental main effects taken fixed. For the phenotypic response of genotype i ($i = 1 \ldots n_g$) in the r-th replicate ($r = 1 \ldots n_r$) of environment j ($j = 1 \ldots n_e$) we write

$$\underline{P}_{ijr} = \mu + \underline{G}_i + E_j + \underline{e}_{ijr}, \qquad (1)$$

where μ is the general mean, $\underline{G}_i$ is the random genotypic main effect (random effects will be underlined), which is assumed to be independently distributed and to have constant variance, E_j is the fixed environmental main effect (with sum to zero identification constraint) and $\underline{e}_{ijr}$ is an error term, again independently distributed. The specification of the mixed model must include an explicit formulation of the variance-covariance structure for the random effects, and thus the response, because writing the response as a sum of fixed and random terms, as in (1) is not sufficient for unequivocal model identification. Even for the simple additive model (1) various choices can be made. Usually the genetic variance, here the variance of $\underline{G}_j$, is assumed to be constant over environments: $\text{var}(\underline{G}_j) = V_G$, or, $V_{G_j} = V_G$, for all environments j. When the error variance is chosen to be constant over environments, $var(\underline{e}_{ijr}) = V_e$, or, $V_{e_j} = V_e$, and the heritability for environment j becomes $h_j^2 = \frac{V_G}{V_G + \frac{V_e}{n_r}}$. As the genetic covariance between environments is defined by $Cov_{G_{jj^*}} = V_G$, the genetic correlation between environments, for homogeneous error variance, becomes $r_{G_{jj^*}} = \frac{Cov_{Gjj^*}}{V_{G_j}^{1/2} V_{G_{j^*}}^{1/2}} = \frac{V_G}{V_G^{1/2} V_G^{1/2}} = 1$, leading to $\frac{CR}{DR} = 1$ (suppressing the standard index, T, for DR and CR). When a mixed additive model of the above specifications, with constant V_G and V_e, fits

well, the conclusion is that selection for whichever target environment can be exercised in whichever selection environment desired without loss of selection response (efficiency). However, even under the mixed additive model, (1), with constant genetic variance and perfect genetic correlation, the ratio of responses to selection, $\frac{CR}{DR}$, will not be equal to 1 whenever the error variance becomes heterogeneous. Actually, in that case $\frac{CR}{DR} = \frac{h_{j^*}}{h_j}$, meaning that selection should be performed in the environment with the lowest experimental error, i.e., the environment with the highest heritability.

The additive mixed model is only rarely satisfactory because it does not allow genotypes to react differentially to changes in the environmental conditions. A straightforward way of extending the additive mixed model is by the inclusion of a random interaction term $\underline{GE}_{ij}$:

$$\underline{P}_{ijr} = \mu + \underline{G}_i + E_j + \underline{GE}_{ij} + \underline{e}_{ijr}, \qquad (2)$$

with the interaction having mean zero and constant variance V_{GE}. $\underline{GE}_{ij}$ effects are assumed to be independent between environments. The genetic variance in environment j is $V_{G_j} = V_G + V_{GE}$. Assuming heterogeneous error variances, the heritability becomes $h_j^2 = \frac{V_G + V_{GE}}{V_G + V_{GE} + \frac{V_{e_j}}{nr}}$. Since the genetic covariance between environments is still $Cov_{G_{jj^*}} = \mathrm{V}_G$, the genetic correlation is $r_{G_{jj^*}} = \frac{V_G}{V_G + V_{GE}}$ making the ratio of responses $\frac{CR}{DR} = r_{G_{jj^*}} \frac{h_{j^*}}{h_j} = \left(\frac{V_G}{V_G + V_{GE}}\right) \frac{h_{j^*}}{h_j}$, with j the target environment and j^* the selection environment. As in the additive mixed model, (1), in the mixed model with interaction, (2), all pairs of environments are genetically equally correlated, but in contrast to (1) the correlation is not perfect anymore. Model (2) is referred to in the literature as the uniform correlation model (Cullis et al., 1998), or the compound symmetry model (Neter et al., 1996; SAS, 1997).

The uniform correlation model could be useful when the genetics underlying the phenotypic response are as follows. On the one hand there is a group of genes that consistently expresses itself under all environmental conditions, these are the genes responsible for V_G. On the other hand there are groups of environmentally specific genes that express themselves in just one environment, their behaviour over environments is described by V_{GE}. The environment specific genes cause the genetic correlation to be less than perfect.

When error variances are homogeneous, $\mathrm{V}_{e_j} = \mathrm{V}_e$, in both model (1) and model (2), direct responses due to selection in target environments will always be at least as large as correlated responses due to selection in other environments. However, neither model (1), nor model (2) is very credible for real life data (see next section for motivation). For the description of actual data, more elaborate models are needed for adequate description of variances, covariances and correlations.

Mixed models with variances and covariances depending on environments

In models (1) and (2), genetic variances and covariances (and error variances) were assumed to be constant over environments. We now define the unstructured mixed model as a model with genetic variances being typical for each environment j, and genetic covariances being typical for each pair of environments, j and j^*. The model can be written as

$$\underline{P}_{ijr} = \mu + \underline{G}_{ij} + E_j + \underline{e}_{ijr}, \qquad (3)$$

where $\underline{G}_{ij}$ represents the genetic (main) effect in environment j, with $var(\underline{G}_{ij}) = V_{G_j}$, and $cov(\underline{G}_{ij}, \underline{G}_{ij^*}) = Cov_{G_{jj^*}}$, making $r_{G_j j^*} = \frac{Cov_{G_{jj^*}}}{V_{G_j}^{1/2} V_{G_{j^*}}^{1/2}}$. The error variance can be made environment specific as well, $var(\underline{e}_{ijr}) = V_{e_j}$. As a consequence $h_j^2 = \frac{V_{G_j}}{V_{G_j} + \frac{V_{e_j}}{nr}}$. The ratio $\frac{CR}{DR}$ can be found upon substitution of the genetic correlation and the square root heritabilities in $\frac{CR}{DR} = r_{G_{jj^*}} \frac{h_{j^*}}{h_j}$. In model (3) there is no model-induced restriction on the $\frac{CR}{DR}$ ratio. When this ratio is bigger than 1 for a target environment j and a selection environment j^*, it will be worthwhile to select in another environment than the target environment. For example, when the genetic correlation is 0.8 and the heritability in the selection environment is a factor $1/0.8^2 = 1.5625$ larger than that in the target environment, it would be advisable to perform the selection not in the target environment. Such situations do occur in METs, see Cooper et al. (1997). A good example of the application of model (3) can be found in Cooper & DeLacy (1994). As we will see below there are also indications that these situations occurred in the ICARDA barley trials.

The unstructured model may be appropriate when there are differences in the sets of genes that are responsible for performance in each of the environments

sampled in the MET. Usually, there is a common set of genes expressing itself to a certain degree in every environment, which causes genetic correlations to be moderately to highly positive. In addition to the common set of genes shaping the basic response, environment specific sets of genes may be required for specific adaptations. In some cases, alternative alleles of genes may be better suited to the conditions of different environments and could give rise to negative genetic correlations between environments. For example, where a gene is important in determining flowering time and alternative alleles condition early and late flowering time, under environmental conditions of terminal drought stress, the allele determining early flowering may be best suited, whereas in environments without drought, the allele determining late flowering may be better suited. However, it is also possible that there is a mixture of different environmental factors influencing genotype performance at a particular site-year combination, referred to here as an environment, where the genes respond to some of the environmental factors that characterize the environment type. As one example, take rainfall and level of drought, where the phenotypic response is based on genes responsible for traits contributing to drought resistance. As another example, take incidence of disease, where genes related to disease resistance determine the response. For such genotype-environment systems there is merit in investigating the variance-covariance structure describing the cross classification of environment types with genotype response patterns. For this purpose a number of approaches can be adopted. Cooper & DeLacy (1994) discussed the use of pattern analysis (the combined use of cluster and ordination methods). Here we consider the specification of a variance-covariance model, following approaches described in Denis et al. (1997), Piepho (1997) and Smith (1999).

In between the uniform correlation model with homogeneous genetic variance (2) and the fully unstructured model (3), two intermediate models can be defined. First, we can allow heterogeneity of variance over the environments by giving each environment its own interaction variance, V_{GE_j}, but retain a common genetic covariance term in the form of V_G. Thus, genetic variances, $V_G + V_{GE_j}$, are environment dependent, genetic covariances are uniform, V_G, while genetic correlations are non-uniform, $\frac{V_G}{(V_G+V_{GE_j})^{1/2}(V_G+V_{GE_{j^*}})^{1/2}}$. Second, we can allow heterogeneity of genetic variance by giving each environment its proper genetic variance, V_{G_j}, while assuming the genetic correlation, ρ, to be uniform, thus forcing the genetic covariances to be equal to $\rho V_{G_j}^{1/2} V_{G_{j^*}}^{1/2}$. Both of these models have limited practical value as they are not very flexible and still contain heavy restrictions on the covariances and/or correlations. However, for sets of environments that are not too heterogeneous they may sometimes provide an adequate description.

A drawback of the use of the unstructured model (3) is the large amount of variance-covariance parameters that must be fitted. Standard MET data may not permit all parameters to be estimated with sufficient precision. A class of models that combines high flexibility in modelling the variance-covariance structure with less demands on the amount of data than model (3), is the class of mixed multiplicative models, also called factor-analytic models (Oman, 1991; Gogel, 1995; Piepho, 1997; Smith, 1999). The model for the response is

$$\underline{P}_{ijr} = \mu + (\sum_{k=1}^{K} \underline{f}_{ik}\lambda_{jk} + \underline{d}_{ij}) + E_j + \underline{e}_{ijr}. \quad (4)$$

In comparison with (3), the term $\underline{G}_{ij}$ is replaced by a sum of multiplicative terms of the type $\underline{f}_{ikjk}$ ($k = 1 \ldots K$) and an independently distributed residual, $\underline{d}_{ij}$, where K is chosen such as to minimize the discrepancy between the variance-covariance structure of (4) with that of the unstructured model (3). The parameters $\underline{f}_{ik}$ can be interpreted as random genotypic sensitivities to hypothetical environmental variables λ_{jk}, although one must be cautious not to restrict interpretation to individual multiplicative terms, since these terms are only correctly interpreted when interpreted simultaneously (van Eeuwijk, 1995). Parameters in (4) can be estimated by REstricted Maximum Likelihood, or REML (Piepho, 1997; Smith, 1999). For convenience the variance of the $\underline{f}_k$ is often chosen to be 1, $\text{var}(\underline{f}_k) = V_{f_k} = 1$. The variance of the residual $\underline{d}_{ij}$ is, for most practical purposes, best taken as being dependent on the environment, $\text{var}(\underline{d}_{jj}) = V_{d_j}$. Then the genetic variance for environment j is $V_{G_j} = \sum_{k=1}^{K} \lambda_{jk}^2 + V_{d_j}$. With heterogeneous error variance, $var(\underline{e}_{ijr}) = V_{e_j}$, and the expression for the heritability is $h_j^2 = \frac{\sum_{k=1}^{K} \lambda_{jk}^2 + V_{d_j}}{\sum_{k=1}^{K} \lambda_{jk}^2 + V_{d_j} + \frac{V_{e_j}}{r}}$. The genetic covariance between the environments j and j^* is $Cov_{G_{jj^*}} = \sum_{k=1}^{K} \lambda_{jk}\lambda_{j^*k}$, lead-

ing to $r_{G_{jj^*}} = \frac{\sum_{k=1}^{K} \lambda_{jk}\lambda_{j^*k}}{\left(\sum_{k=1}^{K} \lambda_{jk}+V_{d_j}\right)^{1/2}\left(\sum_{k=1}^{K} \lambda_{j^*k}+V_{d_{j^*}}\right)^{1/2}}$ for the genetic correlation between environments j and j^*. When all V_{d_j}'s are zero, expressions simplify: $V_{G_j} = \sum_{k=1}^{K} \lambda_{jk}^2$ and $r_{G_{jj^*}} = \frac{\sum_{k=1}^{K} \lambda_{jk}\lambda_{j^*k}}{\left(\sum_{k=1}^{K} \lambda_{jk}^2\right)^{1/2}\left(\sum_{k=1}^{K} \lambda_{j^*k}^2\right)^{1/2}}$, and for $K = 1$ genetic correlations will all be perfect, i.e., $r_{G_{jj^*}} = 1$. Model (4) provides flexibility in modelling genetic correlations and variances, like model (3), but at a considerably lower price, because fewer variance-covariance parameters need estimation. The intermediate models with heterogeneous variance and either uniform covariance or uniform correlation are special cases of a multiplicative model with only one term ($K = 1$). The uniform covariance model (with heterogeneous variance) results from a multiplicative model with one term when λ_j is constant over environments, or, $\lambda_j = V_G^{1/2}$. The uniform correlation model is similarly found from a factor-analytic model containing one multiplicative component and constant λ_j, but now after data have been environment standardized.

Comparison of mixed model fits and assessment of an adequate number of multiplicative terms can be done for nested models by comparing deviances (-2 × loglikelihood). Akaike's lnformation Criterion (AIC; loglikelihood – number of parameters) is useful for comparing both nested and non-nested models (SAS, 1997). Differences in deviance are approximately Chi-square distributed with the degrees of freedom given by the difference in the number of parameters. An exception occurs when the variances are constrained to be non-negative under the null hypothesis, because the distribution for the difference in deviances will then have a more complicated form (Stram & Lee, 1994).

A convenient graphical tool in the search for an appropriate variance-covariance model is the biplot. The biplot is a simultaneous display of genotypes and environments providing approximations to the genotypic responses in particular environments, and to variances per environment, and correlations between environments (Gabriel, 1971; Gower & Hand, 1996). For application rules in plant breeding see Kempton (1984), van Eeuwijk (1995), and DeLacy et al. (1996). We will use a special type of biplot suitable for exploring the variance-covariance structure of data, a variance-covariance plot. Environments are depicted by arrows starting at the origin and the (squared) lengths of the arrows are proportional to variances, while the (cosines of the) angles between the arrows are indicative of correlations. Acute angles stand for high correlations, obtuse angles indicate low correlations, and orthogonal angles represent zero correlations (in the approximation).

Useful references for more extensive treatments of mixed models with factor analytic variance-covariance structures are Gogel et al. (1995), Longford (1993), Piepho (1997), Piepho et al. (1998) and Smith (1999).

Data description

The data used for illustration were collected from a series of barley trials conducted by ICARDA. The trials were designed to investigate the ability of farmers to select among suitable germplasm for their own conditions. The barley production environments of Syria have predominantly winter rainfall and range from well watered (over 450 mm rainfall in the growing season, winter) to areas where water stress is severe (down to 150 mm rainfall in the growing season). In the semi-arid areas, where barley is the predominant crop, important abiotic stresses consist in low annual rainfall, unfavourable rainfall distribution, low winter temperatures, and high temperatures coupled with hot winds from anthesis to grain filling. Although both the frequency and the duration of these stresses varies from place to place and from year to year, pre-anthesis water stress is common and post anthesis water stress is the rule, though each increases in frequency as the average annual rainfall decreases.

Nine farms covering a range of water stress environments were selected for trials (Table 1, locations 1 to 9). In 1996 a wide range of 208 breeding lines thought to cover the spectrum of responses necessary to cope with the spectrum of possible water stresses was planted on each of the nine farms and in addition at two research sites; Breda, a research site leased from a local landholder (Table 1, location 11), and Tel Hayda, a research station at ICARDA headquarters (Table 1, location 12). Overall, the 11 sites cover an area of over 500 Km in length, from Central to North East Syria. Based on cropping patterns, long term rainfall data, and types of barley grown, sites 1, 2, 9 and 12 are relatively high yielding sites growing the white seeded (more productive) barley landrace. Locations 6 and 8 also grow the white seeded landrace, but yields are limited by poor soils. Eventually, sites 3, 4, 5, and

Table 1. Location statistics with V_p = phenotypic variance, V_g = genetic variance, V_e = error variance, and h^2 is heritability. V_e is estimated from 1997/1998 trials at the same location

Location identifier	Location name	Rain	Mean	V_p	V_g	V_e	h^2
1	Abbien	436	3.248	1.368	1.144	0.224	0.837
2	Ebla	460	2.857	0.701	0.506	0.195	0.722
3	Tel Brak	278	3.685	0.984	0.728	0.256	0.740
4	Jern al Aswad	284	1.415	0.534	0.442	0.092	0.827
5	Belounan	192	0.280	0.037	0.021	0.016	0.557
6	Al Bab	350	0.376	0.049	0.027	0.022	0.542
7	Melabya	241	0.713	0.172	0.128	0.044	0.742
8	Salamia	248	1.017	0.267	0.202	0.065	0.757
9	Souran	303	2.515	0.436	0.266	0.170	0.610
11	Breda	233	0.811	0.067	0.016	0.051	0.235
12	Tel Hadya	434	4.495	0.826	0.510	0.316	0.618
Mean		314	1.947	0.495	0.363	0.132	0.653

7 represent the more marginal environments where the more drought tolerant black-seeded landrace is grown.

Each of the 11 trials grown in the season of 1996/1997 consisted of an unreplicated 4×52 grid without checks. To facilitate farmers to observe changes in performance between sites, the order of the lines was the same at each of the 11 sites. The 208 entries included 158 two-row and 50 six-row types, 100 fixed lines and 100 segregating bulks (F3 bulks), and 8 landrace populations, 161 white grain, 28 black grain and 19 lines segregating for grain colour, and with a range of maturity from 105 to 136 days at Tel Hayda in 1997 (Ceccarelli et al., 2000). The entries were deliberately chosen to represent a wide range of maturity, plant height, tillering, spike length and shape, in order to test farmers' preferences.

The entries selected during the 1996/97 season, were grown again in 1997/98 on the same nine farms and two research sites. The 1997/1998 trials were unreplicated with systematic checks every ten plots, and in the first and last plot. In this paper we will look primarily at yield data from the 1996/1997 trials in an attempt to estimate genetic variances and correlations, which we need for a comparison of direct responses due to selection at farms proper with indirect responses following from selection at the research stations. It should be remarked that our analysis is slightly at odds with the actual goal of the trials, which was the comparison of the selection behaviour of the farmers with that of the official breeder.

Analyses of ICARDA data

The unreplicated nature of the individual trials in combination with the absence of randomization made a rigid statistical analysis rather difficult. For most statistical procedures observations should be independent, and non-randomization may violate this assumption. However, the non-randomization was probably not too undermining for subsequent analyses, because competition between neighbouring plots was believed to be absent or of very low intensity. Some support for this assumption was given by the results of the fit of a spatial model for the lowest level of variation in the data (AR(1) × AR(1); Cullis & Gleeson, 1991), where only a weak error correlation of 0.35 between neighbouring columns was found, while correlation between rows was almost absent, 0.05. Furthermore, as trials were laid out at different farms no consistent benefit for particular lines from common fertility trends can have occurred.

An independent error estimate is a prerequisite for the assessment of differences between lines and for the estimation of genetic variances (= phenotypic variance – error variance). To this end, it is recommended to always include partial or complete replications in at least some trials representative of PPB and FPB conditions and management. The non-replicated character of the trials of our example data forced us to come up with some ad hoc solutions for finding error estimates. Two different strategies were followed. The first approach

Table 2. Results of fits of various variance-covariance models

Model nr	Nested in	Model description	Variances	Covariances	Correlations	Nr param	Deviance	Dev. Diff	Df	AIC
1	2	V_g+V_{ge}	Homogeneous	Uniform	Uniform	2	4850.3			–2427
2	4	V_g+$diag(V_{ge})$	Heterogeneous	Uniform	Specific	12	3646.6	1203.7	10	–1835
3	4	diag(V_g)+uniform correlation	Heterogeneous	Specific	Uniform	12	3616.8	1233.5	10	–1820
4	5	FA1+diag(residual)	Heterogeneous	Specific	One	22	3449.5	197.1	10	–1747
5	6	FA2+diag(residual)	Heterogeneous	Specific	Specific	32	3377.8	71.7	10	–1721
6	7	FA3+diag(residual)	Heterogeneous	Specific	Specific	41	3345.4	32.4	9	–1714
7		Unstructured VCOV	Heterogeneous	Specific	Specific	66	3306.7	38.7	25	–1719

was as follows. Individual error variances for the 1996/1997 trials were predicted from the 1997/1998 trials. The 1997/1998 trials were replicated for some checks and thus an error could directly be estimated for these trials. The errors over the 1997/1998 trials were subsequently regressed on the trial means of the 1997/1998 trials by means of a generalized linear regression (log link, gamma distribution; see McCullagh & Nelder, 1989). The parameters for this mean – variance relationship together with the 1996/1997 trial means were subsequently used to estimate error variances for the 1996/1997 trials. Genetic variances for individual 1996/1997 trials (unstructured model) were obtained by subtraction of the predicted error from the observed phenotypic variances (Table 1). This scenario led to rather high heritabilities, higher than commonly found. A second way of separating genetic variances from the observed phenotypic variances was by the use of factor-analytic models for the variance-covariance structure of the 1996/1997 data as a whole (over all trials). For unreplicated data, the estimates for the residual variance, $\hat{V}_{d_j}$, will be the sum of a specific genetic variance and experimental error. The only way to disentangle these confounded variances is by introducing the, admittedly strong, assumption of the genetic part in $\hat{V}_{d_j}$ being zero, making $\hat{V}_{d_j}$ to represent exclusively experimental error, $\frac{\hat{V}_{e_j}}{n_r}$. Then, genetic variances can become equal to the sum over the multiplicative terms, $V_{G_j} = \sum_{k=1}^{K} \lambda_{jk}^2$. Heritability estimates will be biassed downwards by the assumption of $\hat{V}_{d_j}$ to be an estimate of experimental error.

In Table 1 we present some basic statistics for the individual trials. V_P is the phenotypic variance, calculated as the variance over the 208 entries for each trial. The environments differed considerably in phenotypic variance. V_e was obtained from the regression equation described in the last section. The heritability, h^2, is calculated from the quotient V_G/V_P. Heritabilities appear to be rather high, probably because the estimates for V_{e_j} are too low, but also because lines were selected to represent a wider range of genetic variation than is common for both FPB and PPB trials. V_P and mean were correlated (Pearson product moment correlation = 0.86), so trials with higher mean yields also exhibited more phenotypic variation. Mean yield and phenotypic variance were positively related to average rainfall, 0.69 and 0.67, respectively. Heritability was not highly correlated to mean yield or rain, 0.34 and 0.31, respectively.

As the phenotypic and genetic variances varied strongly (Table 1) it was not to be expected that an additive model or a uniform correlation model would fit well.

This expectation was confirmed. In Table 2 the deviance (-2 × loglikelihood) for a model based on V_G + V_{GE} (Table 2, model 1) was very much higher, 1203.7 on 10 df ($p \ll 0.0001$), than that of a model allowing heterogeneity of the interaction variance; V_G + V_{GE_j} (Table 2, model 2), whereas the latter model provided itself a very poor fit when compared with more elaborate models (Table 2, models 4 to 7). Tests on the necessity of more variance-covariance terms can be constructed by taking the difference in deviance between the nested and nesting model, for example 2–4, and compare this difference with the percentiles of a Chi-square distribution with df equal to the difference in number of parameters. Strictly speaking the estimated interaction variances $\hat{V}_{GE}$ and $\hat{V}_{GE_j}$ contain both genotype by environment interaction and pure error. We could have used our error estimates to find unbiassed estimates for the interaction variances. However, this exercise is futile in the light of the bad fit of the models 1 and 2 of Table 2. (Note that the deviances and AICs in Table 2 ignore the estimation of

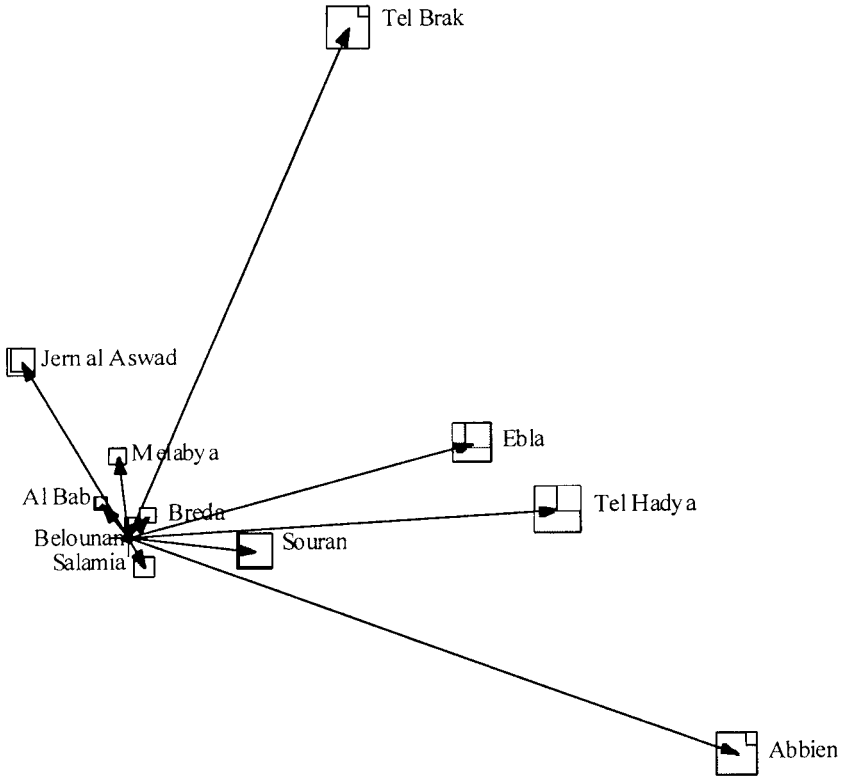

Figure 1. Plot of variances and correlations for yield. Total described variation over environments, 60%. Squares prop. to mean yield, cut-outs prop. to non-described variation per environment. Lengths arrows prop. to standard deviation, angles prop. to correlation.

trial errors.) Also the model with heterogeneous variance and uniform correlation (Table 2, model 3) was found to be inadequate in comparison with a factor-analytic model with one (Table 2, model 4) or more terms (Table 2, model 5 and 6), or in comparison with the unstructured model (Table 2, model 7). (Model 3 is only nested in model 4 when the data are first environment standardized, however, we will ignore this complication here.) Table 2 shows that the pattern of heterogeneity of variances and non-uniformity of covariances and correlations required either a completely unstructured variance-covariance model (Table 2, model 7) or a close approximation as given by the factor analytic model with 3 multiplicative terms (Table 2, model 6). The p-value for the difference between model 6 and model 7 was 0.04, proving the proximity of the models. The AIC puts a penalty on the number of parameters where the deviance does not. Model 6 is therefore preferred over model 7 when AIC would be used as the criterion for model choice.

Like the testing procedure described above a variance-covariance plot of the phenotypic data also revealed the severe heterogeneity of both variances and correlations (Figure 1). The lengths of the environmental arrows give the impression that the variances at Tel Brak, Ebla, Tel Hadya and Abbien were considerably higher than those at the other trials (the longer the arrow, the higher the standard deviation). The sizes of the squares show that these trials had also higher mean yields (the larger the square the larger the mean yield). The cut-outs in the upper-right corners tell us that in Figure 1 especially the high mean yield (= high variance) trials are well represented, whereas the low mean yield (= low variance) trials are very poorly represented (the larger the cut-out, the lower the proportion of variation represented). Figure 1 contains 60% of the total variation in the '208 lines $\times$ 11 environments' table of observations. As an overall measure 60% is acceptable, but inevitably some environments will be badly represented, and this will especially be true for the low variance environments. For that reason plots like Figure 1 should only be used to generate ideas about plausible structures, but these ideas should always be verified by going back to the data

Table 3. Phenotypic correlations above diagonal, genetic correlations below diagonal. Unstructured model

		1 Abbien	2 Ebla	3 Tel Brak	4 Jern al Aswad	5 Belounan	6 Al Bab	7 Melabya	8 Salamia	9 Souran	11 Breda	12 Tel Hady
1	Abbien		0.49	0.11	–0.27	–0.05	–0.26	–0.10	0.07	0.19	0.04	0.59
2	Ebla	0.63		0.31	–0.03	0.08	–0.01	0.07	0.13	0.29	0.10	0.45
3	Tel Brak	0.13	0.43		0.10	0.11	0.11	0.25	–0.06	0.03	0.15	0.29
4	Jern al Aswad	–0.32	–0.04	0.13		–0.03	0.19	0.09	–0.03	–0.05	0.07	–0.15
5	Belounan	–0.07	0.13	0.17	–0.05		0.05	0.00	–0.14	0.11	0.16	0.07
6	Al Bab	–0.39	–0.02	0.17	0.28	0.09		0.28	–0.06	0.06	0.07	–0.17
7	Melabya	–0.13	0.09	0.33	0.12	0.00	0.44		–0.12	–0.13	–0.11	–0.05
8	Salamia	0.08	0.18	–0.07	–0.03	–0.22	–0.10	–0.16		0.00	0.08	–0.02
9	Souran	0.26	0.43	0.05	–0.07	0.19	0.10	–0.19	0.00		0.21	0.28
11	Breda	0.08	0.25	0.36	0.15	0.46	0.20	–0.26	0.18	0.54		0.19
12	Tel Hadya	0.83	0.67	0.43	–0.21	0.12	–0.29	–0.08	–0.03	0.45	0.51	

themselves, in this case the matrix of variances and covariances/ correlations (see Table 1 for phenotypic variances and Table 3 for phenotypic correlations). Correlations between trials can be observed in Figure 1 as angles between environmental arrows. Thus the pair Tel Hadya – Abbien seems positively correlated, just like the pair Tel Hadya – Ebla. In contrast, Abbien and Tel Brak seem uncorrelated (orthogonal angle). For trials other than Tel Brak, Ebla, Tel Hadya and Abbien, conclusions about variances and correlations should be drawn with some care due to the low quality of representation of these trials. Badly represented trials will generally have low variance. With respect to the correlations of badly represented trials we can at least say that the correlations between the well represented trials and the badly represented trials will be low, while we can not conclude very much about the correlations between badly represented trials. The overall lesson from Figure 1 is that an elaborate model is necessary for an adequate description of variances and correlations. This is the same conclusion as arrived at by explicit fitting of the models and by comparing deviances and AICs. The utility of Figure 1 is that it provides a quick and dirty method, where explicit modelling can be time-consuming. We advise to use a plot like that in Figure 1 as a screening device to reduce the number of candidate models.

On the basis of AIC and deviance the unstructured model and the factor analytic model with 3 multiplicative terms were selected as adequate variance-covariance models for the trial data. Descriptive statistics for the unstructured model are given in Tables 1 and 3. Similar information for the factor analytic model (with 3 multiplicative terms) can be found in the Tables 5 and 6. Although the fit of both models to the observed (phenotypic) variance-covariance was very comparable, derived quantities as genetic variances and correlations, error variances and heritabilities deviate substantially. The main reason for that is our choice to estimate trial error variances for use with the unstructured model from the 1997/1998 trials (by using the estimated coefficients of the 1997/1998 regression of trial error variance on trial mean for prediction of the 1996/1997 trials), while we estimate the trial errors for use with the factor analytic model from the residual variances, V_{d_j}, within the factor analytic model. The latter being a natural option proper to factor analytic models.

Heritabilities as presented in Table 5 (factor-analytic model) correspond better with experience (Tinker et al., 1996) than those of Table 1 (unstructured model). The differences in genetic parameters between the unstructured model and the factor analytic model have strong consequences for the evaluation of the *CR/DR*-ratios, the ultimate aim of the whole modelling exercise. Tables 4 and 7 show the *CR/DR*-ratios following from the unstructured model and the factor analytic model, respectively. Rows are indexed by selection environment, columns by target environment. For example, Table 4 shows that selection at Tel Hadya will result in an indirect response at Abbien that is only 0.71 of the response that would have been obtained by direct selection at Abbien, when calculations are based on the unstructured model. Concentrating

Table 4. Unstructured model. Ratio *CR/DR*. Rows = selection environment, columns = target environment

		1 Abbien	2 Ebla	3 Tel Brak	4 Jern al Aswad	5 Belounan	6 Al Bab	7 Melabya	8 Salamia	9 Souran	11 Breda	12 Tel Hady
1	Abbien	1	0.68	0.14	–0.32	–0.09	–0.48	–0.14	0.09	0.31	0.16	0.96
2	Ebla	0.59	1	0.42	–0.04	0.15	–0.02	0.09	0.18	0.47	0.43	0.72
3	Tel Brak	0.13	0.43	1	0.13	0.20	0.20	0.33	–0.07	0.05	0.64	0.47
4	Jern al Aswad	–0.32	–0.04	0.14	1	–0.06	0.35	0.13	–0.03	–0.08	0.28	–0.24
5	Belounan	–0.06	0.12	0.15	–0.04	1	0.09	0.00	–0.19	0.18	0.70	0.11
6	Al Bab	–0.31	–0.02	0.15	0.23	0.09	1	0.37	–0.08	0.09	0.30	–0.27
7	Melabya	–0.12	0.09	0.33	0.11	0.00	0.51	1	–0.16	–0.21	–0.47	–0.08
8	Salamia	0.08	0.19	–0.08	–0.03	–0.26	–0.12	–0.16	1	0.00	0.33	–0.04
9	Souran	0.23	0.40	0.04	–0.06	0.19	0.10	–0.17	0.00	1	0.88	0.45
11	Breda	0.04	0.14	0.20	0.08	0.30	0.13	–0.15	0.10	0.34	1	0.32
12	Tel Hadya	0.71	0.62	0.39	–0.18	0.12	–0.31	–0.07	–0.03	0.46	0.83	1

Table 5. Location statistics with genetic and error variance derived from factor analytic model with 3 terms

Location identifier	Location name	Mean	V_p	V_g	V_e	h^2
1	Abbien	3.248	1.368	1.096	0.273	0.801
2	Ebla	2.857	0.701	0.309	0.392	0.440
3	Tel Brak	3.685	0.984	0.328	0.655	0.334
4	Jern al Aswad	1.415	0.534	0.069	0.465	0.130
5	Belounan	0.280	0.037	0.003	0.034	0.089
6	Al Bab	0.376	0.049	0.011	0.038	0.226
7	Melabya	0.713	0.172	0.092	0.080	0.534
8	Salamia	1.017	0.267	0.006	0.261	0.021
9	Souran	2.515	0.436	0.086	0.350	0.198
11	Breda	0.811	0.067	0.017	0.049	0.257
12	Tel Hadya	4.495	0.826	0.468	0.359	0.566
Mean		1.947	0.495	0.226	0.269	0.327

on *CR/DR*-ratios for the research centres Breda and Tel Hadya we note that Table 4 does not give arguments for using Breda as selection environment for farmers fields as none of the *CR/DR*-ratios exceeds one. Recalling that $\frac{CR}{DR} = \frac{h_S r_{G_{ST}}}{h_T}$ and observing that heritability, h_S^2, at Breda was low, 0.235, it could be expected that there would be no farmer's trials for which it would make sense to select at Breda. For Tel Hadya the situation was slightly different as the heritability here was 0.618, implying that when the genetical correlation, $r_{G_{ST}}$ would not be too low, it might be beneficial to perform selection at Tel Hadya instead of at a farm. The only farms possessing a sizeable genetic correlation with Tel Hadya are Abbien and Ebla, but because these farms had both higher heritabilities than Tel Hadya the *CR/DR* ratio remains smaller than one, and no argument for selection at Tel Hadya does arise.

When we now turn to the inspection of *CR/DR*-ratios following from the factor analytic model (Table 7), we observe that selection for Ebla could just as well take place at Tel Hadya, *CR/DR* = 1.08, and selection for Salamia and Souran could probably better take place at Tel Hadya, with *CR/DR* = 1.92 and 1.38, respectively. With respect to Breda, selection for Souran can equally well be done at Breda as at

Table 6. Genetic correlations from factor analytic model with 3 terms

		1 Abbien	2 Ebla	3 Tel Brak	4 Jern al Aswad	5 Belounan	6 Al Bab	7 Melabya	8 Salamia	9 Souran	11 Breda
2	Ebla	0.81									
3	Tel Brak	0.23	0.75								
4	Jern al Aswad	–0.81	–0.32	0.38							
5	Belounan	–0.15	0.32	0.70	0.57						
6	Al Bab	–0.64	–0.10	0.56	0.94	0.49					
7	Melabya	–0.16	0.22	0.55	0.47	–0.02	0.73				
8	Salamia	0.55	0.08	–0.49	–0.80	–0.21	–0.94	–0.90			
9	Souran	0.50	0.68	0.57	–0.12	0.74	–0.15	–0.37	0.38		
11	Breda	0.07	0.38	0.56	0.29	0.94	0.17	–0.32	0.13	0.90	
12	Tel Hadya	0.88	0.95	0.59	–0.48	0.33	–0.33	–0.10	0.37	0.81	0.49

Table 7. Ratio *CR/DR* from factor analytic model. Rows = selection environment, columns = target environment

		1 Abbien	2 Ebla	3 Tel Brak	4 Jern al Aswad	5 Belounan	6 Al Bab	7 Melabya	8 Salamia	9 Souran	11 Breda	12 Tel Hady
1	Abbien	1	1.10	0.36	–2.02	–0.45	–1.20	–0.20	3.37	1.01	0.12	1.05
2	Ebla	0.60	1	0.86	–0.60	0.70	–0.13	0.20	0.38	1.02	0.50	0.84
3	Tel Brak	0.15	0.66	1	0.61	1.35	0.69	0.43	–1.92	0.74	0.64	0.45
4	Jern al Aswad	–0.33	–0.18	0.24	1	0.69	0.71	0.23	–1.97	–0.10	0.21	–0.23
5	Belounan	–0.05	0.14	0.36	0.47	1	0.31	–0.01	–0.42	0.50	0.55	0.13
6	Al Bab	–0.34	–0.07	0.46	1.24	0.78	1	0.48	–3.06	–0.16	0.16	–0.21
7	Melabya	–0.13	0.24	0.69	0.95	–0.06	1.13	1	–4.47	–0.61	–0.46	–0.10
8	Salamia	0.09	0.02	–0.12	–0.33	–0.10	–0.29	–0.18	1	0.12	0.04	0.07
9	Souran	0.25	0.46	0.44	–0.15	1.11	–0.14	–0.22	1.15	1	0.79	0.48
11	Breda	0.04	0.29	0.49	0.41	1.60	0.18	–0.22	0.47	1.02	1	0.33
12	Tel Hadya	0.74	1.08	0.77	–1.01	0.83	–0.53	–0.11	1.92	1.38	0.73	1

Souran itself, *CR/DR* = 1.02, while selection for Belounan could better be performed at Breda, *CR/DR* = 1.60. There was no evident relationship between the size of the *CR/DR*-ratio and mean yield or rainfall. One would expect that the high yield/ high rainfall environment of Tel Hadya would be suitable for indirect selection for other high yield and/or high rainfall environments. Similarly, the low yield/ low rainfall environment of Breda might be expected to be suitable for indirect selection for other low yield/ low rainfall environments. These suppositions were not confirmed, even though Tel Hadya seems an unsuitable selection site for marginal environments (3, 4, 5, 7, and 11), and Breda seems unsuitable to select for the high yielding environments with the exception of Souran.

The general conclusion from the *CR/DR*-ratios in Table 7 is that sometimes trials at research centres could serve equally well for genetic improvement at particular farms as selection at these farms themselves, but at other times direct selection at the farm could not be replaced by selection at the research centre. Conditions at the research centres often did not seem to mimick those of the target environments on farm. We were unable to identify the conditions under which selection at research centres does at least as well as selection on farm.

Final comments

A decision on what will be the best breeding strategy depends on the statistical model for the variance-

covariance structure. Therefore, it is important to select an adequate model. Such a selection can be based on a combination of exploratory, informal techniques like the biplot and formal tests based on the comparison of deviances of nested models. In this paper we have restricted ourselves to models that are principally descriptive. In the unstructured model, variances and covariances/ correlations are defined for individual environments and pairs of environments. No further interpretation is possible. In the factor-analytic model these variances and covariances are approximated by sums of product terms and residual variances. The product terms allow some interpretation of the variance-covariance generating mechanisms. The λ_{jk}'s, environmental scores, can be interpreted as hypothetical environmental variables to which the genotypes differ in sensitivity ($\underline{f}_{ik}$). We can try to interpret these hypothetical environmental variables by regressing them on explicit environmental variables. The best way to do this is by multivariate multiple regression of the environmental scores on a number of explicit environmental variables. The aim is to replace the hypothetical variables of the factor analytic model by interpretable environmental variables in a (random) regression model. The result of such an exercise would make genetic variances, correlations and heritabilities dependent on known environmental variables (Denis et al., 1997). The identification of the most important environmental variables shaping genetic variances and correlations would allow us to calculate *CR/DR*-ratios from just the values on these environmental variables, and to assess the feasibility of selection in other environments than the target environment, for example, at research stations for farmers. An important issue in such an approach is the characterisation of the environment in terms that are meaningful to the plant. Especially the factors that induce stresses and the frequencies with which they occur should be revealed. Interesting work in this direction has recently been done for grain sorghum in northeast Australia by Chapman et al. (2000a,b,c).

Unfortunately, for our barley example data we were unable to identify environmental variables that could form the basis for a description of the variance-covariance structure. We had to be content with the factor analytic model with its hypothetical environmental variables. The latter model described the variance-covariance structure adequately with a moderate amount of parameters and at least allowed us to get some insight in the suitability of research centres as selection environments for farm conditions. The reasons for our failure to identify the conditions for successful indirect selection can be the following. Firstly, we didn't have much of a description of the meteorological and soil conditions in the trials. In that sense the approach followed by Chapman et al. (2000a,b,c) to characterize environments by kinds and frequencies of stresses might be used as precursor to the statistical analyses presented in this paper. Secondly, the trials, both for the research centres and the on farm trials, were not replicated, and data from only one year were analyzed. As heritabilities and genetic correlations were calculated for individual trials, the precision of the estimates for these parameters was low, which in turn complicated the modelling of variances and correlations on environmental variables. Furthermore, individual trials varied so heavily from each other that it was not obvious how to group trials to arrive at more precise estimates by using averages over trials.

We have restricted ourselves to multi-location data from one year, to illustrate the statistical machinery for modelling genetic variance-covariance structures in a slightly simplified context. Extending the analyses to multi-location multi-year data does not present new methodological issues. Multiplicative models like those described above can be formulated for each type of genotype by environment interaction occurring in multi-environment trials, be it genotype by location interaction, genotype by year interaction, or genotype by location by year interaction (Piepho & van Eeuwijk, 2001). A simpler, but often satisfactory, strategy would be to first fit a three-way mixed model to genotype by location by year data, and use the three-way model to form a two-way genotype by location table of best linear unbiassed predictions. The latter two-way table can then be analyzed by the two-way methods described in this paper.

Finally, the issue is not so much to compare participatory and formal plant breeding approaches as if they were alternative, mutually exclusive, approaches, but rather to develop a common framework and language that can help in improving on farm production against minimal costs. As soon as we have knowledge of genetic variances and correlations and as soon as we know how these quantities depend on the environment, we will be able to design an optimal breeding strategy. It is to be expected that conditions on research centres are such that here higher heritabilities can be realized than at farmer's fields. In that case centralized breeding would be the preferred option provided that the genetic correlation between central trials and farmer's fields is substantial. The key is-

sue is to define the target environment as precisely as possible. When it is impossible to mimick farmers fields at research stations, the only way ahead in a sensible breeding strategy is to include the particular farmer's fields explicitly in the evaluation trials coordinated by research centres. Thus on farm trials can be completely compatible with formal plant breeding. For a successful application of this idea see Bänziger & Cooper (2001).

References

Atlin, G.N., M. Cooper, A. Bjørnstad & F.A. van Eeuwijk, 2001. A comparison of formal and participatory breeding approaches using selection theory. Euphytica: This Volume.

Bänziger, M. & M. Cooper, 2001. Breeding for low-input conditions and consequences for participatory plant breeding – Examples from tropical maize and wheat. Euphytica: This Volume.

Ceccarelli, S., 1996. Positive interpretation of genotype by environment interactions in relation to sustainability and biodiversity. In: M. Cooper & G.L. Hammer (Eds.), Plant Adaptation and Crop Improvement, pp. 467–486. CAB International, Wallingford, England.

Ceccarelli, S., S. Grando, R. Tutwiler, J. Baha, A.M. Martini, H. Salahieh, A. Goodchild & M. Michael, 2000. A methodological study on participatory barley breeding. I. Selection phase. Euphytica 111: 91–104.

Chapman, S.C., M. Cooper, D. Butler & R.G. Henzell, 2000a. Genotype by environment interactions affecting grain sorghum. I. Characteristics that confound interpretation of hybrid yield. Austr J Agric Res 51: 197–207.

Chapman, S.C., M. Cooper, G.L. Hammer & D. Butler, 2000b. Genotype by environment interactions affecting grain sorghum. II. Frequencies of different seasonal patterns of drought stress are related to location effects on hybrid yields. Austr J Agric Res 51: 209–221.

Chapman, S.C., G.L. Hammer, D.G. Butler & M. Cooper, 2000c. Genotype by environment interactions affecting grain sorghum. III. Temporal sequences and spatial patterns in the target population of environments. Austr J Agric Res 51: 223–233.

Cooper, M., R.E. Stucker, I.H. DeLacy & B.D. Harch, 1997. Wheat breeding nurseries, target environments, and indirect selection for grain yield. Crop Sci 37: 1168–1176.

Cooper, M., & I.H. DeLacy, 1994. Relationships among analytical methods used to study genotypic variation and genotype-by-environment interaction in plant breeding multi-environment experiments. Theor Appl Genet 88: 561–572.

Cullis, B.S. & A.C. Gleeson, 1991. Spatial analysis of field experiments – An extension to two dimensions. Biometrics 47: 1449–1460.

Cullis, B.S., S.J. Welham, R. Thompson & A.R. Gilmour, 1998. New Developments in REML and its Implementation in Genstat 5 Release 4.1. Orange Agricultural lnstitute, Orange, NSW 2800, Australia.

DeLacy, I.H., K.E. Basford, M. Cooper, J.K. Buil & C.G. McLaren, 1996. Analysis of multi-environment trials – An historical perspective. In: M. Cooper & G.L. Hammer (Eds.), Plant Adaptation and Crop lmprovement, pp. 39–124. CAB International, Wallingford, England.

Denis, J.B., H.P. Piepho & F.A. van Eeuwijk, 1997. Modelling expectation and variance for genotype by environment data. Heredity 79: 162–171.

Eyzaguirre, P. & M. Iwanaga, 1996. Participatory Plant Breeding. Proc Workshop on Participatory Plant Breeding, 26–29 July 1995, Wageningen, Netherlands. IPGRI, Rome, Italy.

Gabriel, K.R., 1971. The biplot graphic display of matrices with applications to principal components analysis. Biometrika 58: 453–467.

Gower, J.C. & D.J. Hand, 1996. Biplots. Chapman and Hall, London.

Falconer, D.S., 1981. Introduction to quantitative genetics, 2nd edn. Longman, London.

Gilmour, A.R., R. Thompson & B.R. Cullis, 1995. Average information REML: An efficient algorithm for variance parameter estimation in linear mixed models. Biometrics 51: 1440–1450.

Gogel, B.J., B.R. Cullis & A.P. Verbyla, 1995. REML estimation of multiplicative effects in multi-environment variety trials. Biometrics 51: 744–449.

Joshi, A. & J.R. Witcombe, 1996. Farmer participatory crop improvement. II. Participatory varietal selection, a case study in India. Experimental-Agriculture 32(4): 461–477.

Kempton, RA., 1984. The use of biplots in interpreting variety by environment interactions. J Agric Sci Cambridge 103: 123–135.

Longford, N.T., 1993. Random Coefficient Models. Clarendon Press, Oxford.

McCullagh, P. & J.A. Nelder, 1989. Generalized linear models, 2nd edn. Chapman and Hall, London.

Neter, J., M.H. Kutner, C.J. Nachtsheim & W. Wasserman, 1996. Applied linear statistical models, 4th edn. Irwin, Chicago.

Oman, S.D., 1991. Multiplicative effects in mixed model analysis of variance. Biometrika 78: 729–739.

Piepho, H.-P., 1997. Analyzing genotype-environment data by mixed models with multiplicative terms. Biometrics 53: 761–766.

Piepho, H.-P., J.-B. Denis & F.A. van Eeuwijk, 1998. Mixed biadditive models. In: Proc 19th Int Biometric Conf, Capetown, pp. 79–89.

Piepho, H.-P. & F.A. van Eeuwijk, 2001. Stability analysis in crop performance evaluation. In: M.S. Kang (Ed.), Crop Improvement, Challenges in the 21st Century, Chpt II. Haworth Press, New York.

SAS Institute, 1997. SAS/STAT Software: Changes and Enhancements through Release 6.12. SAS Institute, Cary, NC.

Smith, A.B., 1999. Multiplicative Mixed Models for the Analysis of Multi-Environment Trial Data. Ph.D. Thesis Dpt of Statistics, The University of Adelaide, Adelaide, South Australia, 5005, Australia.

Stram, D.O. & J.W. Lee, 1994. Variance components testing in the longitudinal mixed effects setting. Biometrics 50: 1171–1177.

Tinker, N.A., D.E. Mather, B.G. Rossnagel, K.J. Kasha, A. Kleinhofs, P.M. Hayes, D.E. Falk, T. Ferguson, L.P. Shugar, W.G. Legge, R.B. Irvine, T.M. Choo, K.G. Briggs, S.E. Ullrich, J.D. Franckowiak, T.K. Blake, R.J. Graf, S.M. Dofing, M.A. Saghai Maroof, G.J. Scoles, D. Hoffman, L.S. Dahleen, A. Kilian, F. Chen, R.M. Biyashev, D.A. Kudrna & B.J. Steffenson, 1996. Regions of the genome that affect agronomic performance in two-row barley. Crop Sci 36: 1053–1062.

van Eeuwijk, F.A., 1995. Linear and bilinear models for the analysis of multi-environment trials: I. An inventory of models. Euphytica 84: 1–7.