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R. L. Wu · D. M. O'Malley

Nonlinear genotypic response to macro- and microenvironments

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Abstract Environmental variation can be due to macro- and microenvironments. Whereas macroenvironments, such as climate, density, and nutritional levels, are distinguishable, microenvironments including external random errors or internal "accidents" of an organism cannot be well specified. A quantitative genetic model is proposed to estimate the genetic control of response of genotypes to these two kinds of environments. The model extends Gimelfarb's additive-multiplicative model for genotype × environment interaction by considering both macro- and microenvironmental variation and the mechanistic basis of genotypic response to a particular macroenvironmental factor. It is further extended to estimate genetic correlations between quantitative traits under the additive-multiplicative model. An example from a forest tree was used to illustrate the application and power of the new model. In many aspects, the model displays remarkable advantages over the traditional analysis of variance used to study genotype × environment interaction.

Key words Additive-multiplicative model · ANOVA · Macroenvironment · Microenvironment · Genotype × environment interaction

Introduction

Genotype × environment (G × E) interactions have long been the object of much research by students in plant breeding and evolutionary biology (Comstock and Moll 1963; Finlay and Wilkinson 1963; Via and Lande 1985; Jinks and Pooni 1988; Gillespie and Turelli 1989; Wu and Stettler 1997). Traditional analysis of G × E is to use the two-way analysis of variance (ANOVA) with which the effect due to interactions between genotypes and environments can be estimated. By making use of F-statistic calculated on the structure of the mean squares, the significance of G × E interaction is further tested. If this effect is statistically significant, this means that different genotypes respond differently, or to different extent, to a change in the environment (Caligari and Mather 1975) or that a specific difference in the environment has a greater effect on some genotypes than on others (Falconer and Mackay 1996). The imprecision of these two interpretations on G × E interaction can be corrected by partitioning G × E interaction variance into two components, one due to heterogeneity of genetic variance and the other to the lack of genetic correlation among environments (Cockerham 1963; Wu and Stettler 1997). It is important to distinguish between these two components, because the former does not directly affect selection decisions while the latter does.

Recently, the traditional ANOVA method has been questioned for its application to analyzing G × E interactions in the case where environment cannot be specified (Gimelfarb 1994). There are two categories of environmental variation: macro and micro (Allard and Bradshaw 1964; Jinks and Pooni 1988; Wu 1997). Macroenvironments, e.g., temperature, density, and nutritional level, can be specified and, therefore, the ANOVA method provides a general means for dissecting genotype × macroenvironment interactions (Perkins and Jinks 1973). On the other hand, this method is less powerful to deal with interactions between genotypes and microenvironments, since it is impossible to specify fluctuating microenvironments, such as external stochastic errors or internal "accidents" of an organism (Gavrilets and Hastings 1994). In the existing studies, genotype × microenvironment

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R. L. Wu (✉) · D. M. O'Malley
Forest Biotechnology Group, Department of Forestry, Box 8008, North Carolina State University, Raleigh, NC 27695-8008, USA
Fax: (919)-515-7801
E-mail: rwu@unity.ncsu.edu

interactions have been largely neglected. However, given its major contribution to the total phenotypic variance, this kind of interactions may be an important force in the evolution of quantitative traits (Gavrilets and Hastings 1994).

The ANOVA method cannot consider the biological mechanisms of character development in varying environments, as early observed (Lewontin 1957). In general, response of genotypes to macroenvironments follows some biological rule, which can be approximated by mathematical functions. Consider, as an example, the response of the photosynthetic rate of plants to irradiation. At low levels of irradiation, photosynthetic rate respond proportionately to light intensity, whereas photosynthetic rate can be reduced if plants are over-radiated to the point of light saturation. This relationship between photosynthetic rate and irradiation has been described using a non-rectangular hyperbolic function (Marshall and Biscoe 1980). From the viewpoint of evolutionary biology, the relationship like this is virtually the function for the shape of reaction norms that describe the phenotypic expression of given genotypes across some set of environmental states (Gavrilets and Scheiner 1993; Van Tienderen and Koelewijn 1994; Via et al. 1995).

In this paper, we propose an additive-multiplicative model to handle G × E interactions in order to overcome the two major limitations of ANOVA described above. In agricultural studies, additive-multiplicative models have been used to examine G × E interactions (Zobel et al. 1988; Gauch and Zobel 1988, 1990; Gauch 1990; van Eeuwijk and van Eeuwijk 1995; van Eeuwijk et al. 1995). However, the agricultural models generally do not include microenvironmental interaction, nor do they use mathematical functions to model the macroenvironmental gradient (e.g., Finlay and Wilkinson 1963). We further extend the additive-multiplicative model to estimate genetic parameters, such as broad-sense heritability and genetic correlation between quantitative traits. A numerical example in a forest tree is used to illustrate the application and power of the new model.

The nonlinear model

Within an environment, the relationship between the phenotype of an individual (P) and its genotype is described by a linear equation

$$P = G + E_\varepsilon + I_\varepsilon \tag{1}$$

where G and E_ε are the main effects of genotype and microenvironment (or random error), respectively; and I_ε is their interaction effect. When the genotypes are reared in multiple macroenvironments, the linear relationship of Eq. 1 can be generalized as

$$P = G + E_e + I_e + E_\varepsilon + I_\varepsilon \tag{2}$$

where E_e and I_e are the main effect of macroenvironment and its interaction effect with genotype, respectively. Under the assumption that the genotype is independent of the environment, the phenotypic variance can be partitioned into its genetic, macroenvironmental, genotype × macroenvironment interaction, microenvironmental, and genotype × microenvironmental interaction variance components

$$V_P = V_G + V_{E_e} + V_{I_e} + V_{E_\varepsilon} + V_{I_\varepsilon} \tag{3}$$

However, using the traditional two-way ANOVA model, the last two terms are mix estimated, i.e., the variance component of genotype × microenvironment (V_{I_e}) cannot be separated from the microenvironmental variance, because microenvironments involved cannot be specified. We shall solve this problem by extending the additive-multiplicative model of G × E interaction, as suggested by Gimelfarb (1994).

Because no genotype can display its value independently of the environment in which it is grown, the phenotype explained by the genotypic effect virtually represents its contribution to the phenotype in this specific environment (Falconer and Mackay 1996). Similarly, the effect of the environment on phenotype could be different among individual genotypes, which means that the environmental effect should be interpreted as the contribution of the environment to the phenotype of a particular genotype. When this nature of gene and environmental action is taken into consideration, the phenotype of an individual may be broken down more appropriately into the contributions rather than the strict effect by genotype and environment. In a mathematical term, this can be described as

$$P = g + e + \xi ge + \varepsilon + \zeta g\varepsilon \tag{4}$$

where g, e, and ε are the contributions to the phenotype by the genotype, and macro- and microenvironment, respectively; and parameters ξ and ζ, which can each take any value from $-\infty$ to $+\infty$, describe the interactive relationship between genotype and its macro- and microenvironment, respectively. There is no interaction if ξ or $\zeta = 0$, whereas larger values of ξ or ζ indicate a more multiplicative interaction. Thus, the contribution by genotype environment interactions is represented as a multiplicative term of these two variables. Clearly, an advantage of Eq. 4 is that it incorporates interactions between genotype and microenvironment, which is not possible with the ANOVA model. However, Eq. 4 is not still adequate to reflect the biological mechanism of trait development over distinct macroenvironments. Assuming that the environment-dependent development of a trait can be approximated by a mathematical function, Eq. 4 is changed as:

$$P = g + f(e) + \xi ge + \varepsilon + \zeta g\varepsilon \tag{5}$$

where $f(e)$ represents a general function of some macroenvironmental gradient which is supposed to reveal the biological relationship between the phenotypes and environments. To set an example, we assume that the function is a second degree of polynomial in an environmental factor, i.e., $f(e) = bf + cf^2$ where b and c are the linear and quadratic regression coefficients of response function of genotypes to environmental gradients, respectively.

Assume that the genotypic, and macro- and microenvironmental contributions are distributed independently of each other with the mean and variance of g denoted as m_g and v_g, with the mean and variance of e denoted as m_e and v_e, and with the mean and variance of ε denoted as m_ε and v_ε. The phenotypic mean (m_P) and the phenotypic variance (V_P) of the trait can be derived from Eq. 5 (Stuart and Ord 1987):

$$m_P = m_g + bm_f + cm_{f^2} + \xi m_g m_e + m_\varepsilon + \zeta m_g m_\varepsilon \tag{6a}$$

$$V_P = v_g[1 + (\xi m_e)^2 + (\zeta m_\varepsilon)^2] + v_e(\xi m_g)^2 + v_\varepsilon[1 + (\zeta m_g)^2] + \xi^2 v_g v_e + \zeta^2 v_g v_\varepsilon + b^2 V_f + c^2 V_{f^2} \tag{6b}$$

where m_f and V_f are the mean and variance of the environmental factor across macroenvironments, respectively; and m_{f^2} and V_{f^2} are the mean and variance of the square of the environmental factor

across macroenvironments, respectively. The phenotypic covariance between a pair of traits (x, y) is expressed as

$$W_{P_{xy}} = w_{g_{xy}}(1 + \xi_x\xi_y m_{e_x} m_{e_y} + \zeta_x\zeta_y m_{\varepsilon_x} m_{\varepsilon_y}) + w_{e_{xy}}(\xi_x\xi_y m_{g_x} m_{g_y}) + w_{\varepsilon_{xy}}(1 + \xi_x\xi_y m_{g_x} m_{g_y}) + \xi_x\xi_y w_{g_{xy}} w_{e_{xy}} + \zeta_x\zeta_y w_{g_{xy}} w_{\varepsilon_{xy}} + b_x b_y V_f + c_x c_y V_{f^2} \quad (6c)$$

where $w_{g_{xy}}$, $w_{e_{xy}}$ and $w_{\varepsilon_{xy}}$ are the covariances of the genotypic, and macro- and microenvironmental contributions between traits x and y, respectively. The means of genotypic, and macro- and microenvironmental contributions, and other parameters, b, c, ξ, and ζ, for the two traits are expressed by the corresponding subscripts.

As microenvironmental contributions are suggested to result from internal or external errors of an organism, it is assumed that they follow a normal distribution with the mean set to zero without loss of generality (by linear rescaling of the phenotypes). Thus, Eq. 6a can be rewritten as:

$$m_P = m_g + bm_f + cm_{f^2} + \xi m_g m_e \quad (7)$$

It can be seen that the trait mean in a population is a function of the mean of genotypic and macroenvironmental contributions.

Given a group of genotypically identical individuals, the phenotypic variance of a character among them can be regarded as the sum of macro- and microenvironmental variance of their genotype. If environment is strictly additive, this sum is determined only by the variance of environmental contributions and, hence, it is the same for any genotype. This is not true, however, if environment is not additive. Consider a genotype i exposed to multiple macroenvironments in each of which the genotype is reared with replicates. Because of the same genotype, the variance of genotypic contribution for genotype i is zero, i.e., $v_g = 0$. Thus, the sum of macro- and microenvironmental variances, i.e., phenotypic variance, of this genotype is obtained as

$$V_{P|i} = v_e(\xi m_{g|i})^2 + v_\varepsilon[1 + (\zeta m_{g|i})^2] + b^2 V_f + c^2 V_{f^2} \quad (8)$$

where the mean of genotypic contribution for genotype i is expressed, from Eq. 7, as:

$$m_{g|i} = \frac{m_{P|i} - bm_f - cm_{f^2}}{1 + \xi m_e} \quad (9)$$

where $m_{P|i}$ is the mean phenotypic value of genotype i. Combining Eqs. 8 and 9, we obtain a nonlinear function with $V_{P|i}$ as the dependent variable and $m_{P|i}$, m_f, m_{f^2}, V_f, and V_{f^2} as independent variables. All these dependent and independent variables can be calculated from a real data set. Our focus now is on how to obtain the solutions of seven unknown parameters, b, c, m_e, ξ, ζ, v_e, and v_ε, included in equations. It is found that these parameters can be estimated by using the least squares fit of nonlinear regression model in Eqs. 8 and 9 to observations, $V_{P|i}$ on $m_{P|i}$, m_f, m_{f^2}, V_f, and V_{f^2}. The SAS program for doing so is given as follows

```
PROC NLIN;
MODEL combining Eqs. 8 and 9;

DER. b = -2m_f(m_P|i - bm_f - cm_f^2)·(ξ^2 v_e + ζ^2 v_ε)/(1 + ξm_e)^2 + 2bV_f;

DER. c = -2m_f^2(m_P|i - bm_f - cm_f^2)·(ξ^2 v_e + ζ_ε^2)/(1 + ξm_e)^2 + 2cV_f^2;

DER. m_e = 2ξ(m_P|i - bm_f - cm_f^2)^2·(ξ^2 v_e + ζ^2 v_ε)/(1 + ξm_e)^3;

DER. ξ = 2(m_P|i - bm_f - cm_f^2)^2·((1 + 2ξm_e)ξv_e + ζ^2 m_e v_ε)/(1 + ξm_e)^3;

DER. ζ = 2(m_P|i - bm_f - cm_f^2)^2·ζv_ε/(1 + ξm_e)^2;

DER. v_e = (m_P|i - bm_f - cm_f^2)^2·(ξ/(1 + ξm_e))^2;

DER. v_ε = 1 + (m_P|i - bm_f - cm_f^2)^2·(ζ/(1 + ξm_e))^2;
```

where the expression in the DER statement is not written in a form valid for a SAS expression but should be so in a real application. The sampling variances for the estimators of these parameters will also be given in the OUTPUT of PROC NLIN. The variance of genotypic contributions can be estimated by substituting the estimated parameters into Eq. 6b:

$$v_g = \frac{V_P - v_e(\xi m_g)^2 - v_\varepsilon[1 + (\zeta m_g)^2] - b^2 V_f - c^2 V_{f^2}}{1 + (\xi m_e)^2 + (\zeta m_\varepsilon)^2 + \xi^2 v_e + \zeta^2 v_\varepsilon}$$

The sampling variance of the variance of genotypic contributions is estimated from the Taylor expansion on a ratio estimate and retaining the first-order terms (Stuart and Ord 1987).

If the phenotypic covariance between a pair of traits, x and y, is calculated for genotype , the procedure can be extended to estimate the covariances of macro- and microenvironmental contributions between the two traits. Given $w_{g_{xy}} = 0$, the phenotypic covariance of genotype can be expressed, based on Eq. 6c, as:

$$W_{P_{xy|i}} = w_{e_{xy}}(\hat{\xi}_x\hat{\xi}_y\hat{m}_{g_x|i}\hat{m}_{g_y|i}) + w_{\varepsilon_{xy}}(\hat{\zeta}_x\hat{\zeta}_y\hat{m}_{g_x|i}\hat{m}_{g_y|i}) + \hat{b}_x\hat{b}_y V_f + \hat{c}_x\hat{c}_y V_{f^2} \quad (10)$$

where $\hat{}$ denoted the least squares estimator of parameters b, c, $m_{g|i}$, ξ, and ζ. Unknown parameters, $w_{e_{xy}}$ and $w_{\varepsilon_{xy}}$, are estimated by regressing $W_{P_{xy}|i}$ and on $m_{g_x|i}$ and $m_{g_y|i}$ in the nonlinear form of Eq. 8. The SAS program to calculate $w_{e_{xy}}$ and $w_{\varepsilon_{xy}}$ is given:

```
PROC NLIN;
MODEL Eq. 10;

DER. w_e_xy = ξ̂_x ξ̂_y m̂_g_x|i m̂_g_y|i;

DER. w_ε_xy = 1 + ξ̂_x ξ̂_y m̂_g_x|i m̂_g_y|i.
```

The sampling variance of $w_{e_{xy}}$ and $w_{\varepsilon_{xy}}$ can be read from the OUTPUT. After these two covariances are estimated, the covariance of genotypic contributions between trait x and y is estimated using Eq. 6c:

$$w_{g_{xy}} = \frac{W_{P_{xy}} - w_{e_{xy}}(\xi_x\xi_y m_{g_x} m_{g_y}) - w_{\varepsilon_{xy}}(1 + \zeta_x\zeta_y m_{g_x} m_{g_y}) - b_x b_y V_f + c_x c_y V_{f^2}}{(1 + \xi_x\xi_y m_{e_x} m_{e_y}) + \zeta_x\zeta_y + m_{\varepsilon_x} m_{\varepsilon_y}) + \xi_x\xi_y w_{e_{xy}} + \zeta_x\zeta_y w_{\varepsilon_{xy}}}$$

The sampling variance of the covariance of genotypic contributions between traits x and y is estimated from the Taylor expansion on a ratio estimate and retaining the first-order (Stuart and Ord 1987).

The partitioning of phenotypic variance and covariance

In the statistical decomposition model of Eq. 2, the main genotypic effect, G, is determined strictly by the genotype, whereas the main macro- and microenvironmental effects, E_e and E_ε, are determined strictly by macro- and microenvironment, respectively. The sum of G, E_e and E_ε gives the least squares fit to the phenotypes in the population. Hence, in order to obtain the main effects for a character with phenotypic values determined by Eq. 5, it is necessary to find functions $G(g)$, $E(e)$, and $E'(\varepsilon)$ such that they deliver the minimum of the integral

$$\int_g\int_e\int_\varepsilon [g + e + \xi ge + \varepsilon + \zeta g\varepsilon - G(g) - E(e) - E'(\varepsilon)]^2 \, p(g)q(e)q'(\varepsilon)dg\,de\,d\varepsilon \quad (11)$$

under the constraints

$$\int_g G(g)p(g)\,dg = m_g,$$

$$\int_e E(g)q(g)\,de = m_e, \tag{12}$$

$$\int_\varepsilon E'(\varepsilon)q'(\varepsilon)\,d\varepsilon = m_\varepsilon,$$

where $p(g)$, $q(e)$, and $q'(\varepsilon)$ are the distributions of the genotypic, and macro- and microenvironmental contributions, respectively. Let us consider the functions $G(g)$, $E(e)$, and $E'(\varepsilon)$ in a linear form:

$$G(g) = \alpha g + \beta,$$

$$E(e) = \gamma e + \delta, \tag{13}$$

$$E'(\varepsilon) = u\varepsilon + v.$$

It follows from Eq. 12 that

$$\beta = m_g - \alpha m_g,$$

$$\delta = m_e - \gamma m_e, \tag{14}$$

$$v = m_\varepsilon - u m_\varepsilon.$$

Substituting Eqs. 13 and 14 into Eq. 11 and differentiating 11 with respect to α, γ, and u, we obtain the following equations:

$$\int_g\int_e\int_\varepsilon (g - m_g)[(1-\alpha)(g-m_g) + (1-\gamma)(e-m_e) + (1-u)(\varepsilon - m_\varepsilon) + \xi ge + \zeta g\varepsilon]p(g)q(e)q'(\varepsilon)\,dg\,de\,d\varepsilon = 0,$$

$$\int_g\int_e\int_\varepsilon (e - m_e)[(1-\alpha)(g-m_g) + (1-\gamma)(e-m_e) + (1-u)(\varepsilon - m_\varepsilon) + \xi ge + \zeta g\varepsilon]p(g)q(e)q'(\varepsilon)\,dg\,de\,d\varepsilon = 0, \tag{15}$$

$$\int_g\int_e\int_\varepsilon (\varepsilon - m_\varepsilon)[(1-\alpha)(g-m_g) + (1-\gamma)(e-m_e) + (1-u)(\varepsilon - m_\varepsilon) + \xi ge + \zeta g\varepsilon]p(g)q(e)q'(\varepsilon)\,dg\,de\,d\varepsilon = 0.$$

After integration, we have:

$$v_g(1 - \alpha + \xi m_e + \zeta m_\varepsilon) = 0,$$

$$v_e(1 - \gamma + \xi m_g + \zeta m_\varepsilon) = 0, \tag{16}$$

$$v_\varepsilon(1 - u + \xi m_e + \zeta m_g) = 0,$$

which yield:

$$\alpha = 1 + \xi m_e + \zeta m_\varepsilon,$$

$$\gamma = 1 + \xi m_g + \zeta m_\varepsilon, \tag{17}$$

$$u = 1 + \xi m_e + \zeta m_g.$$

Consequently, the main genotypic, and macro- and microenvironmental effects are:

$$G = g(1 + \xi m_e + \zeta m_\varepsilon) - \xi\, m_g m_e - \zeta m_g m_\varepsilon,$$

$$E_e = e(1 + \xi m_g + \zeta m_\varepsilon) - \xi\, m_g m_e - \zeta m_e m_\varepsilon, \tag{18}$$

$$E_\varepsilon = \varepsilon(1 + \xi m_e + \zeta m_g) - \xi\, m_e m_\varepsilon - \zeta m_g m_\varepsilon.$$

The interaction effect between genotype and environment is then calculated by substituting the sum of G, E_e, and E_ε from the right side of Eq. 4:

$$I = \xi[m_g m_e + (g - m_g)(e - m_e)] - \xi m_e(\varepsilon - m_\varepsilon) + \zeta[m_g m_\varepsilon + (g - m_g)(\varepsilon - m_\varepsilon)] - \zeta m_\varepsilon(e - m_e) \tag{19}$$

In fact, Eq. 19 represents the sum of genotype × macroenvironment and genotype × microenvironment interactions. However, according to the definition of ξ and ζ, the first two terms with ξ can be regarded as the genotype × macroenvironment interaction (I_e) and the last two terms with ζ as the genotype × microenvironment interaction (I_ε). Since the mean of microenvironmental contribution is set to zero, Eqs. 18 and 19 are rewritten as:

$$G = g(1 + \xi m_e) - \xi m_g m_e,$$

$$E_e = e(1 + \xi m_g) - \xi m_g m_e,$$

$$E_\varepsilon = \varepsilon(1 + \xi m_e + \zeta m_g), \tag{20}$$

$$I_e = \xi[m_g m_e + (g - m_g)(e - m_e)] - \xi m_e\varepsilon,$$

$$I_\varepsilon = \zeta(g - m_g)\varepsilon.$$

The variance components of various factors in Eq. 2 can be obtained following the above equation:

$$V_G = v_g(1 + \xi m_e)^2,$$

$$V_{E_e} = v_e(1 + \xi m_g)^2,$$

$$V_{E_\varepsilon} = v_\varepsilon(1 + \xi m_e + \zeta m_g)^2, \tag{21}$$

$$V_{I_e} = v_g v_e \xi^2 + v_\varepsilon(\xi m_e)^2,$$

$$V_{I_\varepsilon} = v_g v_e \zeta^2.$$

If all parameters at the right side of Eq. 21 are distributed independently of each other, the sampling variances for these variance components are derived as

$$V(V_G) = V(v_g)[(1 + \xi m_e)^2 + V(\xi)V(m_e)]^2 + V(\xi)V(m_e)[(v_g)^2 + V(v_g)][2(1 + \xi m_e)^2 + V(\xi)V(m_e)]$$

$$V(V_{E_e}) = V(v_e)[(1 + \xi m_g)^2 + V(\xi)V(m_g)]^2 + V(\xi)V(m_g)[(v_e)^2 + V(v_e)][2(1 + \xi m_g)^2 + V(\xi)V(m_g)]$$

$$V(V_{E_\varepsilon}) = V(v_\varepsilon)[(1 + \xi m_e + \zeta m_g)^2 + V(\xi)V(m_g) + V(\zeta)V(m_g)]^2 + [(v_\varepsilon)^2 + V(v_\varepsilon)][V(\xi)V(m_e) + V(\zeta)V(m_g)] [2(1 + \xi m_e + \zeta m_g)^2 + V(\xi)V(m_e) + V(\zeta)V(m_g)]$$

$$V(V_{I_e}) = (v_g v_e)^2 V(\xi)[2\xi + V(\xi)] + V(v_g)V(v_e)[\xi + V(\xi)]^2 + [(\xi m_e)^2 + V(\xi)V(m_e)]^2 + V(\xi)V(m_e)[(v_\varepsilon)^2 + V(v_\varepsilon)][2\xi m_e + V(\xi)V(m_e)]$$

$$V(V_{I_\varepsilon}) = (v_g v_e)^2 V(\zeta)[2\zeta + V(\zeta)] + V(v_g)V(v_e)[\zeta + V(\zeta)]^2$$

where $V(\cdot)$ denotes the sampling variances of parameters. The broad-sense heritability of a quantitative trait is expressed as:

$$H^2 = \frac{V_G}{V_G + V_{E_e} + V_{E_\varepsilon} + V_{I_e} + V_{I_\varepsilon}}, \tag{22}$$

which indicates that in the presence of G × E interaction (ξ and $\zeta \neq 0$), the broad-sense heritability of a trait is determined not only by the variances of the genotypic, and macro- and microenvironmental contributions, but also by the means of the genotypic and macroenvironmental contributions.

Similarly, the phenotypic covariance between traits x and y can also be broken down into the corresponding covariance components:

$$W_{G_{xy}} = w_{g_{xy}}(1 + \xi_x m_{e_x})(1 + \xi_y m_{e_y}),$$
$$W_{Ee_{xy}} = w_{e_{xy}}(1 + \xi_x m_{g_x})(1 + \xi_y m_{g_y}),$$
$$W_{E\varepsilon_{xy}} = w_{\varepsilon_{xy}}(1 + \xi_x m_{e_x} + \zeta_x m_{g_x})(1 + \xi_y m_{e_y} + \zeta_y m_{g_y}), \quad (23)$$
$$W_{Ie_{xy}} = w_{g_{xy}} w_{e_{xy}} \xi_x \xi_y + w_{\varepsilon_{xy}} \xi_x \xi_y m_{e_x} m_{e_y},$$
$$W_{I\varepsilon_{xy}} = w_{g_{xy}} w_{e_{xy}} \zeta_x \zeta_y.$$

Assuming the independence among ξ_x, ξ_y, m_{e_x} and m_{e_y}, the sampling variance of the genetic covariances between traits x and y can be derived as:

$$V(W_{g_{xy}}) = (1 + \xi_x m_{e_x})^2 (1 + \xi_y m_{e_y})^2 V(w_{g_{xy}}) + V(\xi_x) V(m_{e_x}) V(\xi_y) V(m_{e_y}) [(w_{g_{xy}})^2 + V(w_{g_{xy}})]$$

The genetic correlation between the two traits is expressed as

$$r_{g_{xy}} = \frac{W_{G_{xy}}}{\sqrt{V_{G_x}}\sqrt{V_{G_y}}} \quad (24)$$

It can be seen that genetic correlation is only a function of the mean and variance of genotypic contribution for the corresponding traits even when G × E multiplicative interactions exist. The sampling variances of broad-sense heritability and genetic correlation are approximated by the Taylor expansion on a ratio estimate and retaining the first-order terms (Stuart and Ord 1987).

Experimental data

We provide a numerical example of G × E interactions in a F_2 family of 375 genotypes produced by intercrossing two poplar species, *Populus trichocarpa* and *P. deltoides* (Wu and Stettler 1997). In spring 1993, the F_2 hybrids were planted using rooted cuttings in two macroenvironments, one east of the Cascades in Boardman, Oregon, the other west of the Cascades in the lower Columbia River Valley near Clatskanie, Oregon. Both plantations were laid out in a randomized complete block design with three (Clatskanie) or four (Boardman) clonal replicates and two-tree plots at a spacing of 1.5 × 3.0 m and surrounded by two border rows. At Boardman, the fourth replicate received a different watering regime from the other three from year 2, and so it was excluded from all analysis of this example.

Two plantation environments differ markedly. Boardman (117°6′W, 45°42′N) has a continental climate, and Clatskanie (123°40′W, 46°6N′) is in the coastal zone with a strong maritime influence. During the growing season (April–October) of 1994, the average monthly temperature was 18.5°C at Boardman, 14.3°C at Clatskanie. The average solar irradiance in that year at Boardman was 430 $W\,m^{-2}\,day^{-1}$, compared to 280 $W\,m^{-2}\,day^{-1}$ at Clatskanie. Other environmental factors that differ between the two sites include annual precipitation, soil water potential, soil fertility, and wind movement. However, in this example, we will focus on the influence by temperature and irradiance.

We calculated the trait means and phenotypic variance of second-year (1994) stem volume index based on all clonal replicates in the two macroenvironments for each genotype. The phenotypic variance for a genotype is composed of two components: macroenvironmental variance and microenvironmental variance within each macroenvironment. Using the nonlinear function, described by Eqs. 8 and 9, to regress the phenotypic variance of a single genotype on its phenotypic mean and temperature or irradiance, we finally obtained the genotypic, macroenvironmental, genotype × macroenvironment interaction, microenvionmental, and genotype × microenvironment interaction variances for volume index when these two environmental parameters were respectively incorporated into the model of G × E (Table 1). In this example, we estimated the macroenvironmental variance, although the two macroenvironments used were not randomly sampled. It was found that while the genotypic, microenvironmental, and their interaction variances were similar between the two incorporating models, the macroenvironmental and genotype × macroenvironment interaction variances showed larger values in the temperature- than irradiance-incorporated model. This makes sense when the ξ values, characterizing the strength of genotype × macroenvironment interactions, are compared. In the temperature-incorporated model, the ξ value was much larger than that in the irradiance model. However, the ζ values, which characterize the strength of genotype × microenvironment interactions, were quite close between the two models. For these

Table 1 The variance components (± SE) due to the genotypic (V_G), macroenvironmental V_{E_e}, genotype × macroenvironment V_{I_e}, microenvironment V_{E_ε}, and genotype × microenvironment interaction effects V_{E_ε} in second-year stem volume index of a F_2 family of *P. trichocarpa* and *P. deltoides*. All these values were, respectively, estimated from the additive-multiplicative model incorporating the temperature and irrandiance differences between two plantations east and west of the Cascade Ranges, with a comparison to the traditional ANOVA method

Method	The temperature model	The irradiance model
Additive-multiplicative		
V_G	20.97 ± 2.51	21.53 ± 2.59
V_{E_e}	19.25 ± 1.74	11.35 ± 1.07
V_{I_e}	9.33 ± 0.82	4.70 ± 0.33
V_{E_ε}	4.67 ± 0.57	3.51 ± 0.31
V_{I_ε}	13.42 ± 1.40	12.74 ± 1.15
ξ	12.11 ± 1.09	7.25 ± 0.62
ζ	3.43 ± 0.25	2.88 ± 0.20
H^2	0.31 ± 0.11	0.40 ± 0.12
ANOVA		
V_G	16.37 ± 2.12	
V_{E_e}	23.30 ± 3.57	
V_{I_e}	8.19 ± 0.88	
V_{E_ε}	15.11 ± 1.23	
H^2	0.26 ± 0.10	

reasons, the broad-sense heritability (H^2) was larger for volume index based on the irradiance- than on the temperature-incorporated model. The two models incorporating a specific environmental variable, especially temperature, showed a larger H^2 level than that obtained from the traditional ANOVA method (Wu and Stettler 1996). It was found that from both temperature- and irradiance-incorporated models the variance due to genotype × mircoenvironment interactions was much larger than the pure microenvironment variance in second-year volume growth of poplar hybrids (Table 1). However, the interaction variance of this kind cannot be detected using the traditional ANOVA model.

Discussion

Genotype × environment interactions play an important role in evolution and plant and animal breeding. The analytical method to deal with this phenomenon is limited to the application of traditional analysis of variance. As pointed out by Gimelfarb (1994), this method is not adequate to describe the variation in response to microenvironments that cannot be specified and the mechanisms of gene and environmental action as well. Another limitation of ANOVA applied to the analysis of G × E interaction is that the method can only detect the effect strictly by genotype and environment. In other words, it cannot manipulate the mechanistic relationship between the genotype and its environment.

Gimelfarb (1994) suggested the additive-multiplicative model to divide the phenotype of an individual into the genotypic, environmental, and genotype × environment interaction contributions. Gavrilets (1986) interpreted the three contributions in the model as the environment-dependent effect of genes, the effect of a change in the composition of genes, and the effect of a change in the activity of genes, respectively. We generalize Gimelfarb's model to allow both macro- and microenvironments and their interactions with genotype. Macro- and microenvironments should be differentiated because they influence the phenotype of a character in different ways (Falconer and Mackay 1996). Whereas genotypes always respond to macroenvironments in a predictable manner, the microenvironmental sensitivity is unique for different individuals of the same genotype. Based on this variety of facts and ideas, we use a nonlinear function (i.e., second degree of polynomial) to fit the contributions of the macroenvironment. Other response functions can also be employed but should be based on the empirical relationship of the phenotype and environmental factors. For example, a non-rectangular hyperbolic equation could be more appropriate when one attempts to examine the response of genotypes to irradiance for the photosynthetic rate of plants. Simulations on the influence of other environmental factors, such as temperature, moisture, and nutritional level, on the phenotype have also been reported in the current literature (e.g., Namkoong et al. 1992). As demonstrated from an example in poplar, the new method can provide a better insight into the mechanisms of interactions between genotype and a specific environmental factor.

Our model is further extended to the case of multitrait correlation. Despite its importance, how trait correlation is influenced by G × E interactions receives few concerns. There has been much evidence for the change of trait relationships over environments (Schlichting 1989). The present model will make it possible to study the genetic mechanisms underlying developmental integration.

The prerequisite of the application of the present method is to have genetically identical individuals for each genotype. It is possible to produce such individuals experimentally. In annual plants and animals, these materials include inbred lines or isogenic lines; in some long-lived forest trees, clones from cuttings and rootings.

Most previous papers on G × E interactions were restricted to the context of plant and animal breeding (Finlay and Wilkinson 1963; Eberhart and Russell 1966; Pani and Lasley 1972; Wescott 1986). Disputes have arisen in the past about the potential role of interactions between genotype and environment in the evolution of quantitative traits (Bradshaw 1965; Via and Lande 1985; Scheiner et al. 1991; Gomuliewicz and Kirkpatrick 1992; Via 1993). Now G × E interactions can be measured more accurately, and their contributions to phenotypic variance specified, thereby allowing more general theoretical and empirical approaches to this question.

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References

Allard RW, Bradshaw AD (1964) Implications of genotype-environment interactions in applied plant breeding. Crop Sci 4: 503–508

Bradshaw AD (1965) Evolutionary significance of phenotypic plasticity in plants. Adv Genet 13: 115–155

Caligari PDS, Mather K (1975) Genotype-environment interaction. III. Interactions in *Drosophila melanogaster*. Proc R Soc London Ser B 191: 387–411

Cockerham CC (1963) Estimation of genetic variances. In: Hanson WD, Robinson HF (eds) Statistical genetics and plant breeding. Proc Natl Acad Sci – Natl Res Council USA 982: 53–94

Comstock RE, Moll RH (1963) Genotype-by-environment interactions. In: Hanson WD, Robinson HF (eds) Statistical genetics and plant breeding. Proc Natl Acad Sci – Natl Res Council USA 982: 164–196

Eberhart SA, Russell WA (1966) Stability parameters for comparing varieties. Crop Sci 6 : 36–40
Falconer DS, Mackay TFC (1996) Introduction to quantitative genetics, 4th edn. Longman Group, Harlow, Essex
Finlay KW, Wilkinson GN (1963) The analysis of adaptation in a plant-breeding program. Aust J Agri Res 14 : 742–754
Gauch HG Jr (1990) Full and reduced models for yield trials. Theor Appl Genet 80 : 153–160
Gauch HG Jr, Zobel RW (1988) Predictive and postdictive success of statistical analyses of yield trials. Theor Appl Genet 76 : 1–10
Gauch HG Jr, Zobel RW (1990) Imputing missing yield trial data. Theor Appl Genet 79 : 753–761
Gavrilets S (1986) An approach to modeling the evolution of populations with consideration of genotype-environment interaction (translation from Russian). Soviet Genet 22 : 28–36
Gavrilets S, Hastings A (1994) A quantitative-genetic model for selection on developmental noise. Evolution 48 : 1478–1486
Gavrilets S, Scheiner SM (1993) The genetics of phenotypic plasticity. V. Evolution of reaction norm shape. J Evol Biol 6 : 31–48
Gillespie JH, Turelli M (1989) Genotype-environment interaction and the maintenance of polygenic variation. Genetics 121 : 129–138
Gimelfarb A (1994) Additive-multiplicative approximation of genotype-environment interaction. Genetics 138 : 1339–1349
Gomuliewicz R, Kirkpatrick M (1992) Quantitative genetics and the evolution of reaction norms. Evolution 46 : 390–411
Jinks JL, Pooni HS (1988) The genetic basis of environmental sensitivity. In: Weir BS, Eisen EJ, Goodman MM, Namkoong G (eds) Proc 2nd Int Conf Quant Genet. Sinauer, Sunderland, Mass. pp 505–522
Lewontin RC (1957) The adaptations of populations to varying environments. Cold Spring Harbor Symp Quant Biol 22 : 395–408
Marshall B, Biscoe PV (1980) A model for C_3-leaves describing the dependence of net photosynthesis on irradiance. I. Derivation. J Exp Biol 31 : 29–39
Namkoong G, Jonsson A, Eriksson G (1992) Genetic variation in nutrient response functions. Theor Appl Genet 85 : 165–172
Pani SN, Lasley JF (1972) Genotype × environment interactions in animals. Mo Agric Exp Stn Res Bull 992, Columbia, Mo
Perkins JM, Jinks JL (1973) The assessment and specificity of environmental and genotype-environmental components of variability. Heredity 30 : 111–126
Scheiner SM, Gaplan RL, Lyman R (1991) The genetics of phenotypic plasticity. III. Genetic correlations and fluctuating asymmetries. J Evol Biol 4 : 51–68
Schlichting CD (1989) Phenotypic integration and environmental change. BioScience 39 : 460–464
Stuart A, Ord JK (1987) Kendall's advanced theory of statistics. Vol. 1. Distribution Theory. Oxford University Press, New York
van Eeuwijk FA, van Eeuwijk FA (1995) Linear and bilinear models for the analysis of multi-environmental trials: I. An inventory of models. Euphytica 84 : 1–7
van Eeuwijk FA, van Eeuwijk FA, Gauch HG Jr, Goodman LA, Wedderburn RWM (1995) Multiplicative interaction in generalized linear models. Biometrics 51 : 1017–1032
van Tienderen PH, Koelewijn HP (1994) Selection on reaction norms, genetic correlations and constraints. Genet Res 64 : 115–125
Via S (1993) Adaptive phenotypic plasticity: target or by-product of selection in a variable environment? Am Nat 142 : 352–365
Via S, Gomulklewicz R, De Jong G, Scheiner SM, Schlichting CD, van Tienderen PH (1995) Adaptive phenotypic plasticity: consensus and controversy. Trend Ecol Evol 10 : 212–217
Via S, Lande R (1985) Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution 39 : 505–523
Wescott B (1986) Some methods of analyzing genotype-environment interaction. Heredity 56 : 243–253
Wu RL (1997) Genetic control of macro- and micro-environmental sensitivities in *Populus*. Theor Appl Genet 93 : 104–115
Wu R, Stettler RF (1997) Quantitative genetics of growth and development in *Populus*. II. The partitioning of genotype × environment interactions in stem growth. Heredity 78 : 124–134
Zobel RW, Wright MJ, Gauch HG Jr (1988) Statisrical analyses of a yield trial. Agron J 80 : 388–393