**Differential Susceptibility to the Environment:**

**Are Developmental Models Compatible with the Evidence from Twin Studies?**

**Simulation description**

**1. Environmental states**

The simulation describes *k* pairs of MZ twins and *k* pairs of DZ twins. The shared (S) and nonshared (N) components of the state of the environment at time 1 were simulated as vectors of *k* normally distributed random values with *M* = 0 and *SD* = 1:

(1)

where subscripts *A* and *B* identify the two members of the pair. The vector of environmental states at time 1 () was computed as a weighted sum of and :

(2)

(3)

where corresponds to the proportion of the total environmental variance accounted for by shared effects ().

At time 2, shared and nonshared components were updated based on the environmental correlation :

(4)

(5)

(6)

with . Finally, the vector of environmental states at time 2 () was computed as follows:

(7)

(8)

so that both and had *M* = 0 and *SD* =1.

**2. Plasticity development**

The development of plasticity at time 1 was simulated separately for each of the four models of differential susceptibility considered in the paper (see Figure 1). Genetic effects on plasticity were modeled with two independent vectors of genotypic values, for the direct genetic effect and for the interaction effect. This avoids the restrictive assumption that the same loci that directly modulate plasticity also increase the dependence of plasticity on early environmental factors. However, simulation results were not meaningfully affected by imposing the additional constraint . All genotypic values were normally distributed with *M* = 0 and *SD* = 1, consistent with the assumption that phenotypic traits reflect the additive contribution of many small-effect loci:

(9)

In DZ pairs, genotypic values for twin *B* were generated so as to obtain a genotypic correlation of .50 between twins:

(10)

In the following steps of the simulation, some vectors were rescaled to yield a different mean and standard deviation. For compactness, a rescaling function can be defined as follows:

. (11)

(For example, would correspond to standardizing vector **x**.)

**2.1. Model DST-1**

In model DST-1, plasticity is entirely a function of an individual’s genotype. Plasticity vectors were computed as follows:

(12)

(13)

In all the models, plasticity was rescaled to a distribution with *M* = 1 and *SD* = 0.2, effectively restricting it to positive values. This was done to obtain the specific interaction shape postulated by differential susceptibility models, in which the environmental slope may become larger (higher plasticity) or smaller (lower plasticity), but does not change sign (see the right panel of Figure 1).

**2.2. Model DST-2**

In model DST-2, plasticity is a function of an individual’s genotype, the environment at time 1, and their interaction. Plasticity vectors were computed as follows:

(14)

(15)

where denotes the element-by-element product of vectors **x** and **y**. Note that , and

(16)

so that weights , , and correspond to the proportions of variance explained by the respective components.

**2.3. Model BSC-1**

In model BSC-1, plasticity develops as a quadratic function of the environment at time 1. Plasticity vectors were computed as follows:

(17)

(18)

where **x.2**denotes the element-by-element square of vector **x**.

**2.4. Model BSC-2**

In model BSC-2, plasticity is a function of an individual’s genotype, the square of the environment at time 1, and their interaction. Plasticity vectors were computed as follows:

(19)

(20)

**3. Trait development**

Trait values were computed as a function of an individual’s genotype (), the environment at time 2, and the interaction between plasticity and the environment at time 2, as follows:

(21)

(22)

(23)

(24)

**4. Trait measurement**

Measured trait values were computed by adding measurement error to the true value of the trait. The amount of error was determined by measurement reliability , i.e., the proportion of true score variance on the total variance of the measured trait ().

(25)

(26)

**5. Variance components**

ACE variance components were estimated as follows (see Falconer & MacKay, 1996):

(27)

(28)

(29)

where is the bivariate correlation between **x** and **y**.

**6. References**

Falconer, D. S., & MacKay, T. F. C. (1996). *Introduction to quantitative genetics* *(4th ed.).* Harlow, UK: Longmans Green.