

2. Belsky J, Jonassaint C, Pluess M, Stanton M, Brummett B, Williams R: Vulnerability genes or plasticity genes? *Mol Psychiatry* 2009; 14:746–754
3. Belsky J, Pluess M: Beyond diathesis-stress: differential susceptibility to environmental influences. *Psychol Bull* 2009; 135:885–908
4. Kilpatrick DG, Koenen KC, Ruggiero KJ, Acierno R, Galea S, Resnick HS, Roitzsch J, Boyle J, Gelernter J: The serotonin transporter genotype and social support and moderation of post-traumatic stress disorder and depression in hurricane-exposed adults. *Am J Psychiatry* 2007; 164:1693–1699

MICHAEL PLUSS, PH.D.
London
JAY BELSKY, PH.D.
Davis, Calif.

The authors report no financial relationships with commercial interests.

This letter (doi: 10.1176/appi.ajp.2011.11111614) was accepted for publication in December 2011.

Response to Pluess and Belsky Letter

TO THE EDITOR: We appreciate the opportunity to respond to Pluess and Belsky's interesting letter. We would like to make three main points. First, in contrast to Pluess and Belsky's contention, we do not view G×E inquiry exclusively from a diathesis-stress (as opposed to plasticity) perspective. Rather, in writing a review, the focus is necessarily on published stud-

ies, and the diathesis-stress perspective has been the dominant one in the candidate G×E (cG×E) literature. Second, we believe it unlikely that "one reason cG×E findings often do not replicate is the misconceptualization of candidate genes as risk genes." Such misconceptualizations would affect novel investigations and direct replication attempts in an identical manner, so that could not be a reason for the numerous failures to replicate cG×E findings. Third, Pluess and Belsky argue that including both risk and protective variables can lead to the correct identification of higher-order (e.g., three-way) interactions. We agree that this is theoretically possible. However, given that the central problems that were raised in our review—low power and likely high false discovery rate—are likely to be exacerbated in tests of higher-order interactions, we would urge caution before accepting novel reports of such findings. As argued in our original article, well-powered, direct replication attempts are crucial for understanding the legitimacy of novel candidate polymorphism findings. In a field with a poor record of subsequent empirical support for novel findings, such direct replications should be viewed as at least as scientifically important as the novel findings themselves.

LARAMIE E. DUNCAN, PH.D.
Boston
MATTHEW C. KELLER, PH.D.
Boulder, Colo.

The authors' disclosures accompany the original article.

This reply (doi: 10.1176/appi.ajp.2011.11111614r) was accepted for publication in December 2011.

Corrections

In the article "A Nationwide Cohort Study of Oral and Depot Antipsychotics After First Hospitalization for Schizophrenia," by Jari Tiihonen et al. (*Am J Psychiatry* 2011; 168:603–609), in the first sentence of the second paragraph of the Results section, the mean follow-up period of 2 years (5,221 person-years) reflects the potential time frame for discontinuation of medication. However, the actual mean follow-up time in the analysis of all-cause discontinuation was 0.5 years, and the number of person-years was 809, since follow-up for any given patient stopped after discontinuation of medication. The actual numbers of person-years for each antipsychotic are listed in Figure S1 in the online data supplement.

In the article "Treatment of Suicide Attempters With Bipolar Disorder: A Randomized Clinical Trial Comparing Lithium and Valproate in the Prevention of Suicidal Behavior," by Maria A. Oquendo et al. (*Am J Psychiatry* 2011; 168:1050–1056), in the Intervention subsection of the Method section, the units for the target blood level range for lithium were incorrectly reported. The correct range is 0.6–1.0 mEq/liter.