monotherapy and polytherapy patients, we do not feel that any bias in rating affected our comparison of the two groups.

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Completed Suicide After a Suicide Attempt

TO THE EDITOR: Kirsi Suominen, M.D., Ph.D., et al. (1) followed a cohort of 100 self-poisoned Helsinki patients and found out suicides continued to accumulate 37 years after the index attempts. Although it had a long follow-up period, the analysis they carried out was completely based on a clinical study group. We would like to highlight our findings based on a community sample from the United States.

The Baltimore Epidemiologic Catchment Area Study was a community-based longitudinal study starting in 1981. History of suicide attempts was assessed at baseline. Mortality and causes of death were identified by death certificates after deaths were confirmed by searching the National Death Index. Our study analyzed deaths confirmed up to 1998. By the end of 1998, 861 of the 3,481 baseline participants were deceased. Death certificates of 762 deceased participants (88.5%) were collected, and causes of death were identified, with 729 having died of natural causes. Thirty-three had died of nonnatural causes (4.3% among all known causes of death), including 19 from accidents, six from homicides, and two undetermined. The remaining seven died from suicide (0.9% among all known causes of death). The method was firearms in 57% of the suicide deaths, hanging in 29%, and drug overdoses in 14%. Only two suicides were women (29%). The year of suicide did not cluster in any specific years. History of suicide attempts was not associated with overall mortality after sociodemographic characteristics were adjusted.

In our sample, only 1.7% of the participants with a history of attempts eventually completed suicide in the 17-year period. Compared to people without a history of attempts, former attempters had a much higher odds of completing suicide (odds ratio=11.3, $p<0.001$). In a multiple logistic regression model with adjustment for sociodemographic variables, only history of attempts and gender were associated with later suicide: men were six times more likely to commit suicide than women (odds ratio=5.9, $p<0.001$). History of suicide attempts was not associated with the likelihood of other nonnatural deaths.

Although our analysis had a lower percent of eventual suicide than the study by Suominen et al. (13%), our findings overall showed a comparable, although less significant, pattern between gender, history of attempts, and eventual suicide. Our sample also included a comparison group (nonattempters) that the study by Suominen et al. did not have. Our less significant result might have come from our shorter follow-up period and other intrinsic population differences. The newest wave of the Baltimore Epidemiologic Catchment Area Study began in 2004 and will offer more information on longitudinal association between suicide attempts and suicide.

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Reference

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Nonsignificant But Suggestive Results?

TO THE EDITOR: I would like to comment on a study in the April issue by Stephen V. Faraone, Ph.D., et al. (1). It is surprising to me that a study that did not produce a single positive result could elicit such broad-sweeping conclusions from its authors. Is it really acceptable science to tout findings that are nonsignificant but suggestive? We have seen nearly two decades of genetic linkage analysis studies and, as the author of another article in the same issue of the *Journal* concedes, there has yet to be a "confirmed gene related to bipolar disorder" (2, p. 595). One would think that this fact would make genetic researchers more circumspect.

Yet this mountain of failed studies is now cited as evidence for the genetics of bipolar disorder, including the concept of "susceptibility" genes and now the idea of genes influencing when the onset of the disorder is likely to occur. All of this is nothing more than wild speculation. I suggest that those conducting future genome-wide scans for linkage make a good-faith effort to demonstrate that elevated lod score (logarithm of the odds ratio for linkage) results are not merely additional false positives before presenting their findings.

In fact, the very use of lod scores should be brought into question when performing a genome-wide scan. It should hardly be surprising if a few regions of the several hundred being examined have elevated lod scores simply because of random variance from the large sample of loci "scanned." This point could easily be confirmed, and I challenge the authors to take the same subjects, randomly assign the group without regard to diagnosis, and repeat the genome-wide scan. I venture to guess that this will also produce a few elevated lod scores. Would these random results also be described as nonsignificant but suggestive?

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Neurotensin Receptor Agonists and Antagonists for Schizophrenia

TO THE EDITOR: For nearly three decades, researchers have hypothesized that neurotensin is an endogenous neuroleptic