A Twin Study of Recent Life Events and Difficulties

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Objectives: To examine the role of genetic and familial-environmental factors in the origin of stressful life events.

Design: Self-report questionnaires describing stressful life events in the last year.

Participants: Both members of 2315 twin pairs ascertained from the population-based Virginia Twin Registry.

Results: Life events were modestly but significantly correlated in twin pairs, and correlations in monozygotic (MZ) twins consistently exceeded those in dizygotic (DZ) twins. For total life events, the best-fitting twin model indicated that familial-environmental and genetic factors each accounted for around 20% of the total variance. Individual life events could be best divided into “network events” (directly affecting individuals in the respondent’s social network) where twin resemblance was due solely to the familial environment, and “personal” events (directly affecting the response) where most twin resemblance was the result of genetic factors.

Conclusions: While neither genes nor familial environment is likely to directly produce life events, personal and social factors that predispose to life events are substantially influenced by an individual’s genetic and family background. These results, which suggest that stressful life events reflect more than random influences, may have important implications for our understanding of the relationship between stressful life events and psychopathology.

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LIFE EVENTS have been a central concept in psychiatric research since the introduction more than 20 years ago of the Social Readjustment Rating Scale of Holmes and Rahe. The concept remains a popular one because the possibility of precisely measuring exposure to environmental stress is very appealing. A central assumption, usually implicit, of much of life event research is that their occurrence is largely or entirely random: that having a large number of stressful life events is, in essence, bad luck.4

However, several lines of evidence suggest that a “random” model of life events is incorrect. First, the number of recent life events reported by individuals over distinct time periods is significantly and often substantially correlated; with modest consistency, some individuals experience large numbers while others experience small numbers of life events.5-7 Second, the number of life events can be predicted by stable personal characteristics such as social class,8,10 self-esteem, social support, mood, and personality.9,11-13 Third, research on specific events such as automobile accidents, industrial injuries, and criminal victimization has shown consistent interindividual differences in “event-proneness” that in turn relates to personal attributes such as personality, life-style, psychopathologic condition, and drug and alcohol intake.16-18

Because of the ubiquitous influence of familial factors on human behavioral traits, it is of interest to examine, from a genetic epidemiologic perspective, the causes of stressful life events. To our knowledge, three studies to date have taken this approach.

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SUBJECTS AND METHODS

SAMPLE

Data for this article came from an ongoing study of personality, social attitudes, drinking behavior, and common psychiatric disorders in the Virginia Twin Registry, a population-based registry based on a systematic review of all birth records in the Commonwealth of Virginia from 1915 onward. Current addresses are obtained by a variety of means, including matching with state records. Between 1985 and 1988, questionnaires were mailed to members of white twin pairs from the Virginia Twin Registry (MZ, DZ; male, female, and opposite sex) aged 17 to 55 years, with multiple questionnaires being sent to nonrespondents. The final individual response rate was 68.2%, which overstates the true refusal rate, as an unknown proportion of the nonresponding twins, due for example to incorrect addresses or improperly forwarded mail, never received a questionnaire. Usable responses were obtained from 2315 twin pairs. Zygosity was assigned by an algorithm that included for all twins questions about physical similarity and frequency of confusion as children. These questions, when tested against blood typing, have been found to assign zygosity accurately in more than 95% of cases. In addition, for most of the female sample, this information was reviewed along with photographs of each member of the twin pair and the basis of this zygosity was blindly assigned. Our final algorithm assigned 890 twin pairs as MZ (male, 283; female, 607) and 1425 as DZ (male, 308; female, 537; opposite sex, 580). The female preponderance of this sample resulted from an oversampling of female subjects and greater effort in their recruitment in preparation for a field study that would involve only female same-sex twin pairs. Mean (±SD) ages of the five sex-zygosity groups were significantly different (P<0.001), but the differences were small in magnitude: MZ male subjects, 30.8±5.8 years; MZ female subjects, 28.2±5.7 years; DZ male subjects, 32.3±5.8 years; DZ female subjects, 29.2±5.7 years; and DZ opposite sex subjects, 31.7±5.8 years. Years of education (mean±SD) of the entire sample was 13.7±2.4 years and did not differ significantly across twin types.

LIFE EVENTS

Life events over the past year were measured by 44 self-report items, which were divided into “network” events, which had primary impact on individuals in the respondent’s social network (eg, father in an automobile accident) and “personal” events, which had their primary impact on the respondent (eg, respondent in an automobile accident). Eighteen network events consisting of death, illness/injury, or personal crisis for each of six classes of individuals in the respondent’s social network (spouse, children, parents, siblings, other relatives, and “someone else close to you”) were analyzed as three separate categories: network death, network illness/injury, and network crisis. Network events referable to the co-twin were excluded from our analyses. Based on item content and factor analyses, the remaining 26 items were formed into seven categories of personal events: marital difficulties, work difficulties, robbed/assaulted, interpersonal difficulties, financial problems, illness/injury, and legal problems. The individual items making up these subscales are available on request. Life events were not weighted. A twin’s “score” in an individual category reflects the total number of items therein that were positively endorsed.

Several categories of life events were applicable to only a subset of respondents. Analyses of marital and work difficulties were performed only in the subset of respondents who were married or had worked in the preceding year. Not all twins had equal numbers of individuals in their social networks. However, two different approaches to “correcting” for network size produced very modest changes in our results. To avoid complicating our analyses, we restricted them in this report to the raw data on network events, uncorrected for network size. For convenience, we use the term “life events” to refer to both true events, which are usually brief and temporally discrete, and to true difficulties, which are usually enduring with less clear onsets and offsets. We used previously validated questions to assess the similarity of childhood environment of the twins and their frequency of contact as adults.

STATISTICAL ANALYSIS

Total number of life events was treated as a continuous variable, but transformed by the function log(x+1) to normalize its positive skewness. The impact of age, sex, and

In a family study of depression, Bebbington et al found that depressed patients and their relatives had high levels of recent stressful life events. In a community sample, Breslau et al found that exposure to traumatic events was significantly predicted by a family history of psychiatric illness. In nearly 400 elderly Swedish twins reared together and apart, Plomin et al found that life events, reported during the entire life span, were significantly correlated in twin pairs, and the correlation in monozygotic (MZ) twins substantially exceeded that found in dizygotic (DZ) twins.

In this article our goal is to expand on those previous studies that suggest an important role for familial factors in the cause of life events. Specifically, we examine life events and difficulties reported over the last year in a large sample of twins from the population-based Virginia Twin Registry. We wish to address three major questions: (1) Can we replicate previous findings that recent life events are familial? (2) To what extent is the familial resemblance for recent life events and difficulties the result of genetic vs familial environmental factors? (3) Does the importance of these factors vary for different categories of life events?
zygosity on the number of reported life events and the impact of childhood similarity or time difference of reporting on the absolute within-pair difference in number of recorded events was assessed by standard regression for total life events and by logistic regression for the individual categories of life events. In regression analyses with individual twins, the non-independence of observations from members of a twin pair, which does not influence the accuracy of the regression coefficients but produces spuriously low SEs, was corrected as a function of the proportion of the sample that were complete twin pairs and the magnitude of the correlation of the dependent variable in those twin pairs. In calculating the SE, the first member of a complete twin pair is counted as a full individual, but the second member is "discounted" by the amount of variance in the dependent variable shared with the first member. Tests of significance reported herein are based on these corrected SEs.

Because the number of individual events in our categories was small, these categories could not be treated as continuous variables. Instead, we used a "liability-threshold" model which assumes that, underlying the observed discrete distribution of numbers of reported life events, there exists a continuous, normally distributed latent liability. When the discrete distribution contains only two categories, a tetrachoric correlation is calculated from the 2×2 contingency table of twin 1's response and twin 2's response to the single life event making up each category. The tetrachoric correlation obtained from a 2×2 table is a "perfect fit" and provides no test of the liability-threshold model. In life event categories with more than two events, a polychoric correlation is calculated from an N×N table, cross tabulating the responses of the two twins. A χ² goodness-of-fit test is then available to assess the adequacy of the liability-threshold model.

For total life events a variance-covariance matrix and for each separate category of life events a tetrachoric or polychoric correlation for twin 1 and twin 2 were computed separately for MZ and for DZ twins. Models were fitted to these matrices or correlations using the computer program LISREL. The goal of these analyses was to obtain estimates for the proportion of variance in these categories of life events that were due to the following: additive genetic factors (A); familial, shared, or "common" environment (C); those environmental factors shared by twins such as rearing environment, social class, school, etc.; and individual specific environment (E; those environmental influences unique to each member of a twin pair, including any unreliability of measurement). We began by fitting an ACE model that included additive genes, family environment, and individual specific environment. The fit of this model was assessed by a goodness-of-fit χ² test. We then fitted two simpler models that postulate markedly different causes for any observed familial aggregation of life events. The AE model assumes that all familial aggregation results from additive genetic effects, while the CE model assumes that all observed familial aggregation results only from shared environmental influences. The fit of each of these models can be compared, by a likelihood ratio χ² test with 1 df, with that found for the ACE model. The proportion of variance in liability to life events due to additive genes, common environment, and specific environment equals, respectively, a², c², and e², respectively.

Next, sex-dependent models were fitted to the covariance matrix or correlations of the five zygozy-sx-sex twin groups (i.e., MZ male, MZ female, DZ male, DZ female, and DZ opposite sex). These models allow for two kinds of sex differences: (1) differences in magnitude of genetic or environmental effects in the two sexes (sometimes termed the "scalar" sex-limitation or "environmental" model) and (2) differences in the actual genetic or environmental factors that influence the two sexes (sometimes termed the "non-scalar" sex limitation or "independent" model). For the scalar sex-limitation model, we would obtain separate estimates for A, C, and E in male and female subjects (denoted by the subscripts M and F, respectively). The non-scalar model would also include two correlations (rM and rF) that reflected, respectively, the correlation in the additive genetic and common environmental factors influencing male and female subjects. Since all unlike-sex pairs are DZ, it is impossible to estimate rM and rF simultaneously.

For all our twin analyses, the best-fitting model was chosen using the information criterion of Akaike, which equals the χ² value minus twice the df. The model with the lowest value of this parameter has the optimal combination of goodness of fit and parsimony. If a variable is substantially correlated with age, this age effect (which is, by force, shared by both members of a twin pair) can inflate estimates of E. To obtain unbiased parameter estimates, age can be incorporated directly into the structural equation.

In asking these questions, we do not wish to imply that genes "code" for life events as they do for eye color or blood group. It is, however, plausible that a number of human traits, like personality, which are influenced by genetic factors, affect the probability of experiencing life events. These questions are interesting because a major focus of psychiatric research has been to understand the relationship between environmental stress, usually conceptualized as adverse life events, and the onset or recurrence of psychopathologic conditions. It will be difficult to reach a fuller understanding of this key issue if we do not understand the major influences on the occurrence of adverse life events themselves.

RESULTS

SAMPLE

The mean (±SD) number of life events reported by the twins in this sample was 3.0±2.9, with 18.4% reporting no events. The frequency of life events reported in each of the 10 classes is seen in Table 1. Interpersonal dif-
ficulties and network crises were the most frequently reported, while being robbed or assaulted and experiencing legal problems were the least frequent. The endorsement rate for legal problems was so low as to preclude meaningful analysis and was excluded from further analyses.

**EFFECT OF SEX, AGE, AND ZYGOSITY ON FREQUENCY OF LIFE EVENTS**

Female subjects reported significantly more total life events and events in five of the nine categories: network illness/injury, network crises, financial problems, marital problems, and interpersonal difficulties. Total life events and four individual categories of events (interpersonal difficulties and financial, marital, and work problems) were significantly and negatively related to age. Network illness/injury was significantly and positively related to age. Zygosity had no significant effect on total number of life events and a significant difference was seen in two of the nine specific categories: DZ twins reported more network deaths and MZ twins reported more frequent marital difficulties.

**TWIN ANALYSES**

The goodness of fit of the multiple threshold model, assessed in those classes of life events with more than two categories, failed at the 5% level in two of the 42 polytomous correlations, a result close to chance expectation. There is significant familial resemblance for reported total life events. (Table 2): the correlations in MZ twins (+0.429) and DZ twins (+0.35) both differ significantly from zero (P<.0001).

The correlations of liability for MZ and DZ twins and for the five sex-zygosity categories of twins for individual classes of life events are also seen in Table 2. The correlation in MZ twins consistently exceeds that found in DZ twins, although the difference is sometimes small. The correlation in opposite-sex DZ twins varies from being similar to that found for same-sex DZ twins (eg, network deaths) to being much lower (eg, financial difficulties).

**MODEL FITTING TO THE TWIN DATA**

The fit of the three sex-independent models (ACE, CE, and AE) and the parameter estimates for the best-fitting model are seen in Table 3. For total life events, ACE was the best fitting model and estimated that 26% of the variance in total life events resulted from genetic factors, 18% resulted from familial-environment factors, and 57% resulted from individual-specific environmental factors.

Results of model fitting to the individual classes of life events, also seen in Table 3, suggest that the individual categories of life events were divisible into three groups: (1) all the network events (death, illness/injury, and crises) and work difficulties, where CE was the best-fitting model so that twin resemblance appeared to result solely from common environment; (2) four personal events or difficulties (robbed/assaulted, illness/injury, marital problems, and financial problems) where AE was the best-fitting model so that twin resemblance appeared to result only from genes; and (3) interpersonal difficulties where ACE was the best-fitting model and twin resemblance appeared to result from both common environment and genes.

Sex-dependent models, fit to the twin correlations, produced an improvement in fit over the best sex-independent model only for total life events and for illness/injury, financial problems, and marital problems. For total life events, the best-fitting model incorporated nonscalar sex-dependent effects, estimating A, C, and E to be equal in the two sexes and accounting for, respectively, 20%, 23%, and 57% of the variance. The same familial environmental factors influenced both sexes, but different genes were acting in male and female subjects (ie, r_1=1 and r_2=0). As noted above, estimates of r_E and r_A are confounded. This model for total life events fit only marginally better than the model in which r_A=1 and r_E=0.487. While there is evidence that different
Table 2. Twin Correlations for Various Classes of Life Events

<table>
<thead>
<tr>
<th>Life Event/Difficulty</th>
<th>No. of Categories</th>
<th>MZ Male</th>
<th>MZ Female</th>
<th>DZ Male</th>
<th>DZ Female</th>
<th>DZ OS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>2</td>
<td>283</td>
<td>367</td>
<td>308</td>
<td>309</td>
<td>309</td>
</tr>
<tr>
<td>Network</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>3</td>
<td>4.65</td>
<td>4.42</td>
<td>4.16</td>
<td>4.40</td>
<td>4.44</td>
</tr>
<tr>
<td>Illness/injury</td>
<td>4</td>
<td>3.46</td>
<td>3.30</td>
<td>3.42</td>
<td>3.34</td>
<td>2.94</td>
</tr>
<tr>
<td>Crisis</td>
<td>5</td>
<td>3.85</td>
<td>3.26</td>
<td>3.23</td>
<td>3.40</td>
<td>3.38</td>
</tr>
<tr>
<td>Interpersonal</td>
<td>5</td>
<td>3.89</td>
<td>3.02</td>
<td>3.40</td>
<td>3.37</td>
<td>2.97</td>
</tr>
<tr>
<td>Robbed/assaulted</td>
<td>2</td>
<td>2.31</td>
<td>1.86</td>
<td>2.25</td>
<td>2.24</td>
<td>0.66</td>
</tr>
<tr>
<td>Financial</td>
<td>2</td>
<td>2.47</td>
<td>1.21</td>
<td>2.44</td>
<td>2.22</td>
<td>1.81</td>
</tr>
<tr>
<td>Illness/injury</td>
<td>2</td>
<td>2.75</td>
<td>-0.01</td>
<td>3.76</td>
<td>2.28</td>
<td>1.59</td>
</tr>
<tr>
<td>Marriage</td>
<td>3</td>
<td>1.30</td>
<td>0.05</td>
<td>3.08</td>
<td>0.52</td>
<td>-0.63</td>
</tr>
<tr>
<td>Work</td>
<td>3</td>
<td>2.96</td>
<td>2.71</td>
<td>4.38</td>
<td>2.35</td>
<td>3.80</td>
</tr>
</tbody>
</table>

*If few twins endorsed high numbers of events in a given class of events, the upper end of the scale was collapsed into a single category. OS indicates opposite sex.
†Log transformed Pearson product-moment.
‡Goodness-of-fit $\chi^2$ $P<.05$.
§Tetrachoric.
¶Based on smaller sample size.

For financial problems and illness/injury, the best fitting sex-dependent model was also nonscalar. No evidence was found for common environmental effects; equal estimates were obtained for A in the two sexes (+1% for financial difficulties and 24% for illness/injury) and for genetic effects in the two sexes that were uncorrelated (i.e., $r=0$). For marital difficulties, model fitting suggested that twin resemblance in female subjects resulted solely from common environmental factors accounting for 12% of the variance, while in male subjects, twin resemblance resulted entirely from genetic effects, accounting for 15% of the variance.

**INCORPORATING AGE IN THE TWIN MODELS**

The frequency of total life events and six of the individual classes of events were significantly related to age. Since MZ and DZ twins are perfectly correlated for age, age effects can artificially bias upward estimates of familial environment. To assess the magnitude of this bias, age was added to the best-fitting model for the all seven kinds of life events that were significantly related to age. Age accounted for less than 5% of the variance in liability to life events. Estimates of $a^2$ and $c^2$ were essentially unchanged and estimates of $c^2$ were modestly reduced. Detailed results are available on request.

**POSSIBLE VIOLATIONS OF THE EQUAL ENVIRONMENT ASSUMPTION**

Our twin models assume that the trait-relevant environments for members of MZ and DZ twin pairs are equally correlated. In this data set, compared with DZ twins, MZ twins reported significantly more similar childhood environments ($P<.0001$) and had closer contact with one another as adults ($P<.0001$). To examine whether these indexes of environmental resemblance influence twin similarity in reported life events, we examined, in a regression analysis controlling for the effect of age, sex, and zygotism, the relationship between childhood similarity, frequency of contact, and the absolute within-pair difference in reported number of life events. The similarity of childhood environment had no significant effect on twin similarity for total life events and was a significant predictor for only one of the nine individual life event categories—a pattern of results that does not differ from chance expectation. However, frequency of contact as adults significantly predicted twin similarity for total life events and five of the nine individual categories: all three network events (death, illness/injury, and crises) and financial and work difficulties.

Given that MZ twins have significantly more frequent contact than DZ twins, it is possible that greater twin resemblance in MZ twins, which our model attributes to genetic influences, could have resulted from closer contact between the MZ twins. This is unlikely because the categories in which frequency of contact related significantly to twin resemblance were, with one exception, those categories in which model fitting showed that twin resemblance resulted entirely from familial environmental and not genetic factors. We addressed this question more rigorously by matching MZ and DZ twins on frequency of contact and then refitting our twin models. While detailed results are available on request, these analyses showed little difference in final results from the results obtained in our initial analyses of the entire data set.
supporting our previous impression that the greater frequency of contact in MZ vs DZ twins does not produce a substantial upward bias on the estimates of heritability.

**SOURCES OF FAMILIAL ENVIRONMENTAL INFLUENCES**

We performed three further analyses (results available on request) to determine the source of the familial-environmental influences on life events: (1) examining twin correlations in network events that must have been shared by both twins (eg, death of father) vs network events that might have been relevant for only one twin (eg, illness in a friend); (2) exploring the relationship between twin similarity and the time difference in the twins’ completion of the life event survey; and (3) comparing the correlations for life events in twins and within the same person across time. All of these analyses were consistent with the following hypothesis: for network events, common environment was largely the result of both twins reporting the same “contemporary” events, while for the personal difficulties, any common environment that was found was more likely to be the result of “enduring” familial-environmental effects, such as might be the result of the rearing environment.

**COMMENT**

The goal of this article was to clarify the role of familial factors in the etiology of reported life events. Our results support previous evidence that suggests that a variety of personal characteristics, which are influenced by familial factors, affect the probability of experiencing stressful life events. In a large, epidemiologic sample of twins, reported life events and difficulties experienced during the last year were substantially correlated in twin pairs. These correlations were consistently higher in MZ than in DZ pairs. For total life events, the best-fitting twin model suggested that genetic and familial-environmental factors each accounted for around 20% of the variance. For individual classes of life events, analyses suggested that a particularly meaningful distinction was between network and personal life events. Network events appeared to be uninfluenced by genetic factors, with the substantial twin resemblance resulting entirely from familial environmental influences. All personal events (with one exception—work difficulties) were significantly influenced by genetic factors, with common environmental influences playing little or no role.

Our results are somewhat different from those found in the one previous twin study of life events. Plomin et al[1] examined lifetime life events, an approach rarely used in psychiatric epidemiologic studies, rather than those during the last year. Compared with our results, they found for total events, a much higher heritability estimate (40%) and no evidence for familial-environmental effects. They also subdivided their life events, finding much higher heritability estimates for “controllable” than “uncontrollable” events. These findings have some similarity with our results with personal vs network events. Most network events are inherently uncontrollable, being independent of the respondent’s behavior, while many personal events are controllable in that they are potentially dependent on the individual’s own behavior.

**THE EQUAL ENVIRONMENT ASSUMPTION**

The validity of twin analyses of life events may be suspect because of violations of the equal environment assumption. Although similarity of childhood environment and frequency of contact as adults were greater in MZ than in DZ twins in our sample, similarity of childhood environment did not relate systematically to twin resemblance for life events. However, the fre-
frequency of contact as adults significantly predicted twin similarity for total and for five of nine individual classes of life events. Nevertheless, when we fit models to subsets of our data in which MZ and DZ twins were matched for frequency of contact, no significant change of parameter estimates was seen. It is unlikely that our results were substantially biased by violations of the equal environment assumption.

**SEX DIFFERENCES IN LIFE EVENTS**

We found two kinds of sex differences in reported life events: differences in mean number of reported events and differences in sources of variance. Except for being robbed or assaulted, female subjects reported more of all kinds of life events. For six of the nine categories of life events, however, including many that had large mean differences between the sexes, model fitting suggested that the sources of variance were the same in the two sexes. For example, although female subjects were much more likely than male subjects to report network crises or interpersonal difficulties, the magnitude and kind of genetic and/or environmental influences acting in the two sexes appeared to be the same.

This was not, however, the case for total events and for three categories of personal life events (illness/injury, financial difficulties, and marital difficulties) in which the best-fitting models included gender differences in the genetic and/or environmental etiologic factors. For at least some classes of life events, the familial factors that influence their occurrence appear to differ qualitatively in men and women. Our results suggest that different genetic factors influencing the probability of illness/injury in men and women are consistent with the finding that the personality profiles of men and women involved in serious automobile accidents are different.33,34

**SOURCES OF GENETIC AND FAMILIAL INFLUENCES**

We have shown significant genetic influences on total life events and on five of six categories of personal life events. How could genetic variation impact on the number of reported events? Clearly, life events are a "distal" phenotype, far removed from the level of gene expression. However, genetic influences on a variety of human personality traits have been demonstrated repeatedly,22 as has the association between personality variation and the probability of certain classes of life events.13,16,18 It is, therefore, plausible that genetically influenced traits such as impulsiveness, stability, frustration tolerance, and risk taking affect the probability of experiencing adverse life events.

By contrast, few consistent effects of the familial environment on human personality have been found.22,24 Social class may be one important familial-environmental influence. Not only is social class strongly familial, but low social class has been shown consistently to correlate with high levels of life events.30 Brown and Harris3 have written poignantly of a "conveyor belt of adversities" that begins with low social class of origin and poor parental care and leads to high levels of life events and symptoms in adulthood.

A substantial proportion of the life "events" considered in this article are enduring difficulties rather than point-in-time events. Interpersonal, financial, and marital difficulties, for example, are more often long-term problems than short-term events. Factors that influence the reporting of such events could alter the probability of onset or the probability of persistence. Thus, another plausible mode of action of genetic or familial-environmental influences on life events is as a predictor of persistence. We have shown, in this sample, that coping behavior is influenced by genes and familial environment.35 Different coping strategies could lead either to the rapid resolution or the persistence of interpersonal, financial, or marital difficulties.

**IMPLICATIONS**

Our results, which suggest that familial and genetic factors play an important etiologic role in recent life events and difficulties, have important implications for the ways in which life events are conceptualized in psychiatric research. In particular, our results argue strongly against the validity of a random model of life events, in which having lots of life events is just a reflection of "bad luck." We found that more than 40% of the total variance in life events was due to genetic or familial-environmental factors. These findings, coupled with evidence from previous studies, argue for a rethinking of our concept of the "environment" as indexed by life events and difficulties. Epidemiologists often conceptualize the environment as something "out there," which impinges on the organism in a unidirectional manner. Our results require a more dynamic concept of the environment where the individual and the environment influence one another in a bidirectional fashion.36

Psychiatric epidemiology and genetics should consider at least two other major causal pathways between life events and psychiatric illness. First, a common set of familial and genetic factors may influence the vulnerability to both life events and psychiatric illness so that, while correlated, the two are not causally linked.37 Second, the genetic or familial-environmental influences on mental illness may not "directly" increase vulnerability to illness, but may instead increase risk for psychiatric illness by predisposing individuals to create for themselves high-risk environments.38 Twin and family studies will complement standard epidemiologic designs in further unraveling the critical but complex interrelationship between life events and psychiatric disorders.
LIMITATIONS

The results of this article should be interpreted in the context of two potentially important limitations. First, the life events analyzed in this report were obtained by self-report questionnaires. Neither dating nor contextual information was obtained. While contextual information, such as the degree to which they were anticipated or the magnitude of the threat they posed, has been useful in understanding the relationship between life events and psychiatric illness, it is less clear that such information would be helpful in understanding the sources of variation in reported life events. McGuffin et al. in their family study of adversity and depression, used a personal interview that extensively assessed contextual information regarding life events and concluded that the familial aggregation of events could not be explained by the same event impinging on the proband and other family members.

Second, our data consist only of reported life events and difficulties. It is possible, and even plausible, that genetic and familial-environmental factors act not only at the level of experiencing life events but also influence whether the events will be recalled and reported, given that they have been experienced. However, our results remain relevant to the interpretation of the literature on reported life events, which constitute the bulk of previous studies of stressful life events and psychopathologic conditions.

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Leroy Thatcher, MS, assisted in the data analysis.

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