The Heritability of Life Events: An Adolescent Twin and Adoption Study

Heather R. Bemmels,[1] S. Alexandra Burt,[2] Lisa N. Legrand,[1] William G. Iacono,[1] and Matt McGue[1]

[1] Department of Psychology, University of Minnesota, Twin Cities Campus, Minneapolis, United States of America

[2] Department of Psychology, Michigan State University, East Lansing, United States of America

Although life events are often conceptualized as reflecting exogenous risk factors for psychopathology, twin studies have suggested they are heritable. We undertook a mixed twin/adoption study to further explore genetic and environmental contributions to individual differences in the experience of life events. Specifically, a sample of 618 pairs of like-sex adolescent twins, 244 pairs of like-sex adopted adolescent and young adult siblings, and 128 pairs of like-sex biological siblings completed a life events interview. Events were classified as independent (not likely to have been influenced by respondent's behavior), dependent (likely to have been influenced by respondent's behavior), or familial (experienced by a family member), and then summed to form three life event scales. Variance on the scales was assumed to be a function of four factors: additive genetic effects (a^2), shared environmental effects (c^2), twin-specific effects (t^2), and nonshared environmental effects (e^2). Data were analyzed using standard biometrical models. Shared environmental effects were found to be the largest contributor to variance in familial events ($c^2 = .71$; 95% confidence interval of .65, .76); additive genetic effects were the largest contributor to dependent events ($a^2 = .45$; CI = .31, .58); and nonshared environmental effects were found to be the largest contributor independent events ($e^2 = .57$; CI = .51, .64). A significant twin-specific effect was also found for independent life events, indicating that twins are more likely to be exposed to such events than non-twin biological siblings. Findings are discussed in terms of their implication for understanding the nature of psychosocial risk.

Researchers have long been interested in both the influences that help bring about life events, and the effects of the events themselves. This interest is partially due to the well-established association between the occurrence of stressful life events and the subsequent development of a broad range of general health and mental health conditions, from depression (e.g., Costello, 1982; Kendler et al., 1993; Surtees et al., 1986) to the common cold (e.g., Cohen et al., 1993). More specifically, genetically informative studies have allowed researchers to parse out the influence of genes and have established a causal, environmentally mediated link between life events and psychopathology, like depression (e.g., Kendler et al., 1999a). This link appears to be at least in part a gene-environment interaction, whereby environmental stress pushes those genetically vulnerable across a threshold into clinical disorder (Caspi et al., 2003; Kendler et al., 1995). At the same time, behavioral genetic research has determined that some of the association between stressful life events and depression is noncausal (Kendler et al., 1999a; Kendler & Karkowski-Shuman, 1997; Silberg et al., 1999). That is, certain individuals appear to either select or evoke environments that increase the likelihood that they will have stressful experiences.

Such environmental niche picking has been termed 'active' and 'evocative' gene–environment correlation (i.e., rGE; Scarr & McCartney, 1983). The theory is that the environment comes to reflect an individual's genotype through processes by which the individual actively (though possibly inadvertently) selects/evokes experiences consistent with their genetic propensities and evokes reactions from others that complement their genes. For example, those with a predilection towards rule breaking may find that they most enjoy the company of other rule-breakers and so seek out rule-breakers as friends (an active process) and are simultaneously rejected by those not interested in rule-breaking (an evocative process). Genetic predispositions are thus both expressed and magnified via life choices and the reactions that chosen behaviors elicit from others (Burt, in press).

Another reason for continued interest, closely intertwined with the last, is the heritability of life events. Their heritability serves as a reminder that caution must be used when interpreting environmental effects. Life events are only one of a series of examples of 'environmental' measures that have turned out not to be entirely environmental after all (e.g., Jaffee & Price, 2007; Plomin & Bergeman,

Heather R. Bemmels, S. Alexandra Burt, Lisa N. Legrand, William G. Iacono, and Matt McGue

1991). Indeed, it is likely the nonrandom, active and evocative nature of some life events that makes them heritable (e.g., Fergusson & Horwood, 1987; Jockin et al., 1996). As explained above, individuals are often active participants in the creation of their environments (Kendler & Eaves, 1986), opting themselves into situations with more or less probability of resulting in a stressful life event.

This theory of active and evocative mechanisms is supported by different categories of life events demonstrating different levels of heritability. It is common for researchers to distinguish between 'independent' and 'dependent' life events. Independent life events encompass experiences that seem to occur at random, outside of the control of the individual; for example, a close friend moving away. In contrast, dependent life events are those linked to an individual's choices and behaviors and are thus more controllable. These include actions or decisions knowingly made by the individual (e.g., quitting a job), those events that result from the dynamics of an interpersonal relationship (e.g., the ending of a romantic association), and those that are a consequence of the individual's behavior (e.g., getting evicted from an apartment). While not all studies have unequivocally supported the heritability distinction between independent and dependent life events (e.g., Saudino et al., 1997; Thapar & McGuffin, 1996), most have been relatively consistent with the above theory and interpretation. As would be hypothesized, independent life events have generally been found to be either not significantly heritable (Billig et al., 1996; Kendler et al., 1999b; Silberg et al., 2001) or only modestly heritable (Plomin et al., 1990; estimate of 18% heritability). Dependent life events, on the other hand, generally demonstrate heritability around 40 to 50% (Billig et al., 1996; Plomin et al., 1990; Rice et al., 2003; Sobolewski et al., 2001). For both types of events, the remaining environmental variance is largely of the nonshared variety, making this the primary influence for the independent life events.

While a consensus is developing, there remain some anomalous findings (Saudino et al., 1997; Thapar & McGuffin, 1996), suggesting a need for additional data on the topic. Here, we followed the example of Billig et al. (1996) and also added a third category of life events, that of 'familial' life events, to the standard distinction between independent and dependent events. Familial life events are events that happen to individuals within a participant's immediate family, such as the death of a grandparent or the father becoming unemployed. These events are shared by members of a family and, appropriately, have been found to be influenced primarily by the shared- or common-family environment (Billig et al., 1996; shared environmental estimate of 81%). Our data, consequently, will both expand and refine the literature addressing the etiological influences on independent, dependent, and familial life events.

We also add to the literature by addressing these questions using a mixed-sex, adolescent sample containing both twins and adoptees. All previous studies on the heritability of life events have been conducted on samples of twins. Other genetically-informative samples, such as biological and adoptive non-twin siblings, have not yet been examined. Should active and evocative gene–environment correlations actually underlie the experience of particular life events, then independent life events should be less heritable than dependent life events even in non-twin designs. To further increase the generalizability of our findings to non-twin groups, we tested biometric models that included a twin-specific environmental parameter, which factors out variance related to the unique circumstances of being a twin; for example, twins' matched age may increase the likelihood that they will experience the same life events. Additionally, we determined whether gender was an important moderator of the heritability of life events. Finally, ours is an adolescent and young adult sample. As life events are largely of interest because of their link to numerous disorders related to mental health,, and many of those disorders (e.g. depression, schizophrenia, eating disorders) often emerge during adolescence, the results of this sample will add valuable knowledge to the existing research literature.

We tested specific hypotheses derived from the literature. We presumed that dependent life events would be influenced by both genetic and nonshared environmental factors, while independent life events would be primarily influenced by the nonshared environment and show little evidence of heritable or shared environmental influence. Familial life events, on the other hand, were expected to be primarily influenced by the shared-family environment.

Methods

Participants

Participants were 626 pairs of same-sex twins and 617 pairs of non-twin siblings recruited to the Minnesota Center for Twin and Family Research (MCTFR; see Iacono, et al., 2006, for more information about the study). Complete life events information was available for both members of 618 twin pairs (monozygotic (MZ): female, $n = 221$; male, $n = 185$ pairs; dizygotic (DZ): female, $n = 114$; male, $n = 98$ like-sex pairs). To make the sibling sample comparable to the twin sample, only data from same-sex sibling pairs were used; this reduced the sibling sample from 617 to 376 pairs, which was further reduced to 372 with the requirement of complete life events information. Of these 372 pairs of siblings, 128 pairs were biological siblings (female, $n = 67$; male, $n = 61$). The remaining 244 pairs (female, $n = 148$; male, $n = 96$) were biologically unrelated to each other. The twins, born between 1972 and 1984, ranged in age from 16 to 18 (mean = 17.5 years; $SD = 0.5$); the siblings, born between 1978

and 1991, ranged in age from 11 to 21 (mean = 14.3; *SD* = 1.97) at the time of their assessment.

The recruitment of twin participants was made possible through State of Minnesota birth certificates which were used in conjunction with other publicly available resources (e.g., phone directories, internet search engines) to locate families who were then recruited by phone. Families were eligible to participate if they lived no more than a day's drive from the University of Minnesota campus and neither of the twins had a cognitive or physical disability that might interfere with their completing the assessment. Recruitment of non-twin siblings from adoptive and biological families was conducted in a manner similar to the twin recruitment, except that adoptive participants were recruited through records from the three largest adoption agencies in Minnesota. Eligibility requirements for the sibling families were also similar to those for the twin families, except they included having, instead of twins, two adolescents no more than five years apart in age who were between the ages of 11 and 21 for their intake assessment. For the adoptive families, we additionally required that the sibling pair be biologically unrelated, although one sibling could be the biological offspring of the adoptive parents. All adoptees were placed permanently in their adoptive home prior to the age of two years. For the nonadoptive families, we required that the siblings be full, biological siblings. More detailed information on assessments, inclusion criteria, as well as comparisons of participating to nonparticipating contacted families can be found in Iacono and McGue (2002) for the twins and McGue et al. (2007) for the sibling pairs.

To examine the generalizability of our sample, a comparison of MCTFR participants to United States Census data for Minnesota was conducted (Holdcraft & Iacono, 2004; McGue et al., 2007). The analysis of census data showed that twin participants were comparable to the Minnesota state population in ethnicity (i.e., 95% Caucasian) and socioeconomic status (SES). For the sibling families, the comparison showed a slightly higher rate of education in participating adoptive parents than the general population of the Twin Cities Metropolitan area from which they were recruited. However, the biologically related families were found to be comparable to standard demographics for married couples living with children in the state. The ethnicity of the adopted siblings, though, differed substantially, due to a large number of international adoptions: 67% of all adopted siblings were Asian–American, 21% Caucasian, 4% mixed race, 3% Hispanic/Latino, 2% African–American, 2% East Indian, 1% South Central American, and 0.1% other ethnicities.

Zygosity

Zygosity of the twins was determined through the agreement of three separate estimates: parents report on a standard zygosity questionnaire, staff evaluation of the twins' physical similarity, and an algorithm of physical measurement. Disagreements were resolved with a serological blood analysis. A previous analysis of the accuracy of this method, using 50 twin pairs, showed that the assessments perfectly predicted the blood-based classifications for that sample.

Measures

We used questions from the Life Events (LE) interview that were common across the twins' and siblings' assessments. Lifetime events were categorized into three nonoverlapping classes: independent, dependent, and familial, following the criteria specified by Billig et al. (1996). Specifically, events were classified as: (1) dependent if the participant had control over or inadvertently caused the event (e.g., Have you failed a course in school?; 14 events); (2) familial if the event was something experienced by everyone in the respondent's family but independent of the respondent's behavior (e.g., Are your parents divorced?; 19 events); or (3) independent if the event was not familial and the participant's behavior was not likely to have influenced the likelihood they had experienced the event (e.g., When did your body begin to change or develop due to puberty?; 8 events). Of the 41 events used in the present study, 37 were the essentially the same as those used by Billig et al. (1996), so that the classification used in that study, based on the consensus ratings of three psychology graduate students, was used here. The 4 events unique to this study (e.g., Have you ever had intercourse?, Have you ever gotten into trouble because of your use of drugs or alcohol?) were classified using the Billig criteria by the lead author of this paper. The full classification of the life events can be found in the Appendix.

Three LE scores were computed for each participant by summing the number of *yes* responses for each type of event. An analysis testing whether events were endorsed differently for twin and sibling groups revealed a significant ($p < .01$) group (MZ twin vs. DZ twin vs. biological sibling vs. adoptive sibling) effect across the three classes of life events, with both sibling groups reporting fewer events on average than MZ and DZ twins (i.e. roughly 1.5 fewer familial events, 1.25 fewer dependent events, and 1.25 fewer independent events). Age differences between the sibling and twin samples accounted for much but not all of this difference, as age is significantly correlated with each of the LE scores (familial $r = .25$, dependent $r = .41$, independent $r = .22$).

Analyses

Standard twin and adoption study methodology were the basis of the statistical analyses for this research. Specifically, we modeled the phenotypic variance for each life event scale as an additive function of: an additive genetic component (A), a shared environmental component (C), and nonshared environmental component (E), and a twin-specific environmental component (T). Sibling/twin correlations for the A component were 1.0 for MZ, 0.5 for DZ and biological siblings, and 0.0 for adoptive siblings. Since all our

Heather R. Bemmels, S. Alexandra Burt, Lisa N. Legrand, William G. Iacono, and Matt McGue

twins and siblings were reared together, we assumed that the correlation for C equaled 1.0 for all groups. We also assumed that the contribution of the E component to differences in twin/sibling pairs was the same across groups. The T component is correlated 1.0 for twins and 0.0 for non-twin siblings and was included to investigate possible differences across the twin and sibling data. Scales were log transformed prior to analyses to reduce positive skew.

Biometric models were fit to raw data using the structural equation modeling program, *Mx* (Neale et al., 1999). The Chi-square goodness-of-fit index (χ^2) was used to compare model fit; it was calculated as the difference in minus twice the log-likelihood values (–2lnL), which is distributed as a chi-square random variable under the null hypothesis of the more restrictive model. Chi-square values compared, for the three types of variables, the ACTE model with the standard ACE model to test for twin effects. Chi-square values were also used to compare models in which the two genders were allowed to vary from each other to models in which the genders were constrained to be equal to determine whether subjects' gender influenced the findings. To examine the influence of the sibling age differences in our sibling sample, we also compared models when sibling age difference was allowed to moderate sibling similarity for each life event scale. Convention dictates that if restricting a model does not result in a significant deterioration in fit, as evaluated by a chi-square difference test, then the more restrictive model (i.e., the more parsimonious model) is preferred.

Results

Prior to model-fitting analyses, intraclass correlations were computed for each class of life events. These correlations are computed separately for each twin/sibling type. When the MZ correlation exceeds the DZ correlation, genetic influences are implicated. Shared environmental influences are implied whenever the MZ correlation is less than twice the DZ correlation. The unrelated sibling (URT Sibs) correlation is also a direct estimate of shared environmental influences. Prominent nonshared environmental influences are implicated by small MZ correlations. Lastly, the biological sibling (Bio Sibs) correlation is expected to resemble the DZ correlation under the assumption that fraternal twins are no more similar, genetically or environmentally, than ordinary siblings. However, any differences in the similarity of DZ twins and Bio Sibs would be captured by the T effect.

Twin and sibling intraclass correlations are presented in Table 1. As seen there, dependent life events, especially in males, appear to be significantly influenced by genetic effects (i.e., *r*MZ > *r*DZ), though there is little evidence for genetic effects on the other classes of life events. Familial life events appear to be predominantly shared environmental in origin (*r*MZ = *r*DZ; *r*URT > 0). Independent life events, by contrast, appear to be largely nonshared environmental in origin (*r*MZ is rather

Table 1

Intraclass Twin and Sibling Correlations for the Three Categories of Life Events

	Class of life event		
	Familial	Dependent	Independent
Males			
MZ twins	0.79 (0.73, 0.84)	0.72 (0.64, 0.78)	0.33 (0.19, 0.45)
DZ twins	0.81 (0.73, 0.87)	0.43 (0.25, 0.58)	0.42 (0.24, 0.57)
Bio sibs	0.64 (0.46, 0.76)	0.3 (0.06, 0.51)	0.07 (–0.18, 0.31)
URT sibs	0.55 (0.40, 0.68)	0.33 (0.14, 0.49)	–0.01 (-0.21, 0.17)
Females			
MZ Twins	0.79 (0.74, 0.84)	0.65 (0.57, 0.72)	0.41 (0.30, 0.52)
DZ twins	0.69 (0.58, 0.78)	0.47 (0.31, 0.60)	0.38 (0.22, 0.53)
Bio sibs	0.64 (0.48, 0.76)	0.54 (0.35, 0.69)	0.19 (–0.05, 0.41)
URT sibs	0.62 (0.51, 0.71)	0.22 (0.06, 0.36)	–0.09 (–0.25, 0.07)

small). Finally, close inspection of the correlations suggests that there is something unusual about the twin condition for independent life events in particular. Namely, the Bio Sibs' correlation estimates do not fall within the confidence intervals of the DZ twins' correlation estimates, suggesting that the T parameter may contribute to independent life events.

Table 2 presents selected model fit comparisons. For both the familial and dependent life events, dropping the T parameter did not result in a significant deterioration in model fit; consequently, the ACE model was chosen for additional testing (i.e., effects of gender and age on familial and dependent life events were tested using ACE models). By contrast, T was determined to be a significant parameter for the independent life events. Thus, the ACTE model was used in all subsequent models assessing independent life events.

Next, we compared the fit of models in which parameter estimates were constrained to be equal across gender to models in which parameter estimates were free to vary across gender. Constraining the model parameters to be equal across gender led to nonsignificant changes in chi-square for all three types of life events. These results thus suggest that the AC(T)E estimates are invariant across gender.

The test of whether the age difference among the sibling sample affected our results, which is the comparison of models with and without an age-moderator variable, suggested no significant effects for sibling age differences. To summarize, we found no significant gender or sibling age difference effects, but did find that independent life events were best modeled with the inclusion of a twin-specific environmental parameter.

Table 2

Model-Fitting Tests for Gender, Age, and Twin Effects

Model	Fit statistics	Class of life event		
		Familial	Dependent	Independent
Twin effect	χ^2	0.09	0	9.99
	df	1	1	1
	p	ns	ns	0.002
Gender effect	χ^2	1.95	1.50	2.89
	df	3	3	4
	p	ns	ns	ns
Sibling age difference effect	χ^2	1.51	0.80	0.49
	df	1	1	1
	p	ns	ns	ns

Note: Twin Effect compares the ACTE models with the ACE models where T is fixed to zero. Gender Effect compares models in which parameter estimates were constrained to be equal across gender to models where estimates are free to vary. Age Difference Effect compares models with versus without a sibling age difference-moderator variable. Note that because a twin effect was only found for independent life events, Gender and Age models were run using ACE models for the familial and dependent life events but were run using an ACTE model for the independent life events.

Given the above results, biometric model-fitting results are presented for the entire sample (i.e., across age and gender). Standardized variance estimates, plus 95% confidence intervals, for the genetic (a^2), shared environmental (c^2), nonshared or unique environmental (e^2), and twin-specific environmental (t^2) components of variance are shown in Table 3 for each of the three classes of life events. As noted, the twin-specific environmental parameter was not included in the table for the familial and dependent life events as it was only significant for the independent life events (of note however, when included, T was estimated to be precisely zero for nonindependent life events).

The results indicate that, as hypothesized, genetic factors are most important for dependent life events, explaining 45% of the variance. Nonshared environmental influences also contributed (33% of the variance). There was also a moderate but significant effect of the shared environment on dependent life events (22%). For independent life events, the nonshared environment was most important, as hypothesized, explaining 57% of the variance. However, the remaining variance (i.e., 32%) was specifically associated with being a twin, and indicated that DZ twin similarity was greater than expected given what was observed in the biological sibling data. Genetic and shared environmental contributions to independent life events were not statistically different from zero, as both had confidence intervals containing zero. Finally, familial life events were, as expected, primarily a consequence of shared (71%) and nonshared (24%) environmental factors.

Discussion

Our results corroborate prior work in suggesting that individual differences in dependent life events, and only dependent life events, are significantly influenced by

Table 3

Parameter Estimates and 95% Confidence Intervals for Additive Genetic (a^2), Shared Environmental (c^2), Nonshared Environmental (e^2) and Twin-Specific Environmental (t^2) Components of Variance

Scale	a^2	c^2	e^2	t^2
Familial	0.05	**0.71**	**0.24**	—
	(0.00, 0.12)	(0.65, 0.76)	(0.21, 0.27)	
Dependent	**0.45**	**0.22**	**0.33**	—
	(0.31, 0.58)	(0.11, 0.32)	(0.29, 0.38)	
Independent	0.07	0.04	**0.57**	**0.32**
	(0.00, 0.30)	(0.00, 0.15)	(0.51, 0.64)	(0.11, 0.47)

Note: Confidence intervals that do not overlap with zero indicate that the parameter is statistically significant at $p < .05$. These parameters are highlighted in bold.

genetic factors. Familial life events were, perhaps not surprisingly, predominantly shared environmental in origin. By contrast, independent life events were largely nonshared environmental in origin, consistent with the notion that random events or events that appear to be outside of one's control are not heritable. These results thus constructively replicate those of prior studies (e.g., Billig et al., 1996; Plomin et al., 1990).

One result that was not altogether anticipated by the existing literature was our finding that, in addition to genetic influences, shared environmental factors appear to play a moderate but significant role (22%) in dependent life events. Research with adult samples has found no evidence of shared environmental influences on dependent life events (e.g., Plomin et al., 1990; Saudino et al., 1997). Importantly, however, analyses on samples with participants less than 18 years of age (Billig et al., 1996; Rice et al., 2003; Silberg et al., 1999) have revealed shared environmental influences to be an important factor in dependent life events. Thus, it may be that common or familial environmental influences do play a role, at least prior to adulthood, in individuals' choices and voluntary experiences.

Though they did not form part of our original hypotheses such findings, nevertheless, support a current theory in the field of behavioral genetics: that the impact of the shared family environment is at its strongest in childhood and adolescence and then fades to nonsignificance by adulthood. It is believed that as individuals leave their childhood home and shape their own destinies, shared environmental influences decrease while genetic influences increase. This shift in importance during development, from shared environmental influences to genetic influences, has been supported by studies on antisocial behaviors and traits (Lyons, et al.; 1995; Rhee & Waldman, 2002), alcohol use (Rose et al., 2001), cognitive abilities (McGue et al., 1993), and various personality traits and related behaviors (Eaves et al., 1997; Koenig et al., 2007; McGue et al., 1993). The dependent life event scale used in the present study included items like, 'Have you ever been suspended from school?', 'Have you

Heather R. Bemmels, S. Alexandra Burt, Lisa N. Legrand, William G. Iacono, and Matt McGue

ever run away from home?' Items like these are likely related to the externalizing construct, which Bergen et al. (2007) recently showed in a meta analysis to increase in heritability but decrease in shared environmental influence from adolescence to early adulthood. Thus, our results are consistent with prior work in suggesting that shared environmental influences may make important contributions to human behaviors and experiences prior to adulthood (Burt et al., 2007).

The results for independent life events were noteworthy in that we were able to show that this type of life event is significantly influenced by the circumstance of being a twin. While we categorized life events as independent based upon their being random or outside of the subject's control (e.g., 'Has a close friend of yours died?'), it seems plausible that twins, because of their matched age, are more apt to be exposed to the same life events. The DZ twins in our sample were markedly more similar for independent life events than were Bio Sibs, even though both types of sibling share a family environment and 50% of their segregating genes. When we ran a basic ACE model for independent life events, heritability was estimated at 40%; however, this fell to a nonsignificant 7% when a twin-specific environmental parameter (T) was added to the model, an addition which improved model fit. The model including T was also run for the familial and dependent life events, but findings from this analysis (t^2 = 0) revealed that the twin effect seems to only influence the independent events in our sample.

The significance of the twin-specific environmental parameter for independent life events underscores the value of including non-twin siblings as well as twins in the analyses. Ours is the first study to examine life events in both an adopted and biological sibling sample, and the first to combine twins and non-twin siblings (both biologically-related and adoptive). Accordingly, the consistency of findings across our twin and sibling samples (seen in the intraclass correlations), particularly, when combined with our population-based design, highlights the validity of our final estimates. For example, the correlations among biologically unrelated siblings (URT Sibs) serve as direct measures of shared-environmental effects, and these correlations fully support the results of the formal modeling in demonstrating substantial and significant shared environmental contributions to both familial and dependent life events.

An additional strength of the present study is its inclusion of both male and female adolescents. Several previous studies have either investigated only one gender (e.g., Billig et al., 1996; Plomin et al., 1990) or found conflicting results across gender (e.g., Saudino et al., 1997). In contrast, the patterns of correlations observed in Table 2 are very similar across males and females, and formal biometric modeling determined that the magnitudes of the genetic and environmental components of variance do not differ significantly by gender. We can thus conclude that although there are

recorded differences in numbers of life events experienced based on gender (Kendler et al., 2001), the etiological nature of these life events does not vary across gender.

These findings also provide support for theories of active and evocative rGE in that events that are influenced by our choices and behaviors are more heritable than those that are not. This information can be use to expand the understanding of psychological traits and disorders in future research. For example, several previous studies found that participants frequently experienced a dependent life event during or before onset of a psychological disorder (Harkness & Luther, 2001; Kendler, et al., 1999a; Poulton & Andrews, 1992; Williamson et al., 1995), suggesting individuals are effectively choosing (either intentionally or inadvertently) environments and behaviors consistent with their genotype. If this is the case, heritability for psychological disorders, like depression, would be partially mediated through genetically influenced behaviors that increase the probability of experiencing a dependent event. This heritable event, when sufficiently influential, would then increase the likelihood that a susceptible person would experience an episode of a given disorder. Whether it is the genetic susceptibility to the disorder itself that influences the individual to select situations that increase the chance of experiencing a disorder-triggering event is one question that could also be answered through future research.

In conclusion, our findings are important on several levels. Foremost, they supported our hypotheses, thereby providing additional evidence for theories proposed in the literature. Namely, life events over which we have some control appear to be partially genetic in origin, whereas those over which we do not have any control (i.e., familial and independent) are predominantly environmental in origin (though the type of environment appears to be specific to each class of life event). Such results bolster theories regarding active and evocative rGE. Importantly, our findings are reinforced by the inclusion of adoptive and biological siblings, groups that have never before been used to test influences on life events. Adding these groups to our analyses likely serves to increase the generalizability of our findings. Future research should continue to explore extended twin designs such as that used here.

References

Bergen, S. E., Gardner, C. O., & Kendler, K. S. (2007). Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: A meta-analysis. *Twin Research and Human Genetics, 10,* 423–433.

Billig, J. P., Hershberger, W. G., Iacono, W. G., & McGue, M. (1996). Life events and personality in late adolescence: Genetic and environmental relations. *Behavioral Genetics, 26*, 543–554.

Burt, S. A. (in press). Genes and popularity: Evidence of an evocative gene–environment correlation. *Psychological Science*.

Burt, S. A., McGue, M., Krueger, R., & Iacono, W. G. (2007). Environmental contributions to adolescent delinquency: A fresh look at the shared environment. *Journal of Abnormal Child Psychology, 35*, 787–800.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301*, 386–389.

Cohen, S., Tyrrell, D. A., & Smith, A. P. (1993). Negative life events, perceived stress, negative affects, and susceptibility to the common cold. *Journal of Personality and Social Psychology, 64*, 131–140.

Costello, C.G. (1982). Social factors associated with depression: A retrospective community study. *Psychological Medicine, 12*, 329–339.

Eaves, L., Martin, N., Heath, A., Schieken, R., Meyer, J., Silberg, J., Neale, M., & Corey, L. (1997). Age changes in the causes of individual differences in conservatism. Age changes in the causes of individual differences in conservatism. *Behavior Genetics, 27*, 121–124.

Fergusson, D. M. & Horwood, L. J. (1987). Vulnerability to life event exposure. *Psychological Medicine, 17*, 739–749.

Harkness, K. L., & Luther, J. (2001). Clinical risk factors for the generation of life events in major depression. *Journal of Abnormal Psychology, 110*, 564–572.

Holdcraft, L. C. & Iacono, W. G. (2004). Cross-generational effects on gender differences in psychoactive drug abuse and dependence. *Drug and Alcohol Dependence, 74*, 147–158.

Iacono, W. G., & McGue, M. (2002). Minnesota Twin Family Study. *Twin Research, 5*, 482–487.

Iacono, W. G., McGue, M. & Krueger, R. F. (2006) Minnesota Center for Twin and Family Research. *Twin Research and Human Genetics, 9*, 978–984.

Jaffee, S. R. & Price, T. S. (2007). Gene-environment correlations: A review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry, 12*, 432–442.

Jockin, V., McGue, M., & Lykken, D. T. (1996). Personality and divorce: A genetic analysis. *Journal of Personality and Social Psychology, 71*, 288–299.

Kendler, K. S. & Eaves, L. J. (1986). Models for the joint effect of genotype and environment on liability to psychiatric illness. *American Journal of Psychiatry, 143*, 273–289.

Kendler, K. S. & Karkowski-Shuman, L. (1997). Stressful life events and genetic liability to major depression: Genetic control of exposure to the environment? *Psychological Medicine, 27*, 539–547.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999a). Casual relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry, 156*, 837–841.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999b). The assessment of dependence in the study of stressful life events: Validation using a twin design. *Psychological Medicine, 29*, 1455–1460.

Kendler, K. S., Kessler, R. C., Neale, M. C., Heath, A. C., & Eaves, L. J. (1993). The prediction of major depression in women: Toward an integrated etiologic model. *American Journal of Psychiatry, 150*, 1139–1148.

Kendler, K. S., Kessler, R. C., Walters, E. E., MacLean, C., Neale, M. C., Health, A. C., & Eaves, L. J. (1995). Stressful life events, genetic liability and onset of an episode of major depression in women. *American Journal of Psychiatry, 152*, 833–842.

Kendler, K. S., Thornton, L. M., & Prescott, C. A. (2001). Gender differences in the rates of exposure to stressful life events and sensitivity to their depressogenic effects. *American Journal of Psychiatry, 158*, 587–593.

Koenig, L. B., McGue, M., Kruger, R. F., & Bouchard, T. J. (2007). Genetic and environmental influences on religiousness: Findings for retrospective and current religiousness ratings. *Journal of Personality, 73*, 471–488.

Lyons, M. J., True, W. R., Eisen, A., Goldberg, J., Meyer, J. M.., Faraone, S. V., Eaves, L. J., & Tsuang, M. T. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry, 52*, 906–915.

McGue, M. (1993). Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental Psychology, 29*, 96–109.

McGue, M., Bouchard, T. J., Iacono, W. G., & Lykken, D. T. (1993). Behavior genetics of cognitive ability: A life span perspective. In R. Plomin & G. E. McClearn (Eds.), Nature, nurture, & psychology (pp. 59–76). Washington, DC: American Psychological Association.

McGue, M., Keyes, M., Sharma, A., Elkins, I., Legrand, L., Johnson, W., & Iacono, W. G. (2007). The environments of adopted and non-adopted youth: Evidence on range restriction for the Sibling Interaction and Behavior Study (SIBS). *Behavior Genetics, 37*, 449–462.

Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (1999). *Mx: Statistical modeling* (5th ed.). Richmond, VA: Department of Psychiatry.

Plomin, R. & Bergeman, C. S. (1991). The nature of nurture: Genetic influence on "environmental" measures. *Behavior and Brain Sciences, 14*, 373–386.

Plomin, R., Lichtenstein, P., Pedersen, N., McClearn, G.E., & Nesselroade, J.R. (1990). Genetic influences on life events during the last half of the life span. *Psychology and Aging*, 5, 25–30.

Poulton, R. G., & Andrews, G. (1992). Personality as a cause of adverse life events. *Acta Psychiatrica Scandinavica, 85*, 35–38.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin, 128*, 490–529.

Rice, F., Harold, G. T., & Thapar, A. (2003). Negative life events as an account of age-related differences in the genetic aetiology of depression in childhood and adolescence. *Journal of Child Psychology and Psychiatry, 44*, 977–987.

Rose, R. J., Dick, D. M., Viken, R. J., & Kaprio, J. (2001). Gene-environment interaction in patterns of adolescent drinking: Regional residency moderates longitudinal influences on alcohol use. *Alcoholism: Clinical & Experimental Research, 25*, 637–643.

Saudino, K. J., Pedersen, N. L., Lichtenstein, P., McClearn, G. E., & Plomin, R. (1997). Can personality explain genetic influences on life events? *Journal of Personality and Social Psychology, 72*, 196–206

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype à environment effects. *Child Development, 54*, 424–435.

Silberg, J., Pickles, A., Rutter, M., Hewitt, J., Simonoff, E., Maes, H., Carbonneau, R., Murrelle, L., Foley, D., & Eaves, L. (1999). The influence of genetic factors and life stress on depression among adolescent girls. *Archives of General Psychiatry*, 56, 225–232.

Silberg, J., Rutter, M., Neale, M., & Eaves, L. (2001). Genetic moderation of environmental risk for depression and anxiety in adolescent girls. *British Journal of Psychiatry*, 179, 116–121.

Sobolewski, A., Strelau, J., & Zawadzki, B. (2001). The temperamental determinants of stressors as life changes: A genetic analysis. *European Psychologist, 6*, 287–295.

Surtees, P. G., Miller, P. M., Ingham, J. G., Kreitman, N. G. Rennie, D., & Sashidharan, S. P. (1986). Life events and the onset of affective disorder: A longitudinal general population study. *Journal of Affective Disorders*, 10, 37–50.

Thapar, A. & McGuffin, P. (1996). Genetic influences on life events in childhood. *Psychological Medicine, 26*, 813–820.

Williamson, D. E., Birmaher, B., Anderson, B. P., Al-Shabbout, M., & Ryan, N. D. (1995). Stressful life events in depressed adolescents: The role of dependent events during the depressive episode. *Journal of American Academic Child and Adolescent Psychiatry, 34*, 591–598.

Appendix A

The Heritability of Life Events: An Adolescent Twin and Adoption Study

Event class	Specific event
Independent Life Events (8 items)	Have any of your close friends ever moved away so you couldn't see them much anymore?
	Was a close friend of yours ever seriously ill or hurt?
	Has a close friend of yours died?
	Has your body begun to change or develop due to puberty?
	Have you been teased a lot because your body is changing too slowly or too quickly?
	Have you worn or started wearing braces?
	Have you started to get pimples?
	Were you ever mugged or robbed?
Dependent Life Events (14 items)	Have you ever been suspended or expelled from school?
	Did you ever not make an after school activity (sport, club, or group) that you wanted to participate in?
	Have you ever had a serious problem with a close friend?
	Have you ever run away from home overnight?
	Have you started dating?
	Have you and a romantic partner ever broken up?
	Have you had intercourse?
	Have you or your romantic partner ever become pregnant?
	Have you or your romantic partner given birth to a child?
	Did you or your romantic partner ever have an abortion?
	Have you ever gotten into trouble because of your use of drugs or alcohol?
	Have you ever been in trouble with the police (for traffic violations or any other reason)?
	Have you ever had to go to court?
	Were you ever sent to a juvenile detention center?
Familial Life Events (19 items)	Has your family ever moved to a new neighborhood?
	Have you ever changed schools because your family moved or because your parents sent you to a different school (e.g., parochial school, home schooling)?
	Have you ever had a family, pet, like a dog or a cat that died?
	Have any of your close relatives died?
	Have any of your brothers or sisters ever run away from home overnight?
	Has your family ever had problems with money?
	Has your family ever received money from a government agency (welfare, food stamps, AFDC, disability)?
	Has your family ever had money cut off by a government agency?
	Have there been times when your parents (or other adults living in your home) argued a lot?
	Have your parents ever lived apart because they couldn't get along?
	Since you were born, have your parents ever dated other people?
	Has a new adult come to live with your family?
	Did you ever go live with another parent or guardian?
	Has either of your parents not been available very much?
	Have any of your brothers or sisters not been available very much?
	Have your parents, brothers, or sisters ever gotten into trouble because of their use of drugs or alcohol?
	Have your parents, brothers, or sisters ever been arrested or sent to jail?
	Has anyone in your family ever tried to kill himself or herself?
	Has a member of your family killed himself or herself?

Note: Bemmels, Burt, Legrand, Iacono, & McGue (submitted)