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Gene–Environment Correlations in the Stress–Depression Relationship

Jason Schnittker[1]

Abstract

A critical feature of the social stress model is the apparent relationship between stress and depression. Although many studies have demonstrated a connection between the two, the relationship may be contaminated by genes affecting both stress and depression. Using a sample of identical and fraternal twins, this study explores genetic influences on depression and assorted sources of stress while explicitly estimating, and thereby controlling for, gene–environment correlations. I consider both stress and depression in a fine-grained fashion. For the former, the study explores assorted sources of stress, including health and disability, family, unemployment, discrimination, and perceived neighborhood safety, as gene–environment correlations may be stronger for some forms of stress than others. For the latter, the study explores both depressive symptoms and major depressive disorders, as each may entail a different epidemiological process, especially with respect to genes. The results reveal that most, but not all, measures of stress have moderate heritabilities, suggesting that genes influence exposure to the environment in a broad fashion. Yet, despite this, the relationship between stress and depression is generally robust to gene–environment correlations. There are some notable exceptions. For example, allowing for gene–environment correlations, marital conflict is generally unrelated to depression. Moreover, gene–environment correlations are generally stronger for major depression than for depressive symptoms, encouraging further elaboration of the distinction between the onset of depression and its recurrence, especially in the context of genes. These exceptions do not put limits on environmental influence, but do suggest that genes operate in a complex life-course fashion.

Keywords

genes, depression, stress, behavioral genetics

A critical feature of the social stress model is the apparent relationship between stress and depressive symptoms (Pearlin and Schooler 1978; Turner and Lloyd 1999). Stress is defined as any environmental, social, or psychological demand that necessitates readjustment, a definition that aligns a psychological and physiological concept with the environmental focus of sociology (Selye 1956). The evidence linking stress to depression is voluminous, and virtually all of it suggests that both recent stressful events and, especially, chronic stress increase depressive symptoms (Kessler 1997; Mazure 1998). Although most people who experience a negative life event do not develop major depression, the relationship between stress and depression is nevertheless strong and compelling, providing sociologists with a mechanism whereby the social environment "gets under the skin," as well as an explanation for assorted differentials in well-being. Emphasizing the view that depression is fundamentally social in nature, sociologists regularly interpret stress as a "socially modifiable contingency" (Turner 2003).

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Yet a good deal hangs on the "nature" of environmental influence. There remains lingering concern over whether the stress–depression association is, in fact, causal and, if it is, whether the average association revealed in conventional regression models obscures more complex pathways (Hammen 1992, 2005; Kessler 1997; Mazure 1998). Concern over causality is not new, of course. Indeed, causality has been part of the debate since scholars began studying the linkages between stress and depression (Dohrenwend 1974). Nevertheless, recent skepticism has been expressed in new ways, with much of the concern stemming from evidence for the pervasive influence of genes. At a minimum, we know that genes influence depression, with most estimates of the heritability of major depression lying somewhere between 30 and 40 percent (Caspi et al. 2003; Kendler and Prescott 2006; Plomin et al. 2005). Perhaps more importantly, we know that genes also influence many behaviors and putative features of the environment, putting genetic influences front and center in the minds of most skeptics (Kendler, Gardner, and Prescott 2003; Kendler and Baker 2007; Rutter 2000). A key question remains concerning whether the same genetic influences underlie both stress and depression. At a general level, this seems very likely, and some sociologists encourage analysts to assume, as a matter of course, that the estimated relationship between any social cause and outcome will be biased by genetic influences (Freese 2008). This is no less the case in the stress literature, wherein there are both conceptual and empirical clues pointing to the relevance of gene–environment correlations. Indeed, some of the most important stressors from the standpoint of sociological theory may be the most vulnerable. Chronic stress, for example, may be rooted in strong genetic propensities insofar as it implies persistent influences related to the environment (Fergusson and Horwood 1987).

In this study, I examine the relationship between stress and depression using a genetically sensitive design. I use a sample of identical and fraternal twins, from which I estimate models that allow for independent genetic influences on both stress and depression and, more importantly, correlations among these influences. Other aspects of the analysis allow me to unpack the stress–depression relationship even further. For one, I explore assorted sources of stress, including chronic and eventful stress, and stress to both the individual and his or her social network. Furthermore, I explore both depressive symptoms, reflecting an additive continuum of depression severity, and major depressive disorder, reflecting a categorical assessment based on clinical criteria. Recent scholarship has encouraged the simultaneous use of categorical and dimensional measures of depression, as each may imply different epidemiological processes (Kessler 2002) and these distinctions may be particularly sharp with respect to genes (Kendler, Thornton, and Gardner 2001).

BACKGROUND

Gene–environment correlations are usually conceptualized as genetic control over environmental exposure, although they refer only to the co-occurrence of certain genotypes with certain environments (Kendler and Eaves 1986; Plomin, DeFries, and Loehlin 1977). Such correlations can take three forms: (1) they may be passive, when individuals inherit family environments that are correlated with genes; (2) they may be active, when individuals select or modify their experiences in ways that are correlated with their genes; or (3) they may be reactive, when individuals evoke reactions from their environment on the basis of their genetic endowment. All three correlations are likely present in the stress–depression relationship, but active and reactive correlations are particularly relevant to sociology, wherein a primary concern is with how contemporaneous environments impinge upon the individual (Selye 1956; Thoits 1995), as well as how individual agency shapes exposure to stress and its consequences (Thoits 2006). These concerns encourage those interested in genetic influences to think about current environmental exposures in light of genetic propensities, as genes are related to traits relevant to behavior.

Although research from psychiatry and behavioral genetics at this point provides the bulk of the evidence pertaining to gene–environment correlations, the influence of these correlations can be found within sociology as well. One potential sign of their influence is that most indicators of environmental stress show some heritability. Heritability refers to the fraction of variance in a phenotype within a population due to genetic influences. Reviewing twin studies, Kendler and colleagues (2007) find that genetic influences over behaviors and environments are moderate in impact but pervasive in extent, with an average weighted heritability of just under 30 percent. Genetic influences are apparent across different domains of stress (including life events and marriage quality), across

different types of reports (including self, observer, and informant reports), and across both dependent and independent events (events for which the individual could plausibly and directly exert agency). The fact that genetic influences are so widely diffused across areas of life suggests that no type of stress is entirely immune from the actions of genes. While most now accept the idea that many phenotypes and behaviors are at least partially heritable (Turkheim 2000), the implications of heritability remain contested.

Heritability alone does not mean that the stress–depression relationship is contaminated. Genetic influences are important only insofar as the same genetic influences behind stress are also behind depression. Although not always expressed in terms of shared influences, there are assorted pieces of evidence for at least partial co-determination of this sort. Some individuals, for example, consistently report more stressful life events than others, and these individual differences have been linked, in turn, to assorted genetically influenced traits, such as personality and mood (Fanous et al. 2002; Jocklin, McGue, and Lykken 1996; Kendler and Eaves 1986; Kendler and Karkowski-Shuman 1997; Plomin 1994; Plomin et al. 1990). Even apart from these specific traits, general psychological influences can shape the environment in appreciable ways, as sociologists have long recognized, occasionally in an explicitly genetic fashion (Freese 2008; Pescosolido et al. 2008; Shanahan et al. 2008). Those in "good mental health," for example, may be better able to engage in problem-solving and actively pursue a desired course of action (Thoits 2006). Similarly, in a wide-ranging review, Turner (2003) notes that many established risk factors for depression may matter only because they serve as markers for other risk factors, including those rooted in personal character. Although this comment was meant to emphasize the relevance of stress, it implicitly highlights the relevance of gene–environment correlations by invoking comorbid conditions as markers for dispositional factors. Other features of the sociological literature likewise hint at the potential relevance of gene–environment correlations.

Depressive Symptoms versus Depressive Disorders

Previous research notes a distinction between depressive symptoms (so-called "dimensional" conceptualizations) and depressive disorders (or "categorical" conceptualizations), but within sociology dimensional conceptualizations have assumed preeminence as they are thought to best capture the influence of social factors (Horwitz 2002). Although many of the risk factors for depressive symptoms are in any case associated with depressive disorders as well, it is important to reconsider the distinction when discussing gene–environment correlations. The genetic influences behind disorders may be different from those behind symptoms, necessitating the integration of categorical and dimensional assessments (Kessler 2002).

The "kindling" hypothesis is the most well-studied conceptualization of the relationship between depression and stress in a genetic and life-course context. The hypothesis posits that the relationship between stress and major depression declines with previous episodes of the disorder, while the risk of recurrence increases. Although major depression and stress are initially related, depression becomes independent of stress with multiple episodes of the disorder, as individuals are already "kindled" for the depressive response and therefore do not require much environmental "spark" for a new episode (Post 1992). Research using twins shows that the kindled state may be reached either through many previous depressive episodes driven by stress or through a high genetic risk (Kendler et al. 2001). In the context of genetically informative data, the kindling hypothesis implies a distinction between current major depression and depressive symptoms, as the former usually represents the recurrence of an earlier episode whereas the latter is merely the sum total of current depressive symptoms. Given a difference in their implied courses, there may be stronger gene–environment correlations for major depression insofar as such correlations capture earlier influences more in the case of major depression than in the case of depressive symptoms. Those who are genetically at risk for major depression may require environmental stress to initiate the disorder, but when eliminating gene–environment correlations, the analyst estimates the influence of environmental stress on the recurrence of depression, thereby reducing the apparent effect of stress relative to models that implicitly combine onset and recurrence.

Bivariate ACE Models with and without Gene–Environment Correlations

To explore gene–environment correlations, this study uses bivariate ACE models (Neale and Cardon 1992;

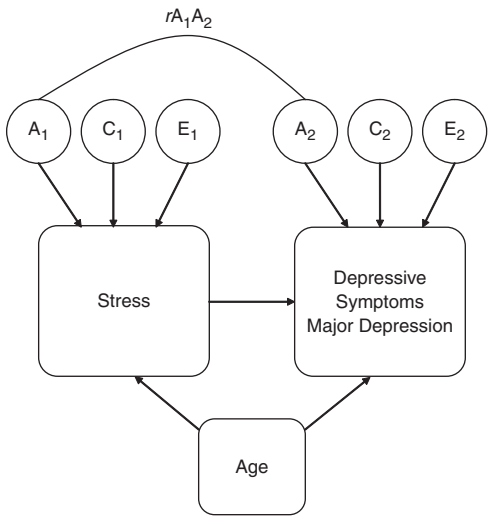

Figure 1. Conceptual Model of Relationships among Genetic Factors, Stress, and Depression

Plomin et al. 2005). Figure 1 presents the basic model for a single twin. The ACE framework is a two-group structural equation fit to covariance matrices for different twin types in which additive genetic (A), common environment (C), and unique environment (E) influences are estimated as latent variables. Separate ACE structures are estimated for stress, on the right-hand side of the equation, and depression, on the left-hand side of the equation. Although Figure 1 presents the structure of the bivariate model for one twin, an identical model is estimated for the other member of a twin pair. In this structure, the contribution of genes to a phenotype is identified by correlating the A factors at different levels for different twin types, reflecting their different degrees of relatedness. Specifically, the A factors are correlated at 1 for monozygotic (MZ) twins and .5 for dizygotic (DZ) twins (since MZ twins share 100% of their genes at conception, whereas DZ twins share on average 50%).[1] So specified, the ACE model assumes additive genetic influences, but this assumption is appropriate for this study: Models for additive genetic influences fit virtually all the symptoms of depression better than models for dominance effects (Kendler et al. 1986; Sullivan, Neale, and Kendler 2000). The C factors, meanwhile, are correlated at 1 for both MZ and DZ twins, while the E factors are uncorrelated. The E factor captures the influence of unique environments, including measurement error, as well as many contemporaneous environments, as most adult twins no longer share many common environments in their day-to-day lives.

Although the ACE structure provides the foundation for the models presented below, I am interested primarily in bivariate relationships rather than univariate biometric quantities. That is, I am interested in the correlation between the A factors for stress and depression more than the A factors themselves. The key to identifying this correlation is in the cross-trait, cross-twin correlations. In the univariate case, a trait has a high heritability if the within-trait, cross-twin correlation is larger for MZ twins than for DZ twins, implying greater similarity based on genetic relatedness. In the bivariate case, the cross-trait, cross-twin correlation is used to decompose the covariance between two traits. If, for example, the cross-twin stress–depression correlation is higher for MZ twins than DZ twins, then the within-twin correlation between stress and depression is likely to reflect genetic factors. Because they rely in part on different quantities, the results of bivariate covariance decomposition need not reflect those of univariate variance decomposition. A correlation between two variables can result from genetic factors even if the influence of genes on the variables comprising the correlation is weak. There are, of course, other possibilities. If the cross-twin stress–depression correlation differs from zero and is similar for MZ and DZ twins, then shared environmental factors contribute to the correlation. And if the same correlation is zero for both MZ and DZ twins, then individual-specific environments contribute. The expected biometric variance-covariance matrix for the bivariate case is presented in Plomin's (2005) *Behavioral Genetics*, Table A.4 (p. 362).

In the analyses that follow, I estimate two models for each source of stress. In the first model, I assume no gene–environment correlation, and thus the correlation between the genes related to stress and those related to depression (denoted rA_1A_2) is zero. This assumption matches the assumption of most sociological studies: Most studies do not identify genetic influences, and factors relegated to the residual are assumed to be uncorrelated with the independent variables. In this model, I regress depression on the stress source and, as a control variable, age. In the second model, I relax the assumption of no gene–environment correlation. Everything else in the model remains the same. A key question is whether the coefficient for stress changes between the first and second specifications, given that the second allows for gene–environment correlations. I present the regression coefficients,

the estimated gene–environment correlations, and model chi-squares. Because model 1 is nested in model 2 (differing only by one degree of freedom), I present chi-square tests comparing the two specifications. Readers can interpret the coefficients as linear regression coefficients in the case of depressive symptoms, and linear probability coefficients in the case of major depression. I fit these models using M-Plus software.

DATA

Data are from The National Survey of Midlife Development in the United States (MIDUS) (Brim et al. 2007). MIDUS data consist of multiple samples. A sample of twins is derived from a main sample of unrelated persons. The main sample is a nationally representative sample of noninstitutionalized, English-speaking adults aged 25 to 74. Twins were identified within this sample directly or within the main sample respondent's immediate family, and given the infrequency of twinning, some additional households were screened specifically for twins. Twin respondents were asked a series of questions to evaluate their zygosity, including questions on shared physical characteristics and whether other people found them difficult to distinguish. Although apparent similarity is not a perfect indicator of zygosity, studies reveal a high concordance between zygosity reports based on physical traits and those based on more sophisticated molecular tests (Chen et al. 1999). In order to increase statistical power, I use same-sex and opposite-sex DZ twins, adjusting for sex differences using a mean offset. The final sample consists of 634 MZ twins and 964 DZ twins. The main and twin samples were similar with respect to this study's key variables: The twin sample is slightly younger (45.4 versus 47.1), slightly less depressed (12.4% prevalence of major depression versus 13.8%, and .498 for depressive symptoms versus .571), and has somewhat more females (56% versus 52%), but none of these differences is statistically significant.

Depressive Symptoms and Major Depression

MIDUS uses two approaches to assess depression. *Depressive symptoms* were evaluated using six symptom-based items, culled from assorted psychometric sources. Respondents were asked, "During the past 30 days, how much of the time did you feel: so sad nothing could cheer you up; nervous, restless, or fidgety; hopeless; that everything was an effort; and worthless?" These items have been employed in other studies concerned with depression (Kessler, Mickelson, and Williams 1999). Response categories ranged from "none of the time" (coded 0) to "all of the time" (4), and respondents were assigned their mean score across the five items. Exploratory factor analysis revealed only one meaningful dimension for the items and, consistent with this, the Chronbach's alpha for the scale was high. Alphas for the scales are presented along with other descriptive statistics in the Appendix.

MIDUS also assessed the presence of clinically significant depression. *Major depression* is based on criteria established in the Diagnostic and Statistical Manual (Version III-R) of the American Psychiatric Association and the World Health Organization's International Classification of Disease (Kessler et al. 1998). According to these criteria, major depression requires a period of at least two weeks of either anhedonia or depressed mood most of the day, nearly every day. In addition, it requires at least four other associated symptoms during the same period, including suicidal thoughts or actions, problems with eating, sleeping, energy, or concentration, and feelings of self-worth. MIDUS data contain items adapted from the Composite International Diagnostic Inventory Short Form (CIDI), which asks symptom-specific questions sufficient to derive a diagnosis of major depression based on diagnostic criteria. Respondents who report symptoms sufficient to reach the clinical criteria for major depression need not have been diagnosed by a psychiatrist, although methodological studies have found good clinical validity for the CIDI (Blazer et al. 1994). The instrument included in MIDUS assesses current (or 12-month) major depression, although, for the reasons outlined above, current major depression often reflects the recurrence of an earlier disorder.

Stress Measures

I explore four sources of stress in this study: health stress, work/financial stress, network/family stress, and discrimination stress.

Health. Respondents were asked about the presence of assorted chronic conditions, as well as any disability resulting from health problems. The measure of chronic conditions corresponds to the sum of ten common conditions, including asthma or other lung problems; arthritis and other bone

diseases; recurring backache; allergies; stomach problems; bladder problems; foot problems; hypertension; migraine headaches; and ulcer. I selected items from a longer list based on exploratory factor analysis; this analysis revealed a single meaningful factor wherein a condition's loading was roughly proportional to its prevalence. Although ten potential conditions were included in the sum, few respondents reported more than three, and no one reported more than nine.

Respondents were asked six questions regarding *activities of daily living*. They were asked, "How much does your health limit you in: lifting or carrying groceries; bathing or dressing yourself; climbing several flights of stairs; bending, kneeling, or stooping; walking one block; and moderate activities, like bowling or vacuuming." Response categories ranged from "not at all" (coded 0) to "a lot" (3). Once again, I assigned respondents their mean response, given the high average inter-item correlation, reflected in the high alpha (.89).

Work and finances. Respondents were asked if they were ever unemployed in the preceding 12 months. Although they were asked to report the number of weeks in which they were unemployed, supplementary analyses revealed a cut-point with respect to depression: *Any* experience with unemployment was distressing. Given this result, unemployment was coded as a dummy variable. Respondents were also asked about *financial strain*. Respondents were asked, "How difficult is it for you (and your family) to pay your monthly bills?" Response categories ranged from "not at all difficult" (coded 0) to "very difficult" (3). This source of stress consists of only one item, which makes it empirically weak relative to others, but it has an unusually strong relationship with depression. In general, the reliability of measurement, while high for most measures, was not strongly associated with statistical significance.

Network/family stress. A series of questions were asked about network-related stress, including ongoing marital stress, events occurring to a spouse or child, and the perceived safety of the neighborhood in which the respondent lives.

Marital disagreement was assessed using the three questions: "How much do you and your spouse or partner disagree on the following issues?" with stem questions for (1) money matters, (2) household tasks, and (3) leisure time activities. Response categories ranged from "not at all" (coded 0) to "a lot" (3), from which respondents were assigned their mean response. When appropriate, respondents were asked about any events occurring within the last 12 months to their spouse or children. For both categories, respondents were asked about ten problems: chronic disease or disability; frequent minor illnesses; emotional problems; alcohol or substance abuse; financial problems; problems at school or work; difficulty finding or keeping a job; marital or partner problems; legal problems; and difficulty getting along with people. *Spouse events* and *child events* represent the sum total of these items for each category. Models that include these items were estimated only for twin pairs wherein both twins had a spouse or child.

Beyond family and friends, neighborhoods contribute significantly to psychological well-being. Two items assessed *perceived neighborhood safety*: (1) "I feel safe being out alone in my neighborhood during the daytime" and (2) "I feel safe being out alone in my neighborhood at night." For both items, response categories ranged from "not at all" (coded 0) to "a lot" (3), and as before, respondents were assigned their mean.

Discrimination. MIDUS discrimination questions were explicitly designed to assess unfair treatment on any basis, rather than discrimination based on a specific source, such as race. Two types of questions were asked. The first asked about any *lifetime* experiences with discrimination and proffered eleven events, including discrimination with respect to education (e.g., denied a scholarship or discouraged from seeking higher education), employment (e.g., fired or not given a promotion), criminal justice (e.g., hassled by the police), and services (e.g., given inferior medical care). Because many of these events are uncommon and, in any case, have effects on depression that are similar regardless of prevalence (Kessler et al. 1999), the models employ a count of the number of lifetime events. The second type of question asked about *chronic* or-day-to-day discrimination. Respondents were asked about nine types of ongoing discrimination: (1) "you are treated with less courtesy than other people"; (2) "you are treated with less respect"; (3) "you receive poorer service"; (4) "people act as if you are not smart"; (5) "people act as if they are afraid of you"; (6) "people act as if they think you are dishonest"; (7) "people act as if they think you are not as good as they are"; (8) "you are called names or insulted"; and (9) "you are threatened or harassed." Response categories ranged from "never" (coded 0) to "often" (3), and respondents were assigned their mean response. Although some studies assign a cut-point to these items, after which respondents are thought to

Table 1. ACE Model for Key Variables

Variable	h^2	c^2	e^2
Depression			
Depressive Symptoms	.288	.083	.629
	(.124)	(.098)	(.045)
Major Depression	.221	.048	.730
	(.133)	(.102)	(.050)
Health and Disability			
Chronic Conditions	.340	.031	.630
	(.127)	(.096)	(.049)
Activities of Daily Living	.252	.055	.693
	(.143)	(.098)	(.061)
Employment			
Not Working	.086	.000	.914
	(.051)	(.000)	(.051)
Financial Strain	.259	.000	.741
	(.045)	(.001)	(.045)
Network and Relationships			
Marital Disagreement	.090	.000	.910
	(.057)	(.000)	(.057)
Spouse Events	.000	.168	.832
	(.000)	(.042)	(.042)
Child Events	.249	.061	.691
	(.061)	(.119)	(.057)
Neighborhood and Discrimination			
Perceived Neighborhood Safety	.200	.000	.800
	(.049)	(.000)	(.049)
Lifetime Discrimination	.255	.000	.745
	(.048)	(.000)	(.048)
Chronic Discrimination	.366	.015	.619
	(.127)	(.099)	(.047)

Note: All quantities are based on univariate ACE models.

experience regular discrimination, I maintained the continuity of the index in order to conserve statistical power. These items, too, showed high unidimensionality.

RESULTS

Univariate ACE Models

Table 1 presents results from the univariate ACE models for each of the variables used in the analysis, beginning with depressive symptoms and major depression. Although based on the ACE pathways described above, the quantities presented in the table sum to zero, reflecting the percentage of the total variance explained by additive genetic influences (h^2), common environments shared by twins (c^2), and unique environments (e^2). Both depressive symptoms and major depression show moderate heritability, at .29 and .22, respectively. This means that, of the variation in depression found in MIDUS, 29 percent and 22 percent is due to variation in genes. These figures are somewhat lower than the heritabilities found in previous studies, although they are certainly not outside the typical range (see Sullivan et al. 2000 for a meta-analysis). Previous studies generally find a heritability of major depression around 37 percent, but the standard errors in most studies are quite large.

Most of the stress measures show moderate heritability, although there is substantial variation between them. The influence of common environments is in many cases zero or near zero, which is consistent with previous studies (Turkheim 2000), meaning that the experiences twins have in common, including families or neighborhoods, explain very little of the variation in stress. To the extent that twins are similar, they are similar because of

Table 2. Bivariate ACE Model Predicting Depressive Symptoms and Depressive Disorder Using Chronic Conditions and Activities of Daily Living, with and without Gene–Environment and Environment–Environment Correlations

	b		rA_1A_2		Model Chi-square			Model 2 vs. Model 1, *p*-value	
	Symptoms	Disorder	Symptoms	Disorder	Symptoms	Disorder	df	Symptoms	Disorder
Chronic Conditions									
Model 1	.114***	.032***			198.095	186.228	96		
	(.010)	(.006)							
Model 2	.071***	–.006	.468	.658**	191.543	172.153	95	.010	.000
	(.019)	(.011)	(.244)	(.269)					
Activities of Daily Living									
Model 1	.302***	.073***			265.754	239.309	96		
	(.025)	(.014)							
Model 2	.174***	.018	.505*	.414	254.845	233.842	95	.001	.019
	(.046)	(.027)	(.215)	(.229)					

*$p < .05$; ** $p < .01$; *** $p < .001$ (standard errors in parentheses).

common genes rather than common environments. Also consistent with a straightforward genetic story, the heritability of events occurring to one's child (or children) is higher (25%) than the heritability of events occurring to one's spouse (0%). Yet even many events removed from reproduction demonstrate moderate heritability. Chronic and lifetime discrimination, for example, have heritabilities over 25 percent. The heritability of chronic discrimination is, in fact, on par with the heritability of chronic health problems (over 30% for each). Even classically environmental measures show moderate heritability, including neighborhood safety (20%). The most common source of variance for all the measures is unique environments, which is what one would expect if one believes that stress is, above all, a reflection of one's contemporaneous environment. But this fact alone does not imply that genetic influences are irrelevant to the stress–depression relationship. The key test lies in the bivariate results.

Bivariate ACE Models

The bivariate ACE models reveal several key facets of the stress–depression relationship: (1) the stress–depression relationship is generally robust to gene–environment correlations; (2) the relationship between stress and major depression is more sensitive to gene–environment correlations than is the relationship between stress and depressive symptoms; and (3) the heritability of a stress measure bears little relationship to its sensitivity to gene–environment correlations.

Tables 2 through 5 present a number of quantities, the most important of which are those presented in the first two columns. This set of columns corresponds to the effects of stress on depressive symptoms (the first column) and depressive disorder (the second column). The next set presents the correlation between the A component for the source of stress and the A component for depression (and, thus, the gene–environment correlation) for depressive symptoms (again, the first column) and major depression (the second). The next set of columns presents model chi-square tests, comparing the second and first models, while the final set of columns presents the difference in these chi-squares, thereby testing whether models that allow for gene–environment correlations (model 2) provide a significantly better fit than models that do not (model 1). Because the total influence of the gene–environment correlation reflects the effects of genes on each phenotype (the A pathways) and not just the gene–environment correlation, the significance of the gene–environment correlation is best evaluated using both the model chi-square tests and the gene–environment correlations themselves.

Table 2 begins with health-related stress. The patterns shown here are similar to those presented in the remaining tables, although genes are especially powerful in this case. With no gene–environment correlation, model 1 reveals that chronic conditions are strongly related to both depressive

Table 3. Bivariate ACE Model Predicting Depressive Symptoms and Depressive Disorder Using Employment Conditions, with and without Gene–Environment Correlations

	b		rA_1A_2		Model Chi-square			Model 2 vs. Model 1, *p*-value	
	Symptoms	Disorder	Symptoms	Disorder	Symptoms	Disorder	df	Symptoms	Disorder
Not Working									
Model 1	.146**	.109***			185.523	199.699	96		
	(.054)	(.030)							
Model 2	.164	.047	.063	.434	185.452	197.311	95	.790	.122
	(.086)	(.050)	(.234)	(.325)					
Financial Strain									
Model 1	.151***	.045***			175.956	173.072	96		
	(.017)	(.010)							
Model 2	.085**	.007	.384**	.416*	168.139	165.673	95	.005	.007
	(.029)	(.017)	(.155)	(.179)					

*$p < .05$; ** $p < .01$; *** $p < .001$ (standard errors in parentheses).

symptoms ($b = .114$) and major depression ($b = .032$). Because the outcomes are modeled linearly, the interpretation of the coefficients is straightforward: Each additional chronic condition increases the probability of major depression by .032 and increases depressive symptoms by .114 units (nearly one-fifth of a standard deviation). Allowing for a gene–environment correlation, however, reduces this association, meaning that the relationship between chronic conditions and depression is partly determined by genetic influences they share. In a result that will be found across most of the remaining model, the gene–environment correlation is stronger for major depression than it is for depressive symptoms. The correlation is .468 for depressive symptoms and .658 for depressive disorder. These correlations eliminate the statistically significant association between chronic conditions and major depression, while reducing the association between chronic conditions and depressive symptoms by 38 percent. For both outcomes, model 2 provides a significantly improved fit over model 1, even if the estimated gene–environment correlation is insignificant for depressive symptoms.

This pattern is also apparent with respect to activities of daily living. Allowing for a gene–environment correlation reduces the effects of activities of daily living on depressive symptoms by 42 percent, from .302 to .174, although the coefficient remains significant. In the case of major depression, however, the coefficient is reduced by 75 percent and, as before, to statistical insignificance. Also as before, the model that allows for gene–environment correlations fits better than the model that does not, meaning that gene–environment correlations are an essential component to understanding gene–environment relations.

Unemployment is among the most popular subjects of stress research, and analysts remain concerned with estimating its effects appropriately (Burgard, Brand, and House 2007). Table 3 explores the relationship between unemployment and depression, as well as between financial strain and depression. Relative to the other sources of stress considered here, gene–environment correlations play a smaller role in the relationship between unemployment and depressive symptoms. In this sense, a genetically-sensitive design coheres with much of the existing sociological literature. The coefficient changes little between models 1 and 2 (and, in fact, increases slightly). For major depression, however, the coefficient is reduced by more than half and to statistical insignificance. Although model 2 does not provide a significantly better fit than model 1, the gene–environment correlation for major depression is much larger than that for depressive symptoms. The results for financial strain are more definitive. The coefficient for financial strain declines from .151 to .085, although it remains significant despite the significant gene–environment correlation (.384). For major depression, however, the coefficient is reduced to statistical insignificance. In both cases, model 2 fits better than model 1.

Table 4 turns to network and family stressors. Because the influence of genes on spouse events is

Table 4. Bivariate ACE Model Predicting Depressive Symptoms and Depressive Disorder Using Family Life Characteristics, with and without Gene–Environment Correlations

	b		rA_1A_2		Model Chi-square			Model 2 vs. Model 1, *p*-value	
	Symptoms	Disorder	Symptoms	Disorder	Symptoms	Disorder	df	Symptoms	Disorder
Marital Disagreement									
Model 1	.166***	.025			149.611	131.629	96		
	(.022)	(.013)							
Model 2	.055	.002	.930**	.376	130.214	129.664	95	.000	.161
	(.033)	(.021)	(.343)	(.284)					
Spouse Events									
Model 1	.073***	.033***			215.809	206.704	96		
	(.012)	(.007)							
Child Events									
Model 1	.050***	.026***			171.480	198.441	96		
	(.010)	(.005)							
Model 2	.017	.021*	.347	.124	166.806	197.999	95	.031	.483
	(.018)	(.010)	(.184)	(.192)					

*$p < .05$; ** $p < .01$; *** $p < .001$ (standard errors in parentheses).

zero, I did not estimate gene–environment correlation models for spouse events, although, for purposes of comparison with other equations, the coefficients from model 1 are presented. Marital disagreement is a different matter. In model 1, marital disagreement has a strong relationship with depressive symptoms (.166), but this effect is reduced to statistical insignificance in model 2 because of the gene–environment correlation. This result raises an important consideration. Although the heritability of marital disagreement is relatively small (.09), the large gene–environment correlation suggests that the genetic influences behind marital discord overlap with those behind depression. In the case of major depression, the coefficient is also reduced to statistical insignificance (from .025 to .002), although the gene–environment correlation is less. In the case of childhood events, the gene–environment correlation is also stronger for depressive symptoms than major depression, reducing the coefficient to insignificance for the former but not the latter.

Table 5 explores the relationship between perceived neighborhood dangerousness and depression and between discrimination and depression. It, too, illustrates the potential discontinuity between univariate heritability and bivariate quantities. Recall that depression, dangerousness, and discrimination all three have relatively strong heritabilities, especially chronic discrimination. Yet allowing for gene–environment correlations does little to diminish the effects of perceived neighborhood dangerousness, lifetime discrimination, or chronic discrimination on depressive symptoms. For lifetime discrimination, the coefficient is reduced to statistical insignificance, but the magnitude of the reduction is small and the model that allows for gene–environment correlations does not fit significantly better than a model that does not allow for gene–environment correlations. The gene–environment correlation for perceived neighborhood dangerousness is among the smallest in the study. The gene–environment correlations are much stronger with respect to major depression, and in each case the effect on major depression is reduced to statistical significance.

DISCUSSION

Although the relationship between stress and depression is well established, there has been renewed skepticism surrounding the notion that stress causes depression, especially in the context of evidence for the pervasive influence of genes. The twin design is well-suited to addressing this concern, as multivariate twin models allow the analyst to estimate gene–environment correlations directly. Although a good deal of attention has focused on the (mostly) descriptive question of univariate heritability, the influence of genes in a

Table 5. Bivariate ACE Model Predicting Depressive Symptoms and Depressive Disorder Using Neighborhood Perceptions and Discrimination, with and without Gene–Environment and Environment–Environment Correlations

	b		rA_1A_2		Model Chi-square			Model 2 vs. Model 1, *p*-value	
	Symptoms	Disorder	Symptoms	Disorder	Symptoms	Disorder	df	Symptoms	Disorder
Perceived Neighborhood Dangerousness									
Model 1	.136***	.037**			205.010	191.978	96		
	(.026)	(.015)							
Model 2	.128**	.023	.037	.127	204.962	191.504	95	.827	.491
	(.044)	(.026)	(.169)	(.189)					
Lifetime Discrimination									
Model 1	.042***	.021***			220.310	231.680	96		
	(.010)	(.006)							
Model 2	.035	–.004	.075	.497*	220.091	224.190	95	.640	.006
	(.018)	(.011)	(.161)	(.223)					
Chronic Discrimination									
Model 1	.310***	.086***			208.818	185.946	96		
	(.030)	(.017)							
Model 2	.267***	.043	.120	.222	208.045	183.804	95	.379	.143
	(.057)	(.034)	(.141)	(.153)					

*$p < .05$; ** $p < .01$; *** $p < .001$ (standard errors in parentheses)

multivariate context is especially important for furthering sociological theory.

At a basic level, the results validate the effects of most forms of stress on depressive symptoms. Despite the pervasive influence of genes on assorted indicators of stress, the relationship between stress and depressive symptoms is generally robust to genetic influences. This is true across stressors in different domains, across stressors referring to different time frames, and across stressors occurring to the self and those occurring to others. The reductions observed in this study between models that do and do not allow for gene–environment correlations are generally similar to the reductions observed in previous studies that used genetically-insensitive designs but multiple control variables, suggesting that the set of control variables sociologists routinely use to account for co-determination may capture some of the most important genetic influences behind stress and depression (see, for example, Turner and Lloyd 1999:385).

In the same vein, the results help to combat the still-popular notion that heritability implies destiny, although in this case without questioning the basic assumptions of twin studies. Virtually all of the variables explored here show moderate heritability, but the degree of heritability is imperfectly related to the influence of gene–environment correlations in the stress–depression relationship. Some of the most heritable sources of stress have effects on depression that are largely invulnerable to gene–environment correlations (e.g., chronic conditions), while some of the least heritable sources of stress have effects that essentially disappear once gene–environment correlations are considered (e.g., marital disagreement). The influence of genes may be pervasive, but the complexity of genetic influence makes discontinuities of this sort the rule rather than the exception.

The analyses also speak to the importance of distinguishing major depression from depressive symptoms. The heritability of major depression differs only slightly from that of depressive symptoms, but gene–environment correlations are much more influential in the case of the former than the latter. Although it is tempting to interpret this in terms of genes trumping the environment, this result does not mean that the environment is less influential in the case of major depression than in the case of depressive symptoms. The result perhaps says less about the absolute influence of genes than it does about the life-course epidemiology of major depression. There are at least two possibilities. Following the logic of the kindling

hypothesis, it is possible that major depression is initiated by social stress, but that it becomes progressively less associated with environmental influences with repeated episodes (Kendler et al. 2001). In this case, stress is relevant to major depression, but mostly with respect to the onset of depression rather than its recurrence and, therefore, operating before the point at which sociology has focused much of its attention. On the other hand, it is possible that major depression originates almost entirely in genetic factors, meaning that much of the relationship between stress and depression is an artifact of genes that have kindled both. It is difficult to differentiate these two perspectives empirically, but, in either case, the results encourage sociologists to consider the life-course epidemiology of depression, especially in the context of genes.

There are several limitations to this study, some related to using twins. First, the analysis was concerned only with the direct additive effects of stress on depression. This simple bivariate relationship is a building block of stress research, but there are more complicated and contingent specifications, especially surrounding coping. Alternative specifications would help to further pin down the influence of genes, but it is important to note that complexity of this sort does not of necessity provide a window for more environmental (or genetic) influences. Indeed, genetic influences are perhaps just as relevant for coping with stress as they are for exposure to stress (Kendler 1997). Similarly, while this study uses an assortment of stress measures, there are certainly others, and the particularity of patterns can reveal important features of gene–environment relationships.

At the same time, this study quantifies general genetic influences and hints at pleiotropy (i.e., the same genes having effects on multiple phenotypes), but it does not speak to which genes, in particular, are influential. Having established the heritability of depression, psychiatric research has moved in an increasingly molecular direction, attempting to identify which specific polymorphisms are relevant for stress and coping (Caspi et al. 2003). Investigations of this sort are path-breaking and garner a good deal of attention, but they tend to suffer from poor replication and may not serve sociology especially well. Because the specific genes implicated in environmental exposure are not well understood (and certainly not exhaustively known), it is perhaps more useful for sociology to quantify the general influence of genes now, rather than wait for better molecular information later on. Indeed, sociologists seem to be interested in general genetic influences even when they focus on specific polymorphisms. For example, Pescosolido and colleagues (2008) explore the specific relationship between the GABRA2 gene and clinically significant alcohol problems, but they end their study with a discussion of the general influence of genes on stressful situations, concluding in light of their results that gene–environment correlations may be irrelevant (p. S192). Elisions between the particular and the general are perhaps to be expected, given the novelty of genetic research in sociology, but if sociologists are serious about rethinking social pathways to illness in light of genes, then the category comprising "genetics" must be given its full empirical due. By the same token, some sociologists have been critical of the basic assumptions of twin studies, especially (and often only) the equal environments assumption (Horwitz et al. 2003), but it is important to recognize that twin studies can be deployed in multiple ways toward various ends, including demonstrating the influence of the environment, as was done here. Given the interdisciplinary nature of research on depression, sociology will be better served thinking about which gene–environment correlations matter most, rather than the narrow question of whether genes matter at all.

In the end, addressing the role of genes in social processes will help to advance medical sociology's epidemiological enterprise, and there is little to suggest that the discipline's core concepts have no utility in a genetic framework. Indeed, the serious consideration of genetic influences might sharpen the discipline's theoretical apparatus insofar as it points to specific areas where genes do or do not matter with respect to the environment. This can, in turn, reveal new facets of the environment not readily apparent in genetic-insensitive research designs. In the end, the concepts used in the genetic study of depression are of a piece with the concepts routinely used in sociology. The present study demonstrates that genetic influences are not an overwhelming threat to sociology's first-order claims regarding the relationship between stress and depression, but it does argue for a more complex approach focused on a consideration of the life-course.

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Appendix. Descriptive Statistics

Variable	Sample Mean	Standard Deviation	Range	Chronbach's Alpha
Depression				
Depressive Symptoms	.498	.603	0–4	.87
Major Depression	.124		0,1	
Health				
Chronic Conditions	1.318	1.484	0–9	
Activities of Daily Living	.316	.582	0–3	.89
Employment				
Not Working	.078		0,1	
Financial Strain	1.018	.858	0–3	
Network and Relationships				
Marital Disagreement	1.071	.736	0–3	.72
Spouse Events	.950	1.415	0–9	
Child Events	1.265	1.715	0–10	
Neighborhood and Discrimination				
Perceived Neighborhood Safety	.407	.575	0–3	.62
Lifetime Discrimination	.377	.491	0–3	.72
Chronic Discrimination	.787	1.439	0–9	.91

NOTE

1. A common critique of twin studies is that MZ twins share more similar environments than DZ twins, thereby artificially inflating heritability. Although there is some evidence that the equal environments assumption is violated for some features of the environment, the assumption is phenotype-specific, and there is little evidence that violations of the assumption are related to depression in particular (Hettema, Neale, and Kendler 1995; Kendler and Gardner 1998; Kendler et al. 1993; McGuffin et al. 1996; Xian et al. 2000).

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