Socioeconomic Status, Genetic Risk Factors, and Psychological Distress: Exploring Independent, Correlated, and Interactive Effects

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ABSTRACT

Research shows that both socioeconomic status (SES) and genetic risk factors are associated with psychological distress. To date, however, very little research has explored (a) whether, and (b) how, these two predictors might be interconnected. The present study addresses this shortcoming by formulating, and then empirically testing, a series of models designed to clarify how SES and genetic risk factors might function to influence psychological distress. The models draw on twin sibling data from the National Survey of Midlife Development in the United States (MIDUS), and results show (a) that both SES (inversely) and genetic risk (positively) are significantly associated with levels of psychological distress in bivariate models, (b) that the relationship between SES and distress is explained, to a considerable degree, by genetic risk factors, (c) that the correlation between genetic risk factors and distress is stronger at low levels of SES, and (d) that the association between SES and distress exists primarily among individuals who are at high genetic risk. The implications of these findings for sociological inquiry are discussed, and an agenda for future research is outlined.

KEYWORDS

Mental Health, Social Status, Life Stress Paradigm, Confounding, Behavior Genetics, MIDUS

INTRODUCTION

On one front, an enormous body of sociological research documents that socioeconomic status (SES)—e.g., educational attainment, monetary income, etc.—is inversely associated with psychological distress and other forms of poor mental health (House 2002; Kessler 1982, 1979; Miech and Shanahan 2000; Mirowsky and Ross 2003). On another front, psychologists, psychiatrists, biologists, and medical doctors are amassing evidence that genetic risk factors also predict level of distress (Blehar et al. 1988; Caspi et al. 2003; Hamann 2005; Lesch et al. 1996; Pezawas et al. 2005; Plomin and Rende 1991). Importantly, there is growing consensus that mental health is the product of a complex interplay between environmental influences such as SES, and biological ones including genetic differences (Rutter, Moffitt, and Caspi 2006).

To date, however, very few attempts have been made to theorize and empirically examine the various ways in which SES and genetic risk factors might work, either independently or interconnectedly, to influence psychological distress. Thus, several elemental questions remain unanswered, including: Does SES affect distress net of genetic risk factors? Are the effects of SES and genetic factors additive? Do genetic factors confound (i.e., explain) the phenotypic relationship between SES and psychological distress? Is SES a mediator of genetic effects on distress—i.e., is there a correlation between these two predictors of distress? Does SES moderate genetic effects on distress, or does the influence of SES on distress vary across genetic differences? The inability to answer questions such as these represents a sizable gap in our knowledge, and given the current state of the literature, it is unclear—despite their obvious importance—how genetic factors might be integrated into sociological research on psychological distress.

The present study addresses each of these questions and issues by initially formulating five conceptual models of the relationships between SES, genetic risk factors, and psychological distress: (1) SES-only effects; (2) genetic-only effects; (3) independent SES and genetic effects; (4) correlated SES and genetic effects; and (5a) moderation of genetic effects by SES, and (5b) moderation of SES effects by genetic risk. These models are then empirically evaluated using data from the National Survey of Midlife Development in the United States (MIDUS), a national sample of working-age monozygotic and dizygotic twin siblings. Summarizing the results below, the analyses provide statistical evidence that SES and genetic risk factors are not completely independent of one another, but are instead correlated with each other to a considerable degree. Further, SES and genetic factors appear to interactively influence psychological distress, with (a) the association between genetic risk factors and psychological distress being greater among persons with low levels of education and income, and (b) the link between both education and income, and distress, being strongest among persons who are at high genetic risk for distress. Overall, it is concluded that integrating genetic factors into sociological models of psychological distress leads to a deeper understanding than is otherwise possible, and the present study serves as a preliminary roadmap for how to achieve this goal.

[Figure 1 About Here]

THEORETICAL AND EMPIRICAL BACKGROUND

As reviewed in detail below, the literature shows that both SES and genetic risk factors are associated with levels of psychological distress. The various ways in which these two predictors might influence distress, however, have yet to be systematically investigated. The present study addresses this shortcoming in the literature by formulating, and then empirically testing, three

conceptual models of gene-environment influences: independent, correlated, and interactive effects.

Independent Effects

Socioeconomic status affects psychological distress and other mental health outcomes primarily by shaping (a) exposure to social stressors (i.e., chronic and acute conditions that tax individual capacities to respond), and (b) different degrees of vulnerability to those stressors (i.e., the quantity and quality of available resources with which individuals can deal with their problems) (Kristenson et al. 2004; Weinstein et al. 2003). Much of the research in this area has been, or could have been, framed from the perspective of the life stress paradigm (Ellison 1994; Ellison et al. 2001; Ensel and Lin 1991), which, among other things, conceives of social resources such as high SES as potential buffers against stressors that lead to high levels of psychological distress (Glenn and Weaver 1981; Miech et al. 1999). For example, persons with more socioeconomic resources are less prone to face difficulties in meeting personal and family needs, paying bills, and obtaining mental and physical health care, as well as lower risk of legal, interpersonal, familial, and other types of stressors (Pearlin et al. 1981; Ross and Van Willigen 1997). At the same time, these individuals often enjoy larger and more diverse social networks from which to obtain emotional, tangible, and informational assistance that could help them to resolve problems and manage the emotional and physical consequences of their difficulties (Cohen 2004; Lin et al. 1999). In addition, persons with substantial socioeconomic resources typically benefit from a richer array of psychological and cognitive resources such as feelings of personal control, efficacy, and self-worth, which can facilitate successful coping and resilience in the face of stressful life events (Mirowsky and Ross 2003). Persons lacking socioeconomic resources, in

contrast, typically have deficits in all of these areas. This relationship is summarized by Model 1 (Pathway A) in Figure 1.

Although sociological research rarely addresses them, genetic risk factors appear to be important predictors of psychological distress as well. For example, twin and adoption studies suggest that genetic influences may account for a considerable proportion (30-70%) of the individual-level variation on affective disorders such as depression, which are characterized by feelings of worthlessness, sadness, and hopelessness, as well as sleep and appetite irregularities (Eley 1997; Kendler 2001; Plomin 1990; Plomin and Rende 1991; Sullivan et al. 2000). Similarly, researchers have found that panic and anxiety disorders are substantially heritable (Crowe et al. 1983; Finn and Smoller 2001), and it is now firmly established that schizophrenia, a severe mental pathology, is largely the product of genetic factors (Gottesman 1991). There is even evidence showing that personality traits with clear linkages to psychological distress—e.g., neuroticism, psychoticism, negative emotionality, aggression-hostility, harm avoidance, etc.—are influenced by genetic differences (Bouchard and Loehlin 2001). In addition to evidence from family-based studies such as these, which provide heritability estimates, molecular genetic research shows that carriers of the short allele of the functional 5' promoter polymorphism of the serotonin transporter gene suffer from increased levels of depression and anxiety compared with individuals who inherited the long allele (Hamann 2005; Lesch et al, 1996; Pezawas et al. 2005). Other specific genetic differences, many of them polymorphisms of genes related to serotonin and dopamine, have been linked with mental health as well (Deckert et al. 1999). This association is shown as Pathway B in Model 2 (Figure 1).

Despite widespread endorsement and use—typically in different disciplines—these SES-only and genetic-only effects models are oversimplifications of the actual causes of psychological

distress, especially when considered separately. Further, given the various issues involved with examining these two influences in isolation—e.g., explanatory power deficiencies, biased empirical findings, erroneous conclusions, etc. (Bouchard and Loehlin 2001; Caspi et al. 2005a; D'Onofrio et al. 2006; Gottfredson 2003; Lichtenstein et al. 1992; Neiderhiser et al. 1999; Reiss et al. 2000; Udry 1995)—it is important that researchers begin to analyze both of these factors simultaneously, especially since their effects on psychological distress could be either (a) independent; or (b) interconnected (i.e., correlated or interactive).

Model 3 of Figure 1 shows an independent SES and genetic effects model, which simply combines Models 1 and 2 into a single explanatory framework so that gene-environment independence, which was implicitly assumed by all of the studies reviewed above, can actually be examined. If (a) the logic of the current sociological and biomedical paradigms is correct, and (b) the compartmentalization of researchers into distinct environmental and genetic camps is not problematic, we would expect SES and genetic risk factors to influence psychological distress net of the other, and that their effects will be largely additive, and thus will not be substantially altered by the simultaneous examination of both (see Model 3, Pathways A and B).

There is good reason, however, to believe that the effects of SES and genetic risk factors on mental health are not independent of one another, at least not completely, but that are instead likely to be interconnected in some way (D'Onofrio et al. 2006; Johnson and Kreuger 2005a; Lichtenstein et al. 1992; Neiderhiser et al. 1999; Reiss et al. 2000; Shanahan and Hofer 2005). In the words of Rutter, Moffitt, and Caspi (2006: 244), with reference to psychopathology in general: "The traditional notion that strictly additive, non-interactive, effects for genetic and environmental influences would constitute the norm must now be rejected." If their conclusion is correct, the possible interrelations between these two predictors of psychological distress need to

be examined, and these interconnections come in two basic forms: correlated and interactive effects.

Correlated Effects

The most basic manner by which SES and genetic factors might be interconnected is simply by explaining some of the exact same variation (i.e., by having at least some overlap in their explanatory power). If this is true, simultaneously examining both of them will reduce the effect of one or the other (or perhaps both) on psychological distress. This would provide evidence for non-independent, interconnected effects. Such a possibility implies the scenario shown in Model 4 of Figure 1, which competes with Model 3. If this model is correct, we would expect both SES and genetic risk factors to influence psychological distress, but the effects of one or the other, or perhaps both, will be substantially altered by the simultaneous examination of both—i.e., their effects will not be completely independent, but will instead be correlated (Model 4, Pathways A, B, and C).

One of the primary reasons to expect correlated effects for these two predictors is a growing body of evidence showing that—in addition to mental health outcomes—genetic factors also influence environmental variables such as SES. For example, research has found sizable genetic effects (possibly accounting for as much as 50% of the variation) on educational attainment (Behrman et al. 1980; Heath et al. 1985; Tambs et al. 1989; Vogler and Fulker 1983). Similarly, a recent study found significant genetic influences on both grade-point average and college plans (aspirations) among adolescents and young adults (Nielsen 2006). There is even evidence that one's monetary income is substantially influenced by genetic factors, and that the genetic predispositions underlying education and income are partially, although not completely, the same

(Rowe et al. 1998). These findings suggest, therefore, that genetic tendencies toward particular levels of SES might be at least partially responsible for the observed correlation between this aspect of social life and levels of psychological distress, especially if latent genetic factors simultaneously influence both of these outcomes. (Note: Research on social selection effects is consistent with this possibility—e.g., Miech et al. 1999; Mulatu and Schooler 2002.)

This suggests an interesting potential relationship between SES, genetic risk factors, and psychological distress. Looking again at Model 4—a correlated (i.e., mediated / confounded) SES and genetic effects model—genetic factors may directly influence SES, which then indirectly affects psychological distress. Even though this model has yet to be tested specifically for SES and distress, empirical support does exist for several other outcomes. For example, the correlation between both marital quality and social support, and mental health, appears to be at least partially explained by common genetic factors—i.e., genetic effects on mental health may flow, in part, indirectly through marital quality and social support (Spotts et al. 2005). Similarly, genetic influences on physical health have been shown to be correlated with genetic effects on socioeconomic status (Johnson and Krueger 2005b; Lichtenstein et al. 1992). Perhaps the most powerful finding in this area, however, is one showing that genetic risk factors for depression are positively associated with the probability of experiencing stressful life events in the interpersonal and financial domains (Kendler and Karkowski-Shuman 1997). These studies, and others like them (Charles and Almeida 2007; McGue and Lykken 1992; Lyons et al. 1995), suggest that genes are at least partially responsible for the occurrence of deleterious social conditions (including low SES), which are, in turn, associated with poor mental health. Thus, based on Model 4, this possibility suggests that genetic influences on psychological distress are at least

partially indirect via their effects on SES (Pathway C), which then affects psychological distress (Pathway A).

This model also suggests that the relationship between SES and psychological distress may be at least partially the product of one or more exogenous third variables—e.g., genetic factors in the present study—that may lead to a biased and possibly even spurious correlation between them. A model nested within Model 4 suggests that the direct effect of SES on psychological distress (Pathway A) may be weak or absent when genetic factors are taken into consideration (Pathways B and C). In other words, latent genetic influences could be confounding the observed correlation between SES and distress. For example, we know that educational attainment is inversely associated with distress, but in contrast to the typical sociological interpretation—e.g., that high levels of education provide psychosocial resources that buffer against distress, or that low levels create stressful conditions that undermine mental health—it might also be the case that common genetic factors predispose individuals in some way toward (a) both high levels of education and low levels of distress, or (b) both low levels of education and high levels of distress.

Although there are no published studies of this type on SES and psychological distress, support for this model has been shown for other health outcomes (Neiderhiser et al. 1999; Reiss et al. 2000). For example, shared genetic liabilities appear to explain some, although certainly not all, of the correlation between socioeconomic status and chronic illnesses, body mass index, and self-reported health (Johnson and Krueger 2005b; Lichtenstein et al. 1992). The truth is, however, that due to a relative dearth of stringent empirical studies on this topic, we do not know whether, or to what extent, genetic confounding is occurring in sociological studies of SES and psychological distress. Given that researchers in various other disciplines have already argued

that it is an issue (see references above), sociologists need to begin articulating with this body of work. Moreover, it is in sociologists' best interest to do this. Showing that SES has an effect on psychological distress even when potential genetic confounders are taken into consideration will only serve to strengthen sociological arguments (e.g., Carlsson et al. 2007; Caspi et al. 2000, 2005a). Stated in terms of models shown in Figure 1, genetic risk factors potentially influence both SES (Model 4, Pathway C) and psychological distress (Pathway B), and when this effect is considered, the observed correlation between SES and distress (Pathway A) may be reduced, and possibly even eliminated—i.e., genetic factors may be at least partially confounding the phenotypic relationship between SES and psychological distress.

Interactive Effects

In addition to independent and correlated effects, there is one other primary way in which SES and genetic factors might work together to influence psychological distress: gene-environment "interaction." Although much work remains to be done in this area, there is growing evidence that (a) genetic effects on distress may be either more or less pronounced depending upon environmental influences such as SES (Model 5a of Figure 1), and (b) that environmental factors may have different effects based on the genetic makeup of individuals (Model 5b of Figure 1) (e.g., Boomsma et al. 1999; Caspi et al. 2003; Eley et al. 2004a and b; Jaffee et al. 2005; Jang et al. 2001; Kendler et al. 1995; Silberg et al. 2001; Shanahan and Hofer 2005).

With respect to the first type, theory and empirical research suggests (a) that desirable environments such as high SES may buffer against or impede genetic liabilities toward distress via the provision of psychosocial resources, and / or (b) that low SES may trigger or facilitate genetic risks for psychological distress via a dearth of social support and psychological coping

mechanisms. This line of thought is premised on the fact that genetic effects are not fixed blueprints that are destined to unfold, but are instead more accurately conceptualized as "predispositions" that are contingent on environmental conditions.

Although no studies to date have examined these possibilities for SES and distress, empirical evidence for other health outcomes does exist. For example, grounded in previous research showing (a) that chronic illnesses and body mass index (BMI) are both strongly influenced by genetic factors, and (b) that income is correlated with these same two outcomes as well, a recent study found that the association between genetic factors and both aspects of physical health are lower at higher levels of income (Johnson and Krueger 2005a). This suggests that financial assets may provide individuals with psychosocial resources that can be used to buffer against genetic risks for poor physical health. These findings are broadly consistent with sociological theory (e.g., the life stress paradigm, social capital theory, etc.), which argues that individuals who possess adequate psychosocial resources (e.g., a high level of monetary income) may be better suited to deal with undesirable health outcomes, perhaps even if they are genetically predisposed toward them.

In addition to buffering against genetic risks, SES (when low) may also function to trigger or facilitate latent genetic vulnerabilities. Empirical research provides at least some support for this possibility as well by showing that genetic influences on a variety of health outcomes appear to be more influential when accompanied by environmental stressors such as negative life events, social adversity, parental maltreatment, a lack of social support, and unemployment, among others (Cadoret et al. 1995; Caspi et al. 2003; Eley et al. 2004a and b; Grabe et al. 2005; Jaffee et al. 2005; Kaufman et al. 2004; Kendler et al. 1995; Silberg et al. 2001). One of the best examples of gene-environment interaction of this type is research on PKU, a genetic disorder that leads to

mental retardation when triggered by certain dietary (i.e., environmental) habits (Omenn 2000; Shostak 2003). In terms of the models in Figure 1, the argument above suggests that SES moderates (Model 5a, Pathway D) the association between genetic risk factors and psychological distress (Pathway B).

Other research on gene-environment interaction has framed the associations in terms of the moderation of SES by genetic risk factors (Model 5b of Figure 1). Of course, this interpretation is empirically indistinguishable from those shown in Model 5a. Again, although SES and psychological have yet to be studied in this context, support for this possibility is present for other sociologically relevant predictors. For example, a recent study of depression investigated this issue by using information on zygosity and co-twin history of depression to define a gradient of genetic vulnerability toward this form of psychopathology (Kendler et al. 1995). Results showed that the relationship between stressful life events and depression was stronger among individuals at high genetic risk compared with those at low genetic risk. This means that genetic factors may affect individual differences in sensitivity to the depression-inducing effects of stressful life events—i.e., for individuals who are not predisposed toward depression, environmental stressors may not matter much, but for those who inherited innate tendencies toward depression, stressful social situations may be profoundly important. Similarly, recent molecular genetic work has shown that a functional polymorphism in the promoter region of the serotonin transporter (5-HTT) gene moderates the influence of stressful life events on depression (Caspi et al. 2003). Individuals with one or two copies of the 5-HTT short allele exhibited more depressive symptoms, diagnosable depression, and suicidality following stressful life events than individuals homozygous for the long allele. Still other research shows that the relationship between cannabis use and psychosis is moderated by differences in the catechol-O-

methyltransferase gene; in other words, cannabis use appears to be associated with the development of schizophrentiform disorder among individuals with one gene variant, but not others (Caspi et al. 2005b). Overall, then, genetic risk factors may moderate (Model 5b, Pathway E) the influence of SES on psychological distress (Pathway A).

METHODS

Data

Investigating the alternative ways in which socioeconomic status and genetic risk factors combine to influence psychological distress requires a genetically-informed design. The present study makes use of twin sibling data from the National Survey of Midlife Development in the United States (MIDUS), 1995-1996, which was obtained via the Inter-University Consortium for Political and Social Research (ICPSR) (Brim et al. [1996] 2003). The MIDUS study collected data on working-age (25-74) adults to examine the patterns, predictors, and consequences of midlife development in the areas of physical health, psychological well-being, and social responsibility, among others.

Two data collection agencies (ICR / AUS Consultants and Bruskin Associates) were hired by MIDUS personnel to recruit twin pairs by making telephone calls and asking respondents whether they, or any of their immediate family, were members of an intact twin pair. Roughly 50,000 households, constituting a representative sample of the United States, were screened in this manner. Respondents who reported the presence of a twin in the family (14.8%) were then asked if they would allow the research team to contact them again to solicit their participation in the survey. The 60% of the respondents who agreed were then referred to the MIDUS recruitment process. The twin pairs that ultimately participated in the MIDUS Twin Screening

Project represent the first national sample of twins ascertained randomly via the telephone (for additional information on the sample or sampling process see: Brim et al. [1996] 2003).

Twin pairs in the sample were diagnosed as monozygotic (MZ) versus dizygotic (DZ) with self-report data on whether they had the same eye color, natural hair color and complexion, whether individuals mistook them for each other when they were young, and whether they had ever undergone testing or been told by a doctor whether they were genetically identical or fraternal. Pairs were given a series of points for their answers to a number of specific questions and then the points were subsequently totaled. The point system was set up such that "high" scores indicated MZ twin pairs and "low" scores indicated DZ pairs. Similar methods of diagnosing zygosity have been shown to be over 90% accurate (see: Lykken et al. 1990; Maes et al. 1999). The scores for 26 pairs fell directly in the middle of the range, making a definitive classification impossible, and thus these pairs, along with all opposite-sex DZ twin siblings, are excluded from the present study.

Dependent Variable

Psychological distress, the aspect of mental health examined here, is gauged with the K6 scale (Kessler et al. 2002). Respondents were asked the following six questions (Chronbach's alpha = 0.87): "During the past 30 days, how much of the time did you feel… (a) so sad nothing could cheer you up; (b) nervous; (c) restless or fidgety; (d) hopeless; (e) that everything was an effort; and (f) worthless?" This index, which is constructed by taking the mean of each of the six items, distinguishes negative affect based on the type and amount of severity of the problem, rather than diagnosis (i.e., each question was asked of all respondents, not just the ones who preliminarily screened positive for a diagnosed mental illness such as major depression). This measure was

specifically designed for use in surveys such as the MIDUS, and it is excellent for broad-gauged screening of psychological distress, and can discriminate DSM-IV cases from non-cases. To reduce the skewed nature of this variable (skewness = 2.00), it is normalized via an inverse square-root transformation (transformed variable skewness = 0.65). Since this transformation reverses the ordering of the variable so that higher scores represent lower levels of distress, it is then multiplied by -1 to recover the desired direction of coding (i.e., so that higher scores represent more distress).

Independent Variables

Socioeconomic Status

SES, one of the two key independent variables examined here, is operationalized using two measures. Educational attainment is a five-category variable: (1) less than high school, (2) high school, (3) some college, (4) bachelor's degree, and (5) graduate degree. Preliminary analyses indicated that the effect of education on psychological distress can be captured equally well by either (a) treating this measure as a normally-distributed continuous outcome with a range of 1 to 5, or (b) coding it as a series of dummies where less than high school and high school are both compared to a reference category of some college or more. For ease of interpretation, and to be consistent with our other measure of SES—income, which is also continuous—the former version is employed in all of the analyses presented below. All substantive interpretations, however, would be the same if the dummy variable approach had been chosen instead. Personal income is a continuous measure ranging from 1-31, where 1 = less than zero (i.e., lost money) and 31 = $100,000 or more / year. To reduce the loss of a significant number of cases on the income variable, a mean imputation was performed on all missing cases. Additional analyses that

included a flag variable for the imputed cases were conducted, and it was found that this imputation was not statistically significant in any of them.

Genetic Risk

As one might expect, measuring genetic risk factors for psychological distress is not an easy task. Complex phenotypes such as psychological distress are almost certainly influenced by a whole host of different genes (perhaps hundreds or more), each of which exerts minimal effects on the expressed phenotype. Given (a) that we currently understand the functional utility of only a handful of specific genes, and (b) that many of the genes that are likely associated with psychological distress have yet to be identified, it is impossible at the present time to directly measure and empirically examine all of them. This does not mean, however, that genetic effects cannot be studied.

When data are collected on twin siblings, an individual's genetic risk for any outcome, including psychological distress, can be calculated as a function of his or her co-twin's level of distress accompanied by the pair's zygosity (Andrieu and Goldstein 1998; Jaffee et al. 2005; Kendler et al. 1995; Ottman 1994). (Note: These analyses are premised on the assumption that psychological distress is heritable, which was established by the numerous studies reviewed above, as well as the analyses that follow.) Because MZ twins share 100% of their genes, it can be inferred that an individual's genetic risk for psychological distress is high if that individual suffers from a high level of distress and his or her MZ co-twin also has a high level of distress, and low if his or her MZ co-twin does not suffer from high distress. (Note: A high level of psychological distress is defined here as a score in the top quartile of the distribution.) That is, if distress is genetically influenced and an individual and his or her MZ co-twin *both* experience a

high level of distress, then these individuals presumably share all of the susceptibility genes for distress since they are by definition genetically identical; thus, they are at high genetic risk. DZ twin siblings, in contrast, share only half their genes, on average. This means that if an individual who suffers from a high level of distress has a DZ co-twin who also has a high level of distress, that individual's genetic risk for distress is high, but not as high as it is for MZ twin siblings because these two individuals may or may not share the susceptibility genes for distress. Likewise, if an individual who has a high level of distress has a DZ co-twin that does not, that individual's genetic risk for distress is low, but not as low as it is for a MZ individual who has a high level of distress but who has a genetically identical sibling who does not because the DZ individual may have inherited susceptibility genes for distress while his or her co-twin did not since they are only 50% genetically identical on average.

Based on this information, genetic risk toward psychological distress can be constructed as a 4-category variable with a range of 0-3, where: 0 = very low genetic risk (MZ siblings where one twin has a high level of distress and the other does not, plus MZ siblings where neither twin has a high level of distress); 1 = low genetic risk (DZ siblings where one twin has a high level of distress and the other does not, plus DZ siblings where neither twin has a high level of distress); 2 = moderate genetic risk (DZ siblings where both twins have a high level of distress); and 3 = high genetic risk (MZ siblings where both twins have a high level of distress).

Covariates

Given that both psychological distress and socioeconomic status vary by *age* and *gender*, the present study controls for the effects of each of these influences. Age is measured in years (25-74), and gender is a dichotomous variable (female = 1).

Modeling Techniques

Since the outcome examined here is a continuous measure of psychological distress, ordinary least squares (OLS) regression is used to model the relationships between socioeconomic status, genetic risk, and this mental health outcome. A series of models (as outlined above) are fit to the data to explore the potential independent, correlated, and interactive effects. For the interactive relationships, all of the predictors are zero-centered prior to the construction of the cross-product terms between SES and genetic risk (Aiken and West 1991). Since the ordering of the twin pairs (i.e., as first or second) in the data employed here may not be random with respect to the dependent and independent variables (i.e., since this is not a selected sample), both twins of a pair are entered into the data twice: once as the first twin (i.e., the dependent variable), and once as the second twin (i.e., the independent variable). Although this procedure does not bias the parameter estimates, it does affect the standard errors. To take this non-independence of observations into account, the standard errors are adjusted to the correct degrees of freedom prior to the calculation of tests for statistical significance by multiplying them by an adjustment factor of 1.41 (i.e., $\sqrt{2}$) (see Kohler and Rodgers 2001).

[Table 1 About Here]

RESULTS

Table 1 shows descriptive statistics for all of the study variables. Psychological distress has a mean of 1.498 on a 1-5 scale (for the non-transformed version). The mean for the education variable is 2.882, which indicates some college. Personal income has a mean of 18.321 on a 1-31 scale, which is slightly more than $15,000 / year. The mean for genetic risk is 0.658 on a 0-3

scale, indicating that most respondents have a relatively low level of genetic risk toward psychological distress. Supplementary analyses indicate that 48.38% of the sample is at very low genetic risk, 42.25% are at low genetic risk, 4.60% are at moderate genetic risk, and 4.77% are at high genetic risk.

[Table 2 About Here]

Table 2 shows bivariate correlations between these variables. As expected, education is inversely associated with the transformed psychological distress measure ($r = -0.103$). Personal income is also inversely associated with this mental health outcome ($r = -0.111$), while genetic risk is positively correlated ($r = 0.428$). Interestingly, genetic risk factors for psychological distress are also significantly associated with both education ($r = -0.151$) and income ($r = -0.121$).

[Table 3 About Here]

To further examine these relationships, multivariate regression analyses are required. As shown in Model 1 of Table 3, both education ($b = -0.0103$, $p<0.05$) and income ($b = -0.0015$, $p<0.05$) are both inversely associated with psychological distress net of each other (as well as age and gender), indicating that both exert independent effects on levels of distress. These findings are consistent with the extensive sociological literature summarized earlier. Model 2 shows that genetic risk is also associated with psychological distress, with a one unit increase in genetic risk being associated with a 0.0716 unit increase in the transformed psychological distress measure. The magnitude of this effect is actually quite large, with predicted mean levels of distress being more than an entire unit (on a 1-5 scale) higher among individuals at high genetic risk (mean = 2.690 on the non-transformed variable) compared with those at low genetic

risk (mean = 1.365). This finding is consistent with a large biomedical literature on mental health.

Model 3 examines whether the influence of SES and genetic risk factors are independent of one another. One way to think about these associations is that the genetic risk for psychological distress is randomly distributed across levels of socioeconomic status, such that both SES and genetic risk are associated with psychological distress net of each other. The findings shown in Model 3 of Table 3, however, cast doubt on this widely-held assumption. The coefficient for education, which was -0.0103 in Model 1, is reduced to -0.0042 when genetic risk factors are introduced in Model 3. This amounts to a 59% decrease, although the change in the parameter estimate does not appear to be statistically significant. The findings for income also suggest at least some modest dependence between SES and genetic risk, with the coefficient being reduced from -0.0015 in Model 1 to -0.0011 in Model 3 (a 27% reduction). For genetic risk, the coefficient is reduced from 0.0716 to 0.0696: a reduction of 3%. Overall, these findings suggest that SES and genetic risk factors are not be completely independent of one another.

Unfortunately, the mediation of genetic effects via SES, and the confounding of the phenotypic relationship between SES and distress by genetic factors, respectively, cannot be sorted out here. That said, given that genetic risk factors for distress are correlated with both educational attainment and income, it is likely that both scenarios are occurring to some extent. Such a possibility was initially supported by the bivariate findings shown in Table 2, and subsequent analyses (not shown) indicate that genetic risk factors for distress are significantly lower (mean = 0.558) among individuals with some college or more compared with those with only a high school education or less (mean = 0.798; p-value for difference = 0.0001). One potential explanation for this is that genetic risks toward high levels of psychological distress are

functioning as selection effects that also lead to low levels of educational attainment. The findings for income are similar, although not as strong.

[Figure 2 About Here]

The results now examine whether SES moderates the influence of genetic risk factors on psychological distress. More specifically, the present study assesses whether high levels of education and / or income function—via psychological and social resource mobilization—to offset the deleterious effects of genetic risk factors on psychological distress. Among individuals with high levels of education or income, genetic risk factors should have less of an impact on distress. The findings shown in Models 4-6, as well as Figures 2a and 2b, which graphically summarize the statistical results shown in the table, suggest that there is statistical evidence for this hypothesis. As education increases, for example, the effect of genetic risk factors on distress decreases (i.e., the slope gets flatter) (Figure 2a). For income, we see a similar pattern (Model 5 and Figure 2b): at low levels of income, genetic risk factors are more strongly associated with psychological distress, but as income increases, these effects are substantially moderated to the point to where they are considerably less pronounced at very high levels of income.

Model 6 of Table 3 examines the buffering effects of education and income simultaneously, and the results suggest that education is more important than income (i.e., the interaction between income and genetic risk is no longer significant when the one with education is controlled). These results should be interpreted with caution, however, given the substantial multicollinearity that is likely involved in models containing all of these different main and interactive effects.

These findings, axiomatically, also offer support for the idea that genetic factors moderate the influence of SES on levels of distress. Looking at Figures 2c and 2d, the effects of both

education and income on psychological distress are stronger for individuals at high genetic risk. Essentially, among those who did not inherit a genetic susceptibility toward distress, the results suggest that education and income are not strongly associated with distress. This suggests that environmental buffering resources may only matter for a certain segment of the population—i.e., one that is at high genetic risk for psychological distress.

One last set of analyses is required to support these assertions. Empirically, gene-environment interaction is sometimes difficult to disentangle from gene-environment correlation. If zygosity differences exist in education or income, the results reported here could actually support the latter. Additional analyses (not shown) revealed that there are indeed zygosity differences in educational attainment, but not personal income. To address this issue, the models reported above were reanalyzed with zygosity regressed out of the education variable prior to the model fitting. These results are not substantially different from the ones reported here. Further, as shown in Table 2, genetic risks toward psychological distress are correlated with both education and income. This too could imply a form of gene-environment correlation, so in addition to regressing out zygosity differences, genetic risks were also regressed out of the education and income measures. Once again, the results reported above remained basically the same. Thus, we can be fairly confident that the findings described above support the idea that distress is associated with a true gene-environment interaction between socioeconomic resources and genetic risk factors.

DISCUSSION

To date, most research on the socioeconomic and genetic causes of psychological distress has been conducted by scholars in isolated camps, with little attention given to the potential

interconnections between these two predictors. This is unfortunate given that evidence suggests that sociological outcomes such as SES, and biological ones including genetic risk factors, work together to influence a host of health outcomes (Lichtenstein et al. 1992; Reiss et al. 2000; Rutter, Moffitt, and Caspi 2006; Shanahan and Hofer 2005). The present study extends our knowledge on this front by examining six potential relationships between SES, genetic risk factors, and psychological distress: SES-only effects, genetic-only effects, independent SES and genetic effects, correlated SES and genetic effects, moderation of genetic effects by SES, and moderation of SES effects by genetic risk.

Results revealed: (a) that both SES and genetic factors are strong predictors of psychological distress; (b) that a large proportion of these effects are not independent, but are instead interconnected in a correlated manner; (c) that the direct effects of SES (particularly education) are substantially reduced when latent genetic confounders are taken into consideration; (d) that genetic risk factors are not deterministic influences, but are instead contingent upon levels of SES; and (e) that the buffering function of high SES against psychological distress arises primarily among individuals who are at high genetic risk for distress, and not among the population as a whole. Viewed broadly, these findings suggest that sociology's role in the scientific study of mental health is fundamentally important, and that examining sociological associations using a genetically informed design yields important information about population heterogeneity in the sociological associations. Overall, the research reported here poses at least three major implications for the sociological study of psychological distress.

First, the commonly-held sociological assumption that SES and genetic factors are independent of one another, and thus can be studied in isolation, needs to be reconsidered. In the present study, the effect of educational attainment on psychological distress was reduced (by

54% or more) when genetic factors that influence both of these outcomes were introduced into the analysis. The same was true, but to a lesser extent, for personal income. Thus, even though there does appear to be an effect of SES (at least for income) that is net of latent genetic differences, there is a considerable amount more that is inseparable from genetic sources of covariance. To be able to forcefully argue for the importance of environmental influences such as SES, sociologists are going to have to address this issue. Once they begin to do so, however, they will likely find that their arguments are actually stronger than they previously were (Carlsson et al. 2007; Caspi et al. 2005a).

Second, the significant moderation of genetic effects by SES points to a unique role for environmental factors in genetic research on psychological distress. The findings shown here suggest that genetic factors are certainly not the determinative, developmentally-unfolding "blueprints" that many biologists portray them to be (see Lewontin 2000). Instead, they are merely predispositions that manifest themselves in, and in response to, a multitude of social-environmental contexts. The research reviewed above, as well as the empirical analyses provided here, point toward a remarkable contingency of genetic influences on environmental moderators, including SES (as shown in this study), religious involvement (Boomsma et al. 1999), and stressful life events (Kendler et al. 1995), among others.

Third, and axiomatically, this study also provides evidence that the effects of SES are moderated by genetic risk factors. Specifically, these findings suggest that the effects of SES on psychological distress occur primarily among individuals who inherited a genetic risk for poor mental health, not among the population as a whole. The results in Figure 2 (c and d) show that the inverse relationship between both education and income, and psychological distress, becomes less pronounced as genetic risk decreases. In fact, in the lowest category of genetic risk, which

comprises 42.25% of the sample, there is basically no relationship between SES and distress at all. Thus, social resources appear to be particularly important among those who are biologically vulnerable, but not for others.

Given the findings regarding the correlation between genetic risks toward psychological distress and SES, future research now needs to begin examining the pathways by which genetic factors forge their way into aspects of social life such as the achievement processes reflected in education and income—i.e., Model 4 of Figure 1 needs to be intensely scrutinized. In addition to selection effects (e.g., Mulatu and Schooler 2002), which were discussed earlier, it has been proposed, although not adequately tested, that genetic factors likely affect environmental outcomes indirectly via personality and interpersonal characteristics (Alford et al. 2005; Kendler and Prescott 2006). This assertion is based on two empirical findings. First, genetic factors appear to account for a considerable proportion (~50%) of the variation on virtually all personality characteristics, including each of the "Big Five" traits: openness, conscientiousness, extraversion, agreeableness, and neuroticism (Bouchard and Loehlin 2001; Jang et al. 1996). Second, each of these traits has, in turn, been linked with many different environmental variables, including SES (for a review see: Ozer and Benet-Martinez 2006). Thus, given the presence of significant genetic effects on the major personality traits, it is likely that some of these predispositions manifest themselves in the environmental variables with which they are correlated. Research in this area is, unfortunately, quite rare, and much work remains to be done.

In addition, future research should examine other aspects of health and well-being, different environmental outcomes, and biological factors other than genetic predispositions. For example, in addition to psychological distress, research of this type now needs to be conducted on other aspects of mental health, physical well-being, and health risk behaviors such as alcohol and

tobacco use. In addition, environmental factors other than SES—e.g., religious involvement, family relations, occupational characteristics, etc.—have also been linked with health and well-being, and future research using genetically-informed techniques should examine the effects of these areas of social life as well. Further, genetic differences represent only one type of biological influence on social life and health and well-being. For example, SES and other social indicators have been linked with a variety of physiological outcomes including cortisol levels and responses (both at waking and throughout the day), catecholamines (adrenalin and noradrenalin), heart rate, organ breakdown, insulin secretion, coronary heart disease, diabetes, musculo-skeletal systems, lipoprotein profiles, obesity, infectious illness, glucose tolerance, fibrinogen, blood clotting, and blood pressure (Kristenson et al. 2004; Kunz-Ebrecht et al. 2004; Weinstein et al. 2003). Given that each of these biological factors are at least partially influenced by genetic differences, it may be the case that genetic predispositions affect social life, health outcomes, and the correlation between these two indirectly via their influences on physiological processes.

Lastly, it is important that researchers begin to contemplate how the models outlined here might function across the life course. For example, we might expect that genetic risks toward psychological distress are facilitated or exacerbated in early adulthood (a difficult stage in the life course) by stressful environments such as low income, the transition into the labor force, the breaking of old social ties and the forming of new ones, the birth and rearing of children, and the ceasing or declining of religious participation, among others. Likewise, we might also expect these same (and other) environmental factors to become buffers against genetic risks toward poor mental health in adulthood and later life as income and labor force participation become more stable resources, as social ties and support networks become more permanent, as children grow

into adulthood and become potential sources of social support for their parents, and as religious involvement tends to increase following both parenthood and retirement. Possibilities such as these have yet to be systematically investigated.

Despite providing unique insight into the environmental and genetic causes of psychological distress, the present study has at least two important weaknesses. One of these is the inability to identify any of the specific genes related to psychological distress. That said, it is becoming increasingly apparent that individual genes (the few that have actually been identified) make only minor contributions to the total genetic effect on psychological distress—a complex outcome that is likely influenced by countless, difficult to discover, genes (USDHHS 1999). Nonetheless, future research should certainly examine whether specific genes that have been linked to mental health (e.g., the serotonin transporter gene discussed above, etc.) are moderated by SES. Another shortcoming is the cross-sectional nature of the data employed here, which does not allow the exact causal ordering to be determined. Do genetic factors initially influence SES, which then affects changes in mental health over time, or do genetic differences affect mental health, which then poses subsequent implications for SES attainment? Future research should address this weakness.

To conclude, most research on psychological distress has focused on either environmental factors such as SES, or biological ones including genetic differences. Very few studies, in contrast, have explored the potential interconnections between these two predictors of distress. The findings reported here suggest that the failure to simultaneously examine both of these influences could lead to (a) extreme limitations on empirical explanatory power, (b) biased estimates and potentially confounded relationships, and (c) a continued failure to uncover some of the most interesting and theoretically important connections between SES, genetic risk factors,

and psychological distress (e.g., gene-environment correlation and interaction). The study of the complex interplay between these aspects of life promises a much deeper understanding of psychological distress than is currently possible, and future research should continue integrating sociological and biomedical models of mental health.

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Figure 1: Five Conceptual Models of the Relationships Between SES, Genetic Risk Factors, and Psychological Distress

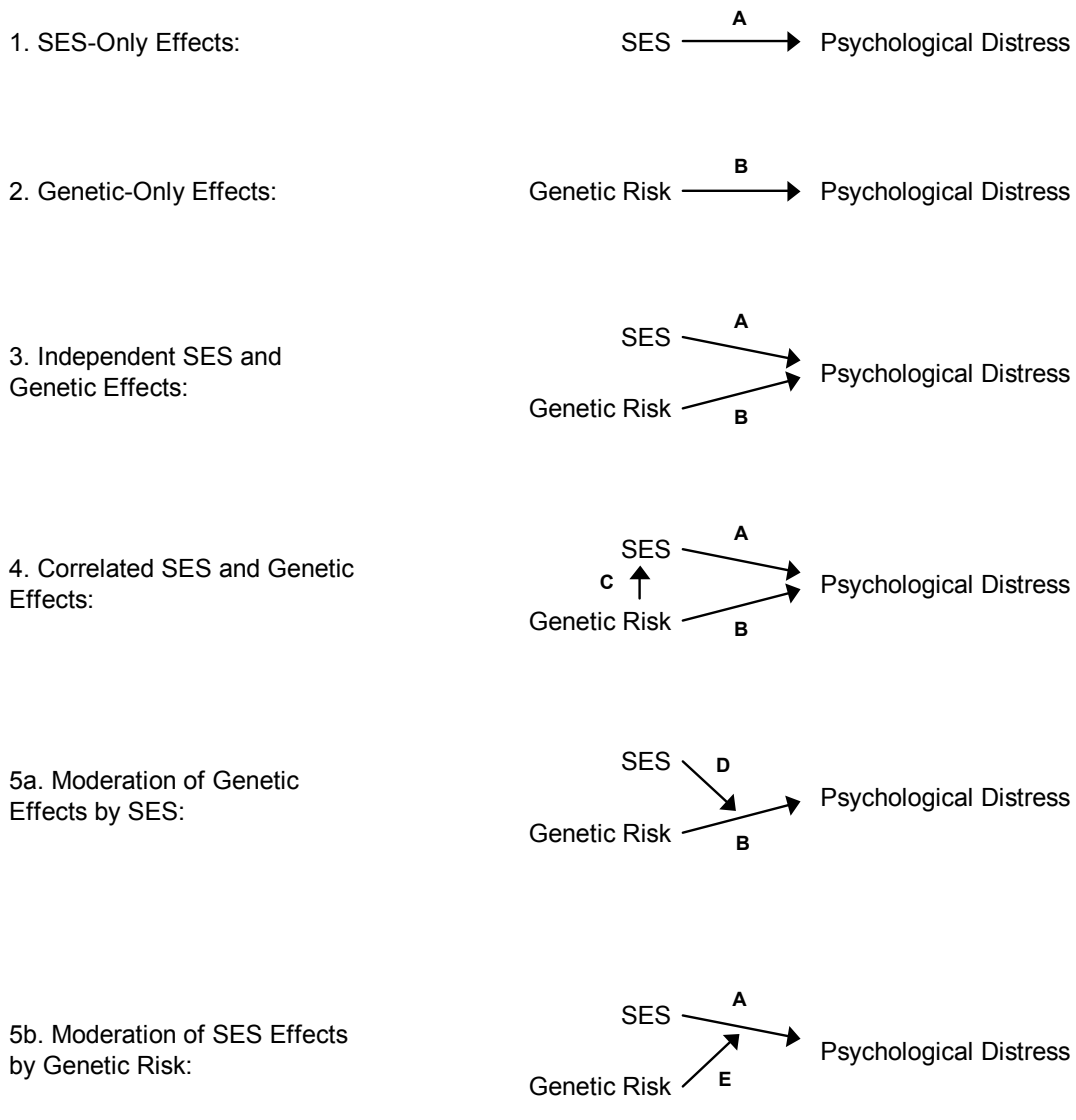


Table 1: Descriptive Statistics

	Mean	Std Dev	Min-Max
Psychological Distress	1.498	0.611	1-5
Age	45.003	12.162	25-74
Gender (Female=1)	0.579	-	0-1
Education	2.882	1.153	1-5
Less Than High School	0.101	-	0-1
High School	0.313	-	0-1
Some College	0.306	-	0-1
Bachelor's Degree	0.164	-	0-1
Graduate Degree	0.116	-	0-1
Income	18.321	9.601	1-31
Genetic Risk	0.658	0.777	0-3

Notes: N = 587 monozygotic and same-sex dizygotic pairs; psychological distress is the non-transformed version.

Table 2: Bivariate Correlations

	Psychological Distress	Education	Income	Genetic Risk
Psychological Distress	1.0000			
Education	-0.1028	1.0000		
Income	-0.1107	0.2663	1.0000	
Genetic Risk	0.4277	-0.1509	-0.1213	1.0000

Note: N = 587 monozygotic and same-sex dizygotic pairs; psychological distress is transformed as described in the text; all correlations are significant ($p<0.0001$ or less).

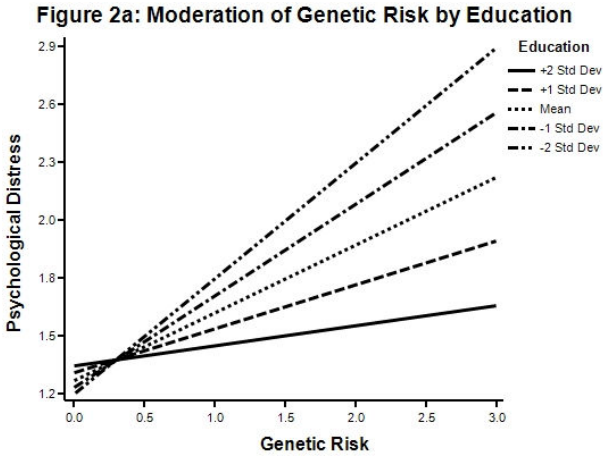
Table 3: OLS Parameter Estimates from the Regression of Socioeconomic Status, Genetic Risk, and the Interaction Between These Two Influences on Psychological Distress

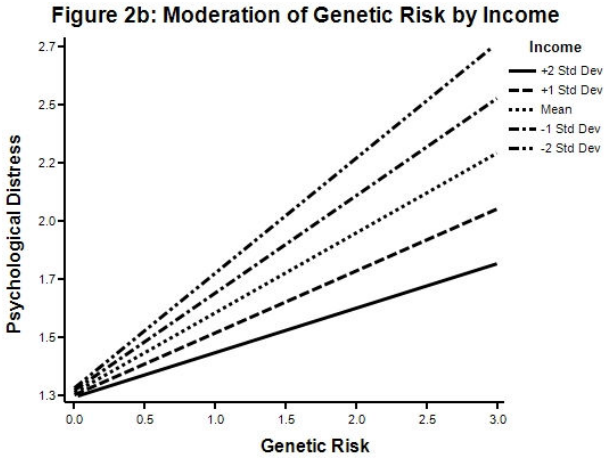
	(1)	(2)	(3)	(4)	(5)	(6)
Education	-0.0103 *	-	-0.0042	-0.0057	-0.0040	-0.0054
	(0.0048)		(0.0044)	(0.0044)	(0.0044)	(0.0044)
Income	-0.0015 *	-	-0.0011 +	-0.0010 +	-0.0012 *	-0.0011 +
	(0.0006)		(0.0006)	(0.0006)	(0.0006)	(0.0006)
Genetic Risk	-	0.0716 *	0.0696 *	0.0671 *	0.0666 *	0.0657 *
		(0.0063)	(0.0063)	(0.0063)	(0.0065)	(0.0065)
Education*Genetic Risk	-	-	-	-0.0176 *	-	-0.0153 *
				(0.0055)		(0.0059)
Income*Genetic Risk	-	-	-	-	-0.0014 *	-0.0008
					(0.0007)	(0.0007)
Intercept	0.7863 *	0.8018 *	0.7865 *	0.7882 *	0.7872 *	0.7884 *
Adjusted R-Square	0.0379	0.1930	0.1990	0.2120	0.2048	0.2132

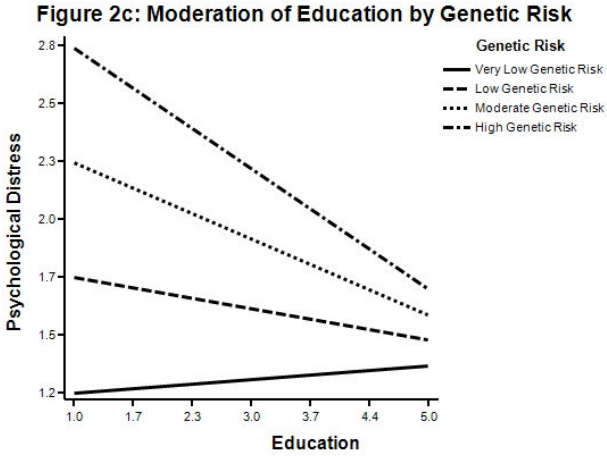
Notes: N = 587 monozygotic and same-sex dizygotic twin pairs; psychological distress is transformed as described in the text; and all models contain adjustments for age and gender (female=1).

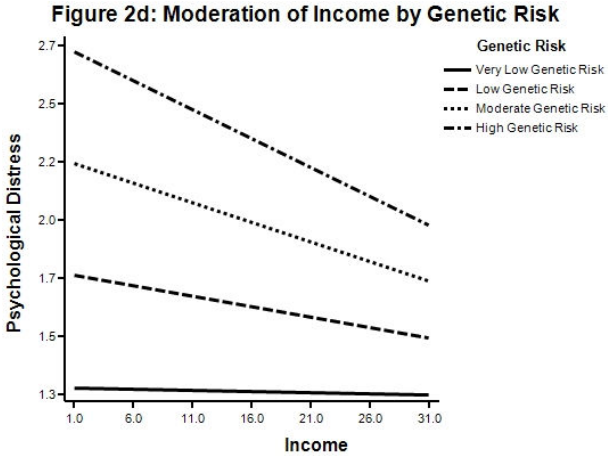
*p < 0.05 or less; +p<0.10 (two-tailed test)

Figure 2: The Interactive Influence of SES and Genetic Risk on Psychological Distress









Appendix 1: Descriptive Statistics for Genetic Risk and Psychological Distress by Education and Income

	Genetic Risk		Psychological Distress	
	Mean	Std Dev	Mean	Std Dev
Education				
High School or Less	0.798a	0.835	1.587a	0.728
Some College or More	0.558a	0.716	1.434a	0.503
Income				
1st Quartile (Lowest)	0.788b	0.843	1.573f	0.711
2nd Quartile	0.693c	0.791	1.579g	0.705
3rd Quartile	0.632d	0.776	1.477h	0.520
4th Quartile (Highest)	0.519e	0.668	1.363i	0.437

a Significantly different from other levels of education
b Significantly different from 3rd and 4th quartiles
c Significantly different from 4th quartile
d Significantly different from 1st quartile
e Significantly different from 1st and 2nd quartiles
f Significantly different from all other quartiles
g Significantly different from 3rd and 4th quartiles
h Significantly different from all other quartiles
i Significantly different from all other quartiles