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Genetic Influences on Social Life:

Evidence, Pathways, and Implications for Sociological Inquiry

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Genetic Influences on Social Life:

Evidence, Pathways, and Implications for Sociological Inquiry

by

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Dedication

To my wife Cerrie, the mother of my son William and my daughter Kailey, I would like to thank you for standing by me. You have always been a loyal partner, and you put your dreams on hold so that I could pursue mine. For that, I will always be indebted to you. I love you. To my beloved mother, who prematurely departed the earth for heaven in May of 2002, I just want to say thank you for all of your sacrifices. You lived your life for others, especially me, and gave no thought to your own needs. You are a truly inspirational role model, and each day I try to pattern my life after the example you set. I love and miss you, and I know you are smiling down on me with pride. To the best sister in the world, Melissa, I want to thank you for never giving up on me, even when I was not a very good big brother. You have been very instrumental in my success. Without your help I would never have learned how to write, or use a computer. This accomplishment is yours as well. Thank you and I love you. To my father, who on many occasions thought I was going insane, but who nonetheless patiently supported me anyway, I want to say thank you. This achievement would not have possible had you been any lesser of a father. You should be proud of yourself for helping make this dream a reality. A special thank you goes out to Chris Ellison, who has become like a brother to me. Without your guidance, patience, financial support, and wisdom, this dissertation would have never been completed. You deserve all of the accolades and rewards you have received—you are truly a one-of-a-kind professor. I thank you. I also thank all of my colleagues and fellow students, both supporters and detractors.

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Genetic Influences on Social Life:

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Scholars in diverse disciplines are currently engaged in debates concerning the causes and consequences of human social interaction in areas including personality development, interpersonal characteristics, social attachments and support, family life, religious involvement, civic engagement, socioeconomic attainment, and health and well-being, among others. Unfortunately, researchers in these areas are compartmentalized into two, largely isolated, camps: (1) social scientists who base their research on the assumption that social outcomes are primarily, if not exclusively, the products of social-environmental influences; and (2) biologists, geneticists, psychiatrists, and some psychologists, all of whom assume that genetic factors are important as well.

The purpose of this dissertation, therefore, is to begin integrating sociological and biomedical research on social life. To facilitate this task, four specific research questions are addressed: Do genetic and environmental factors both influence social life, and if so,

what is the relative contribution of each? Why and how do genetic factors influence social life, and what are the pathways by which they operate? Are genetic and environmental influences on social life correlated (i.e., non-additive), and do genetic factors bias social scientific studies that do not take them into consideration? Do genetic and environmental factors interact to produce social outcomes? To answer these questions, twin sibling data from the National Survey of Midlife Development in the United States (MIDUS) is analyzed.

In response to the first question, results reveal that both genetic and environmental factors are indeed important predictors of individual-level variation on several different aspects of social life, including religious involvement, civic engagement, personality and interpersonal characteristics, family relations, socioeconomic status, community attachment, neighborhood quality, and psychological distress. Further, genetic effects on several of these outcomes (e.g., civic engagement, psychological distress) are mediated by personality, interpersonal characteristics, and social relationships, which provides insight into the second and third research questions. With respect to the final question, the findings presented here suggest that genetic and environmental influences on at least one social outcome—health and well-being—function in both a correlated and interactive manner. Overall, the theoretical and empirical research provided in this dissertation highlights a growing need for research that integrates sociological and biological approaches to the study of social life.

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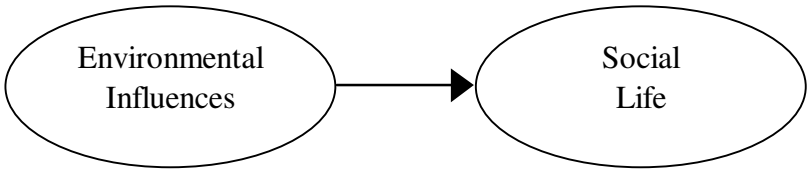
INTRODUCTION

The study of social life is a vibrant area of scientific inquiry. In attempts to improve both individual and societal well-being, researchers and policymakers alike seek to understand the causes and consequences of human social interaction in areas including personality development, interpersonal characteristics, social attachments and support, family life, religious involvement, civic engagement, socioeconomic status, and mental and physical health, among others (Bouchard and Loehlin 2001; Campbell 2006; Clary and Snyder 1999, 1991; Cohen 2004; Ozer and Benet-Martinez 2006; Putnam 2000; Rowe et al. 1998; Stark and Finke 2000; Wuthnow 1990). Despite the utility of the published research on these topics, our understanding of social life is currently being impeded by the compartmentalization of scholars into two, largely isolated, camps.

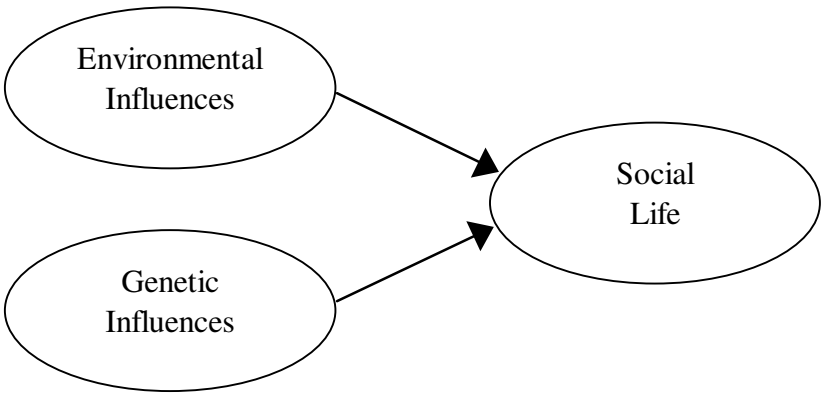
Figure I.1:
Environment-Only Effects



On one front are social scientists from multiple disciplines including sociology, psychology, history, economics, and political science, among others. Scholars in these fields generally base their research on the assumption that social-oriented attitudes and

behaviors are primarily, if not exclusively, the products of environmental influences such as observational learning, positive and negative reinforcement, normative pressures, and social exchanges. Shown conceptually in Figure I.1, examples of this research include: personality and social orientations as products of social structures and contexts (Hartup and van Lieshout 1995; Kohn 1989); religion as a cultural system of meanings and norms (Smith 2003), or a marketplace for exchanges between humans and supernatural others (Stark and Bainbridge 1996); civic engagement as an outgrowth of human, social, and cultural capital (Galston 2001; Wilson and Musick 1997); and health disparities as manifestations of differences in the quality and quantity of social relationships (Mirowsky and Ross 2003).

Figure I.2:
Additive Genetic and Environmental Effects



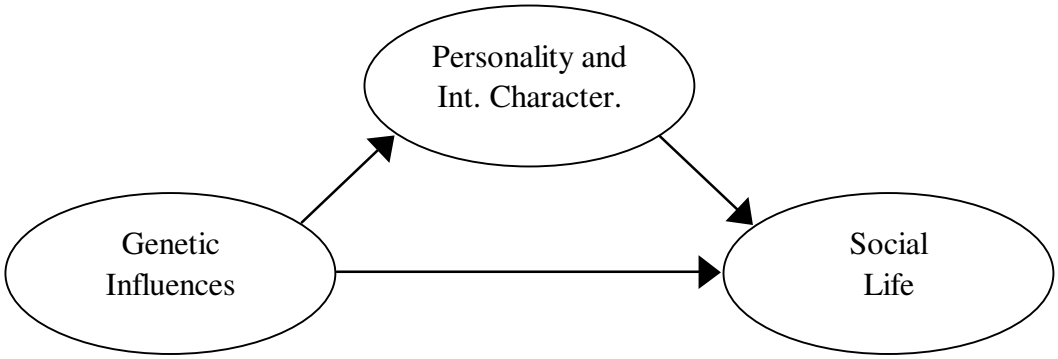
On another front are geneticists, biologists, psychiatrists, and some psychologists, all of whom ground their research in a somewhat competing assumption: that genetic (or other biological) factors also influence social life. Although research in this area is still relatively sparse, findings on several aspects of social life, including personality, interpersonal characteristics, and health and well-being, suggest that genetic influences are indeed important (Bouchard and Loehlin 2001; Caspi et al. 2003; Hamann 2005; Jang et al. 1996; Plomin and Caspi 1998; Plomin and Rende 1991). There is, however, a relative dearth of genetically-informed research on other social outcomes, including religious involvement, civic engagement, family relations, and social support and attachments, among many others. Thus, the first research question addressed in this dissertation is:

- Research Question 1: Do genetic and environmental factors both influence social life, and if so, what is the relative contribution of each?

To empirically examine genetic influences, twin and adoption designs—which seek to disentangle proportional genetic and environmental effects by comparing individuals of known, and differing, genetic relatedness—are typically employed. These approaches, which take the form of the conceptual model shown in Figure I.2, assume that genetic and environmental influences on social outcomes are additive—i.e., that each exerts independent and largely uncorrelated effects. This model has been utilized in almost every genetically-informed study of social life conducted to date, and it is employed here

to examine a variety of social outcomes, including: religious involvement (Chapter 1); personality, interpersonal characteristics, family ties and support, and broad-based social attachments (Chapters 2, 3, and 4); civic engagement (Chapter 2); socioeconomic status (Chapters 3, 4, and 5); and health and well-being (Chapters 3, 4, and 5). Overall, this research will help us to understand whether, and to what extent, both genetic and environmental factors influence individual-level variation on these aspects of social life.

Figure I.3:
Mediation of Genetic Effects by Personality and Interpersonal Characteristics



Originally, this dissertation intended to focus exclusively on Research Question 1, and this introduction would have ended here. Each of the empirical chapters would have presented findings from univariate analyses of the various aspect of social life outline above, and the conclusion of each would have been that genetic effects do indeed exist, and are quite substantial. Given the current dearth of genetically-informed research on many aspects of social life, this would have been an important contribution to the

literature. However, after reviewing much of the published research in this area, and upon completion of the first chapter, which showed sizable and statistically significant genetic effects on eight different measures of religious involvement, it became apparent that simply examining whether or not genetic factors influence social life was not sufficient. In fact, doing so actually raised more questions than it answered. Importantly, these unanticipated questions turned out to be more profound (and interesting) than the original one. Therefore, the focus of this dissertation subsequently shifted from an exploratory analysis of potential genetic effects on social outcomes, to a detailed examination of the pathways by which genetic factors influence social life, and the consequences they might pose for sociological research that does not take them into consideration. Thus, starting in Chapter 2, more complex and theoretically important questions are addressed. The first of these, which follows logically from the initial findings showing that genetic factors influence all of the social outcomes examine here, is:

- Research Question 2: Why and how do genetic factors influence social life, and what are the pathways by which they operate?

To date, virtually no research has addressed this issue, and we currently do not know why or how genetic factors influence social life. Therefore, theory in this area will have to be built from the ground up. This dissertation initiates this task, and thus begins establishing knowledge on this front by developing, and then empirically testing, a preliminary theory suggesting that genetic influences on social outcomes are at least

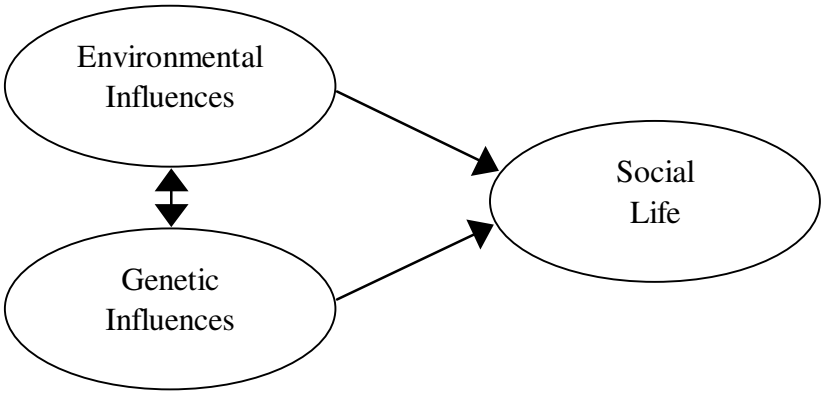
partially indirect via two broad-based mediators: personality and interpersonal characteristics (Figure I.3). For these outcomes to mediate genetic effects on social life, however, two conditions must be met: (1) genetic factors must influence not only social outcomes, but also these mediators; and (2) these mediators must be correlated with social life.

With respect to the first requirement, research shows that genetic factors do indeed account for a considerable proportion of the individual-level variation on: (a) virtually all personality traits, including each of the "Big Five" (i.e., openness, conscientiousness, extraversion, agreeableness, and neuroticism) (Bouchard and Loehlin 2001; Jang et al. 1996); and (b) various measures tapping interpersonal characteristics, including social closeness and intimacy, dependability, adherence to social rules and authority, positive relations with others, social support, network closure, and social ties (Bouchard and Loehlin 2001; Bradshaw 2007; Cleveland and Crosnoe 2004; Kendler 1997; Kenrick et al. 2003; Kirkpatrick 2004; Newberg et al. 2002; Plomin 1990; Wilson 1998). Consistent with the second condition, each of these potential mediators has, in turn, been linked with multiple aspects of social life, including religious involvement, family and peer relations, occupational choice and success, educational attainment, political orientations, volunteering and other forms of prosocial behavior, and health and well-being (Cohen 2004; Ellison and Levin 1998; Glenn and Weaver 1981; Gove, Style, and Hughes 1990; House 2002; Hummer et al. 1999; Lu and Hu 2005; Miech et al. 1999; Mulatu and Schooler 2002; Noor 1996; Ozer and Benet-Martinez 2006; Spotts et al. 2005). Thus, these two potential mediators meet the initial requirements for fulfilling this explanatory

role, and as shown in Figure I.3, it is therefore possible that genetic effects on these outcomes manifest themselves indirectly in the aspects of social life with which they are correlated. In sum, it is theorized that genetic factors influence social life in part by shaping individual differences in personality and interpersonal characteristics, which subsequently affect social outcomes. Here, these ideas will be theoretically developed and empirically tested for two different social outcomes: civic engagement (Chapter 2), and health and well-being (Chapters 3, 4, and 5).

The questions raised by the presence of genetic effects on social life do not end here, however, and in addition to examining (a) whether, and to what extent, genetic factors influence social outcomes, and (b) why and how they function in this capacity, it is important that social scientists begin to think about (c) the potential implications that genetic influences might pose for mainstream sociological research that does not take them into consideration. If the simple additive effects model shown in Figure I.2 is accurate, social scientists can probably continue to ignore genetic influences. Explanatory power deficiencies will obviously be an issue if genetic factors explain a considerable proportion of the variation on any particular social outcome, but other than that, genetic factors would pose very few implications for social science. If this model is inaccurate, however, at least two major issues will come to the forefront, and will need to be addressed.

Figure I.4:
Correlated / Nonadditive Genetic and Environmental Effects



First, genetic and environmental influences on social life might actually be correlated, not additive (Figure I.4). Given that research has already shown that genetic factors influence (a) several different "environmental" variables, and (b) multiple aspects of social life (Bouchard and Loehlin 2001; Bradshaw and Ellison Forthcoming; Cleveland and Crosnoe 2004; Kendler 1997; Spotts et al. 2005), it is therefore possible that that they also affect (c) the "correlations" between environmental influences and social life (Rutter, Moffitt, and Caspi 2006). Previous research suggests that this may indeed be the case for several different associations, including the ones between SES and health (Lichtenstein et al. 1992), family life and human development (Reiss et al. 2000), marital quality and psychological well-being (Spotts et al. 2005), and stressful life events and depression (Kendler and Prescott 2006). If findings such as these generalize to other areas of social life, this would suggest that the failure to control for genetic effects while simultaneously examining environmental ones (and vice-versa) could lead to biased estimates and

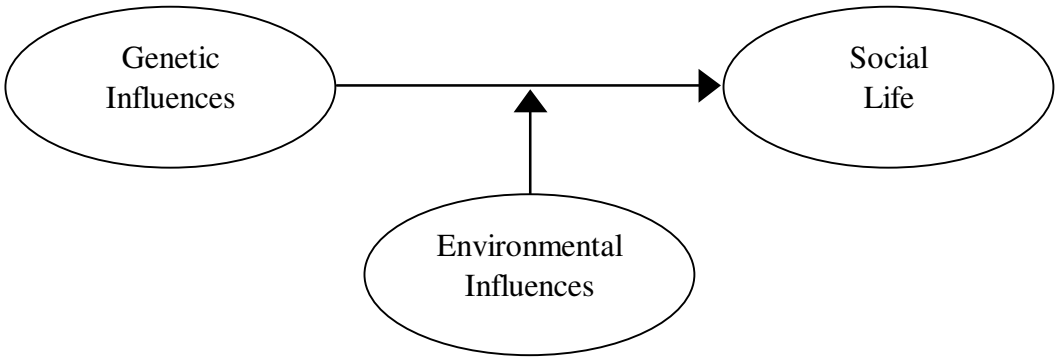
possibly even erroneous conclusions regarding cause and effect (Bouchard and Loehlin 2001; D'Onofrio et al. 2006; Gottfredson 2003; Lichtenstein et al. 1992; Neiderhiser et al. 1999; Reiss et al. 2000; Spotts et al. 2005; Udry 1995). This leads to a third research question:

- Research Question 3: Are genetic and environmental influences on social life correlated (i.e., non-additive), and do genetic factors bias social scientific studies that do not take them into consideration?

In essence, gene-environment correlation (i.e., genetic confounding) occurs when latent genetic factors explain all, or part, of a theoretically important relationship between an environmental influence and a social outcome. To exemplify gene-environment correlation, take the following example for the well-established connection between SES and health and well-being: We know that educational attainment (a commonly-employed measure of the "environment") is positively associated with desirable health outcomes, but in contrast to the typical sociological interpretation—e.g., that high levels of education provide psychosocial resources that buffer against stressors that lead to poor health, or that low levels create stressful conditions that undermine health and well-being—it might also be the case that genetic factors predispose individuals in some way toward (a) both high levels of education and desirable health, or (b) both low levels of education and poor health. In this case, the sociological relationship between the environmental influence (i.e., education) and the social outcome (i.e., health), as shown

in Figure I.4, might be weak or absent when latent genetic differences are taken into consideration. This could occur if the same genetic factors that predict health and well-being also influence educational attainment. In this dissertation, gene-environment correlation will be addressed in detail for health and well-being in Chapter 3, a theoretical expose, and then empirically examined in Chapter 4 for the correlations between many different measures of social relationships (i.e., social-oriented personality traits, interpersonal characteristics, family ties, SES, community attachment, and neighborhood quality), and psychological distress.

Figure I.5:
Environmental Influences as Moderators of Genetic Effects

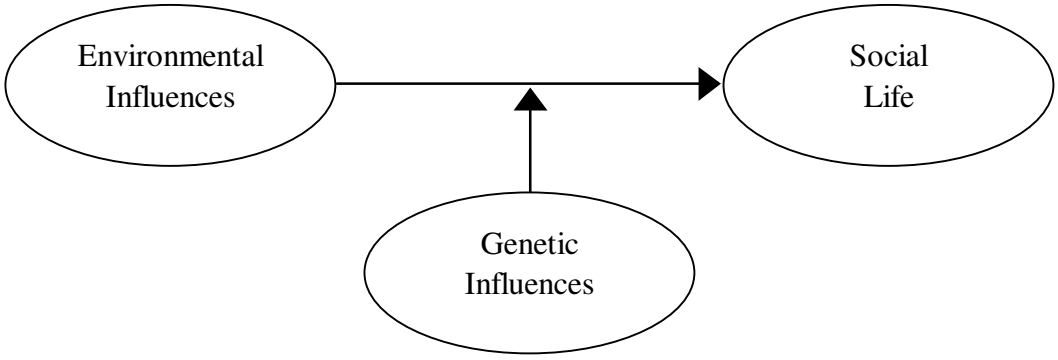


In addition to gene-environment correlation, genetic effects on social outcomes pose a second potential implication for social scientific research: gene-environment interaction. Even though much work remains to be done in this area, there is growing evidence that (a) genetic effects are either more or less pronounced depending upon environmental

influences (Figure I.5), and / or (b) that environmental factors have different effects based on the genetic makeup of individuals (Figure I.6) (Boomsma et al. 1999; Caspi et al. 2005a and b, 2003; Eley et al. 2004a and b; Jaffee et al. 2005; Jang et al. 2001; Kendler 2001, 1999, 1995; Silberg et al. 2001; Shanahan and Hofer 2005).

Once again exemplified for health and well-being, research on the first scenario suggests (a) that desirable environments may buffer against or impede genetic liabilities toward poor health (or, alternatively, enhance ones for good health), and (b) that stressful environments may trigger or facilitate genetic risks for poor health (or, alternatively, offset ones for good health) (Figure I.5). 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 genetic influences on both aspects of physical health were 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. There are several other examples in this area, and they will be reviewed extensively in Chapter 3. An empirical analysis of SES and psychological distress will then be conducted in Chapter 5. Overall, research of this type is critically important for social scientists because: (a) it demonstrates that genetic effects are not determinative, but are instead highly contingent upon environmental contexts; and (b) it provides a deeper understanding of the possible mechanisms by which environmental factors influence social life (i.e., by facilitating or impeding innate predispositions).

Figure I.6:
Genetic Influences as Moderators of Environmental Effects



Other research on gene-environment interaction hints at a theoretically different, yet statistically identical, scenario: genetic moderation of environmental influences (Figure I.6). The literature in this area suggests that the effects of environmental variables on health may actually be stronger among certain individuals compared with others based on genetic differences. For example, research shows that the relationship between stressful life events and depression is stronger among individuals at high genetic risk compared with those at low genetic risk (Kendler et al. 1995). In other words, for individuals who are not genetically 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. Importantly, this means that environmental factors may only be important for certain individuals (i.e., those who are at high genetic risk), not for the population as a whole. Additional examples in this area will be provided in Chapter 3, and this issue will be empirically examined for SES and

psychological distress in Chapter 5. In essence, it is crucial that social scientists begin paying attention to this possibility because: (a) genetic factors may help to identify at-risk populations that could disproportionately benefit from social scientific research; and (b) social scientists are the best-equipped scholars when it comes to understanding the health-related consequences of a vast array of environmental influences, including race, gender, family life, social support religious involvement, socioeconomic status, occupational characteristics, community attachment, neighborhood quality, and stressful life events such as divorce or the loss of a loved one, among others. Many, and perhaps even most, biomedical researchers are ill-equipped to deal with the complexity in these areas. Overall, this literature suggests a final research question:

- Research Question 4: Do genetic and environmental factors interact to produce social outcomes, particularly health and well-being?

To summarize, social scientific research assumes that social outcomes are primarily, if not exclusively, the products of environmental influences. There is, however, a growing body of evidence suggesting that this assumption may be false, and that social life may instead be the result of interconnected genetic and environmental influences. If true, this could pose several potential implications for traditional social science, including explanatory power deficiencies, correlated genetic and environmental effects, environmental effects that are contingent upon genetic differences (and vice-versa), biased estimates, and erroneous conclusions regarding cause and effect, among others.

The purpose of this dissertation, therefore, is to: (a) examine whether, and to what extent, genetic factors predict individual variation on social outcomes (Research Question 1 and Figure I.2); (b) theorize and empirically test potential explanations for genetic effects by identifying the pathways by which they influence social life (Research Question 2 and Figure I.3); (c) provide estimates of the extent to which gene-environment correlation might bias social scientific research that does not take genetic differences into consideration (Research Question 3 and Figure I.4); and (d) explore the possibility that genetic and environmental factors moderate (i.e., facilitate or impede) the influence of each other on social outcomes (Research Question 4 and Figures I.5 and I.6).

In total, 23 different aspects of social life are examined here, including social-oriented personality traits (e.g., extraversion, agreeableness, neuroticism), interpersonal characteristics (e.g., positive relations with others), family life (e.g., marriage, partner support, and family support), religious involvement (e.g., service attendance, salience, various beliefs, and transformations / commitments), civic engagement (e.g., obligations toward civic participation, and volunteering for charity), socioeconomic status (e.g., education, income, and employment status), and broader social ties and contexts (e.g., community attachment, and neighborhood quality). These particular aspects of social life were chosen because (a) they are commonly-examined topics of sociological inquiry, and (b) the amount of genetically-informed research and knowledge on each varies from virtually nothing (for civic engagement, family life, community attachment, and neighborhood quality) to a small amount (for religious involvement, social support, and socioeconomic status) to a great deal (for personality traits, and health and well being).

This variety allows for theoretical and empirical investigations of many different models of gene-environment effects, ranging from simple univariate analyses, to pathbreaking multivariate models of gene-environment correlation and interaction.

DISSERTATION OVERVIEW

Chapter 1

Research Question 1 asked: Do genetic factors influence social life, and if so, to what extent? Currently, we know the answer to this question for several social outcomes, particularly personality traits and health and well-being, but we do not yet know the degree to which genetic factors influence other aspects of social life, including the one examined in this chapter: religious participation. Utilizing data from a national sample of working-age twin siblings, this chapter provides estimates of proportional genetic and environmental effects on four different dimensions (eight measures) of religious involvement: (1) organization-based practices (i.e., service attendance), (2) personal religiosity and spirituality (i.e., salience, identification, spirituality, coping, etc.), (3) conservative ideologies (i.e., Biblical literalism and exclusivist beliefs), and (4) transformations and experiences (i.e., being "born-again" or making a religious commitment). Results from univariate structural equations models reveal sizable and statistically significant genetic effects—explaining 19-65% of the individual-level variation depending upon the particular measure under investigation—on all eight aspects of religious life. Given that the literature in this area is extremely sparse, simply providing evidence for genetic influences on religious involvement is an important step at

the present time. This is especially true given that each of the measures examined here is widely-employed in mainstream social scientific studies of religion, both as an outcome of inquiry, and as a predictor of other aspects of social life including prosocial attitudes and behaviors, family life, and health and well-being.

Chapter 2

This chapter examines another aspect of social life that has been largely ignored by genetically-informed research: civic engagement. To determine whether, and to what extent, genetic factors influence civic engagement (Research Question 1), univariate twin models are fit to two different outcomes: (1) attitudes toward civic engagement, and (2) the frequency of volunteering. Results reveal significant genetic effects on individual-level variation on both of these variables: 46 and 34%, respectively. In addition to these univariate findings, a theoretical model linking genetic effects with civic engagement indirectly via personality and interpersonal characteristics is also examined in an attempt to address Research Question 2. Multivariate structural equations modeling findings provide strong support for this model for obligations toward civic engagement, but not volunteering for charity. More specifically, a considerable proportion of the total genetic effect on obligations toward civic engagement is mediated by (i.e., correlated with) extraversion, agreeableness, positive relations with others, and community attachment. With respect to volunteering for charity, however, these potential mediators were not found to be very effective.

Chapter 3

The two previous chapters exemplified the utility of a genetically-informed approach for two social outcomes that have been given very little consideration in the literature: religious involvement and civic engagement. In contrast, this chapter (and subsequent ones as well) transitions to a topic of inquiry that has received considerable attention from geneticists, biologists, psychologists, psychiatrists, and sociologists alike: health and well-being. That said, even though (a) scholars from multiple disciplines are working in this area, and (b) there is considerable evidence in support of both genetic and environmental influences on virtually all health outcomes, there is nonetheless a relative dearth of research on the potential ways in which these two influences might be interconnected. Therefore, this chapter addresses Research Questions 1, 2, 3, and 4 by providing a detailed theoretical formulation of five possible models of gene-environment effects on health and well-being: (1) genetic-only effects; (2) environment-only effects; (3) additive genetic and environmental effects; (4) correlated genetic and environmental effects; (5a) environmental moderation of genetic effects; and (5b) genetic moderation of environmental effects. The theoretical insight provided in this chapter will help to (a) organize the existing literature in this area, and (b) guide and frame future scholarship on genes, environments, and health.

Chapter 4

The theoretical expose provided in the previous chapter raised several important issues regarding genetic and environmental effects on health and well-being, including gene-

environment correlation and gene-environment interaction. Motivated by Research Question 3, this chapter examines the former for the widely-noted correlation between social relationships and psychological distress. Given that genetic factors appear to predict both social relationships and levels of distress, it is therefore possible that they explain at least part of the correlation between these two outcomes as well. Empirical findings show: (a) that many different aspects of social life, including personality, interpersonal characteristics, family ties, socioeconomic status, and broader social contexts such as community attachment and neighborhood quality, are indeed associated with psychological distress; (b) that genetic factors account for non-trivial proportions (15-52%) of all of these relationships; (c) that most of the aspects of social life employed here (besides education) are correlated with distress net of genetic influences; and (d) that at least part of the genetic effect on distress is indirect via social relationships. It is concluded that the failure to address genetic influences could pose at least two major implications for the sociological study of psychological distress: (a) biased empirical findings, and (b) erroneous conclusions regarding cause and effect.

Chapter 5

Chapter 5, the final empirical analysis, addresses Research Question 4 and the other main issue raised in Chapter 3: gene-environment interaction. More specifically, it examines five conceptual models of the relationship between SES and genetic risk factors as they pertain to psychological distress: (1) SES-only effects; (2) genetic-only effects; (3) additive SES and genetic effects; (4) correlated SES and genetic effects; (5a) moderation

of genetic effects by SES; and (5b) moderation of SES effects by genetic risk. Multivariate analyses provide support for each of these models, and thus indicate that all four of the research questions raised in this dissertation deserve intense scrutiny by scholars of health and well-being. With particular emphasis on Research Question 4, it was found that SES (both education and income) interacts with genetic effects on psychological distress, which suggests (a) that SES can help to buffer against the negative impact of genetic risk factors on psychological distress, and (b) that SES is associated with distress primarily among individuals who are genetically at risk, not among the population as a whole. The implications of these findings for social scientific inquiry are discussed in detail.

Conclusion

To conclude, the purpose of this dissertation is fourfold. First, to examine whether, and to what extent, genetic factors influence individual variation on several different aspects of social life, including religious involvement, personality and interpersonal characteristics, civic engagement, family relations, socioeconomic status, and health and well-being, among others. Second, to theorize, and then empirically test, a conceptual model linking genetic influences with social life indirectly via personality and interpersonal characteristics. Third, to determine the extent to which gene-environment correlation (i.e., genetic confounding) might bias social scientific research that does not take genetic differences into consideration. And fourth, to explore the possibility that genetic and environmental factors moderate the influence of each other on social life. This

concluding chapter summarizes the findings in these areas, provides a detailed discussion of the implications posed by these findings, and outlines a detailed agenda for future research in this area.

CHAPTER 1

Do Genetic Factors Influence Religious Life?

Findings from a Behavior Genetic Analysis of Twin Siblings

SUMMARY

Social scientific research assumes that religious involvement is primarily, if not exclusively, the product of social-environmental influences. There is growing evidence, however, that genetic or other biological factors also play a role. Analyzing twin sibling data from National Survey of Midlife Development in the United States (MIDUS), this chapter addresses this issue, as well as Research Question 1, by showing that individual-level variation on four different aspects of religious life—organizational involvement, personal religiosity and spirituality, conservative ideologies, and transformations and commitments—is indeed the product of both genetic and environmental influences. Specifically, genetic effects explain 19-65% of the variation, while environmental factors account for the remaining 35-81%, depending upon the aspect of religion under investigation. It is concluded that research of this type enhances contemporary social science by providing a new perspective that nicely supplements existing ones, but that it also highlights potential implications, including explanatory power deficiencies and potentially biased empirical findings.

INTRODUCTION

Social scientific research assumes that religious involvement is primarily, if not exclusively, the product of social-environmental influences—e.g., observational learning, positive and negative reinforcement, normative and cultural pressures, social exchanges, etc. (Batson, Schoenrade, and Ventis 1993; Ellison and Sherkat 1995; Hayes and Pittelkow 1993; Myers 1996; Sherkat 1997; Smith 2003; Stark and Finke 2000). There is growing evidence, however, that this assumption is false, and that genetic or other biological factors also influence religious life.

For example, a handful of twin and adoption studies have reported statistically significant genetic effects on several religious outcomes including service attendance, conservative beliefs, and spirituality, among others (Beer et al. 1998; D'Onofrio et al. 1999a; Kirk, Eaves, and Martin 1999; Koenig et al. 2005; Loehlin and Nichols 1976; Winter et al. 1999). In addition, Miller and Stark (2002) recently invoked biology as a potential explanation for one of the most replicated findings in the literature: the fact that women are, on average, more religious than men. Further, while critiquing the overly deterministic approaches of sociobiology and evolutionary psychology, Smith (2003: 38-39) nonetheless acknowledged that biological factors play some sort of role in the construction of "moral (i.e., religious), believing animals":

> Rejecting the reductionism of explaining human morality as serving the interests of selfish genes, however, does not require the dismissal of an interest in biological factors in human social life. Nor does it necessitate the rejection of

> possible evolutionary frameworks of explanation. In whatever other ways that human animals may be mental or moral or spiritual beings, we are also clearly biological organisms, and there is no reason to think that biology and mental, moral, and spiritual life do not interact. Of course they do.

Focusing on one particular biological influence—genetic similarities / differences—the present study extends our knowledge on this front by addressing an elemental question: Is individual-level religious variation the product of both genetic and environmental influences, and if so, what is the relative contribution of each? If religious involvement is indeed influenced by genetic factors, traditional social scientific models may suffer from explanatory power deficiencies if they ignore sizable genetic effects. Furthermore, studies employing religious participation as a predictor of other outcomes such as prosocial attitudes and behaviors, family relations, and health and well-being, among others, may report findings that are biased by latent genetic influences (Bouchard and Loehlin 2001). Although largely ignored to date, the profound nature of these issues has been plainly stated by at least one sociologist (Udry 1995: 1274): "In the sociological literature I read, there appears to be a complete ignorance of behavior-genetic findings that threaten the validity of conclusions from traditional sociological research designs."

The present study addresses these issues by performing four tasks. First, the relatively small, but growing, literature in this area will be reviewed. Second, the theory, methods, and statistical techniques used to conduct genetically-informed research on religious involvement will be described. Third, using data from the National Survey of Midlife

Development in the United States (MIDUS)—a large national sample of working-age (25-74) twin siblings—genetic and environmental influences on individual-level religious variation will be estimated for four unique aspects (eight measures) of religious life: organizational involvement, personal religiosity and spirituality, conservative ideologies, and transformations and commitments. And fourth, a detailed discussion of the relevance of these findings for the social scientific study of religion will be provided, and an agenda for future research will be outlined.

BACKGROUND

In support of the widely-held assumption that religion is a social phenomenon, research has shown that individual differences in religious involvement are influenced by an array of environmental factors including parental religiosity and moral emphasis (Francis and Brown 1991; Hayes and Pittelkow 1993; Hoge, Petrillo, and Smith 1982; Myers 1996), school and church settings (Francis and Brown 1991; Regnerus, Smith, and Smith 2004), community and cultural contexts (Berger 1969; Ellison and Sherkat 1995; Geertz 1973; Pattillo-McCoy 1998; Smith 2003; Wuthnow 1987), and even religious market structures (Finke and Stark 1992; Iannaccone 1994; Stark 1997; Stark and Bainbridge 1996; Stark and Finke 2000). Although the utility of this literature is undeniable, a potential problem exists: religious socialization occurs primarily in family contexts, and family members share both genes and social environments.

It is therefore imperative that we begin to examine (a) whether, and to what extent, genetic factors influence individual-level variation on religious outcomes, and (b) if the

relative contribution of genetic and social-environmental influences varies depending upon the aspect of religious life under consideration (i.e., whether genetic influences are more important for some religious outcomes compared with others). An approach referred to as behavior genetics provides the theoretical, methodological, and statistical tools required to investigate these issues (Carson and Rothstein 1999; Plomin and Rende 1991). Specifically, by simultaneously analyzing data on multiple family members with varying degrees of genetic relatedness (e.g., identical versus fraternal twin pairs, biological versus adopted siblings, etc.), behavior genetic techniques allow researchers to decompose observed (i.e., phenotypic) individual-level variation on any outcome, including religious ones, into three proportional variance components: genetic, shared environmental, and nonshared environmental influences (Neale and Cardon 1992; Plomin 1990).

Within this framework, *genetic influences* gauge the effects of biologically-inherited individual differences in DNA and related products (e.g., proteins, hormones, anatomical structures, etc.). When applied to "social" outcomes such as religious involvement, genetic influences—which function to make individuals who share them similar to each other—tell us what proportion of the variation among individuals is explained by gene-based predispositions, needs, wants, desires, or motivations. In other words, the genetic component of the behavior genetic model is best conceptualized as an indicator of, or a proxy for, the manifestation of individual free-will or freedom of choice (Udry 1996).

Shared environmental influences in the behavior genetic framework are non-genetic, family-level social influences that are common to individuals in a family unit, perfectly

correlated between them, and that function to make those who share them similar to each other. In most cases, this reflects the influence of parents and siblings, but it also gauges other factors such as shared friends, teachers, religious leaders, family socioeconomic status, and even certain cultural or ecological influences such as living in a religiously homogeneous community. In essence, this component of the behavior genetic model taps the effects of family-level socialization as conceptualized by most social scientific theories, and thus it is of crucial importance to scholars in this area.

Nonshared environmental influences, in contrast to shared ones, are environmental sources of variation that are unique to individuals, uncorrelated between them, and that function to make individuals different from one another. This is a heterogeneous residual category that contains not only environmental influences not shared by individuals—i.e., different friends, role models, interpretations of culture, opportunities, etc.—but also measurement error in empirical analyses. Unlike shared genes and shared environmental influences, which both function to increase family similarity, nonshared environmental influences or unique experiences function to differentiate family members.

Although still relatively sparse, behavior genetic research has actually been conducted on religious involvement, and the current literature suggests three broad trends. First, religious affiliation appears to be transmitted primarily via social and cultural mechanisms (e.g., family-based socialization, normative pressures, memes, etc.), not genetic ones (Boomsma et al. 1999; Eaves et al. 1990). That said, at least one study has reported a small genetic effect on being a member of a conservative religious institution (12%), but the majority of the variation on this outcome was nonetheless shown to be the

product of shared (51%) and nonshared (37%) environmental influences (Kendler, Gardner, and Prescott 1997).

Second, several behavior genetic studies have examined organizational religious involvement (i.e., religious or spiritual service attendance), but the results to date have been somewhat inconsistent. For example, while one study found no support for genetic influences on individual-level variation on religious attendance (Truett et al. 1992), other publications have reported statistically significant genetic effects on this outcome (Kendler, Gardner, and Prescott 1997; Kirk et al. 1999). In a recent article, genetic factors were found to account for 39% of the variation on an indexed measure of "external religiousness" that included items tapping service attendance, discussing religious teachings, observing religious holidays, and membership in youth / study groups (Koenig et al. 2005). Given these contradictory findings, additional research will be required before we will be able to confidently make any conclusions regarding the presence, or absence, of genetic effects on this aspect of religious life.

Third, researchers have also explored an assortment of religious beliefs and attitudes, again with somewhat mixed findings. For example, a few studies have reported that genetic factors account for a significant proportion (20-45%) of the individual-level variation on fundamentalist orientations (Beer et al. 1998), as well as attitudes pertaining to the observance of the Sabbath, divine law, church authority, and the truthfulness of the Bible, among others (Martin et al. 1986). Another published manuscript, however, found virtually no genetic contribution to individual variation on personal conservatism (Kendler, Gardner, and Prescott 1997). Meanwhile, a recent study of "internal

religiousness" found that genetic factors accounted for 34% of the variation (Koenig et al. 2005). An article on intrinsic and extrinsic religious motivations reported that slightly more than a third of the individual variation on each was due to genetic differences (Bouchard et al. 1999), while a study of self-transcendence found that genetic influences were responsible for approximately 40% of the variation (Kirk, Eaves, and Martin 1999). Genetic factors even appear to influence attitudes concerning religious leisure time and occupational interests (Waller et al. 1990).

Overall, this small literature, while thought provoking, suffers from several shortcomings including contradictory findings and the lack of detailed information regarding potential genetic effects on a variety of theoretically important and widely-employed (by social scientists) aspects of religious life including personal religious salience, religious coping strategies, the use of religion during daily life, conservative beliefs, and religious transformations and commitments. To date, only religious affiliation, service attendance, and a handful of beliefs and attitudes, have been examined from a behavior genetic perspective, and even in these areas, more research is need because inconsistent results have been reported. Furthermore, very few of the published findings in these areas have been replicated, and certainly not with population-based, national data. Therefore, by analyzing data on numerous aspects of religious life from a large sample of U.S. adults, the present study addresses many of these shortcomings in the literature.

METHODS

Data

In order to examine genetic and environmental influences on individual-level religious variation, the present study analyzes 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). This data was collected 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. This particular data source was selected for the present study for three reasons. First, it contains questions gauging numerous aspects of religious life, including several that have yet to be examined with genetically-informed techniques. Second, it contains data on adults, which is especially important since theory suggests that genetic influences should manifest themselves more strongly among adults because they have more freedom to make choices compared with children and adolescents (D'Onofrio et al. 1999a; Plomin 1990; Udry 1996). And third, it is a large national sample of U.S. adults, which should make the results more generalizable to the U.S. population than previous research on this topic.

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 twin pairs ascertained randomly via the telephone (for additional information on the sample or sampling process see Brim et al. [1996] 2003).

Twin pairs were diagnosed as identical (monozygotic) versus fraternal (dizygotic) with self-report data on whether they had (a) the same eye color, (b) natural hair color, (c) complexion, (d) whether they were mistaken for each other when they were young, and (e) 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 these specific questions and the points were subsequently totaled. High scores indicated identical twin pairs while low scores indicated fraternal twin pairs. Similar methods of diagnosing zygosity have been shown to be over 90% accurate (Lykken et al. 1990; Maes et al. 1999). The scores of 26 pairs fell directly in the middle of the range, making a definitive classification impossible. These pairs, along with all opposite-sex dizygotic twin siblings, were excluded from the present study. Therefore, the findings reported here are based on 193-316 pairs of monozygotic siblings (note: N varies depending upon the particular aspect of religion under investigation) and 176-278 pairs of same-sex dizygotic siblings.

Measures

The MIDUS data contains measures of four distinct aspects of religious life. *Organizational involvement* is gauged with a continuous measure of the frequency of attendance at religious services. Respondents were asked: "In a typical month, about how many times do you attend religious services?" A handful of respondents reported attending at very high rates (i.e., 20 or more times per month). Given that this seems improbable, if not impossible, responses above the 95th percentile are recoded to equal the mean. Ancillary analyses revealed that this did not alter the findings reported here. The range on the recoded variable is 0-11, and due to the skewed nature of this measure even after this recoding, a square root transformation is performed in order to normalize the distribution.

Four different measures are used to gauge *personal religiosity and spirituality*. Childhood religiosity is tapped with the following question (coded 1=not at all to 4=very much): "How important was religion in your home when you were growing up?" Religious salience is a 6-item summed index (Cronbach's alpha=0.88; each item is coded 1=not at all to 4=very). Specific items include: (a) "How religious are you?" (b) "How important is religion in your life?" (c) "How important is it for you—or would it be if you had children now—to send your children for religious or spiritual services for instruction?" (d) "How closely do you identify with being a member of your religion?" (e) "How much do you prefer to be with other people who are the same religion as you?" and (f) "How important do you think it is for people of your religion to marry people who are the same religion?" Spirituality is gauged with two items (alpha=0.91; each item is

coded 1=not at all to 4=very): (a) “How spiritual are you?” and (b) “How important is spirituality in your life?” Daily guidance and coping is tapped with two questions (alpha=0.85; each is coded 1=never to 4=often): (a) “When you have decisions to make in your daily life, how often do you ask yourself what your religious or spiritual beliefs suggest you should do?” and (b) “How often do you seek comfort through religious or spiritual means such as praying, meditating, attending a religious or spiritual service, or talking to a religious or spiritual advisor?”

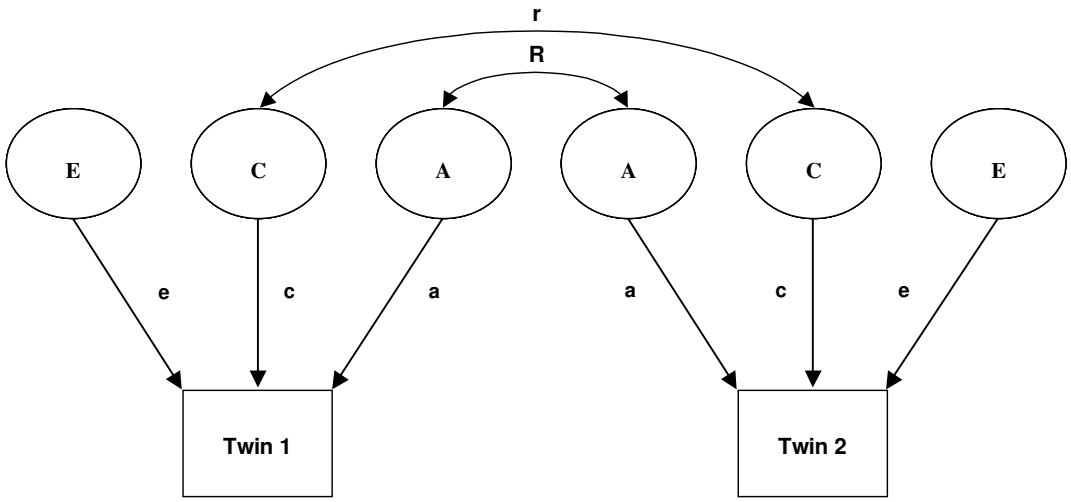
Conservative ideologies are gauged with two single-item measures. Biblical literalism, a commonly-employed measure of religious conservatism, is tapped with the following question: “How much do you agree or disagree with the following statement (coded 1=strongly disagree to 5=strongly agree): "The Bible is the actual Word of God and is to be taken literally, word for word?” Exclusivist beliefs are measured with the following question (coded 1=yes and 0=no): “Do you believe that one should: stick to one faith?” For this variable, respondents who answered “yes” are compared with those who believed that individuals should “explore different teachings,” do “neither” of these things, or “don’t know” the answer to this question.

Transformations and commitments are measured with the following question (coded 1=yes and 0=no): “Have you been ‘born-again,’ that is, had a turning point in your life when you committed yourself to Jesus Christ?”

Behavior Genetic Methods, Twin Studies, and Structural Equations Modeling (SEM)

This study employs a classical twin design. In analyses utilizing twin sibling data, one twin is correlated with their sibling separately for monozygotic and dizygotic twins. These intra-pair correlations are then compared across the two types of twins. If monozygotic twins are more highly correlated with (i.e., more similar to) each other than dizygotic twins, genetic influences are implied. The logic behind this rests on the assumption that no substantive differences exist in the environments of monozygotic versus dizygotic twins. Therefore, if monozygotic twins are more highly correlated with each other than dizygotic ones despite living in similar environments, shared genetic predispositions must be responsible. If the intra-pair correlations are similar for the two types of twins, shared environmental influences are implied. The logic behind this rests on the assumption that the only influence other than shared genes that is capable of producing similarities among twin siblings is shared environments or common experiences. Therefore, if both groups are similarly correlated, yet not that similar genetically, shared environmental influences must be contributing to twin similarities. If the correlations for both groups are relatively low, nonshared environmental influences are implied. The logic behind this rests on the assumption that unique experiences function to differentiate individuals. Overall, using these assumptions and correlations, along with the statistical techniques described below, it is possible to estimate the proportion of individual-level variation on any religious outcome that is due to genetic, shared environmental, and nonshared environmental influences.

Figure 1.1: Univariate Structural Equations Model



Notes: Circles represent latent (unmeasured) variables and squares represent manifest (measured) variables; A = additive genetic factor; C = shared environmental factor; E = nonshared environmental factor; R = genetic relatedness (1.0 for monozygotic twins and 0.5 for same-sex dizygotic twins); and r = twin sibling environmental exposure (1.0 for both types of twins).

Structural equations modeling (SEM) is the most precise and widely-employed method for estimating twin models. In the terminology of SEM, twin designs are two-population models composed of monozygotic and dizygotic pairs of twin siblings. In structural models like the one shown in Figure 1.1, the correlations between monozygotic siblings are fixed to 1.0 on the genetic factors because these siblings are, by definition, genetically identical (i.e., they are perfectly correlated with each other genetically). The correlations between dizygotic twin siblings, in contrast, are fixed to 0.5 on the genetic factors because they share, on average, half their genes. Shared environmental influences are contexts that twin siblings have in common regardless of zygosity, so these factors

are fixed to 1.0 for both types of twins. Nonshared environmental influences, on the other hand, are contexts that are unique to each twin of a pair, which means that there are no correlations between siblings on these factors for either monozygotic or dizygotic twins.

Using the publicly available SEM package Mx (Neale et al. 2003), the following three models are fit to the data (using maximum likelihood analysis of variance-covariance matrices) for each of the eight religion measures (separately): a full model that decomposes observed individual-level variation into latent genetic, shared environmental, and nonshared environmental components; a reduced model that drops the shared environmental component; and a reduced model that drops the genetic component. Estimating a nonshared environment-only model would assume that there is no family resemblance on these religious outcomes, and that twins are not correlated. This is obviously not the case, so this model is not estimated for any of the measures employed here. Additionally, models that do not contain a nonshared environmental component are not estimated because they would assume that that no differences exist between monozygotic twins (i.e., that monozygotic siblings are perfectly correlated with each other). This is also clearly false.

Model fit for each of the three models is assessed using the -2 log likelihood (-2LL), chi-square statistic and associated p-value, and Akaike's Information Criterion (AIC=χ^2-2*df*). To test for the statistical significance of the parameters in the full model (genetic and shared environmental influences only), both of the reduced models (i.e., genetic and nonshared environmental factors only, and shared and nonshared environmental factors only) are compared with the full model (separately) using likelihood ratio tests. Since the

resulting test is symtotically distributed as a chi-square, the change in fit between the models can be calculated by taking the difference in -2LL, with degrees of freedom being equal to the difference in the number of parameters estimated in the full versus reduced model. A non-significant change in chi-square test indicates that the reduced model is a better fit to the data, and that the dropped parameter (either the genetic or shared environmental factor) is therefore not statistically significant. Additionally, 95% confidence intervals for each of the three parameters in the full model are also computed as an alternate check of statistical significance.

To obtain proportional estimates of the effects of the three latent factors in the structural model (Figure 1.1), three steps are required. First, each of the path estimates (a, c, and e) is squared. Second, the three squared path estimates are then added together. And third, each individual squared path estimate is then divided by the sum-total of all three of the squared path estimates to obtain a proportional estimate for each path. Once calculated, these estimates provide information on the proportion of individual-level variation on any outcome that is due to genetic (A), shared environmental (C), and nonshared environmental (E) influences (for additional details on twin studies, structural equations modeling, fit statistics, or the ACE model, see: Cleveland and Crosnoe 2004; Neale and Cardon 1992; Neale et al. 2003; Plomin 1990).

Table 1.1: Descriptive Statistics for Four Dimensions (Eight Measures) of Religious Involvement

Religion Measures	N (Pairs)	Min-Max	Mean	St Dev	Covariance	Correlation
ORGANIZATIONAL INVOLVEMENT						
Religious or Spiritual Service Attendance	305 / 266	0-11	2.04 / 2.30	2.23 / 2.40	0.52 / 0.37	0.53 / 0.37
PERSONAL RELIGIOSITY AND SPIRITUALITY						
Childhood Religiosity	316 / 278	1-4	3.18 / 3.23	0.85 / 0.80	0.53 / 0.38	0.73 / 0.60
Religious Salience	306 / 255	6-24	16.69 / 17.17	4.70 / 4.13	14.00 / 7.19	0.63 / 0.42
Spirituality	305 / 261	2-8	6.15 / 6.36	1.65 / 1.52	1.39 / 0.73	0.51 / 0.32
Daily Guidance and Coping	310 / 270	2-8	5.52 / 5.64	2.10 / 2.13	2.57 / 1.64	0.58 / 0.37
CONSERVATIVE IDEOLOGIES						
Biblical Literalism	198 / 177	1-5	3.58 / 3.66	1.42 / 1.38	1.16 / 0.65	0.58 / 0.34
Exclusivist Beliefs	309 / 267	0-1	0.45 / 0.48	- / -	0.11 / 0.06	0.44 / 0.24
TRANSFORMATIONS AND COMMITMENT						
Born-Again / Religious or Spiritual Commitment	193 / 176	0-1	0.56 / 0.55	- / -	0.18 / 0.09	0.71 / 0.38

Notes: All notes apply to the entire column (all eight religion measures), not just religious or spiritual service attendance.

a N=The number of cases remaining after listwise deletion of missing values.

b Same for monozygotic and same-sex dizygotic twin pairs (larger numbers represent higher levels of religious involvement).

c Twin 1 versus twin 2 separately for monozygotic / same-sex dizygotic twin pairs (all correlations are significant at $p<0.001$ or less).

RESULTS

Table 1.1 displays descriptive statistics for each of the MIDUS religion measures (separately for monozygotic and same-sex dizygotic twins). From these results, we can see that very little variation exists between the two types of twins with respect to the means and standard deviations of these religious outcomes. The intra-pair correlations, however, reveal that monozygotic twin siblings are more highly correlated with (i.e., similar to) each other on all eight measures compared with dizygotic twin siblings. In some cases these differences are not extremely large, as is the case for childhood religiosity (0.73 and 0.60 for monozygotic and dizygotic twins, respectively), but for others such as being born-again or making a religious or spiritual commitment, the difference is quite large (0.71 and 0.38 for monozygotic and dizygotic twins, respectively). As described above, larger correlations for monozygotic siblings compared with dizygotic ones suggests that genetic factors may be important, while comparable correlations hint at the importance of shared environmental influences. Thus, these findings preliminarily suggest that genetic factors likely contribute to individual-level variation on all eight religious outcomes examined here. (Note: The correlations for the two dichotomous variables—exclusivist beliefs and being born-again or making a religious or spiritual commitment—are reported as Pearson's correlation coefficients instead of the more appropriate tetrachoric correlations for two reasons: simplicity, and to be consistent with the other measures. The substantive findings reported here, and below, are virtually identical, however, regardless of the estimation procedure used.)

Table 1.2: Goodness-of-Fit-Criterion for Structual Equations Models Fit to Covariance Matrices for Four Dimensions (Eight Measures) of Religious Involvement

Religion Measures	Chi-Square	p-value (Chi-Square)	df (Chi-Square)	AIC
ORGANIZATIONAL INVOLVEMENT				
Religious or Spiritual Service Attendance	0.04 / 3.72 / 6.86	1.00 / 0.45 / 0.14	3 / 4 / 4	-5.96 / -4.28 / -1.14
PERSONAL RELIGIOSITY AND SPIRITUALITY				
Childhood Religiosity	4.52 / 37.66 / 10.64	0.21 / 0.00 / 0.03	3 / 4 / 4	-1.49 / 29.66 / 2.64
Religious Salience	10.21 / 18.43 / 16.41	0.02 / 0.00 / 0.00	3 / 4 / 4	4.21 / 10.43 / 8.41
Spirituality	5.95 / 8.58 / 10.83	0.11 / 0.07 / 0.03	3 / 4 / 4	-0.05 / 0.06 / 2.83
Daily Guidance and Coping	1.11 / 3.36 / 14.30	0.78 / 0.50 / 0.01	3 / 4 / 4	-4.90 / -4.64 / 6.30
CONSERVATIVE IDEOLOGIES				
Biblical Literalism	0.43 / 1.32 / 9.14	0.94 / 0.86 / 0.06	3 / 4 / 4	-5.57 / -6.68 / 1.14
Exclusivist Beliefs	0.05 / 0.16 / 8.71	1.00 / 1.00 / 0.07	3 / 4 / 4	-5.95 / -7.84 / 0.71
TRANSFORMATIONS AND COMMITMENT				
Born-Again / Religious or Spiritual Commitment	0.03 / 0.21 / 27.71	1.00 / 1.00 / 0.00	3 / 4 / 4	-5.97 / -7.79 / 19.71

Notes: Insignificant p-values ($p>0.05$) and low AIC (relative to other models) indicate acceptable model fit. Cell entries are: full model (containing genetic, shared environmental, and nonshared environmental factors) / reduced model (shared environmental factor dropped) / reduced model (genetic factor dropped).

Turning now to the SEM results, Table 1.2 shows goodness-of-fit criterion for each of the three models discussed above (one full and two reduced) for each of the eight religion measures separately. According to the χ^2 p-values and AIC statistics, models containing a genetic component (columns 1 and 2 under each subheading) fit better for each of the measures than models without this component (column 3 under each subheading). For religious or spiritual service attendance, a reduced model that includes only shared and nonshared environmental influences also fits the data well, but for even this religious outcome, taking genetic effects into consideration adds to the overall model fit. For each of the remaining seven measures of religion, however, a genetic factor is required to adequately explain the data.

That said, overall model fit statistics such as χ^2 and AIC can sometimes be misleading with respect to the importance of particular parameters in the model (especially for simple models like the ones estimated here), and dropping parameters (whether significant or not) can lead to biased estimates on the remaining parameters (Edwin et al. 2000). Therefore, as an additional check of statistical significance, 95% confidence intervals are estimated for each of the three parameters in the full model. Statistically significant paths—i.e., those where the confidence intervals do not contain zero—are indicated by stars in Table 1.3, which shows proportional genetic, shared environmental, and nonshared environmental influences on religious variables.

Table 1.3: Proportional Genetic, Shared Environmental, and Nonshared Environmental Influences on Individual Variation for Four Dimensions (Eight Measures) of Religious Involvement

Religion Measures	Proportional Genetic Influences	Proportional Shared Environmental Influences	Proportional Nonshared Environmental Influences
ORGANIZATIONAL INVOLVEMENT			
Religious or Spiritual Service Attendance	0.32 *	0.21 a	0.47 *
PERSONAL RELIGIOSITY AND SPIRITUALITY			
Childhood Religiosity	0.19 *	0.53 *	0.28 *
Religious Salience	0.27 *	0.33 *	0.40 *
Spirituality	0.29 *	0.20	0.51 *
Daily Guidance and Coping	0.42 *	0.16	0.42 *
CONSERVATIVE IDEOLOGIES			
Biblical Literalism	0.44 *	0.13	0.43 *
Exclusivist Beliefs	0.41 *	0.04	0.55 *
TRANSFORMATIONS AND COMMITMENT			
Born-Again / Religious or Spiritual Commitment	0.65 *	0.06	0.29 *

*p<0.05 (Based on 95% Confidence Intervals)

a p<0.06

From this table we can see that proportional genetic effects on individual religious variation range from 19% for childhood religiosity, to 65% for being born-again or making a religious or spiritual commitment. For all eight measures, the confidence intervals reveal that the genetic factor is statistically significant at $p<0.05$ or less. Genetic influences are sizable for several commonly employed measures of religion including religious or spiritual service attendance (32%), religious salience (27%), spirituality (29%), daily guidance and coping (42%), biblical literalism (44%), exclusivist beliefs (41%), and being born-again or making a religious or spiritual commitment (65%). The last finding, which shows a proportional genetic effect that is considerably larger than it is for any of the other measures, is particularly noteworthy.

With respect to shared environmental influences—i.e., family-level socialization effects, which represent the only source of similarity among twin siblings except for genetic influences—the effects shown in Table 1.3 range from 4% for exclusivist beliefs, to 53% for childhood religiosity. Confidence intervals indicate that the estimates for religious or spiritual service attendance (21%; $p<0.06$), childhood religiosity (53%), and religious salience (33%), are statistically significant at $p<0.05$ or less, while the ones for spirituality, daily guidance and coping, biblical literalism, exclusivist beliefs, and being born-again or making a religious or spiritual commitment, are not.

Nonshared environmental influences, which function to differentiate twin siblings on religious outcomes, range from 28% for childhood religiosity, to 55% for exclusivist beliefs. Importantly, this factor is statistically significant ($p<0.05$ or less) for all eight outcomes. These numbers represent the proportion of the individual-level variation that cannot be explained by either genetic or family-level socializing forces. Nonshared environmental influences are almost always statistically significant in twin models—as they are here—because siblings rarely, if ever, display identical characteristics on any outcome. Unfortunately, these estimates also contain measurement error, which may be relatively large in the present study due to the utilization of several single-item measures of religious involvement.

Overall, these findings demonstrate the utility of a behavior genetic approach for both biology-based and social scientific research alike. The aspects of religion that all owe a considerable proportion of their individual variation to genetic effects—i.e., spirituality, daily guidance and coping, biblical literalism, exclusivist beliefs, and being born-again or

making a religious or spiritual commitment)—are the ones that should be expected to be more internally motivated, and thus less responsive to social influences or constraints. In contrast, shared environmental influences are important for religious or spiritual service attendance, childhood religiosity, and religious salience: aspects of religious life that are obviously subject to powerful social-environmental pressures. These findings are intuitive, and their ramifications are discussed in detail below.

DISCUSSION

This study began by asking: Is individual-level religious variation the product of both genetic and social-environmental influences, and if so, what is the relative contribution of each? An analysis of data from a national sample of adult twin siblings strongly suggests that the answer is yes, and that the proportion of each varies depending upon the particular aspect of religious life under investigation. Specifically, genetic effects appear to explain large proportions of the variation on several religious outcomes including daily guidance and coping (42%), conservative religious ideologies (44% and 41%, respectively, for biblical literalism and exclusivist beliefs), and religious transformations such as being born-again or making a religious or spiritual commitment (65%). Proportional genetic influences are also sizable for measures of religious or spiritual service attendance (32%), religious salience (27%), and spirituality (29%). Shared environmental influences, or family-level social effects, appear to be important for several outcomes as well, explaining 21, 53, and 33% of the variation, respectively, on religious or spiritual service attendance, childhood religiosity, and religious salience.

Nonshared environmental influences also account for considerable proportions, ranging from 28% of the variation on childhood religiosity, to 55% on exclusivist beliefs. In sum, statistically significant genetic and environmental effects were found for all eight of the outcomes examined here. Given these findings, the next question is: What does it mean to say that genetic factors, in addition to environmental contexts, influence religious life?

One useful way to think about, and interpret, the findings reported here is to frame them in terms of individual free-will versus social constraint. To begin with, genetic influences on "social" outcomes such as religious involvement (as tapped by twin studies) likely represent the manifestation of gene-based, innate predispositions, needs, wants, desires, or motivations, while shared and nonshared environmental influences broadly tap family-level social influences and wider social contexts, respectively. Here, the present study draws on the work of Udry (1996; see also Guo and Stearns 2002; Jencks 1980), who suggested: (a) the higher the level of social constraints experienced by individuals in an environment, the more variance is likely controlled by social forces; and (b) conversely, the more opportunities to exercise free-will an individual has, the more variance is likely controlled by genetic factors. In other words, proportionally large genetic effects reflect the relative weakness of social-environmental influences, whereas smaller genetic effects imply that social influences are particularly powerful. Thus, when behavior genetic techniques are employed to analyze social outcomes such as religion, the genetic component is not seen as "determining" outcomes, but instead appears to tap the ability to think or behave in accordance with one's internal, biological motivations.

When this interpretive framework is applied to the findings reported above, the results are supportive. For instance, social scientific theory predicts that religious practices such as service attendance are subject to social influences or constraints to a much greater degree than are other, more private, aspects of religion such as beliefs or experiences. In essence, individuals may alter their outward practices (e.g., attendance at services) to accommodate the wishes of others in part because these expressions of conformity can be observed and monitored (Sherkat 1997), while they may be less prone to adopt particular religious beliefs, to draw on religious resources in their everyday lives, to experience religious transformations, or to make religious commitments, based on external social pressures. These latter aspects of religious life may flow, instead, from internal motivations or desires to a larger degree. The findings presented above support this idea by showing that proportional genetic influences are relatively smaller (and thus social-environmental ones are comparably larger) on religious attendance (32%), while the inverse is true for conservative beliefs (41-44%), religious coping strategies (42%), and transformation and commitments (65%).

Behavior genetic techniques and twin models like the ones employed here are also useful for understanding family-level socialization. Shared environmental influences, as previously discussed, are social processes that function to make individuals who share them similar to each other. In essence, these are the microcontexts in which theories of social learning argue that the majority of socialization and reinforcement occurs (Carson and Rothstein 1999; D'Onofrio et al. 1999a). Although behavior genetic research has seldom found that these environments explain much of the individual variation on diverse

attitudes and behaviors (Plomin 1990), they do appear to be important for certain facets of religion, particularly organizational forms such as religious attendance. In the present study, family-level socialization arguments were partially supported—i.e., shared environmental influences were found to explain 21, 53, and 33% of the individual variation, respectively, on religious or spiritual service attendance, childhood religiosity, and religious salience. For these three outcomes, family contexts are obviously important, but for the other aspects of religion examined here, little or no evidence of family-based socialization effects was found. It should be noted, however, that these findings are based on a sample of adults, and that shared environmental influences tend to become less important (and thus genetic ones more important) as individuals progress through the life course. It is therefore likely that shared environmental influences are relatively more important during the earlier stages of life (i.e., childhood and adolescence), while genetic factors become more influential in adulthood and old age (Eaves et al. 2008).

Although the present study nicely supplements existing social scientific research on religion (i.e., by examining free-will and family socialization effects), it also highlights two potential implications for social scientific approaches. First, it is likely that any model of religion that does not consider all three of the components discussed here—genetic, shared environmental, and nonshared environmental influences—will fail to explain a large proportion of the individual-level variation on religious practices, salience, beliefs, and experiences. This assertion is based on the fact that each of the eight aspects of religion examined here appears to be influenced by both genetic and environmental factors. This suggests that traditional sociological models of religious life,

which focus exclusively on the latter, may suffer from explanatory power deficiencies, especially when trying to predict individual-level variation on the more internally-motivated (i.e., less social) aspects of religious participation such as beliefs, transformations, and commitments.

Second, and perhaps equally important, it is possible that genetic factors may be biasing the findings of studies that employ religious variables as predictors of other outcomes (Bouchard and Loehlin 2001; Udry 1995). For example, some scientists (e.g., Sloan, Bagiella, and Powell 1999) have voiced concern that findings showing a connection between religion and health (see Ellison and Levin 1998 for a review) may be spurious due to the lack of control of important covariates, including potential genetic confounders. Currently, we are unable to definitively establish whether or not religion is a "cause" of other individual and social outcomes, or whether it is simply a "marker" that is tapping some unobserved set of influences, possibly including genetic factors. As is the case with explanatory power deficiencies, this potential challenge to social scientific research varies depending upon which facets of religion are under investigation—i.e., this may be a bigger issue for aspects of religious life such as beliefs and commitments that appear to be strongly influenced by genetic factors, compared with affiliation and organization-based activities, which are influenced by genetic differences to a smaller degree. Important topics of scholarship that could be vulnerable to this concern include the relationships between religious involvement and prosocial attitudes and behaviors, family relations, health and well-being, civic engagement, crime and delinquency, and socioeconomic attainment, among others. In order for the growing corpus of literature in

these areas to be accepted by important segments of the larger scientific community, researchers will eventually need to show that these associations are not the spurious results of unobserved genetic predispositions. Efforts to address this issue on a large scale will require additional data collection projects that take both religious and potential genetic influences seriously.

Although informative, the present study is unable to explain *why* and *how* genetic factors influence religious outcomes. Future research should begin addressing these issues by identifying the pathways by which genetic influences forge their way into religious life. One way to achieve this goal is to examine potential mediators—i.e., influences that intervene between genetic predispositions and religious outcomes (Jang et al. 2001; Plomin 1990; Scarr and McCartney 1983). For example, it is likely that genetic effects on social outcomes such as religious involvement are indirect via a whole host of personality and interpersonal characteristics including extraversion, agreeableness, openness to experience, and the ability to form and maintain positive relationships with others, to name just a few (Alford, Funk, and Hibbing 2005; Bradshaw 2007). This contention is based on research showing (a) that genetic factors are significant predictors of individual variation on all personality traits, and (b) that these characteristics are, in turn, correlated with religious outcomes (Bouchard and Loehlin 2001; Bouchard et al. 1999; James 1997; Jang, Livesley, and Vernon 1996; Kirkpatrick 1999; Saroglou 2002). In other words, it is likely that genetic effects at least partially function via the shaping of individuals differences in personality, which then subsequently affect religious life.

To conclude, individual-level variation on a diverse assortment of religious outcomes—i.e., service attendance, childhood religiosity, religious salience, spirituality, daily guidance and coping, biblical literalism, exclusivist beliefs, and being born-again or making a religious or spiritual commitment—appears to be the product of both genetic and social-environmental influences. These findings, which are in contrast to most of the published research on religious participation, as well as social scientific theory in general, demonstrate the need to begin integrating genetic and other biological factors into existing theoretical and empirical models of religious life. Behavior genetics, the approach employed here, is capable of doing this, and thus offers the potential to shed new light not only on potential genetic influences, but also on the arguments social scientific theory by addressing the longstanding question: To what extent are innate motivations (i.e., free-will) versus social influences or constraints responsible for the observed variation on religious outcomes? The social scientific study of religion has either much to gain by capitalizing on the strengths of this approach, or much to lose by ignoring them. The next step forward in the multidisciplinary struggle to understand religious phenomena will undoubtedly involve research on the role of genetic and other biological influences, and social scientists should therefore seek to become more involved in this area of scholarship.

CHAPTER 2

Do Genetic Factors Influence Civic Engagement: If So, Are They Explained by Correlations with Personality and Interpersonal Characteristics?

SUMMARY

Previous research on the causes of civic engagement has focused almost exclusively on social-environmental influences. A handful of recent studies, however, have suggested that genetic or other biological factors may be important as well. This chapter, which utilizes twin sibling data from the National Survey of Midlife Development in the United States (MIDUS), extends our knowledge in this area by: (a) providing evidence that genetic factors may indeed influence individual-level variation on civic-oriented attitudes and behaviors (in response to Research Question 1); (b) showing that at least part of these genetic effects can be explained by genetic-level correlations with social-oriented personality and interpersonal characteristics (i.e., sociality) (in response to Research Question 2); and (c) empirically examining the equal environments assumption of twin studies. It is concluded that in order to fully understand civic engagement, scholars must begin to examine both genetic and environmental influences, and that the failure to address the former may result in explanatory power deficiencies and potentially biased empirical relationships.

INTRODUCTION

A sizable literature has examined the causes of civic-oriented attitudes and behaviors such as voting, keeping informed about social and political issues, the willingness to testify in court, and volunteering for community organizations, among others (Bekkers 2005; Beyerlein and Hipp 2006; Campbell 2006; Clary and Snyder 1999; Galston 2001; Putnam 2000; Theiss-Morse and Hibbing 2004; Wilson and Musick 1997; Ziemek 2006). Even though virtually all of this research has focused on social-environmental influences, a handful of recent studies have actually suggested that genetic or other biological factors may be important as well (Alford, Funk, and Hibbing 2005; Alford and Hibbing 2004; Hibbing and Alford 2004; Masters 2001; Wilson 2006). If true, this could pose profound implications—in the form of explanatory power deficiencies and possibly even genetically confounded relationships, among others—for research on civic engagement that does not take these latent influences into consideration.

The present study extends our knowledge in this area in three ways. First, it provides evidence that genetic influences may indeed be important predictors of individual-level variation on two different aspects of civic life: (a) feelings of obligation toward civic participation; and (b) the frequency of volunteering in community settings. Previous research has analyzed social and political orientations from a genetically-informed perspective, but no studies to date have examined either of these two aspects of civic engagement. Second, this study offers a potential explanation for genetic effects on civic engagement: genetic-level correlations with several measures of sociality (i.e., the tendency to associate with others and to form social groups). Specifically, it examines

whether genetic effects on civic obligations and volunteering are correlated with genetic predispositions toward three different indicators of sociality: an extraverted personality, positive relations with others, and community attachment. And third, the present study empirically examines the equal environments assumption. Twin studies like this one are based on the assumption that monozygotic (genetically identical) and dizygotic (fraternal / genetically 50% identical, on average) twin siblings experience substantively similar environments. Some have argued that monozygotic (MZ) siblings are treated more similarly and have closer relationships with each other than dizygotic (DZ) siblings, and that this biases the estimates of twin studies. This possibility will be examined here for civic engagement.

These three tasks will be achieved by first reviewing the relevant literature. Based on this theoretical and empirical background, several hypotheses will be formulated, and then empirically tested using data from the National Survey of Midlife Development in the United States (MIDUS), a national sample of working-age twin siblings. A summary of the results will then be presented, and a discussion of their relevance for research in this area will be provided. An agenda for future research will then be outlined.

THE SOCIAL (AND GENETIC?) CAUSES OF CIVIC ENGAGEMENT

In modern, industrialized societies like the United States, civic engagement is central to good citizenship and the maintenance of democracy (Bekkers 2005; Galston 2001; Putnam 2000; Theiss-Morse and Hibbing 2005). The famous words of Alexis de Tocqueville (1956: 201) convey this message nicely:

> These Americans are the most peculiar people in the world. You'll not believe it when I tell you how they behave. In a local community in their country, a citizen may conceive of some need that is not being met. What does he do? He goes across the street and discusses it with his neighbor. Then what happens? A committee begins functioning on behalf of that need. All of this is done by private citizens on their own initiative. The health of a democratic society may be measured by the quality of functions performed by private citizens.

In essence, Western-style democracies are built upon the expectation that individuals will be intimately involved in public life, and that they will perform certain duties such as serving on juries, testifying in court, keeping informed about social and political issues, voting in elections, and volunteering their time and energy to community causes, among many others. Without the unpaid support of large numbers of citizens, there can be little doubt that democratic societies would cease to function properly.

While these facts are not in dispute, the causes of civic engagement are not completely understood. Given that this participation is rarely financially rewarding—in fact, it actually involves costs in terms of both time and money—free-riding should be widespread, and apathy should be the norm. The truth is, however, that many individuals are actively involved in civic life, which begs the question: Why?

Drawing on diverse theoretical perspectives including symbolic interaction theory, role theory, social learning theory, rational choice and social exchange theory, conflict

theory, social capital theory, and cultural theory, among others, virtually all social scientists, from Tocqueville on, have assumed that individuals engage in civic activities because they are socialized, in one way or another, into doing so (Campbell 2006; Clary and Snyder 1999, 1991; Galston 2001; Hodgkinson 1995; Putnam 2000; Smith 2003; Sundeen and Raskoff 1994; Uslaner 2002; Wilson and Janoski 1995; Wilson and Musick 1997; Wuthnow 1990). In the words of Galston (2001: 217): "…it is reasonably clear that good citizens are made, not born." In support of this claim, he reviews research linking civic engagement with numerous social influences including residential stability, home ownership, professional interests, marriage, parenthood, socioeconomic status, and feelings of community, to name just a few. Similarly, other social influences such as a historical heritage of land ownership and profit-seeking, population density and heterogeneity, religious involvement, political values and behaviors, and media consumption, among others, also appear to be important (Bekkers 2005; Beyerlein and Hipp 2006; Campbell 2006; Wilson and Janoski 1995; Wilson and Musick 1997).

Although sociological approaches such as these currently dominate the scientific discourse in this area, several recent studies have actually challenged the assumption that social influences account for most, if not all, of the variation on civic-oriented attitudes and behaviors by suggesting that genetic factors might be important as well (Hibbing and Alford 2004; Masters 2001; Wilson 2006). For example, a recent study by Hatemi and colleagues (2007) found that genetic factors accounted for 24 percent of the individual variation on voting preferences. Another relevant example comes from Alford, Funk, and Hibbing (2005), who found that genetic influences explained 43 percent of the

individual-level variation on a 28-item index of political orientations (i.e., conservative versus liberal ideologies). Social-environmental influences were found to account for the remaining 57 percent of the variation, with shared environmental influences (i.e., family-level social influences) explaining 22 percent, and nonshared environmental influences (i.e., social influences that twin siblings do not have in common) accounting for 35 percent (note: detailed information on twins studies will be provided below). Other scholars have found similar results for related outcomes and relevant personality characteristics including fundamentalism, orthodoxy, and openness to experience (Beer et al. 1998; Bouchard et al. 1990; Eaves, Eysenck, and Martin 1989; Martin et al. 1986). One study has even shown that genetic factors influence attachments to others in one's community (Bradshaw 2007).

Even though research of this type has analyzed social and political orientations from a genetically-informed perspective, general feelings of obligation toward civic participation, as well as the actual frequency of volunteering in one's community, have been completely neglected. The present study addresses this weakness in the literature by examining whether both genetic and environmental factors influence these two aspects of civic life. It is therefore hypothesized that:

- Proposition 1: Individual-level variation on obligations toward civic participation and the frequency of volunteering will be the product of both genetic and environmental influences.

EXPLAINING GENETIC INFLUENCES ON CIVIC ENGAGEMENT

Even among scholars who may acknowledge a role for genetic influences on civic-oriented outcomes, the findings of univariate twin studies (or other genetically-informed designs) may be theoretically ambiguous, and thus may actually raise more questions than they actually answer. In other words, when research finds that genes matter, readers are routinely left to ponder what it means to say that they matter (i.e., Why and how do they influence a particular outcome?). Unlike genetic influences on traits such as height, hair color, certain types of diabetes, and even severe forms of psychopathology such as schizophrenia, which may correctly be conceived of as hardwired effects, genetic influences on civic engagement almost certainly take the form of innate predispositions or tendencies (as opposed to deterministic effects), either toward, or away from, social and public life. Based on this assumption, an interesting possibility emerges.

Although no research to date has empirically examined this possibility, several scholars (e.g., Alford, Funk, and Hibbing 2005; Alford and Hibbing 2004) have suggested that genetic influences on one or more latent personality characteristics may indirectly influence contemporary social and political attitudes and behaviors. This argument is based on (a) the fact that virtually all personality traits, including those related to civic life—e.g., extraversion, the ability to form and maintain positive relations with others, attachments to others, etc.—are strongly influenced by genetic factors (Bouchard and Loehlin 2001), and (b) that these characteristics are correlated with civic engagement (Bekkers 2005). For the purposes of the present study, characteristics such as

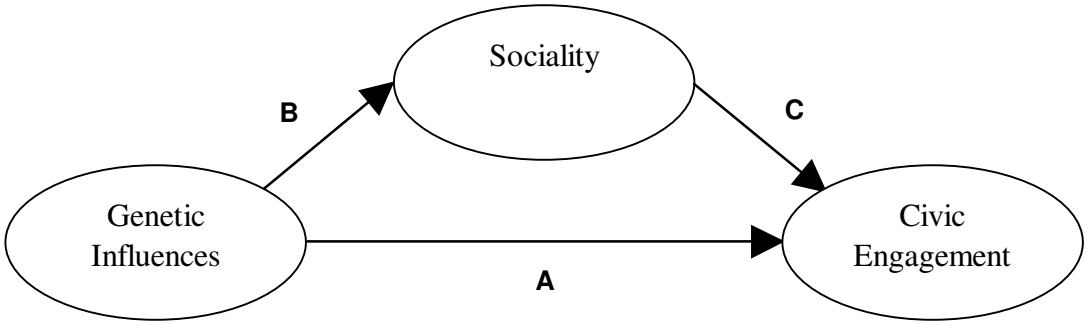
these are broadly referred to as sociality. Let us take a closer look at the genetically-informed research on this topic.

Utilizing both twin and adoption designs, researchers have shown that genetic influences are important predictors of an extraverted personality, one particular aspect of sociality (Baker and Daniels 1990; Bouchard and Loehlin 2001; Eaves and Eysenck 1975). A study based on data from MZ and DZ twin siblings reared apart has even been published, and it found substantial genetic effects on extraversion as well (Pedersen et al. 1988). Research also suggests that other dimensions of sociality, such as the ability to form and maintain positive relations with others, may also be strongly influenced by genetic or other biological factors (Boorman and Levitt 1980). Specifically, the literature shows that genetic differences predict individual-level variation on social closeness and intimacy, openness, agreeableness, dependability, cooperativeness, social integration, altruism, interpersonal trust, and the adherence to social rules and authority, among others (Axelrod and Hamilton 1981; Bouchard and Loehlin 2001; Kenrick, Li, and Butner 2003; Kirkpatrick 2004; Newberg, D'Aquili, and Rause 2002; Plomin 1990). Research even suggests that genetic factors influence social network closure (Cleveland and Crosnoe 2004), social support (Kendler 1997), and even marital quality (Spotts et al. 2004).

In addition to being influenced by genetic factors, for social-oriented personality and interpersonal characteristics to mediate genetic effects on civic engagement, these outcomes must actually be correlated with civic attitudes and behaviors. Research has indeed linked traits such as openness, conscientiousness, extraversion, and empathy with

volunteering for civic organizations, and conscientiousness and empathy with voluntary association membership (Bekkers 2005). In addition, individuals who are intimately connected to others in their community, who have high levels of interpersonal trust, and who do not perceive of others as threatening, are generally more involved in civic life than individuals without these characteristics (Putnam, Leonardi, and Nanetti 1993; Sullivan and Transue 1999). It has even been shown that social connections, orientations, and compassion facilitate involvement in civic society (Lerner 2004).

Figure 2.1: A Conceptual Model of the Potential Relationships Between Genetic Influences, Sociality, and Civic Engagement



Given these findings, it is therefore possible that genetic effects on personality traits and interpersonal characteristics (i.e., sociality) may mediate genetic influences on civic engagement. In other words, genetic effects on contemporary beliefs and behaviors may be the indirect "byproducts" of correlations between them and one or more broad-based psychosocial orientations (Alford, Funk, and Hibbing 2005; Alford and Hibbing 2004; Kirkpatrick 2004). Shown diagrammatically in Figure 2.1, Pathway A represents direct

genetic effects on civic engagement, as theorized in the previous section. Pathway B represents the argument that sociality is at least partially a gene-based trait. Pathway C represents a traditional sociological model linking sociality with civic engagement that does not take genetic effects into consideration. Tracing Pathways B and C represents the model outlined in this section, which predicts that genetic influences on civic engagement are at least partially mediated by sociality. If this model is correct, taking the [genetic] relationship between civic engagement and sociality into consideration will reduce, and possibly even eliminate, direct genetic effects on the former. Stated in proposition form:

- Proposition 2: Genetic influences on individual-level variation on obligations toward civic participation and the frequency of volunteering will be at least partially mediated by (i.e., correlated with) genetic influences on sociality.

METHODS

Twin Studies and the Equal Environments Assumption

To examine genetic influences, scholars commonly employ twin designs. When utilizing this approach, one twin is correlated with their sibling separately for MZ and DZ twins. These "intra-pair correlations" are then compared across the two types of twins in an attempt to disentangle proportional genetic and environmental influences. Briefly, if MZ twins are more highly correlated with each other than DZ twins, genetic influences are implied. The logic behind this rests on the assumption that no substantive differences exist in the environments of MZ versus DZ twins. If both groups are not similarly

correlated, yet live in (or came from) similar environments, something besides shared environments must be making MZ twins more similar than DZ twins—i.e., shared genes. This is the so-called "equal environments assumption." If the correlations between siblings are similar across the two types of twins, shared environmental influences are implied. The logic behind this rests on the assumption that the only influence other than shared genes that is capable of producing similarities among siblings is shared environments or common social experiences. If both groups are similarly correlated, yet not similar genetically, something besides shared genes must be making DZ twins as similar as MZ twins—i.e., shared environmental influences. If the correlations for both types of twins are relatively low, nonshared environmental influences are implied. The logic behind this rests on the assumption that nonshared environments or unique social experiences function to differentiate siblings, including genetically identical ones (for additional information on twin designs, as well as their strengths and weakness, see Alford, Funk, and Hibbing 2005; Neale and Cardon 1992; Plomin 1990).

The most controversial portion of this framework is the equal environments assumption. If MZ twin siblings experience environments that are substantively different from DZ siblings, the greater similarity among the former—a finding that is typically interpreted as evidence for genetic effects—could actually be due to environmental differences. In the present study, this assumption could be violated if MZ siblings have more contact with, or have closer attachments to, each other compared with DZ twins in a manner that leads them to have more similar levels of civic engagement. This possibility will be empirically examined here. It is important to realize that even though this

assumption is made in all twin studies, empirical tests of it must be conducted separately for all different outcomes. Research has found it to be valid for personality (Borkenau et al. 2002; Loehlin and Nichols 1976), psychopathology (Cronk et al. 2002), physical health (Zondervan et al. 2005), eating disorders (Klump et al. 2000), and sexual orientation (Kendler et al. 2000), but not studies to date have examined it for civic engagement.

Data

To test the two hypotheses outlined above, 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), will be analyzed. This data was collected 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 MZ versus 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. These pairs, along with 263 pairs of opposite-sex DZ twin siblings, are excluded from the present study.

This particular data source was selected for the following reasons. First, it contains good measures of civic obligations, voluntary activities, twin sibling environmental similarity (i.e., equal environments), and sociality. Second, it contains data on adults, which is especially important since theory suggests that genetic influences should manifest themselves more strongly among adults because they have more freedom to make choices compared to children and adolescents, who have less (D'Onofrio et al.

1999; Plomin 1990). And third, it is a large national sample of twin siblings, which is capable of supporting complex statistical analyses.

Measures

This data contains five questions gauging *civic obligations* (i.e., obligations toward civic engagement). Respondents were asked to indicate: "How much obligation they would feel to: (a) serve on a jury if called; (b) keep fully informed about national news and public issues; (c) testify in court about an accident they witnessed; (d) vote in local and national elections; and (e) volunteer time or money to social causes they support?" These questions, each of which is coded 0 = none to 10 = very great, are combined into a five-item mean index (Cronbach's alpha = 0.79) referred to as civic obligations (Rossi 2004). Due to the negatively skewed nature of this measure, a squared version of it is employed in all analyses.

Frequency of volunteering, a measure of civic practices, is gauged with four items pertaining to the respondents' time spent doing volunteer work in their community. Specific items include the average time per month spent doing formal volunteer work: (a) in a hospital, nursing home, or other health-care-oriented facility; (b) in a school or other youth-related organization; (c) for a political organization or cause; and (d) for any other organization, cause, or charity. These four items are used to construct a summed index of the frequency of volunteering.

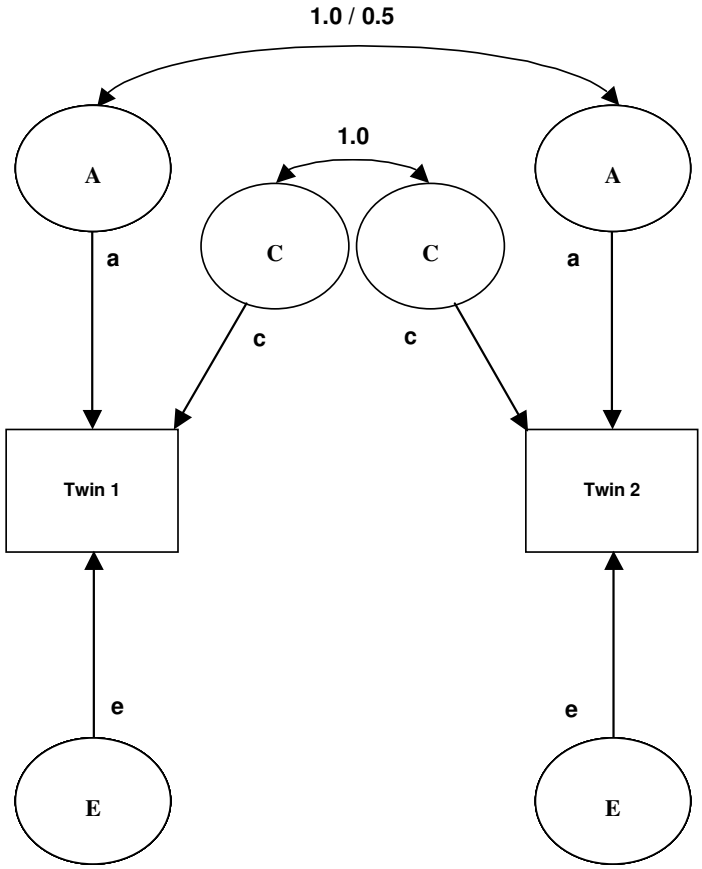
Since *sociality* is defined as (a) the "tendency" to associate with others and (b) to "form" social groups, three different measures of this multidimensional construct are

employed: an extraverted personality taps the innate desire (i.e., tendency) for social interaction or contact, while positive relations with others and community attachment tap the material manifestation (i.e., formation) of this tendency. To gauge extraversion, respondents were asked to indicate how well each of the following described them: outgoing, friendly, lively, active, and talkative (Lachman and Weaver 1997). A mean index (alpha = 0.79) is constructed from these five items (Lachman and Weaver 1997), all of which are coded 1 = not a lot to 4 = a lot. Positive relations with others is gauged with a three-item mean index constructed from the following questions (alpha = 0.62) (Ryff 1989; Ryff and Keyes 1995): (a)"Maintaining close relationships has been difficult and frustrating for me (reverse coded)." (b) "People would describe me as a giving person, willing to share my time with others." and (c) "I have not experienced many warm and trusting relationships with others (reverse coded)." Each of these measures is coded 1 = disagree strongly to 7 = agree strongly. Community attachment is a three-item mean index (alpha = 0.74) (Keyes 1998). This measure, which gauges the respondent's level of integration into (or attachment to) their community, is constructed from the following questions, each of which is coded 1 = disagree strongly to 7 = agree strongly: (a) "I don't feel I belong to anything I'd call a community (reverse coded)." (b) "I feel close to other people in my community." and (c) "My community is a source of comfort."

The MIDUS data contains five single-item questions that can be used to empirically examine the *equal environments assumption* of twin studies—i.e., the assumption that environmental measures related to civic engagement are perfectly correlated for MZ versus DZ twins (more information on this assumption is provided below). Three items

tap childhood relations: “When you were children…(a) how often did you and your twin have the same playmates? (b) how often were you in the same classrooms at school? and (c) how often did you dress alike?.” Each of these is coded 1 = never to 4 = always. Two items tap current relations: (a) “How often do you and your twin see each other?” and (b) “Not including visits, how frequently are you in contact with your twin—including phone calls, letters, or electronic mail messages?.” Both are coded 1 = less than once a week to 2 = once a week or more. Each of these questions is examined both as single-item measures, and as two different indices: one tapping childhood environments (3 items), and another gauging the frequency of adult contact (2 items). In addition, *educational attainment* (coded in years), which is strongly influenced by family environments, is included as an additional measure of the importance of family environments.

Figure 2.2:
Univariate Structural Equations Model for Twin Data

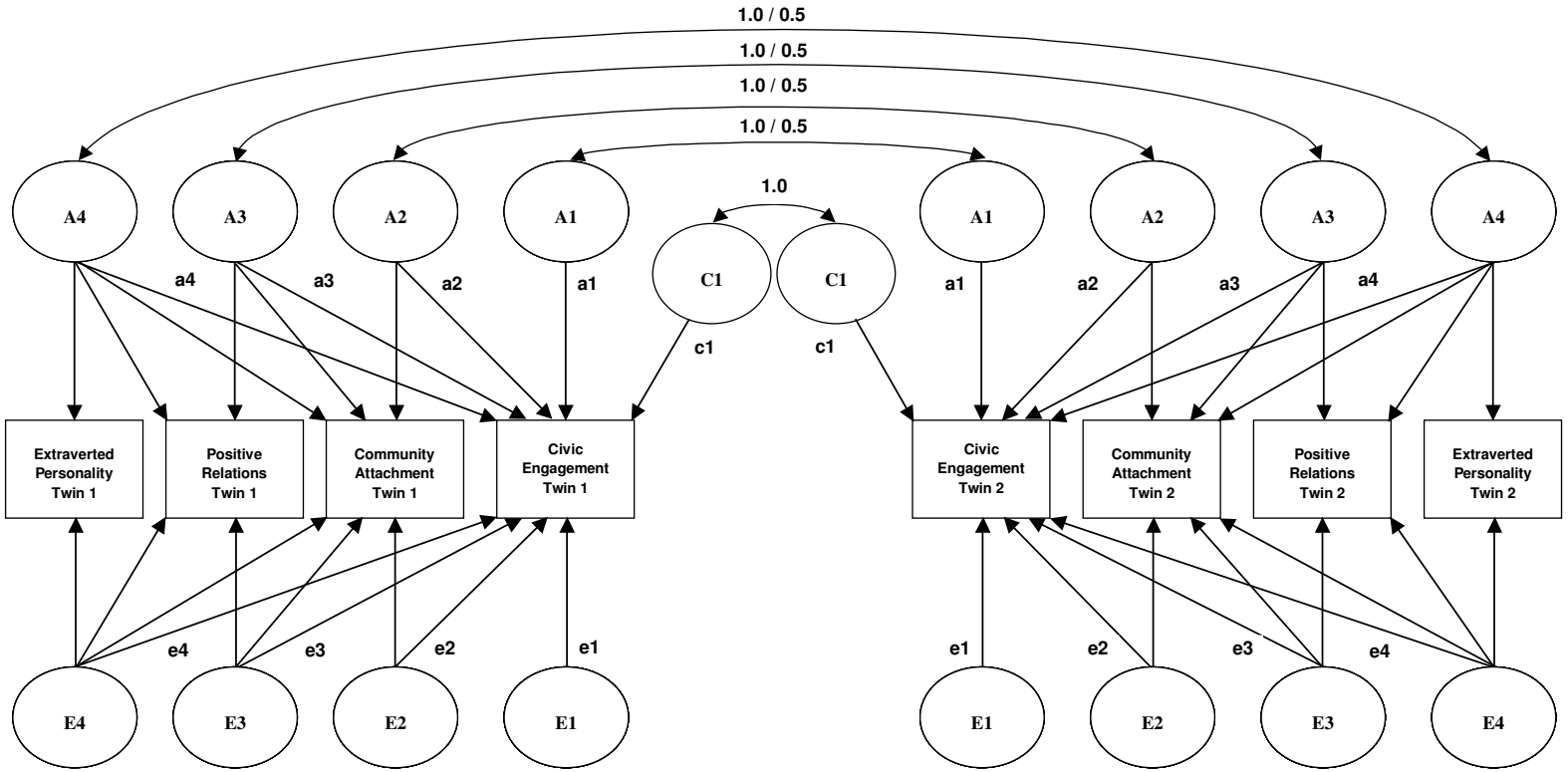


Model-Fitting Techniques

Structural equations modeling (SEM) is the most widely employed and precise method for estimating twin models. In the terminology of SEM, twin models are two-population models composed of a population of MZ twin pairs and a population of DZ twin pairs. In structural models like the ones shown in Figures 2.2 and 2.3, the correlation between MZ siblings is fixed to 1.0 on the genetic factor because these siblings are, by definition,

genetically identical (i.e., they are perfectly correlated with each other genetically). The correlation between DZ twin siblings, in contrast, is fixed to 0.5 on the genetic factor because they share, on average, half their genes. Shared environmental influences are contexts that twin siblings have in common regardless of zygosity, so these factors are fixed to 1.0 for both types of twins. Nonshared environmental influences, on the other hand, are contexts that are unique to each twin of a pair, which means that there is no correlation between siblings on these factors for either MZ or DZ twins.

Figure 2.3: Multivariate Structural Equations Model of the Genetic and Environmental Relationships Between an Extraverted Personality, Positive Relations with Others, Community Attachment, and Civic Engagement



Using the publicly available SEM package Mx (Neale et al. 2003), two different models are fit to variance-covariance matrices constructed from the data. Figure 2.2 shows the simple univariate twin model, which estimates proportional genetic (a^2), shared environmental (c^2), and nonshared environmental (e^2) effects on a single variable. 95-percent confidence intervals, as well as change in chi-square tests for nested / reduced models, are used to determine the statistical significance of each parameter. This model is fit to the data separately for each of the civic engagement and sociality measures employed here in order to establish baseline univariate estimates.

Multivariate Cholesky decomposition models are also fit to the data (Neale and Cardon 1992; Snieder et al. 1999). The full model, which is shown in Figure 2.3, simultaneously examines the effects of all three aspects of sociality on civic engagement (obligations and volunteering, separately). Specifically, it provides estimates of: (a) independent genetic, shared environmental, and nonshared environmental influences ($a1^2$, $c1^2$, and $e1^2$, respectively) on individual variation on obligations / volunteering; and (b) genetic and nonshared environmental effects on obligations / volunteering that are mediated by an extraverted personality ($a4^2$ and $e4^2$, respectively), positive relations with others net of extraversion ($a3^2$ and $e3^2$, respectively), and community attachment net of extraversion and positive relations ($a2^2$ and $e2^2$, respectively). (Note: In the univariate analyses described below, shared environmental influences, $c1^2$, were not found to be important predictors of any of the independent or dependent variables. Thus, mediating factors for these variables are not included in the multivariate models. Also, the paths on the diagrams that are not labeled are nonetheless estimated, but since they are irrelevant

for the purposes of the present study, they are not given any attention here.) Total genetic, shared environmental, and nonshared environmental effects on obligations and volunteering (separately) are obtained with the following formulas, respectively: $a1^2 + a2^2 + a3^2 + a4^2$; $c1^2$; $e1^2 + e2^2 + e3^2 + e4^2$.

Two different multivariate models are fit: one for civic obligations, extraversion, positive relations with others, and community attachment; and another for the frequency of volunteering, extraversion, positive relations with others, and community attachment. It is important to note that the interpretation of the parameter estimates in Cholesky models depends on the [rationally-defined] ordering of the variables (Hatemi et al. 2007; Loehlin 1996). Since civic engagement is the outcome under investigation here, it is always entered into the models last. The ordering of the three independent variables—extraversion first, positive relations with others second, and community attachment third—is based on theory. Specifically, these three measures were chosen because they tap three different levels (i.e., individual, interpersonal, and macrosocial) of social life. In line with the fact that general constructs generally incorporate specific ones more strongly than the inverse (see Hatemi et al. 2007), it is assumed that broad-based individual-level characteristics such as extraversion are strongly influenced by genetic factors, and that they will subsequently influence more specific social characteristics at the other levels. Interpersonal relations, or the quality of intimate relationships one is able to establish, are likely to, in turn, influence one's integration into larger social structures such as communities. In essence, this order flows from very broad psychological characteristics, to more specific types of social relationships. There is undoubtedly some

feedback among these three levels, but there is almost certainly some causal influence in the direction assumed here. Used in this way, we will be able to see: (a) whether genetic effects on extraversion are correlated with (i.e., explain) genetic effects on civic engagement; (b) if genetic effects on interpersonal relations explain some additional genetic variation on civic engagement once the effects of extraversion are removed; and (c) whether macrolevel factors such as community attachment offer any explanatory power above and beyond extraversion and intimate social relationships (for additional information on these models or formulas see: Neale and Cardon 1992; Snieder et al. 1999).

RESULTS

Before the results are presented, a brief discussion of the equal environments assumption is warranted. To empirically examine the validity of this assumption, two different methods were employed here. First, controls for several measures of the rearing environment were introduced into all of the statistical models to examine whether genetic effects were reduced or eliminated. Specifically, measures gauging common playmates during childhood, being in the same classrooms at school, being dressed alike during childhood, seeing each other currently, and current communications via phone, letters, or electronic mail, as well as educational attainment, were included in the models. Results indicated that proportional genetic and environmental effects on civic engagement were virtually identical in models with and without these controls. This provides at least some support for the validity of the equal environments assumption. Second, difference-scores

for each of the equal environments assumption measures were regressed on difference-scores on civic engagement to examine whether differences in childhood environments, adult contact, and educational attainment differentiated twin siblings on civic engagement. No significant relationships were found. This provides additional support for the equal environments assumption. Therefore, given (a) the lack of explanatory power provided by these variables, (b) that the inclusion of these measures introduces a considerable number of missing cases into the analyses, and (c) that the following models are much more parsimonious, the analyses presented below do not contain controls for these measures. This does not, however, bias any of the substantive findings.

Table 2.1: Proportional Genetic, Shared Environmental, and Nonshared Environmental Effects on Individual-Level Variation on Civic Obligations, Frequency of Volunteering, Extraversion, Positive Relations with Others, and Community Attachment (Best-Fit Univariate Models)

	Genetic Effects	Shared Environmental Effects	Nonshared Environmental Effects
Model 1: Civic Obligations	0.46 *	-	0.54 *
Model 2: Frequency of Volunteering	0.34 *	-	0.66 *
Model 3: Extraversion	0.40 *	-	0.60 *
Model 4: Positive Relations with Others	0.38 *	-	0.62 *
Model 5: Community Attachment	0.38 *	-	0.62 *

Notes: (Fit statistics for Models 2-5 are based on the analyses from frequency of volunteering: N = 262 MZ pairs and 216 DZ pairs. N = 287 MZ pairs and 230 DZ pairs for Model 1)

*p < 0.05 based on 95% confidence intervals and change in chi-square tests

Model 1 Fit Statistics: chi-square = 0.02; p-value = 1.00; df / parameters = 4 / 2; AIC = -7.99
Model 2 Fit Statistics: chi-square = 0.14; p-value = 1.00; df / parameters = 4 / 2; AIC = -7.86
Model 3 Fit Statistics: chi-square = 3.11; p-value = 0.54; df / parameters = 4 / 2; AIC = -4.89
Model 4 Fit Statistics: chi-square = 1.38; p-value = 0.85; df / parameters = 4 / 2; AIC = -6.62
Model 5 Fit Statistics: chi-square = 0.05; p-value = 1.00; df / parameters = 4 / 2; AIC = -7.95

Table 2.1 provides the results of the univariate genetic analyses of civic obligations, volunteering, and sociality. The best-fit models for each of the five measures contain two factors: genetic and nonshared environmental influences. Shared environmental influences, in contrast, do not appear to be important. Specifically, proportional genetic effects on civic obligations were estimated to be 46%, while nonshared environmental influences accounted for the remaining 54%. For the frequency of volunteering, findings indicated that genetic factors explained 34% of the variation, while nonshared environmental influences explained the remaining 66%. Overall, these results provide strong support for Proposition 1.

With respect to genetic and environmental influences on sociality, the proposed mediator of genetic effects on civic engagement, results show that genetic factors explain 40, 38, and 38% of the individual variation on extraversion, positive relations with others, and community attachment, respectively. Nonshared environmental influences explain the remaining 60, 62, and 62% of the variation, respectively, on these three outcomes. Although these univariate results are not central to the present study, which focuses on civic engagement, they are important for Proposition 2, which argues that sociality may mediate (i.e., explain) genetic effects on obligations toward civic participation and the frequency of volunteering. For this to actually be occurring, however, sociality needs to be influenced by genetic factors as well, and these results suggest that this may indeed be the case.

Table 2.2: Proportional Direct and Indirect (Via Extraversion, Positive Relations with Others, and Community Attachment) Genetic, Shared Environmental, and Nonshared Environmental Effects on Individual-Level Variation on Civic Obligations (N = 287 Monozygotic and 230 Same-Sex Dizygotic Pairs)

	Genetic Effects	Shared Environmental Effects	Nonshared Environmental Effects
Model 1: Univariate Model for Civic Obligations	0.46 *	-	0.54 *
Model 2: Multivariate Model for Civic Obligations, Extraversion, Positive Relations with Others, and Community Attachment			
Independent Effects on Civic Obligations	0.26 ns	-	0.52 *
Effects Mediated by Extraversion	0.06 *	-	0.00 ns
Effects Mediated by Positive Relations with Others	0.05 *	-	0.00 ns
Effects Mediated by Community Attachment	0.09 *	-	0.02 *

Notes:

*p < 0.05 based on 95% confidence intervals and change in chi-square tests

Model 1 Fit Statistics: chi-square = 0.02; p-value = 1.00; df / parameters = 4 / 2; AIC = -7.99

Model 2 Fit Statistics: chi-square = 48.21; p-value = 0.62; df / parameters = 52 / 20; AIC = -55.79

To actually test Proposition 2, however, it is necessary to fit multivariate twin models such as the one shown in Figure 2.3 to the data. Model 2 of Table 2.2 shows the multivariate results for civic obligations (note: Model 1 is shown simply for comparison purposes, and is identical to the one shown in Table 2.1). This model estimates four different genetic effects: (a) direct genetic effects on civic obligations that are net of those in common with the three measures of sociality; (b) genetic effects that are shard with extraversion; (c) genetic effects that are shared with positive relations with others and net of extraversion; and (d) genetic effects that are shared with community attachment and net of extraversion and positive relations with others. These results show that the 46% genetic effect on civic obligations shown in Model 1 is reduced to 26% when these three aspects of sociality are controlled. This amounts to a 43% (26% / 46%) reduction in the

total genetic effect, which means almost half of the total genetic effect on civic obligations is mediated by (i.e., shared with) these three outcomes. For each aspect of sociality specifically, extraversion explains 13%, positive relations with others explains 11%, and community attachment explains 20% of the genetic effect on civic obligations. These findings provide strong support for Proposition 2. Interestingly, when these three outcomes are controlled, the path for direct genetic influences, despite being estimated to be 26%, is reduced to statistical non-significance, meaning that it could actually be 0. Although this could be interpreted as evidence that sociality completely mediates genetic effects on civic obligations, one should use caution when assuming this since it is a reflection of the confidence interval around the 26% estimate being wide, which is not the same thing as the estimate actually being 0.

Table 2.3: Proportional Direct and Indirect (Via Extraversion, Positive Relations with Others, and Community Attachment) Genetic, Shared Environmental, and Nonshared Environmental Effects on Individual-Level Variation on Frequency of Volunteering (N = 262 Monozygotic and 216 Same-Sex Dizygotic Pairs)

	Genetic Effects	Shared Environmental Effects	Nonshared Environmental Effects
Model 1: Univariate Model for Frequency of Volunteering	0.34 *	-	0.66 *
Model 2: Multivariate Model for Frequency of Volunteering, Extraversion, Positive Relations with Others, and Community Attachment			
Independent Effects on Frequency of Volunteering	0.28 ns	-	0.64 *
Effects Mediated by Extraversion	0.01 ns	-	0.00 ns
Effects Mediated by Positive Relations with Others	0.03 *	-	0.00 ns
Effects Mediated by Community Attachment	0.01 ns	-	0.01 *

Notes:

*p < 0.05 based on 95% confidence intervals and change in chi-square tests

Model 1 Fit Statistics: chi-square = 0.14; p-value = 1.00; df / parameters = 4 / 2; AIC = -7.86

Model 2 Fit Statistics: chi-square = 36.67; p-value = 0.95; df / parameters = 52 / 20; AIC = -67.33

The findings for volunteering, which are displayed in Table 2.3, are not nearly as strong as they are for obligations, but there is some evidence that sociality mediates genetic effects on this outcome. Looking at Model 2, the 34% genetic effect on volunteering shown in Model 1 (the univariate model) is reduced to 28% in the multivariate model that adjusts for genetic effects in common with the three measures of sociality. This might represent a 15% reduction in the total genetic effect, but since the path estimates for extraversion (0.01) and community attachment (0.01) are not statistically significant, we can only be confident that positive relations with others (0.03) is actually mediating genetic effects on volunteering. Specifically, this outcome explains 9% (3% / 34%) of the total genetic effect on volunteering.

DISCUSSION

To date, research on civic engagement has focused almost exclusively on social-environmental influences. This could be problematic, however, given that several recent studies have suggested that genetic factors may also be important (Alford, Funk, and Hibbing 2005; Alford and Hibbing 2004; Hibbing and Alford 2004; Masters 2001; Wilson 2006). The present study addressed this possibility, and extended this bourgeoning literature, in three ways.

First, it moved beyond examining political orientations (i.e., attitudes and ideologies), and instead focused on more general obligations toward civic engagement, as well as actual behaviors in this area. An analysis of a national sample of working-age twin siblings showed that genetic “and” environmental influences were both important

predictors of individual-level variation on (a) an indexed measure tapping obligations toward serving on a jury, testifying in court, keeping informed about social issues, voting, and volunteering, as well as (b) a measure of the actual frequency with which individuals engage in voluntary activities in their community. Specifically, genetic influences were found to account for 42 percent of the variation on obligations, and 34% of the variation on voluntary activities. Nonshared environmental influences, as opposed to shared environmental factors, accounted for the remaining variation on each. The proportional genetic effects found here are comparable to previous findings for social and political orientations, as well as numerous social-oriented personality characteristics (Alford, Funk, and Hibbing 2005; Beer et al. 1998; Bouchard et al. 1990; Eaves, Eysenck, and Martin 1989; Martin et al. 1986).

Second, this research outlined a preliminary theory suggesting that genetic effects on contemporary social and political attitudes and behaviors such as civic engagement may be real in their effects, but that they may actually be indirect result (i.e., byproduct) of correlations with sociality—i.e., social-oriented personality and interpersonal relations characteristics (Alford, Funk, and Hibbing 2005). In other words, personality traits such as extraversion and positive relations with others, as well as more macrolevel aspects of social life such as community attachment, may express themselves in many different social and psychological arenas, including those pertaining to civic participation. This theory was supported with empirical data showing that 43 percent of the total genetic effect on civic obligations was mediated by these three characteristics. The findings for

the frequency of volunteering were not as strong, but they revealed that at least some proportion (9-15%) of the genetic effect on volunteering was mediated by sociality.

Third, this study empirically tested one of the foundational assumptions of twin studies: the "equal environments assumption." Briefly, if MZ twin siblings experience environments that are substantively different from DZ siblings, the greater similarity among them compared with DZ siblings—a finding that is typically interpreted as evidence for a genetic effect—could actually be due to environmental differences. In this study, controlling for several environmental variables—i.e., having the same playmates, attending the same classes, and being dressed alike in childhood, as well as frequency of contact during adulthood and educational attainment—did not decrease the magnitude of the genetic effects on civic obligations or volunteering. Further, twin difference score analyses revealed that differences in these family-level environmental outcomes did not predict differences in civic engagement. Thus, unequal treatment or contact does not appear to explain why genetically-identical siblings are more similar on civic obligations and volunteering than their non-identical counterparts.

It is important to note that even though this study supports a role for genetic factors in civic life, it does not imply that civic engagement is genetically pre-programmed as opposed to learned from environmental influences; quite the contrary. Rather, research of this type focuses on the question of whether individual differences in "socially learned" attitudes and behaviors are the result of genetic makeup and / or common or unique environmental influences. Even strict biological determinists now concede that most, if not all, attitudes and behaviors, especially complex ones such as those involving civic

life, must be learned from environmental sources. Therefore, since genetic influences are probably not blueprints from which the attitudes and behaviors of individuals are forced to unfold, they are best portrayed as environmentally-shaped yet innate needs, wants, motivations, preferences, tastes, or desires. Given this conceptualization, it might be useful to view the components of the twin model in terms of an economic / rational choice model of individual free-will (the genetic component) versus social constraints (the two environmental components). Proportionally large genetic effects on outcomes such as civic participation may reflect the relative weakness of environmental influences, while smaller genetic effects likely imply that social influences are particularly powerful. In the case of civic engagement, both appear to be powerful.

In addition to arguing that genetic factors matter, this study also supports a significant role for social influences on civic life. According to the findings, 56 percent of the variation on civic obligations, and 66% of the variation on volunteering, is explained by the variables of interest to social scientists. Recall that previous research has linked a host of environmental influences—e.g., professional interests, stable residency, home ownership, marriage, parenthood, socioeconomic status, verbal ability, community ties, population density and heterogeneity, religious involvement, and media consumption, among others—with civic participation (Beyerlein and Hipp 2006; Campbell 2006; Galston 2001; Wilson and Janoski 1995; Wilson and Musick 1997). In no way does the presence of genetic influences on civic engagement discount the importance of these factors, and they deserve more attention in the future.

That said, these findings do highlight two potential implications or problems with social scientific research in this area. First, given that these results, as well as those of previous research, suggest that genetic factors account for roughly 40 percent of the individual-level variation on social and political attitudes and behaviors, models that do not consider these influences will likely fail to explain a large proportion of the variation. This suggests that traditional social scientific models may suffer from explanatory power deficiencies. Second, and perhaps more importantly, is the possibility that genetic factors may be confounding social scientific studies of civic participation (Bouchard and Loehlin 2001; Glymour 1997; Udry 1995). In other words, omitted variable bias may be problematic for outcomes—including civic obligations and volunteering—that appear to be influenced by genetic factors. A preliminary suggestion: Social scientists should employ genetically-informed designs that control for potential genetic confounders while simultaneously examining theoretically important social influences.

Although informative, this study is unable to adequately explain why and how genetic factors influence civic engagement. Additional research should address potential direct and indirect effects. Potential direct genetic influences might include specific genes or groups of genes, and biochemical processes such as hormones and neurotransmitters, among others (see Udry 2000 for a related example). Potential indirect influences or mediators—i.e., influences that intervene between genetic predispositions and civic participation—might include the following, all of which have been shown to be at least partially the result of genetic factors: altruism, risk-aversion, reward-seeking and competitive instincts, interpersonal attachments, and various personality types, among

many others (Alford, Funk, and Hibbing 2005; Bouchard and Loehlin 2001; Carson and Rothstein 1999; Kirkpatrick 1999). The present study has already demonstrated the importance of social-oriented personality characteristics and interpersonal relations, but other mediators likely exist as well, and should be addressed.

Future research should also begin examining the complex intersection between genetic and social influences on civic participation. This necessarily entails examining potential gene-environment correlations and interactions (see Plomin 1990; Scarr and McCartney 1983). If genetic influences on civic engagement are indeed genuine, it is likely that they are either facilitated or impeded by any number of environmental factors. For example, genetic effects on civic engagement may be facilitated in contexts where individuals are socially integrated, whereas they may be impeded in anomic situations. Other environmental factors including socioeconomic status, region, media consumption, and religious involvement, among others, may also be important environmental moderators. Despite an emphasis on gene-environment interactions in others areas of scholarship, particularly health, no research of this type has been conducted on civic participation.

To conclude, individual-level variation on obligations toward civic participation, as well as the frequency of volunteering, appears to be the product of both genetic and social-environmental influences. Additional research of this type is definitely warranted, especially since it can shed fresh light on the arguments of social scientific research on this topic. It can tell us whether, and to what extent, innate motivations versus family- and nonfamily-level social influences are responsible for variation on civic-oriented

outcomes. It can even address the potential relationships, correlative and interactive, between these two forces.

CHAPTER 3

Genes, Environments, and Health:

Theorizing Independent and Interconnected Effects

SUMMARY

The two previous chapters exemplified the utility of a genetically-informed approach for two social outcomes that have been given very little consideration in the literature: religious involvement and civic engagement. In contrast, this chapter (and the remainder) of this dissertation addresses a topic that has received considerable attention from geneticists, biologists, psychologists, psychiatrists, and sociologists alike: health and well-being. Specifically, it theorizes the potential ways in which genetic and environmental influences on health and well-being might be interconnected. In essence, this chapter provides insight into Research Questions 1, 2, 3, and 4 of the dissertation by providing a detailed theoretical formulation of five possible models of gene-environment effects on health and well-being: (1) genetic-only effects; (2) environment-only effects; (3) additive genetic and environmental effects; (4) correlated genetic and environmental effects; (5a) environmental moderation of genetic effects; and (5b) genetic moderation of environmental effects. The theoretical insight provided in this chapter will help to (a) organize the existing literature in this area, and (b) guide and frame future scholarship on genes, environments, and health, including the final two chapters of this dissertation.

INTRODUCTION

Medical sociologists have linked various aspects of social life—e.g., social-oriented personality characteristics, social support, community ties, marital and parent-child relations, religious involvement, and socioeconomic status, to name just a few—with diverse health outcomes including depression, hypertension, alcohol and tobacco use, obesity, cancer, and even mortality (Cohen 2004; Ellison and Levin 1998; Glenn and Weaver 1981; Gove, Style, and Hughes 1990; House 2002; Hummer et al. 1999; Lu and Hu 2005; Noor 1996). Virtually all of these studies show that socially-integrated individuals experience better health than individuals who lack social ties.

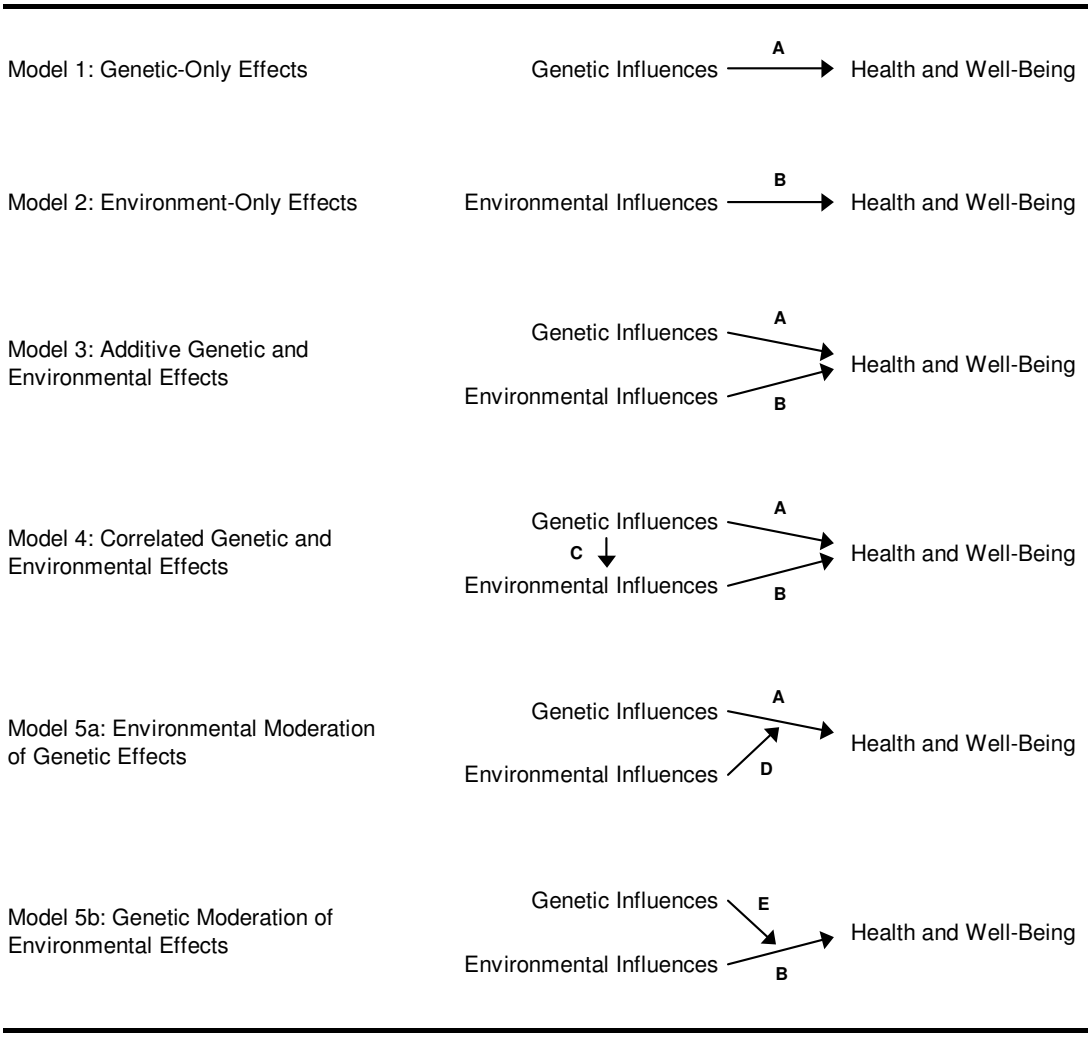
Despite the utility of this literature, it suffers from at least one weakness: the failure to adequately address genetic or other biological influences. Why should medical sociologists, who purposefully and exclusively focus on social-environmental factors, be concerned with genetic influences? The answer is simple: genetic differences appear to predict (a) virtually all health outcomes (Caspi et al. 2003; Hamann 2005; Johnson and Kreuger 2005a and b; Kendler, Gardner, and Prescott 1997; Lesch et al. 1996; Pezawas et al. 2005), (b) many different "environmental" variables of consequence to health (Cleveland and Crosnoe 2004; Kendler 1997; Kenrick et al. 2003; Spotts et al. 2004), and (c) even the correlation between social relationships and health (Lichtenstein et al. 1992; Reiss et al. 2000; Spotts et al. 2005). It may even be the case that the connection between environmental influences and health is contingent upon (i.e., moderated by) genetic factors (Kendler et al. 1995; Rutter, Moffitt, and Caspi 2006). In essence, considerable evidence shows that health and well-being is the product of both genetic and

environmental influences, which means that it will be difficult, if not impossible, for sociologists to fully understand health disparities without simultaneously examining both of these powerful influences. Further, empirical findings now suggest that the failure to control for genetic effects while simultaneously examining environmental ones (and vice-versa) could lead to biased estimates, confounded relationships, and possibly even erroneous conclusions (Bouchard and Loehlin 2001; Caspi et al. 2005a and b; D'Onofrio et al. 2006; Gottfredson 2003; Lichtenstein et al. 1992; Neiderhiser et al. 1999; Reiss et al. 2000; Spotts et al. 2005; Udry 1995).

Thus, medical sociologists and other scholars of health need to move beyond overly simplistic questions—e.g., Do genetic and environmental factors both influence health, and what is the proportional contribution of each?—and toward more informative ones such as: Are genetic and environmental influences on health independent of one another (i.e., are their effects additive), or are they interconnected in some way? How might genetic and environmental factors work together to produce health outcomes? Are sociological studies of health confounded by genetic factors, and are biomedical studies biased by unmeasured social influences? Do environmental variables mediate genetic effects on health? Are genetic influences on health moderated by environmental contexts, and does the association between environmental variables and health vary depending upon genetic differences? The present study begins addressing questions such as these by formulating five distinct conceptual models of gene-environment effects: (1) genetic-only effects; (2) environment-only effects; (3) additive genetic and environmental effects; (4) correlated genetic and environmental effects; (5a) environmental moderation of genetic

effects; and (5b) genetic moderation of environmental effects. Each of these models is shown diagrammatically in Figure 3.1, and discussed in detail below. Overall, integrating genetic factors into sociological research will lead to a deeper understanding of the causes of health and well-being than is currently possible, and the models outlined here provide preliminary theoretical direction to scholars seeking to achieve this goal.

Figure 3.1: Five Models of Gene-Environment Effects on Health and Well-Being



INDEPENDENT GENETIC AND ENVIRONMENTAL EFFECTS

Model 1: Genetic-Only Effects

Much of our knowledge of genetic effects is based on comparisons of health outcomes across individuals of known, and differing, genetic relatedness—i.e., twin siblings and / or adopted versus biological family members. Using data on multiple individuals with varying degrees of genetic similarity, researchers are able to roughly estimate the proportion of individual-level variation on health outcomes that is due to genetic and environmental influences (Neale and Cardon 1992). Basically, if genetically similar individuals are more concordant on health outcomes compared with individuals who are less similar genetically, this provides evidence for the presence of genetic effects. Findings obtained using this approach suggest that genetic factors are significant predictors of both mental and physical health, as well as health-risk behaviors.

With respect to mental health, research shows that genetic factors account for a considerable proportion (30-70 percent) of the individual-level variation on affective disorders such as depression (Eley 1997; Kendler 2001; Kendler 2001; Plomin 1990; Plomin and Rende 1991; Sullivan et al. 2000). Similarly, scholars have found that the first-degree relatives of persons suffering from anxiety attacks have a risk of developing these symptoms that is almost seven times greater than it is in the population as a whole: a fact that is strongly suggestive of sizable genetic effects (Crowe et al. 1983; Finn and Smoller 2001). In the realm of severe psychopathology, schizophrenia is largely, although not exclusively, the product of genetic factors (Gottesman 1991).

For physical health, there is a large literature showing that cancer is at least partially the product of an unfortunate genetic heritage (Benhamou and Sarasin 2005). Hypertension also appears to be strongly influenced by genetic factors, with proportional genetic effects on systolic and diastolic blood pressure ranging from 44-64 and 34-73 percent, respectively (see Whitfield and McClearn 2005). Forced expiratory volume, which gauges lung capacity and is commonly-employed as an indicator of overall physical vitality among older adults, has also been shown to be influenced by genetic differences (McClearn et al. 1994; Whitfield et al. 1999). Findings on other aspects of physical health suggest that genetic factors predict variation on outcomes including chronic pain (Zondervan et al. 2005), body mass index (BMI) (Johnson and Kreuger 2005a), eating disorders (Kendler 2001; Polivy and Herman 2002), and insulin and glucose levels in the body (Sneider et al. 1999), among others.

Health-risk behaviors, which affect both mental and physical well-being, also appear to be influenced by genetic differences. For example, considerable research has shown that alcohol consumption patterns are at least partially the product of genetic influences (Kendler and Prescott 2006). Tobacco use, which is predictive of many different health outcomes, also appears to be influenced by one's genetic makeup, with research showing that the age at which one first experiments with cigarettes, as well as eventual dependence, are both substantially heritable (Heath et al. 1999). Delinquency, behavior problems, risky activities, and other forms of health-risk behaviors also appear to be influenced by genetic factors (Guo, Roettger, and Shih 2007; Jaffee et al. 2005).

To date, twin and adoption studies such as these have been fruitfully applied to the study of health and well-being, and they will continue to be employed into the foreseeable future for one simple reason: most (and perhaps all) health outcomes are complex phenotypes that are influenced by a whole host of different genes (plus countless environmental influences), each of which exerts a relatively small effect on the final, observable characteristic (Guo 2005; Plomin and Caspi 1998; Plomin and Rende 1991). In other words, most health outcomes are polygenic traits, and given that we currently understand the functions of only a small percentage of the literally thousands of human genes, actually measuring genetic differences and then correlating them with health outcomes is currently somewhat difficult. Thus, at the present time family-based studies are particularly useful for studying complex traits. That said, techniques such as twin designs are based on assumptions—e.g., the equal environments assumption, which claims that the environments of monozygotic and dizygotic siblings are not substantively different, and thus if the former are more similar, it must be due to genetic influences—that have been questioned, particularly by sociologists (see Freese and Powell 2003; Horowitz et al. 2003; Neale and Cardon 1992).

In the coming years, however, the dismissal of genetic influences because of methodological objections will not be sufficient. Molecular genetic techniques are already reinforcing the findings of twin and adoptions studies, and they promise to provide even more (and perhaps stronger) evidence for the importance of genetic influences on health outcomes in the near future (Guo 2005). Research in this vein (i.e., molecular genetics) poses more direct challenges to medical sociology than the twin and

adoption studies reviewed above because there are no controversial assumptions associated with this research. In molecular studies, genetic differences are actually "measured" via genotype (i.e., DNA) sequencing or scanning procedures, and health disparities are then examined across differences in genetic makeup.

Although most of the specific genes in the human genome remain unidentified, polymorphisms in several different genes have in fact already been discovered and linked with health outcomes. For example, several studies have found that different variants of the serotonin transporter gene predict differences in levels of depression and anxiety (Hamann 2005; Lesch et al, 1996; Pezawas et al. 2005). Other specific genetic differences, many of them polymorphisms of genes related to serotonin, dopamine, or other biochemicals, have been linked with various other health outcomes including schizophrenia (Sullivan 2005) and anxiety and panic disorders (Deckert et al. 1999). Overall, even though our knowledge on this front is still somewhat limited, an enormous amount of research of this type is currently being conducted, and the expansion of the literature in this area is going to be rapid in the coming years. Broadly, this type of research suggests the following proposition, which accompanies Model 1 of Figure 3.1:

- Proposition 1: Genetic factors influence health and well-being (Model 1, Pathway A).

Model 2: Environment-Only Effects

In addition to genetic differences, many different social-environmental influences have also been linked with health and well-being. Most of this research is grounded in social

capital theory and / or the life-stress paradigm, both of which conceive of social relationships as either (a) psychological or material resources that help to buffer individuals against poor health (e.g., a supportive spouse, high levels of education, etc.), or (b) stressors that have deleterious effects on health (e.g., divorce, low levels of income, etc.) (Ellison 1994; Ellison et al. 2001; Ensel and Lin 1991). Research in this area is premised on the fact that inequities exist in the number of problems individuals have, as well as the amount of psychosocial resources they can draw on to deal with their problems. Within this literature, social-environmental influences take many forms, and the present study reviews a diverse assortment of these including social orientations, interpersonal characteristics, social support and community ties, marriage and family life, religious involvement, and socioeconomic status.

Various social-oriented personality and interpersonal characteristics—e.g., traits that reflect one's general ability to form and maintain positive relations with others—appear to pose implications for health and well-being. For example, extraverted and agreeable individuals (compared with introverted and disagreeable ones) tend to be more enthusiastic, talkative, assertive, gregarious, cooperative, and empathetic, and they also tend to find greater pleasure in social activities such as community get-togethers, political rallies, and organization-based religious activities, among many others (Baker and Daniels 1990; Bouchard and Loehlin 2001; Eaves and Eysenck 1975; Eysenck 1990; Lachman and Weaver 1997). Given their desire for, and success at engaging in, social life, it follows that extraverted and agreeable individuals are more socially integrated than others, and since social ties have been linked with a variety of health outcomes, it comes

as no surprise that traits such as these are correlated with both mental and physical well-being (Costa and McCrae 1980; Duckitt 1984; Larsen and Kasimatis 1990; Lu and Hu 2005; Noor 1996; Ozer and Benet-Martinez 2006; Tanaka et al. 1998).

Social support and community attachment have also received some attention in the health literature (Fitzpatrick and LaGory 2003). Individuals who are connected to supportive others are less likely to suffer from poor health compared with socially isolated persons. A considerable amount of research in this area has focused on the types of support—e.g., instrumental, informational, and emotional—individuals receive from those close to them, and virtually all of it has found a strong positive correlation between levels of support and desirable health outcomes (Cohen 2004). More recent work has now extended this research beyond intimate, personal relations to include those at the community level as well (O'Brien et al. 1994). Findings in this area show that community satisfaction and attachment are both independently and positively correlated with individual well-being net of other forms of social life (Theodori 2001). In a related area of scholarship, research suggests that neighborhood characteristics are also correlated with health and well-being (Mirowsky and Ross 2003).

Considerable evidence has also shown that family life has an affect on health. For example, married individuals report better health than single adults (Mirowsky and Ross 2003). One reason for this association is that spouses are a primary source of social support that can be utilized during difficult times (Beach et al. 1993). In addition to merely being married, several studies have also linked marital quality with health and well-being (Glenn and Weaver 1981; Keicolt-Glaser and Newton 2001; Prigerson et al.

1999). This research shows that individuals who report being in good marriages are happier (Gove, Hughes, and Style 1983), and may even experience fewer depressive symptoms (Spotts et al. 2004), than individuals in poor marriages. Another aspect of family life—parent-child relations and the presence of children in the home—has also been examined by medical sociologists. Findings in area suggest that children can exert both desirable and deleterious effects on one's health (Mirowsky and Ross 2003).

Religious involvement—another aspect of social life that has been linked with health and well-being—is a complex, multidimensional social outcome. Consequently, various facets of religious participation may differ in their association with a given variable of interest. In studies of health and well-being, two of the most important dimensions of religiousness are (a) organizational involvement (often gauged in terms of attendance at worship services or other congregational activities), and (b) non-organizational involvement (including private practices such as prayer and mediation, as well as religious identification and personal salience). Organization-based religious participation is linked with health and well-being for a variety of reasons, including the ability to provide (a) monetary support in times of trouble, (b) assistance with finding and maintaining employment, (c) discouragement of health-risk behaviors such as smoking, drinking, and sexual promiscuity, (d) information pertaining to medical treatment, and (e) the presence of like-minded others with whom to share one's problems, among many others (Ellison and Levin 1998). Aspects of religion that are not necessarily linked with formal organizations—e.g., prayer, mediation, salience, beliefs, etc.—also pose implications for health and well-being in many ways. For example, feelings of connection

to an omnipotent and loving God (or gods) (Poloma and Gallup 1991; Wikstrom 1987) may help to facilitate self-esteem and personal efficacy, both of which have been linked with desirable health outcomes. Similarly, intimate relations with the divine may instill positive emotions such as forgiveness and empathy in individuals, and it may even facilitate healthy beliefs such as hope and optimism. For these reasons, among others, religious involvement has been linked with a diverse assortment of health outcomes including depression, anxiety, alcohol addiction, tobacco use, life satisfaction, and even mortality (Ellison and Levin 1998).

Socioeconomic status—one of the most commonly-employed outcomes in sociological studies—influences health and well-being by shaping (a) individual differences in 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 resources available to individuals) (Kristenson et al. 2004; Weinstein et al. 2003). Briefly, individuals with high SES tend to have relatively few problems and many resources at their disposure, while people with low SES have more problems and fewer resources (Glenn and Weaver 1981; Miech et al. 1999; Pearlin et al. 1981). For example, upper SES individuals are less prone to face difficulties in meeting financial, healthcare, and legal needs, and thus face fewer stressors in these areas. Individuals with high levels of education and income also tend to be embedded in larger social networks, which facilitate emotional, tangible, or informational resources. It is also well-established that individuals with high SES possess more psychological and cognitive resources, such as feelings of personal control, efficacy, and

self-worth, which can help them cope with stressful life conditions when they arise (Mirowsky and Ross 2003). Thus, in studies of health scholars have firmly established that SES is positively associated with both mental and physical well-being, and inversely associated with a variety of poor health outcomes (Adler and Ostrove 1999; Kessler 1982; Marmot and Wilkinson 1999; Taylor 2002). Overall, this brief review of the sociological literature suggests the following proposition, which is shown as Model 2 of Figure 3.1:

- Proposition 2: Environmental factors influence health and well-being (Model 2, Pathway B).

Model 3: Additive Genetic and Environmental Effects

Despite widespread endorsement and utilization, genetic-only and environment-only effects models are probably drastic oversimplifications of the actual causes of health and well-being, especially when considered separately as they have been in most of the studies outlined above. Given the various issues involved with examining genetic and environmental influences in isolation—e.g., explanatory power deficiencies, biased and possibly even confounded empirical relationships, erroneous conclusions, etc.—it is important that researchers begin to analyze both of these factors simultaneously, especially since their effects on health could be either independent or interconnected. With respect to the first possibility, Model 3 of Figure 3.1 depicts an additive genetic and environmental effects model, which is simply a combination of Models 1 and 2. This

model allows the gene-environment independence assumption, which was made by virtually all of the studies reviewed in the previous two sections, to actually be examined. If (a) the theoretical and empirical foundations of the current biomedical and sociological paradigms are correct, and (b) the compartmentalization of researchers into distinct genetic and environmental camps is not problematic, the following proposition should be supported by the data:

- Proposition 3: Genetic and environmental factors influence health and well-being net of the other, and their effects will be largely additive, and thus will not be substantially altered by the simultaneous examination of both (Model 3, Pathways A and B).

INTERCONNECTED GENETIC AND ENVIRONMENTAL EFFECTS

Model 4: Correlated Genetic and Environmental Effects

Despite the size and importance of both the biomedical and sociological literatures on health and well-being, there is good reason to believe that genetic and environmental influences are not independent of one another, at least not completely (D'Onofrio et al. 2006; Johnson and Kreuger 2005a and b; Lichtenstein et al. 1992; Neiderhiser et al. 1999; Reiss et al. 2000; Rutter, Moffitt, and Caspi 2006; Shanahan and Hofer 2005; Spotts et al. 2005). If this is true, the possible interrelations between these two predictors need to be examined, especially if we are to obtain a deeper understanding of the pathways by which each affects health and well-being.

The simplest way in which genetic and environmental factors might be interconnected is by having at least some overlap in their explanatory power. If this is indeed occurring, simultaneously examining both of them will reduce the effect of one or the other (or perhaps both) on health and well-being: something that would provide evidence for non-independent effects. Such a possibility implies the model shown in Model 4 of Figure 3.1, which competes with Model 3. In proposition form:

- Proposition 4a: Genetic and environmental factors influence health and well-being, 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 3, Pathways A and B).

If Proposition 4a is correct, the independence assumption made by biomedical and sociological researchers alike will need to be reconsidered. One of the primary reasons to question this widely-held premise is a growing body of evidence showing that—in addition to health outcomes themselves—many of the seemingly "environmental" predictors of health and well-being are also influenced, to some degree, by genetic factors (Cleveland and Crosnoe 2004; D'Onofrio et al. 1999; Kendler 1997; Kenrick et al. 2003; Reiss et al. 2000; Spotts et al. 2004). If true, this would suggest that genetic influences help to predict the environments in which individuals are situated: environments that subsequently affect health and well-being. Let us take a look at the findings in this area for several different aspects of social life, including social-oriented personality and

interpersonal characteristics, social support and community networks, marriage and family life, religious involvement, socioeconomic status, and the occurrence of stressful events, among others.

Considerable research has shown that broad-based social orientations and characteristics—e.g., a general ability to form and maintain positive relations with others—are influenced by both genetic and environmental factors. Specifically, the literature shows that genetic differences are important predictors—explaining roughly 50% of the variation—of outcomes including an extraverted personality, social closeness and intimacy, openness, agreeableness, dependability, cooperativeness, and the adherence to social rules and authority, among others (Bouchard and Loehlin 2001; Kenrick et al. 2003; Kirkpatrick 2004; Newberg et al. 2002; Plomin 1990; Wilson 1998). Research has even shown that genetic factors influence social support (Kendler 1997) and possibly even degrees of social network closure (Cleveland and Crosnoe 2004). Moreover, social orientations and characteristics such as these have been linked with health and well-being (Williams et al. 2004), and despite the presence of significant genetic effects, these relationships are typically interpreted in terms of purely social influences.

The family rearing environment—a social context where many sociological theories argue that learning and reinforcement occurs—has also been examined using genetically-informed techniques. To date, most social scientists have assumed that the interactions between spouses, parents, and their children could be measured without reference to genetic influences, and that they represented true environmental factors. There is, however, evidence suggesting significant genetic effects in this area of social life. For

example, research shows that three different domains of parent-child relations—positivity (e.g., warmth, empathy, etc.), negativity (e.g., disputes, anger, etc.), and control (e.g., monitoring, knowledge of activities, etc.)—are all influenced by both genetic and environmental factors (Plomin et al. 1994). These same authors also found that the interaction between siblings was influenced by genetic differences as well. Importantly, these genetic effects arise from reports from taken from mothers, fathers, and children alike. Another seemingly environmental outcome that stems from the family home—marital relations—has also been examined. In one study, data from twin women and their partners was analyzed, and the results provided evidence for significant genetic effects on marital quality (Spotts et al. 2004). Further, research has shown that genetic factors are important predictors of individual variation on other aspects of romantic life as well, including pair bonding (Trumbetta and Gottesman 2000), the likelihood of ever marrying (Jerskey et al. 2001), and even divorce (Jerskey et al. 2001; McGue and Lykken 1992). Importantly, both parent-child relations and marriage appear to be associated with health and well-being, perhaps because of both genetic and environmental factors.

Religious involvement, an aspect of social life that is routinely employed as a predictor of health in sociological studies (Ellison and Levin 1998), has also been examined using genetic techniques. The findings for organizational involvement (i.e., religious service attendance, the most commonly used measure in studies of religion and health in the Christian-dominated United States) are not unequivocal, but the weight of the evidence suggests that genetic influences explain roughly 20-30 percent, and possibly more, of the individual-level variation on this outcome (Boomsma et al. 1999; Bradshaw

and Ellison Forthcoming; D'Onofrio et al. 1999; Heath et al. 1999; Kendler, Gardner, and Prescott 1997; Kirk et al. 1999). With respect to more private aspects of religious participation, research has found that genetic factors explain a third or more of the variation on outcomes such as personal religious devotion (Kendler, Gardner, and Prescott 1997), intrinsic versus extrinsic religious orientations (i.e., devout commitment versus self-serving means to ends other than religion, respectively) (Bouchard et al. 1999), personal religiosity (Winter et al. 1999), and subjective religiousness / religious identification (Bradshaw and Ellison Forthcoming). These findings may be important given that both organization-based and private aspects of religion have been linked with many different health outcomes (Koenig et al. 2000).

Socioeconomic status, perhaps the most widely-studied sociological outcome, has even been examined in this context. Research shows sizable genetic effects (possibly explaining 50% or more of the variation) on educational attainment (Heath et al. 1985; Tambs et al. 1989; Vogler and Fulker 1983). Monetary income also appears to be influenced by genetic factors (Rowe et al. 1998). Importantly, SES has been shown to be a major predictor of health and illness, and this research is commonly interpreted in terms of a causal influence from the former to the latter. There is, however, some evidence already suggesting that SES and health are correlated in part because of common genetic factors (Lichtenstein et al. 1992). Further, given that genetic variation contributes to health and well-being (reviewed above), and that health disparities predict socioeconomic differences in a bidirectional manner (Miech et al. 1999; Mulatu and Schooler 2002), it follows that genetic factors may influence SES indirectly via health outcomes.

Additional research has found significant genetic effects on other seemingly environmental variables as well. For example, fertility-related outcomes have been shown to be influenced by genetic factors (Rodgers et al. 2001). Given that the presence of children in the home appears to be associated with health and well-being—both positively and negatively—these findings may pose important implications for sociological research in this area. In addition, stressful life events show strong negative correlations with health and well-being, particularly mental health (Kendler 2001; Kessler 1997). Important for the present study, several studies have found that genetic factors contribute to both the occurrence of stressful life events and to the resiliency individuals have to the negative effects of such environmental stressors (Charles and Almeida 2007; McGue and Lykken 1992; Kendler and Prescott 2006; Lyons et al. 1995). On another front, aspects of adolescent social life such as peer popularity also appear to be influenced by genetic factors (Iervolino et al. 2002).

What implications might these findings pose for traditional sociological research on health and well-being? To begin with, sociologists commonly assume that the observed correlation between measures of the environment and health provides support the social causation of health and well-being. Although this is undoubtedly true to some extent (the exact degree, however, is currently unknown), what if latent genetic factors actually do influence the very environmental predictors employed by sociologists? Would this not imply that genetic predispositions toward particular environmental outcomes might be at least partially responsible for the observed correlations with health? The present section reviewed research that already hints at the possibility that genetic differences predict

many of the social-environmental variables utilized by medical sociologists. These findings, which are akin to [genetic] selection effects, could challenge traditional sociological models in a manner that may even be more profound than the results reviewed above concerning direct genetic influences on health. The question now becomes: How can sociologists of health incorporate these findings into their explanatory frameworks?

Although not immediately obvious, this literature suggests an interesting potential relationship between genes, environments, and health. Shown as Model 4 in Figure 3.1—a correlated (i.e., mediated / confounded) genetic and environmental effects model—genetic factors may directly influence environmental variables, which then indirectly affect health and well-being. This implies that environmental factors might explain some of the genetic effects on health outcomes.

Even though this model has been largely ignored by sociologists of health, empirical support for it does exist in the literature. 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 (Spotts et al. 2005). Similarly, genetic influences on physical health have been shown to be correlated with genetic effects on socioeconomic status, a finding that suggests that genetic effects on physical may flow at least partially indirectly through SES (Lichtenstein et al. 1992). The best example of research in this area, however, is a study showing that genetic risk toward major depression increases the probability of experiencing stressful life events (Kendler and Karkowski-Shuman 1997). In essence, studies in this area suggest that genes are at least

partially responsible for the occurrence of deleterious social conditions in the lives of individuals, which are, in turn, associated with poor health (Charles and Almeida 2007; McGue and Lykken 1992; Kendler and Prescott 2006; Lyons et al. 1995). Overall, gene-environment correlation offers a potential pathway for genetic effects on health—i.e., they might be at least partially explained via environmental mediation, and thus may not be interpretable without reference to social environments. In propositional form, and based on Model 4 in Figure 3.1, this possibility suggests:

- Proposition 4b: Genetic influences on health are at least partially indirect via their effects on environmental variables (Pathway C), which then influence health and well-being (Model 4, Pathway B).

This model also suggests something troubling to traditional sociological research: the possibility that the relationship between environmental variables and health may be at least partially the result of exogenous genetic factors that explain at least part of the correlation between them. A submodel of Model 4 suggests that the direct effect of environmental influences on health (Pathway B) may be weak or absent when genetic factors are taken into consideration (Pathways A and C). In other words, genetic predispositions could help to account for the observed relationship between environmental variables and health. For example, we know that social support is phenotypically associated with desirable health outcomes (reviewed above), but in contrast to the typical sociological interpretation—e.g., that social support helps to buffer

against poor health or enhance mental and physical well-being—it might also be the case that genetic factors predispose individuals in some way toward (a) both high levels of social support and desirable health, or (b) both low levels of social support and poor health. (Note: Research of this type may also help to illuminate the much-debated causal order between these variables.)

Research on several aspects of health and well-being provide empirical evidence for this possibility (Neiderhiser et al. 1999; Reiss et al. 2000). For example, the associations between marital quality, social support and mental health appear to be explained in part by common genetic factors (Spotts et al. 2005), as does the relationships between parental divorce and earlier initiation of drug use (D'Onofrio et al. 2006). That said, much more research in this area is needed, and we do not currently know the degree to which this might pose problems for research that does not take genetic factors into consideration. Thus, this should be a major focus of research in the coming years. Stated in proposition form:

- Proposition 4c: Genetic factors influence both environmental variables (Model 4, Pathway C) and health outcomes (Pathway A), and when this effect is considered, the observed correlation between environmental variables and health outcomes (Model 4, Pathway B) may be reduced, and possibly even eliminated—i.e., genetic factors may be at least partially confounding the phenotypic relationship between environmental variables and health.

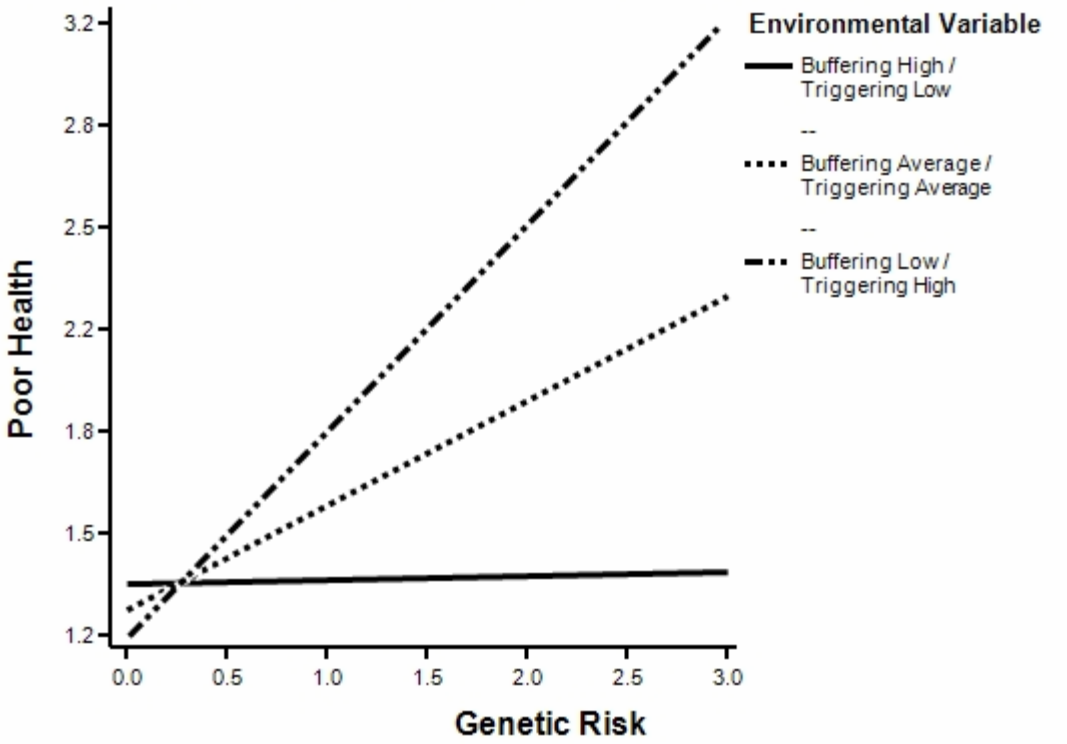
Models 5a and 5b: Gene-Environment Interaction

In addition to additive and correlated effects, there is at least one other way (two forms) in which genetic and environmental factors might influence health and well-being: gene-environment "interaction." Essentially, some published research suggests that (a) genetic effects on health may either be more or less pronounced depending upon environmental influences (Model 5a of Figure 3.1), and / or (b) that environmental influences may have different effects on health based on the genetic makeup of individuals (Model 5b of Figure 3.1) (Boomsma et al. 1999; Caspi et al. 2005a and b, 2003; Eley et al. 2004a and b; Jaffee et al. 2005; Jang et al. 2001; Kendler 2001; Kendler et al. 1999, 1995; Silberg et al. 2001; Shanahan and Hofer 2005).

Model 5a: Environmental Moderation of Genetic Effects

With respect to the first form—environmental moderation of genetic effects—research suggests (a) that desirable social environments may buffer against genetic liabilities toward poor health (or, alternatively, enhance ones for good health), and / or (b) that stressful social conditions may facilitate genetic risks for poor health (or, alternatively, offset ones for good health). These possibilities are grounded in an important assumption: genetic influences on health and well-being are not determinative, but are instead "predispositions" that are interconnected with, and perhaps even contingent upon, environmental conditions.

Figure 3.2: Environmental Moderation of Genetic Effects



To better understand this model, Figure 3.2 displays a hypothetical example for an undesirable health outcome such as depression or hypertension. The x-axis represents genetic risk defined as a continuous measure, while the y-axis shows poor health. In typical biomedical studies, genetic risk shows a strong positive correlation with levels of poor health. As depicted here, however, the influence of genetic risk may vary depending upon on or more environmental variables, which come in one of two primary forms: buffers against, and facilitators of, poor health (both of these are represented in the figure). In the case of environmental buffering—via variables such as support from a loving spouse, high levels of educational attainment, etc.—genetic predispositions toward

poor health may actually be nullified, to some extent, by environmental influences. In other words, genetic factors may have less of an influence on poor health when levels of a buffering environmental variable are high. Looking at the graph, at low levels of the environmental buffer, genetic risk is strongly positively associated with poor health, but as levels of the environmental variable increase, this relationship gets weaker (i.e., the slope gets flatter). At very high levels, genetic risk may actually bear no relationship at all with poor health.

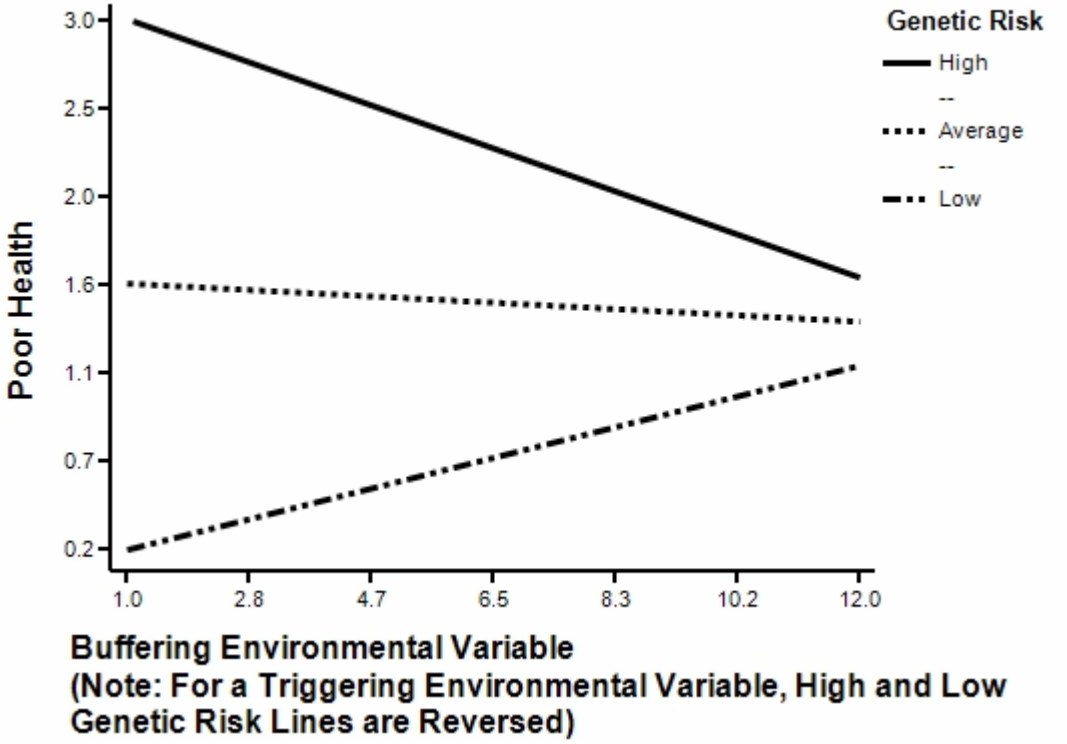
Empirical evidence, although not extensive, does support this possibility. For example, a recent study found that genetic influences on both chronic illnesses and body mass index (BMI) were lower at higher levels of income, suggesting that financial assets may provide individuals with resources that can be used to buffer against genetic risks for poor physical health (Johnson and Krueger 2005a). Other research has shown that genetic predispositions toward several undesirable outcomes, including disinhibition, neuroticism, anxiety, and depression, are moderated (i.e., impeded) by differences in one's religious upbringing (Boomsma et al. 1999). Likewise, the realization of genetic potential for intellectual development depends greatly on advantageous social environments (Guo and Stearns 2002). These findings are consistent with sociological theory (e.g., social capital theory or the life-stress paradigm), which suggests that individuals who possess adequate social resources (e.g., a high level of monetary income and a connection to others in a religious community) may be better suited to deal with undesirable health outcomes, perhaps even if they are genetically predisposed toward them.

As mentioned briefly above, in addition to buffering against genetic risks, environmental factors may also function to facilitate (i.e., trigger) genetic risk factors. In this case, which is also shown in Figure 3.2, genetic influences would be more strongly associated with poor health in the presence of environmental stressors—e.g., divorce, job loss, low levels of income, the death of a loved one, etc. Thus, in contrast to the buffering model where environments serve as resources that help to offset an unfortunate genetic heritage, in the triggering model environmental factors function to exacerbate the deleterious effects of genetic risk factors.

Empirical research suggests support for this model by showing that negative life events, family problems, a lack of social connections, and unemployment, among others, help to exacerbate (and possibly even trigger) genetic influences on poor health outcomes (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. 1999, 1995; Silberg et al. 2001). The most cited example in this area is that of PKU, which is a genetic disorder that facilitates mental retardation only when accompanied by certain dietary (i.e., environmental) stressors (Omenn 2000; Shostak 2003). In propositional form, the environmental moderation of genetic effects from Model 5a of Figure 3.1 suggests:

- Proposition 5a: Environmental influences moderate (Model 5a, Pathway D) genetic influences on health (Pathway A): some environmental variables serve as buffers against genetic risks for poor health, while others serve as triggers or facilitators of genetic risks.

Figure 3.3: Genetic Moderation of Environmental Effects



Model 5b: Genetic Moderation of Environmental Effects

Other research on gene-environment interaction hints at a statistically identical yet theoretically distinct possibility: genetic moderation of environmental influences (Model 5b of Figure 3.1). The literature suggests that the effects of environmental variables on health may actually be stronger among certain individuals compared with others based on genetic differences. To clarify this possibility, a hypothetical graphical representation is depicted in Figure 3.3. In many sociological studies (reviewed above), buffering

environmental variables such as high levels of social support or educational attainment, the x-axis, are shown to be inversely associated with poor health, the y-axis. This association may, however, be contingent upon genetic risk factors. Looking at the graph, among individuals at high genetic risk, the negative relationship between the environmental buffer and poor health is clearly evident. At low genetic risk, however, that association no longer holds. Thus, environmental buffering may only be important among individuals who are at high genetic risk for poor health, not among the population as a whole. A comparable triggering or facilitating effect may also be occurring (see the note in Figure 3.3). In this case, environmental stressors such as divorce or low income may be especially problematic among individuals with high genetic risks for poor health, but among others, they may not be as important. This triggering model can be graphically represented by reversing the high and low genetic risk lines in Figure 3.3.

Since (a) most of the research in this area has focused on the impact of environmental stressors (i.e., facilitators) on genetic effects, and (b) a relative dearth of research has addressed how genetic factors might moderate the effects of beneficial environments, empirical support for the buffering version of Figure 3.3 is difficult to marshal. That said, many of the studies reviewed above likely support it despite the fact that they were interpreted solely in terms of environmental moderation of genetic effects. This is unfortunate given the fact that a significant gene-environment interaction implies (at least statistically) both environmental moderation of genetic effects and genetic moderation of environmental effects. Most scholars, however, have inexplicably ignored the latter possibility, at least with respect to buffering contexts.

Considerable support does exist, however, for the triggering version of Figure 3.3. For example, 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). In this study, 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 suggests 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). Another study showed that the relationship between stressful life events and depression was stronger among individuals at high genetic risk compared with individuals at low genetic risk (Kendler et al. 1995).

As reviewed elsewhere (Moffitt et al. 2005, 2006), there is also support for gene-environment interactions of this type for a variety of physical health outcomes. For example, some individuals (but not others) who have high dietary fat intakes develop cholesterol problems and cardiovascular disease depending upon their genotype on the polymorphic hepatic lipase gene promoter. Some tobacco smokers develop coronary heart disease, but others do not, apparently based on variants in the lipoprotein lipase and apolipoprotein E4 (APOE4) genes. There is even evidence for similar gene-environment interactions in the manifestation of cancer, as well as infectious diseases such as malaria, human immunodeficiency virus and AIDS, leprosy, and tuberculosis.

In addition to mental and physical health, research on human development in general is important here as well. For example, the literature shows that some individuals are more resilient to the negative impact of socioeconomic deprivation than others (Elder 1999). Recent evidence suggests that this may be due to the fact that resiliency to social stressors is at least partly heritable. Further, a molecular genetic study in this area found that a functional polymorphism in the promoter region of a gene encoding monoamine oxidase A moderated the effect of child maltreatment on the cycle of violence (Caspi et al. 2002). These results showed that maltreated children whose genotype conferred low levels of monoamine oxidase A expression more often developed conduct disorder, antisocial personality, and adult violent crime than children with a high-activity monoamine oxidase A genotype. Although much work remains to be done in this area, there is certainly enough evidence to suggest that environmental influences may be contingent upon genetic differences for a variety of health outcomes. In propositional form, Model 5b of Figure 3.1 suggests:

- Proposition 5b: Genetic influences moderate (Model 5b, Pathway E) environmental influences on health (Pathway B): some environmental variables serve as buffers against genetic for poor health among certain individuals but not others based on their level of genetic risk, while others serve as triggers or facilitators of poor health among certain individuals but not others based on their level of genetic risk.

CONCLUSION

In light of the relative dearth of theoretical research on the potential interconnections between genetic and environmental influences on health and well-being, this chapter outlined five possible models: (1) genetic-only effects; (2) environment-only effects; (3) additive genetic and environmental effects; (4) correlated genetic and environmental effects; (5a) environmental moderation of genetic effects; and (5b) genetic moderation of environmental effects. The final two empirical chapters will examine each of these models in detail for several different social-environmental variables, and one specific health outcome: psychological distress.

CHAPTER 4

Gene-Environment Correlation in Research on Social Relationships and Psychological Distress: Evidence, Theory, and Implications

SUMMARY

A considerable amount of research has examined the social and genetic causes of psychological distress. As described in the previous chapter, however, very little research has simultaneously examined both of these influences, and it is currently unknown whether their effects are (a) additive, or (b) correlated (Research Question 3). Given that genetic factors have been linked with both social relationships and psychological distress, it is likely that the latter is occurring, to at least some extent. Results presented in this chapter show: (a) that many different social relationship variables are phenotypically correlated with psychological distress; (b) that genetic factors account for non-trivial proportions (15-52%) of all of these associations; (c) that social relationships, for the most part, are correlated with distress net of genetic influences; and (d) that at least part of the genetic effect on psychological distress is likely indirect via social relationships. It is concluded that the failure to address genetic influences could pose at least two major implications for research on social relationships and psychological distress: (a) gene-environment correlation and possibly even genetic-confounding, and (b) potentially erroneous conclusions regarding cause and effect.

INTRODUCTION

On one front, a large sociological literature has examined the connection between social relationships and psychological distress (Ellison and Levin 1998; Glenn and Weaver 1981; Gove, Style, and Hughes 1990; Lu and Hu 2005; Mirowsky and Ross 2003; Noor 1996; Regnerus et al. 2004). Virtually all of this research shows that socially integrated individuals—i.e., those who possess extensive, high-quality social relationships—experience better mental health than individuals who lack social attachments (Cohen 2004; House 2002). On another front, a considerable number of biomedical studies have linked genetic differences with psychological distress as well (Caspi et al. 2003; Eley 1997; Hamann 2005; Lesch et al. 1996; Pezawas et al. 2005). To date, virtually all of this research has assumed that these two influences are independent of one another (i.e., that their effects are additive and orthogonal).

Based on a bourgeoning literature, the present study argues that this assumption is likely to be false, and that social and genetic influences on psychological distress are instead correlated (Kendler and Prescott 2006). This contention is based on research showing that genetic influences predict individual-level variation on: (a) social relationships (Boorman and Levitt 1980; Bouchard and Loehlin 2001; Bradshaw and Ellison Forthcoming; Cleveland and Crosnoe 2004; Kendler 1997); (b) psychological distress (Caspi et al. 2003; Gottesman 1991; Hamann 2005; Lesch et al. 1996; Pezawas et al. 2005); and (c) even the "correlation" between these two aspects of social life (Reiss et al. 2000; Rutter, Moffitt, and Caspi 2006; Spotts et al. 2005). These findings suggest several, currently unanswered, questions: Do social relationships and genetic differences

influence psychological distress independently, or is there at least some overlap in their explanatory power? Do unmeasured genetic factors bias sociological research on social relationships and psychological distress; if so, to what extent? Do social relationships influence psychological distress net of latent genetic factors? Do genetic differences influence social relationships; if so, do they explain why social relationships are correlated with psychological distress? Are genetic influences on psychological distress indirect via their effects on social relationships?

To address these questions, three distinct, and largely isolated, areas of scholarship will be initially reviewed: (a) the sociological literature on this topic; (b) biomedical research on genetic influences and psychological distress; and (c) a small but rapidly growing body of findings showing that genetic factors also influence social relationships. Based on this theoretical and empirical background, a conceptual model and four theoretical propositions will be formulated, and then empirically tested using genetically-informative twin sibling data from the National Survey of Midlife Development in the United States (MIDUS). The results of these analyses will then be summarized, and their implications for the sociological study of psychological distress will be discussed. The paper will conclude by outlining an agenda for future research in this area.

THEORETICAL AND EMPIRICAL BACKGROUND

Social Relationships and Psychological Distress

Grounded in theoretical insight from the life stress paradigm, human capital theory, and social capital theory, among others, sociological research argues that social relationships

influence psychological distress and other mental health outcomes primarily by shaping (a) exposure to social stressors (i.e., social conditions that strain one's ability to respond), and (b) different degrees of vulnerability to those stressors (i.e., the quantity and quality of available psychosocial resources that one can draw on when dealing with life's difficulties) (Cohen 2004; Ellison 1994; Ellison et al. 2001; Ensel and Lin 1991; Mirowsky and Ross 2003). In this literature, social relationships are measured with a diverse assortment of outcomes, including socially-linked personality characteristics, interpersonal relations, family ties, socioeconomic status, and broader social contexts such as community attachment and neighborhood quality, among others (Cohen 2004; Glenn and Weaver 1981; Gove, Style, and Hughes 1990; House 2002; Lu and Hu 2005; Noor 1996; Regnerus et al. 2004). Let us take a look at the published research in each of these areas.

To begin with, several different social-oriented personality traits have been linked with psychological distress. Extraverted and agreeable individuals tend to be more enthusiastic, talkative, cooperative, and empathetic compared introverted and disagreeable individuals (Baker and Daniels 1990; Bouchard and Loehlin 2001; Eaves and Eysenck 1975; Eysenck 1990; Lachman and Weaver 1997). Importantly, these characteristics have, in turn, been linked with desirable mental health (Costa and McCrae 1980; Duckitt 1984; Larsen and Kasimatis 1990; Lu and Hu 2005; Noor 1996; Ozer and Benet-Martinez 2006; Tanaka et al. 1998). In contrast, neuroticism—which is characterized by traits such as moody, worrying, and nervous—is associated with poor

mental outcomes, including the development of major depression over time (Kendler et al. 2006).

In a related area of scholarship, research also shows that individuals who are able to form and maintain positive relations with others experience less distress than those who are not (Beach and O'Leary 1993; Glenn and Weaver 1981; Gove, Hughes, and Style 1983; Keicolt-Glaser and Newton 2001; Mirowsky and Ross 2003; Prigerson, Maciejewski, and Rosenheck 1999). Similarly, research has consistently shown that individuals who interact well with others have larger and denser social networks, as well as enhanced socioemotional support systems, both of which pose implications for psychological well-being (Ellison and Levin 1998; House 2002).

Family relations also matter, with research showing that married individuals experience lower levels of psychological distress than single persons (Mirowsky and Ross 2003). In addition, individuals in good marriages report being happier (Gove, Hughes, and Style 1983), and experiencing fewer depressive symptoms (Spotts et al. 2004), than those in poor marriages. The primary sociological explanation for these findings is the fact that spouses are a major source of psychological and social support (Beach and O'Leary 1993).

Socioeconomic status also affects psychological distress, primarily by shaping differences in social stressors and vulnerabilities. Individuals with high SES tend to have fewer difficulties in paying bills, obtaining health care, and meeting family needs (Pearlin et al. 1981; Ross and Van Willigen 1997). Upper SES individuals also benefit from more extensive and functional social networks and support systems, which provide emotional,

tangible, and informational support that may help to buffer against the many difficulties that life presents (Cohen 2004; Lin et al. 1999). It has even been shown that SES is positively associated with the possession of psychological and cognitive resources such as feelings of self-worth, a sense of control, and personal efficacy, as well as successful coping strategies, all of which are linked with psychological well-being (Mirowsky and Ross 2003).

Social contexts at more macro levels also appear to influence psychological distress. For example, research has shown that community satisfaction and attachment are both associated with individual well-being and depressive symptomology (Fitzpatrick and LaGory 2003; O'Brien, Hassinger, and Dershem 1994; Theodori 2001). In a related area of scholarship, several studies have found that neighborhood characteristics (e.g., living in an area with graffiti, run down buildings, etc., compared with an aesthetically pleasing one that is perceived as safe) are associated with mental health outcomes as well (Mirowsky and Ross 2003). Overall, then, many different aspects of social life have been linked with psychological distress.

Genetic Influences on Psychological Distress

In addition to social relationships, a sizable literature has demonstrated that genetic factors also influence levels of psychological distress. For example, based on findings from twin an adoption studies, affective disorders such as depression—which are characterized by feelings of worthlessness, sadness, and hopelessness, as well as sleep and appetite irregularities—appear to be strongly influenced by both genetic and

environmental factors (Baker, Cesa, and Gatz 1992; Blehar et al. 1988; Eley 1997; Hamer and Copeland 1998; Plomin 1990; Plomin and Rende 1991). This research broadly suggests that genetic influences explain 30% or more of the individual-level variation on affective disorders. Anxiety and panic disorders have also been examined in this context, and research suggests sizable genetic effects on these outcomes as well (Crowe et al. 1983; Eley 1997).

Several different personality correlates of psychological distress and other mental health outcomes also appear to be strongly influenced by genetic factors (Caspi et al. 2005a and b; Jang, Livesley, and Vernon 1996). In a review of this literature, Bouchard and Loehlin (2001) reported that genetic influences predict individual variation on neuroticism, psychoticism, negative emotionality, aggression-hostility, anti-social behavior, and harm avoidance, among others. Importantly, many of these traits, particularly neuroticism and harm-avoidance, are associated with psychological distress (Ozer and Benet-Martinez 2006; Lesch et al. 1996). In general, genetic and environmental influences each appear to explain roughly half of the variation on all personality characteristics, and it is presumed that much of this genetic effect is manifested in a variety of mental health outcomes.

In addition to evidence from twin and adoption designs, a growing number of molecular genetic studies have linked differences at the molecular (i.e., DNA) level with psychological distress as well. For example, several studies have found that levels of anxiety and depression vary depending upon which version (i.e., allele) of the polymorphic serotonin transporter gene an individual inherited (Hamann 2005; Lesch et

al. 1996; Pezawas et al. 2005). Additional examples of this type of research can be found in Binder et al. (2008) and Gross and Hen (2004), among many others. Overall, then, genetic differences appear to be important predictors of many, and perhaps all, aspects of psychological well-being.

Genetic Influences on Personality and Social Relationships

In addition to psychological distress, a small but rapidly growing literature shows that genetic factors also influence the quality and quantity of one's social relationships. To begin with, several publications have shown that individual-level variation on social-oriented personality traits, including extraversion and agreeableness, among others, is the product of both genetic and environmental influences (Baker and Daniels 1990; Bouchard and Loehlin 2001; Eaves and Eysenck 1975). Research on this topic has been conducted using twin, adoption, and molecular genetic designs, and all three suggest that genetic differences are important predictors of each of these outcomes (Jang, Livesley, and Vernon 1996; Plomin and Caspi 1998).

Other prosocial orientations, such as positive interpersonal relations with others, cooperation, and dependability, among others, are also inluenced by both genetic and environmental factors (Axelrod and Hamilton 1981; Boorman and Levitt 1980; Bouchard and Loehlin 2001; Kenrick, Li, and Butner 2003; Kirkpatrick 2004; Newberg, D'Aquili, and Rause 2002; Plomin 1990; Wilson 1998). Research has even shown that genetic factors influence variation on social network characteristics (Cleveland and Crosnoe 2004), as well as the amount of social support of individual receives (Kendler 1997).

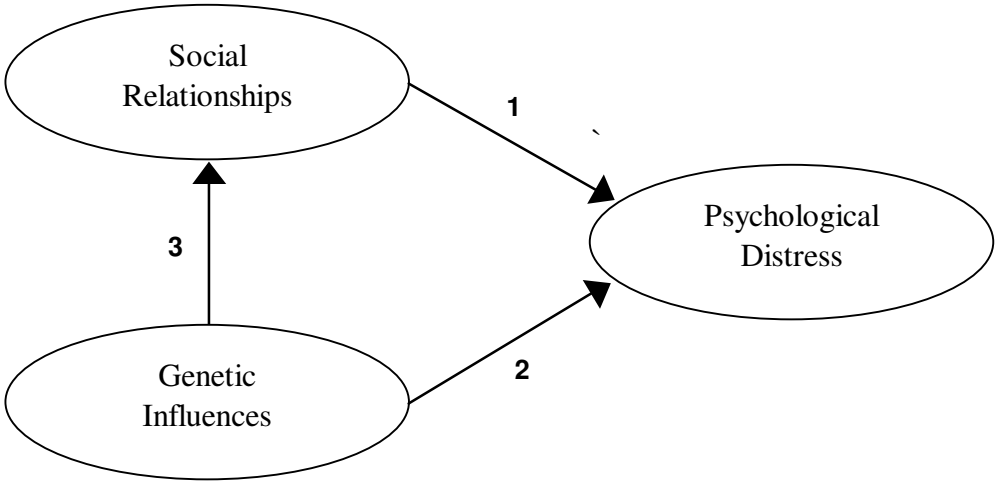
Genetically-informed techniques have even been applied to family-related outcomes. For example, research shows that genetic factors influence individual variation on three different domains of parent-child relations: positivity (e.g., warmth, empathy, etc.), negativity (e.g., disputes, anger, etc.), and control (e.g., monitoring, knowledge of activities, etc.) (Plomin et al. 1994). In addition, data from twin women and their partners was recently used to provide evidence for genetic effects on another aspect of family life: marital quality (Spotts et al. 2004). Genetic effects have even been reported for divorce (Jerskey et al. 2001; McGue and Lykken 1992) and other relevant outcomes such as the likelihood of ever marrying (Jerskey et al. 2001; Trumbetta and Gottesman 2000).

Genetically-informed research on socioeconomic status has also been conducted, and genetic influences appear to account for as much as half of the variation on educational attainment (Behrman et al. 1980; Heath et al. 1985; Tambs et al. 1989; Vogler and Fulker 1983). Similarly, both grade-point average and college aspirations among adolescents and young adults appear to be influenced by genetic differences (Nielsen 2006). Individual variation on personal income seems to be at least partially the product of latent genetic predispositions as well (Rowe et al. 1998).

To date, no studies have examined whether genetic factors influence individual variation on broader measures of one's social context such as community attachment or neighborhood quality. Given that genetic effects have been found on virtually all other aspects of social life, however, it is likely that they are important for these outcomes as well, especially when theorized within the context of research showing that group-forming behaviors of all types are at least partially the products of genetic predispositions

(Axelrod and Hamilton 1981; Boorman and Levitt 1980; Bouchard and Loehlin 2001; Kenrick, Li, and Butner 2003; Kirkpatrick 2004; Newberg, D'Aquili, and Rause 2002; Taylor 2002; Wilson 1998). Overall, then, genetic factors appear to influence many different aspects of social life, including several that have been linked with psychological distress.

Figure 4.1: A Conceptual Model of Social Relationships, Genetic Influences, and Psychological Distress



A Conceptual Model and Four Theoretical Propositions

Viewed in their entirety, these diverse findings suggest the conceptual model shown in Figure 4.1, which implies four propositions concerning the connections between social relationships, genetic influences, and psychological distress. The first three are grounded in the three disparate literatures reviewed above, respectively:

- Proposition 1: There is a phenotypic correlation between social relationships and psychological distress (Pathway 1).

- Proposition 2: Genetic differences predict individual-level variation on psychological distress (Pathway 2).

- Proposition 3: Genetic differences predict individual-level variation on the quality and quantity of social relationships (Pathway 3).

Since genetic factors are theorized to predict both the independent "and" dependent variables in this model (i.e., social relationships and psychological distress, respectively), it is therefore possible that they influence the "correlation" between these two outcomes as well. This insight suggests an additional proposition concerning the potential impact of genetic influences on the sociological (i.e., phenotypic) association between social relationships and psychological distress:

- Proposition 4: Genetic influences common to both social relationships (Pathway 3) and psychological distress (Pathway 2) account for at least a portion of the phenotypic correlation between these two outcomes (Pathway 1).

Findings in support of this proposition, which suggests gene-environment correlation, could be interpreted in two different ways. The first one concerns the mediation of genetic effects on psychological distress by social relationships. Looking at the conceptual model shown in Figure 4.1, and based on the literature reviewed above, it is possible that genetic factors directly influence social relationships (Pathway 3), which, in turn, indirectly affect psychological distress (Pathway 1) (Reiss et al. 2000; Spotts et al. 2005). If this scenario is supported by the data, it would offer a potential "explanation" for genetic effects on psychological distress—i.e., they are at least partially indirect via their influence on social relationships. The second interpretation takes the form of genetic confounding, where genetic differences account for all or part of the connection between social relationships and psychological distress. In other words, social relationships and psychological distress could be correlated merely by virtue of their shared associations with one or more common exogenous third variables—i.e., genetic factors—that lead to a correlation that is not the result of the former influencing the latter. If this is occurring, the effect of Pathway 1 would be weak or absent when Pathways 2 and 3 are taken into consideration. The implications of these two interpretations are discussed in detail below.

METHODS

Data

To empirically examine these four propositions, the present study employs 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). This data was collected 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 (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. The final N used in all of the analyses is 600 twin pairs (1,200 individuals).

Dependent Variable

Psychological distress, the mental health outcome employed here, is gauged with a mean index composed of the following six questions (Cronbach'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 is the K6 scale of psychological distress developed by Kessler and colleagues (2002). It distinguishes negative affect based on the type and amount of severity of the problem, rather than diagnosis (i.e., each question of the index is asked of all respondents, not just the ones who preliminarily screened positive for a diagnosed mental illness). This measure was specifically designed for use in surveys such as the MIDUS, and it is excellent for broad-gauged screening of negative affect, and can discriminate DSM-IV cases from non-cases. To reduce the skewed nature of this variable (normal variable skewness = 2.00), it is normalized via an inverse square-root transformation (transformed variable skewness = 0.65), and the order is then reversed so that higher scores indicate more distress.

Independent Variables

Genetic Risk for Psychological Distress

As one might expect, measuring genetic risk factors for psychological distress is not an easy task. Complex outcomes such as 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 1996). (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 genetically-identical MZ co-twin both

experience a high level of distress, then these individuals must 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 who 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).

Personality

Social-oriented personality characteristics are gauged with three different outcomes. To measure extraversion, which taps an innate desire for social relationships, respondents were asked to indicate how well each of the following described them: "outgoing, friendly, lively, active, and talkative." A mean index (alpha = 0.78) is constructed from these five items (Lachman and Weaver 1997), all of which are coded 1 = not a lot to 4 = a lot. The measure of agreeableness is similar, but the adjectives employed are: "helpful, warm, caring, softhearted, and sympathetic" (alpha = 0.80). The adjectives used to describe neuroticism are: "moody, worrying, nervous, and calm (reverse coded)" (alpha = 0.74).

Interpersonal Relations

Positive relations with others—a broad-based measure of social relationships at the interpersonal level—is gauged with a three-item mean indexed measure constructed from the following questions (alpha = 0.62) (Ryff 1989; Ryff and Keyes 1995): (a) "Maintaining close relationships has been difficult and frustrating for me (reverse coded)." (b) "People would describe me as a giving person, willing to share my time with others." and (c) "I have not experienced many warm and trusting relationships with others (reverse coded)." Each of these items is coded 1 = disagree strongly to 7 = agree strongly.

Family Ties

Family relations are tapped with three different measures. Marriage is a dichotomous variable (married = 1). Partner support is constructed from the following questions, each of which is coded 1 = not at all to 4 = a lot (alpha = 0.86): (a) “How much does your spouse or partner really care about you?” (b) “How much does he or she understand the way you feel about things?” (c) “How much does he or she appreciate you?” (d) "How much do you rely on him or her for help if you have a serious problem?” (e) “How much can you open up to him or her if you need to talk about your worries?” and (f) “How much can you relax and be yourself around him or her?” Given that quite a few individuals reported not having a significant other, there were a considerable number of missing cases on this measure. To keep from loosing these cases, a mean imputation was performed. Additional analyses (not shown), which included a flag variable (which was not statistically significant), revealed that this imputation did not alter the findings reported in this paper. Non-partner support is constructed from the following questions, each of which is coded 1 = not at all to 4 = a lot (alpha = 0.82): (a) “Not including your spouse or partner, how much do members of your family really care about you?” (b) “How much do they understand the way you feel about things?” (c) “How much can you rely on them for help if you have a serious problem?” and (d) “How much can you open up to them if you need to talk about your worries?”

Socioeconomic Status and Employment

SES is operationalized with three 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 the income measure, 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. Unemployment is a dichotomous variable (unemployed = 1).

Social Context

Community attachment, a measure of social relationship quality at a more macrosocial level, is a 3-item mean index (alpha = 0.74) (Keyes 1998). This measure, which gauges the respondent's assessment of their level of integration into their community, is

constructed from the following questions, each of which is coded 1 = disagree strongly to 7 = agree strongly: (a) "I don't feel I belong to anything I'd call a community (reverse coded)." (b) "I feel close to other people in my community." and (c) "My community is a source of comfort." Neighborhood quality is gauged with the following questions, each of which is coded 1 = not at all to 4 = a lot (alpha = 0.73): (a) "I feel safe being out alone in my neighborhood during the daytime." (b) "I feel safe being out alone in my neighborhood at night." (c) "I could call on a neighbor for help if I needed it." and (d) "People in my neighborhood trust each other."

Covariates

Previous research suggests that levels of psychological distress vary by both *age* and *gender* (Mirowsky and Ross 2003; Mroczek and Kolarz 1998), so the present study controls for the effects of each.

Modeling Techniques

Bivariate and multivariate OLS, ordered logistic, and binary logistic regression techniques are used to examine (a) the phenotypic correlations between social relationships and psychological distress, (b) the influence of genetic risk factors on psychological distress, (c) the influence of genetic risk factors on social relationships, (d) the proportion of the correlation between social relationships and distress that is explained by genetic risk factors, and (e) the proportion of the genetic effect on distress that is mediated by social relationships.

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 4.1: Descriptive Statistics

	Mean	Std Dev	Min-Max
Psychological Distress	1.5018	0.6031	1-5
Genetic Risk for Psychological Distress	0.6453	0.7643	0-3
Personality			
Extraversion	3.2204	0.5604	1-4
Agreeableness	3.5302	0.4618	1-4
Neuroticism	2.2373	0.6552	1-4
Interpersonal Relations			
Positive Relations with Others	5.5024	1.3425	1-7
Family Ties			
Married	0.7467	-	0-1
Partner Support	3.6127	0.5483	1-4
Other Family Support	3.9799	0.6513	1-5
Socioeconomic Status and Employment			
Education	2.8893	1.1351	1-5
Income	18.2301	9.6087	1-31
Unemployed	0.0183	-	0-1
Social Context			
Community Attachment	4.8433	1.4292	1-7
Neighborhood Quality	3.4260	0.5358	1-4
Age	44.8943	12.1229	25-74
Sex (Female=1)	0.5803	-	0-1

Notes: N=600 twin pairs (1,200 individuals)

RESULTS

Table 4.1 shows descriptive statistics for all of the study variables. With respect to psychological distress, the mean is quite low: 1.50 on a scale with a range of 1-5. The average genetic risk is also low, with a mean of 0.65 on a 0-3 scale. Respondents report relatively high levels of extraversion and agreeableness (means = 3.22 and 3.53, respectively, on 1-4 scales), and the mean for neuroticism is 2.24 on a 1-4 scale. Positive relations with others has a mean of 5.50 on a 1-7 scale. Roughly 75% of the sample is

married, and support from family members, both partners and others, is quite high: means = 3.61 on a 1-4 scale and 3.98 on a 1-5 scale, respectively. The average respondent has obtained some college education, and the mean for personal income is 18.23, which represents slightly more than $15,000 / year. Only about 2% of this sample is unemployed. Community attachments are quite common (mean = 4.84 on a 1-7 scale), and so is perceived neighborhood quality (mean = 3.43 on a 1-4 scale). With respect to other demographics, the average age is 44.89 years, and 58% of the sample is female.

Table 4.2: OLS Parameter Estimates from Bivariate (Model 1) and Multivariate (Model 2) Regressions of Social Relationship Variables (Independent Variables) on Psychological Distress (Dependent Variable)

	(1) Bivariate Analyses		(2) Multivariate Analysis[1]	
	Parameter Estimates	Adj. R-Square	Parameter Estimates	Standardized Estimates
Personality				
Extraversion	-0.0606 ***	0.0651	-0.0393 ***	-0.1672
Agreeableness	-0.0286 **	0.0082	0.0210 +	0.0738
Neuroticism	0.0840 ***	0.1700	0.0637 ***	0.3175
Interpersonal Relations				
Positive Relations with Others	-0.0291 ***	0.0842	-0.0078 *	-0.0791
Family Ties				
Married	-0.0148 ns	0.0015	0.0046 ns	0.0152
Partner Support	-0.0702 ***	0.0674	-0.0401 ***	-0.1502
Other Family Support	-0.0435 ***	0.0439	-0.0126 +	-0.0623
Socioeconomic Status and Employment				
Education	-0.0120 **	0.0094	-0.0032 ns	-0.0276
Income	-0.0018 ***	0.0134	-0.0012 **	-0.0891
Unemployed	0.1161 ***	0.0132	0.0648 +	0.0662
Social Context				
Community Attachment	-0.0027 ***	0.0642	-0.0008 +	-0.0756
Neighborhood Quality	-0.0531 ***	0.0443	-0.0152 +	-0.0619

Notes: N=600 twin pairs (1,200 individuals); all models are adjusted for age and sex, and Adjusted R-squares are net of these two covariates, which explain 1.98% of the variation.

1. Multivariate model estimates all social relationship variables net of the others, and the Adj. R-square for the combined effects of these variables is 0.2846.

***p<0.001 **p<0.01 *p<0.05 +p<0.10 (two-tailed tests)

Table 4.2 shows OLS parameter estimates from bivariate and multivariate regressions of each of the social relationship measures on psychological distress. As expected, and in line with previous research, the bivariate analyses show that extraversion (b = -0.0606, p<0.001) and agreeableness (b = -0.0286, p<0.01) are both inversely associated with distress, while neuroticism (b = 0.0840, p<0.001) is positively associated with this mental health outcome. Positive relations with others is also inversely associated with

psychological distress (b = -0.0291, p<0.001). In contrast to previous research, there does not appear to be a strong relationship between being married and levels of distress (b = -0.0148, ns), but both partner (b = -0.0702, p<0.001) and other family (b = -0.0435, p<0.001) support are inversely correlated with levels of psychological distress. Consistent with a large number of studies, socioeconomic status has a strong relationship with distress, with both education (b = -0.0120, p<0.01) and personal income (b = -0.0018, p<0.001) being inversely associated with it, and unemployment showing a positive correlation (b = 0.1161, p<0.001). Finally, both community attachment (b = -0.0027, p<0.001) and neighborhood quality (b = -0.0531, p<0.001) are inversely associated with psychological distress in this sample. Model 2 shows multivariate estimates of each of these associations net of the others. These analyses are shown solely for readers interested in making comparisons of the explanatory power of each of the predictors relative to the others. They should, however, be viewed with caution since (a) each of these measures is highly correlated with the others, and (b) there is no reason to expected them to be completely independent of one another. Overall, the findings shown in this table provide considerable support for Proposition 1, which claims that social relationships are correlated with psychological distress.

Table 4.3: OLS Parameter Estimate from the Bivariate Regression of Genetic Risk for Psychological Distress (Independent Variable) on Psychological Distress (Dependent Variable)

	Parameter Estimate	Adj. R-Square
Genetic Risk for Psychological Distress	0.0673 ***	0.1482

Notes: N=600 twin pairs (1,200 individuals); model is adjusted for age and sex, and the adjusted R-square is net of these two covariates, which explain 1.98% of the variation.

***p<0.001 **p<0.01 *p<0.05 +p<0.10 (two-tailed test)

For genetic factors to bias the sociological findings shown in Table 4.2, two conditions must be met: (1) genetic difference must predict psychological distress; and (2) the same genetic factors must also influence social relationships. Table 4.3 addresses the first condition by providing results from a bivariate OLS regression analysis of the effect of genetic influences on psychological distress. Consistent with Proposition 2, genetic factors are indeed strongly associated with distress ($b = 0.0673$, $p<0.001$). The magnitude of this effect is actually quite large, with predicted mean levels of distress being more than an entire unit higher (on the 1-5 unit scale of the non-transformed distress variable) among individuals at high genetic risk (mean = 2.690) compared with those at low genetic risk (mean = 1.365).

Table 4.4: OLS and Logistic Parameter Estimates from Bivariate Regressions of Genetic Risk for Psychological Distress (Independent Variable) on Social Relationship Variables (Dependent Variables)

	Parameter Estimates	Adj. R-Square
Personality		
Extraversion	-0.0781 ***	0.0104
Agreeableness	0.0042 ns	0.0008
Neuroticism	0.2331 ***	0.0722
Interpersonal Relations		
Positive Relations with Others	-0.3010 ***	0.0278
Family Ties		
Married[1]	-0.1143 ns	0.0022
Partner Support	-0.0940 ***	0.0203
Other Family Support	-0.0599 +	0.0041
Socioeconomic Status and Employment		
Education[2]	-0.3550 ***	0.0225
Income	-1.4114 ***	0.0115
Unemployed[1]	0.5901 **	0.0316
Social Context		
Community Attachment	-0.2665 ***	0.0186
Neighborhood Quality	-0.0761 ***	0.0114

Notes: N=600 twin pairs (1,200 individuals).

1. Estimated with binary logistic regression.

2. Estimated with ordered logistic regression.

***p<0.001 **p<0.01 *p<0.05 +p<0.10 (two-tailed tests)

To address the second condition, the next step in the analytic process is to examine whether genetic risk for psychological distress also influences social relationships. Table 4.4 shows results from bivariate regression analyses predicting each of the social relationship variables (separately) with genetic risk for psychological distress (note: the social relationship variables are the dependent variables in this table, even though they are in the same position in the table as they were when they were utilized previously as

independent variables). From this table we can see that a correlation exists between genetic risks for distress and all of the measures of social relationships except for two: agreeableness and marriage. Even though these effects may initially look small (Adj. R-squares range from 0.00 to 0.07), when we take into account the amount of the variation on psychological distress that is explained by each of these social relationship variables, these estimates are in fact quite large. As we will see below, these effects are large enough to cause profound implications for sociological studies that do not take them into consideration. Thus, consistent with Proposition 3, these findings suggest that the same genetic factors that predispose individuals toward high levels of psychological distress are also responsible for non-trivial proportions of the variation on many different aspects of social life, including extraversion, neuroticism, positive relations with others, family support, education, personal income, unemployment, community ties, and even perceptions of neighborhood quality. What we have here, then, is a scenario where (a) social relationships are correlated with psychological distress, and (b) the same genetic factors simultaneously affect both social relationships and distress. This means that it is possible that genetic factors are confounding the observed relationships between these outcomes. To examine the extent of this influence, however, requires one more step.

Table 4.5: OLS Parameter Estimates from Bivariate and Multivariate Regressions of Social Relationship Variables (Independent Variables) on Psychological Distress (Dependent Variable) Without (Models 1 and 3) and With (Models 2 and 4) Controls for Genetic Risk for Psychological Distress (Independent Variable), as Well as the Percentage of Each Relationship that is Explained by Genetic Risk for Psychological Distress

	Bivariate Analyses			Multivariate Analyses		
	(1) Without Considering Genetic Effects[1]	(2) Net of Genetic Effects	% Explained by Genetic Effects	(3) Without Considering Genetic Effects[1]	(4) Net of Genetic Effects	% Explained by Genetic Effects
Personality						
Extraversion	-0.0606 ***	-0.0507 ***	0.1634	-0.0393 ***	-0.0360 ***	0.0840
Agreeableness	-0.0286 **	-0.0243 **	0.1503	0.0210 +	0.0184 ns	0.1238
Neuroticism	0.0840 ***	0.0677 ***	0.1940	0.0637 ***	0.0537 ***	0.1570
Interpersonal Relations						
Positive Relations with Others	-0.0291 ***	-0.0226 ***	0.2234	-0.0078 *	-0.0061 +	0.2179
Family Ties						
Married	-0.0146 ns	-0.0113 ns	0.2260	0.0046 ns	0.0045 ns	0.0217
Partner Support	-0.0702 ***	-0.0573 ***	0.1838	-0.0401 ***	-0.0353 ***	0.1197
Other Family Support	-0.0435 ***	-0.0365 ***	0.1609	-0.0126 +	-0.0123 +	0.0238
Socioeconomic Status and Employment						
Education	-0.0120 **	-0.0058 ns	0.5167	-0.0032 ns	-0.0001 ns	0.9688
Income	-0.0018 ***	-0.0013 **	0.2778	-0.0012 **	-0.0010 *	0.1667
Unemployed	0.1161 ***	0.0893 **	0.2308	0.0648 +	0.0553 +	0.1466
Social Context						
Community Attachment	-0.0027 ***	-0.0022 ***	0.1852	-0.0008 +	-0.0006 ns	0.2500
Neighborhood Quality	-0.0531 ***	-0.0442 ***	0.1676	-0.0152 +	-0.0151 ns	0.0066

Notes: N=600 twin pairs (1,200 individuals); all models are adjusted for age and sex; multivariate analyses provide estimates of all social relationship variables net of the others.

***p<0.001 **p<0.01 *p<0.05 +p<0.10 (two-tailed tests)

1. Models 1 and 3 are replicated from Table 2, and are shown here for comparisons with Models 2 and 4.

Table 4.5 extends the findings shown in Tables 4.2, 4.3, and 4.4 by simultaneously examining the correlations between social relationships, genetic risk factors, and psychological distress. Model 1 replicates the results from Table 4.2, and is shown for comparison purposes only. In Model 2, the bivariate associations established in Model 1 are re-estimated with controls for genetic risk for psychological distress so that the percentage of the relationship that is explained by latent genetic confounders can be calculated. Looking at Model 2, we see that the coefficients are substantially reduced for all of the social relationship variables (examined one at a time) once genetic risk factors are held constant. For some predictors (e.g., extraversion, agreeableness, other family support, neighborhood quality, etc.), this reduction is not very large—16, 15, 16, and 16%, respectively. For others, however, such as socioeconomic status, the proportion of their relationship with psychological distress that is explained by genetic influences is quite large—e.g., 52 and 28% for education and income, respectively. In fact, more than half, and perhaps all (since the coefficient is no longer statistically significant), of the relationship between education and distress is explained by common genetic factors. That said, for all of the other social relationship variables, independent effects (net of genetic confounders) are present as well. As discussed previously, multivariate models are also shown, but should be interpreted with caution. Essentially, in the multivariate model, the effects of agreeableness, education, community ties, and neighborhood quality are completely explained when controls for the other social relationship variables, as well as genetic risk factors, are included in the models. The effects of positive relationships with others, other family support, and unemployment are reduced to borderline statistical

significance in this full model, while the remaining variables—i.e., extraversion, neuroticism, partner support, and income—all exert effects on distress that are net of all of the other social relationship variables and potential genetic confounders. These latter variables are obviously the most powerful predictors of psychological distress. Overall, then, these results provide some support for the genetic confounding claims of Proposition 4, but genetic factors by no means explain all of the correlation between social relationships and psychological distress.

Table 4.6: OLS Parameter Estimates from Bivariate Regressions of Genetic Risk for Psychological Distress (Independent Variable) on Psychological Distress (Dependent Variable) Net of Social Relationship Variables One at a Time

	Parameter Estimates[1]	% Explained by Social Relationship Variable[2]
Genetic Risk for Psychological Distress (Baseline Parameter Estimate = 0.0673)[1]		
Personality		
Extraversion	0.0627 ***	0.0642
Agreeableness	0.0665 ***	0.0075
Neuroticism	0.0515 ***	0.2313
Interpersonal Relations		
Positive Relations with Others	0.0596 ***	0.1104
Family Ties		
Married	0.0668 ***	0.0030
Partner Support	0.0620 ***	0.0746
Other Family Support	0.0642 ***	0.0418
Socioeconomic Status and Employment		
Education	0.0658 ***	0.0179
Income	0.0657 ***	0.0194
Unemployed	0.0658 ***	0.0179
Social Context		
Community Attachment	0.0619 ***	0.0761
Neighborhood Quality	0.0640 ***	0.0448

Notes: N=600 twin pairs (1,200 individuals); all models are adjusted for age and sex.

1. As shown in Table 3, the parameter estimate for genetic risk for psychological distress is 0.0673 when none of the social relationship variables are considered. This is the baseline for comparisons in this table.

2. In a multivariate model, where all of the social relationship variables are estimated net of the others, the coefficient on the genetic risk variable is reduced to 0.0418, which is a 37.61% reduction.

***p<0.001 **p<0.01 *p<0.05 +p<0.10 (two-tailed tests)

Table 4.6 provides results that further test Proposition 4, with specific attention devoted to the possibility that social relationships might mediate at least a portion of the

genetic effect on psychological distress. This proposition raises an interesting possibility that has not yet been examined in the literature—i.e., that genetic factors might influence distress indirectly via their effects on social relationships. The results in this table provide considerable support for this proposition. The coefficient from the bivariate OLS regression of genetic risk for psychological distress on distress, which was shown previously in Table 4.3, is 0.0673. This estimate is used as a baseline for comparison in Table 4.6, which provides parameter estimates in models that control for the social relationship variables one at a time. These findings show that a sizable percentage of the total genetic effect is indeed mediated by social relationships. Neuroticism explains the largest proportion of the genetic effect (23%), while extraversion (6%), partner support (7%), and community attachment (8%) also explain relatively large amounts. When all of the social relationship variables are included in the model simultaneously (not shown), the coefficient on the genetic risk measure is reduced to 0.0418, which amounts to a 37.61% reduction in the total genetic effect. This means that more than a third of the total genetic effect on observed psychological distress is mediated via social relationships.

DISCUSSION

This study began by suggesting that sociological research on psychological distress suffers from a potential shortcoming: the failure to adequately address latent genetic influences, which have also been linked with mental health. This argument was grounded in empirical research showing that genetic factors influence individual-level variation on (a) social relationships (the independent variables), (b) psychological distress (the

dependent variable), and (c) even the "correlations" between these two aspects of social life. Based on these findings, it was further argued that genetic influences could pose at least two potential implications for social science: (1) gene-environment correlation and possibly genetic confounding, and (2) potentially erroneous conclusions regarding the causal influence of social relationships on psychological distress. Despite the profound nature of these two issues, we do not currently know, however, whether, or to what extent, genetic factors play a role in correlation between social relationships and psychological distress.

Using a genetically-informed twin sibling design and data from a sample of U.S. adults, the present study addressed this shortcoming in the literature by showing that genetic factors do indeed influence (i.e., bias, partially explain, etc.) the phenotypic correlations between several different social relationship variables—i.e., social-oriented personality traits, interpersonal relations, family ties, socioeconomic status, and broader social contexts including community attachment and neighborhood quality—and psychological distress (one specific aspect of mental health). The extent of this effect depends on the specific type of social relationship under investigation, with genetic factors accounting for as little as 15% (for the correlation between agreeableness and distress), to as much as 52% (for the association between education and distress). In total, twelve different correlations were examined, and genetic factors were found to explain significant proportions of all of them. For one, the relationship between education and psychological distress, the main effect of education was reduced to statistical non-significance when genetic factors were taken into consideration. Genetic factors played a

sizable explanatory role in the correlations between distress and many other predictors as well, including neuroticism, positive relations with others, partner support, income, unemployment, and community ties. Given these findings, the question now becomes: What implications do genetic influences pose for traditional sociological research on psychological distress that does not take them into consideration?

As mentioned above, there are two different interpretations for these results. The first one—the mediation of genetic effects by social relationships—suggests that genetic factors directly influence personality and social resources, which, in turn, indirectly affect psychological distress. If this scenario is accurate, sociological theory will necessarily need to begin expanding the theoretical and empirical causes of both social relationships and psychological distress to include genetic, as well as environmental, influences. The social scientific argument that differences in psychosocial resources and support systems differentiate individuals on psychological distress would still be a feasible theory, but the reasons why some people have more of these resources than others, and thus have better mental health than others, would need to be revised to incorporate genetic influences.

The second one takes the form of genetic confounding, where the correlation between social relationships and psychological distress may be at least partially the product of one or more exogenous third variables—i.e., genetic factors—that lead to a correlation between social relationships and psychological distress that is not the result of the former influencing the latter. If true, this could pose profound implications for sociological research, including issues of explanatory power, biased empirical findings, and spurious associations. In the present study, genetic confounding appears to exist to at least some

degree in all of the correlations examined. That said, it certainly does not explain the entire link between social relationships and psychological distress, at least not for most of the predictors examined here.

Unfortunately, the present study is unable to determine exactly which of these two interpretations is the best explanation for the empirical findings—i.e., they are statistically inseparable. It is likely, however, that both are accurate to some extent. For example, genetic effects on psychological distress were diminished by almost 38% when all of the social relationship variables were held constant: a finding that suggests that the gene-environment mediation interpretation is at least partially correct. That said, in the empirical analyses conducted above, the size of the parameter estimates on all of the social relationship variables was substantially reduced when genetic risk for psychological distress was introduced into the models. This suggests that a common genetic predisposition influences both social relationships and psychological distress, which is consistent with the genetic confounding interpretation. Although distinguishing between these two interpretations is not possible here, one thing is obvious: genetic risk plays an important role in the connection between social relationships and psychological distress.

That said, the findings reported here also provide evidence that social relationships are important predictors of psychological distress, even net of potential genetic confounders. Despite the fact that genetic factors explain non-trivial proportions of the correlations between social relationships and psychological distress, in every case except for one (i.e., educational attainment, along with marriage, which was never significant), a statistically

significant main effect still exists even after potential genetic confounders are taken into consideration. Although sociologists (a) always assume that this is the case, and (b) oftentimes even forcefully argue in favor of social causation, the present study is actually one of the few empirical papers to actually demonstrate the presence of social influences net of genetic differences.

Regardless of the implications these findings pose for sociological research on psychological distress, it is important to note that they do not suggest that individuals with undesirable genetic predispositions are destined to experience poor mental health. In fact, they suggest exactly the opposite. Since genetic effects on psychological distress appear to be linked with social relationships, it is feasible that they could be altered (i.e., impeded) by policies or interventions aimed at enhancing the psychosocial resources and support systems available to individuals. This implies, as others have argued (Reiss et al. 2000; Shanahan and Hofer 2005), that genetic influences are not immutable, but are instead shaped by the social contexts in which they are situated. If one of the reasons why sociologists are averse to genetic explanations is that they view them as fatalistic or deterministic, the findings reported here should help to alleviate these false perceptions.

It is also important to note that the present study is only one of a handful of publications that have empirically addressed these complicated and controversial issues. Thus, it should not be taken as definitive proof for either (a) genetic confounding of sociological research, or (b) environmental effects net of genetic influences. Much more research will need to be conducted before we will fully understand the extent to which genetic factors affect the correlations between social relationships and psychological

distress. This will necessarily entail examining additional aspects of both social life and mental health. It will also include employing data on "measured" genes that have been linked with mental health, such as the serotonin transporter gene.

Moreover, it is extremely important not to under or overstate the empirical findings reported here. At the present time, our ability to measure genetic risk factors is still quite limited. Great progress has been made on this front over the last decade, and the near future promises a rapid advance in our knowledge in this area, but we are not there yet. Basically, the findings reported here could actually underestimate the true magnitude of genetic effects for two reasons. First, the measure of genetic risk employed here is somewhat crude, and it provides a very conservative estimate of genetic effects (i.e., it explains 15% of the variation on psychological distress in this data). Previous findings from twin and adoption studies suggest that this number should be closer to 30% or more (Blehar et al. 1988; Eley 1997; Kendler and Prescott 2006; Plomin 1990; Plomin and Rende 1991). Given that this measure actually explains 15-52% of the phenotypic correlations between social relationships and psychological distress, it is reasonable to think that if genetic effects are actually larger than indicated here, that their effects on the correlations examined here might be more pronounced as well. Second, in addition to the possibility that it underestimates the true magnitude of genetic effects on psychological distress, the genetic risk measure employed here was constructed solely from the psychological distress variable, and thus it taps only predispositions toward high levels of distress. There are almost certainly genetic effects on social relationships that are not correlated with those for distress, and these potential influences have not been taken into

consideration here. That said, since confounding requires that genetic factors influence both the independent and dependent variables simultaneously, these additional genetic effects may not matter that much, however, if they are not correlated with distress.

Further, the cross-sectional nature of the present study does not allow the causal order to be precisely determined. Although the theory and analyses were aimed at examining how social relationships influence psychological distress, it is possible that psychological distress also affects social relationships (Forthofer et al. 1996; Johnson 1991; Miech et al. 1999; Wade and Pevalin 2004). The findings reported here would be consistent with that possibility since genetic predispositions toward psychological distress would almost certainly lead individuals to experience poor social relationships. In fact, this causal order is quite reasonable given the current results. Future research with genetically-informed longitudinal designs will, however, be needed to address this issue further.

Scholars interested in examining the linkages between genetic and environmental influences on psychological distress in more detail also need to consider the potential ability of social relationships to moderate (i.e., facilitate and / or impede) genetic effects. In other words, in addition to gene-environment "correlation" or "mediation," as examined here, gene-environment "interaction" also needs to be analyzed. This is especially important given that several publications have already shown that genetic and environmental factors work together in an interactive manner to influence mental health (Boomsma et al. 1999; Hamer and Copeland 1998; Omenn 2000; Plomin 1990; Rutter, Moffitt, and Caspi 2006; Shanahan and Hofer 2005; Shostak 2003).

To conclude, the literature reviewed above, the conceptual model and propositions formulated from it, and the empirical analyses conducted, all point to a crucial need for social scientists to incorporate genetic influences into their research. Genetic risk factors not only exert sizable effects on psychological distress, they also appear to influence virtually all types of social relationships as well. This means that they might function in a confounding or explanatory manner with respect to the phenotypic correlation between these two outcomes as well. The findings reported here suggest that this is indeed an issue, but that social relationships (for the most part) also exert independent effects on distress net of potential genetic confounders. The main exception is the result for education—i.e., this aspect of social life appears to be linked with psychological distress almost completely because of common genetic influences. Future research should certainly explore these issues further, but for an outcome like psychological distress, where there can be little doubt that genetic influences play some sort of role, social scientists need to begin giving them the attention they deserve.

CHAPTER 5

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

SUMMARY

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 combine to influence psychological distress. The models draw on twin sibling data from the National Survey of Midlife Development in the United States (MIDUS), and address Research Questions 1-4 of this dissertation (particularly #4). Results show (a) that both SES (inversely) and genetic risk (positively) are significantly associated with 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 relationship 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.

INTRODUCTION

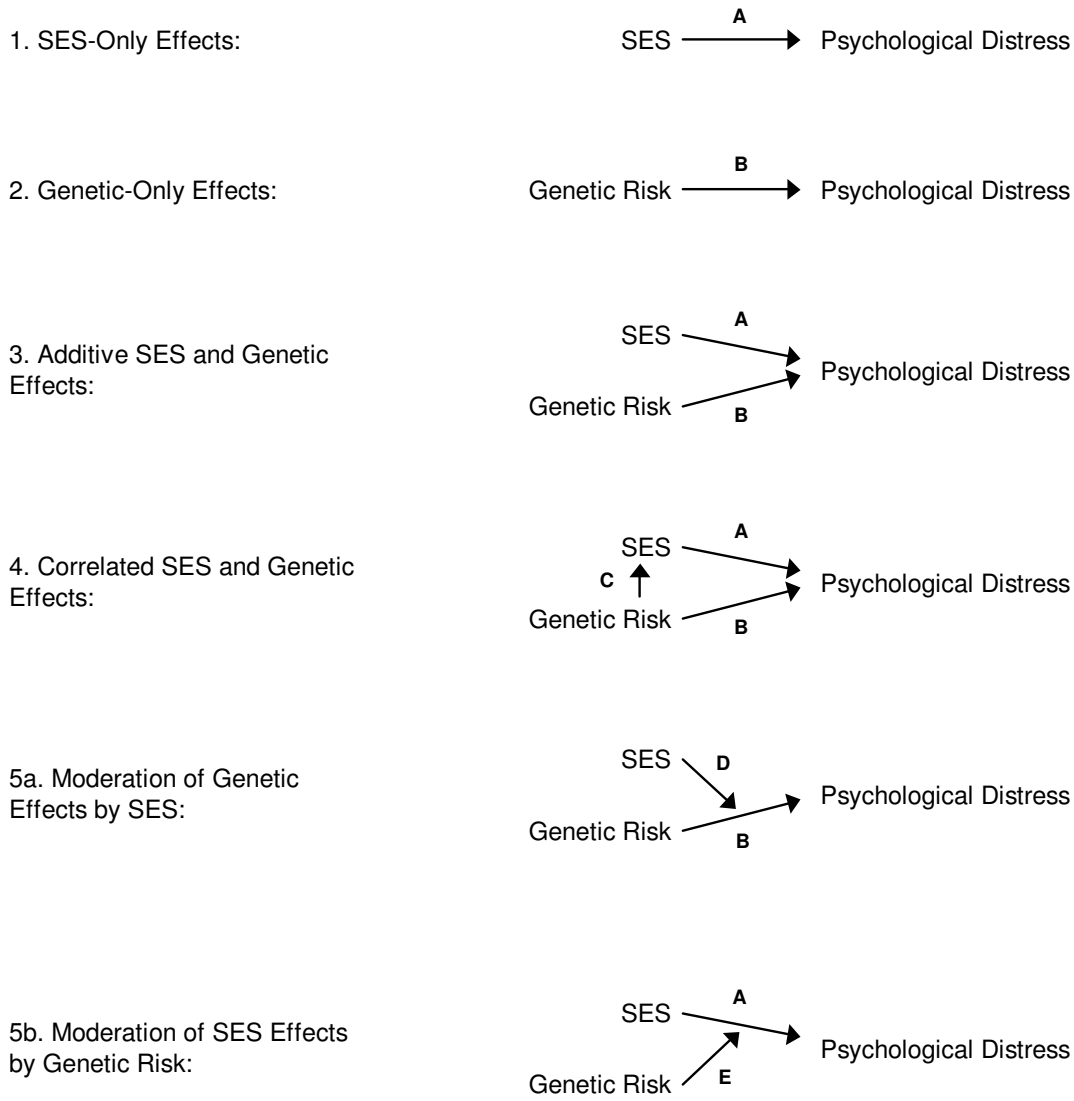
An enormous body of sociological research documents that socioeconomic status (SES)—e.g., educational attainment, monetary income, etc.—is inversely associated with psychological distress and other forms of poor mental health (House 2002; Kessler 1982, 1979; Miech and Shanahan 2000; Mirowsky and Ross 2003). At the same time, psychologists, psychiatrists, biologists, and medical doctors are amassing evidence showing 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 together 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? Does SES mediate 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 evaluating five possible conceptual models of the relationships between SES, genetic risk factors, and psychological distress: (1) SES-only effects; (2) genetic-only effects; (3) additive 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 will be 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 interactively influence psychological distress in the following ways: (a) the association between genetic risk factors and psychological distress is greater among persons with low levels of education and income, and (b) the association between education and income, and distress, is lowest among persons who have very low genetic risk. From a sociological perspective, the results suggest that the SES association with psychological distress is greatest among those persons who are genetically at risk. The association between SES and distress at the population level may reflect the combination of varying gradients in the association depending on the level of genetic risk. Overall, integrating genetic factors into sociological models of distress appears to lead to a deeper understanding than is typically possible because of data that is not genetically informed, and the present study serves as a preliminary roadmap for how to achieve this goal.

Figure 5.1: Five Conceptual Models of the Relationships Between SES, Genetic Risk Factors, and Psychological Distress



THEORETICAL AND EMPIRICAL BACKGROUND

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 stressors (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 in Figure 5.1.

Although sociological research rarely mentions genetic risk factors in the context of examining the association between SES and psychological distress, 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 family-based studies such as these, which provide heritability estimates, molecular genetic research on mental health provides evidence that that carriers of the short allele of the functional 5' promoter polymorphism of the serotonin transporter gene have 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 5.1).

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) additive (i.e., independent) or (b) interconnected (i.e., correlated or interactive).

With respect to the first possibility, Model 3 of Figure 5.1 shows an additive 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 that SES and genetic risk factors 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 (Model 3, Pathways A and B).

With respect to the second possibility, there is good reason to believe that the effects of SES and genetic risk factors on mental health are not independent of one another, at least not completely (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, especially if we are to obtain a deeper understanding of the pathways by which each operates.

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-additive, interconnected effects. Such a possibility implies the scenario shown in Model 4 of Figure 5.1, 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 additive, but will instead be correlated (Model 4, Pathways A, B, and C).

One of the primary reasons to expect non-additive 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 are 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—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 5.1, 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.

In addition to additive 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 5.1), and (b) that environmental factors may have different effects based on the genetic makeup of individuals (Model 5b of Figure 5.1) (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 5.1, 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 5.1). Of course, this interpretation is empirically indistinguishable from the associations shown in 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. Recent molecular genetic work has similarly 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 reported in MIDUS as 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 outcomes 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 additive, 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 5.1: 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.

RESULTS

Table 5.1 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 our 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 5.2: 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 5.2 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 5.3: 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)

To further examine these relationships, multivariate regression analyses are required. As shown in Model 1 of Table 5.3, 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, 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; a one unit increase in genetic risk is 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.

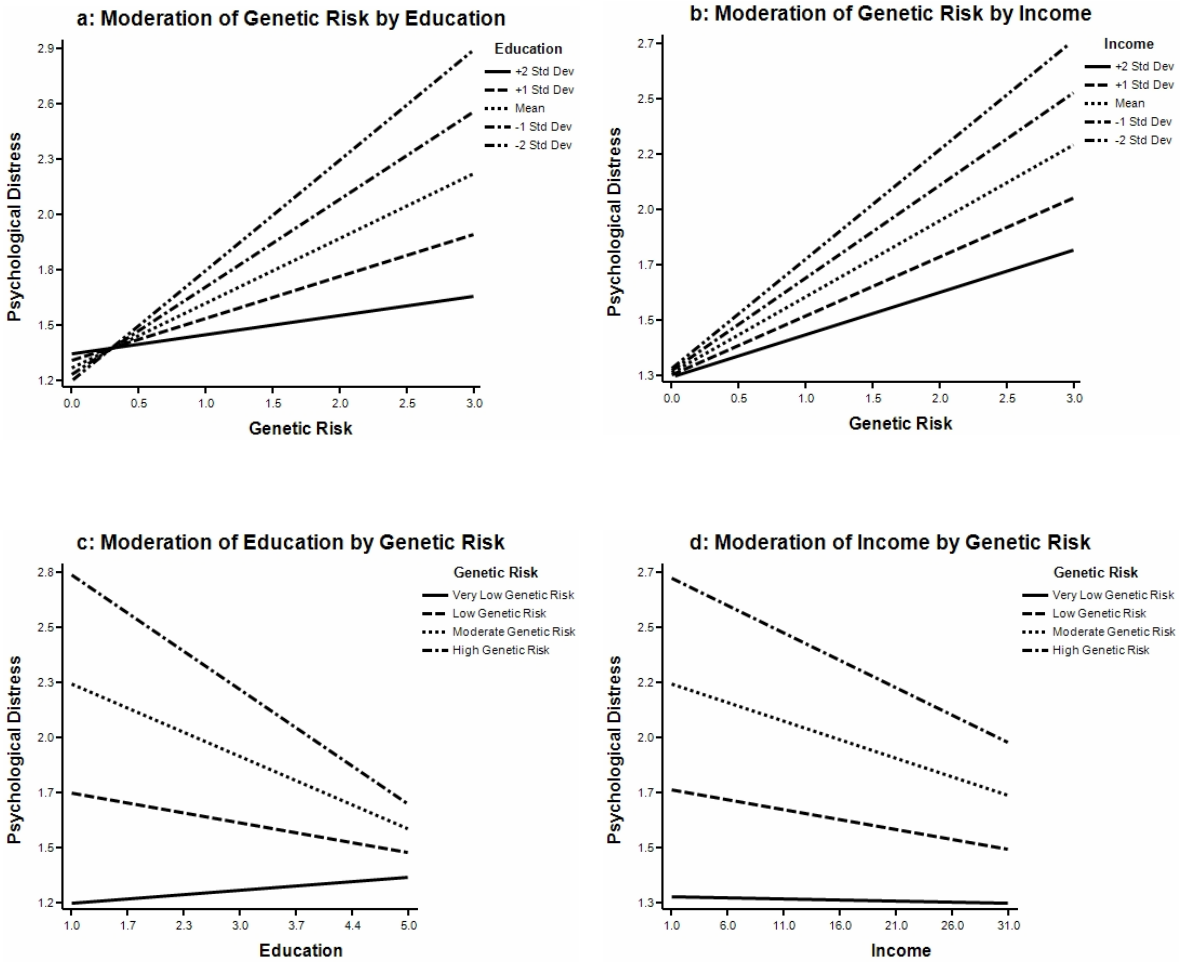
Model 3 tests the idea that the influence of SES and genetic risk factors are largely independent of one another, even when simultaneously examined. Another way to think about these associations is that the genetic risk for psychological distress is randomly distributed across levels of socioeconomic status, such that both SES and genetic risk are associated with psychological distress. The findings shown in Model 3 of Table 5.3, 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 5.2, 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 5.2:

The Interactive Influence of SES and Genetic Risk on Psychological Distress



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 5.2a and 5.2b, 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 5.2a). For income, we see a similar pattern (Model 5 and Figure 5.2b): 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 5.3 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 5.2c and 5.2d, 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 5.2, 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, additive 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 additive, 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 5.2 (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 5.1 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.

CONCLUSION

DISSERTATION SUMMARY

This dissertation began by asking a simple question (Research Question 1): Do both genetic and environmental factors influence social life, and if so, what is the relative contribution of each? One would think that this question would have been asked many times by biologists, geneticists, psychiatrists, and psychologists interested in understanding the causes and consequences of human social interaction. A detailed examination of the literature, however, reveals that this is the case only for social-oriented personality characteristics and health and well-being, and that, unfortunately, this question has motivated surprisingly few studies of other aspects of social life.

To address this shortcoming in the literature, this research examined—from a genetically-informed perspective—a variety of distinct social outcomes. Specifically, analyses of data from the National Survey of Midlife Development in the Untied States (MIDUS), a large sample of working-age (25-74) twin siblings, showed that genetic factors do indeed explain sizable and statistically significant proportions (15-65%) of the individual-level variation on: (a) four different aspects of religious participation (i.e., organizational involvement, personal religiosity, beliefs, and transformations and commitments) (Chapter 1); (b) two different facets of civic engagement (i.e., feelings of obligation to participate, and actual volunteering) (Chapter 2); (c) various personality and interpersonal characteristics (e.g., extraversion, positive relations with others, social attachments, etc.) (Chapters 2 and 4); and (d) levels of psychological distress (Chapters 4

and 5). In addition, genetic risk factors for high levels of distress were also found to predict other aspects of social life including neuroticism, family relations and support, SES, community ties, and neighborhood quality (Chapters 4 and 5). Based on these findings, the answer to Research Question 1 appears to be: (a) yes, genetic influences do indeed matter; and (b) that both genetic and environmental factors explain considerable proportions of the variation on virtually all social outcomes, with the relative effect of each varying depending upon the specific aspect of social life under investigation. These findings are a significant contribution to the literature, especially for outcomes such as religious involvement, civic engagement, family relations, and community attachments, where it is assumed that they are primarily, if not exclusively, the products of social-environmental influences.

That said, the presence of genetic effects on these outcomes also raises several additional questions, including (Research Question 2): Why and how do genetic factors influence social life, and what are the pathways by which they operate? Even though the literature provides very little insight into this question, it has been proposed that genetic influences on social outcomes might be mediated by personality and interpersonal characteristics (Alford, Funk, and Hibbing 2005; Kendler and Prescott 2006). In other words, we know (a) that genetic factors are strong predictors of virtually all personality traits, and (b) that personality differences are important explanatory factors for many different aspects of social life, which implies (c) that genetic effects on personality might indirectly affect social outcomes. In this dissertation, this theory was formulated (Chapters 2, 3, 4, and 5), and then empirically tested for two particular aspects social life:

civic engagement (Chapter 2) and psychological distress (Chapters 4 and 5). Results provided considerable support for it, and future research should therefore consider this possibility for other social outcomes as well.

In addition to examining whether genetic factors influence social life, as well as the potential pathways by which they operate, this dissertation also addressed two other issues: gene-environment correlation and gene-environment interaction (specifically as they pertain to health and well-being). In essence, large bodies of research have shown that virtually all health outcomes are influenced by both genetic and environmental factors, which means that there is not really a need to ask whether genetic influences are important. There is, however, a relative dearth of research on the possible ways in which genetic and environmental influences might be interconnected (see Chapter 3), and addressing this issue raised two additional questions.

The first one was (Research Question 3): Are genetic and environmental influences correlated (i.e., non-additive), and do genetic factors bias social scientific studies that do not take them into consideration? Here, this issue was examined theoretically for health and well-being in general (Chapter 3), as well as empirically for the specific correlations between social relationships (many different types) and psychological distress (Chapters 4 and 5). Results showed: (a) that a whole host of different social relationship variables (i.e., extraversion, neuroticism, positive relations with others, family relations, socioeconomic status, community attachments, and neighborhood quality) are phenotypically correlated with psychological distress; (b) that genetic factors do indeed account for non-trivial proportions (15-52%) of these associations; (c) that social

relationships, for the most part, are correlated with distress net of genetic influences; and (d) that at least part of the genetic effect on psychological distress appears to be mediated by social relationships. These findings pose at least two major implications for research on social relationships and psychological distress that does not consider genetic factors: (a) partially biased or confounded empirical findings, and (b) potentially erroneous conclusions regarding cause and effect.

In addition to gene-environment correlation, it is also possible that genetic and environmental influences on health and well-being are interconnected in a different way: gene-environment interaction. In this area, the final research question was (Research Question 4): Do genetic and environmental factors interact to produce social outcomes, particularly health and well-being? To address this issue empirically, psychological distress was once again employed as the outcome of interest (Chapter 5). The two predictors in this analysis were genetic risk toward high levels of distress and socioeconomic status. Basically, these two variables have been linked with psychological distress in literally hundreds of previous studies, but to date, they have not been simultaneously examined. Thus, we do not currently know whether, or how, they might work together to influence levels of distress. This dissertation addressed this shortcoming by formulating and then empirically testing a series of models, and results showed: (a) that both SES (inversely) and genetic risk (positively) are significantly associated with psychological distress; (b) that the association between SES and distress is explained, to a considerable degree, by genetic risk factors; (c) that the relationship between genetic risk factors and distress is stronger at low levels of SES; and (d) that the inverse correlation

between SES and distress exists primarily among individuals who are at high genetic risk. These findings are important for biomedical researchers and social scientists alike because they indicate both of these factors are needed to fully understand the causes of psychological distress, and that the effects of each is contingent upon the other.

Overall, this dissertation has provided valuable information on the genetic and environmental causes of many different aspects of social life. Much remains to be done, however, and the next section outlines some issues that need to be addressed, as well as some promising directions for future research.

WHERE DO WE GO FROM HERE?

Measuring Genetic Influences

As mentioned throughout this dissertation, most (and perhaps all) social outcomes are complex phenotypes that are likely influenced by many different genes (plus countless environmental influences), each of which exerts a relatively small effect. In other words, most attitudinal and behavioral outcomes are polygenic traits, and given that we currently understand the functions of only a small percentage of the literally tens of thousands of human genes, actually measuring genetic differences and then correlating them with social outcomes is somewhat difficult at the present time. This is why twin and other family-based designs are currently so important, and will continue to be so into the foreseeable future (Guo 2005; Kendler and Prescott 2006; Plomin and Caspi 1998; Plomin and Rende 1991). That said, they are not perfect, and other methods are needed to supplement the findings based on these approaches.

Given that the entire human genome has now been mapped, progress in the area of measured genetic differences at the molecular (i.e., DNA) level will be rapid in the coming years. To date, most of the polymorphic human genes that have been discovered and studied are associated in some form or another with the serotonin or dopamine systems. Since these biochemicals affect brain structure and functioning, differences in their levels (which are at least partially under genetic control) pose severe consequences for virtually all aspects of human existence, including social life. In the current literature, specific genes such as the serotonin transporter gene and the various dopamine receptor genes have been linked with personality, health and well-being, and deviant behavior (Guo, Roettger, and Shih 2007; Hamann 2005; Lesch et al, 1996; Pezawas et al. 2005; Plomin and Caspi 1998). To date, however, virtually no studies have utilized molecular genetic data in an attempt to understand individual differences on other aspects of social life such as religious involvement, civic engagement, family relations, socioeconomic status, or social attachments, among many others. Given that sizable genetic effects have been demonstrated by family-based studies for these, there is justifiable reason to search for linkages between DNA differences and individual variation in these areas.

Overall, then, twin and other family-based studies—because of their ability to tap latent genetic influences that are difficult to measure—will remain important tools for studying population processes, but they will increasingly be supplemented with findings from molecular genetic studies of social outcomes as well. Future research should therefore pursue research on social life using all available genetically-informed techniques, including twin studies, adoption designs, chromosomal linkage techniques,

genome-wide scans, and information on known polymorphic genes. Each approach has their own strengths and weaknesses, and together they are capable of providing detailed information on genetic effects even at this early stage of research in this area.

Measuring Environmental Influences

To date, many of the genetically-informed studies in the literature have measured "environmental influences" simply as two latent factors: shared and nonshared environmental influences (see Chapters 1 and 2). As this dissertation has shown throughout, however, the environments in which individuals are embedded are extremely diverse and complex, and they include family life, friends, school, region of residence, urban versus rural areas, marital status and quality, the presence of children in the home, stressful life events such as unemployment or the loss of a loved one, socioeconomic status, religious participation, social support, and neighborhood quality, among many others. This dissertation has examined several of these, but future research on the intersection between genetic predispositions and environmental influences should begin detailed analyses of all of these facets of human social existence. Importantly, these factors can be easily integrated into genetically-informed approaches such as twin and adoption studies, and even molecular genetic designs.

Examining Additional Outcomes

This dissertation began by analyzing the genetic and environmental causes of two aspects of social life that have received very little attention in the genetically-informed literature:

religious involvement and civic engagement. Despite the knowledge gained here, we still know very little about these outcomes, and future research should delve deeper into these social variables. Other aspects of social life that deserve either initial, or further, attention as well include marriage and romantic life, parenting attitudes and behaviors, attachment relations, political ideologies, school success and trajectories, and socioeconomic attainment, among others. Very little, and in some cases no, genetically-informed scholarship has been conducted on these topics, and virtually no research in these areas to date has looked into gene-environment correlation or interaction.

For example, do certain environmental factors facilitate or impede genetic predispositions toward religious involvement, civic engagement, and other social outcomes? Do social influences in these areas vary depending upon genetic differences (i.e., are environmental factors more important for certain individuals who are already genetically predisposed toward social life, or are they equally effective for all people)? To date, questions such as these have only been addressed for health and well-being and educational attainment, and virtually all other aspects of social life have been completely ignored.

With respect to health and well-being more specifically, genetic factors have been found to predict individual-level variation on many different outcomes including positive and negative affect, depression, anxiety, alcohol consumption, tobacco use, antisocial behavior and conduct problems, hypertension, intellectual development, cancer, BMI, and heart-related outcomes, to name just a few (Bouchard and Loehlin 2001; Guo and Stearns 2002; Jaffe et al. 2005; Jang et al. 2001; Johnson and Krueger 2005a; Kendler

2001; Li et al. 2003; Tuvblad et al. 2006). This dissertation has focused exclusively on psychological distress, but future research should explore the possibility that genetic and environmental influences function in correlated and interactive ways to predict these and other outcomes as well. Future research should also examine whether environmental influences other than SES, such as religious involvement, poor family relations, a lack of community ties, etc., also moderate (and are moderated by) genetic effects on health and well-being. It is likely that desirable environmental conditions such as integration into religious networks, having supportive parents or spouses, and being connected to friends and neighbors, may help to offset genetic risks toward poor health, while undesirable ones such as family conflict and social isolation, among others, may function to exacerbate genetic risk factors.

In addition, it is an unfortunate fact that most "positive" or desirable aspects of social life, particularly health outcomes (e.g., happiness, positive affect, a sense of control, etc.), have been largely neglected in the genetically-informed literature, which has focused instead on undesirable ones such psychological distress, depression, and substance abuse. Given that positive outcomes are not simply the lack of negative ones (i.e., the absence of undesirable outcomes), research needs to begin addressing this weakness in the literature.

Aging and the Life Course

There is some, although not a lot, of evidence suggesting that genetic effects on social outcomes, particularly health and well-being, may vary across the life course (Charles and Almeida 2007; Neiss and Almeida 2004). According to this research, genetic effects

appear to become more pronounced (relative to environmental ones) as individuals progress through the life course, especially following the transition from adolescence into adulthood, and there is even evidence (albeit in the form of only a few studies) that some genetic effects may not manifest themselves until midlife or later (Bortz 2005; Gillespie et al. 2004; Harris et al. 1992). The reasons for this phenomenon are, unfortunately, unknown at the present time. It may be that certain biological processes turn genes on an off at different ages, but environmental factors may also be playing a role.

For example, deleterious environmental influences such as a conflict-laden home environment or monetary difficulties may facilitate genetic risk factors early in the life course, while others such as a loving spouse or a high level of income may impede these same risks later in the life course. Given that many different environmental predictors of social outcomes vary with age—e.g., religious involvement, personal income, marital stability and quality, and social integration increase following adolescence and early adulthood and into midlife—it is therefore possible that they also affect genetic risk factors in different ways during different life stages as well. To date, however, this possibility has received very little attention in the literature, but it should be a focus of future research (see Shanahan and Hofer 2005).

Race and Gender Differences

Despite ample theoretical justification, we do not know yet whether there are gender or racial / ethnic differences in genetic and environmental influences on the aspects of social life examined here. We do know that gender and racial differences exist in many different

social outcomes including religious involvement, personal income, the quality and quantity of social networks and support systems, and health and well-being, among many others (Kendler and Prescott 2006; Miller and Stark 2002; Mirowsky and Ross 2003), but to date, very little research has explored variations in genetic effects by gender or race for these variables. This issue should certainly be the topics of future studies.

For example, research should begin addressing questions such as: Can genetic factors account for the empirical fact that both women and African Americans are more religious than men and whites in the U.S.? Such a possibility has already been implied in the literature (Miller and Stark 2002). Do genetic factors play a role in the differential voting and volunteering habits of younger versus older individuals? Are women more genetically predisposed toward poor mental health than men, and if so, do personality differences account for this phenomenon? How might genetic and environmental factors function together to produce higher levels of hypertension among African Americans compared with whites? Questions such as these have yet to be answered in the literature, and genetically-informed designs may offer unique insight.

IN CLOSING

This dissertation examined the potential role of genetic factors in social life: a neglected area of scientific inquiry. It is important to note that this research was (a) written by a social scientist, (b) supervised by social scientists, and (c) intended to be read by, and to influence, social scientists. For these reasons, it has tried to demonstrate how genetically-informed research can be informative in social scientific research, as well as how

information on genetic differences can be integrated into existing sociological frameworks.

In pursuit of this goal, it was shown that at least 23 different aspects of social life are influenced by genetic factors—some to a considerable degree. It was further shown that in most cases, environmental factors are important predictors of social outcomes above and beyond genetic effects, and that in some instances, genetic and environmental influences function in an interconnected (i.e., correlated and / or interactive) manner. These findings are in sharp contrast to the independent or additive effects models that biomedical and social scientific researchers alike are currently working with. This suggests that there is a need to begin integrating genetic and environmental factors into a single explanatory framework. Doing so will not only provide insight into social life that would otherwise remain undiscovered, it will also address several issues with the current literature including explanatory power deficiencies, potentially biased empirical findings, and possibly erroneous conclusions regarding cause and effect, among others.

Despite aversion to genetic explanations on the part of some social scientists, if the findings presented here are any indication, they have little to fear. In fact, it is likely that addressing genetic influences will only function to strengthen sociological arguments concerning the importance of environmental factors. It is the author's hope that this research will stimulate debate and motivate additional research of this type, and if this occurs, this dissertation will have been a success.

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