#### CHAPTER FIVE

# Health, Well-Being, and Social Responsibility in the MIDUS Twin and Sibling Subsamples

Ronald C. Kessler, Stephen E. Gilman, Laura M. Thornton, and Kenneth S. Kendler

How healthy are we? A national study of well-being at midlife. (2004) Brim, OG, Ryff, CD, & Kessler, RC (eds). Chicago, IL The University of Chicago Press 124-152

One of the special design features of MIDUS was the augmentation of the general population sample to include nationally representative subsamples of twins and nontwin sib-pairs (Kendler et al. 2000). The respondents in these subsamples were administered the same interview and questionnaires as other MIDUS respondents and also received a separate short interview that asked them about the extent to which they were treated similarly or differently from their sibs during their childhood years. Twins were also asked a series of questions about biological similarity that allowed us to classify them as either identical monozygotic (MZ) or nonidentical dizygotic (DZ). Finally, cheek scrape samples were collected from twins and sibs to extract DNA for future molecular genetic analyses. Although no results are yet available from the molecular genetic analyses, behavior genetic analyses have been completed for all three of the central MIDUS outcome domains of self-reported health, psychological well-being, and social responsibility. These behavior genetic analyses are the focus of this chapter.

The analyses reported here are limited to univariate investigations of heritability. Heritability is the percentage of variance in observed variables as a result of between-respondent variation in genes. Documentation of significant heritability is a necessary first step before performing more complex multivariate behavior genetic analyses and molecular genetic analyses. As described in more detail later in the chapter, the estimation of heritability in behavior genetic analyses is usually based on comparisons of the similarities of scores on observed variables in pairs of MZ twins versus DZ twins (Loehlin 1989). Greater similarities in the former than the latter are taken as evidence of genetic influences, because MZ twins share 100 percent of their genes identically by descent, whereas DZ twins share, on average, 50 percent of their genes. A variety of tests described later in this chapter are typically made to rule out competing explanations before concluding that genes are at work. These tests deal with the possibility that MZ twins might be treated more similarly than DZ twins or that

MZ twins might want to differentiate themselves more than DZ twins. When information from nontwin sib-pairs is included in these analyses, it becomes possible to evaluate the effects of being a twin, because nontwin sibs are the same as DZ twins in sharing an average of 50 percent of their genes. Finally, special types of structural equation models can be used to estimate the relative influences of genes and environment in explaining variance in specific outcomes and to perform multivariate analyses (Neale and Cardone 1992).

### The Heritability of Self-Reported Health

Previous studies comparing MZ and DZ twins have consistently shown that a variety of physical illnesses are heritable. For example, in the NAS-NRC twin study (Jablon et al. 1967), Kendler and Robinette (1983) estimated that the heritability of ischemic heart disease is .37 and that the heritabilities of diabetes and hypertension are both .58. Similar findings have been reported by a number of other investigators on the basis of analyses of other twin samples and documenting significant heritabilities of ischemic heart disease (Carroll et al. 1985; Nora et al. 1980), hypertension (Borhani et al. 1976; Carroll et al. 1985; Lichtenstein et al. 2000; Selby et al. 1991), a variety of cancers (Hemminki, Dong, and Vaittinen 1999; Holm, Hauge, and Harvald 1980; Page et al. 1997; Partin et al. 1994; Schneider, Williams, and Chaganti 1986), and obesity (Allison et al. 1994, 1996; Fabsitz, Sholinsky, and Carmelli 1994; Selby et al. 1990; Stunkard 1991; Stunkard, Foch, and Hrubec 1986; Turula et al. 1990).

The heritability of self-reported health has been a subject of much less research. Measures of self-reported health, which are the health measures included in MIDUS, are important because they significantly predict survival even after adjusting for the effects of diagnosed health problems (Mossey and Shapiro 1982). In addition, summary measures of self-reported health are useful proxies for overall objective health, because self-reported health is strongly predicted by a combination of medical morbidities, physical and psychological symptoms, and measures of functional status (Barsky, Cleary, and Klerman 1992; Fylkesnes and Forde 1992; Jylha et al. 1986; Moum 1992).

Two aspects of self-reported health are considered in this chapter: self-rated health perceptions and functional status. Self-rated health perceptions were assessed with global questions that asked about overall health and then separately about physical health and mental health. Global questions like these are widely used in health surveys, although it is rare to ask separate questions for physical and mental health. We are aware of only

one study that estimated the heritability of global self-rated health. This study was performed in four age cohorts of the Swedish Adoption/Twin Study of Aging (Harris et al. 1992). Evidence for statistically significant heritability was found in the older cohorts (.25) but not in the younger cohorts. This specification is interesting in light of previous evidence that there is a statistically significant inverse relationship between self-rated health and age, controlling for objective measures of physical health and psychological distress (Levkoff, Cleary, and Wetle 1987).

Functional status, the second aspect of self-reported health considered here, is the extent to which health limits routine daily activities. Two groups of investigators have estimated the heritability of functional status. Yashin et al. (1998) assessed multiple domains of functional status in a sample of Danish twins and showed that the correlations of these measures among twins were significantly stronger for MZ than DZ twins. The estimated heritability of functional status was approximately .30 for both males and females. Miles (1997), in a separate small study of MZ and DZ twins, documented the existence of significant familial aggregation of self-reported functional status based on substantial correlations between the scores of respondents with their cotwins. However, the sample was so small that no data were reported on differences in the correlations of MZ and DZ twin-pairs or on the estimated heritability of self-reported functional status.

In comparison with the scant amount of literature on the heritability of self-reported physical health, a large number of studies have investigated the familial aggregation of self-reported psychiatric disorders. These studies consistently find that most commonly occurring psychiatric disorders are moderately heritable (Kendler, Walters, et al. 1995). For depression, the mental disorder that has been the subject of the most extensive behavior genetic studies, the majority of familial aggregation of the disorder has consistently been shown to be the result of genes (e.g., Kendler et al. 1993; Kendler, Pedersen, et al. 1995; McGuffin et al. 1996). Genes also appear to play an important role in the familial aggregation of generalized anxiety disorder and phobias (Kendler et al. 1992a, 1992b). A number of studies have also estimated the heritability of dimensional scales of nonspecific psychological distress and have found consistent evidence of significant heritability (e.g., Kessler et al. 1992; Silberg et al. 1990).

# The Heritability of Psychological Well-Being

Ryff(1989) proposed a multidimensional model of psychological wellbeing that included six dimensions: self-acceptance, personal growth, purpose in life, positive relations with others, environmental mastery, and autonomy. Although we are unaware of any studies that have examined the familial aggregation of these six dimensions as a set, there has been some research on the heritability of constructs related to two of these dimensions: self-esteem and social support.

Self-esteem is a personality characteristic that is strongly related to the Ryff dimension of self-acceptance. Two studies have examined the heritability of self-esteem using twin data (Kendler, Gardner, and Prescott 1998; Roy, Neale, and Kendler 1995). Both found that twin concordance for self-esteem was explained by a combination of genetic and individual-specific environmental factors. In the earlier of these studies, based on a sample of same-sex female twins, additive genetic factors accounted for 53 percent of the variance; in the latter study, based on same-sex and opposite-sex pairs, the estimated heritability was approximately .30 for both males and females. There was no evidence in either study that shared environmental influences were related to twin concordance for self-esteem.

Several studies have examined the heritability of various aspects of social support, including measures of support that are conceptually identical to the Ryff and Keyes dimension of positive relations with others (Bergeman et al. 1990; Kendler 1997; Kessler et al. 1992). Heritability estimates for measures of positive social relations have consistently been significant in these studies and in the range .25–.50.

Although they are less closely mapped onto individual dimensions in the Ryff model of psychological well-being, commonly studied dimensions of personality, such as neuroticism, extraversion, conscientiousness, openness to experience, and agreeableness, have all been shown to have heritabilities in the range .20–.40 (Heath et al. 1992; Loehlin 1992; Viken et al. 1994). This is indirectly relevant to the investigation of well-being because Schmutte and Ryff (1997) showed that these dimensions of personality are strong correlates of the well-being dimensions considered here.

# The Heritability of Social Responsibility

Four domains of social responsibility proposed by Rossi (2001) are considered in this chapter: normative altruistic obligation, civic-job obligation, primary obligation, and contribution to others. The only previous studies that examined the heritability of any of these dimensions focused on the personality trait of altruism (Loehlin and Nichols 1976; Matthews et al. 1981; Rushton et al. 1984) and estimated the heritability of this construct to be between .40 and .72. As noted in the previous

subsection, personality characteristics that one might expect to be related to social responsibility, such as conscientiousness and extraversion, have also been shown to be heritable. One might expect, therefore, that there is at least some evidence for significant heritability of social responsibility.

# Sample and Methods Sampling Procedures for the Twin and Sib Samples

Twin-pairs were recruited using a separate two-part sampling design from the main MIDUS sample. The first part of the design involved screening a representative national sample of approximately 50,000 households for the presence of a twin. This was done as part of ongoing national omnibus surveys conducted by ICR/AUS Consultants and Bruskin Associates. Respondents who indicated that twins resided in the household or that they themselves were part of a twin-pair were asked permission to be contacted by our research team for inclusion in MIDUS. One-seventh (14.8 percent) of respondents reported the presence of a twin in the family, of which 60.0 percent gave permission to be contacted for the twin study.

The second part of the twin sample design involved student recruiters from the University of Michigan contacting the twins to participate in MIDUS. Cooperating twins were asked to provide contact information for their cotwins, who were also recruited by the students. The final response rate for twin pairs (i.e., the probability of obtaining complete interviews from both members of the pairs detected in the 50,000 screening interviews) varied dramatically, depending on whether the initial person contacted in the screening interview was a member of a twin pair (60.4 percent response rate) or the relative of twins (20.6 percent response rate). This dramatic variation is due to the fact that the relatives of twins were often reluctant to provide contact information, whereas the twins themselves were much more willing to participate in the survey and to contact their cotwins to encourage them to participate. The final MIDUS twin sample included 794 twin-pairs from 763 distinct families, with 29 families contributing two twin pairs and one family contributing three pairs.

Nontwin sib-pairs were recruited in a different manner. We began by sending a postcard to all MIDUS respondents, telling them of our interest in including siblings in the survey. The card asked respondents to provide contact information for their siblings and to communicate with their siblings about participation before a recruiter made a contact attempt. Because the family study was a secondary aim of the project, no follow-up procedures were employed to increase the low proportion of eligible

MIDUS respondents who provided names and addresses of their siblings (19.7 percent). As with the twin sample, though, the cooperation rate of siblings was much higher once they were contacted (69.3 percent). In total, 529 original MIDUS respondents returned the postcards, providing us with contact information for their siblings. There were 1372 siblings listed on these return postcards, 951 of whom completed the MIDUS survey. The number of additional siblings recruited from any single family ranged from one to six. These included one additional sibling from 272 families, two from 146 families, three from 75 families, four from 22 families, five from 10 families, and six from 4 families.

Of the 951 sibs who completed the MIDUS telephone interview, all but 81 also completed the mail questionnaires. As described later in the chapter, in the discussion of the analysis method, the data were analyzed for pairs of respondents. As a result, sibships in which complete data were collected on four or six sibs were divided up into either two or three randomly constituted pairs. We deleted the data from one random sib in sibships with complete data on three, five, or seven sibs. The remaining data in the sibships that originally had five or seven sibs were then organized into two or three random sib-pairs. The resulting dataset contained 1220 sibs that were divided into 610 sibling pairs. Although the majority of sibling pairs came from distinct families, there were 82 families (16.1 percent) that contributed two pairs of siblings, and 9 families that contributed three pairs of siblings.

# Zygosity Determination

At the time of initial recruitment into the survey, same-sex twins were administered a number of standard questions about whether they were identical (MZ) or fraternal (DZ). (Opposite-sex twins are always DZ.) None of these questions yields a perfect assessment of zygosity. The latter can only be obtained by performing molecular genetic analysis. Because the molecular genetic analysis of the MIDUS twin data did not begin until well after we performed the initial behavior genetic analyses, a classification rule was developed based on comparison with data from members of the Virginia Twin Registry (VTR) who were previously included in molecular genetic analyses. Eight self-report measures about whether the twins were identical or fraternal were included both in MIDUS and in the VTR. These variables were used to estimate a logistic regression equation in the VTR data to predict zygosity that used a classification of MZ and DZ based on molecular genetic analysis. The coefficients from this prediction equation were then used to generate predicted probabilities of being

MZ versus DZ in the MIDUS data. A strong classification (defined as a predicted probability of being MZ either less than 10 percent or greater than 90 percent) was obtained for 86 percent of the pairs, while a likely classification (defined as a predicted probability of being MZ outside of the 40 percent to 60 percent range) was obtained for an additional 10.5 percent. The remaining 3.5 percent were excluded from the study.

#### Measures

Four measures were used as indicators of self-reported health. Three focused on self-rated health perceptions and the fourth on functional status. The first of the three self-rated health measures asked about overall health without distinguishing mental and physical. The wording was as follows: "How would you rate your health these days on a 0–10 scale where 0 is the worst possible health and 10 is perfect health?" Separate singleitem questions were then asked about rating physical health and mental health as either poor, fair, good, very good, or excellent (coded 1–5). The functional status scale was made up of responses to a question about how much health problems "limit you in doing each of the following activities." Response categories were a lot, some, a little, and not at all (coded 1–4). Seven representative intermediate activities of daily living were included in the assessment (e.g., bending, kneeling, or stooping; lifting or carrying groceries). Responses were summed into a scale with a 7–28 range. All four self-reported health measures were standardized to have a mean of 0 and variance of 1 in the nationally representative component of the MIDUS sample. Means and variances were not constrained to be equal across the twin and sib subsamples, making it possible to study betweensubsample differences in means and variances.

Short forms of the six Ryff–Keyes psychological well-being scales (Ryff and Keyes 1995) were used to measure psychological well-being. As noted in the introduction, these six dimensions (with illustrative items in parentheses) include the following: (1) self-acceptance ("I like most parts of my personality"); (2) personal growth ("I think it is important to have new experiences that challenge how I think about myself and the world"); (3) purpose in life ("Some people wander aimlessly through life, but I am not one of them"); (4) positive relations with others ("People would describe me as a giving person, willing to share my time with others"); (5) environmental mastery ("In general, I feel that I am in charge of the situation in which I live"); and (6) autonomy ("I judge myself by what I think is important, not by the values of what others think is important"). The questions asked respondents to indicate how strongly they agreed or

disagreed with each of the statements. Response options included agreeing or disagreeing either strongly, somewhat, or only a little. A "don't know" category was also included as the midpoint on the 1–7 scale, with 1 being "strongly agree" and 7 being "strongly disagree." Each of the six dimensions was measured with either three or four items that were selected on the basis of extensive pilot work to be optimal indicators of the more extensive scales developed by Ryff and Keyes (1995). As with the measures of self-reported health, these scales were standardized to have a mean of 0 and a variance of 1 in the nationally representative component of the MIDUS sample.

Social responsibility, finally, was assessed with scales developed by Rossi (2001) to tap four key dimensions of social responsibility. The first three of these four dimensions were assessed by asking respondents to "rate how much obligation you would feel" in each of a number of hypothetical situations on a 0–10 scale, where 0 means "no obligation at all" and 10 means "a very great obligation." The scales with representative items in parentheses include the following: (1) normative altruistic obligation ("to volunteer time or money to social causes you support"); (2) normative civic and job obligation ("to testify in court about an accident you witnessed"); and (3) normative primary obligation ("to raise the child of a close friend if the friend died"). Four items were used to measure each of the first two dimensions and eight to measure the third. The fourth measure was a singe-item question about (4) contributions to others ("How would you rate your contribution to the welfare and well-being of other people these days?"). Responses were recorded on a 0-10 selfanchoring scale, where 0 represented "the worst" and 10 represented "the best." As in the health and psychological well-being domains, each social responsibility scale was standardized to have a mean of 0 and a variance of 1 in the nationally representative component of the MIDUS sample.

# Analysis Methods

The logic of behavior genetic analysis of twin data hinges on comparing similarities in measured variables of MZ twins, who share 100 percent of their genes identical by descent, and other sib-pairs, usually same-sex DZ pairs, who share, on average, 50 percent of their genes. Differences in similarity are interpreted as the result of genetic influences (Loehlin 1989). This interpretation implicitly assumes that environmental factors are not more similar for MZ twins than for other sib-pairs. This "equal-environment" assumption is plausible for narrowly defined environmental exposures, such as fluoride in water or lead in house paint.

However, the assumption can be called into question for broader environmental variables, such as preferential treatment of one twin by parents, which might be less likely to occur in MZ pairs than in other sib-pairs (Rowe 1983). As described in the first part of the results section, a special investigation of this issue was undertaken before computing correlations. In addition, a special analysis was undertaken of a second plausible possibility that could lead to differential nongenetic influences on differential similarities of MZ twins in comparison to other sib-pairs: the possibility of behavioral differentiation based on twin status (Plomin and Daniels 1987). The notion here is that MZ twins, because of their physical similarities, might be more inclined to differentiate themselves than other sibs might be.

Once the equal-environment assumption and twin differentiation assumption were investigated and either adjusted for (in the case of the equal-environment assumption, which, as described in the first part of the results section, was found to be violated) or shown not to be a problem (in the case of the twin differentiation hypothesis), twin and sibling resemblance on the outcome measures was assessed in two ways. The first consisted of a simple comparison of Pearson correlations for similarities in the outcomes among MZ twin-pairs, same-sex DZ pairs, and same-sex nontwin sib-pairs. If strong genetic influences were at work in explaining variance in these measures in the population, we would expect the correlations to be much larger for MZ pairs than for same-sex DZ and sibling pairs, but we would not expect to find any difference between same-sex DZ and nontwin sib-pairs. If being a twin itself affects similarity for reasons unrelated to genes, then we would expect to find stronger correlations in same-sex DZ pairs than nontwin pairs. If the relevant genes were not the same in males and females, we would expect higher correlations in same-sex versus opposite-sex pairs. It is also possible that the genes are the same but have different effects in same-sex male versus same-sex female pairs.

The second method used to study twin and sibling resemblance was linear structural equation analysis (Neale and Cardone 1992). The goal was to reproduce the within-pair variances and covariances of the eight subsamples with maximum parsimony. The eight subsamples included separate male and female same-sex MZ, DZ, and nontwin sib-pairs as well as opposite-sex DZ and nontwin pairs. There are three observed pieces of information in each of the eight matrices (two variances and one covariance), for a total of twenty-four pieces of information that we seek to reproduce. We began the modeling by assuming that four broad classes

of influences contribute to these observed variances and covariances: (1) additive genes, which we denote as A ("additive" in the sense that we assume the effect to be twice as strong for MZ pairs than other pairs because MZ twins share 100 percent of their genes rather than the average of 50 percent for other sib-pairs); (2) common environmental influences, which we denote as C, that affect the similarity of all twin-pairs and sibpairs equally; (3) special twin environment influences, which we denote as T, that lead twins (whether MZ or DZ) to be more similar to each other than nontwins are; and (4) an individual-specific environmental component, which we denote as E, that represents all the unique experiences an individual has that are not shared with his or her siblings. The models were subsequently expanded to allow for gender differences in parameter values as well as for special genetic (A') and common environmental (E') effects that are different for males than females. All models were fit using the Mx software package (Neale 1991). Comparative model fit was assessed using both the Akaike information criterion (AIC; Akaike 1987) and the Bayesian information criterion (BIC; Schwarz 1978). However, because these two criteria always yielded the same conclusions about the best-fitting models in this particular investigation, only the AIC is reported here. The model with the lowest AIC is defined as having the best balance of fit and parsimony.

# RESULTS The Equal-Environment Assumption

Given the importance of the equal-environmental assumption to the accurate interpretation of twin data, we began by testing the validity of this assumption. This was done by using a standard method that begins with questions included in the twin questionnaire that asked twins about three aspects of environmental similarity during their childhood years: how often they played together, how often they were dressed identically, and how often they were placed in the same classroom in school. We recognize that these are only superficial marker items of the complex ways in which twins are treated similarly or differently. Nonetheless, items similar to these have been shown to be sensitive in previous research comparing the environments of MZ and DZ twins.

We found that MIDUS MZ twins repeatedly reported significantly greater similarities than did same-sex DZ twins on all three measures, which is consistent with the previous research. This was expected. However, the research question of real interest was whether these greater similarities in childhood experiences are relevant to the outcomes under

consideration in this chapter. Not all environmental similarities are meaningful ones. If, for example, being dressed in identical clothing as children is unrelated to adult health, well-being, and social responsibility, then the greater similarity of MZ than DZ in this experience is irrelevant for our purposes. To evaluate this issue, we estimated a series of linear regression equations in which MZ-DZ differences in similarity on outcome variables were related to differences in similarity of childhood experiences. This analysis of differences in similarity required us to begin by computing the absolute value of within-pair differences on the outcome measures and using these difference scores as dependent variables in regression analyses that treated the twin-pairs as units of analysis. Predictors included a dummy variable coded 1 for MZ pairs and 0 for DZ pairs and the average within-pair scores on the three questions regarding similarity of childhood environmental experiences. The critical test was whether these three scores significantly predicted within-pair similarities in the outcomes after controlling for zygosity.

We would expect a mostly positive sign pattern of associations of the scores with the outcomes if the equal-environment assumption were violated, because similarity in childhood experience should predict similarity in the outcomes. As shown in table 1, a pattern of this sort was found with 27 (64 percent) of the 42 coefficients positive. However, this sign pattern was confined to having similar playmates (86 percent of coefficients positive), while there was no meaningful preponderance of positive coefficients for the effects of being in the same school classroom (57 percent) or dressing identically (50 percent). Consistent with the sign patterns, 36 percent of the coefficients for similar playmates are significant at the .05 level, and all of these significant coefficients are positive in sign. Only 7 percent of the coefficients for the other predictors are significant, and the sign pattern of these coefficients is inconsistent.

On the basis of these results, we concluded that the equal-environment assumption is violated for the outcomes considered here. It is worth noting that this violation is small in magnitude, with correlations averaging only .07 for the relationships between similarity in childhood playmates and similarity in the outcomes. Nonetheless, because the childhood experience measures are nothing more than crude indicators rather than fine-grained measures of environmental similarity, the disattenuated correlation could be substantial. In an effort to adjust for this bias to the extent possible in the data, we weighted the twin data so that the distribution of the MZ twins on responses to the question about childhood playmates was set equal to the distribution found among same-sex DZ twins. All further

TABLE 1 Assessment of the Equal-Environment Assumption in Same-Sex Twin-Pairs

|                                    | Child     | hood Similariti | es    |                 |
|------------------------------------|-----------|-----------------|-------|-----------------|
|                                    | Playmates | School Class    | Dress | $F_{3,582}^{a}$ |
| Self-reported health               |           |                 |       | -               |
| Health perception                  | .11*      | 03              | 05    | 1.85            |
| Self-rated physical health         | .12*      | 03              | 03    | 1.59            |
| Self-rated mental health           | .08       | .05             | .07   | 3.96*           |
| Functional status                  | .13*      | 01*             | 09    | 4.34*           |
| Social responsibility              |           |                 |       |                 |
| Normative altruism obligation      | .09       | .00             | .01   | 1.00            |
| Normative civic and job obligation | .04       | .13*            | .03   | 5.53*           |
| Normative primary obligation       | .15*      | 05              | 01    | 2.42            |
| Contribution to others             | 08        | .00             | .00   | 0.65            |
| Psychological well-being           |           |                 |       |                 |
| Autonomy                           | .09       | .01             | 03    | 1.04            |
| Environmental mastery              | .05       | .50             | 04    | 1.06            |
| Personal growth                    | .05       | <b></b> 05      | .06   | 1.55            |
| Positive relations with others     | .08       | .07             | .02   | 3.08*           |
| Purpose in life                    | .14*      | .05             | 01    | 2.35            |
| Self-acceptance                    | 02        | .06             | .05   | 2.06            |

Notes: Results are based on a series of fourteen separate linear regression equations with twin-pairs as the unit of analyses, each including a dummy variable for zygosity and the three childhood similarity measures to predict the absolute value of the difference in the outcome scores between cotwins. A total of 594 twin-pairs were included in the analysis. The coefficients reported in the table are standardized linear regression coefficients.

<sup>a</sup>The *F*-tests evaluate the joint significance of the three childhood similarity measures in predicting the outcome.

results reported in this chapter are based on analyses of these weighted data. To the extent that this adjustment was inadequate, the analyses presented later in the chapter overestimate the effects of genes because excess similarities of MZ pairs in comparison to DZ pairs will be interpreted as the result of genetic influences rather than environmental influences.

#### Behavioral Differentiation Based on Twin Status

Another important issue to consider before turning to an analysis of genetic influences is the possibility that MZ twins might be more likely than DZ twins to make themselves distinct from their cotwins by consciously selecting environmental experiences that are different from those selected by their identical brother or sister. If one twin chooses to play baseball, in such a scenario, the other would avoid baseball at all costs. If this kind of systematic differentiation occurs, it would violate the assumptions of the models considered here by introducing a systematic

<sup>\*</sup>Significant at the .05 level, two-sided test.

TABLE 2 Within-Pair Correlations of

|                                    | 1     | MZ     |
|------------------------------------|-------|--------|
|                                    | Male  | Female |
| Self-reported health               |       |        |
| Health perception                  | .26*  | .38*   |
| Self-rated physical health         | .20*  | .38*   |
| Self-rated mental health           | .18*  | .26*   |
| Functional status                  | .19*  | .31*   |
| Social responsibility              |       |        |
| Normative altruism obligation      | .35*  | .26*   |
| Normative civic and job obligation | .37*  | .45*   |
| Normative primary obligation       | .14*  | .26*   |
| Contribution to others             | .25*  | .07    |
| Psychological well-being           |       |        |
| Autonomy                           | .30*  | .39*   |
| Environmental mastery              | .35*  | .37*   |
| Personal growth                    | .24*  | .47*   |
| Positive relations with others     | .35*  | .35*   |
| Purpose in life                    | .22*  | .39*   |
| Self-acceptance                    | .39*  | .47*   |
| (n)                                | (149) | (186)  |

<sup>\*</sup>Significant at the .05 level, two-sided test.

negative correlation within MZ pairs. This bias, in turn, would lead to an underestimation of genetic effects. We would not expect a direct bias of this sort with respect to the health outcomes considered here. There is no reason to believe, for example, that one cotwin will decide to choose poor mental health to differentiate himself from his mentally healthy cotwin, but such a possibility is more plausible for some aspects of well-being and social responsibility. For example, if one twin in a pair takes on the role of leader and develops a very high level of environmental mastery, this might lead his cotwin to respond by taking on the role of follower and developing a low level of environmental mastery. Similar processes could occur with respect to such aspects of social responsibility as normative primary obligations (i.e., one twin becomes the family caregiver, while the cotwin develops a low sense of familial obligation).

We know that differentiation processes of this sort occur in families (Plomin and Daniels 1987). However, as with the equal-environment assumption, the important question for our purposes is whether these processes are more pronounced for MZ twins than for DZ twins or nontwin sibs in ways that affect the outcomes of consideration here. To evaluate this question, we turned to an inspection of the variances of the outcome measures. Greater within-pair differentiation among MZ twins will lead

Outcomes Stratified by Zygosity and Sex

|       | DZ     |          |       | Nontwin Sibs | 3        |
|-------|--------|----------|-------|--------------|----------|
| Male  | Female | Opposite | Male  | Female       | Opposite |
| 22*   | 20*    | 0.7      |       | 1 = 4        | 0.1      |
| .22*  | .30*   | .05      | .11   | .15*         | 01       |
| .26*  | .33*   | .12      | .10   | .38*         | .09      |
| .22*  | .18*   | .10      | .17   | .09          | .10      |
| .24*  | .34*   | .17*     | .15   | .43*         | .17*     |
| .25*  | .38*   | .08      | .13   | .16*         | .13*     |
| .27*  | .26*   | .11      | 02    | .18*         | .24*     |
| .20*  | .33*   | .11      | .11   | .09          | .06      |
| .10   | .06    | .10      | .12   | .07          | .12*     |
| .01   | .13    | 03       | .14   | .03          | .04      |
| .07   | .04    | .12      | .21*  | .06          |          |
| .27*  | .29*   | .13      | .18*  | .05          | .28*     |
| .21*  | .15*   | .06      | .03   | .10          | .22*     |
| .06   | .30*   | .06      | .10   | .12          | 16*      |
| .10   | .16*   | .08      | .03   | 02           | .16*     |
| (103) | (156)  | (200)    | (128) | (192)        | (290)    |

to variances being higher, even if means are similar for MZ twins in comparison to DZ twins and nontwin siblings. An analysis was performed that examined means and variances of the ten outcome variables within sex for MZ, same-sex DZ, and same-sex nontwin sib-pairs. No evidence was found for meaningful differences either in means or in variances across these three subsamples for any of the outcomes either for male pairs or for female pairs. On the basis of this consistent result, we put aside concerns that greater behavioral differentiation among identical twins might introduce bias into our analyses of these outcome domains.

#### Correlations

The within-pair correlations for measures in all three outcome domains are reported in table 2 separately by sex for MZ twins (weighted to adjust for violation of the equal-environment assumption), DZ twins, and nontwin sib-pairs. Conventional twin analyses work largely with same-sex pairs of MZ and DZ twins and generally do not distinguish male pairs from female pairs. As a result, there is only one critical comparison in such analyses: between MZ pairs and DZ pairs. As described in the section on analysis methods, evidence of higher correlations in the former than the latter are taken to mean that genetic influences are at work based

on the assumption that greater similarity in genetic makeup is the only factor that differentiates MZ pairs from DZ pairs (i.e., the assumption that environmental similarity is identical for MZ and DZ pairs).

The results in the first four rows of table 2 present comparisons between MZ and same-sex DZ pairs by sex. Every correlation is positive, and 84 percent are significant at the .05 level, showing that there is meaningful familial aggregation for these outcomes. However, the correlations are modest in magnitude, with a range of .01-.47, and the majority in the range of .20–.40. There is mixed evidence for genetic effects. The weakest evidence is in the self-reported health domain for males and the social responsibility domain for females, in each of which only one of the four correlations is higher for MZ than DZ pairs. The strongest evidence is in the psychological well-being domain, where five of six correlations are higher for MZ rather than DZ male pairs and six of six for MZ rather than DZ female pairs. It is important to note that the well-being MZ–DZ correlation differences are quite substantial in most cases, with eight of the twelve MZ correlations more than twice as large as the DZ correlations. Differences as large as this are important if they are the result of more than sampling error, because they go beyond the 2:1 ratio we would expect on the basis of additive genetic effects, raising the possibility of nonadditive genetic effects. Evidence for genetic effects is intermediate in the self-reported health domain among females and the social responsibility domain for males, in each of which three of the four correlations are higher for MZ than DZ pairs, but with differences almost always less than a 2:1 ratio.

The inclusion of nontwin sib-pairs in the analysis also allows us to evaluate the environmental effects of being a twin by comparing correlations for MZ twins with those for nontwin sibs. This can be done for each of the 14 outcomes separately for male, female, and opposite-sex pairs, for a total of 42 comparisons. An inspection of the last six columns of table 2 shows a clear trend for higher correlations among same-sex twins than same-sex nontwin sibs (71 percent of comparisons), but not among opposite-sex twins compared with opposite-sex sibs (29 percent of comparisons). One potentially important biasing factor here is that cotwins are always the same age, but nontwin siblings differ in age. To control for this potential bias, we recomputed the sib-pair correlations for pairs that do not differ by more than three years in age. The same general pattern continued to hold as the one seen in table 2.

It is interesting to compare differences in correlations to get a rough sense of the relative importance of genetic influences (MZ versus samesex DZ) compared with environmental influences of being a twin (DZ versus nontwin sibs). In the case of self-rated health among males, for example, the MZ–DZ difference is .04 (.26–.22), suggesting that there is no meaningful genetic effect on this outcome. The DZ-sib difference, in comparison, is .11 (.22–.11), suggesting that environmental experiences associated with being a twin have a modest influence on perceptions of self-rated health. A systematic comparison of this sort across all fourteen outcomes both for male and female same-sex pairs shows that the MZ–DZ difference is larger than the DZ-sib difference in roughly half the cases, while the DZ-sib difference is larger than the MZ–DZ difference in the other half. This rough comparison suggests that environmental effects of being a twin are as common as genetic effects for this set of outcomes.

#### Heritability

A comprehensive series of structural equation models was fit to the eight  $2 \times 2$  covariance matrices of each outcome measure. An illustration is presented in table 3 for one of the outcomes—self-rated mental health. A total of twenty-three models were fit to the covariance matrices for this outcome. These represent all logically possible models that could be identified and were substantively plausible for these data. The most complex model, with eleven parameters, is model AA'DD'TE. This model allows for additive (A) and nonadditive (D) genetic effects that are the same for men and women as well as for sex-specific additive (A') and nonadditive (D') genetic effects, sex-specific effects of being a twin (T), environmental effects that are unique to individuals (E), and constant variance across subsamples. Other less complex models included subsets of these parameters either with or without a common environment (C) parameter.

It is noteworthy that none of these models included both nonadditive genetic effects (D) and common environmental effects (C). This is because nonadditive genetic effects only make sense to estimate when the MZ correlation is more than twice as large as the same-sex DZ correlation. In cases of this sort, a model that includes all additive environmental and genetic effects always estimates a value of 0 for C. Importantly, to identify a model including D, it is necessary to constrain C to some fixed value, which is usually set to 0. It is important to note that there can be situations in which nonadditive genetic effects and common environmental effects are both at work. However, a model including parameters for both of these influences cannot be estimated with the data available to us here. As a result, a model that includes a D effect should be interpreted as indicating the existence of nonadditive genetic effects but leaving uncertain whether or not there are also common environmental effects.

TABLE 3 Summary Evaluations of

|                  |          |    |       |      |     |      | ···· |     |     |
|------------------|----------|----|-------|------|-----|------|------|-----|-----|
|                  |          |    |       |      |     | M    | ale  |     | _ ; |
| Model            | $\chi^2$ | df | AIC   | Α    | A'  | С    | C′   | D   |     |
| AA'CTE           | 16.3     | 15 | -13.7 | .02  | .00 | .16  |      |     |     |
| ACC'TE           | 16.3     | 15 | -13.7 | .02  |     | .16  | .00  |     |     |
| ACTE             | 16.3     | 16 | -15.7 | .02  |     | .16  |      |     |     |
| $ACTE^a$         | 18.0     | 20 | -22.0 | .17  |     | .02  |      |     |     |
| ACE              | 17.1     | 18 | -18.9 | .05  |     | .15  |      |     |     |
| $ACE^a$          | 18.8     | 21 | -23.2 | .23  |     | .01  |      |     |     |
| ATE              | 17.7     | 18 | -18.3 | .24* |     |      |      |     |     |
| $ATE^a$          | 18.1     | 21 | -23.9 | .20* |     |      |      |     |     |
| CTE              | 17.6     | 18 | -18.4 |      |     | .19* |      |     |     |
| $CTE^a$          | 19.4     | 21 | -22.6 |      |     | .11* |      |     |     |
| AE               | 18.7     | 20 | -21.3 | .23* |     |      |      |     |     |
| $AE^a$           | 18.8     | 22 | -25.2 | .25* |     |      |      |     |     |
| CE               | 22.1     | 20 | -17.9 |      |     | .16* |      |     |     |
| $CE^a$           | 22.2     | 22 | -21.8 |      |     | .15* |      |     |     |
| TE               | 26.9     | 20 | 13.1  |      |     |      |      |     |     |
| $TE^a$           | 27.1     | 22 | -16.9 |      |     |      |      |     |     |
| E                | 53.9     | 22 | 9.9   |      |     |      |      |     |     |
| $E^a$            | 53.9     | 23 | 7.9   |      |     |      |      |     |     |
| AA'DD'TE         | 17.6     | 14 | -10.4 | .19  | .05 |      |      | .00 |     |
| ADTE             | 17.6     | 16 | -14.4 | .24  |     |      |      | .00 |     |
| $ADTE^a$         | 18.1     | 20 | -21.9 | .20  |     |      |      | .00 |     |
| ADE              | 18.3     | 18 | -17.7 | .25* |     |      |      | .00 |     |
| ADE <sup>a</sup> | 18.8     | 21 | -23.2 | .25* |     |      |      | .00 | _   |

Notes: Results are based on a series of twenty three separate structural equation models, each of which attempted to fit the covariance structure of the eight subsample matrices with the series of parameters specified in the first column of the table. The AE model is the best-fitting model based on the AIC. A, additive genetic effect; A', sex-specific additive genetic effect; D, nonadditive genetic effect; D', sex-specific nonadditive genetic effect; C, common environmental effect; C', sex-specific common environmental effect; T, environmental effect of being a twin; and E, individual-specific environmental effect.

"Models in which parameters were constrained to be equal for males and females.

The best-fitting model for self-rated mental health is the AE model that constrains the coefficients to have the same values for males and females. The AIC of -25.2 for this model is considerably lower than that for any other model, indicating that the preference for this model over others is not sensitive to minor differences in model fit. The estimate for A is .25, which means that 25 percent of the variance in self-rated mental health is estimated to be the result of additive genetic effects. The remaining 75 percent of the variance is estimated to be the result of individual-specific

<sup>\*</sup>Significant at the .05 level, two-sided test.

Univariate Model Fit

| Explained Variance |      |       |      |      |        |      |       |  |
|--------------------|------|-------|------|------|--------|------|-------|--|
|                    |      |       |      |      | Female |      |       |  |
| D'                 | T    | Е     | A    | С    | D      | T    | Е     |  |
|                    | .03  | .79*  | .18  | .01  |        | .07  | .74*  |  |
|                    | .03  | .79*  | .18  | .01  |        | .07  | .74*  |  |
|                    | .03  | .79*  | .18  | .01  |        | .07  | .74*  |  |
|                    | .05  | .76*  | .17  | .02  |        | .05  | .76*  |  |
|                    |      | .79*  | .25* | .01  |        |      | .74*  |  |
|                    |      | .76*  | .23  | .01  |        |      | .76*  |  |
|                    | .00  | .75*  | .17* |      |        | .09  | .74*  |  |
|                    | .05  | .75*  | .20* |      |        | .05  | .75*  |  |
|                    | .00  | .81*  |      | .07  |        | .15  | .78*  |  |
|                    | .09  | .81*  |      | .11* |        | .09  | .81*  |  |
|                    |      | .77*  | .26* |      |        |      | .74*  |  |
|                    |      | .75*  | .25* |      |        |      | .75*  |  |
|                    |      | .84*  |      | .15* |        |      | .85*  |  |
|                    |      | .85*  |      | .15* |        |      | .85*  |  |
|                    | .18* | .82*  |      |      |        | .20* | .80*  |  |
|                    | .19* | .81*  |      |      |        | .19* | .81*  |  |
|                    |      | 1.00* |      |      |        |      | 1.00* |  |
|                    |      | 1.00* |      |      |        |      | 1.00* |  |
| .00                | .01  | .75*  | .16  |      | .03    | .08  | .73*  |  |
|                    | .01  | .75*  | .14  |      | .05    | .08  | .73*  |  |
|                    | .05  | .75*  | .20  |      | .00    | .05  | .75*  |  |
|                    |      | .75*  | .18  |      | .10    |      | .72*  |  |
|                    |      | .75*  | .25* |      | .00    |      | .75*  |  |

environmental effects. It is instructive to go back to the fourth line in table 2 to compare the correlations with the model parameters in order to see the way in which constraints are being imposed. The fact that sex-specific effects are assumed not to exist in the best-fitting AE model means that the three male versus female same-sex pair comparisons in table 2 were judged to be insignificant as a set. This, in turn, means that the .18 MZ male correlation and the .26 MZ female correlation were averaged in the model to a correlation of approximately .22. The fact that the environmental effect of being a twin is assumed not to exist in the AE model means that the DZ and nontwin sibling correlations were also averaged and treated as a single correlation. A comparison of the averaged MZ correlation (.22) and the pooled correlation for the other six subsamples (.12) yields a ratio close to 2:1, which is consistent with a genetic effect that linearly extrapolates to a common environmental effect of 0. In other words, if 100 percent of genetic similarity leads to a

TABLE 4 Model Statistics and Standardized Estimates

|                                    | Model            | $\chi^2$ | df | AIC              |
|------------------------------------|------------------|----------|----|------------------|
| Self-reported health               |                  |          |    |                  |
| Health perception                  | ATE              | 22.9     | 18 | -13.2            |
| •                                  | ADE              | 22.9     | 18 | -13.1            |
| Self-rated physical health         | CTE              | 16.0     | 18 | -20.0            |
| Self-rated mental health           | $AE^a$           | 18.8     | 22 | -25.2            |
| Functional status                  | CE               | 60.3     | 20 | 20.3             |
|                                    | CTE              | 55.7     | 18 | 19.7             |
| Social responsibility              |                  |          |    |                  |
| Normative altruism obligation      | $AE^a$           | 31.2     | 22 | -12.8            |
| 8                                  | $CTE^a$          | 29.6     | 21 | -12.4            |
| Normative civic and job obligation | $AE^a$           | 55.4     | 22 | 11.4             |
| Normative primary obligation       | $CTE^a$          | 17.5     | 21 | -24.5            |
| Contribution to others             | CE               | 11.9     | 20 | -28.1            |
|                                    | AE               | 11.2     | 20 | -28.8            |
| Psychological well-being           |                  |          |    |                  |
| Autonomy                           | ADE              | 17.1     | 18 | -18.9            |
| Environmental mastery              | $ADE^a$          | 26.7     | 21 | -15.3            |
| Personal growth                    | AE               | 29.5     | 20 | <del></del> 10.5 |
|                                    | $AE^a$           | 33.2     | 22 | -10.8            |
| Positive relations with others     | $AE^a$           | 12.3     | 22 | -31.7            |
| Purpose in life                    | AE               | 13.7     | 20 | -26.3            |
| Self-acceptance                    | ADE <sup>a</sup> | 27.3     | 21 | -14.7            |

*Notes:* Best-fitting models were selected using the AIC. See footnote b in table 3 for definitions of symbols. Additive and nonadditive genetic variance components are combined in a single measure of heritability (H).

.22 correlation and 50 percent genetic similarity reduces the correlation by .10 (i.e., from .22 for MZ to .12 for all other sib-pairs), then a linear extrapolation would lead to the conclusion that 0 percent genetic similarity would reduce the correlation by another .10, resulting in an effect of common environment equal to .02 (i.e., the observed .12 correlation in the pooled non-MZ subsamples minus the extrapolated .10 based on a comparison between the averaged MZ correlation) and the pooled non-MZ correlation. An effect of .02 is nonsignificant with a sample of the size analyzed here, leading to the conclusion that only additive genetic effects are involved in the familial aggregation of self-reported mental health.

The same model-fitting logic was applied to the remaining outcomes, and a best-fitting model was selected for each. Summary results are

<sup>&</sup>lt;sup>a</sup>Models in which parameters were constrained to be equal across sexes.

<sup>\*</sup> Significant at the .05 level, two-sided test.

of Best-Fitting Models for Each Outcome

|      |      | Ex     | plained Vari | ance |      |      |      |
|------|------|--------|--------------|------|------|------|------|
|      | N    | lale . |              | ,    | Fer  | nale |      |
| Н    | С    | T      | Е            | Н    | С    | Т    | Е    |
| .00  |      | .26*   | .74*         | .37* |      | .03  | .60* |
| .34* |      |        | .66*         | .40* |      | .05  | .60* |
|      | .03  | .18*   | .78*         |      | .37* | .00  | .63* |
| .25* |      |        | .75*         | .25* |      |      | .75* |
|      | .12* |        | .88*         |      | .36* |      | .64* |
|      | .07* | .17*   | .76*         |      | .37* | .00  | .63* |
| .30* |      |        | .70*         | .30* |      |      | .70* |
|      | .15* | .09    | .76*         |      | .15* | .09  | .76* |
| .37* |      |        | .63*         | .37* |      |      | .63* |
|      | .08* | .12*   | .80*         |      | .08* | .12* | .80* |
|      | .17* |        | .83*         |      | .07* |      | .93* |
| .25* |      |        | .75*         | .11* |      |      | .89* |
| .27  |      |        | .73*         | .37* |      |      | .63* |
| .33* |      |        | .67*         | .33* |      |      | .67* |
| .33* |      |        | .67*         | .43* |      |      | .57* |
| .38* |      |        | .62*         | .38* |      |      | .62* |
| .32* |      |        | .68*         | .32* |      |      | .68* |
| .19* |      |        | .81*         | .39* |      |      | .61* |
| .41  |      |        | .59*         | .41* |      |      | .59* |

reported in table 4. In cases where the best-fitting model had an AIC less than one point lower than that of competing models, the results for the competing models are also reported in the summary table. Results are reported for a total of nineteen models across the fourteen outcomes. Consistent with the pattern seen in table 2, evidence for a statistically significant genetic effect is more consistent in the psychological well-being domain (100 percent of models) than in either the self-reported health domain (50 percent) or the social responsibility domain (50 percent). The magnitudes of the significant genetic effects—with additive and nonadditive effect size estimates combined in the table into a total heritability (H) estimate—are fairly consistent across the three domains: .25–.40 for self-reported health, .11–.43 for psychological well-being, and .25–.37 for social responsibility. It is important to note that in interpreting this range, measurement error is included in the total estimate of variance. If only 70–80 percent of the variance in the observed outcomes is reliable, heritabilities in the .11–.43 range represent between 14 percent and 61 percent of true score variance.

Even with adjustments for plausible levels of measurement error, the majority of variance in all but a few of the outcomes is estimated to be environmental. In only 50 percent of the models, however, is this variance estimated to be responsible for a significant part of familial aggregation (i.e., significant C or T) effects. In the others, only individual specific environmental influences appear to be at work. It is noteworthy, though, that a significant nonadditive genetic effect is found in one-third of the models that omit common environmental effects. As noted in the last subsection, it is not possible to estimate nonadditive genetic effects and common environmental effects in the same model, given the structure of the data being analyzed here. As a result, the possibility of common environmental effects cannot be ruled out in these cases.

More than half of the best-fitting models include variance estimates that are significantly different for males and females. In general, the heritability estimates are larger for females than males (five of six) in sex-specific models that include significant genetic effects. There is no consistent pattern of sex differences, in comparison, in the magnitudes of common environmental effects in sex-specific models that include significant common environmental components (three with males larger and three with females larger). Taken together, these results mean that the effects of unique environmental experiences are generally larger for males than females in models that allow sex differences in parameter estimates (seven of nine).

#### Discussion

The results are broadly consistent with previous studies in finding significant heritabilities of self-rated health and certain aspects of psychological well-being. However, the results also go beyond these earlier studies both in specifying some previously documented associations and in finding significant heritabilities for aspects of well-being and social responsibility that have not previously been the subject of behavior genetic research.

Before turning to a comment on the specific results, it is important to note that three of the outcomes in table 4 have two models that have very similar AIC values, although the parameters in these models are very different in their implications. These three outcomes are health perception, normative altruistic obligation, and contribution to others. One model for each of these outcomes includes a term for a genetic effect but no term for a common environmental effect, while the other model includes a term for a common environmental effect but not for a genetic

effect. This kind of situation often occurs in analyses of data obtained from moderately sized samples in which there are true modest effects of both genes and common environment. In cases of this sort, there is not enough statistical power to detect both effects, and stochastic factors can lead to one model being favored over the other. It is important to keep this sensitivity of results in mind when interpreting the findings.

The finding that the MIDUS global measure of self-rated health is heritable is consistent with previous research reviewed in the introduction. However, unlike previous studies, MIDUS also included separate questions about self-rated physical and mental health. Only self-rated mental health was significantly heritable. It is understandable that self-rated mental health would be heritable in light of the substantial evidence for the heritability of psychiatric disorders that was reviewed in the introduction. However, the failure to find significant heritability of self-rated physical health is surprising in light of the fact, noted in the introduction, that a number of chronic physical illnesses are known to be heritable. Apparently the perceptions that influence self-rated physical health are sufficiently independent of heritable physical illnesses not to show significant heritability.

Given this important difference in the determinants of self-rated physical health and self-rated mental health, it would seem wise for future health surveys to keep the two measures separate rather than combine them into a single global self-rated health question. The Center for Disease Control's Behavioral Risk Factor Surveillance System Survey is currently the only major ongoing health survey in the United States that asks separately about self-rated physical health and mental health. It would be useful if other major health surveys followed this lead in expanding the assessment of self-rated health to distinguish physical and mental components.

Our failure to find that self-reported functional status is heritable is inconsistent with the one study reviewed in the introduction that investigated this issue. As with self-rated physical health, this failure is surprising in light of evidence that many of the serious physical illnesses that are largely responsible for impairments in the domains assessed in MIDUS (e.g., difficulties climbing a flight of stairs or carrying a bag of groceries) are heritable. It is conceivable that the wide age range of the sample is at least partly responsible for this result, because the causes of limitations in physical functioning among younger people tend to be acute (e.g., a broken leg) and environmentally determined, thus diminishing the effects of heritable factors.

The finding that all six dimensions of psychological well-being are heritable extends the results of previous studies that documented heritabilities of some correlates of these dimensions. Importantly, common environmental effects are absent for all six measures of well-being. Although environmental factors are largely responsible for variance in all the aspects of well-being, these factors are associated with unique environmental experiences rather than with experiences that sibs share in common.

The finding of significant heritabilities for social responsibility, finally, is consistent with evidence reviewed in the introduction for the heritability of altruism. It is interesting to note that genetic effects were limited to perceptions of responsibility regarding community and civic life (normative altruistic obligation and normative civic and job obligation). There was no genetic influence, in comparison, on feelings of responsibility to close friends and relatives (normative primary obligation). Familial factors of a nongenetic kind played the key role for these more personal obligations. However, the caution noted at the beginning of this section has to be invoked in considering this specification. With a sample of the size available here and both genetic and common environmental effects of modest size, stochastic factors alone could explain this seeming specification. Independent replication is needed before concluding that social responsibility to close loved ones is less heritable or more strongly affected by common environmental factors than is community or civic social responsibility.

We are unaware of any research that has systematically investigated male-female differences in heritabilities in these outcome domains. It is noteworthy that the higher heritabilities for women than men are largely concentrated in the psychological well-being domain, with the largest difference being the much greater heritability among women than men for purpose in life. An obvious speculation is that this might be related to gender differences in role expectations involving affiliative rather than instrumental activities. There is no way to evaluate this possibility, though, with the MIDUS data. Serious investigation of this topic would require information to be obtained about differences in the nature of the purpose perceived by male and female respondents who report that they have a strong purpose in life. An expanded investigation of this sort in future research is warranted in light of the strong gender difference in the heritability of purpose in life.

Caution is needed in interpreting the heritability estimates presented here because of violation of the equal-environment assumption. Although we attempted to compensate for this violation by weighting the data, this was only a rough adjustment because of the coarseness of the measures of within-pair similarity in childhood environment. Some sense of the magnitude of the uncertainty introduced by this problem can be obtained by evaluating the implications of the equal-environment assumption for the estimation of genetic effects. This is possible because it is not necessary to assume that the environments of MZ and DZ twins are equally similar to identify the models examined here. It is merely necessary to assume that the MZ: DZ environmental similarity ratio (ESR) is known. Although this ratio is set to 1:1 when the equal-environment assumption is made, it can also be set to any other fixed value. If ESR is set to 2:1, then the A and C parameters are collinear, in which case separate effects of genes and common environment cannot be estimated. When ESR is less than 2:1, however, separate estimates of A and C can be obtained.

To investigate the sensitivity of the results reported here to the equalenvironment assumption, best-fitting models that contained significant genetic effects were re-estimated a number of times, with ESR values varied across the range between 1.0 and 2.0. As expected, the estimated heritability of the outcomes decreased and the standard error of the heritability estimate increased as the ESR approached 2:1. Most heritability estimates became nonsignificant when ESR exceeded 1.5:1. This means that the assumption that genetic influences exist hinges on our willingness to assume that within-pair environmental similarity is less than 50 percent greater among MZ twins than among non-MZ twins and sibs. This assumption has to take into account the fact that genetic similarity of MZ twins can elicit similarities in environmental response that should be considered aspects of genetic influence rather than aspects of environmental exposure (Scarr and McCartney 1983).

It is possible to get a more direct estimate of the effects of common environments net of genes by working with research designs that vary environments and hold genes constant. The design used in this chapter, in comparison, varied genetic similarity (i.e., MZ twins who share 100 percent of their genes compared with DZ twins and sibs who share an average of 50 percent of their genes) and treated environmental similarity as a residual category. An example of a design that varies environments and controls genes is one that compares half-sibs (who share 25 percent of their genes, on average) with pairs of cousins who are the offspring of identical twins. This is an informative comparison because these cousins are genetically the same as half-sibs (i.e., they share 25 percent of their genes, on average, compared with 12.5 percent for most cousins) because they have the "same" mother in terms of genes. However, we would expect

true half-sibs to be more similar if common environment plays a part in the outcomes under investigation because half-sibs are raised in the same household whereas cousins are not.

The half-sib design is rarely used in behavior genetics research because it has low power to estimate genetic effects (25 percent versus 12.5 percent shared genes, on average, for cousins who are versus those who are not the offspring of identical twins). An adoption design is much more powerful in this regard because, at least theoretically, it completely separates genetic effects from environmental effects. However, as a practical matter, the adoption design is limited in a number of ways. Some parents, especially fathers, are unknown and cannot be used in adoption studies. Adoption agencies try to match the racial-ethnic characteristics of children who are being adopted with the characteristics of their adopted parents so that their genes and environments are not strictly independent. Adoption samples are usually unrepresentative because many adoption agencies are unwilling to cooperate with genetic researchers. And the range of environments in adoptive families is quite restricted, because adoption agencies try to choose good homes for their adoptees.

In light of these considerations, many behavior geneticists have concluded that the discordant MZ-cotwin design is the preferred approach for performing studies that examine environmental influences on outcomes like those considered in this chapter in a way that accounts for genetic effects. This design uses the logic of a matched case-control design (Schlesselman 1982) in which pairs of identical twins are used to "control" for genes in studying the effects of environmental factors on within-pair differences in outcomes. It is impossible in this design to evaluate the effects of environmental experiences that are always shared by twins, such as the early death of a parent or other childhood adversities. Adoption designs are much better suited to evaluate those effects in a way that excludes the possibility of genetic effects. However, the effects of experiences that are not always shared by cotwins, such as getting good grades in school, on later outcomes, such as adult socioeconomic achievement and the health consequences of this achievement, can be evaluated in this design in a way that is uniquely able to exclude the possibility of bias as a result of uncontrolled genetic influences. Planned future analyses of the MIDUS data to investigate predictors of the outcomes considered here will use this design in a confirmatory mode. That is, they will attempt to replicate results about the effects of predictors found in the more conventional analyses of the full MIDUS sample by using the discordant MZ-cotwin design applied to the 335 MIDUS MZ twin-pairs. This approach could be especially useful in studying the predictors of psychological well-being, because the results presented in this chapter suggest that the environmental determinants of these outcomes are largely unique and, therefore, ideally suited to investigate with the discordant MZ-cotwin design.

#### REFERENCES

- Akaike, H. 1987. Factor analysis and AIC. Psychometrika 52:317–32.
- Allison, D. B., S. Heshka, M. C. Neale, D. T. Lykken, and S. B. Heymsfield. 1994. A genetic analysis of relative weight among 4,020 twin pairs, with an emphasis on sex effects. *Health Psychology* 13:362–65.
- Allison, D. B., J. Kaprio, M. Korkeila, M. Koskenvuo, M. C. Neale, and K. Hayakawa. 1996. The heritability of body mass index among an international sample of monozygotic twins reared apart. *International Journal of Obesity and Related Metabolic Disorders* 20:501–6.
- Barsky, A. J., P. D. Cleary, and G. L. Klerman. 1992. Determinants of perceived health status of medical outpatients. *Social Science and Medicine* 34:1147–54.
- Bergeman, C. S., R. Plomin, N. L. Pedersen, G. E. McClearn, and J. R. Nesselroade. 1990. Genetic and environmental influences on social support: The Swedish Adoption/Twin Study of Aging. *Journal of Gerontology* 45:101–6.
- Borhani, N. O., M. Feinleib, R. J. Garrison, J. C. Christian, and R. H. Rosenman. 1976. Genetic variance in blood pressure. *Acta Geneticae Medicae et Gemellologiae* 25:137–44.
- Carroll, D., J. K. Hewitt, K. A. Last, J. R. Turner, and J. Sims. 1985. A twin study of cardiac reactivity and its relationship to parental blood pressure. *Physiology and Behavior* 34:103–6.
- Fabsitz, R. R., P. Sholinsky, and D. Carmelli. 1994. Genetic influences on adult weight gain and maximum body mass index in male twins. *American Journal of Epidemiology* 140:711–20.
- Fylkesnes, K., and O. H. Forde. 1992. Determinants and dimensions involved in self-evaluation of health. *Social Science and Medicine* 35:271–79.
- Harris, J. R., N. L. Pedersen, G. E. McClearn, R. Plomin, and J. R. Nesselroade. 1992. Age differences in genetic and environmental influences for health from the Swedish Adoption/Twin Study of Aging. *Journal of Gerontology* 47:213–20.
- Heath, A. C., M. C. Neale, R. C. Kessler, L. J. Eaves, and K. S. Kendler. 1992. Evidence for genetic influences on personality from self-reports and informant ratings. *Journal of Personality and Social Psychology* 63:85–96.
- Hemminki, K., C. Dong, and P. Vaittinen. 1999. Familial risks in cervical cancer: Is there a hereditary component? *International Journal of Cancer* 82:775–81.
- Holm, N. V., M. Hauge, and B. Harvald. 1980. Etiologic factors of breast cancer elucidated by a study of unselected twins. *Journal of the National Cancer Institute* 65:285–98.
- Jablon, S., J. V. Neel, H. Gershowitz, and G. F. Atkinson. 1967. The NAS-NRC twin panel: Methods of construction of the panel, zygosity diagnosis, and proposed use. *American Journal of Human Genetics* 19:133–61.

- Jylha, M., E. Leskinen, E. Alanen, A. L. Leskinen, and E. Heikkinen. 1986. Self-rated health and associated factors among men of different ages. *Journal of Gerontology* 41:710–17.
- Kendler, K. S. 1997. Social support: A genetic-epidemiologic analysis. *American Journal of Psychiatry* 154:1398–1404.
- Kendler, K. S., C. O. Gardner, and C. A. Prescott. 1998. A population-based twin study of self-esteem and gender. *Psychological Medicine* 28:1403–9.
- Kendler, K. S., M. C. Neale, R. C. Kessler, A. C. Heath, and L. J. Eaves. 1992a. Generalized anxiety disorder in women. A population-based twin study. *Archives of General Psychiatry* 49:267–72.
- ——. 1992b. The genetic epidemiology of phobias in women. The interrelationship of agoraphobia, social phobia, situational phobia, and simple phobia. *Archives of General Psychiatry* 49:273–81.
- ——. 1993. A longitudinal twin study of personality and major depression in women. *Archives of General Psychiatry* 50:853–62.
- Kendler, K. S., N. L. Pedersen, M. C. Neale, and A. A. Mathe. 1995. A pilot Swedish twin study of affective illness including hospital- and population-ascertained subsamples: Results of model fitting. *Behavior Genetics* 25:217–32.
- Kendler, K. S., and C. D. Robinette. 1983. Schizophrenia in the National Academy of Sciences–National Research Council Twin Registry: A 16-year update. *American Journal of Psychiatry* 140:1551–63.
- Kendler, K. S., L. M. Thornton, S. E. Gilman, and R. C. Kessler. 2000. Sexual orientation in a U.S. national sample of twin and nontwin sibling pairs. *American Journal of Psychiatry* 157:1843–46.
- Kendler, K. S., E. E. Walters, M. C. Neale, R. C. Kessler, A. C. Heath, and L. J. Eaves. 1995. The structure of the genetic and environmental risk factors for six major psychiatric disorders in women: Phobia, generalized anxiety disorder, panic disorder, bulimia, major depression, and alcoholism. *Archives of General Psychiatry* 52:374–83.
- Kessler, R. C., K. S. Kendler, A. Heath, M. C. Neale, and L. J. Eaves. 1992. Social support, depressed mood, and adjustment to stress: A genetic epidemiologic investigation. *Journal of Personality and Social Psychology* 62:257–72.
- Levkoff, S. E., P. D. Cleary, and T. Wetle. 1987. Differences in the appraisal of health between aged and middle-aged adults. *Journal of Gerontology* 42:114–20.
- Lichtenstein P., N. V. Holm, P. K. Verkasalo, A. Iliadou, J. Kapiro, M. Koskenvuo, E. Pukkala, A. Skytthe, and K. Hemminki. 2000. Environmental and heritable factors in the causation of cancer: Analyses of cohorts of twins from Sweden, Denmark, and Finland. *New England Journal of Medicine* 343:78–85.
- Loehlin, J. C. 1989. Partitioning environmental and genetic contributions to behavioral development. *American Psychologist* 44:1285–94.
- ———. 1992. Genes and environment in personality development. Newbury Park, Calif.: Sage Publications.
- Loehlin, J. C., and R. C. Nichols. 1976. *Heredity, environment, and personality: A study of 850 sets of twins.* Austin: University of Texas Press.
- Matthews, K. A., C. D. Batson, J. Horn, and R. H. Rosenman. 1981. "Principles in his nature which interest him in the fortune of others ...": The heritability of

- empathic concern for others. Journal of Personality 49:237-47.
- McGuffin, P., R. Katz, S. Watkins, and J. Rutherford. 1996. A hospital-based twin register of the heritability of DSM-IV unipolar depression. *Archives of General Psychiatry* 53:129–36.
- Miles, T. P. 1997. Population-based, genetically informative sample for studies of physical frailty and aging: Black elderly twin study. *Human Biology* 69:107–20.
- Mossey, J. M., and E. Shapiro. 1982. Self-rated health: A predictor of mortality among the elderly. *American Journal of Public Health* 72:800–808.
- Moum, T. 1992. Self-assessed health among Norwegian adults. *Social Science and Medicine* 35:935–47.
- Neale, M. C. 1991. *Statistical modeling with Mx*. Richmond: Department of Psychiatry, Medical College of Virginia/Virginia Commonwealth University.
- Neale, M. C., and L. R. Cardone. 1992. *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Nora, J. J., R. H. Lortscher, R. D. Spangler, A. H. Nora, and W. J. Kimberling. 1980. Genetic-epidemiologic study of early-onset ischemic heart disease. *Circulation* 61:503–8.
- Page, W. F., M. M. Braun, A. W. Partin, N. Caporaso, and P. Walsh. 1997. Heredity and prostate cancer: A study of World War II veteran twins. *Prostate* 33:240–45.
- Partin, A. W., W. F. Page, B. R. Lee, M. G. Sanda, R. N. Miller, and P. C. Walsh. 1994. Concordance rates for benign prostatic disease among twins suggest hereditary influence. *Urology* 44:646–50.
- Plomin, R., and D. Daniels. 1987. Why are children in the same family so different from one another? *Behavioral and Brain Sciences* 10:1–16.
- Rossi, A. S. 2001. Domains and dimensions of social responsibility: A sociodemographic profile. In *Caring and doing for others: Social responsibility in the domains of family, work, and community,* ed. A. S. Rossi, 97–134. Chicago: University of Chicago Press.
- Rowe, D. C. 1983. A biometrical analysis of perceptions of family environment: A study of twin and singleton siblings. *Developmental Psychology* 17:203–8.
- Roy, M. A., M. C. Neale, and K. S. Kendler. 1995. The genetic epidemiology of self-esteem. *British Journal of Psychiatry* 166:813–20.
- Rushton, J. P., D. W. Fulker, M. C. Neale, R. A. Blizard, and H. J. Eysenck. 1984. Altruism and genetics. *Acta Geneticae Medicae et Gemellologiae* 33:265–71.
- Ryff, C. D. 1989. Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology* 57:1069–81.
- Ryff, C. D., and C. L. Keyes. 1995. The structure of psychological well-being revisited. Journal of Personality and Social Psychology 69:719–27.
- Scarr, S., and K. McCartney. 1983. How people make their own environments: A theory of genotype greater than environment effects. *Child Development* 54:424–35.
- Schlesselman, J. J. 1982. Case-control studies: Design, conduct, analysis. New York: Oxford University Press.
- Schmutte, P. S., and C. D. Ryff. 1997. Personality and well-being: Reexamining methods and meanings. *Journal of Personality and Social Psychology* 73:549–59.
- Schneider, N. R., W. R. Williams, and R. S. Chaganti. 1986. Genetic epidemiology of familial aggregation of cancer. *Advances in Cancer Research* 47:1–36.

- Schwarz, G. 1978. Estimating the dimensions of a model. Annals of Statistics 6:461-64.
- Selby, J. V., B. Newman, C. P. Quesenberry, Jr., R. R. Fabsitz, D. Carmelli, F. J. Meaney, and C. Slemenda. 1990. Genetic and behavioral influences on body fat distribution. *International Journal of Obesity* 14:593–602.
- Selby, J. V., B. Newman, J. Quiroga, J. C. Christian, M. A. Austin, and R. R. Fabsitz. 1991. Concordance for dyslipidemic hypertension in male twins. *Journal of American Medical Association* 265:2079–84.
- Silberg, J. L., A. C. Heath, R. C. Kessler, K. S. Kendler, M. C. Neale, and L. J. Eaves. 1990. Genetic and environmental effects on self-reported depressive symptoms in a general population twin sample. *Journal of Psychiatric Research* 24:197–212.
- Stunkard, A. J. 1991. Genetic contributions to human obesity. *Research Publications:* Association for Research in Nervous and Mental Disease 69:205–18.
- Stunkard, A. J., T. T. Foch, and Z. Hrubec. 1986. A twin study of human obesity. *Journal of American Medical Association* 256:51–54.
- Turula, M., J. Kaprio, A. Rissanen, and M. Koskenvuo. 1990. Body weight in the Finnish Twin Cohort. *Diabetes Research and Clinical Practice* 10:S33–S36.
- Viken, R. J., R. J. Rose, J. Kaprio, and M. Koskenvuo. 1994. A developmental genetic analysis of adult personality: Extraversion and neuroticism from 18 to 59 years of age. *Journal of Personality and Social Psychology* 66:722–30.
- Yashin, A. I., I. A. Iachine, K. Christensen, N. V. Holm, and J. W. Vaupel. 1998. The genetic component of discrete disability traits: An analysis using liability models with age-dependent thresholds. *Behavior Genetics* 28:207–14.