Social Support: A Genetic-Epidemiologic Analysis

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Objective: *Social support is a widely used construct in the fields of mental health, sociology, and medicine and has typically been conceptualized as an environmental factor that influences the risk for dysfunction and disease. In this study a longitudinal twin design was used to clarify the etiology of social support.* *Method:* *A 16-item social support inventory was administered at personal interview to a population-based sample of female twins twice, approximately 5 years apart. A twin measurement model—which permits an estimation of the etiologic role of genetic and environmental factors correcting for errors of measurement or short-term temporal fluctuations—was applied to these data.* *Results:* *Six factors, which were moderately stable over time, were found: relative problems, friend problems, relative support, confidants, friend support, and social integration. The best-fitting twin measurement models indicated that genetic factors were of substantial etiologic significance for all six social support scales. Heritabilities of the stable component of social support ranged from 43% to 75%. Familial-environmental factors contributed to twin resemblance only for relative problems and relative support. No evidence was found for significant biases in the twin method.* *Conclusions:* *Measures of social support are moderately stable over time. When short-term fluctuations are corrected for, heritable factors are of substantial etiologic importance for social support as measured at personal interview. Treating social support solely as an environmental measure is probably incorrect. Through genetically influenced traits such as temperament, individuals play a substantial role in creating their own social environments.*

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Social support has been a widely used concept in the field of mental health, as well as in medicine and the social sciences more broadly (1). The quality of social relationships predicts general health and mortality (2), psychiatric symptoms (3, 4), and the emotional adjustment to stress (3).

In these wide-ranging studies, social support has typically been conceptualized as an environmental variable—an individual's social support deriving from the caring and sustenance provided by the social environment. Four lines of evidence suggest that this unidirectional model—in which the social environment impinges on the individual but not vice versa—may be unrealistic.

First, levels of perceived social support are significantly correlated with personality, positively with extraversion and negatively with neuroticism (3, 5–7). Second, the quality of social support is moderately stable over time (6), so that social support can be conceptualized "as an individual difference variable as well as an environmental provision" (6). Third, positive social interactions emerge in part as a result of the active effort of individuals to develop and sustain reciprocally supportive relationships (8). Fourth, two prior twin studies (4, 9), both using questionnaire measures, suggested that genetic factors influence aspects of social support.

In this article I report on the use of a longitudinal twin design to further clarify the etiology of social support. Three methodologic features are noteworthy. First, social support was assessed at personal interview on two occasions. Second, multiple dimensions of social support were measured. Third, the "twin measurement model" used permits estimates of the etiologic importance of genetic and environmental factors *correcting* for the effect of measurement error. That is, this model

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TABLE 1. Factor Loadings for Six Factors Identified From Items in a Social Interaction Scale[a] for a Population-Based Sample of Female Twins (N=2,163)

Summary of Item	Factor Loading					
	Relative Problems	Friend Problems	Relative Support	Confidants	Friend Support	Social Integration
Contact with friends	–0.03	0.21	0.02	0.03	0.25	0.59[b]
Number of friends[c]	–0.10	0.11	–0.03	0.20	–0.08	0.57[b]
Friends care about you	0.01	–0.09	0.07	0.06	0.79[b]	0.12
Friends make too many demands	0.11	0.72[b]	–0.03	–0.05	–0.12	0.18
Friends criticize you	0.24	0.71[b]	0.01	0.02	0.02	–0.01
Friends express interest	0.04	–0.07	0.11	0.07	0.81[b]	0.01
Friends create tension	0.17	0.75[b]	0.02	0.01	–0.07	0.06
Contact with relatives	0.33	–0.27	0.64[b]	–0.05	–0.18	0.21
Relatives care about you	–0.30	0.08	0.78[b]	0.09	0.14	0.06
Relatives make too many demands	0.80[b]	0.17	–0.02	–0.01	–0.03	0.01
Relatives criticize you	0.79[b]	0.21	–0.13	–0.01	0.05	–0.05
Relatives express an interest	–0.24	0.07	0.78[b]	0.07	0.23	–0.05
Relatives create tension	0.78[b]	0.18	–0.17	0.03	0.04	–0.02
Meetings of clubs and organizations	0.07	–0.06	0.02	–0.04	0.04	0.73[b]
Have a confidant	0.02	–0.01	0.01	0.88[b]	0.05	–0.01
Number of confidants[c]	0.00	–0.01	0.10	0.86[b]	0.08	0.18

[a]Scale developed at the Institute for Social Research (13).
[b]Factor loading >0.55.
[c]Log transformed.

permits an examination of the role of genes and environment in the etiology of the stable, reliable component of social support.

METHOD

The data come from a study of genetic and environmental risk factors for common psychiatric disorders in Caucasian female same-sex twin pairs from the Virginia Twin Registry (10)—a population-based register formed from a systematic review of all birth certificates in the Commonwealth of Virginia. Twins were eligible to participate if they were born during 1934–1971 and both members had previously responded to a mailed questionnaire, to which the individual response rate was ~64%. In the first interview, 92% of the eligible individuals were assessed (N=2,163), 90% face to face, the rest by telephone. Zygosity was determined blindly by standard questions (11), photographs, and when necessary, DNA (10, 12).

We have completed two additional waves of telephone interviews with 2,001 (93%) and 1,898 (88%) of the original sample, respectively. The mean number of months between the first and third interviews was 61.3 (SD=5.1). In the third interview, we assessed both members of 854 pairs, 497 of which were monozygotic, 354 of which were dizygotic, and three of which had unknown zygosity. The mean age of the participating twins in the third wave of interviews was 34.6 years (SD=7.5), and the ages ranged from 22 to 59. In each case the interviewer was blind to information about the co-twin. Written informed consent was obtained before the face-to-face interviews, and oral assent was obtained before the telephone interviews.

Measure

We assessed the dimensions of social support by using a 16-item social interaction scale previously developed and used at the Institute for Social Research (13). Abbreviated versions of these items are seen in table 1. The full text for two of the items was as follows:

1. One of the things we'd like to know is how people spend their time. First, how often do you have contact with your friends—either see them, talk to them on the phone, or write letters? Would you say more than once a week, once a week, a few times a month, once a month, less than once a month, or never?

2. When you are in contact with these friends, how often do they make you feel that they care about you? Would you say often, sometimes, rarely, or never?

The same items were administered during the first and third waves of personal interviews. The working definitions of "friend" and "confidant" were, respectively, "someone with whom you have regular contact and some emotional connection—but not counting relatives" and "someone with whom you have a close relationship and can share your most private feelings." Frequency of contact with relatives was assessed for relatives the respondent was *not* living with, while the quality of the respondent's relationship with relatives was assessed for *all* relatives in regular contact with the respondent. The definition of meetings of clubs or other organizations included "church-related activities which were not worship-oriented, as well as sports teams, bridge or poker groups, community organizations, PTA, sewing circles, bowling teams, etc."

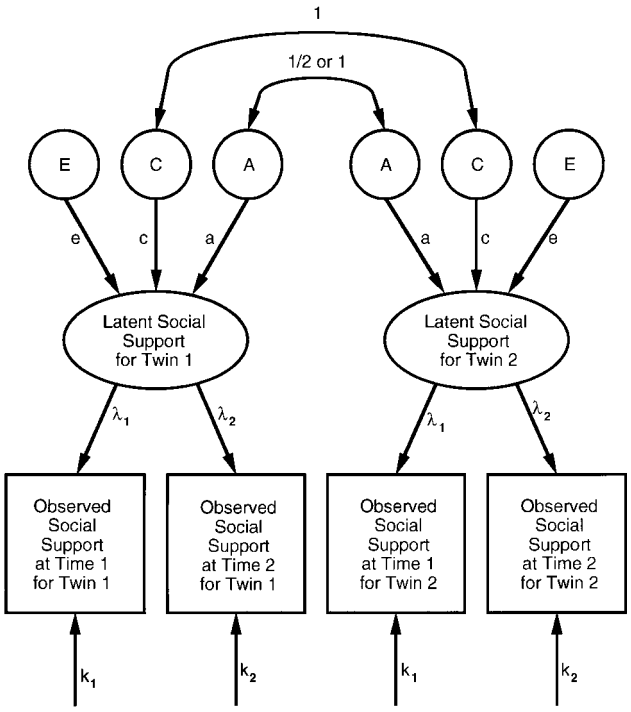
Factor Analysis

The distributions of the variables "number of friends" and "number of confidants" had a strong rightward skew. Before the analyses, therefore, for these variables only, we took the natural logarithm of n+1, where n equaled the number of friends or confidants. For the purposes of data reduction before twin analysis, the 16 items were submitted to a factor analysis followed by VARIMAX rotation using the PROCFACTOR procedure in SAS (14). The number of factors was determined by traditional eigen value criteria. Factor-derived scales were developed from this factor analysis, and each item was assigned to the factor-derived scale on which it loaded most strongly.

Twin Analyses

The twin measurement model used in this study has been described previously (15). Briefly, as in other standard biometrical twin models (16, 17), it is assumed that the variation in the assessed dimensions of social support can be divided into three classes: 1) additive genetic factors (A), which contribute twice as much to the correlation in monozygotic twins as to the correlation in dizygotic twins (because monozygotic twins share all their genes identical by descent, while dizygotic twins share on average half their genes); 2) family or "common" environmental factors (familial factors that make twins similar in their levels of social support) (C), which contribute equally to the correlations in monozygotic and dizygotic twins; and 3) individual-specific environmental factors (E), which traditionally reflect environ-

FIGURE 1. Twin Model for Heritability of Social Support Including Error of Measurement[a]



[a]This model assumes that individual twins have a *true* latent (or stable) level of social support that is imperfectly indexed by two assessments, at time 1 and time 2. The paths λ_1 and λ_2 represent the degree to which these two assessments, respectively, reflect the true level of social support. The other paths to the observed levels of social support at times 1 and 2 (k_1 and k_2) represent error and/or short-term temporal fluctuations in the measure. The model is constrained so that for each twin assessment $\lambda^2+k^2=1.0$.

The latent level of social support is modeled as in a standard biometrical twin design (16), with the sources of variance in liability divided between additive genetic factors (A), common environmental factors (C), and individual-specific environmental factors (E). By definition, the common environmental components are perfectly correlated in twin pairs, while the individual-specific environments are uncorrelated. The correlation of additive genetic factors is unity for monozygotic twins and 0.5 for dizygotic twins. Lower-case letters (a, c, and e) are used to label the paths from these factors. The individual paths represent standardized regression coefficients, so that the proportion of variance in the dependent variables accounted for by the independent variable is equal to the square of the connecting path. Heritability, for example, equals a^2. Observed variables are depicted in boxes, and latent variables are shown in circles and ellipses.

mental experiences not shared by both members of a twin pair and therefore contribute to *differences* between them in their reported social support.

Several of the social support dimensions had relatively nonnormal distributions, in part because they contained so few individual items. Therefore, the twin analyses assumed a liability-threshold model. That is, individual scores on the social support dimensions were the result of thresholds imposed on a latent, normally distributed liability. The fit of this model was tested by a chi-square goodness-of-fit test.

The twin measurement model uses the twins' scores on the social support measures at both time points (figure 1). The model assumes that each twin has a true but unobserved (or latent) level of social support. We measured social support twice in these twins, but each measurement is fallible, in that it partially reflects the true level of social support and partly reflects error. The paths λ_1 and λ_2 represent the degree to which the assessments of social support obtained at the first and second time points, respectively, reflect this true liability. The higher the value of λ, the more accurately any one measure of social support reflects an individual's true level of social support. The other paths to the assessments of social support at the two time points (k_1 and k_2, respectively) represent error in the individual assessments. By definition, $\lambda^2+k^2=1.0$. These models assume that the errors of measurement are uncorrelated within time.

The true, or latent, social support is then modeled in a standard twin design, as already outlined, with the sources of variance in liability divided between additive genetic, common environmental, and individual-specific environmental factors.

Three differences between this measurement model and the standard twin model, based on a single time of assessment, are noteworthy. First, this model provides *separate* estimates for error of measurement (k) and true individual-specific environment (e) that are conflated in the standard twin model. Second, it provides a direct estimate of the reliability of the assessment of social support (λ). Third, while a standard twin model estimates the heritability of the *observed* phenotype (including error), the measurement model estimates the heritability of the *latent* phenotype, correcting for the effects of error. The latter will always be greater than the former.

These analyses assume that 1) levels of social support are similar in monozygotic and dizygotic twins and 2) exposure to environmental factors that influence levels of social support are similarly correlated in monozygotic and dizygotic twins. These two assumptions were tested by examining, by means of regression analysis, whether 1) zygosity could predict levels of social support and 2) when zygosity is controlled for, measures of childhood or adult environmental similarity (18) could predict the difference in social support between members of a twin pair.

Model fitting was performed by using the program Mx (19) by the method of asymptotic weighted least squares (20). The best-fit model was selected by using Akaike's information criterion (21).

RESULTS

Factor Analysis

Six factors were identified, the factor loadings for which are seen in table 1. The first two factors, termed "relative problems" and "friend problems," reflected the degree to which the twin reported, respectively, relatives and friends as "making too many demands," "criticizing," and "creating tension or arguments." The third factor, "relative support," had the highest loadings on the items for the frequency of contact with relatives and the degree to which relatives made the twins feel "they care about you" and expressed "interest in how you are doing." The fourth, or "confidants," factor was made up of two items assessing the presence of and number of confidants. The fifth factor, termed "friend support," had the highest loadings on the two items assessing positive emotional relations with friends ("caring about you" and "expressing interest"). The final factor, which we termed "social integration," reflected the number of friends, the frequency of contact with them, and the frequency of club attendance.

As each item unambiguously loaded highly on a single factor, it was easy to construct six factor-derived scales, as seen in table 1. These six factors reflected the positive and negative emotional quality of social relations (relative and friend support and relative and friend problems, respectively) and the quantity of so-

cial contacts both at an intimate level and at a more general level (confidants and social integration factors, respectively).

A PROMAX oblique factor rotation was performed. The factor structure looked very similar; the mean of the absolute values of the 15 interfactor correlations was 0.09 (SD=0.05), and the range was –0.15 to 0.17.

TABLE 2. Model Fitting and Parameter Estimates for Six Social Support Factors Found for a Population-Based Sample of Female Twins (N=2,163)

Social Support Factor	Fit of Model[a] (χ^2) ACE (df=9)	AE (df=10)	CE (df=10)	Parameter Estimates for Best-Fitting Model[b] a^2	c^2	e^2	λ^2	k^2
Relative problems	16.5[c]	19.8	30.3	0.49	0.20	0.30	0.53	0.47
Friend problems	14.9	14.9[c]	31.5	0.59	—	0.41	0.38	0.62
Relative support	5.8[c]	9.1	11.3	0.44	0.28	0.28	0.42	0.58
Confidants	16.5	16.5[c]	32.0	0.66	—	0.34	0.42	0.58
Friend support	5.5	5.5[c]	8.4	0.43	—	0.57	0.43	0.57
Social integration[d]	7.2	7.4[c]	27.1	0.75	—	0.25	—[d]	—[d]

[a]Models tested influence of combinations of additive genetic factors (A), common environmental factors (C), and individual-specific environmental factors (E).
[b]Paths a, c, and e from factors A, C, and E are standardized regression coefficients; λ^2 represents the reliability of the assessment of social support; and k^2 represents error and/or short-term temporal fluctuations in the measure.
[c]Best-fitting model by Akaike's information criterion (21).
[d]Best-fitting model required separate estimates of λ^2 and k^2 for times 1 and 2—time 1: λ^2=0.58, k^2=0.42; time 2: λ^2=0.36, k^2=0.64; df=8, df=9, and df=9, respectively.

Stability of Social Support

The polychoric correlations of the dimensions of social support between waves were moderate and positive: relative problems, 0.51; friend problems, 0.39; relative support, 0.42; confidants, 0.42; friend support, 0.44; and social integration, 0.45.

Testing Assumptions

The representativeness of the sample was examined in two ways. First, since levels of social support correlate in twin pairs, if low social support predicts noncooperation, then twins with nonresponding co-twins should have lower levels of social support than twins with responding co-twins. This was tested for the six factors at the two time points. Of these 12 tests, one finding was significant at the 5% level, and this finding was not replicated across occasions of measurement. The second test was to determine whether social support at the first assessment predicted subsequent cooperation. None of the six factor scores assessed at time 1 significantly predicted cooperation at time 2.

The impact of zygosity on the six social support dimensions assessed at the two time points was examined in 12 tests. One of these differences was significant at the 5% level but was not stable across times of measurement. The next issue examined was whether an excess similarity for these measures of social support in monozygotic versus dizygotic twins could be due to environmental factors. Specifically, with zygosity controlled for it was determined, separately at each wave, whether twin resemblance for these factors could be accounted for by the similarity of the environmental experiences of the twins in childhood or frequency of contact in adulthood. From the 24 tests, five findings were significant. However, none of the five differences was replicated across times of measurement. The closest to a replicated finding was found for increased frequency of current twin contact predicting greater twin resemblance for social integration. At time 1 this relationship was statistically significant (F=4.83, df=1, 819, p=0.03), and a trend in the same direction was observed at time 2 (F=3.20, df=1, 817, p=0.07).

Finally, whether the multiple-threshold model provided a good explanation for the distribution of social support scores was determined by means of 24 individual chi-square goodness-of-fit tests—six dimensions at two time points separately in monozygotic and dizygotic pairs. From these tests, two findings were significant at the 5% level—but these moderately poor fits replicated neither across times nor across zygosities.

Twin Measurement Model

As previously outlined, the twin measurement model differs from standard twin models. The standard twin model decomposes the variance in liability—into its genetic and environmental components—for an *observed* variable. The measurement model also decomposes variance in liability but now for a *latent* variable that is itself indexed imperfectly by two occasions of measurement.

As seen in table 2, the results of fitting the twin measurement model to the data in this study suggest that the six factors of social support can be divided into two groups. For two of the factors, relative problems and relative support, twin resemblance for the latent factors was due to both genetic and familial-environmental factors. For both these factors, the estimates of a^2 and c^2 were ~45% and ~25%, respectively.

For the four remaining factors—friend problems, friend support, confidants, and social integration—twin resemblance was due entirely to genetic factors. Familial-environmental factors made no apparent contribution to twin resemblance for these factors. Estimates of a^2 for these latent variables were substantial, ranging from 0.43 for friend support to 0.75 for social integration.

For five of the six factors, the best-fitting model assumed the equivalence of the two waves of interviews. However, for social integration the best-fitting model indicated that the face-to-face interview was a significantly better index of the latent variable than was the telephone interview.

Estimates of λ—an approximate measure of reliabil-

ity—were within a narrow range, from 0.61 for friend problems to 0.76 for the wave 1 assessment of social integration.

DISCUSSION

Social support was assessed on two occasions by personal interview in a population-based sample of female twins. It was found that 1) social support is a multidimensional concept, 2) social support is moderately stable over time, and 3) the latent or stable component of all dimensions of social support is substantially heritable. Consistent with findings in earlier studies (4–6, 8, 9), these results suggest that treating social support solely as an environmental measure is unsustainable. Indeed, these results suggest that between 40% and 80% of the temporally stable or reliable variance in dimensions of social support is due to genetic differences between individuals.

How might such genetic differences evolve? Social integration was likely a vital function in human evolution as our ancestors evolved in small hunter-gatherer bands (22). On the one hand, selection was likely for "uncooperative" traits such as aggressiveness that led to greater resource access. On the other hand, there was also selection for an easy, cooperative temperament that worked well in group activities such as hunting or child rearing. This kind of stabilizing selection with maximum fitness reflecting a mixture of cooperative and uncooperative traits is the kind predicted to produce substantial genetic variation (23–25).

These speculations suggest that broadly defined "personality" is a likely mediator between genetic factors and reported social support. In accord with prior observations (3, 5, 6), in this sample there were also substantial correlations between dimensions of social support and the personality dimensions of neuroticism and extraversion, as assessed by Eysenck's personality questionnaire (26). Increasing neuroticism—perhaps indicative of a "difficult temperament"—strongly predicted higher levels of relative problems and friend problems, lower levels of relative and friend support, fewer confidants, and less social integration. Increasing extraversion, perhaps indexing an easy, cooperative temperament, predicted the reverse—higher levels of interpersonal support and lower levels of interpersonal difficulties.

Familial-environmental factors played a modest etiologic role in two social support factors: relative problems and relative support. These findings could have resulted from the continued effects into adulthood of a shared rearing environment that shaped the pattern of interpersonal interactions. Alternatively, these findings could result from contemporary effects—as adult twins still share some of their relatives (e.g., their parents and siblings but not, if married, their in-laws). The latter is a more plausible interpretation because it explains why familial-environmental effects are seen only for the factors involving relatives. If the shared rearing environment influenced patterns of social interaction, it would be difficult to explain why the enduring effect of these patterns would be manifest only in relationships with relatives and not with friends.

True Versus Perceived Social Support

An important methodologic issue in social support research is the distinction between objective versus subjective measures (27). That is, have we measured how the world truly treats the twins or only how the twins see themselves as being treated?

The study was based solely on respondent reports in structured interviews, so all of the data are subjective. Therefore, it cannot be determined definitively whether there is evidence for genetic factors that actually influence how individuals interact with their social environment or for genetic factors that only influence the ways in which the twin perceives the social environment.

However, a useful distinction can be made between four of the scales—on which the twins reported the emotional nature of their social relations (e.g., relative problems and support and friend problems and support)—and the remaining two scales (confidants and social integration)—on which the twins enumerated the number and frequency of their social contacts. If the observed heritability of social support was truly a reflection of a "plaintive set" (e.g., "nobody likes me, everybody hates me"), this would predict *higher* heritability estimates for the four scales reflecting the emotional nature of social relations. However, the *exact opposite* was observed. The highest heritability estimates were for the two scales that measure more objective phenomena: confidants and social integration. These results provide some evidence to suggest that, in addition to measures heavily influenced by the twins' perception of their social world, some features of their objective social reality were also successfully assessed.

Implications

Traditional models for the impact of genetic factors on psychiatric disorders have concentrated on physiological aspects of gene expression. That is, our concept of gene action has been largely "within the skin." These findings suggest that this is an overly simplistic model of gene action for complex human traits such as social support. Consistent with the predictions of evolutionary theory, it is likely that gene action for an animal as quintessentially social as homo sapiens can often reach "beyond the skin" to affect the interpersonal environment as part of an "extended" phenotype (28). In support of this is accumulating evidence that a range of putative environmental measures used in the behavioral sciences are influenced by genetic factors (29, 30). Social support has been shown to predict a wide variety of medical and psychiatric outcomes, including coronary artery disease (31, 32), pregnancy complications (33), depression (34), and alcoholism (35), although the mechanisms by which social support affects these diseases may not be straightforward (36).

We should continue to strive to understand gene expression in psychiatric and medical illness at the physiological level. However, a complete understanding of the role of genes in these disorders will require clarifying how they influence risk by causing individuals to place themselves in high-risk environments, a process Eaves and I have previously termed "genetic control of exposure to the environment" (37).

Limitations

These results should be interpreted in the context of four potentially important methodologic limitations. First, all of the twins studied were female and it would be inappropriate to assume that these findings would extrapolate to a male sample.

Second, this study did not examine all possible dimensions of social support (1). For example, neither instrumental social support (38) (e.g., accessibility of practical help from the social environment, such as lending money or caring for children in a crisis) nor the number or quality of more diffuse social relationships (e.g., acquaintances) was assessed.

Third, it cannot be ruled out that biases in the method inflated heritability estimates, although the observed differences in monozygotic and dizygotic twins on the measures of social support (one of 12 significant tests) and the representativeness of the sample with respect to levels of social support (one of 18 significant tests) did not differ from chance expectation (39). However, tests for violation of the equal-environment assumption did obtain more significant findings (five of 24 results) than would be expected by chance (39). Yet none of these findings replicated across times of measurement, with one possible exception—increased frequency of contact predicting greater twin resemblance for level of social integration. To assess the possible magnitude of the bias introduced in heritability estimates for this dimension of social support, we randomly chose a subset of twin pairs in which monozygotic and dizygotic twins were matched for their frequency of contact. Refitting the best-fitting model resulted in a decline of the heritability estimate from 0.75 to 0.70. The substantial estimates for the heritability of the reliable or latent component of social support obtained in this twin study are unlikely to result, to any notable degree, from biases in the twin method.

Finally, the model assumed that the changes in social support between the two occasions of measurement were random and hence uncorrelated in twins. To test this assumption, a path correlating the error terms for members of a twin pair at the second time of measurement was added. The magnitude of this new path was low in all six dimensions of social support (0.03 to 0.07) and resulted in an improvement in fit for only one: friend problems. For this dimension, inclusion of this path resulted in a modest decrease in the heritability estimate, from 0.59 to 0.48. These results support the validity of the assumption that changes in social support across waves were largely random.

REFERENCES

1. Sarason BR, Sarason IG, Pierce GR (eds): Social Support: An Interactional View. New York, John Wiley & Sons, 1990
2. House JS, Landis KR, Umberson D: Social relationships and health. Science 1988; 241:540–544
3. Monroe SM, Steiner SC: Social support and psychopathology: interrelations with preexisting disorder, stress, and personality. J Abnorm Psychol 1986; 95:29–39
4. Kessler RC, Kendler KS, Heath AC, Neale MC, Eaves LJ: Social support, depressed mood, and adjustment to stress: a genetic epidemiologic investigation. J Pers Soc Psychol 1992; 62:257–272
5. Henderson S, Byrne DG, Duncan-Jones P: Neurosis and the Social Environment. New York, Academic Press, 1981
6. Sarason IG, Sarason BR, Shearin EN: Social support as an individual difference variable: its stability, origins, and relational aspects. J Pers Soc Psychol 1986; 50:845–855
7. Windle M: Temperament and social support in adolescence: interrelations with depressive symptoms and delinquent behaviors. J Youth and Adolescence 1992; 21:1–21
8. Antonucci TC, Jackson JS: The role of reciprocity in social support, in Social Support: An Interactional View. Edited by Sarason BR, Sarason IG, Pierce GR. New York, John Wiley & Sons, 1990, pp 173–198
9. Bergeman CS, Plomin R, Pedersen NL, McClearn GE, Nesselroade JR: Genetic and environmental influences on social support: the Swedish Adoption Twin Study of Aging (SATSA). J Gerontol 1990; 45:101–106
10. Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ: A population-based twin study of major depression in women: the impact of varying definitions of illness. Arch Gen Psychiatry 1992; 49:257–266
11. Eaves LJ, Eysenck HJ, Martin NG, Jardine R, Heath AC, Feingold L, Young PA, Kendler KS: Genes, Culture and Personality: An Empirical Approach. London, Oxford University Press, 1989
12. Spence JE, Corey LA, Nance WE, Marazita ML, Kendler KS, Schieken RM: Molecular analysis of twin zygosity using VNTR DNA probes (abstract). Am J Hum Genet 1988; 43:A159
13. Schuster TL, Kessler RC, Aseltine RH Jr: Supportive interactions, negative interactions, and depressed mood. Am J Community Psychol 1990; 18:423–438
14. SAS/STAT User's Guide, version 6, 4th ed, vols 1, 2. Cary, NC, SAS Institute, 1990
15. Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ: The lifetime history of major depression in women: reliability of diagnosis and heritability. Arch Gen Psychiatry 1993; 50:863–870
16. Kendler KS: Twin studies of psychiatric illness: current status and future directions. Arch Gen Psychiatry 1993; 50:905–915
17. Neale MC, Cardon LR: Methodology for Genetic Studies of Twins and Families. Dordrecht, the Netherlands, Kluwer Academic, 1992
18. Loehlin JC, Nichols RC: Heredity, Environment and Personality: A Study of 850 Sets of Twins. Austin, University of Texas Press, 1976
19. Neale MC: Mx: Statistical Modeling, 2nd ed. Richmond, Virginia Commonwealth University, Department of Psychiatry, 1994
20. Browne MW: Asymptotically distribution-free methods for the analysis of covariance structures. Br J Math Stat Psychol 1984; 37:62–83
21. Akaike H: Factor analysis and AIC. Psychometrika 1987; 52: 317–332
22. Klein RG: The Human Career: Human Biological and Cultural Origins. Chicago, University of Chicago Press, 1989
23. Mather K: Polygenic inheritance and natural selection, in Biological Reviews, vol 18. Edited by Fox HM. London, Cambridge University Press, 1943, pp 32–64
24. Lande R: Natural selection and random genetic drift in phenotypic evolution. Evolution 1976; 30:314–334
25. Buss DM: Evolutionary personality psychology. Annu Rev Psychol 1991; 42:459–491

26. Nystrom S, Lindegard B: Predisposition for mental syndromes: a study comparing predisposition for depression, neurasthenia and anxiety state. Acta Psychiatr Scand 1975; 51:69–76
27. Henderson S: The significance of social relationships in the etiology of neurosis, in The Place of Attachment in Human Behavior. Edited by Parkes CM, Stevenson-Hinde J. New York, Basic Books, 1982, pp 205–231
28. Dawkins R: The Extended Phenotype: The Gene as the Unit of Selection. Oxford, England, Oxford University Press, 1982
29. Plomin R, Bergeman CS: The nature of nurture: genetic influences on "environmental" measures. Behav Brain Sci 1991; 14: 373–385
30. Plomin R: Genetics and Experience: The Interplay Between Nature and Nurture. Thousand Oaks, Calif, Sage Publications, 1994
31. Haynes SG, Feinleib M, Kannel WB: The relationship of psychosocial factors to coronary heart disease in the Framingham study, III: eight-year incidence of coronary heart disease. Am J Epidemiol 1980; 111:37–58
32. Medalie JH, Goldbourt U: Angina pectoris among 10,000 men, II: psychosocial and other risk factors as evidenced by a multivariate analysis of a five year incidence study. Am J Med 1976; 60:910–921
33. Norbeck JS, Tilden V: Life stress, social support and emotional equilibrium in complications of pregnancy: a prospective multivariate study. J Health Soc Behav 1981; 60:30–46
34. Brown GW, Harris T: Social Origins of Depression: A Study of Psychiatric Disorder in Women. New York, Free Press, 1978
35. Ohannessian CM, Hesselbrock VM: The influence of perceived social support on the relationship between family history of alcoholism and drinking behaviors. Addiction 1993; 88:1651–1658
36. Kessler RC, Kendler KS, Heath AC, Neale MC, Eaves LJ: Perceived support and adjustment to stress in a general population sample of female twins. Psychol Med 1994; 24:317–334
37. Kendler KS, Eaves LJ: Models for the joint effect of genotype and environment on liability to psychiatric illness. Am J Psychiatry 1986; 143:279–289
38. Cohen S, Wills TA: Stress, social support, and the buffering hypothesis. Psychol Bull 1985; 98:310–357
39. Feild HS, Armenakis AA: On use of multiple tests of significance in psychological research. Psychol Rep 1974; 35:427–431