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## Genetic Influences on Midlife Functioning

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Regardless of the definition of “midlife,” most of the research in this area is typically normative. Behavioral genetic designs, on the other hand, focus on individual differences, on partitioning genetic and environmental sources. Our goal in this chapter is to provide a review of behavioral genetic studies of midlife, focusing, when possible, on longitudinal findings. Surprisingly little research of this nature has been explicitly devoted to the adult years (between 30 and 65 years of age), although several twin and family studies are either maturing into midlife, have included individuals in midlife because they were parents of adolescents, or have included participants under the age of 65 in studies of aging.

### Behavioral Genetic Designs and What They Can Tell Us

Before beginning our review of the research on genetic and environmental influences in midlife, we felt that we should provide some background on

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behavioral genetics in general. Specifically, we wanted to define some key terms, discuss basic aspects of the methodology involved, and present several different study designs.

### Key Terms

Behavioral genetic studies are designed to partition variance into genetic and environmental components. Estimates of genetic influences are referred to as *heritability* estimates and are the proportion of total variation that can be attributed to genetic differences among individuals in the population. It is important to remember that heritability, as well as estimates of environmental influences, are statistics based on variability in a population, not on mean or group differences.

Environmental variance can be partitioned into two types: shared and nonshared. *Shared environmental influences* are those nongenetic factors that make family members similar, and *nonshared environmental influences* are those nongenetic factors that make family members different.

Genetic and environmental influences may not always have direct effects; rather, there might be interplay of some sort between genetic and environmental factors. This interplay might take the form of a *genotype-environment (GE) correlation*, simply a correlation between a genotype and the environment to which the genotype is exposed. There are three ways that a genotype and an environment can be correlated: passively, evocatively, and actively (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). Passive GE correlation refers to the transmission of both genes and environment from parents to their children. For example, parents who have a very loving and warm marriage may pass on to their children those genetically influenced characteristics that have both helped to create their good marriage and also provided an environment for their child that teaches the skills needed for a warm and loving marriage. Evocative GE correlation refers to environments that are elicited by an individual's genotype. To again use the example of marriage, an adult who is warm and loving is likely to elicit warm, positive responses from their spouse, resulting in a satisfying marriage. Finally, active GE correlation occurs when individuals actively select environments that are correlated with their genotype. A caring adult is more likely to seek out other people who are also warm, rather than befriend those who tend toward more negative behaviors.

Genes and environments might also have moderating effects on each other. Genotype-environment (GxE) interactions refer to the sensitivity of one's genotype to the environment, such that genetic propensities might be modified by environmental circumstances. Searching for GxE interactions has been more successful in nonhuman primates than in humans because researchers can breed strains of animals uniform for the genes that influence particular behaviors and then randomly assign these strains to different environments. Regardless, there has been some recent evidence in human populations for GxE interaction (Caspi et al., 2003; Heath, Eaves, & Martin, 1998; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003).

## Methodology and Designs

Behavioral genetic studies are based on quantitative genetic theory, which means that the model fitting that is done is based on expectations about familial similarity. For example, monozygotic (MZ) twins share all of their segregating genes, whereas dizygotic (DZ) twins share, on average, 50% of their genetic makeup, just as full siblings do. Half siblings share 25% of their genetic makeup. Unrelated siblings (either adopted siblings, or unrelated step siblings) share none of their genetic makeup, but they are excellent indicators of the shared environment because their similarity can only be explained by experiences that they have shared. By including these expectations in the model fitting, we can arrive at estimates of genetic and environmental influences for a given population.

There are a number of different study designs that behavioral geneticists use. One is the family design. Here a proband is selected, usually matched with a nonaffected control. Researchers then examine the incidence of a disease (or other phenomenon) in the proband's and control's relatives. Finding a higher incidence in relatives of the proband than in relatives of the nonaffected control suggests that the disease is familial in origin. By familial, we mean that genetic and/or shared environmental factors are influencing the disease. This design does not allow us to distinguish between the two types of factors.

Twin and sibling designs do allow us to distinguish between genetic, shared environmental, and nonshared environmental influences. If genetic factors were influencing a particular trait, we would expect correlations between each member of a sibling pair to follow this pattern:

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MZ > DZ = full sibling > half sibling > unrelated sibling. If shared environmental influences played a role, we would expect correlations between each sibling of a pair to be of the same magnitude across sibling types. Nonshared environmental influences are indicated by an MZ sibling correlation that is not equal to one because any differences between MZ twins can only be due to different environmental influences as they share the same genotype.

Finally, adoption studies provide a unique way of disentangling genetic and environmental effects by using two different “types” of parents, so to speak. The biological parents of the adopted-away child serve as a marker of genetic influence, while the adoptive parents serve as a marker of environmental influences. Any correlation between the adopted child and his biological parents for a given trait is assumed to be due to genetic influences since the biological parent has had little if any contact with the child. Conversely, any association between the adoptive parent and the adopted child suggests an environmental contribution to the parent-child resemblance. For example, if antisocial behavior were largely heritable, we would expect to see similarities between birth parents and adopted children, but no similarity between adopting parents and adopted children.

### What Behavioral Genetic Studies Can Tell Us

Behavior genetic studies allow us access to very crucial information about development over the life course that other types of studies are not able to provide. Namely, behavioral genetic studies are able to estimate genetic and environmental influences rather than assume their presence. Using these studies, we can get a better handle on the etiology of a wide range of phenomena, as well as better understand why different traits, disorders, and other constructs are interrelated. We have been able to clarify the links between interpersonal factors and mental and physical health. Genetically sensitive designs also allow us to identify influences on developmental processes. Not only have we been able to highlight the role of genes in individual development, but we have also been able to elucidate the role of the environment by searching for sources of shared and nonshared environmental influences. Genetically sensitive studies are necessary to gain the most complete understanding of development.

## Behavioral Genetic Studies of Midlife

To our knowledge, the Twin Offspring Study in Sweden (TOSS) is the only behavioral genetic study specifically designed to capture information about adjustment in midlife. In this study, female and male twin pairs who have adolescent children are the heart of the study design. At the present time, the study is not longitudinal. The numerous studies based on the Virginia Twin Registry and the "Virginia 30,000," based at the Virginia Institute of Psychiatric and Behavioral Genetics, have provided a wealth of information about psychiatric phenotypes in twins who were born between 1915 and 1971. Only some studies from these cohorts are longitudinal. Other twin registries, such as those in the Netherlands and Australia, have cohorts of twins, some of whom are currently in midlife, although most efforts for the former have focused on development of children and adolescents (Boomsma et al., 2002; Croft, Read, de Klerk, Hansen, & Kurunczuk, 2002). The Scandinavian twin registries have the largest cohorts of twins in midlife at the time of data collection (Bergem, 2002; Kaprio & Koskenvuo, 2002; Pedersen, Lichtenstein, & Svedberg, 2002; Skytthe, Kyvik, Holm, Vaupel, & Christensen, 2002), but most of the analyses from these cohorts are cross-sectional and primarily on medical end points. Two studies of aging, the Minnesota Twin Study of Adult Development and Aging (MTSADA; Finkel & McGue, 1993) and the Swedish Adoption/Twin Study of Aging (SATSA; Pedersen et al., 1991; see also Finkel & Pedersen, 2004) have sufficient numbers of pairs in midlife to provide comparisons of younger and older cohorts within the studies. All told, these studies with genetically informative populations have covered a broad array of behavioral and biomedical phenotypes and numerous environmental exposures. Among the many domains of relevance were social aspects (including education and occupation, environmental impingement by stress and life events), interpersonal relationships (parenting, marriage, social support), cognition, personality and adjustment, psychopathology and substance abuse, and health. In the following, we will focus on a limited number of traits that we believe are highly pertinent to understanding individual differences in midlife: interpersonal relationships, social aspects, self-rated health, and cognitive abilities. When possible, we will highlight longitudinal findings and multivariate analyses that go beyond the relatively simplistic characterization of heritabilities.

## Interpersonal Relationships and Adjustment

Interpersonal relationships are of great importance in midlife. Marital relationships and parent-child relationships can become a primary focus for many people during this time. Support from friends and family is also an important factor in coping with life changes, such as aging of parents and one's own aging. This section describes the behavioral genetic research that has considered interpersonal relationships in midlife. Most studies included in this section have used samples not specifically geared toward studying midlife, but rather have used samples that happen to include subjects who are in that stage of development. The exception might be the Twin Moms study (Reiss, Cederblad, et al., 2001; Reiss, Pedersen, et al., 2001), which by default focused on parents in midlife because selection criteria required their children be between the ages of 10 and 18 years. We will first discuss behavioral genetic studies of parenting; then, we will move on to social support and then marriage. We will then discuss studies that have tried to account for genetic influences on interpersonal relationships, ending with studies that have examined underlying genetic and environmental influences on links between interpersonal relationships and outcomes.

### Parenting

To date, the field of parenting research has probably been the area most affected by behavioral genetic inquiry. Traditional psychosocial research has long posited that parenting styles directly affect child outcome (e.g., Baumrind, 1973). Genetically informed studies do not necessarily support those claims. Both child-based (genetic information comes from the children) and parent-based (genetic information comes from the parents) studies support the notion that children influence how parents parent. Rowe was the first to examine the genetic and environmental influences on parenting using samples of adolescents (Rowe, 1981, 1983). He found that the adolescents' genetic influences contributed to their reports of their parents' warmth, and shared environmental influences contributed to parental control, with nonshared environmental influences contributing to about half of the variance of each. These findings have been replicated in several other studies (Braungart, 1994; Elkins, McGue, & Iacono, 1997; Goodman & Stevenson, 1989; Plomin, McClearn, Pedersen, Nesselroade, & Bergeman, 1989).

These findings come from child-based designs, so they only tell us about the influences of the child's genes. What about the parents' genes? Findings from the Twins Moms study—a sample of Swedish female twins, their spouses, and their adolescent child—tell us that the parents' own genetic factors also contribute to their parenting, for all aspects except for attempted and actual control (Neiderhiser et al., 2004).

Through what processes do these genetic factors influence parenting? There are several possibilities, all of which fall under the rubric of GE correlations. GE correlations are correlations between genetic factors of an individual and the environment. There are three types: passive, active, and evocative. Passive GE correlations refer to the fact that each parent provides 50% of their child's genetic makeup. So parents who exhibit warm parenting styles pass along the genes that influence their warmth, resulting in a child who also tends to be warm and caring, thereby contributing to the already pleasant family environment. In a case like this, it is difficult to untangle how much the children are influencing their parents' parenting or are reflecting shared genes. However, genetic characteristics of the child might influence their tendency to be caring and, as a result, lead them to seek out positive environments. This is called an active GE correlation. Finally, genetic characteristics of the child might elicit warm parenting; this is referred to as an evocative GE correlation. Here, the child influences parenting by evoking particular reactions from their parents. Much could be said on this topic, but space considerations limit us to a brief summary of findings from a study that attempted to tease apart these different processes. A comparison of the child-based Nonshared Environment and Adolescent Development study (Reiss et al., 1994; Reiss, Neiderhiser, Hetherington, & Plomin, 2000) and the parent-based Twin Moms study suggested that passive GE correlations may be important for parental negativity and positivity but not for parental control (Neiderhiser et al., 2004). The implications of these findings contradict traditional socialization research suggesting that parents have a direct influence on their child's outcome.

## Remembered Parenting

Several studies have addressed genetic and environmental influences on how adults remember their childhood family environment and parenting. MZ twins report their rearing environment as being more similar than the rearing environments of DZ twins, suggesting genetic influences on

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remembered parenting (Hur & Bouchard, 1995). A study using the Twin Moms sample found genetic influences on all subscales of remembered parenting, as well as shared environmental and nonshared environmental influences (Lichtenstein et al., 2003). Parental warmth was most strongly influenced by genetic influences, which could be accounted for by personality characteristics. Shared environmental influences were explained as an increasing importance of family-level experiences over time.

### Social Support

High-quality social support is robustly associated with improved health and well-being, whereas a lack of social support is linked with poorer health (Ganster & Victor, 1988; Kessler, Kendler, Heath, Neale, & Eaves, 1992; Wade & Kendler, 2000). A number of genetically sensitive studies have begun to examine genetic and environmental factors involved in social support.

The first study to examine genetic and environmental influences on social support came from SATSA. This sample included men and women with an average age of 65.6, 62% of whom were female. Because this sample includes twins separated at birth and reared apart, not only could estimates of genetic and nonshared environmental effects be computed, but also shared environmental variance could be separated into shared rearing environment and correlated environment (similarity of twins beyond that accounted for by genetic influences or shared rearing environments). Findings differed for availability and adequacy of support. The availability of social support was influenced entirely by environmental factors: Primarily nonshared environments, but also shared rearing environments and correlated environments, played a substantial role. Adequacy of support was influenced by genetic and, predominantly, nonshared environmental influences.

A series of studies using a sample of twins drawn from the Virginia Twin Registry (now the Mid-Atlantic Twin Registry) have examined more detailed aspects of social support. In the first study, Kessler and colleagues (1992) examined more specific aspects of social support (perceived support from spouse, from other relatives, and from friends; access to a confidant; frequency of interaction with relatives and with friends; frequency of church attendance and of club attendance) in a sample of same-sex female twins aged 11 to 53 years. The aim was to examine the homogeneity versus heterogeneity of social support as a construct. Genetic factors played a role



in perceived relative support, perceived friend support, having a confidant, frequency of church attendance, and frequency of club attendance. Shared environmental influences were important for perceived spouse support, perceived relative support, frequency of interaction with relatives, frequency of interaction with friends, and frequency of attending church. Nonshared environmental factors accounted for at least 50% of the variance for all aspects of social support, with the exception of frequency of church attendance (20%) and club attendance (48%). Given the differing patterns of influence across different types of social support, it seemed clear that social support is a heterogeneous construct.

A second study was conducted after an additional wave of data was collected on the female twins. Here, the reliable components of different aspects of social support were examined in a genetically informed way (Kendler, 1997). Using six slightly different categories than the first study (i.e., relative problems and support, friend problems and support, confidants, and social integration), this study found that relative problems and support were influenced by genetic, shared, and nonshared environmental influences, whereas the remaining four categories were influenced by only genetic and nonshared environmental influences. Findings of shared environmental influences on the relative problems and support were explained by the fact that twins share the same relatives.

A sample of same-sex male twins and opposite-sex twins were collected to correspond with the first wave of same-sex female twins. This sample was used to examine sex differences in social support, using the same constructs described in the previous paragraph (Agrawal, Jacobson, Prescott, & Kendler, 2002). Findings indicated no qualitative gender differences for any of the factors. In other words, there were no differences in the sources of genetic influences for men and women. On the other hand, quantitative differences in genetic influences between men and women were found for relative support and confidants. Consistent with previous studies, shared environmental influences were only found for relative support and problems.

## Marital Relationships

Most genetically informed studies of marriage focus on marital *status* rather than marital *quality*, so those studies will be reviewed first. The first study to examine a component of marital status was a study of the genetic

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and environmental influences on divorce, using a sample drawn from the Minnesota Twins Registry (McGue & Lykken, 1992). Participants were between the ages of 34 and 53 years. Both male and female MZ twins were more similar for divorce status than DZ twins, indicating genetic influences. Having a divorced MZ co-twin increased one's odds of getting a divorce nearly sixfold, whereas having a divorced parent or DZ co-twin only increased risk by less than twofold. Heritability estimates for divorce were nearly equally divided between genetic and nonshared environmental influences, for both men and women.

The question has also been asked about whether people who have never married and people who have divorced can both be categorized as having "failed to marry successfully." If they are two sides of the same coin, MZ co-twins of those who have never married should have higher rates of divorce than DZ co-twins, and the MZ co-twins of those who have divorced should have a higher rate of never marrying than DZ co-twins. This question was addressed using the National Academy of Sciences–National Research Council (NAS–NRC) sample of World War II veterans (Trumbetta & Gottesman, 1997). These males were born between 1917 and 1927 and were surveyed in 1972 and 1985, when the men were 45 to 55 and 58 to 68 years old, respectively. Never marrying and divorcing were not co-heritable, though there was a nonsignificant trend for more co-twins of those who had divorced to have never married. The authors suggest that this indicates the possibility of a continuum of pair bondedness. The same authors later explored marital status in the evolutionary terms of pair bondedness and mate diversification, saying that being married resembles the former and divorce maps onto the latter (Trumbetta & Gottesman, 2000). Again using the sample of World War II veterans, they found that pair bonding seemed to be more genetically influenced than mate diversification, though the heritability declined over time.

The Vietnam Era Twin Registry was used to examine the heritability of marital status, so again the sample was composed only of males. These men served in the U.S. military between the years of 1965 and 1975 and were assessed in 1987. This study examined never marrying versus getting divorced, and whether the same factors that influenced never marrying also influenced divorce (Jerskey et al., 2001). Jerskey and colleagues found that there were no genetic influences on never marrying, but that if one was to marry, there were genetic influences on getting divorced, replicating previous work (McGue & Lykken, 1992). In other words, one twin's being married was independent of his co-twin's likelihood of divorce.

The propensity to marry seems to be a different story. A study using a sample from the Minnesota Twin Registry examined the genetic and environmental influences on getting married (Johnson, McGue, Krueger, & Bouchard, 2004). The total heritability, including additive and dominant effects, was .70. Gender differences were examined, and it was found that the heritability for men and women were similar, but the genetic components of variance were not the same for men and women. In other words, different genetic factors influence men's propensity to marry than influence women's.

Only recently has the genetic and environmental influences on marital *quality* been explored. One of the aims of the Twin Moms study described above was to examine how marital satisfaction impacted maternal mental health. Toward this aim, the Dyadic Adjustment Scale (DAS; Spanier, 1976) and the Marital Adjustment Test (Locke & Wallace, 1987), both often-used measures of marital quality, were administered to both the twin women and their spouses. Most measures of wife's marital quality were at least moderately influenced by genetic factors (standardized parameter estimates of .24–.33) and primarily influenced by nonshared environmental influences (standardized parameter estimates of .67–.85; E. L. Spotts et al., 2004). One exception was DAS Affectional Expression, which showed negligible amounts of genetic influence. Shared environmental influences played no role in the twin women's reports of their marital quality. Genetic influences on husbands' reports of their marital quality were also examined, with results similar to those of wives' reports. It needs to be noted that influences on husband reports were those of the *wife's* genetically and environmentally influenced characteristics, so these findings of genetic influences represent the effects of wife's genetically influenced characteristics on their husband's perceptions of their marriage. This could be the result of either active GE correlations, whereby the wife actively selects a husband that fits with her genetically influenced characteristics, or evocative GE correlation, whereby the wife's genetically influenced characteristics elicit a particular response from her husband.

## Accounting for Genetic Influences on Social Relationships

This section will discuss studies that have set about trying to identify what accounts for genetic and/or environmental influences on social

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relationships. These studies use multivariate analyses to examine the covariance among variables.

What accounts for the genetic influences on aspects of marriage? It appears that personality plays a large role. Using a sample drawn from the Minnesota Twin Registry, Jockin and colleagues extended McGue and Lykken's (1992) study of divorce to examine the extent to which personality accounted for the genetic influences on divorce (Jockin, McGue, & Lykken, 1996). They found that 30% and 42% of the genetic influences on divorce could be accounted for by personality for women and men, respectively. The authors suggested that the genetic influences on divorce actually influence personality, which in turn influences divorce risk. They also found that the environmental factors influencing personality and divorce were almost entirely independent of each other.

Another study using the same sample examined the extent to which personality characteristics accounted for the genetic influences (Johnson et al., 2004). Using composite scales of the Multidimensional Personality Questionnaire, they found that genetic influences accounted for 68% and 83% of the covariance between personality and the propensity to marry for men and women, respectively.

Another study using the Twin Moms sample tried to account for the genetic influences on marital quality and found that the personality characteristics aggression and optimism accounted for all of the genetic influences on wives' reports of marital quality (E. Spotts et al., in press). It had also been hypothesized that husbands' personality characteristics would account for a substantial portion of the nonshared environmental influences on wives' reports of marital quality, but this was not supported.

### Associations Between Relationships and Adjustment

There is a large literature linking interpersonal relationships with various forms of adjustment. Behavioral geneticists are beginning to explore the underlying mechanisms of these associations by examining what types of influences account for the links.

Parenting is usually discussed in conjunction with child outcome. Because the focus of this book is on midlife, a discussion of these studies will be omitted. However, one study used causal modeling to examine the link between remembered parenting and psychological distress in adulthood (Gillespie, Zue, Neale, Heath, & Martin, 2003). The sample of females was drawn from the Australian National Health and Medical Research Council Twin Registry and ranged in age from 18 to 45 years.

Genetic and nonshared environmental factors were found to account for the association between remembered parenting and depressive and anxious symptoms. The best fitting causal model suggested that remembered parenting influenced psychological distress rather than the opposite.

Levels of social support are robustly associated with positive and negative outcomes. What is less clear are the mechanisms underlying this association. In addition to the possibility of high-quality social support leading to better health, there might also be genetic factors that influence both social support and adjustment. Behavioral genetic studies are uniquely equipped to test these possibilities.

Bergeman and colleagues used the SATSA sample to examine the links between social support and well-being, as measured by self-reports of depressive symptoms and life satisfaction (Bergeman, Plomin, Pedersen, & McClearn, 1991). Genetic and nonshared environmental influences were found to contribute to the associations between social support and each of the two outcome measures. This suggests underlying genetically influenced factors influence both social support and mental health, while factors that make the twins different affect both their relationships and their mental health. Such nonshared factors might include husbands, jobs, or children.

Studies using the Twin Moms sample expanded on this research by including both marital quality and adequacy of social support in examinations of associations among interpersonal relationships and both depressive symptoms and well-being in women. This was done to see if marital quality and social support made independent contributions to mental health. When looking at depressive symptoms, genetic influences were shared among marital quality, social support, and depressive symptoms (E. L. Spotts et al., 2005). Social support shared genetic and nonshared environmental variance with depressive symptoms beyond that common to all three variables. Two aspects of positive mental health were examined: well-being and global self-worth (E. Spotts et al., 2005); different patterns of genetic and environmental influences were found for each. For well-being, marital quality and social support accounted for the same genetic and nonshared environmental variance, with no additional variance explained by social support. On the other hand, social support was independently associated with global self-worth by genetic and nonshared environmental factors after taking marital quality into account. Several conclusions can be drawn from these findings. First, there are genetic factors that are common to relationships and mental health. Second, these findings suggest that marital quality is not the only source of social support influential to a sense of self-worth, though other aspects

of marriage may be important. Third, it seems that influences on global self-worth may be wider ranging than those on well-being, as indicated by social support being independently associated with global self-worth. Finally, we can speculate on the findings of nonshared environmental influences. For each of these analyses, husbands' reports of marital quality were substituted for the wives' reports, and in all cases, nonshared environmental influences were shared among marital quality, social support, and the measure of mental health (E. Spotts et al., 2005; E. L. Spotts et al., 2005). These findings suggest that the husband is an important source of nonshared environmental influence on his wife's feelings about her interpersonal relationships and her mental health.

Kessler, Kendler, Heath, Neale, and Eaves (1992) used the female twin sample drawn from the Virginia Twin Registry to examine the processes by which perceived social support and adjustment to stress, as indicated by onset of major depression (MD), were linked. Among the models tested was one hypothesizing that the link could be explained by a common genetic cause; the model was not supported. At first glance, these results seem to contradict the findings from the Twin Moms study reported above. However, the two studies used very different outcome measures: depressive symptomatology versus acute onset of MD. Also, the nature of the variables used in the analyses resulted in very different sample sizes, with the Virginia sample being reduced to 22 cases.

Wade and Kendler (2000) tested several possibilities explaining associations between lifetime MD (as indicated by an onset of MD within the past 12 months at either Time 1 or Time 2) and social support using the Virginia female twin sample. Three hypotheses were tested, but the one discussed here postulates that social support and the risk for MD may be linked by common, genetically influenced traits. To test this hypothesis, the level of social support in one twin was predicted by MD in her co-twin. A significant association was found, and it was stronger for MZ than DZ twins, indicating genetic influences on the association.

## **Measures of the Environment and Health**

### **Education and Occupation**

Not only family and social relationships but also socioeconomic status characterize midlife. Of several ways to operationalize socioeconomic status, education and occupation are referred to and examined most frequently.

A great deal of research has demonstrated that socioeconomic status relates to health and well-being as well as financial success in life. It is therefore of interest to discuss here why there are individual differences in socioeconomic status and how genes and familial environment contribute to that variation.

One of the early attempts in this area was from studies using twins born in Norway between 1915 and 1960. Heath and colleagues revealed that the relative contributions from genetic and environmental sources varied across cohorts for education (Heath et al., 1985). Heritability was greatest (74% and 45% of the variance in men and women, respectively) for subjects born between 1940 and 1949, slightly lower for the younger subjects born after 1950, and lowest for those born before 1939. Similarly, Tambs and colleagues also reported secular change in the importance of genetic influences on education using part of the same cohorts of Norwegian twins (Tambs, Sundet, Magnus, & Berg, 1989).

Behavioral genetic research with a more powerful study design was conducted using Swedish twins including those who were reared apart. Lichtenstein and colleagues first used this study design to explore the sources of individual differences in educational achievement (Lichtenstein, Pedersen, & McClearn, 1992). Having divided the sample at the age of 60 years, they found that younger and older groups showed significant differences in the relative importance of genetic and environmental effects. Their findings suggested that genetic influences seem to have a greater impact on education for the younger age group than the older group, which was in accord with the Norwegian studies mentioned in the previous paragraph. By using a similar sample, Lichtenstein and Pedersen then investigated genetic and environmental contributions to educational achievement as part of multivariate analyses (Lichtenstein & Pedersen, 1997). Genetic factors for educational achievement were estimated as more important in this study than in the earlier study.

A large sample of Australian twins has provided longitudinal data to study genetic and environmental effects on educational achievement (Baker, Treloar, Reynolds, Heath, & Martin, 1996). Baker and colleagues evaluated the heritability of self-reported educational achievement using twins from older (born between 1893 and 1950) and younger (born after 1950) cohorts. Based on self-reports in 1981 and 1989, they estimated genetic variance in the longitudinal correlation of educational achievement measured at these two occasions as 57%. In contrast to the Norwegian studies, there were no differences in the estimates for the



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younger and older cohorts in this Australian study. Baker and colleagues pointed out that the discrepancy in findings could be attributable to cultural differences, sampling differences, or statistical power.

Occupational status and work environment have also been studied, using twins primarily from socioeconomic status points of view. Arvey and colleagues assessed genetic component to job satisfaction based on the Minnesota Study of Twins Reared Apart, another unique resource for studying twins reared apart (Arvey, Bouchard, Segal, & Abraham, 1989). Having administered the Minnesota Job Satisfaction Questionnaire to reared-apart twins whose mean age was 42 years, they reported that approximately 30% of the variance in general job satisfaction was due to genetic factors, based on intraclass correlations. Keller and colleagues extended this study to evaluate the heritability of work values (Keller, Arvey, Dawis, Bouchard, & Segal, 1992). With the Minnesota Importance Questionnaire, they estimated genetic effects on the total variance as approximately 40%.

In the aforementioned study in SATSA (Lichtenstein et al., 1992), the authors investigated relative importance of genetic and environmental effects on occupational status as well as educational achievement. Substantial sex differences in the estimates were observed for this measure, although differences between younger and older groups were not tested. Based on the same sample, Hershberger and colleagues examined genetic and environmental influences on perceptions of organizational climate, using the Work Environment Scale and another measure of job satisfaction (Hershberger, Lichtenstein, & Knox, 1994). Genetic effects were significant for Supportive Climate (22% of the variance) but not for Time Pressure, implying difference in genetic effects on controllable and uncontrollable environment. Unlike Arvey et al. (1989), genetic effects were not significant for job satisfaction. Lichtenstein and colleagues then analyzed the dimensions of occupation and distances between occupational categories, by using intrapair differences in adult occupational position (Lichtenstein, Hershberger, & Pedersen, 1995). Genetic factors showed substantial influences (60% of the variance) on occupational status for men, while shared and nonshared environmental factors were of about equal importance. For women, genetic effects were less important (12% of the variance), and shared and nonshared environmental effects accounted for more of the variation. The results pointed to the importance of genetic effects for sources of familial resemblance in occupational status for men.



Lichtenstein and Pedersen further extended the previous SATSA studies of occupational and educational achievements in a multivariate study design with a measure of general cognitive ability (Lichtenstein & Pedersen, 1997). They found that educational achievement and occupational status showed significant genetic variance both in common with and independent of genetic variance for cognitive ability. The findings indicated that, although genetic influences for cognitive ability were important for socioeconomic status, a substantial portion of the genetic variance in socioeconomic status was independent of that for cognitive ability. These results were in agreement with the aforementioned Norwegian study (Tambs et al., 1989), in which the variance in occupational status was accounted for more by specific genetic effects than by those in common with educational achievement and general cognitive ability. In contrast, Lichtenstein and Pedersen also found that rearing environmental effects for educational achievement and occupational status were completely overlapping, which implied that the same factors in the rearing home made family members similar to each other for both education and occupation.

Taken together, there is evidence that genetic effects are responsible for substantial part of individual differences in educational achievement and occupational status. However, estimates for the relative importance of genetic and environmental effects often differ between younger and older cohorts, a difference that is likely to be attributable to cohort differences. There also may be cultural differences that vary the estimates from study to study.

## Stressful Life Events

In general, individuals experience a number of major life changes as they grow older. It is reasonable to think that such events significantly correlate with psychological development and health in later life. Since Holmes and Rahe introduced the Social Readjustment Rating Scale (Holmes & Rahe, 1967), stressful life events have been an important topic among developmental psychologists and behavioral geneticists, due to their potential function in the etiology of various mental disorders.

A SATSA study first explored genetic influence on perceptions of major events later in life (Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990). By using twins reared apart and reared together, whose ages ranged from 27 to older than 80, Plomin et al. estimated genetic influence on the occurrence of life events as 40% of the variance of

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the total life events score. Saudino and colleagues then assessed sex differences in genetic and environmental contributions to life events and genetic influences of personality on its association with life events (Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997). Significant genetic variance was observed only for women in controllable, desirable, and undesirable life events, and no sex differences were found for uncontrollable events. Multivariate analyses of personality (as indexed by Neuroticism, Extraversion, and Openness to Experience) and life events suggested that all of the genetic variance on controllable, desirable, and undesirable life events for women is common to personality. Saudino et al. thus concluded that genetic influences on life events appeared to be entirely mediated by personality in middle-aged and older women.

Besides the SATSA, a number of studies have been conducted to examine the relative contribution of genetic and environmental influences to stressful life events using the Virginia Twin Registry. Kendler and colleagues (Foley, Neale, & Kendler, 1996; Kendler, Neale, Kessler, Heath, & Eaves, 1993) reported that genetic factors and familial environment each accounted for around 20% of the variance of total life events in twins aged from 17 to 55 years. They suggested that stressful life events be classified into "network events" (those which occur within an individual's social network), where twin resemblance was due solely to the familial environment, and "personal" events (those in which the individual is directly involved), where most twin resemblance was the result of genetic factors. Their findings indicated that stable individual differences were more important determinants of personal stressful life events than random (occasion-specific) factors. Bolinskey and colleagues provided additional support for the previous findings with a sex limitation model (Bolinskey, Neale, Jacobson, Prescott, & Kendler, 2004). Unlike Saudino et al. (1997), they found that many of the same genetic factors were acting within both sexes.

Overall, genetic influences have been consistently demonstrated in adult samples on the probability of experiencing stressful life events. On the other hand, the importance of shared environment and sex remains unclear. In addition, longitudinal studies that address age differences are necessary to assessing changes in genetic and environmental effects across age.

### Stress Coping

As we have seen significant genetic influences on stressful life events thus far, it is reasonable to question whether variation in the way adult

individuals cope with stress is also influenced by genetic factors. Stress coping is, therefore, a potentially important area that could shed light on the mechanisms of stress-induced health problems, a major issue in later life.

Kendler and colleagues first investigated genetic and environmental influences on stress coping in young adults (Kendler, Kessler, Heath, Neale, & Eaves, 1991). Data were collected by self-report coping behavior based on 14 items of the Ways of Coping Checklist (WCC; Folkman & Lazarus, 1980) from female twin pairs (mean age, 29 years) in the Virginia Twin Registry. Using factor analysis, three coping factors were identified: Turning to Others, Problem Solving, and Denial. The authors reported that twin resemblance in turning to others and problem solving could be explained entirely by genetic factors with heritability of approximately 30%. For denial, shared environmental factors accounted for 19% of the total variation.

Busjahn and colleagues also attempted to evaluate genetic effects on stress coping by using 19 coping styles as well as four secondary coping factors, based on a German coping questionnaire with twin pairs aged 34 years on average (Busjahn, Faulhaber, Freier, & Luft, 1999). All the four coping factors (Defense, Emotional Coping, Substitution, and Active Coping) showed genetic influences, whereas shared environment had no significant influence. For more specific coping styles, genetic influences were found for 17 of the 19 coping styles, most of which were solely under genetic influences. On the other hand, there was no single genetic factor common to all the specific coping styles.

Whereas the two studies mentioned in the previous paragraphs used relatively young samples, a recent SATSA study explored the relative contribution of genetic and environmental influences to stress coping in middle-aged and older adults (Kato & Pedersen, 2005). The subjects were twins reared apart and reared together of both sexes, aged from 26 to 89 years. Three coping scales (Problem Solving, Turning to Others, and Avoidance) were derived by factor analysis based on the Billings and Moos coping measure (Billings & Moos, 1984). The results indicated not only genetic influences on all three coping styles, a result in line with the previous studies, but also significant sex differences in variance estimates for all of them. Shared rearing environmental influences were observed only for Turning to Others and Avoidance in women. Kato and Pedersen further examined the basis of covariation between coping styles and personality traits (as indexed by Neuroticism, Extraversion, and Openness

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to Experience) and revealed that genetic influences on adults' coping differentially reflected genetic factors in common with personality traits. The sources of covariation also showed significant sex differences.

As yet, little is known about the origin of individual variation in coping styles with middle-aged adult samples, despite the vast amount of coping studies at phenotypic levels. As with stressful life events, longitudinal data are warranted in order to distinguish cohort effects from aging effects on stress coping in adults.

### Self-Rated Health

Self-rated (or self-reported) health is a global measure of an individual's physical and mental health, as a summary of his or her own perception from different aspects. Self-rated health is often measured by using three items based on Duke University's Older Americans Resources Survey (Duke University, 1978). Although it is a relatively simple measure, this subjective health assessment is often superior to clinical assessments for predicting outcomes such as mortality (Idler & Benyamini, 1997). As change in health is another important feature that characterizes midlife, it is rational to discuss behavioral genetic studies of self-rated health in this section.

A series of studies using Swedish twins have demonstrated the importance of genetic factors for individual differences in self-rated health. Harris and colleagues first analyzed cross-sectional data from twins reared apart and reared together, aged 26 to 86 years (Harris, Pedersen, McClearn, Plomin, & Nesselrode, 1992). There were significant age differences in the genetic and environmental influences on self-rated health in the sample: Genetic effects were important for subjects aged 60 years or older, whereas the total variance was accounted for entirely by environmental influences in the younger subgroup.

Using a computer-assisted telephone interview with twin pairs aged 17 to 85 years, Svedberg and colleagues tested age and sex differences in genetic and environmental sources of variation for self-rated health (Svedberg, Lichtenstein, & Pedersen, 2001). Having divided the sample into four age groups, they found that increase in total variance was primarily due to genetic influences in the two middle age groups (45–74 years), whereas no genetic influences were observed in either the youngest or the oldest age group, which was similar to what Harris et al. (1992) reported. In contrast, no sex differences were shown in variance

components in the sample. Svedberg et al. further investigated longitudinal data in the SATSA over a 9-year time period (Svedberg, Lichtenstein, Gatz, Sandin, & Pedersen, in press). They revealed that changes in means and variance of self-rated health were largely influenced by cohort differences, although socioeconomic status in childhood did not account for these cohort differences. The results also indicated that correlations between measurement occasions were explained almost equally by genetic and environmental factors.

In another SATSA study, Lichtenstein and Pedersen examined how the associations between self-rated health, stressful life events, and social relationships were mediated by genetic and environmental influences (Lichtenstein & Pedersen, 1995). For men, environmental influences were solely important for variation in the psychosocial measures and were the primary mediators of the relationship with health. For women, however, a substantial portion of the variance in the psychosocial factors was due to genetic influences, and these influences also contributed to the bulk of the correlations with health. Although inconsistent with the previous results from univariate analyses of self-rated health, these findings suggested the importance of sex differences for the relationships and mediation with the psychosocial factors.

In conclusion, it seems that individual variation in the way of assessing one's own health reflects complex effects of sex, psychosocial factors, and changes in society, as well as genetic predisposition to diseases. Given that the variance of individuals' health status increases with increased age, it can be said that lifestyle in middle age deserves more attention for the betterment of prevention and intervention that will lead to successful aging.

## Cognitive Abilities

Until relatively recently, the greatest amount of longitudinal behavioral genetic research has been on cognitive abilities, both general and specific. Again, most studies concern childhood and adolescence, although currently there are a growing number of programs in "gerontological genetics," which include information on cognitive abilities in midlife. As has been the case historically with most efforts at characterizing the genetic and environmental sources of individual differences, the first analyses comprised cross-sectional estimates of heritability for specific cognitive abilities and composite measures of general abilities or IQ. As these studies were performed at a time when demonstrating that genetic variation

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could be of at least *some* importance for behavioral traits, there was little attention paid to hypothesis testing. The first heritability studies of cognitive abilities were based on twin infants, children, and adolescents, followed by adoption studies of the same part of the life span. As the results from these early developmental behavioral genetic studies emerged, Robert Plomin (1986) ventured a number of expectations about genetic influences across the life span. Among the expectations, he predicted that heritability would increase with increasing age, possibly as a result of amplification of genetic effects existing early in development. This proved to be the case for general cognitive abilities, at least from childhood through midlife (McCartney, Harris, & Bernieri, 1990; Pedersen & Lichtenstein, 1997), predominantly due to a concomitant decrease in shared environmental influences.

SATSA provided some of the first evidence for the relative importance of genetic and environmental influences on cognitive abilities in adults older than 50 years of age. Eighty percent of the individual differences seen for general cognitive ability are due to genetic differences (Pedersen, Plomin, Nesselrode, & McClearn, 1992). This value is somewhat higher than those found in adolescence and early adulthood but is consistent with two reports from middle-aged adults (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Tambs, Sundet, & Magnus, 1984). The SATSA results were based on the first wave of in-person testing data, where the sample was treated as one age group, ranging from 50 to 85 years. However, a further exploration into age differences in heritability estimates found some differences across age groups (Finkel, Pedersen, McGue, & McClearn, 1995). Older Swedish twins (over 65 years) demonstrated a significantly lower heritability for general cognitive abilities, suggesting a possible inverted L-shaped function for the relationship between heritability and IQ later in life. Thus, genetic influences are substantial and stable through midlife but appear to decrease in importance after age 65, at least cross-sectionally.

### Aging Trajectories for Cognitive Abilities

Any exploration of longitudinal changes in genetic and environmental influences on individual differences should first characterize normative (mean) and variance trajectories. If sufficient occasions of measurement are available, latent growth models are ideal for describing such patterns. Latent growth models incorporating two linear slopes were recently

applied to cognitive data from SATSA in order to address several phenotypic issues (Finkel, Reynolds, McArdle, Gatz, & Pedersen, 2003). Results indicated stability or even improvement up to age 70 for measures of crystallized ability, followed by significant decline. Linear age changes were found for many cognitive abilities. For those measures with a large speed component, a growth curve model incorporating two separate slopes was indicated, suggesting stability prior to age 65 but a significant acceleration in linear decline thereafter. Accelerating decline in cognitive performance at age 65 may reflect true aging changes, or it may be a consequence of the transition from an active (presumably stimulating) work life to retirement. Furthermore, it is possible that a more accurate model of cognitive aging includes not one but two transition periods when the rate of decline changes.

### Longitudinal Changes in Heritability of Specific Cognitive Abilities

Early longitudinal analyses of SATSA were cohort-sequential such that sliding-interval-based cohorts and longitudinal information were used to examine changes in genetic and environmental components of variance. Inspection of the longitudinal trends for the separate cohorts clearly demonstrates that heritability is relatively stable longitudinally at approximately 80% in midlife cohorts (50–65 years at first testing) but decreases longitudinally from approximately 80% at Time 1 to 60% at Time 3 in the older cohorts.

Subsequent research has taken advantage of expansions of the latent growth curve models to include twin data to examine genetic and environmental influences on mean level of cognitive ability, as well as rates of change over a 13-year span. Various models have been applied, including a two-slope model with centering based on time rather than age (Reynolds, Finkel, Gatz, & Pedersen, 2002) and most recently, a model with linear change prior to age 65 and a quadratic term for accelerated decline after age 65 (Reynolds et al., in press). In all cases, genetic influences were of greater importance for ability level than for linear change. Furthermore, genetic variance was often of greater importance for quadratic (accelerated) change than for linear change. Regardless of the model or time span, the period of midlife (in this case, 50–65 years) was characterized by relative stability, not only in total variation, but also in sources of individual



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differences. After age 65, decreasing genetic variance, but increasing nonshared environmental variance, was the most typical pattern across cognitive domains. Genetic variance decreases were most apparent for perceptual speed measures, fluid abilities, and general cognitive ability late in life; however, one verbal measure and two memory-related measures showed evidence of both increasing genetic and nonshared environmental variance after age 65. These results demonstrate clearly that the relative influence of genetic factors will change as the dynamic combination of genetic and environmental factors changes over the life span. Furthermore, each component of cognitive aging needs to be examined independently; we will not find a single aging trajectory, nor will we find a single explanation for cognitive aging. Relevant to students of midlife will be explorations of turning points (i.e. when relative stability changes to growth or decline) and the predictors of these events.

### The Role of Speed in Cognition

In the previous descriptions of individual differences and cognition, we have focused on individual abilities and domains, but not at the associations within or among domains. Within cognition, some of the strongest evidence for age-related associations comes from studies of perceptual speed and cognitive performance. The processing-speed theory (Birren, 1964; Salthouse, 1996) posits that cognitive aging results from generalized slowing of perceptual and cognitive processes. Several lines of evidence support this theory (Salthouse, 2004), although relatively little work has been done to explore the role of genetic influences in these processes. Cross-sectional analyses of SATSA indicate that the heritability of cognitive ability in adulthood results, at least in part, from genetic influences associated with perceptual speed instead of genes for cognitive functioning, *per se* (Finkel & Pedersen, 2000). Additional longitudinal analyses were pursued in which latent growth curve models were applied to cognitive data from which the speed had been partialled out (Finkel & Pedersen, 2004). When variance components for original and speed-corrected measures were compared, two patterns of results were found. For Information and Thurstone's Picture Memory, greater nonshared environmental variance and less genetic variance were evident for the speed-corrected measures. In contrast, removing the speed variance from Block Design and General Cognitive Ability resulted in a marked difference in the shape of the age trajectory in genetic influence. For both variables,



estimates of genetic variance began at comparable levels at age 50, but they decline at a much faster rate for the speed-corrected measure. This suggests that already during midlife, speed is an important source of genetic variation for fluid and general cognitive abilities.

## Associations Between Cognitive and Physical Functioning

It is not unlikely that if perceptual speed is important for cognitive functioning in adulthood, motor speed is also important. A multivariate analysis incorporating measures of motor speed, perceptual speed, and cognitive ability was conducted on the middle-aged and young-old cohorts of SATSA (Finkel, Pedersen, & Harris, 2000) to examine possible age differences in the relationship between speed and cognition. Results indicated qualitative differences between cohorts in the nature of the genetic and environmental variance common to motor speed, perceptual speed, and cognitive abilities. The genetic variance in cognitive functioning in the middle-aged cohort was defined primarily by motor speed, whereas genetic variance in the older cohort was defined by perceptual speed.

Recent analyses of SATSA data have focused on the aging trajectory for variables that serve as markers of aging and on the genetic and environmental influences on the rates of decline. Growth curve analysis of five markers of aging indicate a steeper rate of decline for men than women in forced expiratory volume and grip strength and a moderate rate of increase in mean arterial pressure for both men and women (Finkel, Pedersen, et al., 2003). For two variables, motor functioning and well-being, growth curve analysis identified a turning point (age 70) at which functioning changed from stability to decline. Quantitative genetic growth curve analysis indicated genetic influences on the mean level of performance on all five markers of aging (Finkel, Pedersen, et al., 2003). The same was not true for the rates of change with age. Genetic influences on the slope were found for three of the variables: motor functioning, mean arterial pressure, and forced expiratory volume.

## Summary of Cognitive Abilities in Midlife

Even though most of the longitudinal results concerning cognitive abilities in midlife have emanated from longitudinal studies of aging, we are able to discern clear patterns. Throughout the period of midlife, there are

few dramatic changes in means and variances. In other words, midlife is a period of relative stability, at least with regard to cognitive abilities. Not only do mean levels remain stable until age 65, but individual differences also appear to be stable throughout this period. Similarly, sources of individual differences, both genetic and environmental, appear to be quite stable during this period. The exceptions appear to be related to the role that genes have in mediating the speed-cognition relationship. The reasons for this apparent stability, and at the same time considerable variability, may seem somewhat enigmatic. Perhaps our measures are not sensitive enough to pick up the sensitive changes that may reflect progression into dementia. If this is the case, we may not be able to detect whether there are changes in the genetic or environmental mechanisms for these changes. Furthermore, almost none of these studies have examined cognitive trajectories as a function of measured changes in the environment (such as lifestyle transitions) or biological processes (such as menopause or disease onset). It may be that there are myriad GxE interactions that can only be detected in stratified groups. Nevertheless, for the population as a whole, this is a period of relative phenotypic, genotypic, and environmental stability.

## Conclusion

At the outset of this endeavor, we were skeptical as to whether there were sufficient empirical data available not only based, but also in particular focused, on behaviors and outcomes relevant to midlife. Our preconceptions were in part fulfilled: All too few studies focus on midlife, in particular those that are concerned with individual differences rather than normative characterizations. We found that most studies of developmental behavioral genetics included subjects in midlife only by default, as in the case of family studies (parents in midlife); by accident, as in the case of twin cohorts; or as a secondary baseline (aging studies). The lack of focus on midlife is perhaps understandable, as the sine qua non of behavioral genetic analysis is variability. For many outcomes, such as health, there is indeed little variability in midlife, as most individuals can be classified as fitting within a fairly narrow band of "normality." For other characteristics, such as behavior, the few results available indicate relative stability in variation throughout midlife. Furthermore, both theoretical and empirical emphasis has been placed almost exclusively on early and

late development. As a result, there is a dearth of systematic information on sources of individual differences in midlife.

Fortunately, the era of demonstrating that genetic influences *may* be of importance for behavioral characteristics is at an end. The efforts of behavioral geneticists in the 1980s and 1990s, as well as public acceptance of the results from the human genome project, have redirected research inquiry toward understanding mechanisms and multivariate relationships, with the implicit understanding that both genes and environments are important for individual differences. There are enormous gains to be made by approaches that evaluate the mechanisms contributing to associations among characteristics, rather than isolating behaviors as independent entities. The associations between social support and adjustment, psychosocial factors and self-rated health, and cognition and physical functioning described earlier in this chapter exemplify the value of multivariate approaches, and further applications are to be encouraged.

Although little effort has been made to understand associations and mechanisms in midlife, there are notable exceptions, such as the study of interpersonal relationships described earlier in this chapter. These studies exemplify three important aspects that should be encouraged:

1. The behaviors and outcomes of interests are relevant to the ageband of interest (marital relationships, parenting, stress coping).
2. There are identifiable exposures (stressful life events, occupational stressors).
3. It is possible to test hypotheses regarding mechanisms because relevant genetically informative constellations of individuals are available (twin-family designs to test GE correlation).

Nonetheless, there are a number of areas in which improvement can be made. At the very least, greater attention should be paid to phenotypes of relevance in midlife. The study of interpersonal relationships is one good example, but far more can be done to understand other relevant responses, including responses to relevant stressors (e.g., work environment, familial demands, health-related behaviors, medically unexplained disorders such as "burnout"). Despite advancements in methodological techniques to incorporate measured environments in behavioral genetic designs, far too little effort has been placed on identifying and measuring relevant environmental exposures and incorporating these into the models. It is also quite likely that there are considerable gender and cultural differences in the multitude of genetic and environmental interactions

that influence individual differences in midlife. What is the importance of genetic susceptibility to stress for women's and men's ability to adapt to rigidity in occupational structure, or to infectious agents? Finally, the study of variation in midlife must be approached from a longitudinal point of view rather than as a series of cross-sectional snapshots in time.

## References

- Agrawal, A., Jacobson, K. C., Prescott, C. A., & Kendler, K. S. (2002). A twin study of sex differences in social support. *Psychological Medicine, 32*(7), 1155–1164.
- Arvey, R. D., Bouchard, T. J., Segal, N. L., & Abraham, L. M. (1989). Job satisfaction: Environmental and genetic components. *Journal of Applied Psychology, 74*(2), 187–192.
- Baker, L. A., Treloar, S. A., Reynolds, C. A., Heath, A. C., & Martin, N. G. (1996). Genetics of educational attainment in Australian twins: Sex differences and secular changes. *Behavior Genetics, 26*(2), 89–102.
- Baumrind, D. (1973). The development of instrumental competence through socialization. In A. D. Pick (Ed.), *Minnesota symposium on child psychology* (Vol. 7, pp. 3–46). Minneapolis: University of Minnesota Press.
- Bergem, A. L. M. (2002). Norwegian twin registers and Norwegian twin studies—An overview. *Twin Research, 5*(5), 407–414.
- Bergeman, C. S., Plomin, R., Pedersen, N. L., & McClearn, G. E. (1991). Genetic mediation of the relationship between social support and psychological well-being. *Psychology and Aging, 6*(4), 640–646.
- Billings, A. G., & Moos, R. H. (1984). Coping, stress, and social resources among adults with unipolar depression. *Journal of Personality and Social Psychology, 46*(4), 877–891.
- Birren, J. E. (1964). *The psychology of aging*: Englewood Cliffs, NJ: Prentice Hall.
- Bolinskey, P. K., Neale, M. C., Jacobson, K. C., Prescott, C. A., & Kendler, K. S. (2004). Sources of individual differences in stressful life event exposure in male and female twins. *Twin Research, 7*(1), 33–38.
- Boomsma, D., Vink, J., van Beijsterveldt, T., De Geus, E. J., Beem, A. L., Mulder, E. J., et al. (2002). Netherlands Twin Register: A focus on longitudinal research. *Twin Research, 5*(5), 401–406.
- Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: The Minnesota Study of Twins Reared Apart. *Science, 250*(6), 223–228.
- Braungart, J. M. (1994). Genetic influence on “environmental” measures. In J. C. DeFries, R. Plomin, & D. W. Fulker (Eds.), *Nature and nurture during middle childhood* (pp. 233–248). Cambridge, UK: Blackwell.

- Busjahn, A., Faulhaber, H. D., Freier, K., & Luft, F. C. (1999). Genetic and environmental influences on coping styles: A twin study. *Psychosomatic Medicine, 61*(4), 469–475.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301*(5631), 386–389.
- Croft, M., Read, A. W., de Klerk, N., Hansen, J., & Kurunczuk, J. (2002). Population based ascertainment of twins and their siblings, born in Western Australia 1980 to 1992, through the construction and validation of maternally linked database of siblings. *Twin Research, 5*(5), 317–323.
- Duke University Center for the Study of Aging and Human Development. (1978). *Multidimensional functional assessment: The OARS Methodology*. Durham, NC: Duke University Press.
- Elkins, I. J., McGue, M., & Iacono, W. G. (1997). Genetic and environmental influences on parent-son relationships: Evidence for increasing genetic influence during adolescence. *Development Psychology, 32*(2), 351–363.
- Finkel, D., & McGue, M. (1993). The origins of individual differences in memory among the elderly: A behavior genetic analysis. *Psychology and Aging, 8*(4), 527–537.
- Finkel, D., & Pedersen, N. L. (2000). Contribution of age, genes, and environment to the relationship between perceptual speed and cognitive ability. *Psychology and Aging, 15*(1), 56–64.
- Finkel, D., & Pedersen, N. L. (2004). Processing speed and longitudinal trajectories of change for cognitive abilities: The Swedish Adoption/Twin Study of Aging. *Aging, Neuropsychology, and Cognition, 11*(2–3), 325–345.
- Finkel, D., Pedersen, N. L., & Harris, J. R. (2000). Genetic mediation of the associations among motor and perceptual speed and adult cognitive abilities. *Aging, Neuropsychology, and Cognition, 7*(2), 141–155.
- Finkel, D., Pedersen, N. L., McGue, M., & McClearn, G. E. (1995). Heritability of cognitive abilities in adult twins: Comparison of Minnesota and Swedish data. *Behavior Genetics, 25*(5), 421–431.
- Finkel, D., Pedersen, N. L., Reynolds, C. A., Berg, S., deFaire, U., & Svartengren, M. (2003). Genetic and environmental influences on decline in biobehavioral markers of aging. *Behavior Genetics, 33*(2), 107–123.
- Finkel, D., Reynolds, C. A., McArdle, J. J., Gatz, M., & Pedersen, N. L. (2003). Latent growth curve analyses of accelerating decline in cognitive abilities in late adulthood. *Developmental Psychology, 39*(3), 535–550.
- Foley, D. L., Neale, M. C., & Kendler, K. S. (1996). A longitudinal study of stressful life events assessed at interview with an epidemiological sample of adult twins: The basis of individual variation in event exposure. *Psychological Medicine, 26*(6), 1239–1252.

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- Folkman, S., & Lazarus, R. S. (1980). An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behavior*, 21(3), 219–239.
- Ganster, D. C., & Victor, B. (1988). The impact of social support on mental and physical health. *British Journal of Medical Psychology*, 61(1), 17–36.
- Gillespie, N. A., Zue, G., Neale, M., Heath, A. C., & Martin, N. G. (2003). Direction of causation modeling between cross-sectional measures of parenting and psychological distress in female twins. *Behavior Genetics*, 33(4), 383–396.
- Goodman, R., & Stevenson, J. (1989). A twin study of hyperactivity—II. The aetiological role of genes, family relationships and perinatal adversity. *Journal of Child Psychiatry and Psychology and Allied Disciplines*, 30(5), 691–709.
- Harris, J. R., Pedersen, N. L., McClearn, G. E., Plomin, R., & Nesselroade, J. R. (1992). Age differences in genetic and environmental influences for health from the Swedish Adoption/Twin Study of Aging. *Journal of Gerontology: Psychological Sciences*, 47(3), 213–220.
- Heath, A. C., Berg, K., Eaves, L. J., Solaas, M. H., Corey, L. A., Sundet, J., et al. (1985). Education policy and the heritability of educational attainment. *Nature*, 314(6013), 734–736.
- Heath, A. C., Eaves, L. J., & Martin, N. G. (1998). Interaction of marital status and genetic risk for symptoms of depression. *Twin Research*, 1(3), 119–122.
- Hershberger, S. L., Lichtenstein, P., & Knox, S. S. (1994). Genetic and environmental influences on perceptions of organizational climate. *Journal of Applied Psychology*, 79(1), 24–33.
- Holmes, T. H., & Rahe, R. H. (1967). The Social Readjustment Rating Scale. *Journal of Psychosomatic Research*, 11(2), 213–218.
- Hur, Y. M., & Bouchard, T. J. (1995). Genetic influences on perceptions of childhood family environment: A reared apart twin study. *Child Development*, 66(2), 330–345.
- Idler, E. L., & Benyamini, Y. (1997). Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior*, 38(1), 21–37.
- Jerskey, B. A., Lyons, M. J., Lynch, C. E., Hines, D. A., Ascher, S., Nir, T., et al. (2001). *Genetic influence on marital status*. Paper presented at the Tenth International Congress of Twin Studies, Kensington, London, UK.
- Jockin, V., McGue, M., & Lykken, D. T. (1996). Personality and divorce: A genetic analysis. *Journal of Personality and Social Psychology*, 71(2), 288–299.
- Johnson, W., McGue, M., Krueger, R. F., & Bouchard, T. J. (2004). Marriage and personality: A genetic analysis. *Journal of Personality and Social Psychology*, 86(2), 285–294.
- Kaprio, J., & Koskenvuo, M. (2002). Genetic and environmental factors on complex diseases: The older Finnish twin cohort. *Twin Research*, 5(5), 358–371.
- Kato, K., & Pedersen, N. L. (2005). Personality and coping: A study of twins reared apart and twins reared together. *Behavior Genetics*, 35(2), 147–158.

- Keller, L. M., Arvey, R. D., Dawis, R. V., Bouchard, T. J., & Segal, N., L. (1992). Work values: Genetic and environmental influences. *Journal of Applied Psychology, 77*(1), 79–88.
- Kendler, K. S. (1997). Social support: A genetic-epidemiologic analysis. *American Journal of Psychiatry, 154*(10), 1398–1404.
- Kendler, K. S., Kessler, R. C., Heath, A. C., Neale, M. C., & Eaves, L. J. (1991). Coping—A genetic epidemiologic investigation. *Psychological Medicine, 21*(2), 337–346.
- Kendler, K. S., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1993). A twin study of recent life events and difficulties. *Archives of General Psychiatry, 50*(10), 789–796.
- Kessler, R. C., Kendler, K. S., Heath, A. C., Neale, M. C., & Eaves, L. J. (1992). Social support, depressed mood, and adjustment to stress: A genetic epidemiologic investigation. *Journal of Personality and Social Psychology, 62*(2), 257–272.
- Lichtenstein, P., Ganiban, J., Neiderhiser, J. M., Pedersen, N. L., Hansson, K., Cederblad, M., et al. (2003). Remembered parental bonding in adult twins: Genetic and environmental influences. *Behavioral Genetics, 33*(4), 397–408.
- Lichtenstein, P., Hershberger, S. L., & Pedersen, N. L. (1995). Dimensions of occupations: Genetic and environmental influences. *Journal of Biosocial Science, 27*(2), 193–206.
- Lichtenstein, P., & Pedersen, N. L. (1995). Social relationships, stressful life events, and self-reported physical health: Genetic and environmental influences. *Psychology and Health, 10*(4), 295–319.
- Lichtenstein, P., & Pedersen, N. L. (1997). Does genetic variance for cognitive abilities account for genetic variance in educational achievement and occupational status? A study of twins reared apart and twins reared together. *Social Biology, 44*(1–2), 77–90.
- Lichtenstein, P., Pedersen, N. L., & McClearn, G. E. (1992). The origins of individual differences in occupational status and educational level: A study of twins reared apart and together. *Acta Sociologica, 35*(1), 13–31.
- Locke, H., & Wallace, K. (1987). Marital Adjustment Test. In N. Fredman & R. Sherman (Eds.), *Handbook of measurements for marriage and family therapy* (pp. 46–50). New York: Brunner/Mazel.
- McCartney, K., Harris, M. J., & Bernieri, F. (1990). Growing up and growing apart: A developmental meta-analysis of twin studies. *Psychological Bulletin, 107*(2), 226–237.
- McGue, M., & Lykken, D. T. (1992). Genetic influence on the risk of divorce. *Psychological Science, 3*(6), 368–373.
- Neiderhiser, J. M., Reiss, D., Pedersen, N. L., Lichtenstein, P., Spotts, E., Hansson, K., et al. (2004). Genetic and environmental influences on mothering of adolescents: A comparison of two samples. *Developmental Psychology, 40*(3), 335–351.



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- Pedersen, N. L., & Lichtenstein, P. (1997). Biometric analyses of human abilities. In C. Cooper & V. Varma (Eds.), *Processes in individual differences* (pp. 125–147). London: Routledge.
- Pedersen, N. L., Lichtenstein, P., & Svedberg, P. (2002). The Swedish Twin Registry in the Third Millennium. *Twin Research*, 5(5), 427–432.
- Pedersen, N. L., McClearn, G. E., Plomin, R., Nesselroade, J. R., Berg, S., & DeFaire, U. (1991). The Swedish Adoption Twin Study of Aging: An update. *Acta Geneticae Medicae et Gemellologiae*, 40(1), 7–20.
- Pedersen, N. L., Plomin, R., Nesselroade, J. R., & McClearn, G. E. (1992). A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychological Science*, 3(6), 346–353.
- Plomin, R. (1986). *Development, genetics, and psychology*. Hillsdale, NJ: Erlbaum.
- Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-Environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, 84(2), 309–322.
- Plomin, R., Lichtenstein, P., Pedersen, N. L., McClearn, G. E., & Nesselroade, J. R. (1990). Genetic influence on life events during the last half of the life span. *Psychology and Aging*, 5(1), 25–30.
- Plomin, R., McClearn, G. E., Pedersen, N. L., Nesselroade, J. R., & Bergeman, C. S. (1989). Genetic influence on adults' ratings of their current family environment. *Journal of Marriage and the Family*, 51(3), 791–803.
- Reiss, D., Cederblad, M., Pedersen, N. L., Lichtenstein, P., Elthammar, O., Neiderhiser, J. M., et al. (2001). Genetic probes of three theories of maternal adjustment: II. Genetic and environmental influences. *Family Process*, 40(3), 261–272.
- Reiss, D., Neiderhiser, J. M., Hetherington, E. M., & Plomin, R. (2000). *The Relationship Code: Deciphering genetic and social influences on adolescent development*. Cambridge, MA: Harvard University Press.
- Reiss, D., Pedersen, N. L., Cederblad, M., Lichtenstein, P., Hansson, K., Neiderhiser, J. M., et al. (2001). Genetic probes of three theories of maternal adjustment: I. Recent evidence and a model. *Family Process*, 40(3), 247–259.
- Reiss, D., Plomin, R., Hetherington, E. M., Howe G., Rovine, M., Tyron, A., et al. (1994). The separate social worlds of teenage siblings: An introduction to the study of the nonshared environment and adolescent development. In E. M. Hetherington, D. Reiss, & R. Plomin (Eds.), *Separate social worlds of siblings: Impact of the nonshared environment on development* (pp. 63–110). Hillsdale, NJ: Erlbaum.
- Reynolds, C. A., Finkel, D., Gatz, M., & Pedersen, N. L. (2002). Sources of influence on rate of cognitive change over time in Swedish twins: An application of latent growth models. *Experimental Aging Research*, 28(4), 407–433.
- Reynolds, C. A., Finkel, D., McArdle, J. J., Gatz, M., Berg, S., & Pedersen, N. L. (in press). Quantitative genetic analysis of latent growth curve models of cognitive abilities in adulthood. *Developmental Psychology*.



- Rowe, D. C. (1981). Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Developmental Psychology, 17*(2), 203–208.
- Rowe, D. C. (1983). A biometrical analysis of perceptions of family environment: A study of twins and singleton sibling kinships. *Child Development, 54*(2), 416–423.
- Salthouse, T. A. (1996). The processing-speed theory of adult age differences in cognition. *Psychological Review, 103*(3), 403–428.
- Salthouse, T. A. (2004). What and when of cognitive aging. *Current Directions in Psychological Science, 13*(4), 140–144.
- Saudino, K. J., Pedersen, N. L., Lichtenstein, P., McClearn, G. E., & Plomin, R. (1997). Can personality explain genetic influences on life events? *Journal of Personality and Social Psychology, 72*(1), 196–206.
- Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype greater than environment effects. *Child Development, 54*(2), 424–435.
- Skytthe, A., Kyvik, K., Holm, N. V., Vaupel, J. W., & Christensen, K. (2002). The Danish twin registry: 127 birth cohorts of twins. *Twin Research, 5*(5), 352–357.
- Spanier, G. B. (1976). Measuring dyadic adjustment: New scales for assessing quality of marriage and similar dyads. *Journal of Marriage and the Family, 38*(1), 15–28.
- Spotts, E., Lichtenstein, P., Hansson, K., Neiderhiser, J. M., Pedersen, N. L., Cederblad, M., et al. (in press). Personality and marital satisfaction: A behavioral genetic analysis. *European Journal of Personality*.
- Spotts, E., Reiss, D., Pedersen, N., Lichtenstein, P., Cederblad, M., & Hansson, K. (2005). Genetic effects on women's positive mental health: Do marital relationships and social support matter? *Journal of Family Psychology*.
- Spotts, E. L., Neiderhiser, J. M., Ganiban, J., Reiss, D., Lichtenstein, P., Hansson, K., et al. (2005). Accounting for depressive symptoms in women: A twin study of associations with interpersonal relationships. *Journal of Affective Disorders*.
- Spotts, E. L., Neiderhiser, J. M., Towers, H., Hansson, K., Lichtenstein, P., Pedersen, N. L., et al. (2004). Genetic and environmental influences on marital relationships. *Journal of Family Psychology, 18*(1), 107–119.
- Svedberg, P., Lichtenstein, P., Gatz, M., Sandin, S., & Pedersen, N. L. (in press). Self-rated health in a longitudinal perspective: A 9-year follow-up twin study. *Journals of Gerontology: Social Sciences*.
- Svedberg, P., Lichtenstein, P., & Pedersen, N. L. (2001). Age and sex differences in genetic and environmental factors for self-rated health: A twin study. *Journal of Gerontology: Social Sciences, 56*(3), 171–178.
- Tambs, K., Sundet, J. M., & Magnus, P. (1984). Heritability analysis of the WAIS subtests: A study of twins. *Intelligence, 8*(4), 283–293.
- Tambs, K., Sundet, J. M., Magnus, P., & Berg, K. (1989). Genetic and environmental contributions to the covariance between occupational status, educational attainment, and IQ: A study of twins. *Behavior Genetics, 19*(2), 209–222.

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---

- Trumbetta, S., & Gottesman, I. (1997). Pair-bonding deconstructed by twin studies of marital status: What is normative? In N. Segal, G. E. Weisfeld, & C. C. Weisfeld (Eds.), *Uniting psychology and biology: Integrative perspectives on human development*. Washington, DC: American Psychological Association.
- Trumbetta, S., & Gottesman, I. (2000). Endophenotypes for marital status in the NAS-NRC twin registry. In J. L. Rogers & D. C. Rowe (Eds.), *Genetic influences on human fertility and sexuality* (pp. 253–269). Boston: Kluwer Academic.
- Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14*(6), 623–628.
- Wade, T. D., & Kendler, K. S. (2000). The relationship between social support and major depression: Cross-sectional, longitudinal, and genetic perspectives. *Journal of Nervous and Mental Disease*, *188*(5), 251–258.