PART I

Biological and Genetic Processes

CHAPTER ONE

Behavior Genetics and Adolescent Development: A Review of Recent Literature

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Introduction

Behavior genetics is a quantitative method, and adolescent development is a psychological topic. Treating the cross between these two arenas appears, at the surface, to require collecting research in which the method has been applied to study the topic, and reviewing that research for coherence and common themes. But the challenge is rather more difficult than the surface level view might suggest. Below the surface is a great deal of shifting sand, which makes organizing the topic difficult. Because of this instability, it is critical that we carefully and explicitly define a foundational starting point. In the introduction to this article, we begin with some definitions, and then we describe the difficulties inherent in reviewing "behavior genetics and adolescent development." We conclude our introduction with a summary of the foundation on which we will base our review. In the next section, we carefully build that foundation. Following, we summarize the relevant research, and embed it within the organizational foundation.

Definitions: Why Is This Chapter Difficult to Frame?

The starting point for most behavior genetic modeling is the conceptual partitioning of sources of variance into genetic, shared environmental, and nonshared environmental (e.g., Rowe & Plomin, 1981). Some of the similarity between individuals in the same family may be caused by sharing the same genes. For example, monozygotic (MZ) twins share 100 percent of their genes, and should be approximately identical on traits or behaviors that are strongly under genetic control (e.g., height, eye color). Dizygotic (DZ) twins and full siblings on average share .50 of their genes, half siblings share .25, cousins share

.125, etc. Alternatively, some of the similarity between individuals in the same family has as its etiological source the shared environment; this theoretical source of influence causes related individuals to be similar because they share a common environment within the family. For example, parental discipline style may be a shared environmental influence that is in common to all children in a family and that results in similarities between the children in a measure of response to authority. Finally, the nonshared environment is a theoretical source of influence that causes individuals in the same family to be different from one another, that is, these influences are not shared in common among siblings. For example, a particularly outstanding teacher that a child had in first grade (but who was not the teacher of the other children in the family) could create nonshared environmental influences on reading motivation. Or, even within the family, parents may be more authoritative with their sons than with their daughters (which would result in parental discipline style being a nonshared rather than shared environmental influence).

It is well known to behavior geneticists – if not necessarily to the general population of researchers – that measures of variance related to genetic processes (heritabilities, or h^2) and shared environmental processes (sometimes called the common environment, or c^2) are *not* immutable (see Angoff, 1988). The basic biometrical model (e.g., Falconer, 1981) partitions the overall variance in the dependent variable into that attributable to genes, that attributable to the shared environment, and that attributable to the nonshared environment. (Most estimation procedures confound the latter source – nonshared environmental variance, often called e^2 – with measurement error, although there are statistical ways around this problem; see Rodgers, Rowe, & Li, 1994.)

Two critical features of this definition must be appreciated. First, h^2, c^2, and e^2 depend on both the amount of variance relevant to their conceptual domain, and they also depend on the amount of *overall* variance. The conceptual formulas are h^2 = genetic variance/total variance, c^2 = shared environmental variance/total variance, and e^2 = nonshared variance/total variance. (Total variance is often referred to as "phenotypic variance" in the behavior genetic literature.) These three coefficients necessarily add up to 100 percent, so that each one may be interpreted as the proportion of total variance attributable to genetic, shared environment, and nonshared environment sources, respectively. Thus, for example, if genetic variance remains fixed, but overall variance in the dependent variable (phenotypic variance) increases substantially, then h^2 goes down due to the denominator, without any shift in the genetic contribution itself. Second, h^2, c^2, and e^2 *must* be interpreted in a variance context; that is, the genetic and environmental contributions explain individual differences, and not general properties of traits or behaviors. For example, genes perfectly determine eye color. Yet, in a setting with few non-brown-eyed people, heritability would be very low (because there is little phenotypic variation). In other words, h^2 indicates how much *variance* in a trait or behavior is related to genetic sources. It does not indicate whether the existence of the trait or behavior has any genetic etiology. Similarly, c^2 and e^2 indicate how much *variance* in a trait or behavior is attributable to these two environmental sources, not whether the trait or behavior has underlying environment etiology. For example, a person's accent is obviously influenced by the environment. But a measure of accent would show very low c^2, for the simple reason that there is very little overall variance to explain. Even otherwise responsible behavior geneticists often misuse their language, and refer to "genetic or environmental influences on a trait," when

what they really mean is "genetic or environmental influences on *variance in* or *individual differences* in a trait."

The explanation above refers to the basic *additive* genetic model. This model implies that a number of separate genetic sources each contribute separate and additive pieces of influences on the variance in a trait or behavior of interest. Of course, some genetic processes are nonlinear and nonadditive (e.g., basic principles of Mendelian inheritance are inherently non-additive). Quantitative modeling in behavior genetics supports fitting dominance models (e.g., Neale & Cardon, 1992), and the concept of emergenesis has been proposed to account for configural genetic contributions (Lykken et al., 1992). On the other hand, another type of nonlinear genetic process, epistasis, involves genetic interactions of alleles across genetic loci (as opposed to Mendelian dominance, which involves interactions of alleles within a genetic locus). Epistasis is difficult to model in behavioral genetic settings (e.g., Neale & Cardon, 1992). In this review, we will focus on efforts to fit additive genetic models to adolescent data, primarily because little attention has been given to nonlinear or non-additive models in the literature. Besides genetic nonadditivity, a second problem occurs when there are genetic-environmental interactions that are not accounted for within the model. Turkheimer (1998) showed that the effect of failing to account for genetic/environmental interactions is to bias estimates of h^2 and c^2. While there is little published literature, efforts to account for gene-environment interactions are ongoing (and several will be mentioned in reviewing the literature).

Both h^2 and c^2 (and, implicitly, e^2) can shift and change over time and over age, as the variances in both the numerators and the denominators shift and change. At adolescence in particular, phenotypic variance in many traits and behaviors can shift substantially. For a few years, height (and most other physiological measures) becomes more highly variable, as children reach puberty at a wide range of ages. In less obvious ways, there can be shifts in overall variance of intelligence, many personality traits, and especially in social and health behaviors. Smoking provides a good example. Among 8-year-olds, smoking behavior has virtually no heritability. Obviously, this is not because genetic influences do not contribute to smoking in general, but because there is little phenotypic variance in measures of smoking among 8-year-olds. By age 14, smoking heritabilities begin to be detectable in a measure like age-at-first cigarette (but not so much in measures of smoking addiction). By age 20, measures of smoking intensity, smoking duration, and smoking addiction all show heritable components. These points about genetic influences apply equally to both shared and nonshared environmental influences as well. It is in this context that we refer to the "shifting sand" on which we observe genetic and environmental influences.

There are several proper ways to treat these problems. The first is interpretational; authors must be clear about what h^2, c^2, and e^2 mean, and what they don't mean. Second, there are direct measures of these influences; for example, some behavior geneticists emphasize the importance of computing heritabilities, and also computing coefficients of genetic variation, which are not affected by the overall variance of the DV itself (see, e.g., Houle, 1992). Third, the shifts in h^2 and c^2 values can provide a great deal of information in and of themselves. For example, Kohler, Rodgers, and Christensen (1999) documented a substantial shift in h^2 of fertility, using secular twin data from Denmark. The dramatic upward shifts in h^2 values that they observed occurred simultaneously with the

two fertility transitions in Denmark, which provided a framework within which to explain this rapid change in heritability.

The difficulties defined in this introductory section are all related to the method (and, importantly, to improper interpretation of the design and purpose of the method). The "shifting sand" problem of interpreting heritabilities is only a problem if it is allowed to be. We will attempt to carefully extract the proper information from the studies we review, and we invite the reader to bring healthy critical inspection into this investigation with us. As Maccoby (2000) noted, "knowing only the strength of genetic factors . . . is not a sufficient basis for estimating environmental ones" (p. 1). Turkheimer (1998) suggested that heritability studies lead only to the "banal tautology that all behavior is ultimately based in the genotype and brain" (p. 782). While we are more sanguine about the value of such studies than Turkheimer, we applaud the skeptical approach that this position implies.

Finally, we intend to respond to the "shifting sand" problem by firmly tying down our treatment with theoretical orientations from both social and biological domains that focus on the topic and not on the method. The study of adolescence in general, and especially adolescent development, focuses attention on the process of change. We have used three different social theories in our past work to frame the process of adolescent development. More properly, these are closer to motivating orientations than they are to formal theories, because we in no sense test or evaluate the structures. Rather, we let them guide our investigation and review. The three approaches are the Transition Behavior Perspective, the Life Course Perspective, and Problem Behavior Theory. In addition, the biological perspective that best informs the study of adolescent development is to consider hormonal influences that affect both physiological changes and behavioral changes.

Theoretical Framework

Adolescence embedded in a social and temporal ecology

The Transition Behavior Perspective is developed more completely in Ensminger (1987) and in Rodgers and Rowe (1993). This orienting framework views adolescence as a period of expanded behavioral opportunity. Adolescents begin to have choices within a behavioral ecology that were not available to them during childhood. How much to study, whether to smoke and drink, what parties to attend, what school clubs to join, and management of health behaviors are emerging issues within the decision-making framework of many adolescents. In addition, consumer behavior offers expanded opportunity in adolescence as well. Transition behaviors are defined as behaviors that adolescents use – either overtly or implicitly – to signal impending adulthood. These behaviors may have other purposes as well, but at least part of their status is to socially represent the transitional features of adolescence. Some of those behaviors are unhealthy, or at least socially proscribed. Examples include risky sexual behavior, reckless driving, smoking, drinking to excess, drug use, and cheating on homework. Some transition behaviors are healthy and socially normative. Examples include playing in the school band, checking out library

books, playing on a tennis team, and joining a church group. The reason the Transition Behavior Perspective is valuable in the context of the current review is that is orients attention toward behaviors that many consider to be prototypical adolescent behavior.

Most of the attention to transition behaviors in the social science (and behavior genetic) literature is on behaviors that we have previously labeled mildly and severely deviant (see Rodgers, Billy, & Udry, 1984; Rodgers & Rowe, 1993). While the socially appropriate transition behaviors deserve much more attention than they have received, we will focus on those that have received substantial research attention. Those include smoking, drinking, drug use, and dating/sexual behavior.

A second theoretical perspective helps motivate some of the behavior genetic literature in this domain. Problem Behavior Theory (Jessor & Jessor, 1977) suggests that a number of both mildly and severely deviant behaviors may group together. The question of whether problem behaviors group together was expanded by Moffit (1993) into a theory of "adolescence-limited" versus "life-course-persistent" deviant behavior. Rodgers and Rowe (1990) found that sexual behavior had a somewhat unusual status in the context of Problem Behavior Theory; while it did covary with other mildly deviant behaviors (e.g., smoking, drinking, and driving illegally), it also contained substantial unique variance of its own that did not overlap with those other variables. The methodology of behavior genetics provides a powerful approach to investigating this perspective, and we will review several studies that consider the multivariate relationships between two or more problem behaviors.

A third theoretical approach that is useful is the Life Course Perspective, which suggests that norms are established by society to define age-appropriate behavioral transitions; "age differentiation is expressed in the sequence of roles and events, social transitions, and turning points that depict the life course" (Elder, 1975). Hogan and Astone (1986) suggested that transitions from adolescence into and through adulthood have ordered stages, and that society has normative expectations about age-appropriate life transitions; Rindfuss, Swicegood, and Rosenfeld (1987) investigated deviations from those normative ordered stages. Most of the examples of events of high salience in the Life Course Perspective are demographic transitions such as marriage, first child, or education. In combining the Transition Behavior and the Life Course Perspective, we bring a whole new domain of behaviors under this umbrella. In this combined perspective, the first cigarette, the first drink, and loss of virginity can also be considered to have "life course status," in that we organize our thinking and plans in relation to the timing of these events. Further, they are of particular salience in defining the "adolescent experience," an observation that brings Problem Behavior Theory into this integrative framework. Indeed, we suspect that many individuals (both adults and adolescents themselves) would define these mildly and severely deviant behaviors as closer to "adolescent prototype behaviors" than the more socially normative transition behaviors like joining clubs and playing in the band.

Nevertheless, we note the existence of certain cognitive and personality transitions that may also be motivated by Transition Behavior and Life Course perspectives. Certain subjects in school may be perceived in a Life Course perspective. One example would be bright students taking pre-calculus in 11th grade, or Advanced Placement English as seniors. Another would be the transition in junior- or mid-high to having a study hall

(and associated expectations) as part of the school experience. A third would be expanding social autonomy, such as the choice to attend a school dance or to have lunch off-campus. A fourth would be the emergence of leadership in a high-school social/political structure.

Adolescence embedded in a hormonal ecology

The idea of adolescent "raging hormones" has been overplayed; for example, most adolescents are not nearly as sexually active in response to those hormones (even those who are nonvirgins are not nearly as sexually active) as many media portrayals might suggest (see, e.g., Rodgers, 1996). The proper phrase is probably closer to "changing hormones." Indeed, hormones drive many of the physiological changes during adolescence, which in turn have tremendous impacts on behavioral processes.

Buchanan, Eccles, and Becker (1992) developed an integrated framework in which to view the influence that hormonal changes during adolescence have on adolescent behaviors and traits. They noted that

> Historically, most of the changes in mood and behavior were presumed to be negative and to be the result of biological factors, particularly of hormones. . . . More recently, psychologists have questioned both the prevalence of such negative changes and their hypothesized biological roots. . . . Emphasis has shifted to contextual (i.e., family, school, peer group) and psychological (i.e., self-esteem, gender role orientation) factors. (p. 62)

Their review shows hormonal changes to potentially influence adolescent self-esteem, happiness, concentration, aggression and behavior problems, and social relationships. Udry et al. (1985) showed a link between androgenic hormones and male adolescent sexual behavior, and Udry and Talbert (1988) documented personality responses to hormonal changes. Susman et al. (1985) showed a link between adolescent hormone levels and socio-emotional behaviors.

Introductory summary

Behavior genetic modeling too often occurs in its own vacuum. This introduction was designed to create a larger context for the upcoming review of behavior genetic studies of adolescent behaviors and traits. Genes influence hormones. Hormones have both overlapping variance with and can change traits and behaviors. All occur at an organic level, within an individual who is embedded in a social environment of family, friends, school, church, and other social influences. Further, all of these complex interrelations are defined temporally, and may mean different things at different ages and stages. The Life Course and Transition Behavior Perspectives, along with Problem Behavior Theory, can help us appreciate this complex interplay. Recent work in the behavior genetic literature has shifted our orientation away from a strict causal flow from the genome to behavior, and has substituted various complex and fascinating feedback loops involved in the causal

process (see, e.g., Brown, 1999; Gottlieb, 2000). We will return to this broader perspective in a concluding section, where we place in context the results of our review of genetic and environmental influences that emerge from the behavior genetic literature.

Methodology Used in This Review

To identify relevant empirical studies to be reviewed in this chapter, we partitioned our title into three component keywords: "behavior genetic," "adolescence," and "development." Our initial literature review identified primary journals in each area: *Behavior Genetics* for the first, *Adolescence* for the second, and *Developmental Psychology* for the third. We obtained copies of each article that involved behavior genetic analysis of a topic related to adolescent development from 1985 to 2000. In reviewing those articles, we identified a number of additional articles that were obtained as well. In addition, several recent chapters in *Annual Review of Psychology* provided both material and references to support this review (see Maccoby, 2000; Plomin & Rende, 1991; Rose, 1995; Steinberg & Morris, 2001).

By far the majority of our articles reviewed here come from *Behavior Genetics* and *Developmental Psychology*. Further, most are recent articles. Though we searched for relevant articles in each area back to 1985, most that were relevant had been published more recently than that, many in the latter half of the 1990s.

Behavior Genetic Research on Adolescence: The Social/Behavioral Ecology

The social/behavioral domains represented within the articles we have collected include adolescent transition behaviors (smoking, drinking, sexual behavior, and other risk-taking activities), social/mental health among adolescents (including depression and antisocial behavior), indicators of social relationships within the family (parents and siblings), and treatment of cognitive and personality development during adolescence.

Transition behaviors

Behavior genetic studies have shown that there is genetic variance underlying adolescent transition behaviors, and have also indicated some of the dynamics underlying the social/environmental correlates. While a general consideration of problem behaviors spreads out beyond the boundaries covered by transition behavior, they certainly overlap substantially with that domain, especially through alcohol and drug use.

Within the domain of problem behaviors, Gjone et al. (1996) used the Child Behavior Checklist (CBCL) to measure internalizing and externalizing problem behaviors among Norwegian same-sex twins born during the 1970s and 1980s. Their results showed

significant heritabilities for both domains, with increasing h^2 and decreasing c^2 as the severity of the problem behavior increased for both internalizing and externalizing behaviors (although rescaling of those variables dampened this pattern). Van den Oord, Boomsma, and Verhulst (1994) also used the CBCL with a sample of international adopted children in the Netherlands. Like Gjone et al., they found significant heritability for externalizing behaviors; however, they did not match their result for internalizing behaviors. They also found larger variance for males than for females for aggressive behavior, which they speculated might have genetic origins. Eaves et al. (1993) studied conduct disorder items from the Rutter Parent Questionnaire using 8–16-year-old male twins from the Virginia Adolescent Behavioral Development study. Their model identified four underlying latent classes, which were themselves shown to be heritable. However, their model rejected a unidimensional interpretation of the latent processes underlying conduct disorder. Rodgers, Rowe, and Li (1994) studied measures obtained from the Behavioral Problem Index (BPI) using 5–11-year-old children from respondents in the National Longitudinal Survey of Youth (NLSY), nationally representative data from adolescents born between 1958 and 1965. For the six subscales of the BPI, they found strong heritabilities for the more trait-based subscales (Anxiety, Hyperactivity, and Dependent) and weaker heritabilities for the social subscales (Antisocial, Peer Conflict, and Headstrong). Further, the three social subscales showed significant shared environmental variance. The quality of the home environment (measured by the HOME) showed significant nonshared influences on siblings in the same household for all of the subscales except for Peer Conflict. Van der Valk et al. (1998) used an adoption design with 10–18-year-old Dutch children to investigate the longitudinal stability of problem behaviors. Their dependent measures came from the Child Behavior Checklist. They found stability in an externalizing factor, primarily genetic in origin, and more shared environmental influence at the early ages than at the later ones. Rowe, Almeida, and Jacobson (1999) studied adolescent aggression using the Add Health data. They found an $h^2 = .32$, with little shared environmental variance. The heritability increased from this value with increases in family warmth measured at the school level. Finally, van den Oord and Rowe (1997) investigated social maladjustment using the BPI measures in the NLSY. Their findings suggested that nonshared environmental influences had the most effect on children's problem behaviors. They also found support for a "liability model," suggesting "a stable underlying liability may be the 'third variable' that explains the relations between subsequent levels of problem behaviors" (pp. 319–320).

Several studies addressed specific transition behaviors. Koopmans et al. (1999) studied smoking initiation and quantity using a twin study of Dutch adolescents. They fit a bivariate model, and found that there were separate dimensions underlying smoking initiation and smoking quantity. They found substantial $h^2 = .39$ and even greater $c^2 = .54$ for smoking initiation, while h^2 and c^2 became negligible for smoking quantity. This finding – that there are separate (though potentially overlapping) liabilities for smoking onset and smoking persistence – was also obtained by Madden et al. (1999) and Heath et al. (1999) using adult samples.

Viken et al. (1999) used twins from a Finnish birth cohort born in 1975–9 to study self-reported alcohol consumption. Like the smoking results above, they found that shared environmental influences were important in drinking initiation, but that additive genetic

effects became more important in explaining variance in drinking frequency among those who had already begun drinking. Buster and Rodgers (2000) used measures of light and heavy drinking from adolescents in the NLSY. They found significant h^2 for adolescent males in the NLSY for light drinking, with the shift to significant c^2 for heavy drinking. Adolescent females had a strong genetic basis (and non-significant c^2's) for both light and heavy drinking.

Carey (1992) and Meyer and Neale (1992) used a simulated dataset to investigate onset of drug use among teenagers. Carey simulated the dataset to represent three processes, diffusion/exposure, initial use, and persistence. Meyer and Neale fit models to show that, at least in this artificial dataset, the shared environment accounted for twin similarity in drug use onset and timing.

While multiple stage theories that include social influence processes have been developed to explain the spread of adolescent transition behaviors like smoking and drinking (e.g., Rowe & Rodgers, 1991; Rowe et al., 1996), the basic and simple distinction between onset and maintenance appears to be the most abiding and valuable one to emerge from this investigation (e.g., Mayhew, Flay, & Mott, 2000). The coherent result across these studies is that onset is driven socially, but after initiation has occurred, the variance in the amount is genetically based.

As discussed above, not all transition behaviors are necessarily problem behaviors. Participation in sports is a positive transition behavior (along with other extracurricular activities, church programs, hobbies, etc.). Boomsma et al. (1989) studied participation in sports using teenaged twins from Amsterdam and their parents. They found a genetic component to sports participation, and a strong shared environmental component for females (but not for males). Further, they developed a bivariate model between sports participation and heart rate, and found heart rate to have a stronger genetic basis and sports participation to have a stronger environmental basis. We encourage additional research falling into this domain, in which healthy adolescent transition behaviors are evaluated through behavior genetic and other methodologies.

Sexual behavior is a transition behavior that becomes socially normative with increasing age. Several behavior genetic studies have identified significant heritabilities for measures of age-at-first-intercourse (e.g., Dunne et al., 1997; Rodgers, Rowe, & Buster, 1999), which typically occurs during adolescence in the cultures studied. Miller et al. (1999) showed a relationship between dopamine receptors and age at first intercourse, providing information about a potential genetic mechanism to help explain this link.

Social/mental health

Rende et al. (1993) fit biometrical models to measures of depression in adolescents. They found a significant heritability in the overall depression distribution. However, the genetic component disappeared when models were fit to evaluate extreme depression. In other words, there did not seem to be any different or additional genetic component to extreme depression over and above its status as an extreme form of the overall variance in depression. Pike et al. (1996) used US sibling pairs who were within four years of one another in age, including twins, siblings, half-siblings, and unrelated siblings. Depression was

measured in three ways, using the Child Depression Inventory, the BPI-Depression subscale, and the Behavior Events Inventory (BEI). They found that the "model attributes the variance of depressive symptoms to substantial genetic influence, negligible shared environmental influence, and moderate nonshared environmental influence" (p. 597). Further, they also found that mother's negativity was associated with adolescent depression through the nonshared environment, independent of genes and the shared environment. Results for father's negativity were similar. Jacobson and Rowe (1999) used the kinship structure in the National Longitudinal Survey of Adolescent Health (Add Health) to study the relation between social connectedness and adolescent depression. They found different models for males and females. Genetic influences were stronger for females than for males for both depression indicators, and also for the covariation between social connectedness and depression.

Other (overlapping) literature has investigated antisocial behavior in adolescents. The study by Pike et al. (1996) reviewed above that treated depression also investigated antisocial behavior using subscales from the BPI and the BEI. Antisocial behavior showed more shared environmental variance than did depression, but genetic and nonshared environmental influences also accounted for significant variance. As with depression, the nonshared environment accounted for covariation between mother's negativity and antisocial behavior, and genetic and shared environmental influences contributed variance as well. In a study by O'Connor et al. (1998) using Colorado Adoption Project (CAP) data from late childhood and early adolescence (ages 7–12), antisocial behavior was assessed using constructed measures for parental antisocial behavior and the Child Behavior Checklist (CBC) for children. They were interested in the causal directionality of parenting behavior and children's antisocial behavior. They found that the covariation between negative parenting and antisocial behavior was not evoked by the child; however, their results were consistent with a plausible parental effect on children's antisocial behavior.

Finally, a study by Topolski et al. (1997) used data from the Virginia Twin Study of Adolescent Behavioral Development to study separation anxiety disorder (SAD), overanxious disorder, and manifest anxiety. Moderate heritabilities were found for each of the three, with meaningful shared environmental variance for SAD. There were no strong age or gender differences.

Social relationships with family members

There has been substantial research on adolescents from a behavior genetic perspective on family relationships. Both the Transition Behavior and Life Course perspectives provide motivation for changing relationships to emerge between adolescents and their family. Because adolescents have a new and broader behavioral repertoire, because they are beginning the transition into the independence and autonomy of adulthood, and because society notes several specific markers during adolescence as particularly salient (e.g., transition to high school, first car, leaving home, etc.), these social relationships may be subtle, volatile, and/or dynamic.

In an interesting methodological study, Plomin et al. (1994) showed that a number of measures of the family environment in fact contain substantial genetic variation: "On

average, more than a quarter of the variance of these environmental measures can be accounted for by genetic differences among children" (p. 32). This study, a part of the Nonshared Environment in Adolescent Development (NEAD) project, recruited adolescents from both non-divorced and stepfamilies randomly chosen from the US population. This project grew out of a large effort showing the importance of nonshared environmental influences on individual differences in human (including adolescent) behavior. This particular study demonstrated the difficulty in cleanly separating measures into genetic and environmental categories.

Elkins, McGue, and Iacono (1997) took a developmental perspective in a study of parent–son relationships during adolescence. They used the Minnesota Twin Family Study and a Parental Environment Questionnaire that assessed various aspects of the parent–child relationship. They found different etiologies for twins around age 11 compared to those around age 17. Both ages showed heritability of adolescents' perceptions of the quality of parent–son relationships, with substantially higher h^2 for the older twins. These effects were stronger for the father–son relationship than for the mother–son relationship. Neiderhiser et al. (1998) used data from same-sex siblings in the NEAD project described above to study adolescent perceptions of parenting. Adolescent perceptions of parenting did mediate parent conflict measures and adolescent antisocial measures, and the association between parental and child maladjustment had a strong genetic component. Bussell et al. (1999) used the same data source to investigate the basis for the common finding that parent–child relationships are related to the quality of sibling relationships. Most of the covariance between quality of the mother–child relationship and the quality of the sibling relationship was attributable to the shared environment, although significant genetic and nonshared environmental components were identified as well. Neale (1999) challenged some of the assumptions from the Bussell et al. study, but supported the finding of the importance of the shared environment for both sibling and parent–child relationships (see also Neiderhiser et al., 1999, who replied to Neale's criticisms).

Personality

A number of researchers have fit biometrical models based on behavior genetic designs to measures of personality. McGue, Bacon, and Lykken (1993) used data from twins in Minnesota High Schools during the 1970s, measured in late adolescence and then around ten years later. They used the Multidimensional Personality Questionnaire, which has subscales of positive emotionality (similar to extraversion), negative emotionality (similar to neuroticism/aggression), and constraint (harm avoidance and traditionalism). They found reduction in genetic influence over the two age periods for negative emotionality, stability in overall personality structure that was primarily based on genetic processes, and change in personality structure that was primarily based on environmental factors. Billig et al. (1996) used data from 17-year-old male twins from the Minnesota Twin/Family Study with measures of personality (obtained from the Multidimensional Personality Questionnaire) and a second survey called Life Events Interview for Adolescents. In this second instrument, respondents indicated which of a wide variety of life events they had

experienced, which were themselves divided into family events (e.g., the whole family moved into a new house), nonfamily events independent of the respondent's behavior (e.g., a close friend moved away), and nonfamily events not independent of the respondent's behavior (e.g., suspended from school). Biometrical modeling showed a genetic basis to nonindependent nonfamily life events and genetic covariance between nonindependent nonfamily life events and personality (especially with the personality factor, constraint). Finally, Macaskill et al. (1994) used the Eysenck Personality Scales with Australian twins aged 11 to 18. After partialling out age and gender, they found genetic influence for psychoticism and neuroticism.

Other treatment of more specific personality topics can also be found. Koopmans et al. (1995) studied sensation seeking using Dutch twins aged 12–24 and their parents. They used Zuckerman's Sensation Seeking Scale, which has several non-overlapping subscales. They found that "genes play a major role in the individual differences in sensation seeking" (p. 354), replicating results from Fulker, Eysenck, and Zuckerman. (1980). No shared environmental influences were significant. Eaves et al. (1997) used a 28-item social attitude survey to study conservatism. They found an important age difference, with twins younger than 20 having their conservatism related to shared environmental factors, while those older than 20 had conservatism variance that was primarily related to genetic influence.

Cognitive abilities

Using an Egyptian sample of twins aged 12–19, Abdel-Rahim, Nagoshi, and Vandenberg (1990) studied measures from a broad battery of cognitive measures. They found different results from those obtained from Western studies, with little difference between MZ and DZ twin scores and lower MZ correlations in general. They provide a cross-cultural interpretation of this result, although they hasten to note several methodological concerns, including low sample size and absence of height heritability. Nagoshi and Johnson (1993) used family data from the Hawaii Family Study of Cognition along with (age-corrected) measures of verbal ability, spatial ability, perceptual speed, and visual memory. They found a race difference (between those of Caucasian ancestry and those of Japanese ancestry), with similar c^2 for adolescents and adults. Petril and Thompson (1993) used twin data from the Western Reserve Twin Project cognitive measures from the WISC-R, the Colorado Test of Specific Cognitive Abilities, the Metropolitan Achievement Test, and the Cognitive Abilities Test. Univariate analysis showed both genetic and shared environmental variance underlying individual differences in cognition and achievement, and multivariate analysis showing covariance between them (especially genetic influence). Rodgers, Rowe, and May (1994) used PPVT, PIAT, and Digit Span measures from children and those transitioning into adolescents (aged 5–12) in the NLSY-Children dataset to study intelligence/achievement. They found moderate h^2 (median $h^2 = .50$) and smaller c^2 (median $c^2 = .16$) across five ability measures. Their particular focus, however, was on the nonshared environmental influences. They used specific measures of the nonshared environment, including differences among siblings in trips to the museum, owning books, parental reading, spanking, and HOME scores. They found a significant relationship of

books to the PIAT Reading Recognition subscale, and a significant relationship of trips to the museum to the PIAT Math subscale.

Plomin et al. (1997) used a longitudinal sample of Colorado adoptive and biological children to study biometrical stability across ages 1–16. They found that over time, children became more like their parents in cognitive performance. Further, during adolescence, adoptive children became similar to their biological parents, suggesting that "genes that stably affect cognitive abilities in adulthood do not all come into play until adolescence" (p. 442). In a meta-analysis of literature from 1967–85, McCartney, Harris, and Bernieri (1990) found that the importance of shared environment as it contributed to differences in IQ decreased with age.

Behavior Genetic Research on Adolescence: The Biological/Hormonal Ecology

Very recently, the human genome has been mapped. This effort stimulated both knowledge of and interest in the way our human genetic structure influences human behavior. Critics have long decried efforts to link genetic structure to behavioral outcomes. More properly, we should simply understand that genetic influences will show up in virtually all domains. As Turkheimer (1998) notes, "Everything is biological; everything is genetic" (p. 789). He did not mean, of course, that everything is *only* genetic.

Knowledge of the human genome permits specification of mechanisms. Specific genetic loci have been identified as having influence (in a correlational rather than deterministic sense) on a number of adolescent behaviors that have been treated in this review, including risk-taking, smoking, and alcohol use. Behavior genetics offers a less direct indicator of genetic involvement than molecular genetic methods. On the other hand, the QTL (Quantitative Trait Loci) method simply correlates structure in the genome with measured traits of interest. A number of "false leads" have emerged using QTL studies, although the method will certainly be valuable in the long run. Activity in both molecular and behavioral genetic arenas has accelerated during the past decade, and in many ways the two approaches complement one another.

Physical growth

A number of behavior genetic studies have been made of adolescent development from a biological perspective. Most of these studies have treated measures of growth or other biological markers that would be expected to have strong genetic influences. In these cases, the important questions are often whether there are any environmental influences of note. Analysis of weight development and weight gain is a good case in point. While adult weight has a strong heritable component (h^2 equals around .80 in one review; see Grilo & Pogue-Geile, 1991), it seems reasonable that environmental influence might also affect weight, and many of those might reasonably emerge from the family.

Jacobson and Rowe (1998) studied adolescent Body Mass Index (BMI) among US Add Health respondents from a behavior genetic standpoint. They found substantial heritability, consistent with previous studies, and found evidence that the genes influencing BMI are similar for males and females. However, they found some differences in the genetic/environment apportionment for males/females and for blacks/whites. Further, they found an important source of shared environmental influence for white females. Beunen et al. (1998) studied subcutaneous fat distribution using Belgian twin pairs. They measured stature, weight, BMI, and five subcutaneous skinfold indicators. They found genetic and nonshared environmental variance to underlie individual differences in body fat, but no shared environmental influences. Their results suggested that all of the skinfolds were influenced by the same set of genes.

Another growth process is that related to puberty. Studies of pubertal development using behavior genetic methods have been conducted, although those will be reviewed below in our section on human reproduction.

Hormones

Harris, Vernon, and Boomsma (1998) studied testosterone in Dutch twin-parent data. Unlike the BMI findings, they found different genetic influences for males and females for plasma testosterone concentrations, with heritabilities of around $h^2 = .60$ for males and $h^2 = .40$ for females. Different genetic factors appeared to emerge in adulthood for males, while they were the same for females. Other research on hormones that is not directly tied to behavior genetic methods is nevertheless relevant to this treatment. Udry and his colleagues (Udry et al., 1985; Udry, Talbert, & Morris, 1986) showed links between androgens and adolescent sexual behavior among both males and females.

Sexual behavior and human reproduction occur at the boundary between social and biological processes (or, more properly, we should probably say that they substantially cross the boundary). The biological marker signaling reproductive potential is puberty. Doughty and Rodgers (2000) fit biometrical models to measures of age at menarche for US female adolescents from the NLSY. They found a significant and substantial heritability, with the rest of the variance attributable to the nonshared environment/measurement error. They also found that father absence was related to age at menarche, a finding originally developed by Belsky and his colleagues (e.g., Belsky, 2000) and given substantial attention in the evolutionary psychology literature. Rodgers and Buster (1994) found a seasonal component to menarche, with disproportionate numbers of NLSY females reporting first menstruation in the summer. Further, they estimated a large heritability of $h^2 = .62$ for seasonal menarche, and no shared environmental component.

Sexuality and human reproduction

We have reviewed several studies of age at first intercourse earlier in our section on transition behaviors. In addition, Rodgers and Doughty (2000) did a biometrical analysis of NLSY adolescent fertility expectations, fertility ideals, and fertility outcomes. They found

a substantial heritability of $h^2 = .60$ for ideal fertility reported at ages 14–21 in 1979, although the estimate was much lower when it was reassessed two years later. Moderate heritabilities were found for late adolescents (ages 17–24 in 1982) for fertility expectations. Though few of the NLSY respondents had had children by 1982, enough had done so to estimate heritabilities for this cohort of 17–24-year-olds; they found a remarkably high $h^2 = .73$, with no shared environmental variance. Obviously, a number of adolescent sexual and reproductive behaviors have genetic components, a finding that might appear to some to be inconsistent with the tenets of Fisher's Fundamental Theorem of Natural Selection (Fisher, 1930). This inconsistency is only apparent and not real, however. Rodgers, Rowe, and Miller (2000) provide broad empirical treatment and Rodgers et al. (2001) discuss the role of genetic influences on human fitness and resolve the apparent inconsistency with Fisher's theorem, from both behavior genetic and molecular genetic standpoints.

Summary Statements and Conclusion

By themselves, behavioral genetic studies can appear somewhat sterile. At one extreme, such studies often report the "usual moderate heritability," the absence of any meaningful shared environment (or at least the interpretable shared environment), much nonshared environmental and measurement error, and little beyond (see Turkheimer, 2000, for a formalization of this set of findings). But at the other extreme, they can identify processes underlying human behavior, suggest interesting and intricate genetic/environmental interactions, show interpretable gender and race differences, complement studies from other domains (such as developmental psychology, molecular genetics, etc.), and strongly inform our models of human behavior. One of the strongest values of behavior genetic modeling is philosophical; this approach has helped to break researchers out of the extreme and unhealthy tendency toward social determinism. As Plomin and Rende (1991) have noted, behavior genetic models can be as powerful for studying the environment as for studying genetic influences; in that sense, it is a misnomer to call this set of models and methods behavior "genetics."

This review has identified a number of coherent patterns across studies, which will be summarized here. Genetic influences are ubiquitous in the adolescent development process. That statement is not surprising in regard to primarily biological domains like pubertal development, hormones, and physical development. But, interestingly, the heritabilities for many social and behavioral processes are generally of the same magnitude as those for the more biological domains. This result strongly supports the position taken in Rodgers, Rowe, and Li (1994) with regard to studies of genetic or environmental influences: "Each type of influence can [i.e., should] be controlled in the study of the other" (p. 374). In no sense would behavior genetic findings of genetic influence obviate the importance of developing social models of adolescent development. But if those social models do not control for or otherwise account for the automatic similarity among related kin caused by shared genes, then the validity of those findings is threatened at a very fundamental level.

This review has also identified a few shared environmental influences, and many nonshared environmental influences. Turkheimer and Waldron (2000) evaluated the status of nonshared environmental influences, and expressed pessimism that we will identify specific influences that have much importance. Further, Turkheimer (1999) and Molenaar, Boomsma, and Dolan (1997) have shown the effects of failing to account for genetic/environmental interactions, which can bias the many estimates of heritability and shared environmental variance in standard biometrical models. The search is still on for the specific causes of the large portion of variance that behavior geneticists call "the nonshared environment."

We have also specified a number of gender differences, and other demographic subgroups show differences in genetic partitioning as well. In relation to sexual or reproductive behaviors, such differences are virtually axiomatic. In other domains, they can help identify useful treatment approaches (e.g., in relation to mental health), useful interventions (e.g., in relation to problem behaviors), or useful components of behavioral models in basic research.

We conclude with some comments about the three theoretical perspectives we have used repeatedly in past research to inform and organize our thinking: Transition Behavior Theory, the Life Course Perspective, and Problem Behavior Theory. Both Transition Behavior Theory and the Life Course Perspective suggest that there are social markers to which adolescents attend in their developmental process. Examples include starting high school, the first cigarette, puberty, initiation of sexual behavior, and beginning to drive. It seems clear that there is genetic variance underlying virtually all of the individual differences in these various behaviors. Some behavior and molecular genetic research reviewed above has even been able to evaluate whether there are shared genetic influences common to these different domains. In fact, we consider that bivariate and multivariate models showing the genetic and environmental overlap provide some of the most exciting and valuable models to apply to future kinship data using behavior genetic designs. A number of such models have been developed previously, and have provided valuable and exciting findings. Others will follow.

Adolescence provides a fascinating "age-graded laboratory" for the study of developmental processes. Behavior genetic methods have been fruitfully applied within this laboratory. The findings from those studies, and the way those findings interact with those outside the boundaries of behavior genetics, have and will continue to provide stimulating and valuable science.

Key Readings

Buster, M. A., & Rodgers, J. L. (2000). Genetic and environmental influences on alcohol use: DF analysis of NLSY kinship data. *Journal of Biosocial Science*, *32*, 177–189.

This article applies biometrical modeling to adolescent and young adult use of alcohol. DF analysis is a simple regression-based approach to estimating genetic and environmental variance components.

Jacobson, K. C., & Rowe, D. C. (1999). Genetic and environmental influences on the relationships between family connectedness, school connectedness, and adolescent depressed mood: Sex differences. *Developmental Psychology*, *35*(4), 926–939.

This article estimates biometrical models for adolescents relating family, school, and mental health.

McCartney, K., Harris, M. J., & Bernieri, F. (1990). Growing up and growing apart: A developmental meta-analysis of twin studies. *Psychological Bulletin*, *107*, 226–237.
This article is a meta-analysis showing how behavior genetic findings relating genetic and environmental influences to various outcomes must be conditioned on the age of the respondent. It motivates the study of behavior genetic patterns in adolescents as potentially different than for other age groups.

Plomin, R., & Rende, R. (1991). Human behavioral genetics. In M. R. Rosenzweig & L. W. Porter (Eds.), *Annual Review of Psychology* (Vol. 42, pp. 161–190). Palo Alto, CA: Annual Reviews.
This review article accounts broadly for biometrical/behavior genetic findings across many different domains and ages.

Rodgers, J. L., Rowe, D. C., & Li, C. (1994). Beyond nature versus nurture: DF analysis of nonshared influences on problem behaviors. *Developmental Psychology*, *30*(3), 374–384.
This article goes beyond the usual partitioning of influences in genetic and shared environmental influences – a model is defined to account explicitly for measured, nonshared environmental influences. The model is applied to the study of problem behaviors in older childhood and young adolescent respondents.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, *9*, 160–164.
This article presents some of the methodological and empirical difficulties of doing research on behavior genetics. The caveats that emerge should inform all behavior genetic modeling efforts.

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