Nature *and* Nurture: Genetic and Environmental Influences on Behavior

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The appropriate conjunction between the words *nature* and *nurture* is not *versus* but *and*. There is increasing acceptance of the evidence for substantial genetic influence on many behavioral traits, but the same research also provides the best available evidence for the importance of environmental influence and important clues about how the environment works. Because much developmental action is at the interface between genes and environment, genetic research needs to incorporate measures of the environment, and environmental research will be enhanced by collecting DNA.

Keywords: genetics; nature vs. nurture; twin studies; behavioral traits; DNA

After six hundred volumes of *The Annals of the American Academy of Political and Social Science* with hardly a mention of genetics, it is time to consider genetic as well as environmental influences on behavior. The theme of this review is that both genetics and environment, and the interplay between them, contribute importantly to the development of individual differences in behaviors including mental health and cognition. Quantitative genetic research—exemplified by the twin design that compares identical twins (monozygotic, MZ) and fraternal twins (dizygotic, DZ)—has gone beyond merely demonstrating the importance of genetic influence (heritability) to investigating more sophisticated issues such as developmental change and continuity, heterogeneity and comorbidity, and the interplay between genes and environment.

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Most exciting is the flood of molecular genetic research whose goal is to identify the specific DNA sequences responsible for genetic influence on common behavioral disorders such as mental illness and on complex behavioral dimensions such as personality. The latter are influenced by many DNA variants of small effect size, called quantitative trait loci (QTLs) (Plomin, Owen, and McGuffin 1994). The most important implication of this research for social scientists is that as multiple QTLs of small effect size for a particular trait are identified, they can be aggregated in a "QTL set" that can then be used as a genetic risk index in the same way that environmental risk indices such as socioeconomic status or education are used. Unlike quantitative genetic research that requires unique samples such as twins, molecular genetic research only requires DNA, which can be obtained painlessly and inexpensively using cheek swabs. We look forward to exciting advances in understanding the interplay between genes and environment once DNA is incorporated into social science research. Our hope for this article is that it facilitates this integration.

In the first part of the article, we describe quantitative genetic research, emphasizing what it has taught us about the way the environment works to affect behavioral development. The second part considers the future of genetic research, which lies with DNA. The brevity of this article does not permit us to discuss background issues (such as the focus on individual differences rather than population means), methodological issues (such as the twin method or methods for finding QTL associations), or to document thoroughly the research that supports our conclusions (for details, see Plomin et al. 2001).

The History of Nature and Nurture

The application of genetic research to human problems has experienced spectacular highs and lows over the decades since its inception in 1865. The study of nature and nurture in the development of behavioral traits began quietly with work from Francis Galton (1865). Pace and interest increased only gradually until, in 1924, the first twin and adoption studies in developmental psychology were published (Merriman 1924; Theis 1924). Events little more than a decade later complicated the course of progress. The slow but steady growth of genetic science was stopped abruptly by a world sickened by Nazi war crimes and all that was associated with them. The Nazi regime's abuse of genetics was high-profile and terrifying to a world in mourning, a world whose way of life had been threatened. The emergence of behaviorism around the same time (Watson 1930) proved a further roadblock in the development of genetic science, achieving a huge impact on the behavioral sciences with its ostensibly comforting theory of an environmental paradigm based on the assumption that we are what we learn.

Even from this poor midcentury soil, however, molecular genetic research managed to flourish, bursting onto the scientific stage in a Nobel Prize–winning blaze of glory with the discovery of the structure of DNA less than a decade after the end of World War II (Watson and Crick 1953). The genetic code was cracked in 1966,

the four-letter alphabet (G, A, T, C) of DNA creating the three-letter words that code for the twenty amino acids that are the building blocks of proteins. The crowning glory of the century and the beginning of the new millennium was the Human Genome Project's working draft of the sequence of the 3 billion letters of DNA in the human genome, nucleotide bases that are the steps in the spiral staircase of DNA (International Human Genome Sequencing Consortium 2001; Venter et al. 2001).

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During this time, genetic research on human behavior was slowly but steadily building a case for the importance of genetics as well as environment. The first textbook on behavioral genetics was only published in 1960, and it focused largely on research with nonhuman animals (Fuller and Thompson 1960). Since then, for nearly every area of behavior that has been studied, twin and adoption studies have shown strong genetic influence (Plomin et al. 2001). Genetic research has consistently shown heritable influence in many traditional areas of psychological research such as mental illness, personality, cognitive disabilities and abilities, and drug use and abuse. Some areas showing strong genetic influence may be more surprising, such as self-esteem, interests, attitudes, and school achievement.

This research has led to growing acceptance of roles for both genes and the environment in the etiology of individual differences in behavior. This shift can be seen in the growing number of genetics papers in mainstream behavioral journals and in funded research grants. The public also accepts a major contribution of genetics. For example, a recent poll found that more than 90 percent of parents and teachers reported genetics as being at least as important as the environment for mental illness, personality, learning difficulties, and intelligence (Walker and Plomin forthcoming). Before the pendulum of fashion shifts too far from nurture to nature, it is important to emphasize that this same genetic research provides the best available evidence for the importance of the environment. For most of these traits, the parents and teachers have it about right: genetics and environment each account for about half of the variance.

Consider schizophrenia. Until the 1960s, schizophrenia was thought to be environmental in origin, with theories putting the blame on poor parenting to account

for the fact that schizophrenia clearly runs in families. The idea that schizophrenia could run in families for genetic reasons was not seriously considered. Twin and adoption studies successfully changed this view. Twin studies showed that MZ twins are much more similar than DZ twins. This suggests genetic influence as MZ twins are genetically identical, like clones, whereas DZ twins, like nontwin siblings, are only 50 percent similar genetically. If one member of an MZ twin pair is schizophrenic, the chances are 45 percent that the other twin is also schizophrenic. For DZ twins, the chances are 17 percent. Adoption studies showed that the risk of schizophrenia is just as great when children are adopted away from their schizophrenic parents at birth as when children are reared by their schizophrenic parents, providing dramatic evidence for genetic transmission. There are now intense efforts to identify some of the specific genes responsible for genetic influence on schizophrenia (Craddock, O'Donovan, and Owen 2005; Plomin and McGuffin 2003).

Back in the 1960s, when schizophrenia was thought to be caused environmentally, it was important to emphasize the evidence for genetic influence, such as the concordance of 45 percent for identical twins. Now that genetic influence is widely recognized, it is important to emphasize that identical twins are *only* 45 percent concordant for schizophrenia, which means that in more than half of the cases, these pairs of genetic clones are discordant for schizophrenia. This discordance cannot be explained genetically—it must be due to environmental factors. Note that the word *environment* in genetic research really means *nongenetic*, a much broader definition of environment than is usually encountered in the behavioral sciences. That is, environment denotes all nonheritable factors including possible biological events such as prenatal and postnatal illnesses, not just psychosocial factors. Nonetheless, the point is that genetics often explains half of the variance of behavioral traits, but this means that the environment explains the other half.

In addition to providing strong evidence for the importance of environmental influence, two of the most important findings from genetic research involve nurture rather than nature: nonshared environment and what has been called the nature of nurture.

Nonshared environment. Recent research shows that the environment works very differently than we previously supposed. Theories of socialization have generally assumed that aspects of the environment such as socioeconomic status or parental divorce will, for better or worse, make children growing up in the same family similar to each other. Environmental research in genetically sensitive designs has consistently found a different pattern, namely, that the environments that affect behavioral development work by making children in the same family different (Plomin and Daniels 1987). We know this, for example, because genetically unrelated children growing up in the same adoptive family scarcely resemble each other for personality, psychopathology, and cognitive abilities after adolescence. Siblings are often similar, but their similarity is rooted in their genes rather than in the environment they share. Environment is hugely important to human development, but genetic research has shown beyond doubt that the most effective envi-

ronmental influences are those that operate to make children in the same family different, not similar. These environmental influences are called nonshared because they are not shared by children growing up in the same family.

So why are children growing up in the same family so different? Research over almost two decades has attempted to identify specific sources of nonshared environment, exploring differential parenting, friendships, health, and chance experiences in relation to a wide range of behaviors. So far, progress has been modest, with many environments showing significant but small effects (Plomin, Asbury, and Dunn 2001; Turkheimer and Waldron 2000). Although it is proving difficult to find specific nonshared environmental factors that account for large amounts of variance, it should be emphasized that nonshared environment is, in general, how the environment works to influence behavior. Although most research has focused on the family environment, it seems reasonable that experiences outside the family, with peers and individual life events, for example, might prove to be richer sources of nonshared environment (Harris 1998). It is also possible that chance contributes to nonshared environment in the sense of random noise, idiosyncratic experiences, or the subtle interplay of a concatenation of events. Compounded over time, small differences in experience might lead to large differences in outcome. Identifying the factors responsible for nonshared environment remains one of the big challenges for the future of this research.

The nature of nurture. Dozens of twin and adoption studies have shown that genetic factors substantially influence measures of behaviorally relevant environments such as parenting, stress, or social support (Plomin 1994), a phenomenon called "the nature of nurture" (Plomin 1994; Plomin and Bergeman 1991). How can this be true, given that environments have no DNA? The answer is that such environments can be considered as extended phenotypes, reflecting genetic differences between individuals as they select, modify, and construct their own experience of the world. In quantitative genetics, this phenomenon is known as genotype-environment (GE) correlation because it involves correlations between genetic propensities and exposures to environment (Bouchard et al. 1996).

Given that environmental measures as well as behavioral measures show genetic influence, it is reasonable to ask whether associations between environmental and behavioral measures are mediated genetically. Multivariate genetic analysis (discussed later) can be used to analyze genetic and environmental contributions to the correlation between environmental and behavioral measures. Genetic factors can mediate the correlation to the extent that the environment represents a direct response to genetically influenced characteristics. For example, differences in parenting could be the genetic result of child psychopathology, rather than its cause. A general guideline from multivariate genetic research of this sort is that genetic factors tend to be responsible for about half of the phenotypic correlation between measures of the environment and measures of behavior. For this reason, environmental measures cannot be assumed to be truly environmental. A far-reaching implication of this research supports a shift from thinking about passive models of how the environment affects individuals toward models that rec-

ognize the active role we play in selecting, modifying, constructing—and reconstructing in memory—our own experiences (Plomin 1994).

In quantitative genetics, another type of GE interplay is known as GE interaction, which refers to differential sensitivity to experiences, in contrast to GE correlation, which denotes differential exposure to experiences. For example, in psychopathology, a particular type of GE interaction has been examined, called *diathesis-stress* in which heritability is greater in high-risk environments (Asbury, Wachs, and Plomin forthcoming). GE interaction has been difficult to demonstrate in quantitative genetic analyses, in part because of lack of statistical power to detect an overall effect of GE interaction on the variance of a trait (Plomin, DeFries, and Fulker 1988; Wahlsten 1990). However, several recent examples of GE interaction have been reported in analyses using measured genes as well as measured environments (Caspi et al. 2002; Caspi et al. 2003; Harlaar et al. forthcoming). One example especially relevant to political and social science is that a functional polymorphism in the promoter region of the MAOA gene was shown to moderate the effect of child maltreatment on later antisocial behavior and violence (Caspi et al. 2002).

In short, genetic research has made some of the most important discoveries about the environment in recent decades, especially about nonshared environment and the role of genes in experience. More discoveries about environmental mechanisms can be predicted as the environment continues to be investigated in the context of genetically sensitive designs. Nonshared environment and the role of genes in experience are two examples of ways in which quantitative genetic research strategies are being used to go beyond merely asking about the relative importance of nature and nurture. Two other examples of going beyond heritability estimates are developmental analyses of change and continuity and multivariate analyses of heterogeneity and comorbidity.

Developmental change and continuity. Change in genetic effects from during one's development does not necessarily mean that genes are turned on and off during these stages, although this does happen. Genetic change simply means that genetic effects at one age differ from genetic effects at another age; that is, the same genes could have different effects in the brains of eight-year-olds and eighteen-year-olds. For example, developmental change in genetic effects is likely to be responsible for the fact that it is difficult to find behavioral markers in childhood for individuals who later become schizophrenic. Although it is possible that "schizophrenia genes" are not turned on until after adolescence, it is more likely that these genes operate the same way before and after adolescence but that they only express their hallucinatory and paranoid effects after adolescent brain development enables such highly symbolic processing.

One of the more striking findings of genetic change involves general cognitive ability, often called intelligence and assessed by IQ tests. The magnitude of genetic influence increases steadily from infancy to childhood to adolescence to adulthood (McGue et al. 1993). This is surprising because most people would think that environmental factors become increasingly important as experiences accumulate dur-

ing the life course. It is not known why the heritability of general cognitive ability increases during development. It is possible that more genes come into play during development, but it is also possible that the same genes have greater effects. This latter hypothesis receives support from longitudinal genetic research on age-to-age change and continuity that suggests that the same genes are largely responsible for genetic influence throughout development. If the same genes are involved, how can genetic influence increase? One possibility involves *genotype-environment correlation*, which, as mentioned earlier, refers to correlations between genetic propensities and exposure to experiences. That is, increasing heritability may occur because small genetic differences may snowball as we progress through life, creating environments that are correlated with our genetic propensities.

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Multivariate heterogeneity and comorbidity. Another important example in which genetic research is going beyond heritability is multivariate genetic analysis, which, as mentioned earlier, focuses on the covariance (correlation) between traits rather than the variance of each trait considered separately. It estimates the extent to which genetic factors that affect one trait also affect another trait. Multivariate genetic research in psychopathology suggests that genetic diagnoses of disorders often differ greatly from traditional diagnoses based on observable symptoms such as anxiety or depression. For example, several studies have shown that the same genes are largely responsible for anxiety and depression (Kendler et al. 1996). Recent research suggests that this genetic overlap among mental disorders is even broader in that genetic influences yield two broad domains of common psychopathology: internalizing problems that include anxiety, depression, and phobia; and externalizing problems that include antisocial behavior, conduct disorder, and drug abuse (Kendler et al. 2003).

Broad effects of genes have also been found in the cognitive domain. Despite the differences between cognitive abilities such as verbal, spatial, and memory abilities, the same genes largely affect all of these cognitive abilities (Petrill 1997). Genetic overlap is also substantial across learning disabilities such as language, reading, and mathematics disability, leading to a "generalist genes" theory of learning disabilities (Plomin and Kovas forthcoming). Finding such substantial genetic

overlap among cognitive abilities and disabilities has far-reaching implications for understanding the brain mechanisms that mediate these genetic effects (Plomin and Spinath 2002).

Sometimes multivariate genetic research suggests heterogeneity rather than comorbidity. An example of genetic heterogeneity that is especially relevant to the social sciences concerns psychopathic tendencies in childhood and antisocial behavior. Antisocial behavior in childhood and adolescence shows only modest genetic influence and, unlike most traits, it shows some shared environmental influence. However, antisocial behavior in the presence of callous-unemotional personality traits is highly heritable and shows no shared environmental influence (Viding et al. forthcoming). Multivariate genetic analyses support the hypothesis that the genetic core of early-onset antisocial behavior is callous-unemotional personality (Viding, Frick, and Plomin n.d.). Another recent example concerns behaviors characteristic of autistic spectrum disorder (ASD). Diagnoses of ASD require deficits in both social and nonsocial behaviors, but multivariate genetic analysis reveals marked genetic heterogeneity between the social and nonsocial ASD-related traits as assessed by teachers and by parents at seven years (Ronald, Happé, and Plomin forthcoming). In other words, different genes affect the social and nonsocial traits indicative of ASD.

The results of these multivariate genetic analyses have important implications not only for diagnosis but also possibly for treatment and prevention. Their most immediate implication is that these quantitative genetic findings chart the course for molecular genetic research that attempts to identify specific DNA variation responsible for genetic influence. For example, the research showing that callous-unemotional personality is the genetic core of psychopathic tendencies in childhood suggests that this should be the target for molecular genetic research rather than the broader phenotype of antisocial behavior.

DNA

The future of genetic research on behavior lies in molecular genetic studies of DNA that will eventually identify specific DNA variants responsible for the widespread influence of genes in behavioral development. Identifying these DNA variants will make it possible to address questions such as those raised above—about gene-environment, developmental, and multivariate mechanisms—with far greater precision and power. As compared to quantitative genetic studies of twins and adoptees, molecular genetics will have a far greater practical impact on behavioral research because molecular genetic research does not require special populations such as twins or adoptees. DNA can be easily and inexpensively obtained (from cheek swabs rather than blood, for about $10 per individual), and genotyping of a DNA marker is also inexpensive (about 10¢ per individual). Moreover, devices called gene chips (microarrays) are available that can genotype hundreds of thousands of genes for an individual in three days (Butcher et al. 2004).

Finding genes associated with complex traits is difficult and expensive, but using genes already identified is easy and inexpensive and can add a powerful genetic dimension to behavioral research (Plomin et al. 2003b). What has happened in the area of dementia in later life will be played out in many areas of behavioral research. The only known risk factor for common late-onset Alzheimer's disease (LOAD) is a gene, apolipoprotein E (APOE), involved in cholesterol transport. A form of the gene called allele 4 increases the risk fivefold for LOAD. Although the association between APOE allele 4 and LOAD was reported only a decade ago (Corder et al. 1993), much research on dementia now genotypes participants for APOE to ascertain whether results differ for individuals with and without this genetic risk factor (e.g., Laurin et al. 2004; Mukamal et al. 2003; Podewils et al. 2005). Genotyping APOE will also become routine in clinics if this genetic risk factor is found to predict differential response to interventions or treatments. Many large-scale behavioral studies are currently obtaining DNA on their samples in anticipation of the time when genes are identified that are relevant to their area of interest.

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It should be noted that DNA variation has a unique causal status in explaining behavior. When behavior is correlated with anything else, the old adage applies that correlation does not imply causation. For example, parenting is correlated with children's behavioral outcomes, but this does not necessarily mean that the parenting causes the outcome. Genetic research has shown that parenting behavior in part reflects genetic influences on children's behavior. When it comes to interpreting correlations between biology and behavior, such correlations are often mistakenly interpreted as if biology causes behavior. For example, correlations between neurotransmitter physiology and behavior or between neuroimaging indices of brain activation and behavior are often interpreted as if brain differences cause behavioral differences. However, these correlations do not necessarily imply causation because behavioral differences can cause brain differences. In contrast, in the case of correlations between DNA variants and behavior, the behavior of individuals does not change their genome. The DNA sequence itself does not change. For this reason, correlations between DNA differences and behavioral differences can be interpreted causally: DNA differences cause the behavioral differences.

There is no single human genome. We each have a unique genome because one in a thousand of DNA bases, 3 million bases of DNA, vary for at least 1 percent of the population. Most life scientists are interested in the generalities of the genome, but medical and behavioral scientists are more interested in the variations in the genome that are responsible for hereditary differences. Most of these DNA variants have already been identified and have made it possible to begin to attempt to find some of the genes that affect behavioral development, as in the example of the APOE gene that is a risk factor for dementia. Gene identification has been most successful for the thousands of rare single-gene disorders in which a mutation in a single gene is necessary and sufficient to cause a disorder. Many of these single-gene disorders have behavioral effects—for example, a recent review found that 282 of these disorders include cognitive effects among their symptoms (Inlow and Restifo 2004). However, such single-gene disorders are very rare, with frequencies of .0001 or less. The DNA revolution also provides tools to identify genes responsible for the heritability of common behavioral disorders and dimensions. These are usually called complex traits because they are likely to be influenced by multiple genes as well as by multiple environmental factors. Behavioral disorders such as schizophrenia, affective disorders, dementia, autism, reading disability, alcoholism, and hyperactivity are the target of much of this DNA research. Although some genes have been identified for these disorders, the process of identifying genes for complex traits in behavioral science as well as in medicine has been slower and more difficult than anticipated, probably because complex traits are influenced by many more genes of much smaller effect size than has been assumed (Plomin and McGuffin 2003).

The most far-reaching ramifications for behavioral research will come after these genes are identified. When sets of genes are identified that account for a useful portion of the variance in, say, learning disabilities, antisocial behavior, or schizophrenia, research will have a major impact on both the philosophy and practices of our schools, our courts, and our hospitals. They promise to provide etiological diagnoses, individualized treatment programs, and, most important, early prediction of problems that can lead to preventative interventions.

Behavioral science will be central to the new era of genetic research called the *postgenomic era* in which the focus will shift from finding genes to understanding how these genes work. Such postgenomic research is usually considered in relation to the bottom-up strategy of molecular biology in which a gene's product is identified by its DNA sequence and the function of the gene product is traced through cells and then cell systems and eventually the brain. Behavioral research lies at the other end of the continuum in the sense that behavioral research represents an integrationist top-down level of analysis that begins with the behavior of the whole organism rather than a reductionistic bottom-up level of analysis that begins with a single molecule in a single cell. For example, behavioral researchers can ask how the effects of specific genes unfold in development and how they interact and correlate with experience (Plomin et al. 2003a).

This top-down behavioral level of analysis has been called *behavioral genomics* to distinguish it from the often-used phrase *functional genomics* because the latter

phrase has become synonymous with bottom-up molecular biology. We suggest that behavioral genomic research is likely to pay off more quickly in prediction, diagnosis, and intervention, and eventually for behavioral preventions that use genes as early-warning systems. Bottom-up and top-down levels of analysis of gene-behavior pathways will eventually meet in the brain. The grandest implication is that DNA will serve as an integrating force across all of the life sciences, including the behavioral sciences.

Nature *and* Nurture

As discussed earlier, the importance of genes as well as environment in the etiology of individual differences in behavior is increasingly accepted in science as well as in society. We predict that this trend will accelerate in the postgenomic era as specific genes are found that are responsible for the heritability of behavioral disorders and dimensions. However, as is the case with most advances in science, these new findings also raise new problems for medicine, parenting, education, employment, law, and insurance, as well as larger philosophical issues such as human dignity, free will, and moral responsibility. These issues have been addressed recently in a report by the Nuffield Council on Bioethics (2002). The Nuffield report has three parts. The first explains the historical and scientific background to behavioral genetic research, which is also the topic of a book by Steven Pinker called *The Blank Slate* (2002). The second part reviews the findings of behavioral genetics, coming to similar conclusions to those described in this article. The third part is most relevant here: it examines the ethical, legal, and policy implications of this research. With the postgenomic era of DNA, the report is especially concerned about the need to monitor and regulate DNA tests and interventions and the possibilities for enhancing capabilities that could lead to even greater inequalities in society.

A general sense of unease about genetics comes from a feeling that genetic differences contradict equality: are not all men (and women) created equal? Although this was a self-evident truth to the signers of the American Declaration of Independence, the founding fathers of the United States were not so naïve as to think that all people are created identical. The essence of a democracy is that all people should have legal equality despite their individual differences, regardless of the environmental or genetic origins or those differences. Decisions, both good and bad, can be made with or without knowledge, but we believe firmly that better decisions can be made with knowledge than without. There is nothing to be gained by sticking our heads in the sand and pretending that genetic differences do not exist. The basic message of behavioral genetics is that each of us is an individual. Recognition of, and respect for, individual differences is essential to the ethic of individual worth. Proper attention to individual needs, including provision of the environmental circumstances that will optimize the development of each person, is a utopian ideal and no more attainable than other utopias. Nevertheless, we can approach this ideal more closely if we recognize, rather than ignore, individuality.

References

Asbury, K., T. Wachs, and R. Plomin. Forthcoming. Environmental moderators of genetic influence on verbal and nonverbal abilities in early childhood. *Intelligence.*

Bouchard T. J., Jr., D. T. Lykken, A. Tellegen, and M. McGue. 1996. Genes, drives, environment, and experience: EPD theory revisited. In *Intellectual talent: Psychometric and social issues*, ed. C. P. Benbow and D. Lubinski, 5-43. Baltimore: John Hopkins University Press.

Butcher, L. M., E. Meaburn, L. Liu, L. Hill, A. Al-Chalabi, R. Plomin, L. Schalkwyk, and I. W. Craig. 2004. Genotyping pooled DNA on microarrays: A systematic genome screen of thousands of SNPs in large samples to detect QTLs for complex traits. *Behavior Genetics* 34:549-55.

Caspi, A., J. McClay, T. E. Moffitt, J. Mill, J. Martin, I. W. Craig, A. Taylor, and R. Poulton. 2002. Role of genotype in the cycle of violence in maltreated children. *Science* 297:851-54.

Caspi, A., K. Sugden, T. E. Moffitt, A. Taylor, I. W. Craig, H. Harrington, et al. 2003. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science* 301:386-89.

Corder, E. H., A. M. Saunders, W. J. Strittmatter, D. E. Schmechel, P. C. Gaskell, G. W. Small, A. D. Roses, J. L. Haines, and M. A. Pericak Vance. 1993. Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science* 261:921-23.

Craddock, N., M. C. O'Donovan, and M. J. Owen. 2005. The genetics of schizophrenia and bipolar disorder: Dissecting psychosis. *Journal of Medical Genetics* 42:193-204.

Fuller, J. L., and W. R. Thompson. 1960. *Behavior genetics*. New York: Wiley.

Galton, F. 1865. Heredity talent and character. *Macmillan's Magazine* 12:157-66, 318-27.

Harlaar, N., L. Butcher, E. Meaburn, I. W. Craig, and R. Plomin. Forthcoming. A behavioural genomic analysis of DNA markers associated with general cognitive ability in 7-year-olds. *Journal of Child Psychology and Psychiatry*.

Harris, J. R. 1998. *The nurture assumption: Why children turn out the way they do*. New York: Free Press.

Inlow, J. K., and L. L. Restifo. 2004. Molecular and comparative genetics of mental retardation. *Genetics* 166:835-81.

International Human Genome Sequencing Consortium. 2001. Initial sequencing and analysis of the human genome. *Nature* 409:860-921.

Kendler, K. S., M. C. Neale, C. A. Prescott, R. C. Kessler, A. C. Heath, L. A. Corley, and L. J. Eaves. 1996. Childhood parental loss and alcoholism in women: A casual analysis using twin-family design. *Psychological Medicine* 26:79-95.

Kendler, K. S., C. A. Prescott, J. Myers, and M. C. Neale. 2003. The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry* 60:929-37.

Laurin, D., K. H. Masaki, D. J. Foley, L. R. White, and L. J. Launer. 2004. Midlife dietary intake of antioxidants and risk of late-life incident dementia: The Honolulu-Asia Aging Study. *American Journal of Epidemiology* 159:959-67.

McGue M., T. J. Bouchard Jr., W. G. Iacono, and D. T. Lykken. 1993. Behavioral genetics of cognitive ability: A life-span perspective. In *Nature, nurture, and psychology*, ed. R. Plomin and G. E. McClearn, 59-76. Washington, DC: American Psychological Association.

Merriman, C. 1924. The intellectual resemblance of twins. *Psychological Monographs* 33:1-58.

Mukamal, K. J., L. H. Kuller, A. L. Fitzpatrick, W. T. Longstreth Jr., M. A. Mittleman, and D. S. Siscovick. 2003. Prospective study of alcohol consumption and risk of dementia in older adults. *JAMA* 289:1405-13.

Nuffield Council on Bioethics. 2002. *Genetics and human behaviour: The ethical context* London: Nuffield Council on Bioethics.

Petrill, S. A. 1997. Molarity versus modularity of cognitive functioning? A behavioral genetic perspective. *Current Directions in Psychological Science* 6:96-99.

Pinker, S. 2002. *The blank slate: The modern denial of human nature.* New York: Penguin.

Plomin, R. 1994. *Genetics and experience: The interplay between nature and nurture*. Thousand Oaks, CA: Sage.

Plomin, R., K. Asbury, and J. Dunn. 2001. Why are children in the same family so different? Nonshared environment a decade later. *Canadian Journal of Psychiatry* 46:225-33.

Plomin, R., and C. S. Bergeman. 1991. The nature of nurture: Genetic influences on "environmental" measures. *Behavioral and Brain Sciences* 14:373-427.

Plomin, R., and D. Daniels. 1987. Why are children in the same family so different from each other? *Behavioral and Brain Sciences* 10:1-16.

Plomin, R., J. C. DeFries, I. W. Craig, and P. McGuffin. 2003a. *Behavioral genetics in the postgenomic era*. Washington, DC: American Psychological Association.

———. 2003b. Behavioral genomics. In *Behavioral genetics in the postgenomic era*, ed. R. Plomin, J. C. DeFries, I. W. Craig, and P. McGuffin, 531-40. Washington, DC: American Psychological Association.

Plomin, R., J. C. DeFries, and D. W. Fulker. 1988. *Nature and nurture during infancy and early childhood*. Cambridge, UK: Cambridge University Press.

Plomin, R., J. C. DeFries, G. E. McClearn, and P. McGuffin. 2001. *Behavioral genetics*. 4th ed. New York: Worth Publishers.

Plomin, R., and Y. Kovas. Forthcoming. Generalist genes and learning disabilities. *Psychological Bulletin*.

Plomin, R., and P. McGuffin. 2003. Psychopathology in the postgenomic era. *Annual Review of Psychology* 54:205-28.

Plomin, R., M. J. Owen, and P. McGuffin. 1994. The genetic basis of complex human behaviors. *Science* 264:1733-39.

Plomin, R., and F. M. Spinath. 2002. Genetics and general cognitive ability (*g*). *Trends in Cognitive Science* 6:169-76.

Podewils, L. J., E. Guallar, L. H. Kuller, L. P. Fried, O. L. Lopez, M. Carlson, and C. G. Lyketsos. 2005. Physical activity, APOE genotype, and dementia risk: Findings from the Cardiovascular Health Cognition Study. *American Journal of Epidemiology* 161:639-51.

Ronald, A., F. Happé, and R. Plomin. Forthcoming. The genetic relationship between individual differences in social and nonsocial behaviors characteristic of autism. *Developmental Science*.

Theis, S. V. S. 1924. *How foster children turn out*. Publication no. 165. New York: State Charities Aid Association.

Turkheimer, E., and M. Waldron. 2000. Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin* 126:78-108.

Venter, J. C., et al. 2001. The sequence of the human genome. *Science* 291:1304-51.

Viding, E., R. J. R. Blair, T. E. Moffitt, and R. Plomin. Forthcoming. Evidence for substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry*.

Viding, E., P. J. Frick, and R. Plomin. N.d. Genetic influences on the relationship between callous-unemotional traits and conduct problems in 7-year-old twins. Manuscript submitted for publication.

Wahlsten, D. 1990. Insensitivity of the analysis of variance to heredity-environment interaction. *Behavioral and Brain Sciences* 13:109-61.

Walker, S. O., and R. Plomin. Forthcoming. The nature-nurture question: Teachers' perceptions of how genes and the environment influence educationally relevant behavior. *Educational Psychology*.

Watson, J. B. 1930. *Behaviorism*. New York: Norton.

Watson, J. D., and F. H. C. Crick. 1953. Genetical implications of the structure of deoxyribonucleic acid. *Nature* 171:964-67.