Nature–Nurture Integration

The Example of Antisocial Behavior

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Nature and nurture do not operate independently of each other, and, to an important degree, genetic effects on behavior come about because they either influence the extent to which the individual is likely to be exposed to individual differences in environmental risk or they affect how susceptible the individual is to environmental adversities. The time has come for an explicit research focus on the forms of interplay between genes and environment and on how this interplay is involved in the causal mechanisms for the origins of antisocial behavior and for its persistence or desistance over time. Molecular genetics has an even greater potential than quantitative genetics for understanding environmental risk mechanisms and the interplay between nature and nurture.

The very terms *nature* and *nurture* imply a polarity. Not surprisingly, therefore, much research in the field of quantitative genetics has focused on measuring the strength of the contributions of genetic and nongenetic (or environmental) influences on individual differences with respect to a wide range of traits and disorders (Plomin, DeFries, McClearn, & Rutter, 1997). The findings have been important in showing the reality of genetic effects on population variance regarding almost all human behavior. In general, genetic influences have usually accounted for some 20% to 60% of the variance.[1] In other words, the effects of genetics on human behavior are relatively strong but far from overwhelming. The genetic evidence has been as important in pointing to environmental influences on behavior as it has been in its demonstration of genetic effects: Environmentality and heritability are both strong (Plomin, 1994; Plomin et al., 1997).

Although some genetic and environmental influences have effects on behavior that are relatively direct, in many instances, the consequences depend on a more complex interplay between nature and nurture. The purpose of this article is to discuss some of the primary ways in which this interplay comes about, using antisocial behavior as the example. First, there is a brief discussion of some key genetic concepts: the meaning and implications of heritability, the implications for the heterogeneity of behavior, and the possible routes for genetic effects on social behaviors. Second, there is a parallel consideration of key environmental concepts: individual variations in exposure to risk environments, the ways in which genetic factors influence what seem to be environmental effects, the finding that most environmental factors are relatively individual-specific, and the effects of the environment on the organism.

In the third section of the article, nature–nurture interplay is discussed in relation to the three main ways in which genetic factors influence people's exposure to different environments: passive, evocative, and active gene–environment correlations. The fourth section considers the role of genetic factors as influences on people's susceptibility to different environments—gene–environment interactions. The article ends with a call for a shift from a focus on the relative strength of nature and nurture effects on behavior to a concern to understand how the interplay between the two comes about and how the interplay affects behavior.

Genetic Concepts

At one time, some geneticists were inclined to use genetic findings as a basis for arguing both that the strength of genetic effects means that environmental interventions are likely to be ineffective and that, because genetic factors play a major role in variations within groups of people, they are likely to be responsible for differences between groups (Herrnstein & Murray, 1994; Jensen,

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[1]Developmentalists have sometimes argued that the heritability figure is an overestimate because it includes gene–environment covariation and interaction within genetic effects and because most twin and adoptee samples underrepresent families presenting the greatest environmental risk (Baumrind, 1993). The concerns are correct, but the figure will also be an underestimate because error is included in the nonshared environmental term and because most heritability figures do not take lifetime liability adequately into account. Rather than argue about the details of the estimates, it is more appropriate to accept them as reasonable "ball park" figures and instead focus on their implications (as discussed here).

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1969). Thus, it was argued that the finding that the average IQ of African Americans tends to be lower than that of Whites was likely to have a genetic basis and that compensatory education would be ineffective because the heritability of IQ was relatively high. Both arguments are fallacious (Block, 1995; Morton, 1974). Heritability estimates are necessarily specific to the particular time and to the sample from which the measures were collected. Heritability estimates describe only what is the case now and have no implications for what could, or would, happen if circumstances changed. Even very high heritability estimates do not imply that changes in the environment cannot bring about big effects (Dennett, 1995; Tizard, 1975). Height provides a telling example. It constitutes one of the most strongly genetically influenced of human characteristics, but, nevertheless, there has been a massive rise in average height over the course of this century—some 12 cm in London schoolboys over the first 50 years. Almost certainly, this rise has been brought about by improved nutrition. This has made little difference to population variation in height, but it has had a major effect on the absolute level of height in the population.

The example of height is of very direct relevance to antisocial behavior. An increasing amount of empirical research has shown the importance of genetic factors for individual differences in antisocial behavior (Bock & Goode, 1996; Carey, 1994). It is extremely important that social scientists and clinical practitioners appreciate the reality of genetic influences on antisocial behavior. But it is equally important that geneticists accept that individual differences do not constitute the only important causal question about a behavioral phenomenon (Rutter & Smith, 1995). Over the last 50 years, crime rates have increased some fivefold or more in most industrialized Western countries; clearly, that rise has to be attributed to environmental influences because the gene pool cannot change that quickly. Similarly, genetic factors cannot be invoked to account for the fact that the homicide rate in the United States is some 15 times that in any European country. The finding that the difference between the United States and Europe is largely restricted to gun killings obviously points to the huge differences in gun control between these countries as the most plausible explanation (Snyder, Sickmund, & Poe-Yamagata, 1996).

It has gradually come to be accepted that the precise quantification of heritability has little value because it provides no unambiguous implications for theory, policy, or practice. Moreover, the time has passed when there can be any interest in yet another behavioral trait showing an important genetic component. We need to appreciate that all human behavior is based on biology and, hence, will involve some degree of genetic influence. But, equally, all social behavior is bound to be affected by social context and, hence, will involve an important environmental influence. There is little to be gained by merely quantifying the relative importance of the contributions of genetic and environmental influences because any estimates will be specific to the population studied and will be subject to change if environmental circumstances alter.

Quantification of heritability, nevertheless, is of considerable importance with respect to its implications for heterogeneity, not only within the field of antisocial behavior but also within other psychopathological domains. Thus, for example, genetic studies have been important in demonstrating that antisocial behavior associated with early onset hyperactivity–inattention, poor peer relationships, and widespread social malfunction has a strong genetic component, whereas antisocial behavior without these accompanying problems is largely environmental in origin (Rutter, Maughan, et al., in press; Silberg et al., 1996). Equally, genetic findings have been important in showing that (contrary to popular views) there is a much weaker genetic component in violent crime than in petty theft (Bock & Goode, 1996; Carey, 1994). Also, such findings have been informative in showing that the genetic component is greater in the case of antisocial behavior that persists into adult life than in antisocial behavior confined to the teenage period (DiLalla & Gottesman, 1989; Lyons et al., 1995; Rutter, Maughan, et al., in press).

Advances in genetic research have brought about another shift in concept, namely that from a focus on the genetic causes of a discrete categorical disorder to a focus on genetic contributions to the various dimensional risk factors involved in the multifactorial origins of psychopathology (Rutter, 1994b). Regrettably, there is still media hype about a possible "gene for crime." There is not likely to be any gene for crime for a number of reasons: (a) Genes do not code for (i.e., have direct effects on) complex social behaviors (rather, genes code for protein products that are likely to have only indirect associations with socially defined behaviors); (b) the evidence strongly suggests that genetic influences on antisocial behavior affect the probability that such behavior will occur rather than determine it directly; (c) it is likely that multiple genes are involved; and (d) it is probable that the genetic influences affect susceptibility factors for antisocial behavior rather than crime as such (Bock & Goode, 1996; Rutter, Maughan, et al., in press). Thus, research findings show that quite a wide range of features are associated with an increased risk of antisocial behavior (Farrington, 1995; Rutter, Maughan, et al., in press; Stoff, Breiling, & Maser, in press). These features include hyperactivity–inattention, novelty- or sensation-seeking, impulsivity, low physiological reactivity, and cognitive impairment, to mention just a few. The probability is that any genetic influences that are important will operate through these personality, physiological, and cognitive features rather than on any specific tendency to commit crimes.

Environmental Effects

Variations in Environmental Risk Exposure

There have been parallel, equally important developments in the understanding of how environmental influences

operate on behavior. Four advances warrant special mention. First, individuals differ enormously in their exposure to risk environments (Rutter, Champion, Quinton, Maughan, & Pickles, 1995). Life stressors and adversities do not occur in random fashion; rather, they are determined by both societal forces and by the ways in which individuals shape and select their own environments (Scarr, 1992). Any understanding of environmental risk processes must incorporate an appreciation of the origins of the environmental risk situations.

Genetic Influences on Supposedly Environmental Effects

Second, genetic research designs have shown that some of the effects of psychosocial adversities are genetically, rather than environmentally, mediated (Plomin & Bergeman, 1991). Labeling a variable *environmental* does not mean that its effects are environmentally mediated. Thus, from the child's perspective, family discord or parental divorce is obviously an adverse experience. Nevertheless, insofar as the disrupted family relationships derive from the behavior of parents, the effects on the children may stem from the genetic transmission of parental traits that predispose them to both conflict and antisocial behavior rather than from the environmental effects of conflict. The finding is important, but it is necessary to recognize that three very different mechanisms may be involved.

Apparent effects of an adverse environment may be purely methodological. Thus, if conflict is rated by the children, it could simply reflect perceptual biases brought about by the genetically influenced personality characteristics of the child. In those circumstances, the supposed environmental risk does not exist and it is purely an artifact of measurement. Although the possibility of there being a measurement artifact must always be kept in mind, the empirical findings suggest that such perceptual biases in reporting are usually minor (Maughan & Rutter, in press). And second, the mechanism may concern the *origins* of the risk factor but not the way the risk is mediated. It is commonly assumed (both conceptually and in behavior genetic statistical analyses) that the two are the same, but in fact they have no logical or necessary connection with one another (Rutter, Silberg, & Simonoff, 1993). Thus, the reasons why people smoke (personality characteristics, social influences, and availability of cigarettes) have nothing to do with the carcinogenic effects of smoking on lung cancer or the biological effects on risk of coronary artery disease or osteoporosis. It is important to appreciate that most genetic analyses fail to recognize or assess this distinction between origins of a risk factor and the way the risk is mediated. Finally, the mechanism may actually reflect the mode of mediation of risk. In this circumstance, inferring that there is an environmental effect is truly erroneous.

The third advance in understanding environmental effects is the recognition that usually nonshared (or individual-specific) environmental effects are greater than shared ones (Plomin & Daniels, 1987). In other words, most environmental influences impinge differently on each individual and, so, tend to make children in the same family different rather than similar. There is no doubt that this finding is of immense importance both with respect to its conceptual implications and to its messages for research designs. Unfortunately, this finding on individual-specific effects has been widely misinterpreted as meaning that familywide influences have little effect (e.g., Rowe, 1994). That is an error. The evidence refers to an *inferred* effect (albeit a strongly based inference) and not to a measured *variable*. Thus, for example, it does not mean that family discord, conflict, or disruption has no effects on the children. Rather, it implies that the effects are not shared equally among the children. Children vary in the extent to which they are scapegoated or embroiled in the family conflict, and they vary in their susceptibility to this type of environmental hazard. The *variable* is shared but the *effects* are nonshared. The finding that children in the same family differ in how they are influenced by environmental risks is also sometimes assumed to mean that nonfamilial influences are more important than familial ones. That, too, is a misconception. As previously mentioned, family influences may bring about nonshared effects. Also, however, nonfamily influences may bring about shared effects. Thus, siblings (and most especially twins) may share peer groups, and, insofar as they do, the effects may be shared rather than nonshared.

It should be added, however, that antisocial behavior constitutes a partial exception to the generalization that nonshared effects predominate over shared ones (see DiLalla & Gottesman, 1989; Silberg et al., 1996). It is very common for several children in the same family to show antisocial behavior, and the evidence suggests that to an important extent this stems from environmental influences. The reasons why shared effects are greater in the case of antisocial behavior than for other behaviors are not well understood but two main possibilities stand out. First, delinquent behavior in adolescence is essentially a group activity. Accordingly, one may expect both that some antisocial acts will be committed by siblings operating together (Rowe, Rodgers, & Meseck-Bushey, 1992) and that peer group influences will be particularly important (Quinton, Pickles, Maughan, & Rutter, 1993; Rowe, Woulbroun, & Gulley, 1994). The latter may operate as a nonshared effect with siblings of a different age but as a shared effect with twins (who necessarily are of the same age).

The fourth advance is the appreciation that the changes that environmental effects bring about in the organism need to be considered (Rutter, 1989a; Rutter & Rutter, 1993). In many ways, this is the most neglected of all issues in the field of psychosocial research; yet it is crucial for any appreciation of why and under what circumstances effects persist over time. Attention has begun to be paid to neuroendocrine alterations, to mental models, to styles of cognitive processing, and to patterns of interpersonal interaction. However, research in this arena is still in its infancy, and little is known as yet on

the mechanisms involved in the persistence, or nonpersistence, of environmental effects.

But most of all, research findings have been crucial in bringing about an appreciation that nature and nurture are not, in reality, separate. Instead of focusing on quantifying the relative strengths of their contributions, it is necessary to focus on the interplay between genes and environment, on how each operate, on how they influence one another, and on the nature of the causal mechanisms that may be both genetic and environmental.

Gene–Environment Correlations or Covariance

Gene–environment correlations refer to situations in which variations in genetic liability are systematically associated with variations in specific environmental circumstances (Rutter, Dunn, et al., in press). That is, they refer to the variety of ways in which genetic factors influence people's exposure to different environments. Such gene–environment correlations are to be expected because exposure to particular environments may depend on the genotypes of the parents or of siblings making up the social environment or on the ways in which each genetically unique individual adapts and responds to his or her environment. Three main varieties occur (Plomin et al., 1997). First, there are the *passive* correlations that arise because parental genes, insofar as they influence parental behavior, also influence the child's experiences. Second, there are *evocative* correlations that derive from the effect of one person's characteristics in eliciting particular responses in other people. Third, there are *active* correlations that arise from the processes by which individuals shape and select their environments.

Passive Gene–Environment Correlations

The effects of passive gene–environment correlations have been subject to surprisingly little study despite the strong evidence that they are likely to be important. Thus, parental criminality is the best risk indicator for antisocial behavior in the offspring (Farrington, 1995), and parental personality disorder has been found to be associated with a huge increase in the rate of family discord and children's exposure to hostile behavior (Rutter & Quinton, 1984). In addition, there are numerous other ways in which parental mental disorder creates environmental risks for the children—starting in the womb with the effects of alcohol on the neural development of the fetus and extending to later parental neglect or breakdowns in parenting (Rutter, 1989b). It may be inferred that parental personality disorder–criminality carries both genetic and environmental risks for the children. What remains quite uncertain, however, is the relative importance of genetic and of environmental risk mediation. Both are likely to be influential.

It should be appreciated, however, that the psychopathological risks to the children stemming from parental disorder may be environmentally mediated despite the genetic component in the parental disorder. Extended twin-family designs provide one means of separating genetic from shared environmental effects in this situation (Kendler et al., 1996; Meyer et al., 1997). Shared environmental effects can be examined by the introduction of measures of the postulated environmental factor into a multivariate model. The multivariate model needs to take into account the correlation between the phenotypes (i.e., the behavioral expressions of the genes) of parent and child, when assessed for a comparable age period. Using this approach, Meyer et al. were able to show some environmental mediation for a lack of family adaptability and cohesion that was child-focused. Despite the strength of this research strategy, there are three important limitations: (a) It assesses only shared environmental effects; (b) it ignores genetic variations in susceptibility to the environmental risk factor; and (c) it underestimates environmental effects that are brought about by a genetically influenced environmental factor, because the causal path is still environmental even though it is genetically driven.

Nonshared environmental effects can be assessed through the examination of within-pair differences within monozygotic (MZ) pairs. This is because any difference must be nongenetic in origin. Although the approach is conceptually sound, it has three important limitations, and very little use has been made of the strategy with respect to antisocial behavior. First, pairs of MZ twins probably vary much less in their experiences than singleton siblings, because they are of the same age and because they will shape and select their experiences in a similar way. Second, even when parents treat MZ twins differently, they frequently have difficulty in recognizing and reporting that they are doing so. The use of child ratings of differences in treatment may be more satisfactory, but the research reward so far has been quite limited. Third, in the (common) circumstance that a shared family experience impinges on the children in different ways, there are, as yet, inadequate measures of the impact on the children of these differences in treatment.

Blended family designs (which compare unrelated children being reared in the same family with half-siblings, step-siblings, and full siblings) are potentially useful for this purpose. Thus, Reiss et al. (1995) showed that the risks of parental conflict–negativity for antisocial behavior in the offspring arose through child-focused negative feelings. The results reported so far, however, have not distinguished child effects on the family from family effects on the child.

Adoptee studies provide another means of studying environmental effects in the context of passive gene–environment correlations. This line of research is powerful because of the fact that the parental genes that shape the parental behavior creating the family environment are not passed onto the children. Accordingly, the effect must be environmentally mediated (provided that any selective placement of the child has been taken into account). Unfortunately, the strategy has four serious practical limitations: (a) Adoptive parents are usually selected because they appear free of serious psychopathology, so that risks in the environment are likely to be less frequent than in

the general population; (b) very few children today are adopted in infancy, so that adopted children are a much more atypical population than was the case a generation ago; (c) confidentiality rules make it very difficult to have access to adoptee samples in most societies; and (d) unless longitudinal data are available, it may be difficult to differentiate parental effects on the child from child effects on the parents.

It is evident that much has still to be learned about the effects of passive gene–environment correlations. Quite often, both geneticists and psychosocial researchers have side-stepped the need to investigate them; remedying this serious lack of attention to gene–environment correlations constitutes an important research priority.

Evocative Gene–Environment Correlations

There is abundant evidence that a person's characteristics shape other people's reactions to them (Engfer, Walper, & Rutter, 1994). That is, it is well established that there are major evocative person–environment correlations (Rutter, Dunn, et al., in press). The extent to which the person characteristics involved in these correlations are genetically influenced, however, is not known, neither is the extent to which the correlations play a role in the origins or persistence of antisocial behavior (or other forms of psychopathology) in the children.

Three main research designs have been used to examine evocative person–environment correlations in the field of antisocial behavior. First, there have been experimental studies. Thus, Brunk and Henggeler (1984) trained 10-year-old children to behave in anxious–dependent or oppositional ways. The children were then placed with adults who were expected to engage them in particular tasks. The findings showed that children who behaved oppositionally elicited much more disciplinary interventions even when they were being compliant. This finding established that reputations were already being established and were having effects on the adults.

A second, quasi-experimental strategy has used the design of comparing mother–child interactions in dyads in which the child either did or did not have conduct disorder (Anderson, Lytton, & Romney, 1986). Three dyads were compared: (a) mothers interacting with their own child, (b) mothers interacting with someone else's child with similar characteristics (i.e., with or without conduct disorder), and (c) mothers interacting with someone else's child with dissimilar characteristics. The findings showed substantial child and maternal effects.

Third, naturalistic longitudinal designs have shown the effects of negative child characteristics on patterns of family interactions. Thus, Lee and Bates (1985) showed that infants with difficult temperamental characteristics were more likely to elicit repeated parental prohibitions resulting in conflict. Similarly, Rutter (1978) found that older children with difficult temperamental features were more likely to be the focus of parental criticism, hostility, and scapegoating. One of the very few studies to examine the consequences of these effects was that with preschool children undertaken by Martin, Maccoby, and Jacklin (1981). Using a longitudinal design, they showed the operation of a vicious cycle in boys (although not in girls) by which oppositional behavior tended to lead parents to back away from confrontations, and, when this happened, the backing away served to perpetuate the persistence of the oppositional behavior. The important implication of this observation is that although the vicious cycle was started by the child's behavior, the parental response influenced the persistence of the same behavior. This constitutes another example of the error involved in partitioning variance in a single cross-sectional analysis; it is an error because the origins of a risk factor and the way in which it is mediated are not synonymous.

These examples all concern the impact of children's behavior on their parents or other adults. The same considerations apply to interactions among siblings and peers. One child's genotype may shape the environment of his or her brothers and sisters through either contrast or assimilation effects. Thus, one child's dominance or exuberance may cause the sibling to be more submissive or constrained. Conversely, the interaction may serve to bring siblings closer together, as seems to happen with MZ twins (Segal, 1984) and perhaps through the delinquent activities of brothers (Rowe et al., 1992). Children's negative attributes may serve to make them unpopular or rejected by their peer group, and this, in turn, may affect their own later development (Parker & Asher, 1987). The point is that the existence of these evocative effects requires a focus on the operation of dynamic processes as they occur over time, rather than the mere quantifying of genetic and environmental components as if they were separate.

Active Gene–Environment Correlations

The third type of gene–environment correlation is termed *active*. This refers to the process by which individuals select and shape their experiences. As with evocative correlations, there is strong evidence that people do influence their environments, but there is little evidence available on the extent to which genetic factors operate through that route.

The strength of the effects of individual behavior on environmental risk exposure is well brought out by the findings of long-term longitudinal studies. An early example was provided by Robins's (1966) follow-up into middle adulthood of antisocial boys who had attended a Child Guidance Clinic when young. When in their 30s and 40s, they were more likely than control boys living in the same neighborhood to have been divorced more than once, to be unemployed, to have had at least 10 job changes in a decade, to be in a low-level job, and to be practically without friends. In a real sense, these adversities reflected their continuing personality problems. However, equally, they represented the particular environmental stressors known to increase the risk of psychopathology. Similarly, Champion, Goodall, and Rutter (1995), in their follow-up of socially disadvantaged London 10-year-olds, found that those showing antisocial behavior

were more than twice as likely as controls to experience severely negative acute and chronic life events and difficulties some two decades later. Quinton et al. (1993), in a separate set of long-term follow-up studies of high-risk groups showing antisocial problems, found a strong tendency in adult life for antisocial individuals to marry, or have children by, a partner who also exhibited crime, drug, or alcohol problems. There is much evidence that antisocial individuals act in ways that greatly increase the likelihood that they will experience high-risk environments in adult life.

The key issue is whether these high-risk environments play a major or minor role in the persistence, or desistance, of antisocial behavior over time. Although genetically sensitive designs play a key role in research to tackle this issue, these strategies are insufficient in themselves because the origins of a risk factor and the way it is mediated have no necessary connection with one another. The solution lies in the strategy of investigating within-individual change over time in relation to defined and measured environmental risk experiences (Rutter, 1994a, 1996; Rutter, Maughan, et al., in press; Rutter & Pickles, 1991; Sampson & Laub, 1993). The essential requirement for this research approach is to ensure that the supposed change is not a reflection of the person's own prior behavior or circumstances. That strategy necessitates good quality, broad-ranging, multisource measures of both individual characteristics and environmental circumstances. These measures need to be obtained longitudinally and need to cover the period both before and after the postulated environmental risk experience. In addition, this research approach requires the use of statistical methods that can take into account unmeasured aspects of the underlying constructs (such as by the use of latent variable methods or by determining whether the error terms of the person's prior behavior and the environmental risk being studied are intercorrelated). The studies that have met these criteria are definite in showing that, for example, marital quality, unemployment, and alcohol abuse all have a substantial effect on the persistence of antisocial behavior in adult life even after prior behavior and circumstances have been taken into account (Quinton et al., 1993; Rutter, Maughan, et al., in press; Sampson & Laub, 1993). Very little is known on the role of genetic factors in these indirect chain effects. Also, there is very little understanding of the proximal processes by which this nature–nurture interplay operates over time. The elucidation of these indirect chain effects needs to be one of the top research priorities in the field of psychopathology. This is not only because of their theoretical importance but, even more crucially, because understanding is essential if findings on genetic and environmental effects are to be used to improve policy and practice designed to prevent or alleviate psychopathology.

Gene–Environment Interaction

A key feature of evolutionary concepts is that whenever an organism has the capacity to respond adaptively to environmental change (either by responding to it or ignoring it), genetic differences in sensitivity to particular environmental changes are to be expected (Dennett, 1995). That is, the genetic liability operates through its role in making the person unusually vulnerable to particular types of environmental stressors or adversities—a gene–environment interaction. Behavior geneticists have sometimes assumed that this is the same as a significant statistical interaction term in a multivariate analysis of genetic and environmental factors. It is not. A significant statistical interaction term is an indication of gene–environment interaction, but, for reasons that are conceptual, statistical, and genetic, such a term will detect only certain sorts of genetic influences on susceptibility to specific environmental factors (Kendler & Eaves, 1986; Mather & Jinks, 1982; Rutter, 1983; Rutter & Pickles, 1991; Wahlsten, 1990). In particular, statistical interaction effects require variation in both the environment and the genetic background. Accordingly, they will not detect genetic susceptibilities to environmental factors that are all-pervasive (as is obviously the case with many allergens and dietary influences and may well be, too, with many psychosocial influences). Also, they have very weak power for detecting genetic susceptibility to environmental risks that apply only to subsegments of the population.

Behavior geneticists have also stressed the distinction between gene–environment correlations and gene–environment interactions. The distinction provides a convenient framework for research, but it is necessary to recognize that the same genetic factor may provide the basis for both effects. Moreover, whether it results in a correlation or interaction is likely to depend on the environmental context prevailing at the time. A particularly vivid illustration that this is so is provided by the different species of Darwin's finches on the Galapagos Islands (Grant, 1986; see also Weiner, 1994, for an excellent updated nontechnical account of Grant's research). Ordinarily, the genetic differences among species are shown by the variations in the distribution of the different species among the islands (which differ markedly in their features). The genetically different species are mainly (but not exclusively) to be found in the environments that suit them best. This is a gene–environment correlation. After a period of prolonged drought, however, there was a marked difference among species in their mortality. The precise features that led to the geographical variations were also the ones that led to differential susceptibilities to the altered feeding opportunities in the absence of rain—a gene–environment interaction. The need in all cases is to determine the underlying biological processes. The statistical terms are important only with respect to their usefulness as a first step in that direction. This example may seem a long way from antisocial behavior but the principle is a general one. The focus of research needs to be on the relevant processes and not on statistical correlations or interaction terms as such.

Adoptee studies have provided the most striking evidence of gene–environment interaction with respect to antisocial behavior. Thus, Bohman (1996), in a Scandinavian adoptee study of petty criminality, found that just

less than 3% of individuals without a criminal biological parent and without major risk experiences in the adoptive family showed petty criminality. The risk doubled when there was an environmental but not a genetic risk. It went up to 12% with genetic risk in the absence of environmental risk but soared to 40% when both types of risks were present. In other words, it was those individuals at genetic risk who were most likely to be adversely affected by adversities in their adoptive family. It is important to note that, because the adoptee strategy is specifically designed to remove the overlap between genetic and environmental risk, the proportion of variation in the population accounted for by this interaction will necessarily be misleadingly low. That is because the proportion of variation in the population explained by the interaction reflects the proportion of individuals in the key cell (in this case, the subgroup with both genetic and environmental risks); for that reason, it provides a most unsatisfactory measure of the strength of the effects as they operate on individuals (Rutter, 1987).

Several other adoptee studies of antisocial behavior have shown closely comparable gene–environment interaction effects. Thus, Cadoret, Yates, Troughton, Woodworth, and Stewart (1995), in a study of conduct disorder, used the biological parents' drug abuse or antisocial personality disorder as an index of genetic risk and marital, legal, or psychiatric problems in the adoptive family as a reflection of environmental risk. Adoptees at genetic risk were those found to be most likely to develop conduct disturbance when exposed to chronic stresses in the adoptive family in which they grew up.

This finding has important policy and practice implications for two main reasons. First, it shows the error of labeling effects as due to nature or nurture on the assumption that genes and the environment act independently from each other. The alleviation of environmental adversities is most crucial for individuals at genetic risk because they appear to be the people most vulnerable to those adversities. Second, it indicates that it is a serious mistake to think that genetic risk means a deterministic effect about which nothing can be done. To the contrary, it seems that the identification of genetic risk points to the opportunity of successful environmental interventions.

It is also necessary that we appreciate how much remains to be understood; there is an extreme paucity of evidence on just how gene–environment interactions operate. The indexing of genetic risk by a crime record in the biological parent is very crude because it is completely uninformative on the route by which the genetic risk operates. For example, does it create a vulnerability to environmental stressors through effects on hyperactivity, on low autonomic reactivity, or on cognitive processing features? Also, what are the specific environmental features to which there is a genetically influenced susceptibility? Molecular genetic research has its greatest potential as a means of answering such questions on risk and protective mechanisms (Plomin & Rutter, 1997). Once genetic risk can be identified at an individual level, it will be possible to obtain better knowledge of the ways in which environmental risks are mediated. Most especially, it will allow, for the first time, the direct study of the proximal processes involved in environmental vulnerability. Nongeneticists are sometimes suspicious of molecular genetics research because they fear that it will bring about a negative, biologically deterministic view of social behavior. Of course, genetic research, like other forms of research, is open to abuse. It is essential that scientists, together with the general public, take steps to ensure both that genetic research is undertaken ethically and that its findings are not misused. However, genetic findings already run counter to narrowly deterministic concepts. Molecular genetics should be very helpful in enabling a much better understanding of environmental risk mechanisms and, hence, in increasing the opportunities for effective prevention and intervention (Rutter & Plomin, 1997).

Conclusions

Quantitative genetic research has been most informative in showing the importance of genetic influences on virtually all forms of human behavior. Behavior has to have a biological basis, and it is necessary that we understand how the biology functions. Equally, the same research has been crucial in its demonstration that environmental influences are also ubiquitous. Geneticists cannot afford to ignore environmental effects. But, most of all, genetic research has been critical in its message regarding the diverse ways in which genetic influences exert their effects. Nature and nurture do not operate independently of each other, and there needs to be an explicit focus on the interplay between them.

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