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# Families at risk for psychopathology: Who becomes affected and why?

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**Abstract**
We examine resilience in relation to one type of risk—familial risk—from a genetic perspective. Specifically, we ask why only some children growing up with the same familial risk develop psychopathology. Unlike environmental theories, genetics predicts that children in the same family will differ in their outcomes. That is, some children in a family at risk are unaffected not because they are resilient but because they are not at risk genetically. Eventually, some of the genes responsible for genetic risk will be identified, making it possible to predict which children in a family will be affected. Resilience from a genetic perspective can be viewed as the extent to which children at genetic risk are not affected. In addition, there may be genetic contributions to resilience that protect some individuals in high-risk families. Finally, genetic research has shown that salient environment influences often operate in a nonshared manner, making children in the same family different. Research on nonshared environmental factors will advance our understanding of environmental origins of resilience by focusing on environmental reasons why children growing up in high-risk families have such different outcomes.

One of the most potent risk factors for the development of psychopathology is a family history of psychopathology. There is an enormous literature documenting that psychopathology tends to aggregate in families for many forms of disturbance. For example, a recent review suggested familial aggregation for the following childhood psychiatric disorders: autism, attention-deficit disorder/hyperactivity, conduct disorder, depression, and anxiety disorders (Rutter et al., 1990). In addition, research on children at risk for psychopathology by virtue of psychopathology in their parents has also demonstrated the importance of familial transmission. A dramatic example of this work is the consequences of having a depressed parent: Children with a depressed parent are at increased risk for depression (see Rutter et al., 1990).

Although family studies have demonstrated the impact of family history of psychopathology, a topic that has been underrepresented in studies to date is documenting *which* family members become affected. Although family history is a strong predictor of psychopathology, in no study is every family member affected. Because family history is a risk factor and not a perfect predictor of each individual's status, we are now at a point where the next level of conceptualization and analysis may begin. The next question to ask is as follows: Who becomes affected, and why in families at risk for psychopathology? In this article, we will use the concepts of risk and resilience to

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guide this unexplored area of study and also bring new shades of meaning to these concepts by utilizing a quantitative genetic perspective.

## Traditional Designs to Assess Familial Aggregation

We begin with an overview of how family studies have traditionally been conceptualized and analyzed (see Weissman et al., 1986). A predominant approach has been to use a case-control study, in which probands (i.e., individuals affected with a disorder) are selected and matched to control probands (i.e., individuals not affected with the disorder but matched on other salient characteristics). The prevalence of the disorder under study in relatives of affected probands is then compared to the prevalence in relatives of controls. In a variation of this prototypical family study, a high-risk paradigm involves studying the offspring of affected probands (usually longitudinally) and comparing their developmental trajectories to those of offspring of matched control probands. In either a case-control study or a high-risk study, the level of analysis is to determine whether or not aggregate rates of disorders differ in relatives of affected probands versus control probands.

It is from data generated from these designs that the potent risk of family history has been documented (e.g., see Rutter et al., 1990). A glance at the number of affected relatives in family studies, however, reveals that only some, and not all, family members are affected. Based solely on family data, then, we can make two observations: Family history is a risk factor, and some individuals exposed to the risk factor do not become affected. In addition, we should note the one disclaimer that always accompanies family studies: It is not possible from these studies to determine whether the risk is due to genes, environment, or a combination of both. This observation can be extended one step further: Family studies are also unable to determine whether the *lack* of psychopathology in unaffected relatives is due to genes, environment, or a combination of both.

## Redefining Risk and Resilience in a Quantitative Genetic Framework: Some General Comments

The theory and techniques of quantitative genetics are often cited as a powerful approach to understanding developmental psychopathology (e.g., Plomin, Rende, & Rutter, 1991). Generally, this perspective has been applied to disentangle familial aggregation into genetic and environmental components. It may be fruitful, however, to consider how this perspective can be integrated with concepts of risk and resilience and, especially, may be geared to address the issue raised in this article, namely, why only some individuals in families at risk become affected. Before embarking on this specific question, however, a few general comments about quantitative genetic approaches to risk and resilience are in order.

In developmental psychopathology, resilience has often been conceptualized as positive outcomes in the face of environmental risk. For example, Rutter (1983) defined resilience as "people who 'do well' in some sense in spite of having experienced a form of stress which in the population as a whole is known to carry a substantial risk of an adverse outcome" (p. 2). However, it is recognized that familial aggregation of disorders may reflect genetic as well as environmental risk, and quantitative genetic approaches have been suggested as powerful methods for disentangling these forms of risk (e.g., Plomin et al., 1991; Rutter et al., 1990). For example, it has been suggested that the effects of parental depression on children should not be assumed to be environmental in origin (Rutter, 1990). Rather, it is possible that some of the associations between parental depression and negative outcomes in offspring may be mediated by genetic influences. Hence, one general consideration that forms the basis for the issues discussed in this article is that genetic influences may also constitute a significant risk factor; therefore, it is appropriate to discuss

resilience given the presence of genetic risk in the same manner as environmental risk.

A second general consideration is the differentiation between *distal* and *proximal* risk made by researchers studying risk and resilience. Factors such as family history of psychopathology have been classified as distal variables, in that they are not what is experienced by the individual at risk (Baldwin, Baldwin, & Cole, 1990; Luthar, 1993; Richters & Weintraub, 1990). It has been suggested that the risk posed by such distal variables are mediated by proximal variables such as ineffective parenting or family discord (see Luthar, 1993). Furthermore, the point has been made that proximal variables reflect *environmental processes* that reflect the risk factors for individuals with a family history of psychopathology (Richters & Weintraub, 1990).

Although it is possible that proximal risk factors reflect environmental influences on psychopathology, it must be stressed that *genetic influence* should also be considered as a potential proximal risk factor. As an example, it has been argued that offspring of schizophrenic parents are exposed to various environmental influences (such as erratic parenting) that may place them at risk for adverse outcomes. However, it should be appreciated that offspring of schizophrenic parents also may be genetically vulnerable to develop symptoms of schizophrenia. Strong evidence from behavioral genetic studies indicates that schizophrenia is a heritable condition (Gottesman, 1991; Gottesman & Shields, 1982). The conclusion from these studies is that the genetic contribution is a probabilistic (rather than deterministic) factor, in which heritable factors predispose individuals to develop symptoms of schizophrenia (Gottesman, 1991; Rende & Plomin, 1992a). Assuming that specific genes will eventually be uncovered that reflect the anonymous component "heritability" in behavioral genetic studies (Plomin, 1990), the specification of the role these genes play in the expression of schizophrenia will also represent *proximal processes* that place some individuals at risk for adverse outcomes. In summary, it must be appreciated that biological processes should be considered as well as environmental factors when searching for proximal risk factors.

A third general point that has been made in the literature on resilience is that researchers need to move beyond the determination of statistical risk to focus more concretely at factors that reflect risk to individuals (Luthar, 1993). Richters and Weintraub (1990) argued that the concept of statistical risk for offspring of affected parents carries limited meaning for individuals. They argued that although 10–15% of offspring of schizophrenic parents develop schizophrenia, this statistic does not mean that all offspring of schizophrenics are 10–15% along the way to developing schizophrenia. Rather, some offspring develop schizophrenia, whereas others do not and go on to have positive outcomes. The challenge for researchers is to refine our indexes of risk to move beyond statistical probabilities for populations to more concrete factors operating in the lives of individuals at risk. Such a point is especially important because some individuals labeled as resilient may actually not be exposed to the risk factors presumed by population statistics (Luthar, 1993; Richters & Weintraub, 1990). Richters and Weintraub suggested studying nongenetic proximal processes that place some individuals at risk. A primary consideration of this article, as already noted, is that the same argument can be made for genetic influences: Genetic factors may also be regarded as proximal processes that place only some individuals at risk. The main thesis in this article is to consider the ways in which quantitative genetic approaches can assist in this overall goal and to demonstrate how such a framework can be used to explore both genetic and environmental reasons why only some family members in families at risk for psychopathology become affected whereas others go on to have positive outcomes.

## Genetic Risk and Resilience

The first law of genetics is that like begets like. The second law is that like does not be-

get like. Although this might sound like an escape hatch for a weak theory, this is a profoundly important point about heredity. The evolutionary reason for sexual reproduction is to ensure thorough shuffling of the genetic cards with each mating. First-degree relatives are 50% similar for segregating genes, but this also means that they are 50% different. Genetics predicts that heritable dimensions and disorders will show differences as well as resemblances within families. In contrast, extant environmental theories from Freud onward tend to be between-family theories that do not come to grips with differences within families.

The genetic law that like does not beget like carries a specific implication for research on resilience. For genetically influenced disorder, some children in a family at risk are expected to be unaffected, not because they are resilient, but because they are not at risk genetically.

### *Major genes*

This implication applies whether a disorder is due to a major gene or to multiple genes. We are doubtful that major genes will be found for complex dimensions and disorders such as psychopathology (Plomin, 1990; Plomin & Rende, 1991; Plomin et al., 1991). However, even if a single gene were the necessary and sufficient cause for a disorder as in the case of the thousands of classical single-gene disorders such as Huntington's disease (McKusick, 1990), in a family at risk some children will be affected and some will not. When a genetic marker is identified for the disorder, it becomes possible to determine even prenatally which children are at genetic risk and which are not. For single-gene disorders such as Huntington's disease, there is no room for resilience because the gene is the necessary and sufficient cause of the disorder. That is, the gene is lethal for all individuals at genetic risk, regardless of their environmental or genetic background. Some children are at 100% risk, and others are at 0% risk. What might have in the past appeared to be resilience is not due to protective factors but, rather, to the absence of risk.

Such single-gene disorders are not the way to think about genetic risk and resilience in the context of developmental psychopathology. Despite optimism about the power of molecular genetics to identify major genes for psychiatric disorders (Mullen & Murray, 1989; Pardes et al., 1989), there is little evidence for such simple sledgehammer effects of genes for complex behavioral dimensions and disorders and good reasons to doubt that they exist (see Reiss, Plomin, & Hetherington, 1991). Even when single-gene models have been proposed for psychiatric disorders, the construct of "incomplete penetrance" is invariably introduced to take into account the fact that not all individuals who have "the gene" have the disorder. If incomplete penetrance does indeed exist for a single gene with an impact on psychopathology, then the clean case of defining genetic risk no longer holds. Such cases warrant research on resilience and the search for protective factors. For example, environmental factors can be examined that might alter the expression of the gene and perhaps function as either vulnerability or protective factors (Rutter, 1987). The existence of a single gene even with reduced penetrance would be enormously useful in that a specific genetic risk factor would be explicated, which would permit targeting of specific individuals with the risk factor and comparisons between those who become affected and those who do not.

Another implication of incomplete penetrance of a single gene that comes from theoretical and empirical work on resilience is the usefulness of considering not only affected status and maladaptive behavior but also positive outcomes such as social competence (e.g., Garmezy, Masten, & Tellegen, 1984) as well as other psychological factors that moderate the effects of the gene. We raise these issues to emphasize that even deterministic single gene disorders with reduced penetrance can present opportunities for developmental psychopathologists. Indeed, they represent interesting cases because specific mechanisms of risk

are known and can be used in highly focused investigations of resilience.

### *Multiple genes and multifactorial models*

Although single genes have been isolated for many medical disorders, it is becoming increasingly recognized that major genes are unlikely to be found in the population for behavioral disorders or for common diseases (King, Rotter, & Motulsky, 1992). The shift toward recognition of multiple-gene influence is important in countering the naive assumption of earlier linkage studies in psychiatry that psychiatric disorders are like classical Mendelian disorders—dichotomous and due to a single gene that is necessary and sufficient to produce the disorder.

An alternative hypothesis is that many genes may make small contributions toward variability and vulnerability. The genetic quest is to find not *the* gene for a psychiatric disorder, but the *many* genes that increase susceptibility in a probabilistic rather than predetermined manner (i.e., polygenic model). In addition, within this framework it is likely that environmental factors also play a key role in shaping developmental pathways (i.e., multifactorial model). It is here that the relevance of links between genetic risk and resilience comes to the fore.

There is a large and increasing data base of twin and adoption research on psychopathology throughout the life span. Two general conclusions emerge from this work: Genes make a significant but moderate contribution to many forms of psychopathology; and environmental factors also make a significant contribution, but they are factors that are nonshared (i.e., are responsible for differences between individuals in the same family).

Putting together these two conclusions provides a partial tentative answer to our question of who becomes affected and why in families at risk for psychopathology. We are led to suggest that genetic risk is important, but not all-determining, and that there are environmental factors that contribute to differences in developmental outcome for children growing up in the same family. However, as discussed with single-gene models, we require a more detailed answer to the question in terms of actual mechanisms and processes.

A first step is to recognize that quantitative genetic approaches are especially relevant to the study of risk and resilience in families in that they may target specific forms of psychopathology that show evidence of genetic risk. For example, recent evidence suggests that both the dimension of attention problems and the clinically defined attention-deficit disorder/hyperactivity are influenced by genetic factors (e.g., Gillis, Gilger, Pennington, & DeFries, 1992; Rende, Plomin, Edelbrock, Fulker, & DeFries, 1993; Stevenson, 1992). In contrast, less evidence indicates that conduct problems are influenced genetically (see Rutter et al., 1990), and environmental factors shared by siblings seem to be more important. As these examples suggest, quantitative genetic research may help to delineate different classes of risk—that is, genetic versus shared environmental—that form the background upon which other vulnerability or protective factors may operate.

Given findings such as these, the next step is to begin to identify the specific risk and resiliency factors involved in different forms of psychopathology. In terms of genetic risk, quantitative genetic research can identify families at risk but cannot indicate which children in the family are specifically at risk. For multifactorial disorders as well as for single-gene disorders, some children in a family are unaffected not because they are resilient but because they are not at risk. Unless the genetic risk can be pinned down, resilience will remain elusive.

Although it is a much more daunting prospect than in the case of major genes, advances in molecular genetics, especially the generation of thousands of DNA markers, have made it possible to consider identifying genes in multiple-gene systems. Although guaranteed to localize a single-gene disorder, linkage techniques are not capable of finding the chromosome location of genes in multiple-gene systems. Other tech-

niques are being developed that have this potential, especially as DNA markers with functional physiological effects are found (Sobell, Heston, & Sommer, 1992). For example, one approach currently in use is allelic association (Edwards, 1991), which compares allelic frequencies in groups of unrelated individuals and has as its greatest strength the ability to identify genes of small effect size. Allelic association has been especially successful in finding genes that affect diseases (King et al., 1992), especially genes in the major histocompatilibity complex of HLA (Tiwari & Terasaki, 1985). A more controversial example involves associations reported between a DNA marker for dopamine $D_2$ receptor for alcoholics and controls (Cloninger, 1991).

Rather than discuss the specifics of these approaches, we wish to emphasize the conceptual point that several genetic markers that index a significant portion of the genetic risk for psychopathology may eventually be available. The best example to date is serum cholesterol, a risk factor for heart disease. About a quarter of the variance in serum cholesterol can be explained by associations with four apolipoprotein gene markers (Sing & Boerwinkle, 1987). Given the findings from quantitative genetic studies that many forms of psychopathology show genetic influence, and the rate at which developments have occurred in molecular genetics, it is not unreasonable to speculate that at some point in the future specific indexes of genetic markers of *small, probabilistic effect* will be found that represent genetic risk for behavioral maladaptation (Rende & Plomin, in press).

The identification of multiple genes with small effects will have large implications for how we examine risk and resilience in families. As is the case with single genes, identifying multiple genes will redefine specific indexes of risk for individuals within families. Rather than saying that two family members (e.g., siblings in a family at risk for psychopathology) are at equal risk, it may be possible to specify individual genomic profiles of risk for each child and chart how these profiles relate to developmental pathways. To the extent that risk can be established, it is then possible to investigate resilience.

## Genetic Influence on Resilience

Although resilience is often considered in terms of environmental factors such as protective parent–child interactions, the possibility of genetic involvement in resilience merits attention. An especially interesting finding is that some genetic conditions that lead to diseases may nonetheless function as protective factors for other conditions. Genetic factors that affect resistance (resilience) as well as susceptibility (vulnerability) to infectious disease (Childs, Moxon, & Winkelstein, 1992) have been found. Most well known are several genes (sickle cell, glucose-6-phosphate dehydrogenase and the Duffy blood group) that increase resistance to malaria. Another example is that mice have been bred for resistance as well as susceptibility to infection (e.g., Gowen, 1960).

Although there are no comparable examples in the study of psychopathology, it has been argued that genetic effects on psychopathology may nonetheless carry some benefits in terms of other areas of functioning (e.g., Rutter, 1991). For example, it may be that the social deficits seen in autism may arise in part from genetic influences that decrease sociability while increasing the ability to focus intensively and independently on tasks. As this latter ability is clearly useful under some circumstances and may be considered in certain contexts as an adaptive function, it is possible that the genetic factors responsible for deleterious outcomes such as autism may also be involved in skills that may be applied toward adaptive development. Indeed, this argument is presented as a possible explanation of why autism does not "die out" in the population even though autistic individuals rarely reproduce (see Rutter, 1991).

The idea that genetic factors may contribute to resilience may also be applied profitably in the study of resilience in the face of environmental adversity. Two fac-

tors that have been studied extensively in resilience research are temperament and intelligence. It has been recognized that such "dispositional attributes of the child" may serve protective functions and that these attributes may reflect genetically influenced characteristics (Luthar & Zigler, 1991). To the extent that temperament dimensions are heritable traits (e.g., Plomin & Rende, 1991), then, it is the case that genes not only predispose some children to respond badly to adverse circumstances but also may act as protective factors that allow children to not experience negative outcomes. As some recent studies have documented that temperament may moderate the effects of stress (e.g., Rende & Plomin, 1992b; Wertlieb, Weigel, Springer, & Feldstein, 1987), behavioral genetic studies should be conducted to empirically assess the extent to which heritable temperamental traits contribute to susceptibility to environmental stressors.

A similar case can be made for intelligence, which is one of the most widely studied factors in resilience research (Luthar & Zigler, 1991). Intellectual ability shows consistent evidence of genetic influence across many studies (e.g., Plomin, DeFries, & McClearn, 1990). Hence, cognitive ability may be considered as a potential protective factor that is influenced in part by genetic factors, and again behavioral genetic studies should be conducted to examine how heritable abilities contribute to differential responses to various forms of environmental stress.

Considering temperament and intelligence not only as potential protective factors but also as heritable influences that may promote resilience serves as a concrete reminder that genetic influences may directly contribute to adaptive development despite the presence of environmental risk factors. This point is important because genetic influences are often considered in psychopathology as predisposing factors to maladaptation. One of the most prominent general frameworks for the development of psychopathology is a diathesis-stress model, in which a genetic predisposition to a disorder interacts with negative environmental factors to lead to psychopathology. In this framework, genetic influences are usually conceptualized as predisposing to psychopathology, whereas their potential protective function is not considered (Rende & Plomin, 1992a, in press). Hence, when examining the role of genes in the development of psychopathology, it may be important to not only consider genetic risk but also to consider heritable influences that may be protective.

Given these general points about genetic influences on resilience, we need to consider the implications for why only some members of families at risk for psychopathology become affected. The key point to keep in mind is the second law of genetics discussed previously: Like does not beget like. Although heritable traits such as temperamental characteristics and cognitive abilities may serve a protective role in development, all individuals in a family are not identical across these domains. Again, what is necessary is to eventually develop individual-specific indexes of the genetic contributions to the domains. That is, as the case has been made that populationwide indexes of familial risk convey little about the risk posed to individuals in families, this point must be kept in mind when we consider genetic contributions to resilience.

A final point is that the study of protective factors such as intelligence and temperament must consider the complex ways in which these factors play a role under different environmental contexts. Luthar (1993) demonstrated the various ways in which a presumed protective factor intelligence may lead to many outcomes depending on the context. Hence, the argument has been made that there is a need to move beyond labeling factors as "protective" and examine the processes by which dispositional characteristics and environmental circumstances come together in development. This argument may be applied as well to the general question of this article. In addition to identifying individual-specific indexes of heritable traits that may serve protective functions, we need to also consider individ-

ual-specific environments in risk families. This topic of individual-specific environments has become a central issue in behavioral genetics, and we turn now to discussion of this crucial concern.

## Nonshared Environment

Quantitative genetic research on psychopathology suggests a new perspective on environmental risk and resilience that is especially relevant to the question, Who becomes affected within a family? With few exceptions, quantitative genetic research converges on two conclusions concerning the environment. First, the environment is important in the sense that genetic factors only partially account for psychopathology. The second conclusion is more novel: The way in which the environment affects psychopathology is very different from the way in which the environment was assumed to operate in traditional theories of environmental influence. Whatever the salient environment factors might be, they operate to make children in the same family no more similar than children reared in different families (Dunn & Plomin, 1990; Hetherington, Reiss, & Plomin, 1994; Plomin & Daniels, 1987). Such environmental influences are called nonshared in that they are not shared by children in the same family, in contrast to environmental factors shared by siblings that would be expected to make siblings similar. In other words, nonshared environmental influences are experienced differently by children in the same family.

Quantitative genetic theory and methods aim to investigate the extent to which familial resemblance occurs for reasons of shared heredity or shared family environment. The answer that emerged from quantitative genetic research is that familial resemblance is largely due to shared heredity, not to shared family environment. However, heredity by no means explains all psychopathology. Nongenetic factors are important, but these factors are not shared by children growing up in the same family. One example of how this conclusion was reached involves identical twins. Differences within pairs of identical twins directly estimate nonshared environment (plus error of measurement). For schizophrenia, the concordance of identical twins is about 40%. This resemblance suggests strong genetic influence as compared to the population base rate of about 1% for schizophrenia. However, why are these genetically identical pairs of individuals more often than not *discordant* for schizophrenia? The answer is nonshared environment. There can be no genetic reason why identical twins are so often discordant for schizophrenia. Methods for estimating nonshared and shared environmental influence and an update of the evidence for the importance of nonshared environment is available elsewhere (Plomin, Chipuer, & Neiderhiser, 1994).

The message is not that family experiences are unimportant but, rather, that the relevant environmental influences are specific to each child, not general to an entire family. These findings suggest that instead of thinking about the environment on a family-by-family basis, we need to think about the environment on an individual-by-individual basis. So often it has been assumed that the key influences on children's adjustment are factors that are likely to be shared by children in the same family: their parents' personality and psychopathology, the quality of their parents' marriage relationship, the children's educational background, the neighborhood in which they grow up, and their parents' attitude toward child-rearing. Yet, to the extent that these influences are shared, they cannot account for the differences in outcomes of children growing up in the same family. The critical question is, Why are the children in the same family so different? The answer requires that we study more than one child in each family and explore the separate worlds of siblings, their differential experiences both in and out of the family.

Consideration of nonshared environment is likely to advance understanding of both environmental risk and resilience. Because environmental factors responsible for

psychopathology are of the nonshared variety, we must turn up the power of our microscopes to look for environmental risk and resilience not between families but within families. Consider as one example the negative consequences of parental behavior displayed by depressed adults, which have been documented by many researchers (see Rutter, 1990). To date, no studies have examined how a depressed parent interacts with multiple children within the family. One possibility is that, because of the chronic nature of depression, severely depressed adults do not discriminate among children; their behavior during depressive episodes may be consistent across siblings in a family.

Another possibility, however, should be considered: A depressed parent may show differential behavior, perhaps based in part on differences among children. It has been shown that parent irritability, criticism, and hostility—behaviors related to depression—may be focused especially on children with specific temperamental characteristics (Rutter, 1986). Other factors such as age and gender could, of course, be considered in addition to temperamental traits. The fundamental point, however, is that although one child in a family may be especially vulnerable to environmental risk, another child in the family may be protected from *exposure*. Determining the extent to which environmental risk is shared by children in a family is an essential step for understanding why only some individuals in families at risk develop psychopathology. As was the case with genetic influence, we need to determine whether unaffected individuals are resilient in the face of environmental adversity in the family, or in fact in some unaffected individuals are actually not at as high risk as other family members.

As mentioned earlier, the strongest evidence for the importance of nonshared environment comes from studies of discordant identical twins. Discordant identical twins provide a unique opportunity to identify specific sources of nonshared environment. This is because their discordance must be due to nonshared environment, unlike fraternal twins or nontwin siblings, who might differ in outcome because of genetic differences. This approach has been used to explore nonshared environmental factors in schizophrenia, albeit with little success to date (Gottesman, 1991; Gottesman & Shields, 1982). Although little progress has been made in specifying nonshared factors that contribute to discordance among identical twin pairs, the general approach has great potential for examining the environmental contributions to both adaptive and maladaptive development. In addition to searching for negative influences that may lead to schizophrenic symptoms in one co-twin, it is also essential to investigate possible factors that protect the other co-twin from a negative outcome. That is, both risk and protective factors could be examined when assessing discordant identical twins. This approach is intriguing considering recent research on the offspring of twins discordant for schizophrenia. Such work indicates that the offspring of nonaffected identical twins have a risk for schizophrenia similar to that of the offspring of schizophrenic co-twins (Gottesman & Bertelsen, 1989). What this implies is that the same schizophrenia-inducing genes are present in both the normal and schizophrenic twins as well as in their offspring. Most importantly, this finding suggests that environmental factors determine whether or not schizophrenic symptomatology is manifested. Again, because only some of the twins and offspring develop schizophrenia, such environmental factors must be nonshared, and it appears that these nonshared influences help to determine whether an individual has an adaptive or maladaptive outcome.

A final point to consider is that focusing on nonshared processes related to psychopathology may help to clarify the ways in which both genetic and environmental influences contribute to pathways in development. An influential paper by Scarr and McCartney (1983) presented an overview of various developmental processes that in-

volve the interplay of genetic and environmental influences, such as gene–environment correlation. It may be possible to use the discordant sibling method to highlight some of these models. As discussed earlier, two theories could be contrasted concerning the influence of the behavior of depressed parents on children. In one model, depressed parents would be expected to behavior similarly to multiple children in the family because of the chronic nature of depression. In a second model, it is hypothesized that depressed parents may especially target some children in the family, whereas others may not be exposed as strongly to maladaptive parenting. By using informative designs (such as the twin method, or comparisons of full, half, and unrelated siblings), it may be possible to not only test which model is correct but also to determine whether or not there are indeed associations among children's heritable traits, their environment (ie., parental behavior), and their outcomes. Placed within the framework of nonshared environment, such designs could help to clarify etiological influences that lead siblings to discordant developmental pathways. Again, the overall goal would be to develop individual-specific profiles of both genetic and environmental factors and to examine how the interplay between them leads to differential outcomes in families.

## Concluding Remarks

In this article, we have focused on redefining risk and resilience using concepts from quantitative genetics. Our purpose has been to highlight a large theme from studies of families at risk: Not every member within families at risk become affected. We believe that advances in molecular genetics, as well as the study of environmental influences, will eventually allow for more precise models of why this is the case, and we have outlined some general theoretical possibilities of how the concepts of risk and resilience may be applied in this overall framework.

As a concluding theme, we wish to make two points. First, identifying specific genes does not imply that environmental factors are unimportant or that genes alone are responsible for psychopathology. Rather, we emphasize that most genetic influences on psychopathology will be probabilistic and not deterministic. Replacing anonymous components of variance—in terms of both genes and environments—will contribute much to understanding both risk and resilience in families. Ultimately, this will lead to preventive interventive programs that do not involve manipulations on the genome but, rather, attempt to modify environmental factors based on information provided by genetic data and that will be targeted toward individuals at particular risk within families. In addition, nonshared environmental factors will play a key role in understanding environmental risk and resilience. Indeed, we suggest that questions about risk and resilience afford an excellent opportunity to address the interface between nature and nurture in the development of psychopathology.

A second concluding remark is that quantitative genetic research on psychopathology can follow the lead of resilience research by focusing on positive outcomes or competence as well as absence of psychopathology. In this vein, understanding genetically influenced protective factors may highlight how positive developmental pathways can occur even in the face of familial risk. In addition, we may also begin to uncover ways to develop preventive interventive programs for children at specific genetic risk. Current approaches in medicine to complex diseases such as heart disease suggest that identifying specific genetic markers in individuals at global familial risk will help these individuals make wise choices not only related to preventing heart disease but also to promoting good health and quality of life. The same message applies in research on psychopathology. A quantitative genetic framework can help not only to prevent psychopathology but also to promote positive developmental outcomes in individuals in families at risk.

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