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An Interdisciplinary Approach to Studying Gene–Environment Interactions: From Twin Studies to Gene Identification and Back

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Abstract

There has been a surge of interest in studying gene–environment interaction; however, research in this area faces a number of challenges. Interdisciplinary collaborations are critical at this juncture. This article reviews studies that illustrate how findings across different literatures can be synthesized to characterize how genetic and environmental influences impact developmental pathways. Developmental scientists are poised to make important contributions to studying gene–environment interaction. However, for this potential to be realized developmental–genetic studies must incorporate the most recent advances in genetics, and bridge the current schism that exists between genetic research being conducted in the fields of psychology and genetics.

There has been an exponential increase in interest in genetic influences on behavioral outcomes in the past decade. These ideas are not new to the field of development, where a handful of scholars, such as the embryologist Paul Weiss, the geneticist Sewall Wright, and more recently the psychologist Gilbert Gottlieb, championed a systems view of developmental biology that included multiple interacting levels of analysis (as reviewed in Gottlieb, 2002). However, developmental science historically focused primarily on understanding and characterizing environmental influences on development. The notion that genetic influences were important in shaping behavior was sometimes viewed as a hostile "opposing" viewpoint. The field has now largely embraced a more holistic view of development, in which genetic and environmental influences are viewed as inexorably intertwined, and the challenge is to understand how these influences act and interact across development. But though there may be increasing consensus that studying gene–environment (GxE) interaction is important, agreement about just how to go about that—and how to evaluate what interactions we can believe—presents yet another challenge to the field. I propose that interdisciplinary collaborations are particularly critical at this juncture, as there are many different fields that can make important contributions to characterizing GxE interactions. In this article, I review some of the study designs and different areas of research that can contribute to understanding GxE interaction effects. I illustrate how findings across these literatures can be synthesized to characterize how genetic and environmental influences come together to affect developmental pathways.

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WHAT IS GENE–ENVIRONMENT INTERACTION?

It is important to start by noting that people mean different things when they say *gene–environment interaction.* Before studying GxE interaction became so widespread, the phrase *gene–environment interaction* was sometimes used loosely to mean that genes and environments acted together to contribute to the outcome. This is entirely consistent with genes and environments having main effects, but there being no statistical interaction. The idea of there being a statistical interaction between genetic and environmental effects is the more recent and widespread meaning of *gene–environment interaction.* This indicates a situation where genetic and environmental effects are not independent of one another, but rather, the importance of one's genetic predisposition varies as a function of environmental conditions, or alternately, the importance of the environment varies as a function of one's genotype. It is important to note that these two alternate characterizations of GxE interaction are statistically indistinguishable. The key concept is one of dependence: one cannot understand the effect of genes without taking into account the environment, and one cannot understand the effect of the environment without taking into account genetic predispositions. The implications of this dependency are profound and may hint toward why the concept of GxE interaction was met by so much resistance (or ignored) by the fields of development and genetics, respectively, for so long. Most scientists are trained in one area—psychology OR genetics—so the very notion of GxE interaction forces one to recognize and incorporate other areas of science into one's own research. This can be difficult! Yet I propose that this is also where the most exciting advances are likely to happen.

LATENT VERSUS MEASURED GENE–ENVIRONMENT INTERACTION

A further distinction exists in the study of GxE interaction: that of latent versus measured GxE interaction. Although they are both active fields of study it is actually quite striking how different and nonoverlapping the literatures are. Latent genetic influences are inferred by comparing similarity across individuals with different degrees of genetic sharing, for example, different types of family members. Twin studies are one of the most widely used methods for studying latent genetic effects. In the classic twin design, information about the relative importance of genetic and environmental influences is inferred by comparing the similarity of monozygotic (MZ) twins and dizygotic (DZ) twins. MZ twins arise from a single egg, fertilized by a single sperm, and therefore share all of their genetic variation identical by descent. DZ twins result from two eggs, fertilized by two sperm, and therefore share, on average, 50% of their segregating genetic variation, as do ordinary siblings. The basic genetically informative twin model partitions variance in a behavior into additive genetic influences (A), common environmental influences (C) [or dominant genetic influences (D); C and D cannot be simultaneously modeled with only twin data], and unique environmental influences (E). In twin models, genetic influences are set to correlate 1.0 between MZ twins, and 0.5 between DZ twins. *Common environmental effects*, as defined in biometrical twin modeling, refers to all environmental influences that make siblings more similar to one another. Unique environmental influences are those factors that have the effect of decreasing the covariance between siblings and include measurement error.

When information about a particular environment of interest is measured, the basic twin model can be extended to test whether the relative importance of latent genetic and environmental influences varies as a function of that environment. This can be accomplished either using a multiple group model, allowing one to test for differences in genetic and/or environmental influences across different groups (e.g., urban vs. rural environments), or through a subsequent extension of the twin model (Dick, Rose, Viken, Kaprio, & Koskenvuo, 2001; Purcell, 2002) allowing one to test for changes in genetic and environmental influences as a function of a more continuous environmental measure (e.g., scores on a family environment scale). In latent genetic studies, it is important to note that no specific genes are actually measured. Rather, genetic influences are inferred by comparisons of relative pairs. This provides an overview of the total aggregate genetic effect, and how that total genetic effect may differ across different environments, but it tells us nothing about the specific genes involved or the underlying biology. It has been suggested that now that we have the capability to measure specific genes, the utility of twin studies is limited, and they should be resigned as a thing of the past. However, until we have identified all of the specific genes that make up the predisposition to any given trait (a goal that we are not yet close to achieving for any psychological outcome), twin studies will continue to provide the only method of studying overall genetic influence for any given trait.

The other predominant model of studying GxE interaction is via studies that measure specific genotypes. The most well known literatures for specific GxE interactions involve the interaction between the MAOA genotype and childhood abuse in the development of antisocial behavior (Caspi et al., 2002), and the interaction between a polymorphism in the serotonin transporter gene and stressful life events in the development of depression (Caspi et al., 2003). Subsequent to the initial high-profile publications, there has been a literal explosion of measured GxE interaction studies. These literatures have been highly controversial, full of high-profile replications (Kim-Cohen et al., 2006), failures to replicate (Risch et al., 2009), and thoughtful commentaries on the replications and nonreplications that have been observed (Caspi et al., 2010).

Despite the exponential growth in the number of papers published in this area in recent years, most measured GxE interaction studies have been limited to a small number of candidate genes, and more specifically, to a small number of purportedly functional polymorphisms in those candidate genes (Belsky et al., 2009). This is in stark contrast to the shift in genetic strategies that has taken place in the field of genetics over a similar time period. Candidate gene studies have largely fallen out of favor in the field of genetics due to recognition that our understanding of the underlying biology of psychological and behavioral outcomes is woefully limited. With falling genotyping costs and rapid developments in technology for high-throughput genotyping (genotyping large numbers of markers at once), gene identification studies have moved toward more atheoretical approaches that allow one to scan the entire genome (so-called genome-wide association studies or GWAS). By using a systematic approach one is not limited to previously known genes of interest. Indeed, in some of the more successful applications of GWAS to complex disorders, such in the area of type II diabetes, GWAS has uncovered susceptibility genes that have expanded our understanding of the underlying etiology to include biological pathways

previously unrecognized as being involved in the disease pathology (Billings & Florez, 2010).

GWAS have their limitations as well. Severe corrections for multiple testing must be applied when using atheoretical, large-scale approaches, resulting in the need for extraordinarily large numbers of participants. And candidate gene studies have not been abandoned entirely. In fact, it makes good sense to take advantage of what knowledge we do possess about the underlying biology and to further study genes involved in those systems. One of the most robust findings to come out of meta-analyses of GWAS for smoking behavior was to confirm the involvement of a set of candidate genes previously thought to play a role in susceptibility, the nicotine receptor genes (Caporaso et al., 2009). However, a critical difference is that most of the specific GxE interactions that have been studied have been limited to a single purportedly functional marker in a candidate gene of interest. The evidence for the functionality of these markers is often ambiguous (Cirulli & Goldstein, 2007), a factor that is not widely recognized by developmental psychologists. This is understandable as the methods of molecular biology go well beyond the expertise of most psychologists (and indeed, many geneticists!). Establishing functionality of genetic loci is a very challenging area, and one without strong consensus. The ambiguity of the evidence for functionality of the markers that are widely studied in the measured GxE literatures is rarely acknowledged or discussed.

In contrast, candidate gene studies in the field of genetics rarely genotype a single genetic marker in the gene of interest; in fact, this would generally not be publishable in any respectable genetics journal (Pettersson et al., 2009). Rather, with data from the Human Genome Project and the HapMap project, we now know something about the structure of most genes in the human genome (Manolio et al., 2008). Further, there are many polymorphic markers available across most genes of interest. It is possible that multiple locations in a gene could have various forms that lead to differential function of that gene contributing to differential susceptibility to an outcome (McClellan & King, 2010). In fact, we know this to be the case in single gene Mendelian disorders, such as cystic fibrosis, where more than 1,000 different mutations have been discovered in the cystic fibrosis gene that lead to the disease phenotype! Accordingly, to truly evaluate the role of a hypothesized gene of interest it is absolutely necessary to understand the genomic structure in and around that gene (see Dick et al., 2011 for a discussion of this and other issues surrounding basic genetics that social scientists doing research in this area need to understand).

THE WAY FORWARD

Studying GxE interaction has proceeded thus far in a number of fairly independent silos. A growing number of papers are emerging from the field of behavior genetics showing that the importance of (unmeasured) genetic influences on a variety of different outcomes (i.e., heritability) varies as a function of specific measured environments. Largely independent literatures have emerged around a handful of specific measured genes, most notably *MAOA* and *5HTT*. Gene identification projects continue to steamroll forward, embracing a large scale, atheoretical approach with seemingly little intersection with the aforementioned literatures. Although GxE interaction is being discussed far more in the area of gene finding

than in years past (Engelman et al., 2009), these discussions often involve atheoretical approaches and center on methods to adequately detect GxE in the presence of extensive multiple testing (Gauderman, 2002; Gauderman et al., 2010), again reflecting differences in philosophy and methods between the fields of psychology and genetics. The more recent interest in GxE interaction in the gene finding world likely stems from the slow progress in identifying specific genes involved in psychiatric traits, and the failure of genetic methods to date to account for a substantial portion of the heritability of psychiatric conditions, rather than from a deep appreciation of environmental influence. Much of the GxE interaction work in large gene-finding studies is moving forward without the involvement of psychologists or other social scientists with expertise in studying the environment.

The fact that these areas of study have unfolded largely in parallel is unfortunate. Each of these strategies has its own strengths and limitations. Combining these areas of study allows us to capitalize on their respective contributions and help avoid some of the pitfalls associated with each individually. Below, I delineate a program of research that illustrates how findings from twin studies, gene identification projects, and the broader developmental literature can be used to inform and guide measured GxE interaction studies. These studies are all in the area of GxE interaction in alcohol use and related behavioral outcomes. This reflects the fact that this is my own area of study, although a similar integrative strategy could be applied to most psychological outcomes of interest.

LATENT GENE–ENVIRONMENT INTERACTION IN THE AREA OF ALCOHOL USE

A robust finding to emerge from twin studies in the area of alcohol use in recent years is that genetic influences on alcohol use and related problems are dynamic. Throughout this section when I refer to genetic effects, I am referring to aggregate genetic influence, as inferred from twin comparisons, not effects associated with any specific gene. Twin studies indicate that genetic influences change across time, becoming increasingly important as individuals move from early adolescent experimentation to more established patterns of alcohol use later in adolescence/emerging adulthood (Rose, Dick, Viken, & Kaprio, 2001), and they can also change profoundly as a function of the environment. In the Finnish twin studies, we have initiated a program of research aimed at identifying environmental factors that modify the relative importance of genetic and environmental influences on alcohol related outcomes across development. We have found that environments across a number of different domains can play important roles in moderating the importance of genetic effects. Initially we found that a number of different socioregional factors moderated the relative importance of genetic and environmental effects. Genetic influences on frequency of alcohol use in late adolescence (ages 16–18) were stronger in urban settings, neighborhoods with greater regional alcohol sales, those with more migration in and out (which we believe may represent greater anonymity and less community monitoring), and those which had a greater percentage of slightly older adolescents (likely providing more alcohol availability and diversity of selection of role models and activities) (Dick et al., 2001; Rose et al., 2001). Similar socioregional effects were observed earlier in adolescence on behavior problems in girls at age 14 (Dick, Bernard, et al., 2009).

We have also found that parental monitoring can dramatically alter the importance of genetic and environmental influences on adolescent substance use, with genetic effects assuming far greater importance under conditions of lower parental monitoring (Dick, Viken, et al., 2007). Further, we have found that genetic influences on adolescent alcohol use assume greater importance when the adolescent has more peers who also report substance use (Dick, Pagan, et al., 2007), a finding that has been replicated in other independent samples (Harden, Hill, Turkheimer, & Emery, 2008). Similar effects have been demonstrated for more general externalizing behavior: genetic influences on antisocial behavior were higher in the presence of delinquent peers (Button et al., 2007). In studies conducted in other large twin cohorts, genetic influences on alcohol use were greater among unmarried women, whereas having a marriage-like relationship reduced the impact of genetic influences on drinking (Heath, Jardine, & Martin, 1989). Religiosity has also been shown to moderate genetic influences on alcohol use among females, with genetic factors playing a larger role among individuals without a religious upbringing (Koopmans, Slutske, van Baal, & Boomsma, 1999).

A common theme emerges across these findings of GxE interaction from the twin literature, namely that environments that exert more social control (e.g., higher parental monitoring, less migratory neighborhoods, etc.) tend to reduce genetic influences, whereas other environments allow greater opportunity to express genetic predispositions, such as those characterized by more deviant peers and greater alcohol availability. It is likely that many of the important moderating effects of the environment associated with alcohol use and related externalizing behavior reflect differences social control and/or opportunity, resulting in differential expression of individual predispositions (Shanahan & Hofer, 2005).

GENE-IDENTIFICATION EFFORTS IN ALCOHOL DEPENDENCE

A number of gene identification projects are underway to identify the specific genes involved in alcohol dependence and related disorders (Begleiter et al., 1995; Prescott et al., 2005). The use of complementary strategies has contributed to the successful identification of a number of genes associated with alcohol dependence, many of which have now replicated in independent samples (Dick et al., 2006). The candidate gene strategy was used early on to demonstrate robust association with polymorphisms in genes encoding alcohol metabolizing enzymes: aldehyde dehydrogenase (ALDH; Harada, Agarwal, Goedde, Tagaki, & Ishikawa, 1982) and alcohol dehydrogenase (ADH; Whitfield, 1997).

More recently, there have been large-scale gene identification efforts that have targeted affected individuals and their family members and used systematic gene identification strategies, conducting linkage analyses to identify genomic regions likely to harbor susceptibility genes, followed by association analyses to pinpoint the specific genes (Edenberg, 2006). Most recently, these strategies have been joined by GWAS that test ~1 million markers or more across the genome (Bierut et al., 2009; Edenberg et al., 2010).

Next on the horizon is sequencing, with efforts underway to sequence coding regions across the genome, and the possibility of sequencing the entire genome! And though beyond the scope of this article to review all the findings, a growing number of genes have been identified as associated with alcohol dependence (Edwards, Svikis, Pickens, & Dick, 2009).

Despite the fact that broad statements are often made about how gene identification efforts have not been widely successful for identifying genes involved in psychiatric conditions, this reflects the fact that the replicated genetic associations that exist likely only reflect a small proportion of the total number of loci that are believed to be involved. There is far more work to be done. But it does not mean that there has been no progress! There are replicated associations that exist, and these can be integrated into developmental studies to characterize the risk pathways associated with these genes. Further, some of the failures to replicate likely reflect heterogeneity between studies, at the level of the phenotype and with respect to environmental exposure, and these are complexities that developmental psychologists may be able to help resolve.

In the area of alcohol dependence, two genes that we have focused on that come out of the gene identification efforts from the Collaborative Study on the Genetics of Alcoholism (COGA) are *GABRA2* and *CHRM2.* Both genes were targeted because they were plausible biological candidates located near linkage peaks observed in the COGA sample. Chromosome 4 repeatedly emerged with linkage to alcohol dependence diagnoses, quantitative drinking measures, and electrophysiological measures (that are believed to represent endophenotypic markers of a predisposition toward alcohol problems and other externalizing disorders) (Porjesz et al., 2002; Reich et al., 1998; Saccone et al., 2000; Williams et al., 1999). Located under the chromosome 4 linkage peak was a cluster of GABA-A receptor genes. These genes were considered good candidates for potential involvement in alcohol dependence, as evidence from animal, human, and in vitro cell models suggested that Aminobutyric acid (GABA), the major inhibitory neurotransmitter in the human central nervous system, is involved in many of the neurochemical pathways affecting alcohol use and related disorders (Buck, 1996; Grobin et al., 1998). Genetic markers were tested across the four GABA-A receptor genes in the region, and evidence emerged that alcohol-dependent individuals were more likely to carry a particular version of the *GABRA2* receptor gene, suggesting it may be involved in the predisposition to alcohol dependence (Edenberg et al., 2004). This finding was subsequently replicated by multiple independent studies (Covault, Gelernter, Hesselbrock, Nellissery, & Kranzler, 2004; Fehr et al., 2006; Soyka et al., 2008; Xu et al., 2004).

Similarly, chromosome 7 was another region in COGA that had linkage to alcohol dependence diagnoses and electrophysiological endophenotypes (Jones et al., 2004; Wang et al., 2004). Just under the linkage peak was the gene *CHRM2,* which is an acetylcholine muscarin receptor. Muscarinic acetylcholine receptors activate a multitude of signaling pathways, and there is evidence they are involved in many brain functions, such as learning and memory, providing biological plausibility for its role in psychiatric and behavioral outcomes (Volpicelli & Levey, 2004). Like with *GABRA2,* genetic markers were tested across *CHRM2,* and alcohol-dependent individuals were found to be more likely to carry a particular version of the gene (Wang et al., 2004), a finding that was subsequently replicated in an independent sample (Luo et al., 2005). In the case of both genes, association was originally identified with adult alcohol dependence, but subsequent analyses demonstrated broader involvement in a number of externalizing disorders, including childhood conduct problems, adult antisocial behavior, and illicit substance use (Dick, 2007; Dick et al., 2008).

INTEGRATING TWIN STUDIES AND GENE IDENTIFICATION EFFORTS

With mounting evidence for involvement of *GABRA2* and *CHRM2* in alcohol problems and related externalizing disorders, we aimed to further explore the risk associated with these genes in a general, population-based sample. To that end, we genotyped associated markers across both genes in the Child Development Project (CDP), an intensively studied community-based cohort of more than 500 children, followed annually from kindergarten through their mid-twenties. In 2006 and 2007 we collected DNA from the participants via saliva sample and obtained DNA from 452 individuals, representing 93% of the target sample of regular CDP participants. Based on the twin literature indicating that childhood behavior problems and adult alcohol dependence overlap largely due to shared genetic factors (Slutske et al., 1998), and that alcohol dependence symptoms observed very early in adolescence have a very different etiology, being largely environmentally influenced (Rose et al., 2004), we hypothesized that genes originally associated with adult alcohol dependence would be associated with behavior problems at earlier stages of development. We also hypothesized that the association between the gene and behavior problems would be moderated by environmental factors related to social control and opportunity, per the twin literature on GxE interaction effects. In particular, we tested for a moderating role of parental monitoring and peer antisocial behavior, based on our findings from the Finnish twin studies. We hypothesized that the association between the high-risk genotypes and externalizing behavior would be stronger under conditions of lower parental monitoring and higher peer deviance, based on the twin evidence that the overall genetic influence observed in the population was greater under these conditions.

It is important to note that change in the overall heritability across environmental contexts does not necessarily dictate that any one specific susceptibility gene will operate in a parallel manner. However, a change in heritability (which reflects the aggregate influence of all the individual genes) suggests that at least a good portion of the involved genes (assuming many genes of approximately equal and small effect) must be operating in that manner for a difference in heritability by environment to be detected. Thus, it represents a good piece of evidence from which to build hypotheses about gene by environment interaction effects that may be associated with specific candidate genes.

In the CDP, we used latent class analyses to characterize trajectories of externalizing behavior from age 12 to 22 (Dick, Latendresse, et al., 2009). We identified two classes of trajectories of externalizing behavior: the majority of the sample (83%) showed a decrease in externalizing behavior from early adolescence to adulthood, whereas17% of the sample showed consistent, elevated levels of externalizing behavior that persisted into adulthood. The individuals showing this pattern of persistently high externalizing behavior were significantly more likely to carry the variant of *GABRA2* that was originally associated with increased risk for adult alcohol dependence in the COGA sample. Further, we found evidence that the association between *GABRA2* and trajectories of externalizing behavior was moderated by parental monitoring: the effect of the genotype on externalizing behavior was stronger under conditions of lower parental monitoring, weaker under conditions of higher parental monitoring (Dick, Latendresse, et al., 2009).

Subsequent analyses examined the association between *CHRM2* and trajectories of externalizing behavior (extended to a three-class solution with the incorporation of a nonlinear growth term) and tested for moderation by peer antisocial behavior (Latendresse et al., 2010). We found that, relative to the normative lower-risk externalizing trajectory, the likelihood of membership in the two higher risk trajectories increased with each additional copy of the risk allele at *CHRM2.* This association was exacerbated among those exposed to higher levels of peer group antisocial behavior. Accordingly, our findings were consistent with the evidence of moderation from twin studies, in which heritable influences were found to be higher under conditions of lower parental monitoring and higher peer deviance.

Twin studies are not the only place from which to draw hypotheses about environmental influences that are likely to moderate genetic effects. The developmental literature contains a wealth of studies demonstrating differential effects of the environment across children with differing temperaments and/or who differ on family history. Because temperament and family history provide information about the child's genetic predisposition, these kinds of interactions can also serve as starting points for developing hypotheses about GxE effects associated with specific genes.

In addition to the twin evidence suggesting that parental monitoring moderated the importance of genetic effects, there are numerous studies in the developmental literature suggesting the importance of this construct in moderating associations between early temperament/family history and the subsequent development of child behavior problems. For example, Bates and colleagues found that across two independent samples, a difficult childhood temperament was related to the subsequent development of externalizing behavior, but only in the context of lower parental control (Bates, Pettit, Dodge, & Ridge, 1998). Further, Molina and colleagues found that density of family history of alcoholism is related to the development of behavior problems in children, but only in the context of poor parenting (a measure that included reduced parental monitoring) (Molina, Donovan, & Belendiuk, 2010). These studies find that associations between predisposing factors (both known to at least partially reflect genetic influence) and child behavior problems are stronger under conditions of lower parental monitoring, paralleling the finding from twin studies that genetic influences were stronger under conditions of lower parental monitoring. They provided yet another compelling rationale to study parental monitoring as a moderator of the effects associated with specific candidate genes involved in substance use and externalizing behavior, the effect which we subsequently demonstrated with respect to *GABRA2* (Dick, Latendresse, et al., 2009).

GENE–ENVIRONMENT INTERACTION VERSUS GENE–ENVIRONMENT CORRELATION

This article focuses largely on GxE interaction, but discussion of genes and the environment as if they are separate sources of influence represents a clear oversimplification. Genetic and environmental influences are inexorably intertwined. Although some environmental influences may be largely random, such as experiencing a natural disaster, most measures of the environment show some degree of genetic influence, illustrating the active role

that individuals play in selecting and creating their environment (Kendler & Baker, 2007). To the extent that these choices are influenced by an individual's genetically influenced temperament and behavioral characteristics, an individual's environment is not purely exogenous, but rather, in some sense, is yet another extension and reflection of the individual's genotype. This concept is called gene–environment correlation.

The presence of GxE correlation complicates the interpretation of GxE interactions because differences in genetic effects in different environments may reflect differences in gene frequency if individuals are selecting themselves into different environments (rather than differences in the effect of genes as a function of the environment, as GxE would indicate). Accordingly, tests of GxE interaction usually control for GxE correlation, or explicitly test for GxE correlation (to ensure there is none for the variables being studied) before testing for GxE interaction. This is an inherently unsatisfactory solution, but it reflects the fact that our analytic methods are limited for capturing the complexity of developmental transactions.

In some sense, figuring out to what extent something is "genetic" and/or "environmental" is a moot point. What we are ultimately interested in is the unfolding of developmental trajectories that lead to outcomes of interest. If, for example, an individual is genetically predisposed toward sensation seeking, and this makes that individual more likely to spend time in bars (a GxE correlation), and this increases their risk for alcohol problems, are the predisposing "sensation-seeking" genes or the "bar environment" the causal agent? In actuality, the question is moot: they both played a role; it is much more informative to try to understand the pathways of risk than to ask whether genes or the environment were the critical factor. Although this review focuses on GxE interaction, it is important for the reader to be aware that this is but one process by which genetic and environmental influences are intertwined. Excellent reviews covering the nature and importance of GxE correlation also exist (Kendler, 2010).

CONCLUSIONS

Understanding the pathways of risk associated with identified susceptibility genes will be critical to potentially use genetic information in the future to inform prevention and intervention efforts. This must involve understanding how this risk unfolds across development, and in conjunction with environmental factors. Developmental scientists are well-equipped to make important contributions to this endeavor, with a long history of careful research on mechanistic processes, mediating and moderating variables, and articulated theory and conceptual frameworks to guide research. However, for this research to reach its potential, developmental scientists and geneticists need to reach across disciplinary boundaries and integrate the methods and findings from each respective field. For developmental scientists, this must involve going beyond studying "the usual suspects" and working with geneticists involved in gene finding projects to study novel genes currently being identified. It must also involve moving beyond studying one or two purportedly functional polymorphisms, and doing a more careful and thorough job of characterizing the gene of interest based on knowledge about the underlying gene structure.

Genetics has made great progress in recent years with the completion of the Human Genome Project and the International HapMap project. Developmental scientists must take advantage of these advances and utilize this information when integrating genetics into their projects. Until then, psychological genetic research risks being ignored and viewed as naïve, as it fails to reflect the current state of knowledge in genetics. Because genetics is a complex field that is advancing rapidly, collaborations between psychologists and geneticists will be critical to stay abreast of the changing genetic landscape. Further, psychology students who want to do interdisciplinary research should consider training in the area of genetics so that they can be equipped to converse across the fields and take advantages of the strengths of each. This could be accomplished through a postdoctoral experience or interdisciplinary doctoral training. K awards available through the National Institutes of Health represent a mechanism for faculty members at various career levels to obtain additional training; a desire to incorporate genetics into one's research and to learn more about the field seems an ideal foundation for one of these awards. In the surge of interest surrounding genetics, many psychologists know just enough to be dangerous. We must ensure that the contributions of developmental scientists to this area of research represent the best that our field has to offer.

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