Behavioral genetics, also commonly referred to as behavior genetics, can be broadly defined as the study of the inheritance of behavioral phenotypes (Crusio, 2015). Although it is a surprisingly old discipline going back to the 19th century, behavioral genetics is one of the most rapidly expanding areas of contemporary biology. In particular, the breathtaking speed of methodological development in the analysis of genes and genomes has transformed the understanding of the genetic basis of behavior and is paving the way for the emerging field of comparative behavioral genomics. Consequently, the limiting factor in the future of behavioral genetics might not be the gathering of genetic, genomic, or epigenetic data but rather gaining a deeper understanding of the behavioral variation explained by genes, the environment, and their interaction. In this chapter, our goal is to provide a concise introduction to the vast and wide-ranging research field of behavioral genetics and to describe its history and early controversies, conceptual and methodological advances, and outlook for the future.

NATURE VERSUS NURTURE BECOMES NATURE, NURTURE, AND THEIR INTERACTION

E. O. Wilson (1991), the famous evolutionary biologist, once wrote, “Behavior is the most intricate and revealing part of an animal’s natural history. If you know an animal’s behavior well, you know its essence” (p. xi). As in so many other areas of contemporary biology, behavioral genetics can be traced back to the work of Charles Darwin (1859); his Origin of Species included a chapter on instinct in which he described various aspects of animal behavior. Probably even more important than this chapter was the inspiration Francis Galton found in it; he was the first to popularize the terms nature and nurture in his studies of human intelligence (Galton, 1869, 1876), and he was also the first to advocate the use of twin studies (Galton, 1876).

The success of the idea of natural selection and the emphasis on the importance of inheritance in the early 20th century on the one hand and the rise of the social sciences on the other hand led to a strong countermovement advocating that behavior, especially human behavior, was not affected by genes (Boas, 1911; Watson, 1930). This behaviorism was an extremely influential school of thought for the next few decades (Montagu, 1968), and the rather dogmatic views on both sides intensified the nature-versus-nurture debate to sometimes absurd dimensions in the 1970s and 1980s (Lewontin, Rose, & Kamin, 1984), despite Hall’s (1951) conclusion 3 decades earlier that the dichotomy of nature and nurture was a pointless exercise.

The fact that humans have successfully bred domesticated animals such as dogs (Canis familiaris) for desirable behavioral traits indicates that many behaviors must have a genetic component (reviewed...
by Scott & Fuller, 1965). Similarly, a long-term selection experiment in foxes (Vulpes vulpes) has shown that many of the characteristics of domestic animals, both behavioral and morphological, can be brought about solely by selecting for tameness (Trut, 1999). A particularly striking early example of the power of artificial selection to generate behavioral differences comes from a study in which laboratory mice (Mus musculus) were selected over 30 generations for either high or low activity in an illuminated arena called an open field. Because some animals actively explored the arena and others remained largely immobile and showed signs of stress, the outcome of this test has been widely interpreted as a proxy for fearfulness. With each successive generation, the selection lines became more divergent, eventually differing in their activity levels by a factor of more than 30 (DeFries, Gervais, & Thomas, 1978; see Figure 18.1). This result indicates not only a strong response to selection, and hence that open field activity is at least partly under genetic control, but also that the trait must be polygenic; if only one or two genes were involved, the lines would be expected to stabilize after a few generations.

Since then, evidence has been accumulating at an increasing pace for the role of genes in behavior, including in humans (Boomsma, Busjahn, & Peltonen, 2002; Loehlin, 1989; Segal, 1999). However, behavioral variation need not always have a genetic component. For example, a recent study of genetically identical laboratory mice showed that individual differences in behavior can emerge as a result of developmental plasticity during neurogenesis (Freund et al., 2013). It has also been known since the 1950s that behavioral differences often result from the interplay of genes and the environment (so-called Gene × Environment interactions; reviewed by Manuck & McCaffery, 2014).

For example, Cooper and Zubek (1958) showed that rats (Rattus norvegicus) selected over several generations for being either good or bad at learning to find their way through a maze responded differently

![Figure 18.1](image-url)
to environmental enrichment. Thus, by the latter part of the 20th century, nature versus nurture had been largely replaced by nature and nurture and their interaction (Plomin, DeFries, Knopik, & Neiderhiser, 2013), and since then one of the key goals of behavioral genetics has been to quantify the relative contributions of genetic and environmental effects and their interactions across a variety of contexts (see Chapter 11, this volume). For an interesting account of the development of the behavioral genetics field, see Greenspan (2008). Early influential works are showcased by Hirsch and McGuire (1982), and Fuller and Thompson (1960) wrote the first book that explicitly defined behavioral genetics as a discipline.

QUANTITATIVE GENETICS

Quantitative genetics provides some very useful analytical tools for assessing the extent to which genes and the environment are responsible for behavioral variation (Boake, 1994). Quantitative genetic approaches aim to understand the inheritance of phenotypic traits that are expressed on a continuous scale—also referred to as quantitative or complex traits—which behaviors typically are. These traits tend to have a polygenic basis (i.e., they are influenced by many loci), and thus they cannot be readily studied using classical Mendelian genetics, which describes the inheritance patterns of discrete traits caused by the segregation of alleles at a single locus.

Quantitative genetics is a discipline with a long history that, after Galton’s (1869, 1886) initial work on within-family resemblance of human intelligence and height, was formally developed by R. A. Fisher (1919) and Wright (1921) in the early 20th century. In the 1950s, Hirsch introduced quantitative genetic analysis to the study of behavior with his pioneering work on the genetics of geotaxis in Drosophila (Hirsch & Tryon, 1956). Quantitative genetic approaches build on the principles of Mendelian genetics to estimate the magnitude of genetic effects on phenotypic variation while making the assumption that most continuous traits are influenced by numerous genetic loci with small effect sizes (the infinitesimal model). This can be achieved, in a nutshell, through various statistical approaches that attempt to quantify the extent to which known genetic relatedness between individuals is reflected in the similarity of their phenotypes (for an accessible introduction, see Falconer & Mackay, 1996).

An important underlying assumption of quantitative genetic approaches is that the phenotypic similarity of related individuals is due to shared genes, not a common environment. In animal studies, it is possible to control for the potentially confounding influence of the environment through cross-fostering experiments (e.g., Kruuk & Hadfield, 2007), and in humans it is necessary to draw on adoption cases or to contrast the phenotypic resemblance of monozygotic (identical) and dizygotic (fraternal) twin pairs, each of them raised in the same households (Boomsma et al., 2002). A recent comprehensive meta-analysis of these twin studies has pointed toward the shared environment’s having a relatively small influence on the majority of traits (Polderman et al., 2015).

Arguably the most straightforward quantitative genetic approach is parent–offspring regression. Here, values of a given phenotypic trait in offspring are regressed on their parents’ mean trait value (the mid–parent value). The slope of the resulting regression then provides an estimate of the relative magnitude of genetic versus environmental causes underlying the observed phenotypic variation, which is commonly referred to as the (narrow-sense) heritability ($h^2$). Heritability estimates can also be obtained by analyzing the phenotypic similarity (covariance) of other classes of relatives, including siblings and more distantly related individuals (Falconer & Mackay, 1996).

The heritability of a trait provides a measure of the proportion of the total phenotypic variance that is explained by alleles transmitted from the parents, which is also referred to as the additive genetic variance (i.e., $h^2 = \text{additive genetic variance} / \text{total phenotypic variance}$). Heritability scales between zero and one, with high values indicating that the observed phenotypic variation has a strong genetic component and low values indicating a predominant role of the environment. It is important to realize that heritability estimates are population specific because they depend on both the amount of available genetic variation and the specific environment in which this genetic variation is expressed (Visscher, Hill, & Wray, 2008). Obviously, the notion that genetic
effects depend on both the genetic background and the environment in which they are expressed is not restricted solely to quantitative genetic analysis (e.g., Crabbe, Wahlsten, & Dudek, 1999). Heritability estimates in natural animal populations vary substantially among different species and behavioral traits studied, but overall they average around 0.5 (reviewed in Mousseau & Roff, 1987; Postma, 2014). This average is comparable to the magnitude of heritabilities observed for morphological and physiological traits (Postma, 2014) and implies that in general both genes and the environment have an appreciable impact on the expression of behavior.

Evolutionary biologists and animal and crop breeders have a long-standing interest in quantitative genetics and the estimation of trait heritabilities (for a concise discussion of the concept of heritability and its use and limitations, see Visscher et al., 2008). This is not necessarily surprising, because the evolutionary response of a trait under selection is predicted to be proportional to its heritability (but note that in natural populations this prediction is not always straightforward; Morrissey, Kruuk, & Wilson, 2010). The application of quantitative genetics has proven extremely successful in animal and crop improvement (Hill & Kirkpatrick, 2010) and has gone hand in hand with the development of increasingly powerful statistical approaches for estimating quantitative genetic parameters from multigenerational pedigrees (so-called animal models; Henderson, 1975; Lynch & Walsh, 1998).

Animal models are a form of mixed model containing both fixed and random effects (see Chapter 8, this volume), which allow for the partitioning of the total phenotypic variance into its different variance components, including the additive genetic variance, while controlling for potentially confounding effects (e.g., resulting from relatives sharing the same environment) through the inclusion of additional random variables. Fixed variables can also be included to control, for example, sex and age effects. Animal models are particularly powerful because they exploit information from all possible pairwise phenotypic and genetic comparisons provided by the pedigree. Moreover, their flexibility allows for the explicit modeling of, for example, maternal effects, Genotype × Environment interactions and Genotype × Age interactions. They can also be implemented in a multivariate form to allow for the estimation of genetic correlations between traits, which measure the extent to which different traits are influenced by the same genetic factors (Kruuk, 2004; Kruuk, Slate, & Wilson, 2008; Lynch & Walsh, 1998).

In recent years, animal models have been adopted by evolutionary biologists studying natural populations (Kruuk, 2004; A. J. Wilson et al., 2010), leading to a surge in quantitative genetic studies of life history and behavioral variation in the wild (Charmantier, Garant, & Kruuk, 2014; Kruuk et al., 2008). This research makes use of data from long-term individual-based studies of wild animal populations, which provide a wealth of phenotypic and pedigree data that can be integrated within the context of natural environmental variation (Clutton-Brock & Sheldon, 2010). These studies have not only confirmed that behavioral traits tend to have substantial heritabilities in natural populations but have also facilitated more sophisticated analyses, such as of genetic correlations between behavioral and other ecologically important traits. For example, in an expanding population of North American western bluebirds (Sialia mexicana), both male territorial aggression and dispersal tendencies have a heritable component and are genetically correlated, thereby driving population dynamics (Duckworth & Badyaev, 2007; Duckworth & Kruuk, 2009).

Understanding the evolutionary mechanisms leading to the origin and maintenance of behavioral variation among individuals (see Chapter 11, this volume), also commonly referred to as animal personality, is currently a strong focus of behavioral and evolutionary research (Dingemanse & Wolf, 2010; Réale, Reader, Sol, McDougall, & Dingemanse, 2007; see Exhibit 18.1). Understanding this variation could be relevant for animal husbandry and breeding (Adamczyk, Pokorska, Makulska, Earley, & Mazurek, 2013; Friedrich, Brand, & Schwerin, 2015; see also Volume 2, Chapter 35, this handbook), animal conservation, and the control of invasive species (Sih, Cote, Evans, Fogarty, & Pruitt, 2012). It may also inform psychologists and provide additional insights into the evolutionary origin and maintenance of human personality variation (Gosling, 2001; Penke, Denissen, & Miller, 2007). Likewise, research on personality in...
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In question. An interesting case in point is provided for partitioning behavioral variation into its genetic and environmental components but falls short of identifying the specific genes involved. Bottom-up approaches based on candidate genes have therefore been developed. Here, specific loci are selected for screening based on a priori knowledge of the gene’s biological function and potential relevance to the trait in question. An interesting case in point is provided by the neurohypophysial hormone arginine vasopressin (AVP) and its brain receptor subtype (AVPR1a), which are known to be involved in diverse aspects of mammalian social behavior (Goodson & Bass, 2001; see also Chapters 19 and 22, this volume). Administration of AVP increases pair-bonding behavior in monogamous male prairie voles (Macrotus ochrogaster) but not in males of a closely related but nonmonogamous species, the montane vole (M. montanus; Winslow, Hastings, Carter, Harbaugh, & Insel, 1993). This appears to reflect differences in the distribution of AVPR1a in the brain (Young, Winslow, Nilsen, & Insel, 1997), which in turn are associated with a genetic difference between the two species in the promoter region of the AVPR1a gene (Young,
Remarkably, male transgenic mice carrying the prairie vole AVPR1a locus show expression patterns and pair-bonding behaviors resembling those of prairie voles (Young et al., 1999), experimentally confirming the key role of a single locus in generating interspecific variation in behavior.

A further approach to investigate the causal effects of candidate genes on behavioral phenotypes is to “knock out” or render a target gene dysfunctional. Here, a specific gene is adjusted in its sequence in such a way that either it is not transcribed at all or its expression is changed. The altered gene is then transferred into an embryo, and once the resulting population is homozygous for the gene, the effect on phenotypes can be investigated. This sort of approach has been used extensively in Drosophila fruit flies and mice, with several thousand knock-out lines having been established in the latter, including more than a hundred lines alone for evaluating alcohol consumption (Crabbe, Phillips, Harris, Arends, & Koob, 2006). Although this approach is, like any other, not without its problems (Crusio, Goldowitz, Holmes, & Wolfer, 2009), knock-out studies “have undoubtedly contributed enormously to our understanding of how genes influence behavior” (Crusio, 2015, p. 91).

Knock-out studies of laboratory organisms have proven especially useful for exploring associations found in humans, which cannot otherwise be confirmed experimentally. An example of this is provided by recent studies of a polymorphism within the promoter region (5-HTTLPR) of the serotonin transporter gene (SERT). This polymorphism gives rise to two common alleles that differ in their transcriptional activity and thus their serotonin binding capability. Because serotonin is crucial for the regulation of cognition, emotion, sleep, and endocrine activity, among other functions, the polymorphism has long been considered a strong candidate for explaining human behavioral variation (reviewed by Savitz & Ramesar, 2004). Accordingly, some very interesting associations have been found. For instance, it was recently shown that individuals carrying the low-expression allele show a more negative interpretation bias (i.e., are pessimists) than those with the high expression genotype (who are optimists; Fox & Standage, 2012). However, a review of 36 human studies recovered mixed results, with only 18 studies reporting significant associations, and roughly half of these not being in the direction expected given the effects of the two alleles on transcriptional activity (Savitz & Ramesar, 2004). Nevertheless, there is reason to believe the gene may have a causal effect, because a mouse knock-out model has revealed clear effects of gene alteration on anxiety and depression-related behaviors, exploratory behavior, aggression, and the stress response (reviewed by Holmes, Murphy, & Crawley, 2003).

Although candidate gene approaches have in many instances been successful at identifying causal genetic variants, there are also cases in which associations initially reported have proven difficult to reproduce. For example, a recent large-scale study that sought to replicate associations between 55 different candidate genes and major depressive disorder could only replicate four of these associations (Bosker et al., 2011). In general, candidate gene studies are more likely to be successful when applied to traits that are underlain by simple genetic mechanisms and when there is solid a priori physiological information on, for example, the signaling pathways and receptors involved (e.g., Young et al., 1999).

GENE MAPPING

Especially in the absence of a priori information, it becomes desirable to obtain a genomewide perspective on behavior. This perspective can be achieved through a variety of top-down approaches that exploit genetic markers to map the genomic regions responsible for phenotypic variation, with a view toward identifying the causal loci (for a concise overview of gene-mapping approaches, see Schielzeth & Husby, 2014). Conventional quantitative trait locus (QTL) analyses exploit backcrosses between inbred selection lines to facilitate the detection of associations between mapped genetic markers and a phenotypic trait (Mackay, Stone, & Ayroles, 2009). A classic example of QTL mapping to elucidate the genetic basis of behavior comes from a study of open-field activity in mice (Flint et al., 1995). Here, QTLs were identified on three different chromosomes, all of
which could subsequently be replicated (Turri, Henderson, DeFries, & Flint, 2001). Genetic dissection of one of these QTLs identified a mutation within Rgs2, a gene that is widely expressed in the brain and that is known to modulate anxiety (Oliveira-dos-Santos et al., 2000; Yalcin et al., 2004). More recently, QTL mapping has also proven successful at identifying genomic regions associated with complex behaviors, such as burrowing, in natural populations (Weber, Peterson, & Hoekstra, 2013).

QTL mapping studies based on backcrosses or known pedigree information are gradually being superseded by genome-wide association studies (GWAS). Here, very high densities of mapped genetic markers (typically hundreds of thousands to millions of single-nucleotide polymorphisms) are used to search for genetic associations in outbred populations (for a detailed description, see McCarthy et al., 2008). Until now, GWAS have largely concentrated on the pathologies of behavioral and cognitive traits in humans and animal models (Flint & Eskin, 2012; Stranger, Stahl, & Raj, 2011; but see de Moor et al., 2012, for a GWAS of personality traits). An example is schizophrenia, a highly heritable psychiatric condition (Sullivan, Kendler, & Neale, 2003). The largest GWAS on this disorder to date analyzed more than 36,000 schizophrenia cases and 113,000 controls to detect more than 100 schizophrenia-associated loci (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014; see Figure 18.2). This study confirmed the already-known importance of the dopaminergic pathway and also implicated a suite of additional genes, thereby opening up avenues for further research and drug development.

Over the past decade, GWAS studies have increased dramatically, both in number and in scope, and have identified many hundreds of genetic variants associated with disease and other traits (reviewed by Welter et al., 2014). However, this approach has also been criticized because of the difficulty of replicating genetic associations (Ioannidis, 2007) and because the genetic variants identified by GWAS often explain very little of the trait’s heritable variance, also known as the missing heritability problem (Maher, 2008; Manolio et al., 2009). For
example, despite having analyzed a large sample of 7,900 individuals genotyped for 350,000 single-nucleotide polymorphisms, Davis et al. (2010) recently concluded that “the genes associated with childhood cognitive ability remain tantalizingly beyond our current reach” (p. 760).

**COMPARATIVE GENOMICS**

The rapid development of genomic techniques has opened up the possibility of obtaining genomewide sequence data, not only in traditional model species (e.g., humans, mice, fruit flies, nematodes) but essentially in any organism. By now, the genomes of more than 2,400 species of eukaryotes have been sequenced, including at least 164 mammal, 65 bird, 11 reptile, and 62 fish species (National Center for Biotechnology Information; http://www.ncbi.nlm.nih.gov). Comparative genomics uses this growing availability of genomic data to compare sequences among species to infer evolutionary histories and investigate genetic footprints of past selection and adaptation.

A prominent example of how comparative genomics has been applied to human behavior is provided by studies of the evolutionary genetics of speech and language. This work began with the discovery of a defect in a transcription factor coding gene named *FOXP2*, which causes impairment of speech (Lai, Fisher, Hurst, Vargha-Khadem, & Monaco, 2001; for a review, see Preuss, 2012). Subsequently, it was discovered that, although *FOXP2* is highly conserved across primates, two amino acid substitutions are fixed in humans (Enard et al., 2002). Because these mutations seem to have arisen around the first appearance of *Homo sapiens*, it has been speculated that they could have enabled the complex orofacial movements needed for speech. However, *FOXP2* knock-out mice exhibit slowed development, general motor impairment, and reduced spontaneous activity, pointing toward a more complex etiology (reviewed by S. E. Fisher & Scharff, 2009).

**FUTURE OF BEHAVIORAL GENETICS, BEHAVIORAL GENOMICS, AND BEYOND**

The quest has moved on from simply demonstrating that genetic factors are important to estimating the heritability of behavior and pinpointing the genes involved. Increasingly sophisticated analytical methods are providing new perspectives on the interpretation of abundant genetic information provided by GWAS, and emerging fields such as functional genomics and epigenetics are opening new avenues for understanding the precise genetic mechanisms, including gene interactions and expression patterns, that underpin behavior.

Focusing first on analytical developments, new methods have recently emerged for GWAS data that appear to resolve the long-standing problem of the missing heritability (reviewed by Vinkhuyzen, Wray, Yang, Goddard, & Visscher, 2013). GWAS studies apply stringent significance thresholds to minimize false positives. However, this typically results in the discovery of a limited number of genetic variants that collectively explain far less trait variance than expected given the heritability of the trait. Initially, this mismatch was attributed variously to interactions between genes (dominance and epistasis), Gene × Environment interactions, or incorrect estimates of heritability (Manolio et al., 2009). However, new methods rooted in quantitative genetics have been developed to combine the effect sizes across all of the genetic variants, regardless of individual significance, and these effects explain a far larger fraction of the trait variance (Yang et al., 2010). These approaches also allow partitioning of the total phenotypic variance, both according to effect size (see Figure 18.3A) and across chromosomes (see Figure 18.3B), thereby providing insights into the underlying genetic architecture (Moser et al., 2015). For example, a recent study of seven disease-related traits in humans found a marked contrast between bipolar disorder and Type 1 diabetes, the former being attributed to many variants of very small effect size and the latter to relatively fewer variants of larger effect size (see Figure 18.3A; Moser et al., 2015). This is consistent with the idea that many behavioral traits are highly polygenic. Further evidence from GWAS points toward predominantly additive genetic effects contributing to complex trait variance at the population level (Robinson, Wray, & Visscher, 2014; for a different view, see Nelson, Pettersson, & Carlborg, 2013), despite the likely presence of abundant
epistatic effects at the molecular level (Phillips, 2008).

Identifying specific genetic variants and partitioning the phenotypic variance are only the first steps toward understanding the connection between genes and behavior. Functional genomics attempts to bridge the gap using a diverse toolkit of high-throughput methods, such as RNA-seq (Wang, Gerstein, & Snyder, 2009), to investigate gene expression patterns and to characterize gene networks (see Chapter 5, this volume). For example, differences in gene expression can be measured between selection lines for high and low trait values, or between individuals expressing normal and pathological behavior, to link gene transcription to behavioral variation. For example, transcriptome-wide analysis has shown that autism appears to be associated with diverse transcriptional changes in the brain that involve a suite of both immune regulatory and neuronal genes (Gupta et al., 2014).

Finally, a greatly improved understanding of the inheritance of behavioral variation may be obtained by incorporating epigenetics, the study of heritable changes in gene expression and phenotype that occur without changes to the DNA sequence (Jensen, 2015; see also Chapters 11 and 22, this volume). Epigenetics provides a wonderful example of how nurture can influence nature (Powledge, 2011), and the appeal of epigenetics for explaining behavioral variation can hardly be overstated (Miller, 2010). Epigenetic changes, most notably DNA methylation and histone modification, affect all aspects of the behavioral control system, from sensory input to motor output, and may be transmitted through the germline to the next generation, leading to transgenerational effects on behavior (Heard & Martienssen, 2014; Jablonka & Raz, 2009; Jensen, 2015). In rats, the amount that a mother licks and grooms her young affects how these offspring will respond to stress in later life, providing a nice early example of epigenetic programming. This effect is mediated by epigenetic modification of the glucocorticoid receptor gene in the hippocampus (Weaver et al., 2004), which in turn is associated with the differential expression of more than 900 genes (Weaver, Meaney, & Szyf, 2006). Interestingly, similar patterns of hippocampal glucocorticoid receptor methylation are found in suicide victims.

FIGURE 18.3. Inference of the genetic architecture of seven different disease traits in humans from high-density SNP data. A: Proportion of additive genetic variation contributed by SNPs of different effect sizes (indicated by different colors; mixture component). B: The proportion of additive genetic variance for bipolar disorder explained by SNPs of different effect sizes, partitioned by chromosome. BD = bipolar disorder; CAD = coronary artery disease; CD = Crohn’s disease; HT = hypertension; RA = rheumatoid arthritis; SNP = single-nucleotide polymorphism; T1D = Type 1 diabetes; T2D = Type 2 diabetes. From “Simultaneous Discovery, Estimation and Prediction Analysis of Complex Traits Using a Bayesian Mixture Model,” by G. Moser, S. H. Lee, B. J. Hayes, M. E. Goddard, N. R. Wray, and P. M. Visscher, PLOS Genetics, 11, e1004969. Copyright 2015 by G. Moser, S. H. Lee, B. J. Hayes, M. E. Goddard, N. R. Wray, and P. M. Visscher. Adapted with permission.
with a history of childhood abuse (McGowan et al., 2009). The field of behavioral epigenetics has been rapidly expanding, and epigenetic effects have been implicated in anxiety (Kaminsky et al., 2008), learning and cognition (Day & Sweatt, 2010), addiction (Wong, Mill, & Fernandes, 2011), obesity (Campbell, Mill, Uher, & Schmidt, 2011), schizophrenia (Abdolmaleky, Thiagalingam, & Wilcox, 2005), and depression (Pariante & Lightman, 2008).

CONCLUSIONS

This chapter provides a flavor of the long history, enormous breadth, and rapid ongoing development of the study of behavioral genetics. Genomic technologies are developing so rapidly that arguably one of the greatest remaining challenges will be to obtain a better understanding of the behavioral phenotypes themselves. Although laboratory studies are essential for unraveling the genetic mechanisms underlying differences in behavior, studies of natural populations can also help us to understand the functional significance of behavioral variation and the selective pressures explaining its evolutionary origins and maintenance (see Chapter 3, this volume).

References


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