Chapter 1

Why is the topic of genes and behavior controversial?

In this book, I set out to explain why the topic of genetics is so important for all of us, and specifically how it can be very informative with respect to questions about the causes and course of both mental disorders (such as depression or schizophrenia) and normal psychological characteristics (such as variations in scholastic achievement or **personality** characteristics). In the course of outlining the real value of genetics, I will, however, need to point out the "hype" and exaggeration associated with some genetic claims, as well as the widespread misunderstanding of genetics by some critics who have opposed its influence.

Before turning to a consideration of why the topic of genes and behavior has proved to be so surprisingly controversial, thereby setting the scene for the book, I need to say something of the achievements and claims of genetics. These involve both basic laboratory science and more applied studies.

Accomplishments of genetics

The history of genetics goes back to the mid-nineteenth century when, using studies of pea plants, Mendel (the Austrian monk, also trained as a scientist) concluded that genes were particulate factors that were passed on from generation to generation, each **gene** existing in alternate forms, now called **allele**s (see Lewin, 2004). Curiously, the importance of his discovery was not recognized at the time; indeed, it was not appreciated until well after his death. Also, it was not until the mid-twentieth century that it became clear that deoxyribonucleic acid (**DNA**) constituted the relevant genetic material. However, even then, there was no understanding of how it might work.

At the time I was a medical student in the early 1950s it was not even known how many chromosomes humans had (that was discovered in 1956) and there was discussion of how Down syndrome might be a result of stress!

(Its origin in an extra chromosome 21 was discovered only in 1959[1].) With respect to the basic biological mechanisms, the key breakthrough came in 1953, with the discovery by Watson and Crick that DNA had a paired helix (corkscrew) structure. In wonderful understatement, they concluded their paper by stating: "It has not escaped our notice that the specific pairing we have postulated immediately suggests a possible copying mechanism for the genetic material."[2] Another key step was Fred Sanger's description in 1977 of how to determine the precise sequence of **nucleotides** in any strand of DNA. Both these discoveries rightly led to Nobel prizes. Over the second half of the twentieth century, there was an awesome and spectacular series of scientific discoveries in molecular biology (some of which gave rise to further Nobel prizes), leading to a rich understanding of the detailed biology of how genes operated, a few key details of which are outlined in Chapter 7.[3]

Quite apart from the basic science elucidation of the biological mechanisms underlying gene action, technological (and conceptual) advances paved the way for the identification of genes associated with the liability to specific diseases. Perhaps the first crucial step was the discovery that enzymes could be used to cut the DNA at a particular sequence. A further step was the discovery of polymorphic markers (meaning that they took several forms that varied from individual to individual) that extended across the whole genome. The first type was called a restriction fragment length polymorphism (RFLD) but these have been largely superseded by microsatellite simple sequence repeats (SSRs) and more recently by single nucleotide polymorphisms (SNPs) – the advantage of the more recent developments being the far greater number of markers available. Two other advances had also revolutionized molecular genetic possibilities. First, the discovery of the polymerase chain reaction in the mid-1980s made it possible to have selective amplification of specific target **DNA sequence**s and permitted the cloning (i.e., reproduction) of genes, so facilitating their study. Second, high speed robotic methods were developed that enabled rapid screening of the whole genome for the markers being used. In addition, there have been important advances in the statistical methods needed in gene identification. Lastly, it is necessary to emphasize the importance of the discovery of the major extent of the overlap in genes across animal species, thereby making it possible to learn lessons from research on other organisms (including yeast and the fruit fly) and to test hypotheses on gene function through **animal models**.

The consequence of these revolutionary developments has been the identification of the individual genes responsible for a huge number of single-gene medical conditions (meaning those due to genes without the need for specific environmental factors – see Chapter 6). Progress has been slower with respect to the genes involved in susceptibility to multifactorial disorders

in which there is a complex interplay among multiple genetic and environmental **risk factor**s but, as discussed in Chapter 8, progress is now being made.

It might be thought that, although all of this is undoubtedly tremendously exciting from a scientific perspective, it may not have provided much understanding of the genetic issues involved in specific medical conditions. However, there have been important clinical advances, as exemplified by some of the unusual genetic mechanisms discussed in Chapter 6.

Because scientists recognized the huge medical, as well as scientific, potential of an adequate understanding of how individual genes worked and how they brought about their effects, the internationally collaborative Human Genome Project (HGP) was launched in 1990 to sequence the entire human genome. Draft reports were published in 2001 by both the HGP and a rival commercial concern, Celeron Genomics.[4] A further report was published in 2004. One key finding is that the number of protein-coding genes (20–25,000) is quite a lot less than used to be thought. This has implications for an understanding of gene action – discussed in Chapter 7.

In parallel with early developments in genetic mechanisms in the first half of the twentieth century, and the rapidly burgeoning field of molecular biology in the second half of that century, there was the somewhat separate development of population quantitative genetics. In a real sense, Francis Galton's study in the mid-nineteenth century of how talent ran in families provided the forerunner for this field, but it was the statisticians Karl Pearson and Ronald Fisher, together with the geneticist J.B.S. Haldane, who provided the foundation for this branch of genetics. Twin and adoptee studies were used to great effect to determine the relative importance of genetic and environmental influences on psychological **trait**s and on mental disorders. With respect to the latter, Eliot Slater was a key figure, both through his setting up of the Maudsley Hospital Twin Register in 1948 and through the research in his own Medical Research Council Psychiatric Genetics Unit. During the second half of the twentieth century there were crucially important developments in both sampling and statistics and, as a result, an impressive corpus of knowledge was built up on the heritability of a wide range of psychological traits and mental disorders.[5] It was important, too, that there was a much more critical approach to twin and adoptee studies than there had been in the earlier days.[6] In particular, researchers appreciated the necessity of combining different research strategies. The outcome was the demonstration that genetic factors played a significant role in individual differences in the liability to show almost all human psychological traits and to suffer almost all mental disorders. In a few instances (such as **autism** and **schizophrenia**), genetic influences predominate, but in a larger number they

are contributory to a lesser degree (accounting for some 20 to 60 percent of the variance in the general population).

Three key findings were particularly important. First, for the great majority of traits or disorder, *both* genetic and environmental factors were influential – meaning that any neat subdivision into those due to nature and those due to nurture was bound to be misleading. Second, except in rare circumstances, genes were not determinative of either psychological characteristics or mental disorders, and their influence involved a complex mix of direct and indirect effects operating on different parts of the causal chains. Third, the pervasive impact of genetic influences extended to social behaviors and attitudes, and even to the likelihood of experiencing particular types of risk environments.

It might be thought that these spectacular developments in genetics over the past half-century might be universally welcomed for the benefits that might be expected to follow. However, the reactions of professionals and the lay public alike have been quite mixed and I need to turn now to what is entailed in the various controversies.

Supposed lack of medical utility

Le Fanu,[7] whilst appreciative of the science, described "The New Genetics" as one of the major failures of modern medicine. He drew attention to the fact that the promise that genetic research would elucidate the causes of disease and thereby lead to effective new methods of treatment and of prevention had simply not been fulfilled because it had had such extremely limited success in producing successful genetic engineering, genetic screening, and gene therapy. He went on to argue that this was because, on the whole, genes do not play an important role in disease and, when they do (as in single-gene conditions such as cystic fibrosis), the genetic effects are so complex and elusive that not much can be done about them. He was undoubtedly correct in his assessment that gene therapy had been oversold,[8] and that genetics had not led to dramatic gains in drug discovery. Nevertheless, his conclusion was both premature and unduly pessimistic. The mistake was to equate genetic influences with single-gene disorders (it is correct that these account for a tiny proportion of medical conditions) and to assume (following, it has to be said, some genetic evangelists) that gene identification itself will elucidate the causes of disease (it will not). As discussed in Chapter 4, the findings from twin and adoptee studies are compelling in showing that genetic influences are highly important (albeit not determinative) in all medical conditions, including mental disorders. But the genes, in almost all cases, operate together with environmental influences as part of multifactorial causation (see Chapter 2 for a discussion of what this means).

The premature nature of the dismissal of "The New Genetics" arises from two different considerations. First, and most crucially, it ignores the need for biological research that uses pointers from genetics but which goes beyond it in order to elucidate *how* the causal influences operate. Gene discovery on its own will not do that. As discussed in Chapter 7, DNA itself does not cause any kind of disease process and, hence, identifying an individual gene that predisposes to some disease outcome is not directly informative. As Bryson[9] puts it, in his very readable popular gallop across the field of science, the cracking of the human genome constitutes only the beginning because it does not indicate *how* effects come about. Proteins are the workhorses that provide the action and, so far, we know remarkably little about their activity in relation to disease (and even less in relation to behavior). The term "**proteomics**" was introduced a few years ago to cover the new research field of the operation of protein interactions. If we are to understand how genes are involved in the causation of disease we will need major advances in proteomics and that will take time to happen.

However, understanding the chemistry, crucial though that is, will not be enough on its own. There is the further need to elucidate the complex pathways through which the chemical effects play a role in leading to a particular disease or a particular trait or characteristic. That will require an integrative physiology that moves from cell chemistry to whole body physiology, and that develops and tests hypotheses or ideas on how the processes may lead to the outcome being considered. In addition, there will be a need for the rather different field of molecular epidemiology in order to understand the interplay between genes and the environment as a crucial part of the causal processes. All of this is potentially doable but it will take time (many decades and not just months or a few years) and we are only just learning how to pursue the long path from gene discovery to determination of the causal processes.[10] The dismissal of "The New Genetics" was also premature because it failed to appreciate the time span required to identify the genes implicated in multifactorial disorders and traits. Again, the scientists have been responsible for arousing expectations that cannot be met. Thus Plomin and Crabbe[11] some five years ago claimed that we will soon "be awash with susceptibility genes." As Chapter 8 indicates, important progress has been made but it continues to prove quite difficult to identify genes for multifactorial traits (somatic or psychological) because most genes have such small effects and because their effects are often contingent on environmental circumstances (see Chapter 9 for a discussion of **gene–environment interactions**). As I hope this book will demonstrate, there is every reason to suppose that "The New Genetics" will deliver the goods but it will do so only if it combines effectively with other branches of science.

The supposed poor quality of the evidence from twin and adoptee studies

Quantitative behavioral genetics (as distinct from medical genetics) has been subject to particularly scathing and sweeping attacks on the supposed poor quality of twin and adoptee studies, as well as on the basic concept of heritability as applied to individual variations in psychological characteristics.[12] As discussed in Chapters 3 and 4, it has to be accepted that some of the methodological criticisms, particularly of the earlier research, has some validity. Not enough attention has been paid to questioning the assumptions of the twin design[13] and there has often been a failure to appreciate the consequences of the restriction in environmental range in adoptive families,[14] as well as concerns over sampling issues[15] and the effects of biased participation in studies.[16] These criticisms have some validity but the critics who have been keen to dismiss the whole of behavior genetics[17] have been equally guilty of selective attention to research findings. Any dispassionate critic would have to conclude that the evidence in favor of an important genetic influence on individual differences is undeniable, even though there are reasonable uncertainties over the degree of population variance accounted for.[18]

Three main points are relevant. First, particular attention needs to be paid to the studies that have addressed the methodological challenges most successfully (see Chapter 4). Second, attention needs to be paid to the extent to which different studies (with contrasting patterns of strengths and limitations) give rise to the same conclusions. Third, it is necessary to ask how likely it is that environmental influences could account for the overall pattern of findings. It is obvious that they could not. Opinions may reasonably differ on the strength of genetic influences but there can be no reasonable doubt that they are important.

Fraud and bias in behavioral genetics

A further concern stems from the evidence that behavioral genetic research has occasionally involved outright fraud, as exemplified by Cyril Burt's twin data.[19] Burt was a very distinguished British academic psychologist who undertook an important pioneering epidemiological study of mental retardation, who did much to establish applied psychology as a profession, and who played a key role in the development of factor analysis (a statistical method for studying how traits group together). However, he was also a strong proponent of the strength of genetic influences on intelligence and his published twin findings (for a variety of good reasons) came to be

suspected of fraudulent manipulation. Some of the protagonists of behavioral genetics (especially those focusing on IQ) have strenuously sought to deny or downplay the evidence of fraud.[20] However, most dispassionate reviewers have concluded that the evidence of manipulation of data is sufficiently strong for it to be necessary to exclude Burt's data on the grounds of their untrustworthiness. In addition to rank fraud, there has also been concern over the ways in which some behavioral geneticists have been quite biased in their approach to research evidence.[21] These are serious scientific concerns, but it is important that the conclusions on genetic influences are much the same whether or not the disputed data are included or excluded. Nevertheless, the slipperiness has definitely not helped the behavioral geneticists' cause. It has been most unfortunate that, because some behavioral geneticists have been reluctant to accept the reality of fraud and bias, the far greater volume of high quality twin research has been unfairly castigated.

Acceptance of funding from organizations with an axe to grind

A somewhat related concern is that some behavioral geneticists have appeared to support the racist use of genetics and have definitely been willing to accept financial support for their research from highly suspect organizations. Thus, Eysenck and Jensen have seen no problem in their accepting funding from the Pioneer Fund, which has been widely regarded as having racist aims. Hans Eysenck, like Burt, was a very distinguished academic psychologist in London. He undertook some very important quantitative studies of personality dimensions as they related to mental disorder, and through his disciples he pioneered the use of behavioral methods of psychological treatment. He was a brilliant teacher and communicator and was a most effective popularizer of psychology, through a series of very readable paperbacks. However, he was also an enthusiastic controversialist in relation to race and IQ, smoking and cancer, and astrology. Throughout his career, he was suspected of being a bit dodgy in his use of evidence,[22] although he was never formally investigated for fraud. Nevertheless, his employing institution required him to hand back a research grant he had obtained from the Pioneer Fund for a study that he had "overlooked" submitting to the Ethics Committee.

Arthur Jensen, an American academic psychologist, is a world expert on the concepts and findings with respect to the notion of "g" as the central biological core of general intelligence.[23] He has undertaken some very important high quality research on this topic but, with respect to concerns over genetics, he is particularly associated with a scholarly paper that argued

that the on-average lower IQ of African Americans as compared with Whites was likely to be due to their genetic endowment and also that attempts to raise IQ through educational interventions were doomed to fail.[24] Although he has been unwilling publicly to admit it, his arguments are known to be flawed (because it is not justifiable to infer the cause of a between-group difference on the basis of within-group findings[25] and because scarcely any of the twin data was on African Americans). He has argued that there has never been any attempt by the funding organization to censure his reporting of evidence[26] but there is good evidence that the source of funding does influence the ways in which findings are reported – as evident in the source of funding of drug studies.[27] Eysenck[28] similarly always argued that his critical discussion of the links between smoking and lung cancer were uninfluenced by his extensive support from tobacco companies. However, there is good evidence that British American Tobacco did suppress scientific findings[29] and, frankly, it is naïve to suppose that it is irrelevant who funds a particular scientist's research. Quite rightly, recommendations on ethical guidelines now stipulate that funding sources must be taken into account.[30]

In addition, there have been concerns over the misuse of genetic findings in support of discriminatory **eugenics** practices. Thus, for example, on the basis of eugenic principles, in the mid-1930s some 20,000 Americans were sterilized against their will.[31] Nazi Germany carried things even further, with some 322,000 suffering the same fate between 1934 and 1939. Of course, it is true that these abhorrent policies were based on a misunderstanding of the genetic findings but it is the case that they were supported by some very distinguished geneticists. Most people would consider that, although this historic past is both deplorable and extremely regrettable, it is not relevant to the situation today. But is that so? Müller-Hill[32] suggested that when susceptibility genes for IQ are discovered, there may well be a reemergence of concepts of genetic superiority and inferiority (because of views about IQ – see below) with consequent eugenic temptations. Also, there are reasonable concerns over the views of some distinguished (but ethically naïve) geneticists that "designer babies" (chosen on the basis of their genes) are an appropriate way forward.[33]

The holy grail of identifying the genes for intelligence

It is obvious that behavioral genetics has no particular focus on IQ or general intelligence; rather, it is concerned with genetic and environmental influences on all psychological characteristics and mental disorders. Nevertheless, it is the case that controversies have particularly concentrated on claims regarding the **heritability** of IQ. Kamin's book[34] on *"The Science and Politics of IQ"*

includes the claim that: "there exist no data which should lead a prudent man to accept the hypothesis that IQ scores are in any way heritable." In fact, there is abundant evidence to indicate the importance of genetic influences on individual differences in IQ – most estimates put the heritability at about 50 percent. However, the basic critique is less about the precise level of heritability than it is about the tendency of some genetically minded psychologists to argue that a few traits are of such overwhelming importance that it is desirable that everyone should possess the same outstanding qualities. Thus, a regrettably large number of writers have sought to elevate IQ to a superordinate position in which it is seen as *the* human quality that is more important than all others, so that social or ethnic groups that are supposedly lacking in IQ should be treated differently and that the search for the genes that influence IQ should constitute the holy grail of behavioral genetics.[35] Of course, there is no denying that high IQ is quite a strong predictor of worldly success – both educational and occupational. Moreover, this appears to be the case in societies that differ widely in their political and social circumstances.[36]

On the other hand, follow-up studies of very high IQ individuals have shown that they are by no means all universally successful in adult life. Many human qualities other than IQ are vitally important in successful human adaptation. We are social animals, as well as thinking, talking animals, and success in a broad sense is much influenced by skills in social relationships, as well as by general intelligence. It would be foolish indeed to focus exclusively on IQ to the neglect of a much broader range of important adaptive human qualities. Also, however, it would be equally foolish to assume that it is desirable that everyone should be of high IQ and that genetic manipulation should be used to "design" high IQ children. To begin with, that could well mean inadvertent disadvantageous effects on other desirable human qualities. But, also, it is extremely questionable whether it would be either biologically or socially beneficial if everyone were similar with respect to high intelligence. Individual variation is an intrinsic part of biology and it would be ridiculous, as well as completely hopeless, to attempt to remove such individual differences and to seek to make everyone the same.

The supposed inequalities associated with individual differences

From a biological perspective, it is positively desirable to have individuals (both human and other animals) that vary in their skills, qualities, and limitations. There is no one "model" that would be ideal for all conditions, and there never could be. Traits that make for adaptability and success in

one environment may not work so well in others. It is biologically advantageous for there to be individual variation so that there can be successful adaptation to new environmental conditions as and when they arise. That is, of course, one of the key features of how evolution takes place, and it is a central concept in genetics.

Nevertheless, over the years, social reformers and social scientists have often been concerned that individual differences create social inequalities, which are inherently undesirable. There is, indeed, much evidence of the adverse health consequences stemming from wide social inequalities, with the ill-effects evident in those at the bottom of the social hierarchy.[37] The precise causal mechanisms of these effects of social inequity remain ill-understood,[38] although part of the disadvantage stems from limitations in the availability and access to medical and other services[39] and part from lifestyle effects on features such as smoking, diet, and exercise. It is a matter of legitimate concern that social inequities have increased in countries such as the USA (especially) and the UK, with the gap between the rich and poor increasing.

However, this is not at all equivalent to a desire to remove individual differences. Tawney expressed the issue this way: "While . . . natural endowments differ profoundly, it is the mark of a civilized society to aim at eliminating such inequalities as have their own source, not in individual differences, but in its own organization . . . Individual differences that are the source of social energy are more likely to ripen and find expression if social inequalities are, as far as practical, diminished."[40] In other words, the problem lies in the ways in which societies put in place artificial disadvantaging "blocks" that impede people's performance and which prevent them reaching their potential and exercising their skills to their best advantage. It is clear that such "blocks" are brought about by discriminatory housing policies, lack of educational opportunities, and the various forms of racial and religious discrimination that are endemic in most societies. It is crucially important that a focus on the importance of genetic influences does not lead to a neglect of these vitally important societal influences. We need to understand better how they operate and we need to take the appropriate societal actions to deal with the damaging and disadvantaging inequities. But that should not get muddled up with a futile, and damaging, quest to get rid of biologically influenced individual differences.

Nevertheless, some psychologists have been worried that a focus on genetics may divert interest and attention from the important social influences on behavior.[41] It has to be admitted that this worry has both historical and contemporary roots in the writings of genetic enthusiasts. Thus Eliot Slater, who did so much to establish psychiatric genetics in Britain (see above), was notoriously hostile to social psychiatry and to those working in that field.[42]

Also, his championing of biology was associated with an uncritical advocacy of brain surgery as a treatment for mental disorders – so much so that he saw this likely to develop as a distinct specialty.[43] Similarly, Steven Pinker – a most distinguished language expert – set up the ridiculous "straw man" that non-geneticists believed that the mind is "a blank slate" (meaning that nurture can change everything) and thereby condemned the entire field of social research.[44]

The worry that a focus on genetics may lead to a neglect of social influences has some validity in addition because the dominance of genetic, and broader biological, concepts focuses on individual differences rather than on levels of either disorder or psychological functioning as known to vary either over time or between populations.[45] Thus, environmental factors have to be implicated in the major increase over the past half-century or so in levels of crime, substance use and misuse, and suicide in young people. Equally, they are involved in the rise in the average level of intelligence.[46] That does not mean that there are not continuing major genetic influences on individual differences in all these traits but it does mean that there have to be non-genetic factors that are responsible for the changes in level. Changes in the gene pool take place too slowly to account for such major time trends. Equally, it is obviously implausible that genetic factors are responsible for the fact that homicide is at least a dozen times as common in the United States as it is in Europe. In all probability, there are genetic factors involved in propensities to engage in such violent behavior but the national differences are not likely to be attributable to genetic factors; rather, the evidence indicates that they are a function of access to firearms.[47] Behavioral genetics has rightly been criticized for ignoring this evidence. Of course, that does not mean that genetic factors are not involved through interplay with the environment but it does mean that a straightforwardly deterministic view of genetic factors is unwarranted.

Overstatement of genetic claims

A related concern is that not only are the genetic claims overstated, but some geneticists resolutely ignore the evidence that runs counter to their evangelism. Thus, both Baumrind[41] and Jackson[41] drew attention to the limitations in some behavioral geneticists' considerations of the evidence, but also took exception to the claims that only extreme environments matter and that variations in rearing in families is of no real consequence.[48] As discussed in Chapter 5, these sweeping assertions on the irrelevance of the family environment are not supported by the research evidence. It is quite striking that behavioral genetics reviews usually totally ignore the findings

on environmental influences. It is almost as if research by non-geneticists is irrelevant. The underlying problem is that many behavioral geneticists have been reluctant to pay attention to evidence that does not derive directly from the use of genetic designs. The end product has been a rather one-sided approach to research findings.

There is no doubt that some of the proponents of behavioral genetics have been guilty of evangelistic overstatement and misleading claims, but that does not mean that they are wholly wrong in the arguments that they are putting forth. The purpose of this volume is to try to take a dispassionate view of the research evidence and, thereby, to come up with conclusions on the probable role of genes in influencing individual differences in behavior. Inevitably, that will mean a cool hard look at the evidence on what genes actually do, as well as an equally rigorous look at how genetic mechanisms might play a role in shaping individual differences in behavior.

How could there be genes for social behavior?

Critics of behavioral genetics have cast scorn on the apparent absurdity of the idea that there could be genetic influences on behaviors that are manifestly social, such as crime, divorce, and homosexuality. However, this attack rather misses the point. Of course, it is the case that there is not, and could not be, a gene for any of these behaviors, but individuals do vary in their propensity to show those behaviors and, insofar as that is the case, there is every reason to suppose that genetic factors will be implicated (see Chapters 4 and 8). It makes no sense to try to subdivide behaviors into those that are social and those that are not. To a degree, all behavior is influenced by social context and social forces, but that does not mean that it is not also influenced by genetic factors. It would be truly absurd to suppose that, although there are genetic influences on everything else, susceptibility to the environment is unique in not being influenced by genetic factors. Evolutionary concepts make clear that genes are very much involved in adaptation to different environments, and the empirical evidence (see Chapter 9) provides demonstrations of such gene–environment **correlation**s and interactions.

The supposed inappropriateness of neurogenetic determinism

Finally, and perhaps most importantly, there have been critiques of what seems to be neurogenetic determinism.[49] Some of these arguments are better based than others. For example, Rose[50] argued that the behavioral genetics

claims imply a directness of genetic effects (as exemplified by references to genes "for" **schizophrenia** or "for" **autism** or "for" **bipolar disorder**) that are out of keeping with the evidence that genetic pathways are much more indirect than that. The DNA influences the **RNA**, which influences the production of polypeptides and thereby proteins, which influence the metabolic pathways that cause disorder (see Chapter 7), but it is much more complex than implied by statements that genes lead to any kind of disorder. Such statements also ignore the influence of gene–environment correlations and gene–environment interactions and, especially, ignore the effects of environmental influences on gene expression (see Chapters 9 and 10). In all of these respects, the arguments of the critics of behavioral genetics are on target. Genetic influences are indeed all-pervasive, and extremely important, but they are frequently indirect.

But that is exactly what some leading psychiatric geneticists have themselves been arguing. Kendler[51] firmly states that "the strong, clear and direct causal relationship implied by the concept of 'a gene for' does not exist for psychiatric disorders. Although we may wish it to be true, we do not have and are not likely ever to discover 'genes for' psychiatric illnesses." That accepted (as clearly it must be), it is nevertheless important to be clear what neurogenetic determinism does, and does not, mean. A reductionist approach in science implies that, ultimately, everything is derivable from first principles, that everything at one level is explicable in terms of some lower level, and that what appears to be complex will prove to be accountable for in terms of a limited set of concepts and simpler, more basic, constituents.[52] Rose[50] has objected on the grounds that it transfers the burden of explanation from the social to the individual and, within the individual, from the biological system to the molecular. However, that is to take an unduly narrow view of biology. Dennett[53] has argued convincingly that evolution has meant that humans are thinking, feeling beings with the capacity to imagine what might be, to conceptualize the consequences of different actions, and hence the capacity to evaluate the ends and not just the means. In other words, through our thought processes (and their effects on our behavior) we can influence what happens to us. Determinism definitely does not imply inevitability (because avoidance and prevention may be possible); indeterminism would actually provide less room for maneuver (because it is determinism that allows us to decide how to change things), and real options (and not just apparent ones) exist in a deterministic world. Determinism means that there is a logical structure to how genes operate but it does not mean that genes provide direct causal links with any behavior. Manifestly, they do not.

Rose's[50] other objection is that neurogenetic determinism seems to place all mechanisms within the organism, thereby ignoring both environmental influences and social contextual effects. As discussed throughout the book,

that kind of reduction is out of keeping with the evidence.[54] The good side of reductionism lies in the attempt to derive simplifying principles and to identify both organizational constructs and causal pathways. However, the bad side of some forms of reductionism is to seek to do this entirely at the molecular level, ignoring the different levels that have to be considered in terms of what is known about the biology.

Lewontin,[55] whilst noting that holistic explanations cannot provide the answer (because everything is not connected to everything else), emphasizes three main features. First, developmentally there are random effects and not just the specific effects of genes and environment. Second, evolution involves construction, and not just adaptation; in other words, to an important extent organisms shape their environments, just as their environments shape organismic development. Third, there are important feedback loops; a perturbation in one point of a connected system may be the cause of a change in another part, which then leaves a cause for a change in the first part.

Morange[56] put the same point in a slightly different way. He noted that biology is almost always based on a strictly regimented, structured, and dynamic order. Once the processes are properly understood, it is clear that they follow a regular pattern. To that extent, a deterministic view is correct. Genes *do* provide the basis for the process of development and for the functioning of the mature organism.

On the other hand, because the effects of genes are indirect, it is not possible to reduce everything to the molecular level. Organisms are made up of a hierarchy of organizational levels. There is a precise causal chain linking the product of a gene to the actions of that gene within the organism, but this causal chain passes through several different levels of organization. At each level, the chain is transformed and obeys different rules. The complexity starts with the fact that any given gene can have several rather different effects (see Chapter 7). Thus, a given DNA fragment may be involved in making several different messenger RNAs and thus several different proteins, each of which can have different functions. Also, it is misleading to think of a gene as a single thing. The process leading to the production of proteins involves various other genes that do not themselves have a direct effect on proteins but which, nevertheless, exert important effects through their action on genes that do. The route from the protein products to a particular functional feature, such as a behavior, involves yet more indirect links. The finding that a gene is implicated in some way in the pathway leading to a particular behavior does not mean that it causes such a behavior. The protein products of genes do not act in isolation but participate in the formation of complex networks and structures which are then integrated into an overall hierarchical organization. Moreover, with multifactorial traits (and these account for the great majority of behaviors of interest) there is an interplay

with the environment that may involve gene–environment correlations, genetic influences on sensitivity to the environment (see Chapter 9), and the effects of the environment on gene expression (see Chapter 10).

The situation may be summarized by saying that basic science genetic studies have been hugely helpful in identifying some of the key organizing principles of how effects come about, but equally they have emphasized that the causal pathways are often probabilistic and indirect.[56]

Also, the genetic effects operate on causal pathways that will often not be specific to particular diagnostic endpoints. We need to be concerned with what these pathways might be (see Chapter 7) but it would be absurd to restrict attention to particular psychiatric diagnoses. There is every reason to suppose that genetic effects apply across all varieties of individual differences in human functioning and there is absolutely no reason to suppose that genetic effects will operate directly on psychiatric diagnoses.

In addition, many concerns have been expressed over the extravagant claims of protagonists of genetics such as Sandra Scarr[57] or David Rowe[58] or Steven Pinker[44] or Judith Rich Harris.[59]

Again, some psychiatric geneticists have expressed much the same views. Kendler[54] has argued that there is a need for a coherent conceptual and philosophical framework for psychiatry that rejects mind–body dualism; that psychiatry is irrevocably grounded in mental, first-person experiences; that a multilevel systems approach is essential; and that it is necessary to embrace complexity and to support empirically rigorous and pluralistic explanatory models. As he explains, using examples, this is *not* to argue for a compromise "bits of everything" concept, but rather it is an acceptance that, as a U.S. National Research Council entitled their report,[60] the science must extend *"From Neurons to Neighborhoods."*

Conclusions

In summary, behavioral genetics has proved controversial because of much of the "hype" associated with it. It has to be said that this is as much a fault of its protagonists as it is of the media account of genetic findings. In this book, I seek to consider the extent to which, beneath the hype, there is real substance in genetic influences on behavior and that there are important implications of genetic findings for our understanding of causal mechanisms with respect to individual differences in both normal behavior and the occurrence of mental disorders. Before turning to the empirical findings on genetics, however, it is necessary to discuss concepts of risk and protective factors in relation to the variations in behavior for which genetic influences might be relevant (Chapter 2).

Notes

See Reference list for full details.

1 See Valentine, 1986; also McKusick, 2002
2 Watson & Crick, 1953
3 See Lewin, 2004 and Strachan & Read, 2004 for clearly expressed authoritative descriptions of the technical details, and Weatherall, 1995 for a very readable account of some of the scientific highlights and their meaning for medicine.
4 International Human Genome Sequencing Consortium, 2001, 2004; Venter et al., 2001; see Sulston & Ferry, 2002 for a more personal account of what was involved in this pioneering international collaboration.
5 Plomin et al., 2001
6 Rutter et al., 1990 & 1999
7 Le Fanu, 1999
8 See Kimmelman, 2005; Marshall, 1995 a & b; Relph et al., 2004
9 Bryson, 2003
10 See Rutter, 2000 a, for a very brief note on this need in relation to autism and Rutter & Plomin, 1997 and McGuffin & Rutter, 2002 for a broader discussion of the role of genetics in providing an understanding of the neural underpinning of mental disorders.
11 Plomin & Crabbe, 2000
12 See Joseph, 2003; Kamin, 1974; Kamin & Goldberger, 2002
13 Rutter et al., 2001 a
14 Stoolmiller, 1999
15 Devlin et al., 1997
16 Taylor, 2004
17 e.g., Joseph, 2003; Kamin, 1974
18 Kendler, 2005 a
19 See Mackintosh, 1995 for a very clear and fair account of the issues.
20 See Miele, 2002 re Jensen's views
21 See Rutter & Tienda, 2005 with respect to Jensen's dealing with the Minnesota Transracial Adoption Study.
22 See Storms & Sigal, 1958 and Pelosi & Appleby, 1992
23 Jensen, 1998
24 Jensen, 1969
25 See Tizard, 1975
26 See Miele, 2003
27 Antonuccio et al., 2003; Bekelman et al., 2003; Blumenthal, 2003
28 Eysenck, 1965, 1971 & 1980
29 Glantz et al., 1995; Hilts, 1996; Ong & Glantz 2000
30 Royal College of Psychiatrists, 2001
31 Devlin et al., 1997; Black, 2003
32 Müller-Hill, 1993
33 See Rutter, 1999 a; Nuffield Council on Bioethics, 2002
34 Kamin, 1974

35 Herrnstein & Murray, 1994; Jensen, 1998
36 Firkowska-Mankiewicz, 2002
37 Marmot & Wilkinson, 1999
38 Rutter, 1999 b
39 Starfield, 1998
40 Tawney, 1952, p. 49
41 Baumrind, 1993; Jackson, 1993
42 Rutter & McGuffin, 2004
43 Sargant & Slater, 1954
44 Pinker, 2002
45 Rutter & Smith, 1995; Rutter & Tienda, 2005
46 Flynn, 1987; Dickens & Flynn, 2001
47 Rutter & Smith, 1995
48 See the extravagant claims of the protagonists of genetics such as Sandra Scarr (1992) or David Rowe (1994) or Steven Pinker (2002) or Judith Rich Harris (1998)
49 Rose, 1995, 1998; Rose et al., 1984
50 Rose, 1998 – see pp. 272–301
51 Kendler, 2005 c
52 Bock & Goode, 1998
53 Dennett, 2003
54 See Kendler, 2005 b
55 Lewontin, 2000
56 Morange 2001
57 Scarr, 1992
58 Rowe, 1994
59 Harris, 1998
60 Shonkoff & Phillips, 2000

Further reading

Morange, M. (2001). *The misunderstood gene*. Cambridge, MA & London: Harvard University Press.

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

Rutter, M. (2002 b). Nature, nurture, and development: From evangelism through science toward policy and practice. *Child Development*, *73*, 1–21.