

Genes and Behavior

Nature–Nurture Interplay Explained

Michael Rutter



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Preface

For far too long, behavioral genetics and socialization theory have been viewed as necessarily in opposition to one another. Researchers in both “camps” have very rarely referred to studies from the other “camp,” other than to attack their concepts and findings. The result has been much fruitless dispute and serious misunderstandings of what each body of research has to contribute. My main aim in writing this book has been to provide a readable, non-technical account of what is involved in the possible various ways in which genetic influences on behavior may be important. That involves conveying something of how genes operate but, in making this clear, it will be very evident that genes do not operate in isolation. Hence, there will have to be a discussion of what is known about environmental influences and how the interplay between nature and nurture “works” over the course of development. “Nurture” is often used to refer only to patterns of upbringing in the family, but as used in the title of this book it is intended to cover a broader range of environmental experiences.

I am not trained as a geneticist but I have been a user of behavioral genetics for some 30 years¹ and I have made myself informed about genetic mechanisms and genetic issues. However, I have also been a developmental and psychosocial researcher over this same period of time.² The bringing together of these backgrounds has meant that I have ended up being both a strong supporter of behavioral genetics³ and, to some extent, a critic of the excesses of behavioral genetics⁴. Accordingly, I am hopefully well positioned to be a “translator” of specialist genetic concepts and findings. In addition, I have been much involved in ethical considerations with respect to genetic research ethics.⁵ This, too, helps to provide a good background for bringing about the integration that I want to achieve.

The first chapter outlines the major accomplishments of genetics and then goes on to consider why the topic of genes and behavior has proved to be so surprisingly controversial. In part, this arose through misunderstandings and misleading claims, but also it has to be accepted that genetics has been

misused in the past and could be misused again in the future if it is not dealt with properly. Critics of genetics have attacked what they viewed as a damaging biological reductionism. Accordingly, the meaning of this concept is discussed, noting both the positive and negative aspects of reductionist approaches.

As is apparent throughout the book, genetic influences operate in a probabilistic, not deterministic, fashion. In order to indicate what this means, the second chapter discusses the concepts of risk and protective factors as they apply to both genetic and environmental influences. The evidence shows that it is usually necessary to think in dimensional as well as categorical terms with respect to both influences that contribute to causation and to behavior. Traditionally, people have tended to make a sharp distinction between abnormal disorders or conditions and normal variations as they occur in the general population. Modern research has shown that with respect to somatic conditions such as coronary artery disease, or mental disorders such as depression, there is much continuity between the two. That is so even with severe disorders, such as schizophrenia and autism.

Against that backdrop, Chapter 3 provides an account of how twin, family, and adoptee studies can be used to determine the relative strength of genetic and environmental influences on individual differences in behavior (both normal and abnormal) within particular populations. Chapter 4 then summarizes what has been found in relation to a selection of important mental disorders and normal traits or characteristics. Chapter 5 does the same with respect to environmental influences.

Chapter 6 serves a bridging role between the first group of chapters, concerned with quantifying the strength of genetic and environmental influences, and the second group of chapters, concerned with specific identified genes or with how genes work. Thus, Chapter 6 outlines what is known about different patterns of inheritance. Chapter 7 then provides an account of what genes actually do. Although written in a simplified way using non-technical language, Chapters 6 and 7 are likely to involve concepts and approaches that are least familiar to non-geneticists. Readers who do not wish to know the details may decide to omit these chapters. However, it is hoped that they will not make that choice, because an understanding of how genes work is crucial for an appreciation of the importance of genetics in relation to behavior – as well as to an understanding of the limits of genetics. Chapter 8 describes the methods used to find individual genes that may have effects on the behavior and summarizes what has been found with respect to the genes involved in susceptibility to develop certain key mental disorders.

Chapter 9 brings together genes and environment through a discussion of the role of gene–environment correlations and interactions.⁶ In many respects, this constitutes the central issue for the book. That is because it shows that genes and environment cannot be viewed as totally separate and independent.

To an important extent, genes operate *through* the environment. That is, their effects come about first because the likelihood that a person encounters risky environments is influenced by that person's genetically influenced behavior in shaping and selecting their environments. The second way in which the environment is implicated is that genes influence a person's susceptibility to risk environments. People vary in their environmental vulnerability and genes are implicated in their individual differences.

Chapter 10, in effect, turns the tables by focusing on what environments do to genes. In the past, it has usually been assumed that genes do not change as a result of experiences. That is true insofar as gene sequences⁷ are involved, but it is not true in the case of the expression of genes⁸ in body tissues. That is to say, although the DNA is what a person inherits, the effects of that DNA are dependent on a chain reaction that ends up with the expression of the products of the DNA in the structure and function of individual cells (as described in Chapter 7). That is how environments can and do alter genes. The study of gene expression takes us into the realms of quite complicated basic science, but Chapter 10 concentrates on the fruits of that research (which is crucially important and readily understandable), rather than the biochemistry involved in the experiments.

Finally, Chapter 11 seeks to draw the threads together and to look ahead in relation to the likely implications for policy and practice.

Notes

See Reference list for full details.

- 1 Folstein & Rutter, 1977 a & b; Rutter, 1994, 2004
- 2 Rutter, 1972, 2002 a
- 3 Rutter, 1994; Rutter, 2004; Rutter & McGuffin, 2004
- 4 Rutter, 2002 b; Rutter & McGuffin, 2004; Rutter et al., 2001 a
- 5 Royal College of Psychiatrists, 2001; Rutter, 1999 a
- 6 Gene–environment correlations refer to genetic influences on the likelihood that individuals will encounter particular risk or protective environments. The genetic influence operates through effects on those behaviors that serve to shape people's choice of situations or experiences. Gene–environment interactions refer to genetic influences on people's sensitivity or vulnerability to particular risk or protective environments.
- 7 Gene sequences provide the genetic information and they comprise the particular order of the chemical bases (adenine, guanine, cytosine and thymine – A, G, C, and T for short) in the DNA (deoxyribonucleic acid) and then into translated protein (see Chapter 7).
- 8 Gene expression concerns the process by which the DNA exerts its effects through transcription into messenger RNA (ribonucleic acid) and then into translated protein (see Chapter 7).

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Michael Rutter

Contents

Preface	vi
Acknowledgments	ix
1 Why is the topic of genes and behavior controversial?	1
2 Causes and risks	18
3 How much is nature and how much nurture?	40
4 The heritability of different mental disorders and traits	64
5 Environmentally mediated risks	93
6 Patterns of inheritance	117
7 What genes do	144
8 Finding and understanding specific susceptibility genes	156
9 Gene–environment interplay	178
10 What environments do to genes	211
11 Conclusions	221
Complete reference list	226
Glossary	264
Index	271

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 **alleles** (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 1959¹.) 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 sequences** 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 factors** 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 **traits** 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 Bryson⁹ 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 Crabbe¹¹ 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 design¹³ 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 issues¹⁵ 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 genetics¹⁷ 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 findings²⁵ 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 evidence²⁶ 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 Eysenck²⁸ 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 findings²⁹ 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-Hill³² 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 book³⁴ 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