CHAPTER 3

Behavioral Genetics

CHAPTER OUTLINE

LEARNING OBJECTIVES

Studying this chapter should provide you with the knowledge to:

- Realize that knowledge of the genetic underpinnings of behavior is essential to understanding how behavior evolves.
- Comprehend the difference between proximate and ultimate causes in behavioral genetics and be able to apply these concepts.
- Understand how behavioral genetics helps to explain the physiological and neurobiological systems that control behavior.
- Know that most behavior is shaped by a combination of genetic and environmental factors.
- Be aware that single-gene effects on behavior should not be confused with an erroneous idea that complex behavior is "controlled" by those genes.
- Realize quantitative genetics provides better explanations than single-gene models do for most animal behavior traits.
- Understand that molecular approaches to behavior genetics provide important techniques for exploring the regulation of behavior.
- Appreciate the impressive array of behavioral genetic tools available for testing hypotheses about the evolutionary and genetic foundations of behavior.

Animal Behavior.

3.1 INTRODUCTION: PRINCIPLES OF BEHAVIORAL GENETICS AND THE EVOLUTION OF BEHAVIOR

Before Mendel saw his first pea plant, people knew that traits could be inherited. They also knew that the environment could affect inherited traits. Thinkers at least as far back as Shakespeare understood that behavior is usually the outcome of intrinsic influences (***nature***) and environmental ones (***nurture***). Based on this understanding, in *The Tempest* (IV.i.188–189), Prospero bemoans his attempts to reform the intrinsically savage Caliban: "A devil, a born devil, on whose nature/Nurture can never stick…." (In other words, Prospero cannot modify Caliban's nature.) Unlike Shakespeare, scientists have the tools to study the relative contributions of genetics and environment to behavior. What follows in this chapter will not help reform the Calibans of this world (it is to be hoped that there are few!), but it will introduce the genetic basis of behavior, which, in turn, is the key to understanding how behavior evolves.

KEY TERM In the nature–nurture debate, nature is genetics. Nature is the extent to which genetics influence behavior.

KEY TERM In the nature–nurture debate, nurture is environment. Nurture is the extent to which environment influences behavior.

The goal of this chapter is to provide a toolbox of genetic approaches to behavior.[1,2] Genetic studies of behavior take a much stronger problem-solving approach than other areas of behavioral science. An important part of solving a problem is choosing the right tool, and progress through this chapter can be measured by the facility with which the tools are understood and used. Knowledge of genetics gives students of behavior windows into both the evolution of behavior and the physiological regulation of behavior. Assimilating this material gives students powerful problem-solving abilities that can be applied in the study of almost any type of behavior.

CASE STUDY

The "Killer" Bee

The "killer" bee story is a classic example of plans gone awry. In the early 1950s, Brazilian geneticist Warwick Kerr came up with a well-intentioned scheme to introduce honeybees into the tropical Americas. Honeybees provide an outstanding subsistence source of food and wax for farmers, and these products can also be cash crops that supplement income from products such as bananas and papayas. Kerr planned to keep tropically adapted honeybees, imported from southern and eastern Africa, isolated and to use controlled crosses to develop hybrid bees that would be manageable and productive in the Brazilian tropics. Unfortunately, an unintended release of the African bees in 1957 resulted in the spread of very dangerous, unmanageable insects across the Western hemisphere.

One of the most extreme examples of ecotypic differentiation is the Western honeybee, *Apis mellifera*. Because honeybees provide many of the classic examples in behavioral genetics, we'll give some background here on their ***ecotypes***. The geographic distribution of this species, from southern Africa to northern Europe, through the Middle East and into central Asia, encompasses both tropical and temperate environments, as well as arid and mesic (moist) environments. More than two dozen subspecies of *Apis mellifera*—ecotypes differentiated by color, body size, and behavior—have been recognized. This species has been introduced in the Americas and is the common honeybee seen in many North American habitats. One subspecies is also, famously, the "killer" bee, the highly defensive bees now found in much of Central and South America.

KEY TERM An ecotype is a genetically differentiated population within a species that is adapted for a particular habitat.

Honeybees were already present in the Americas, but these bees originated in Europe and are not well adapted to tropical environments. Honeybees in most of North America are the descendants of bees brought by settlers from Europe beginning in the mid-1600s. They are mostly derived from *Apis mellifera*

(*Continued*)

CASE STUDY (CONTINUED)

ligustica, the ecotype that is characteristic of the Italian peninsula, but they also have genetic influences from a variety of other European ecotypes. The "Africanized" or "killer" bee is *Apis mellifera scutellata,* but it has hybridized with other ecotypes as it has spread through South and Central America and, more recently, into Texas, Arizona, and California.

The differences among honeybee ecotypes have a genetic basis; this was well established by beekeepers long before scientists approached the question, but ***common garden*** and controlled crosses of ecotypes by scientists support this conclusion.

KEY TERM Common gardening is a technique in which animals (or plants) are maintained in the same environment. If the animals differ in behavior across their native habitats, then keeping them in a common garden helps to separate environmental from genetic influences. If they all behave the same when in the common garden, then the behavioral differences observed across their native habitats likely are due to environment. If the differences persist in a common garden, then a genetic hypothesis is supported.

FIGURE 3.1
Distribution map of the major ecotypes of the Western honeybee. *Source*: *http://www.sicamm.org/images/Map.jpg.*

The most striking difference between "Africanized" bees and the honeybees to which most North Americans are accustomed is the heightened defensive response of the "Africanized" bees. With bees of European origins, under most conditions you can walk to within a few meters of a hive without risk of being stung, and if approaching the hive from behind, you can probably walk up to it and sit on it without risk. In contrast, "Africanized" bees often respond to an animal's movement 50 to 100 meters from their hive by flying and stinging. Each "Africanized" bee sting is no more potent that a European bee sting, but mass stinging can be lethal to large animals such as humans, dogs, horses, and cattle. A conservative estimate is that several hundred people have died in South and Central America from mass stinging events since the release of the bees in 1957. "Africanized" bees exhibit extreme responses in all phases of nest defense when compared with their European counterparts; the differences between the ecotypes are largely due to genetics (see Figure 3.1 and Table 3.1).[3]

These observations raise the question of why the root African stock from which the "Africanized" bees are derived exhibits such extreme responses. The best hypothesis is that honeybees in Africa have been faced by a combination of predators that includes humans and their evolutionary predecessors.

TABLE 3.1 Comparison of the behavioral attributes of the African ecotype, *Apis mellifera scutellata*, which probably gave rise to "African" bees in the Americas, with those of the Western European ecotype *Apis mellifera mellifera*

	Apis mellifera scutellata	Apis mellifera mellifera
Geographic origin	South and Eastern Africa	Northwestern Europe
Reproduction	Many swarms, absconds from nests to migrate when climate turns unfavorable	One or possibly two swarms a year, rarely absconds
Honey storage	Low	Very high
Defensiveness	Very high	Moderate
Size and color	Small, black	Medium, black

(*Continued*)

CASE STUDY (CONTINUED)

This amounts to strong selection favoring extreme defense. The *scutellata* ecotype has other adaptations for its tropical environment, such as propensity to invest in reproduction rather than storing large quantities of honey, which makes it less attractive to beekeepers; its one strongly positive attribute from a human point of view is its ability to do well in tropical climates, where honeybees of European origin usually fail.

A key lesson from this story is that containment and isolation of potentially dangerous or ecologically damaging introduced animals need to be extremely secure. Knowledge of the behavior of species in their native habitats does not always predict how well they will do in a new habitat, and species as diverse as the "killer" bee, cane toads, Burmese pythons, European starlings, and English sparrows have demonstrated unexpectedly high behavioral abilities to reproduce and disperse in new (to them) habitats.

Why Study the Genetics of Behavior?

Behavior, like all characteristics of animals, is shaped by a combination of genes and environment. This chapter presents techniques used by animal behaviorists to discover how genes and environment come together to determine behavior. This is a surprisingly controversial enterprise, and because of that, the nature–nurture debate and the political overtones possible with such discussions are included.

How Does Behavioral Genetics Inform Studies of Evolution?

Knowing how and why behavior has evolved is a central goal in the study of animal behavior, one that is greatly informed by an understanding of the genetic influences on behavior. Studying genes that seem to influence behavior gives insight into the physiological and neurobiological underpinnings of behavior. In addition, separating genetic from environmental effects allows both identification of the selective forces that shape behavior and determination of how behavior responds to those forces over evolutionary time. In effect, behavioral genetics allows observation of evolution in action.

Natural selection acts on genetic variation; if there is no genetic variation, then traits cannot change over time and evolution cannot occur. It follows from this that behavioral geneticists often focus on understanding how genetic variation affects a behavioral trait. Inevitably, studying the effects of genetic variation leads to considering the effects of environmental variation on the behavior, as well. The behaviors an animal expresses are partly a reflection of the animal's environment and partly a reflection of the animal's genes. Neither environment nor genes *determine* an animal's behavior, but both environment and genes contribute to the behavioral phenotype. A major goal of behavioral genetics is to understand the extent of these environmental and genetic correlations with the behavioral phenotype. Not surprisingly, some behavioral phenotypes, such as signals involved in courtship,[4] often show little variance among environments, whereas others, such as foraging behavior, are typically quite responsive to environmental differences.

Understanding Single-Gene Effects on Behavior

What is the path from genes and their products to the behavior of an animal? Causation is one of the key issues in animal behavior; new techniques allow linking genes, neural mechanisms, and behavior in exciting ways. Genes affect behavior at many levels. While it is tempting to think of genes as guiding behavior ("my genes made me do it"), examples of direct genetic instructions for behavior are rare. Instead, genes most often affect behavior by setting limits, or restraints, on the range of behavior expressed by animals. Thus, behavioral options are most frequently limited by genes, not directed by them.

OF SPECIAL INTEREST: PHENOTYPE AND GENOTYPE

Quick refresher: *Phenotype* is the outward manifestation of a trait, that is, the actual behavior, morphology, or physiology. *Genotype* is the characterization of the genes associated with the phenotype. Phenotype does not always reflect genotype in the same way because of environmental influences on the phenotype. The interplay between genes and environment is often measured using *heritability*, a key concept in genetics. Heritability is the extent to which variation in phenotype in a population of animals is correlated with genetic variation. Calculating heritability allows scientists to investigate the genotypic and environmental roots of phenotypic variability.

Genetically based human diseases rank high in human awareness. One such disease, Huntington's, results from the modification of a repeated nucleotide sequence in a single gene, *huntingtin*. The function of the protein for which *huntingtin* codes is not fully understood, but the modification of the gene has devastating degenerative neuromuscular effects for its human carriers. Many of the symptoms of Huntington's, such as unstable gait, are behavioral, and because of this, it is tempting to suggest that locomotory stability is under the control of a single gene. In fact, modification of the function of a single gene can indeed dramatically affect behavior, but this does not mean that the particular behavior is under control of that gene. Rather, the failure of the gene means that a needed step in the machinery underlying the behavior is absent. That absence causes multiple systems to fail. If any one of many genes other than huntingtin ceases to function, the results might be equally catastrophic.[5]

Fruit flies, *Drosophila melanogaster*, offer another example of one gene that ultimately affects behavior. Fruit flies can be rendered less receptive to mating by modifying the gene (the *Icebox*, or *ibx*, mutation) that is involved in normal formation of brain structures. Unable to respond to a potential mate, the modified flies cannot mate. Does that mean that the *ibx* gene "controls" mating? Not at all. It merely controls one small but crucial step in the mating sequence.[6]

In fact, virtually every behavior is shaped by genes acting in sequence or in a coordinated fashion to produce that behavior. Most investigators in behavioral and neural genetics now feel that genetic control of any one behavioral trait is dispersed over a large number of neural locations, rather than being coordinated by "executive neurons" that integrate the behavior. If many neural elements are involved in the production of a behavior, it logically follows that many genes must underlie the production and regulation of those neural elements. Because behavior is typically shaped by a large number of genes acting in concert, one of the main goals of contemporary behavioral genetics is to understand how multiple interacting genes can shape specific behavioral patterns.

3.2 THE NATURE VERSUS NURTURE DEBATE

What is the balance between genetics (instinct or nature) and learning (nurture) in shaping behavior?[7] Few topics have wasted more emotional energy and created more futile academic fury than the question of behavioral plasticity. An extreme view holds that all animal (including human) behavior is instinctive, with little room for learning and flexibility of response. At the other extreme, some scientists argue that behavior is plastic, modifiable over a great range of possibilities, and that individual experience determines behavior. As is typically the case in such debates, the truth lies between the extremes.

In the animal world, behavior can be envisioned on a continuum between learning and instinct. Depending on the behavior and in some cases, the individual, the explanation may (rarely) involve one of the ends of the continuum, or (more likely) any one of an infinite

array of intermediate locations along that continuum. The appropriate scientific goal is that of understanding the balance between genetic constraints and phenotypic flexibility in shaping the behavior of animals. It is this understanding, and not a squabble of extremes, that gives great insight into how evolutionary forces shape behavior.

What follows is a discussion of the history of ethology, animal behavior, and sociobiology as it specifically relates to the nature–nurture debate. Refer to Chapter 1 for a broader view of the history of the discipline.

The "nature" school of thought came to the forefront in the early to mid-twentieth century among European ethologists, such as Konrad Lorenz. Their studies emphasized the roles of instinct, fixed patterns of behavior, and the influence of evolution on behavior. Looking back at the work of the early ethologists, we can see the most striking feature of their work was its essentially descriptive focus. The leading ethologists—Karl von Frisch, Niko Tinbergen, and Lorenz—were interested in observing behavior in a field, or naturalistic, setting and then in using experimental approaches to explore the neurophysiological basis for the behavior. Lorenz enjoyed self-promotion and capturing the public eye; he used his work on animal behavior to make larger arguments about human behavior in books such as *On Aggression*, *King Solomon's Ring*, *Beyond the Mirror*, and *Civilized Man's Eight Deadly Sins*. Lorenz's penchant for publicity helped to bring attention to his involvement with the Nazi party in prewar Germany, as well as some of his writings in which he appeared to use his scientific findings to support Nazi social theories. For many, this darker side of Lorenz's past cast a shadow on the scientific thought of the ethologists.

Ironically, Niko Tinbergen, the other leading ethologist of the era (von Frisch was a generation older than Lorenz and Tinbergen), was Dutch and lived in the Netherlands through the German occupation of that country. Tinbergen was held at a prison camp (Beekvliet) by the occupying forces for 2 years (1942–1944). Thus, in important ways Tinbergen and Lorenz seem strange bedfellows, but both before and after the war, they were academic collaborators and personal friends.

In the United States, the study of animal behavior had a much stronger laboratory component than it did in Europe. As a result, psychologists contributed a great deal to its history. American psychologists championed the "nurture" school of thought. Because they worked in a laboratory setting, they could study the role of learning in behavior, something that is difficult to address under field conditions. They started with simple models of learning, such as conditioning, and argued that most behavior is learned, modifiable due to experience, and at least in humans, not constrained by evolutionary history.

A critical evaluation of ethology by the American psychologist Daniel Lehrman[8] provided a touchstone for American comparative psychologists who preferred to isolate animals from their natural environments and study behavioral plasticity (learning) in a laboratory context, where all stimuli could be controlled. (Lehrman and his laboratory discovered much of the behavior–environment–endocrine interaction in ring doves introduced in Chapter 2.) Lehrman's harsh reaction to the ethologists and to the study of instinct also must have been shaped by his repugnance against the Nazis, although Lehrman's biographer, Rosenblatt, suggests that Lehrman de-emphasized Lorenz's Nazi sympathies in his 1953 critique of ethology. Lehrman died relatively young (in 1972, aged 53).[9] It is interesting to contemplate the role that he might have had in the synthesis of ethology, neurobiology, and genetics had he lived long enough to participate in these scientific revolutions (see Chapter 1 for more on this aspect of the history of the field).

Application of the nature–nurture question to human behavior nearly always generates trouble. Data interpreted to show genetic bases for differences among humans in intelligence, motor learning capabilities, criminality, and a broad range of other behaviors have, unfortunately, been used to support racism and other forms of bigotry. Advocates for human

social reform are repelled by the thought that human social behavior might be predetermined and might not be subject to modification by social forces. Scientific discoveries should not be ignored if they are disturbing or if they fall outside cultural norms, but neither should they be overinterpreted. It is critical to ask about the costs and benefits of basing policy decisions on an understanding that may be a work in progress. If potential costs—social as well as financial—exceed benefits, then some caution is warranted. Remember that scientific knowledge is a progression based on improvements of methods and collection of more data, and conclusions are less than permanent; as new methods become available, scientific understanding changes. There is danger that much harm can be done by rigidly applying a scientific dogma to human social behavior because that dogma may prove to be incorrect. In short, scientists often do well to avoid stating conclusions in a strongly affirmative manner, especially when those statements may have harmful sociological or political effects on groups of people, unless the benefits of such statements undeniably exceed the cost of being wrong. William Shockley, a Nobel prize–winning physicist who made arguments about racial differences in intelligence and social capabilities, stands as an extreme example of application of "scientific" findings to support negative social agendas.[10]

DISCUSSION POINT: SCIENTISTS' RESPONSIBILITIES TO SOCIETY

Well-meaning scientists asking questions about intelligence and differences among animals and making hypotheses about the evolution of social behavior found their work at the center of a sociocultural and political debate. How responsible are scientists for the uses to which society puts their discoveries? Were strongly worded arguments based on incomplete or inconclusive data?

Sociobiology provided a major arena for the nature–nurture debate in the 1970s and 1980s. This discipline, championed by E. O. Wilson, integrates thought from ethology, ecology, evolution, and genetics in an attempt to develop a deeper understanding of the evolution of behavior.[11] While this approach attracts many behavioral biologists, its detractors, such as R. C. Lewontin,[12] suspect that sociobiology (genetic determinism) ultimately supports racist or class-based justifications for inequities in human societies. This vituperative conflict ranges far outside the boundaries of science and, again, becomes an argument that uses science in the pursuit of policy and, in some cases, power.

A more recent example of the nature–nurture debate is the reaction to the argument that rape is an adaptive reproductive strategy in humans.[13] This assertion not only attributes rape to genetic influences, thus raising questions about individual responsibility, but also challenges the notion that sex criminals can be rehabilitated. Some people think it raises the possibility that potential sex criminals could be genetically identified and segregated from society. These assertions are repugnant to those who think that human behavior is shaped by experience and that all humans are capable of improvement and rehabilitation. No matter what one's stance, this is an understandably volatile question.

It seems that scientists and popularizers who have attempted to construct an interpretation of human behavior around biological principles typically overreach because they do not recognize the limitations of the scientific base from which they draw their conclusions. The vicious responses of critics take the debate wholly out of the realm of science (and of acceptably polite discourse). The resulting standoff does not increase understanding of behavior, nor does it help institutions and individuals who struggle to cope with the real outcomes of aggression and other disturbing conditions. (Taking extreme stands is, however, a behavioral pattern that is frequently seen in human conflict.)

What is clear is that culture stands between humans and their biology in many interesting ways; some aspects of human culture reflect the evolutionary history and biological constraints of humans, whereas other aspects of culture are counterpoised to biology,

regulating or opposing biological forces so that human societies can function. Understanding the complex relationship of biology and culture is beyond current understanding of either. Intellectual surrender in the face of this complexity is wrong-headed—this interplay forms one of the great questions of the twenty-first century!—but caution is reasonable when making sweeping "scientific" statements about the biological bases of human social behavior.

3.3 EVOLUTION AND BEHAVIOR

While a fine-grained discussion of the genetics that underlie behavior may address Tinbergen's causation question, his questions about survival value and evolution are not far away. As discussed in Chapter 1, evolution is a change in frequencies of genes in populations. Such a change normally takes place over the course of generations; the environment, however, may change within the course of a single lifetime. In the face of short-term environmental change, behavior often provides the route for an individual animal to adapt to that change. Migration, for example, is usually a behavioral adaptation to seasonal fluctuations that occur during an animal's lifetime. Similarly, learning the location and types of available food resources allows animals to accommodate shifts in food organisms—again, during a single lifetime. Thus, within-generation phenotypic flexibility is very important to animal survival.

Equally important to the survival of lineages is response to longer-scale environmental change, such as a slowly shifting climate. This type of change invokes evolutionary responses by favoring the survivorship of some phenotypes over others, which in turn leads to shifts in gene frequencies that then produce physiological, morphological, and behavioral modifications. All of these modifications are phenotypic; that is, they are all outward manifestations of genetic traits.

If an animal's behavior allows flexibility, it can be a short-term tool with which an individual animal can respond to a variable environment. If it has a genetic basis, it can also be molded over generations by natural selection, in response to long-term environmental variation.

Evolution is often perceived as taking many generations to have a visible effect. This may be generally true, but there are many examples of rapid evolutionary change in response to strong selection. Strong artificial selection, such as that applied by animal breeders to domestic species (e.g., rabbits, chickens, dogs,[14,15] cats, and cattle), can have substantial effects over three to five generations. This suggests that populations of species in new environments (such as invasive species) or species that are experiencing rapidly changing environmental conditions could have the flexibility to exhibit rapid evolutionary responses if sufficient genetic variation is present.

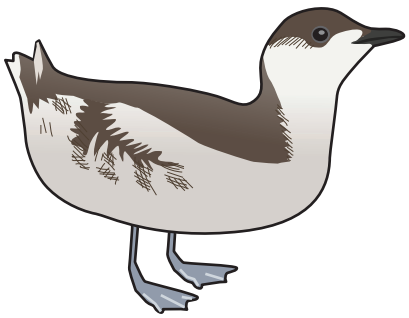

FIGURE 3.2
Marbled murrelet (*Brachyramphus marmoratus*).

It is also possible to use genetics to understand evolutionary history. The history, even the recent history, of sea birds is particularly difficult to infer because of their highly mobile lifestyle (see Figure 3.2). Genetic studies of these animals in the Pacific Northwest of North America have revealed that there are two genetically distinct populations: one on the Aleutian Islands and one from the eastern Alaskan Peninsula. Although they nest in different locations, the genetic distinctions can be traced back to a single ancestral population that expanded in the early Pleistocene period and then was separated by later Pleistocene glaciations.[16]

3.4 THE BEHAVIORAL GENETICS TOOLBOX

Behavioral geneticists have a toolbox, a set of largely experimental approaches that can be used to unravel how genes shape behavior (see Table 3.2). Some of the basic tools come from comparative evolutionary studies; for example, constructing a ***cladogram***, or phylogeny, allows an investigator to follow changes in behavioral patterns in evolutionary

TABLE 3.2 The Behavioral Genetics Toolbox

Tests of Ultimate Causes: The Evolution of Behavioral Phenotype	Proximate Causes: Whole Organism Studies of the Genetic Bases of Behavior	Proximate Causes: The Genetic Dissection of Mechanisms Underlying Behavior
1. Geographic variation, subspecies, and ecotypes	1. Mutational studies	1. Microarrays and other assessments of gene expression
2. Comparative phylogenetic studies	2. Heritability	2. Candidate gene approaches
3. Cross-fostering	3. Quantitative trait locus (QTL) analysis	3. RNA knockout
4. Twin analyses		
5. Artificial selection and inbred lines		
6. Natural selection and behavior		

time and to identify key behavioral innovations.[17] Other tools, such as calculations of heritability, are borrowed from quantitative genetics. Powerful methods for identifying genes that may regulate behavior come from molecular methods of genetic mapping and identification of ***quantitative trait loci*** (QTLs).[18] ***Expressed sequence tags*** (ESTs) provide a direct window into the relationship between gene activity and behavior. This section explores the behavioral geneticists' toolbox and shows how these tools are applied to the central issues of evolution and causation in animal behavior.

KEY TERM A cladogram represents a hypothesis of evolution within a group of species with a tree-like drawing.

KEY TERM A quantitative trait locus (QTL) is a gene that contributes, with other genes, to a phenotype. Because multiple genes contribute to the phenotype, no one gene "determines" the phenotype.

KEY TERM An expressed sequence tag (EST) is a genetic marker that is linked to the gene being studied. When the gene of interest is expressed, the EST "reports" that activation. This allows an investigator to see how gene activation correlates with physiological and behavioral activity.

As noted in Chapter 1, when biologists talk about *ultimate causes,* they are thinking about evolution and the long-term selective pressures that shape an organism's phenotype. Studies of ultimate causes often boil down to understanding where, in the course of evolution, a trait first appears, or where a structure first is used for a particular function. All mammals have digits—fingers and toes—but few have opposable thumbs and the high manipulative ability that comes with having a thumb-like digit. Knowing when opposable digits evolved, and how they were adaptive, helps biologists to understand the ultimate questions associated with these structures, that is, how they evolved. Because genes evolve and help to determine behavioral phenotypes, understanding ultimate causes plays a key role in behavioral genetics.

Often, studies of ultimate causes suggest approaches to finding *proximate causes*, in this case, the present-day physiological or behavioral expressions of gene activity. Biologists who focus on how genes regulate the functions of organisms study the proximate causes of traits. They discover the sequence of events that start with the transcription of DNA and end with the expression of a behavior. The most elegant behavioral genetic studies link the evolutionary roots of behavior to its proximate causes.

Geographic Variation, Subspecies, and Ecotypes

Humans figured out long ago that behavioral, as well as physical, traits run in families.[19] Behavioral genetics sheds the light of modern science on this observation. One starting point is a search for associations between genetic differences and behavioral differences among animals. Comparing families, subspecies, and species and looking at differences at those levels help to indicate the extent to which genes influence behavior.

Strains, sometimes called *ecotypes* or *subspecies*, can be produced naturally or by artificial selection. When subspecies are formed naturally, they can be compared to see if the subspecies differ behaviorally; if so, there is a possibility of genetic influence on those behaviors. In the case of artificial selection, animals are bred to produce strains with differing characteristics, including behavior; comparisons among these strains are also important sources of behavioral genetic information. Because an animal's behavioral phenotype is determined by a combination of environmental and genetic factors, scientists can estimate genetic influences by eliminating environmental variation in the animal's life; if they are successful in doing so, then any between-strain differences can be attributed to genetics. For instance, if different subspecies are kept in shared or identical environments, but behavioral differences nonetheless persist, then one must seriously consider the genetic basis for those differences.

This procedure, called a common garden technique, is frequently used for testing the hypothesis that there is an underlying genetic basis for phenotypic differences (see the Case Study and key term on page 69 for more on this technique). There are several ways to eliminate environmental variation. Moving the animal to a new environment and asking if a behavior persists is one way. If behavior changes with the change in environment, then a strong genetic influence on behavior is doubtful. However, conclusions are more difficult to come by if the behavior persists. Instead of using translocated parents, another method of eliminating environmental variation is to focus on offspring (F1 individuals) reared in the shared environment. This approach may be informative if the offspring are not subject to parental influence, but if parent–offspring interactions are important in shaping the behavior of the F1 generation, then a more sophisticated approach, such as cross-fostering or planned matings between strains (see below), may be required to test the hypothesis that behavioral differences among ecotypes have a genetic basis.

The gray wolf (*Canis lupus*) is an excellent example of a species with population-level behavioral differences that may reflect selective effects of differing environments on those populations. Prior to the spread of humans from Europe into North America, wolves were widely distributed on the continent and could be easily separated into subspecies based on their habitats. The gray wolf ranged across the north-central part of the continent, while the Mexican wolf (*Canis lupus baileyi*) was found in the arid Southwest and was half the weight of the gray wolf (see Figure 3.3). Thus, gray wolves are widespread, and we find much more

FIGURE 3.3
A gray wolf (left), a Mexican wolf (center), and an Arctic fox (right). *Photos: (Left) Frank Wendland, W.O.L.F. Sanctuary, www.wolfsanctuary.net, www.facebook.com/wolf.sanctuary; (Center) U.S. Fish and Wildlife Service; (Right) Keith Morehouse, U.S. Fish and Wildlife Service.*

behavioral variation among such widely distributed populations than among populations with more limited distribution and narrower habitat requirements (e.g., the Arctic fox, *Alopex lagopus*, which is specifically adapted to cold northern climates).

Larger patterns of behavioral evolution can sometimes be visualized by superimposing behavior on a phylogeny or cladogram of a taxonomic group. A phylogeny is a "tree" that expresses supposed relationships among taxa (see Chapter 1). Phylogenies can be constructed in a variety of ways and may follow the intuition of the scientist studying the evolution of the taxa in question. A cladogram is also an evolutionary "tree," but it is a well-defined hypothesis about relationships that is constructed following explicit rules about the analysis of evolutionary relationships; oftentimes, computers are required to implement the "cladistic analysis" of large data sets composed of multiple traits belonging to many species. Cladograms are typically constructed using a set of morphological characteristics, molecular characteristics, or both. Behavioral characteristics can be included in a cladistic analysis, but investigators interested in the evolution of behavior usually avoid circular reasoning by constructing a cladogram using morphological or molecular (nonbehavioral) characteristics and then observing how behavioral patterns fit into the cladogram. Cladistic analysis is most important in helping to test whether a particular behavior or syndrome of behaviors evolved only once in evolutionary history or if it evolved multiple times (convergent evolution). It can also help piece together the evolutionary sequence of events that lead to a behavior; for instance, there may be a shift from a simple behavioral pattern in basal species in the tree to a complex behavioral pattern in more derived species, or the behavior may have become simplified over evolutionary time, with derived species exhibiting only the core attributes of a behavior.

The same principles of cladistic analysis apply to the study of social evolution in sweat bees (family Halictidae). Some species of sweat bees are solitary; females establish nests of their own and do all the work without assistance. Others are social; several females

OF SPECIAL INTEREST: BIRD'S NEST SOUP—THE EVOLUTION OF NESTING BEHAVIOR IN SWIFTLETS

Swiftlets are abundant in South and East Asia. The nests of some swiftlet species are used as the basis for bird's nest soup in Asian cooking. Many swiftlet species build nests that are glued to a tree or a rock face (see Figure 3.4A). The glue is a salivary secretion that is alleged to have marvelous nutritional and medicinal properties and is the key ingredient in the expensive soup. In a few species, the main nesting material is small twigs or feathers. In other species, the glue gains more prominence as a construction material, and in a few species, the glue is the predominant material. Lee et al.[20] chose four characteristics of swiftlet nests: whether glue or a rock ledge serves as the primary support for the nest, whether feathers are used in construction, whether twigs or other vegetation are used, and proximity of nests to each other (whether the birds nest in colonies).

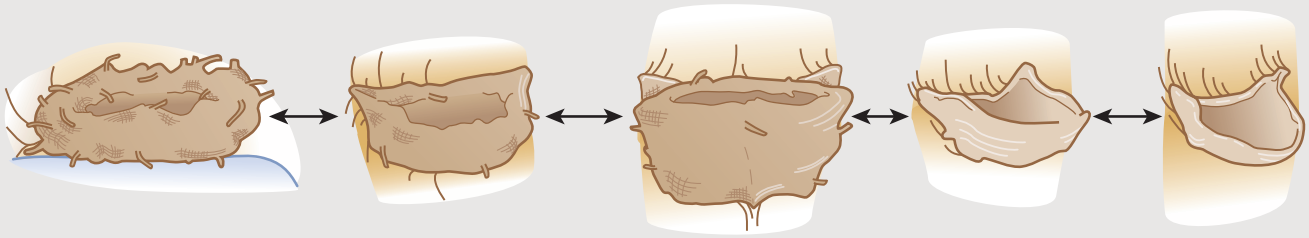

FIGURE 3.4A
Variation in swiftlet nests. Some are made almost entirely with salivary glue and adhere to a rock, whereas others sit on rock and are made of twigs or other plant material.

They then constructed a cladogram for the bird species and examined how well the behavioral variables fit with the pattern of swiftlet evolution (see Figure 3.4B). If the use of salivary glue as the main support for the nest had evolved only once in swiftlets, the expected result would be for this behavior to appear on only one branch of the cladogram. In fact, the behavior appears on three different branches. This evidence shows how a behavior can evolve more than once within a single group of related species.

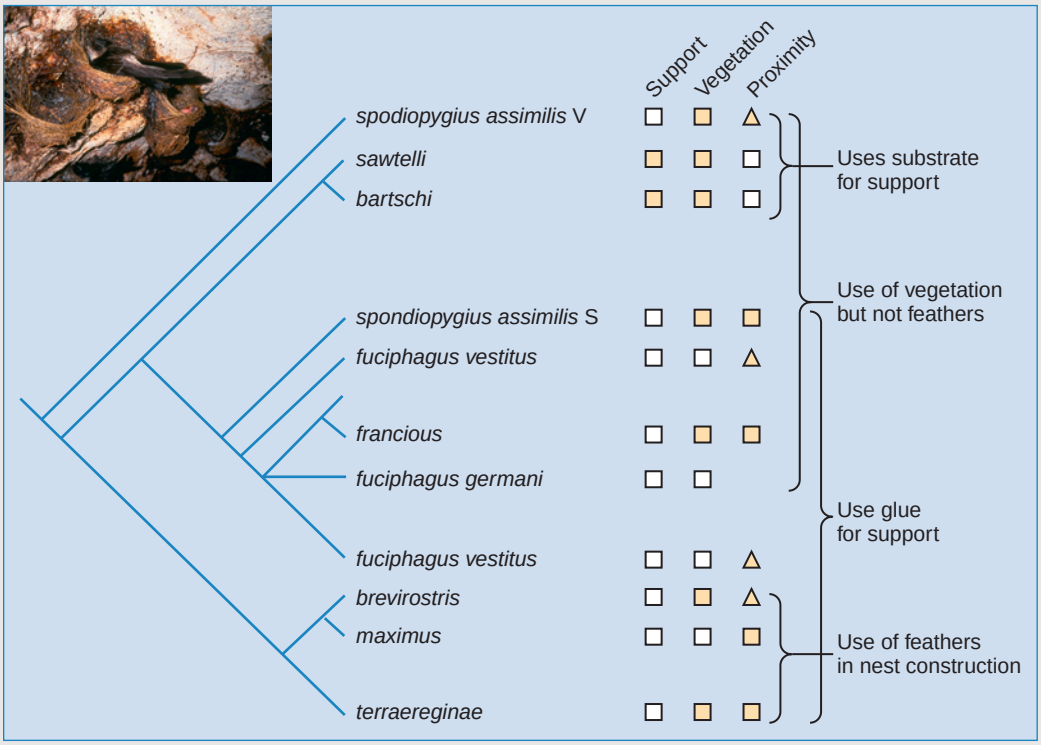

FIGURE 3.4B
A cladogram of swiftlets, with the behavioral traits shown on the right. *Source: Lee, PL, Clayton, DH, Griffiths, R & Page, RD 1996, Does behavior reflect phylogeny in swiftlets (Aves: Apodidae)? A test using cytochrome b mitochondrial DNA sequences. PNAS, Vol. 93, pp. 7091–7096. Photo: Dale Clayton.*

(usually a mother and her daughters or a group of sisters) occupy a nest, and labor in the colony is divided among the colony members. Did this shift from solitary nesting to complex social behavior happen only once in the course of sweat bee evolution, or has it happened repeatedly? Is it possible for the reverse to happen—for a solitary species to be derived from social ancestors? The cladogram (see Figure 3.5) helps to answer these questions; social behavior has evolved more than once in sweat bees, and there is at least one case of reversal, with a solitary species derived from social ancestors.[21]

Phylogenetic inertia is our final stop in thinking about the genetics that underlie the ultimate causes of behavior. Why, for example, does a dog turn around a few times before lying down (see Chapter 1, page 12)? The adaptive roots, or ultimate causes, of this behavior may lie in ancestral preparation for sleep by trampling vegetation or in checking for potential parasites; in a domestic dog, the behavior is charming but meaningless. It is always possible that an observed behavior or phenotype had a function in evolutionary history that is now lost; the behavior persists because it is engrained in the genetic instructions the animal receives from previous generations.

KEY TERM Phylogenetic inertia is the carryover of traits that evolved in previous habitats, even if those traits have little relevance in the current habitat.

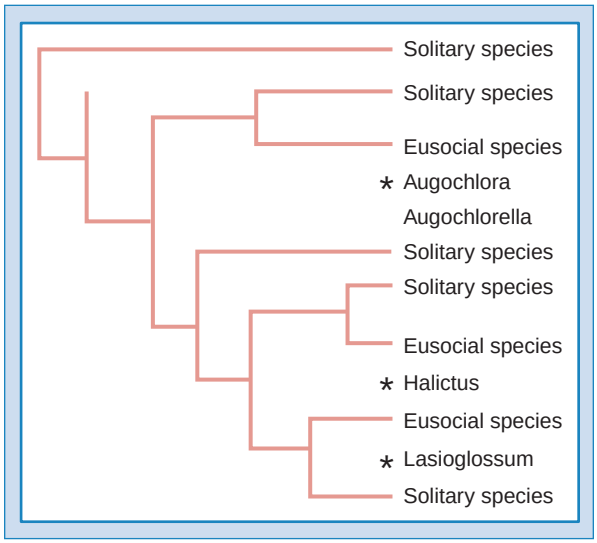

FIGURE 3.5
A cladogram of sweat bees, showing the points at which the worker caste has evolved. This cladogram shows that even very complex behavior can evolve multiple times within a larger taxonomic group. *Source: Brady, S.G., Sipes, S., Pearson, A., & Danforth, B.N., 2006, Recent and simultaneous origins of eusociality in halictid bees, Proceedings of the Royal Society B-Biological Sciences, 273 (1594), pp. 1643–1649.*

Proximate Causes and Correlations: Whole Organism Studies of the Genetic Bases of Behavior

CROSS-FOSTERING

Cross-fostering is a simple and highly effective technique for separating the effect of rearing environment from genetic (instinctive) influences on behavior. Consider two species of swiftlet: species A, which grows up in nests that resemble weaving, and species B, which grows up in well-constructed mud cups. If members of species A are reared in nests of species B, then how they build their own nests will depend on whether experience is important shaping nest-building behavior. If the behavior is entirely genetically programmed, then species A animals will weave nests, even if they are reared in a species B nest. If the behavior is shaped by learning and experience, then species A will learn and adapt to species B's mud construction.

The basic procedure of ***cross-fostering*** involves transferring some newly born or hatched young of species A from their parental nest to the nests of species B. Ideally, if there are multiple offspring from a given mating, the clutch or litter would be subdivided, with some going to B, and some staying home with A. The converse happens as well—that is, some young of species B are moved to homes with A, while others remain with species B. This results in four groups: species A raising A, species A raising B, species B raising A, and species B raising B. This sets the stage for asking what happens to the behavior under these four conditions. Does the behavior reflect their genetic background (the behavior of their parents) or their social background (the behavior of the family that reared them)? After a period of time, during which the behavior of the focal animals develops, the behavior of the transferred (fostered) animals and the non-fostered controls is documented, measured, and compared. If the

KEY TERM Cross-fostering is transfer of young between mothers. This technique is used to separate genetic from environmental influences on behavior.

transferred animals behave like the controls of their species, then the usual conclusion is that genetics dominates, and if they behave like their adopted "family" in the host nest, then environmental influences are predominant.

A recent study highlighted the utility of cross-fostering experiments in separating genetic from environmental (learned) components in bird song (see Figure 3.6).[22] Johannessen and his colleagues wondered if great tits and blue tits, both common in Europe, learned songs from tutors (parental birds) in their own nest. Because these species nest in the same habitat, it would be possible for young birds to pick up songs from birds in their own nest, from neighboring nests of the same species, or from neighboring nests of other species. The investigators located nests and transferred some eggs between nests of the two species. This resulted in a classical cross-fostering design, in which treatment individuals

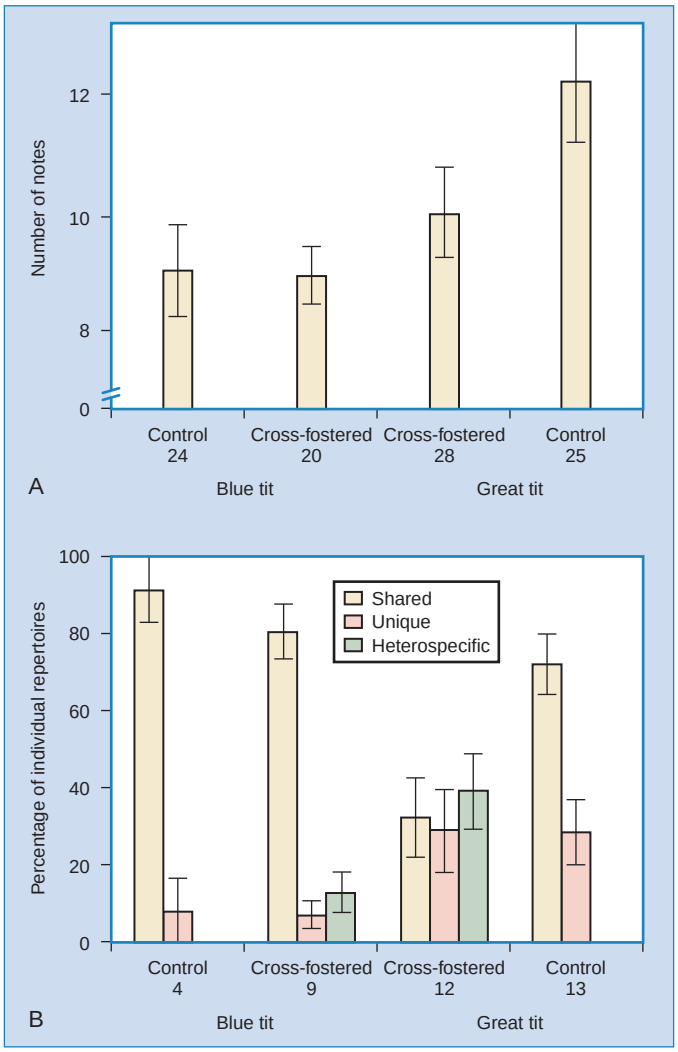

FIGURE 3.6
Results of cross-fostering experiments between blue and great tits in which song-learning of the nestlings was measured. Cross-fostered birds use songs that are intermediate between the two species. (A) Cross-fostered great tits have fewer notes and shorter songs than control birds of the same species. (B) Heterospecific songs appear as part of the repertoire of cross-fostered birds of both species. *Adapted from Johannessen et al., 2006, Animal Behaviour 72: 83–95.*

are reared by birds of other species and control animals are reared by their own species. Because the experiment involved two species, the test had the added dimension of allowing the scientists to ask if song learning in these birds is flexible enough to accommodate songs from a different species. Blue tit songs were very similar between control and cross-fostered birds, whereas the songs of cross-fostered great tits took on many of the characteristics of blue tit foster parents. Elements of the song of the biological (same species) parent remained, but because the experiment was conducted in the field, the results do not indicate if these song elements are genetically derived or if they are learned from birds in neighboring nests. Further experimentation using a cross-fostering design in a more controlled setting would sort this out. After leaving the nest, cross-fostered birds in this experiment appeared to prefer the company of their foster species to that of their own species, suggesting that song learning is part of a larger syndrome of species-specific imprinting (see Chapter 5).[23]

DISCUSSION POINT: CROSS-FOSTERING

What needs to be done to make sure a cross-fostering experiment is adequately controlled so that genetic and environmental components of a behavior can be separated?

Although intrauterine conditions are often not considered when studying environmental effects, they are some of the first environmental conditions than an animal encounters. Can the intrauterine environment affect the later behavior of animals? (See Chapter 2 for more discussion of this topic.) How can maternal influence and genetic effects be distinguished? Cross-fostering can play a role here as well. Embryos implanted into recipient uteri can serve as a control for prebirth effects of maternal physiology on the behavior of offspring. This technique has been used in mice, with the intriguing result that cross-fostered embryos behave like the recipient strain of mice, yet if cross-fostering is performed after birth, then the mice behave like their biological parents (see Figure 3.7). This result does not invalidate cross-fostering experiments done after birth or hatching, but it does raise the interesting caution that prenatal effects need to be considered when designing cross-fostering experiments. In humans, some investigators think that altered maternal well-being (e.g., stress) changes the uterine environment, resulting in lifelong effects on the behavior of the offspring.

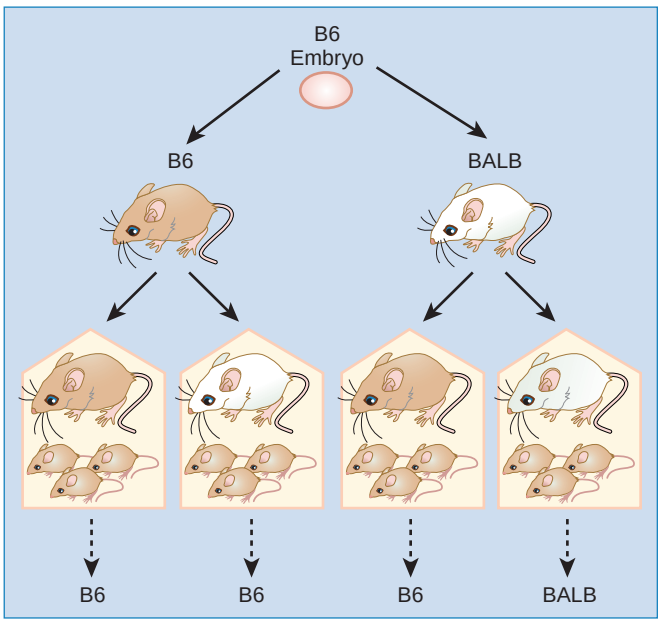

FIGURE 3.7
B6 mice embryos were implanted in mice of two strains (B6 or BALB). The offspring, which were all genetically identical, were raised by either B6 or BALB mothers. The only offspring that exhibited BALB behavioral traits were those that had been both prenatally and postnatally fostered by BALB mothers. *Adapted from Crabbe and Phillips, 2003, Nature Neuroscience.*

In other mammals, placental position can have the same sorts of effects on adult behavior; in mammals with three or more embryos in the uterus, offspring behavior after birth is affected by relative position (front to back) in the uterus and by the gender of adjacent embryos in the uterus. The influence of maternal effects and interactions between embryos on gene expression in offspring is one example of "epigenetic" effects, that is, variable functions of genes that do not involve actual changes in DNA. This variation can be caused by a broad array of environmental influences that are only beginning to be understood but that are clearly critical to attempts to parse the phenotypic contributions of "nature" and "nurture" (see Section 2.3 for more on this topic).

TWIN STUDIES

Twin studies are similar to cross-fostering tests of the relative importance of genes and environment in shaping behavior. These studies are most commonly performed in humans, for whom cross-fostering experiments are ethically questionable. They take advantage of the fact that sometimes human twins are monozygotic (MZ) and sometimes they are dizygotic (DZ). Monozygotic twins come from the same fertilized egg and are genetically identical (see Figure 3.8); DZ twins come from separate fertilized eggs and thus are no more related to each other than if they were siblings born at different times. Assuming that both twins in a pair experience the same rearing environment, then experimenters can compare similarities (concordances) and dissimilarities (discordances) in behavior between MZ and DZ twins; if MZ twins show greater degrees of behavioral concordance than DZ twins do, those differences are probably due to genetics because rearing environments are controlled. For instance, Deater-Deckard and colleagues recently found that behavioral attributes such as task persistence, anger/frustration, and conduct problems in children were much more similar in MZ twins than in DZ twins, although environmental influences also have substantial importance. Perfect concordance of behavioral traits is rare even in MZ twins, suggesting that genes are not the sole influence on behavior; environment and chance play large roles as well. Monozygotic twins occur in at least some nonhuman primates[24] and perhaps in other animals, so the possibility of using twin techniques for behavioral studies extends beyond humans, although detection of MZ offspring in nonhumans requires genetic testing.

KEY TERM Twin studies can segregate environmental from genetic influences because twins are genetically identical, but may be reared in different habitats.

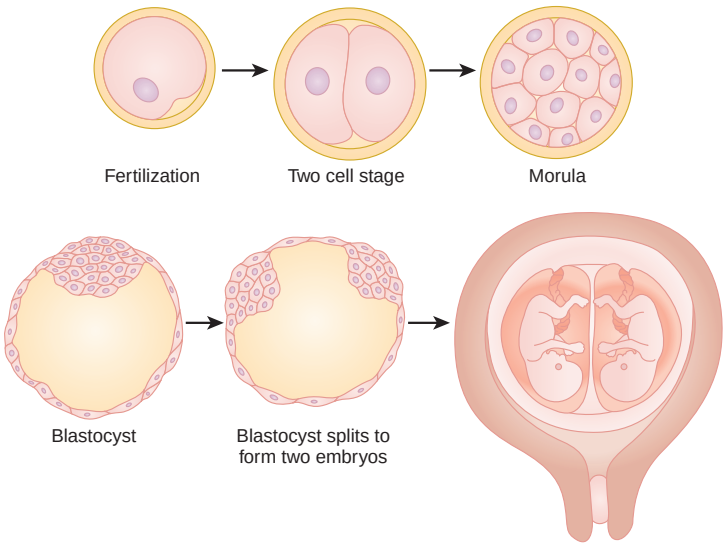

FIGURE 3.8
Embryogenesis of monozygotic twins.

CLONED ANIMALS

Cloned animals offer a major potential for application of the principles of twin analyses in unique ways: a clone offers the opportunity to observe the genetic equivalent of MZ twins born at different times! If a favorite pet is cloned, will the clone's behavior resemble the original pet's behavior in the desired ways? Human monozygotic twins are often quite similar behaviorally, sometimes eerily so in behavioral quirks and mannerisms, but also can differ in significant ways; if cloning of pets gains widespread acceptance, it will be interesting to see how well pet owner expectations are met.

KEY TERM Cloned animals are genetically identical. Use of cloned animals extends the idea of twin studies by allowing larger sample sizes and more controlled conditions.

ARTIFICIAL SELECTION

Artificial selection and ***inbred lines*** allow exploration of behavioral genetics by testing the responses of behavior to selection or to reduction of genetic variation. Recall from Chapter 1 that genetic variation is necessary for either natural or artificial selection to produce shifts in gene frequencies, and the

KEY TERM An inbred line is a population in which closely related animals, such as siblings or parents and offspring, have been repeatedly mated so that nearly all genetic variation is lost. This is similar in effect to cloning.

only traits that can be selected are those found within the range of variation genetic variation present in the population. The potential for selection to modify a trait is assessed by measuring its heritability (see below), one estimate of genetic variation.

Artificial selection, in scientific laboratories and in animal husbandry, has dramatic effects on behavior. Perhaps the broadest range of artificially selected behavior is seen in domestic dogs, which display a wide variety of behavioral attributes. These behavioral patterns are the result of selection for dogs that assist humans in work (e.g., retrievers, shepherds) or as companion animals. Most domestic livestock (such as chickens, horses, cattle, sheep, goats, and swine) reflect the results of artificial selection for manageability in confinement, ease of training, and docility (see Figure 3.9).

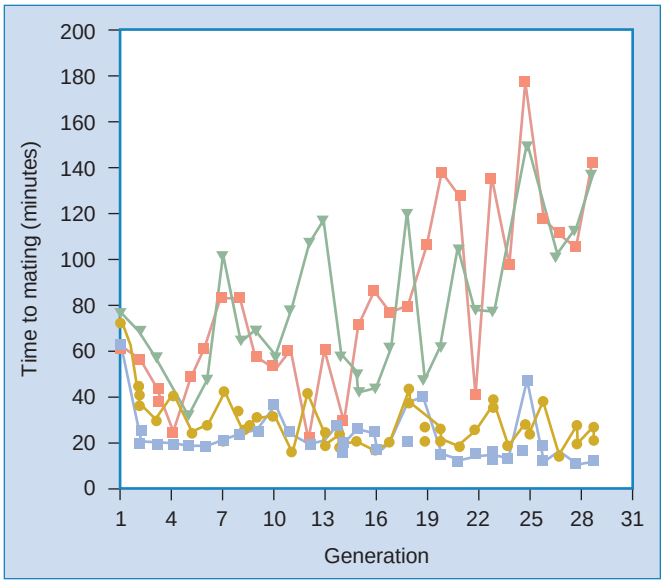

FIGURE 3.9
The response of fruit flies to artificial selection for mating speed. Each symbol represents a line of flies selected for high or low speed. Over generations the lines diverge; after roughly 7–10 generations the differences are apparent and by the 30th generation the differences are extreme. The end-of-chapter discussion of the application of microarrays to behavioral genetics will include this example. *Adapted from Mackay T.F., et al., 2005 Proc Natl Acad Sci U S A May 3 102 Suppl 1:6622–9*

SINGLE-GENE MUTATIONS

A single-gene mutation is a genetic change in just one gene. Such a mutation, spreading rapidly through a broad geographic area, has dramatically changed the social behavior of the black imported fire ant, *Solenopsis richteri*, and the red imported fire ant, *Solenopis invicta* (both introduced via cargo ships to the United States in the twentieth century). The behavior of animals in their natural setting results from many generations of natural selection; the results of this natural selection are observed more often than is natural selection itself in action. Examples of rapid evolutionary change in behavior are relatively rare, but the introduction of exotic and animals (and plants) into ecosystems creates new opportunities for natural selection to act. Animals in new (to them) environments face dramatically changed regimes of selection, which can lead to strikingly rapid evolutionary change.

Colonies of *Solenopsis invicta* are normally monogynous, meaning each colony has a single queen and the workers in the colony are daughters of that queen. Growth of monogynous colonies is limited by the number of eggs this single queen can lay, and by how much food can be collected to feed to the developing larvae. For this ant in its native habitats in Argentina and southern Brazil, the reproductive capacity of a limited number of queens is adequate for colonies to be competitive. Once this species was introduced into the southeastern United States, it encountered a habitat devoid of its natural predators, parasites, and competitors. These conditions favor colonies with the capacity for explosive growth. This growth can be attained by having many queens per colony (polygyny), each laying large numbers of eggs.

The difference between monogynous and polygynous fire ants lies in the expression of a single gene, Gp-9, that codes for a pheromone receptor molecule. The mutation appears to rob the worker ants of the ability to discriminate among queens (see Figure 3.10); consequently, they tolerate a large number of queens, including queens that are genetically unrelated to each other or the workers. This is an excellent example of how a change in a single genetic component that underlies a complex social system can have major effects on the function of the entire social structure. Recent discoveries of the polygynous form of *Solenopsis invicta* in Taiwan, Australia, and China underscore the effectiveness of the mutation in facilitating ant invasions.[25]

Another invasive ant, the Argentine ant (*Linepithema humile*), has been highly successful, possibly because it exhibits reduced intraspecific aggression. A genetic study using microsatellite DNA markers showed that these ants experienced a population bottleneck

FIGURE 3.10
A fire ant queen with workers and eggs. *Photo courtesy of Sanford Porter/USDA-ARS.*

during introduction to California that reduced genetic diversity. This is consistent with the hypothesis that reduced genetic variability may impair nestmate or colony recognition, allowing the invaders to behave like one large colony. This, in turn, allows them rise to higher densities than they do in their native habitat.[26]

The fruit fly, *Drosophila melanogaster*, is the workhorse of laboratory genetics.[27] It is raised easily in large numbers in the laboratory and has a fast generation time. This makes it a good "model system" (see Chapter 1). Many genetic mutations affect fruit fly mating behavior. Male behavior is inhibited by genes named *Nerd* and *fruitless* and is enhanced by *Voila*. Female receptiveness to mating is impaired by genes such as *dissatisfaction*, *spinster*, and *chaste*. The ways that these genes might affect courtship and mating become more apparent when the sequence of courtship behavior and related sensory modes are considered. Visual, auditory, and chemosensory systems may all be affected.

For instance, mutations that affect the visual system reduce fruit fly male mating success by making it difficult for males to find females (see Figure 3.11). Auditory mutations make females less receptive to mating because they cannot hear the male courtship song. Chemosensory mutations can affect the courtship performance of sexes. In fact, *Voila* seems to affect chemosensory cells in the male's front legs. Mutations that affect processes in the brain are even more interesting because they can give insight into the organization of the central nervous system and the generation of complex behaviors such as mating. Fruit flies offer insights into this level of behavioral control as well.

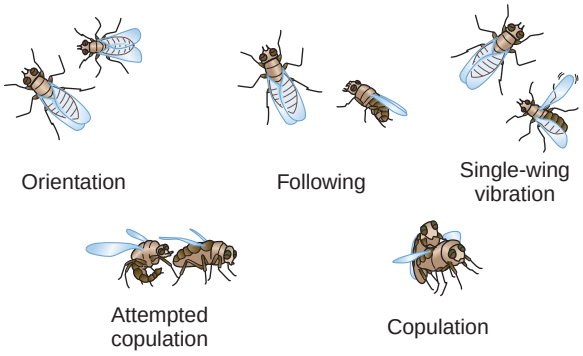

FIGURE 3.11
Drosophila courtship; these are examples of behaviors that mutations may affect.

HERITABILITY

Heritability provides a powerful tool for measuring the variation in a behavior and for allocating it to variation correlated with genes or variation correlated with environment.[28] Heritability is the proportion of phenotypic variation in a population due to genetic variation. *Variation* is the operative word here: It is important to understand and remember that heritability is all about variation such that studies of the heritability of a behavior are actually studies of the variability of that behavior within a population.

Understanding heritability and how it is used rests on the following key concepts:

1. Heritability is a population-level measure, not an individual measure; only populations have heritability.
2. Heritability is NOT a measure of the degree of genetic control of a behavioral trait.
3. Heritability can differ among environments; the same population may show a different heritability for a trait if the environment is changed.
4. The behavior in question must vary among individuals in the population for it to have a measurable heritability; invariable behavior has no heritability.
5. Strong selection (natural, sexual, or artificial) on a behavioral trait reduces the heritability of that trait (because it reduces the variation).

Keep these principles in mind through the following discussion of heritability.

A recurring theme in this chapter has been the fact that phenotype is determined by the collaboration of an animal's genes and the effects of its environment, past and present.

The study of heritability allows students of behavior to parse those influences: heritability indicates how much of the variation in phenotype can be ascribed to genetic variation. Consider a population of animals and its heritability, using the following notation:

Vp = variation of the phenotype
Ve = variation due to the environment
Vg = variation due to genetic effects

With these three notations, a simple equation can be generated that describes the relationship among phenotype, environment, and genes: Vp=Vg+Ve. This is a restatement of a central concept: phenotypic variation is the sum of environmental and genetic variation.

There are two types of ***genetic variation*** (Vg): additive and nonadditive. Recall that animals can differ genetically because they have differing alleles (forms of a gene) at loci (locations on the chromosome) that influence the trait in question. Usually, the effects of the different alleles working together is the sum of their activity, so genetic variation due to allelic differences is called ***additive genetic variation*** (Va). Sometimes one allele is dominant to another in determining the effect of a locus, or loci may interact in ways that change the action of the genes (***epistasis***). Variation due to dominance, epistasis, and other types of interactions among alleles and loci is ***nonadditive*** (Vna).[29] Total genetic variation, Vg, is the sum of the additive and nonadditive components, Vg=Va+Vna.

KEY TERM Genetic variation is a measure of the variation in phenotype that is due to all variation in genotype.

KEY TERM Additive genetic variation is the proportion of genetic variation that is due to simple additive effects among genes.

KEY TERM Nonadditive genetic variation results from interactions between genes and from gene dominance.

KEY TERM Epistasis occurs when genes interact.

Taking another approach, recall that heritability is the proportion—or percentage—of the phenotypic variation that can be attributed to genetic influences. Like any other percentage, to get this proportion, divide the genetic variation by the phenotypic variation (Vg/Vp). This yields a measure called broad-sense heritability. Notice that ***broad-sense heritability***, H^2, involves both additive and nonadditive genetic variation. When only additive genetic variation is used in the calculation, then the result is ***narrow-sense heritability***, $h^2 = Va/Vp$.

KEY TERM Broad-sense heritability is the proportion of phenotypic variation that is explained by genetic variation.

Given this explanation, how might heritability be used in studies of animal behavior? What have studies of heritability of behavior found? Heritability is useful in two ways:

KEY TERM Narrow-sense heritability is the proportion of phenotypic variation that is explained by additive genetic variation.

- Broad-sense heritability is used as a measure of the magnitude of genetic influences on a trait.
- Narrow-sense heritability is particularly useful in predicting how animals will respond to artificial or natural selection. If a trait has a high heritability, selection or controlled breeding can change that trait because high heritability is based on a high level of genetic variation that can be subject to selection. (Remember, low genetic variation does not give selection much "wiggle room.")

BRINGING ANIMAL BEHAVIOR HOME: HERITABILITY AND THE BEHAVIORAL GENETICS OF DOGS

The fact that much of dog behavior has genetic underpinnings is patently obvious. Differences in temperament and ability among breeds are well known. These differences are generally associated with the purposes for which the breed was developed. This brief summary, which is typical of how dog breeds can be classified (see Figure 3.12), suggests some of the traits that have been subject to selection:

1. Sheepdogs (shepherds, collies, and the like) are selected for their keen ability to focus on sheep or cattle, their ability to manipulate the behavior of these animals, and their ability to learn and follow their handler's commands. Some think that much of the herding behavior is derived from predatory behavior, but the actual killing behavior has been suppressed.
2. Terriers are energetic hunters, very attracted to small animals. They are quite willing to follow their prey down burrows, hence their name—derived from *terra*, or earth—not terror, as some owners insist. They dig.
3. Scent hounds (including beagles, bassets, fox and coonhounds) are able to behaviorally exploit their keen sense of smell in tracking prey.
4. Retrievers (including labrador and golden), known also as gun dogs, are selected for retrieving ability. A specific behavior that has been selected is a "soft mouth," the ability to handle prey without damaging the item or attempting to consume it.
5. Companions and Toys (miniature and toy poodles, Pekingese, Chihuahua) display behavioral traits that make them attractive household pets.
6. Sighthounds (afghans, borzois, greyhounds) use their distance vision to track prey. They are also selected for high running speed and endurance.

FIGURE 3.12
The domestic dog, *Canis familiaris*, is strong testimony to the variation that can exist within a biological lineage.

In general, dog breeds that are recognized by groups such as the American Kennel Club (AKC) "breed true." This means that pairing any male and any female in the breed will result in pups with the breed-specific characteristics. If you think about this, you will recognize that the only way to accomplish "breeding true" is through reduction of genetic variation related to those breed-specific traits. Any one breed of dogs will have less genetic variation within the breed than you find if you look across all dogs. At some point, breeding of this sort will eliminate most or all of the additive genetic variation in the breed for some traits. At the point at which the additive genetic variation for a trait has been exhausted, no further "improvement" of the breed (depending on the trait) is possible even through carefully designed pairings.

Thinking about how heritability is calculated, it follows that within any dog breed you would expect to find low heritabilities, particularly for the traits that are thought to characterize the breed. Does this mean that the traits do not have a genetic underpinning? No—not at all. It simply means that further attempts to select for the trait will be futile. Following this line of reasoning, you would expect to observe higher heritabilities for traits (including, of course, behavioral traits) if you include a variety of dog breeds in a study, and lower heritabilities if you focus on only one dog breed.

Most measures of heritabilities of dog behavioral traits are from studies of single breeds. A wide range of behaviors have been measured, such as "willingness," fighting the leash, hare tracking, and "obedience." Nearly all of the studies were performed within breeds (the alternative would be to do controlled matings between breeds). Generally, heritabilities for behavioral traits range from 0 to 0.25. Heritabilities for personality traits of German Shepherds are 0.24, or less.[30]

Does this mean, then, that mixed-breed dogs have more hybrid vigor? A careful breeder of purebred dogs will have used genetic testing and careful pedigree scrutiny to minimize the occurrence of genetic diseases in her kennel's lineage. If the same scrutiny is not applied to the production of a mixed-breed litter, there is no reason to expect such a healthy outcome. A puppy-mill breeder of purebred dogs will often use inbreeding and back-crossing, with no concern for genetics, to produce unhealthy puppies. In short, it is the exclusion (or inclusion) of undesirable genes, and not the heritability of other traits, that contributes to healthy or unhealthy puppies.

How is heritability measured? Assessing heritability often boils down to how much parents resemble their offspring, or offspring resemble each other. Generally, heritability is calculated by looking at associations between the expression of the trait in families. Mechanically, this is easy to do; the average value of the two parents (not surprisingly, this is called the *midparent value*) is graphed on the x-axis, and the value for the offspring goes on the y-axis. If enough parent–offspring pairs are analyzed, the slope of the resulting line is the heritability. In some cases for traits that relate to sexual behavior, such as antlers in deer, the trait is expressed in only one gender; if single-parent values, rather than midparent values, are used, the heritability is twice the slope. Similar techniques can be used if data are available for groups of siblings. The result is a number between 0.0 and 1.0, with the extremes being no phenotypic variation due to additive genetic variation (zero or 0.0) and no phenotypic variation associated with environmental variation (1.0). The most common way of calculating heritability of a trait is by using linear regression analysis, a statistical technique that yields the equation for a line describing the effect of one variable on another. When the phenotypic values for offspring are regressed on the parental values, the slope of the resulting line is the heritability.

Another way of looking at heritability is to explore how a behavioral trait responds to either natural or artificial selection. If selection on the trait results in change from generation to generation, this suggests that the trait is heritable because selection can act only if there is genetic variation for the trait. On the other hand, if selection has no effect on a trait, then its heritability is probably low. Another way of saying this is that a heritable trait is a selectable trait.

OF SPECIAL INTEREST: CALCULATING HERITABILITY

Figure 3.13 shows the heights of 50 male and 50 female students, in centimeters. We also asked these students to give us their parents' heights. We averaged the father's and mother's height; this gives us the *midparent* value for each student.

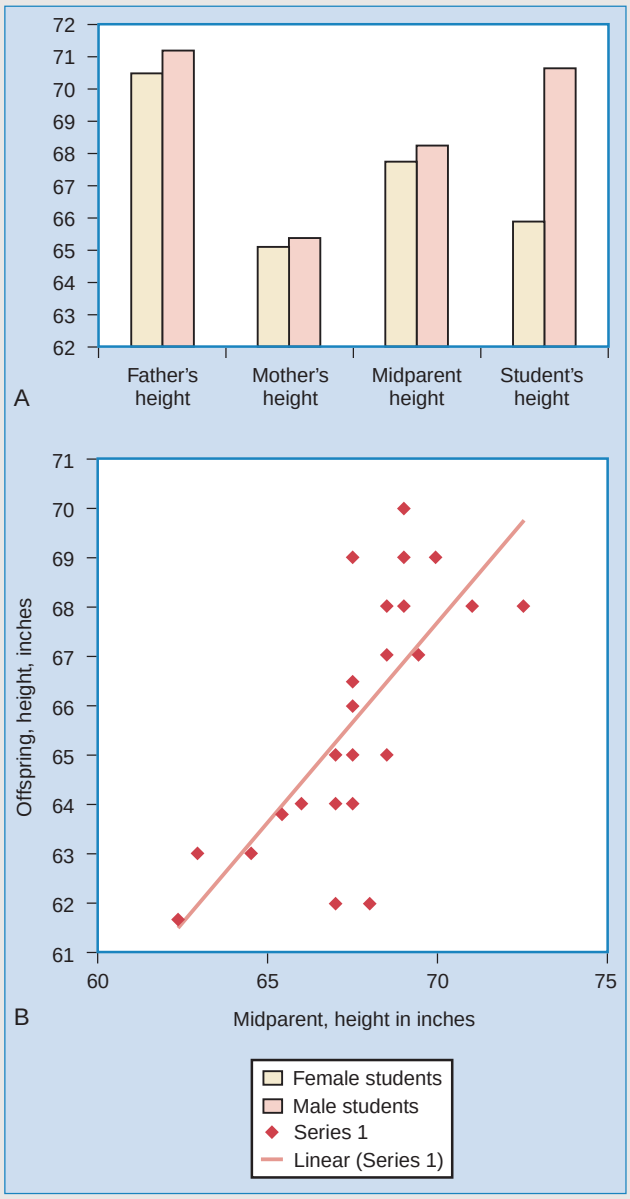

FIGURE 3.13
(A) Bar graph of male student heights and female student heights compared to the height of their parents. Midparent is the average of the two parents' heights. (B) Regression analyses of the male and female student heights against their midparent values. The slope of the regression line and the heritability is 0.82.

Heritability studies have many applications in animal behavior (see Figure 3.14). Indeed, using artificial selection (10 generations) and house mice, Swallow and colleagues[31] showed that voluntary wheel-running could be increased by 75%. Interestingly, these increases were expressed as increased revolutions per minute, not increased minutes per day, meaning that the mice did not run longer, but ran faster.

Following these thoughts about heritability and selection, it is reasonable to predict that strong selection reduces heritability to zero. Why? In theory, strong natural selection should eliminate all additive genetic variation because the alleles favored by selection will be the only ones remaining in the population. Without additive genetic variation, the narrow-sense

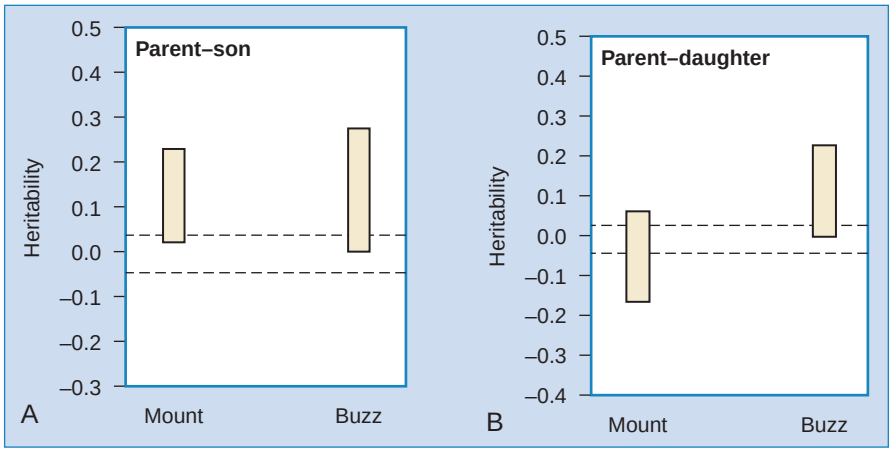

FIGURE 3.14
Heritability analysis of courtship in houseflies. For both behaviors, buzz and mount, the heritability is significant for the parent-son analysis, but only buzz is significant for the parent-daughter treatment. *Adapted from Meffert et al., American Naturalist 160: S198–S213 Suppl. S DEC 2002.*

heritability is zero, and no further evolutionary change in the trait would be expected. This theoretical prediction is supported by data in some cases, whereas in others there is substantial heritability of traits that nonetheless seem to have been under strong selection. In animal behavior, one particular concern is what this prediction means for traits related to behavior such as dominance and mate choice.

KEY TERM Balancing selection occurs when extreme phenotypes are at an advantage, so selection favors the maintenance of both phenotypes. For example, very large and very small animals may be at an advantage in mating, so selection would favor these phenotypes.

Why might additive genetic variation persist over evolutionary time in the face of seemingly strong selection? Four mechanisms commonly explain this (refer to the evolution primer in Chapter 1):

- ***Balancing selection.*** This type of selection occurs when more than one phenotype is favored. It could occur among animals in competition (as in calling males and satellite males in a mating chorus) or between generations, as when environmental conditions shift, so that more than one phenotype is favored over time.
- ***Epistasis.*** These are interactions among genes. While one phenotype may be favored, the alleles' effects on other genes may be deleterious, preventing fixation of the allele. Fixation is elimination of all but one allele; when a character or trait is fixed, no genetic variation remains.
- ***Correlated characteristics in males and females.*** If a trait that is important in one sex, either for competition for mates (as in horns or antlers in male ungulates) or in mate choice, is produced by both sexes, then the deleterious effect on the other sex may outweigh selection for maximizing the trait. For example, in barn swallows tail length varies in both males and females but is a sexually selected trait only in males (see Figure 3.15).[32]
- ***The handicap principle.*** If producing a phenotype is costly enough, selection against extreme individuals may counterbalance selection favoring that phenotype. This is a form of balancing selection, driven by the cost of the phenotype. The basis of this idea has been most strongly promoted by Amotz Zahavi, but it has gained general acceptance in this somewhat modified form.

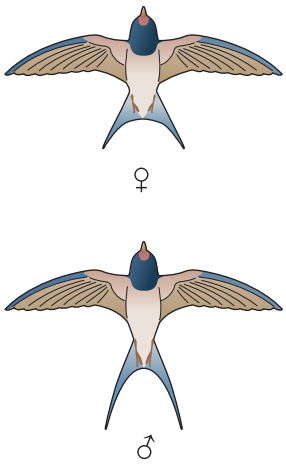

FIGURE 3.15
Male and female barn swallow tails.

What does high or low heritability mean, in terms of history of selection? Low heritability results from strong selection on a trait, so key features for an animal's survival are expected to have low heritability. High heritability, correspondingly, reflects a more benign selective environment; genetic variation is tolerated if selection is not high. The calculation of heritability of traits in Japanese quail is an outstanding example of the use of heritability in a behavioral study.

KEY TERM The handicap principle states that production of a phenotype may be costly, making that phenotype an honest signal of an animal's condition. An honest signal is a phenotype that conveys information about the signaler's genotype or current condition. Dishonest signals may deceive the recipient about the genotype or condition of the signaler.

OF SPECIAL INTEREST: DOMINANCE AND HERITABILITY IN JAPANESE QUAIL

Both male and female Japanese quail (*Coturnix japonica*) start pecking other birds soon after hatching. In adults, males and females establish dominance hierarchies (see Chapter 9). In an interesting study, heritability for a large number of behavioral traits was determined in Japanese quail (see Figure 3.16). Dominant males have more mating success, supporting the hypothesis that strong selection has reduced additive genetic variation for dominance in males. Dominance rank has a higher heritability for males than for females. Heritability may be maintained in females because the trait has no fitness consequences or because there are balancing factors.[33]

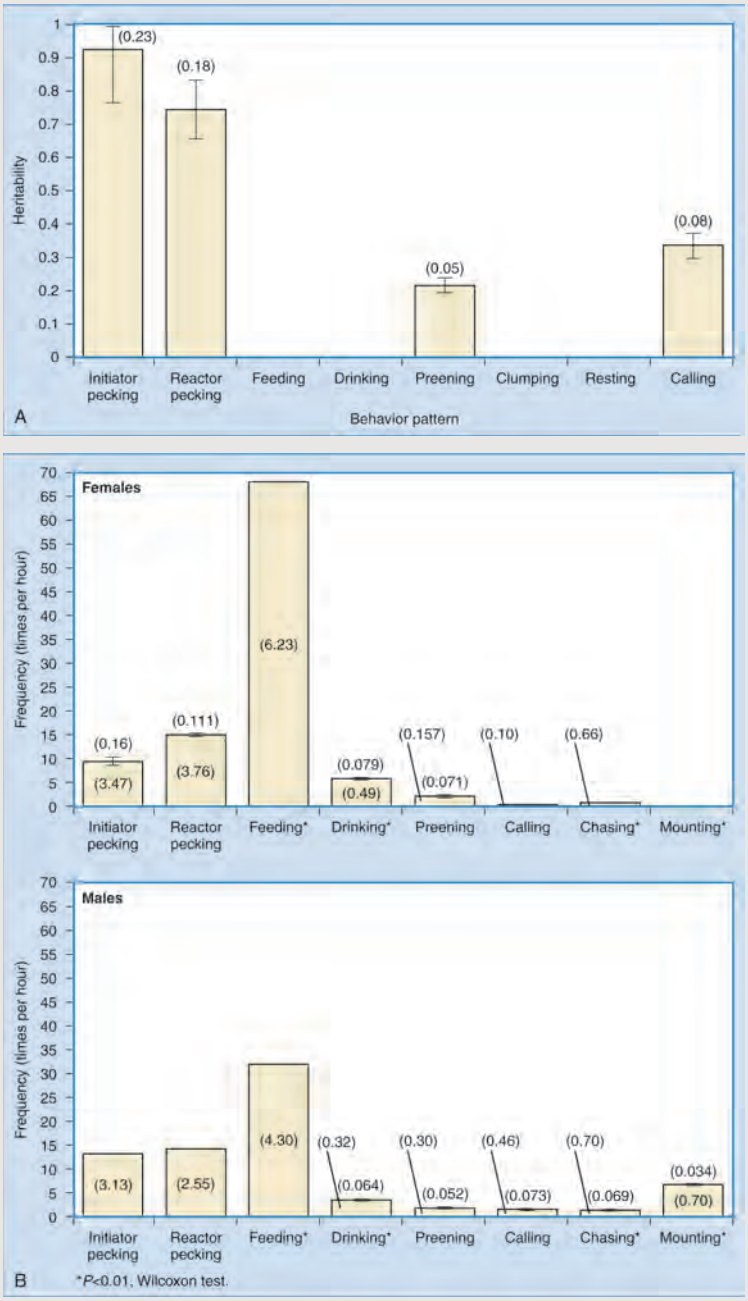

FIGURE 3.16
Heritabilities of four of the behavioral traits of Japanese quail (*Coturnix japonica*). In young birds pecking is heritable in both sexes (0.74 overall). In adults, however, heritability for pecking is high in females (1.33) but 0 in males. Other traits have moderate or low heritabilities. *Adapted from Nol et al., 1966, Animal Behaviour 52: 813–820.*

QUANTITATIVE TRAIT ANALYSIS

What is a quantitative trait? A quantitative trait varies continuously, in contrast to qualitative traits, which have discrete forms. Another aspect of a quantitative trait is that it is determined by a number of genes acting together. A good example of a quantitative trait is height in humans; "normal" adult humans range in height over a span of more than half a meter (more than 2 feet). Human height is greatly influenced by genes, but no one gene is solely responsible for height. Usually, quantitative traits are normally distributed; a graph of the trait results in a bell-shaped curve.

In these cases, a normally distributed trait, whether it is physical, such as height, or behavioral, such as pollen collection, is usually the result of the contribution of a number of genes. Recent advances in genetics now allow scientists to map the genes having the greatest influence on the trait. These genes, as described previously, are called quantitative trait loci, or QTLs. QTL analysis is now a standard format for studying genetic influences on behavior.[34,35]

It follows that a QTL is a location on a chromosome that is thought to regulate an organism's phenotype for quantitative trait. Other experimental approaches will give the same result, but all QTL analyses rely on a linkage map and good behavioral measures. Investigators find numerous markers such as generally single-nucleotide polymorphisms (SNPs), amplified fragment length polymorphisms (AFLs) or random amplification of polymorphic DNA (RAPDs) and determine their linkage group map locations by cross-breeding.

OF SPECIAL INTEREST: HOW IS A QTL ANALYSIS DONE?

To perform a QTL analysis, a behavioral biologist needs three critical sets of information: a linkage map, a behavioral assessment, and a breeding experiment.

First there must be a linkage map of the genome of the study species. This is a significant limiting factor for QTL analyses. In construction of a map, the first step is to find physical (such as the bands on *Drosophila* chromosomes) or molecular markers scattered throughout the animal's genome. This map needs to be well saturated; that is, the markers must be distributed evenly and frequently enough to assort during recombination.

Many types of molecular markers are available; the linkage map for honeybees, which will be our main example in this section, was constructed using RAPDs; these are small DNA segments that can be identified using polymerase chain reaction (PCR) techniques.

To visualize how the data are analyzed, think of a chromosome, remembering that in most animals chromosomes come in pairs. The scientist has genetic markers (short sequences of DNA) scattered along the chromosome; at a given location, the DNA sequences of these markers vary between the two copies of the chromosome. During meiosis, the two members of the chromosome pair cross over and segments of the chromosomes are exchanged, resulting in genetic recombination. Think of two different markers along the chromosome.

Most importantly, the chances of recombination occurring are high if the markers are far apart, and low if the markers are close together. Now what do you think happens to genes that regulate behavior? If they are close to a marker, they likely stay linked with that marker during recombination. If they are far from a marker, then they are much less likely to stay linked. Hundreds, perhaps thousands, of markers are needed to produce a well-saturated map of an animal's genome. In honeybees, mapping has been facilitated by the fact that recombination rates are much higher than in most organisms.

Second there must be a good method available to measure how the behavior varies among animals.

Third all that remains is a breeding experiment. Typically, animals whose behavior has been measured and that have been genotyped are mated. These crossed animals with different behavioral phenotypes allow us to follow how the behavioral phenotype is correlated with the markers. The behavior and genotypes of their offspring are then determined, as well.

The following questions are usually addressed in a QTL analysis:

- How many genes influence the expression of a quantitative trait?
- What is the level of influence of each gene on the trait?
- Where are the genes located on the chromosomes?
- What is the function of each gene?

Answers to these questions will be discussed in the following section. Note that the last question, about function, is the most difficult to answer.

Using a rather elaborate computer program, scientists can ask whether the variation in behavior is correlated with each marker. They do this by focusing on one "family" (a male, female, and their offspring) and asking if the behavior is always high with a certain sequence of a marker and always low with another sequence. In that case, the gene for the behavior is likely to be close to the marker on the chromosome. If the variation in the behavior is more or less random in relation to the marker, then there is little or no linkage between the behavior and that chromosomal location. Most quantitative traits, including behavioral traits, have high correlations with from two to four locations in an organism's genome; that is, they are close to a marker. There are, of course, exceptions, but this is a good rule of thumb. The higher the correlation, the more important a gene may be in regulating the behavior. These chromosomal locations probably do not have genes that code directly for a particular behavior, but rather, ones that code for factors that shape the behavior. For example, a behavioral trait may be influenced by three genes, one of which affects activity (or arousal), another that affects sensory perception, and a third that influences latency to respond to a stimulus.

Many behaviors are quantitative traits. Aggressiveness, for example, varies among individuals in a wide range of animals, such as honeybees, rodents, horses, dogs,[36] and various primates. Another good example is expression of play behavior in mammals. Intelligence, to the extent that it can be measured, is also a quantitative trait. Activity levels are particularly good examples of quantitative traits.

Figure 3.17 shows a typical set of activity measurements—in this case, a QTL analysis of aggressive behavior (stinging) in honeybees. The purpose of this analysis was to identify the locations in the genome of loci responsible for the large difference in stinging behavior between the "African" honeybees in Mexico and the gentler bees found farther north. In an attempt to further focus in on the regulation of aggressiveness in honeybees, a later study sequenced the genome around the sting-2 QTL; this is the ***candidate gene*** approach, which is discussed in more detail in the next section of this chapter. [37,38]

KEY TERM A candidate gene is a gene that has been identified as having a strong possibility of playing a role in regulating a phenotype.[39]

What does knowing the locations of QTLs accomplish? In working with domestic animals and wanting to select for certain behavioral traits, the knowledge of the QTLs would allow the design of an efficient breeding program to achieve the desired result. This has not actually been done in animals, but the principle is commonly applied in crop plants. By knowing which chromosomal locations are important, scientists can, conceivably, track how their genetic manipulations are affecting the regulation of the trait.

BRINGING ANIMAL BEHAVIOR HOME

Canine compulsive disorder (CCD), in which behaviors are executed repeatedly and with some distress, shows a high resemblance to human obsessive-compulsive disorder (OCD). Dogs may lick, tail chase, flank suck, engage in pica (consumption of indigestible substances), and pace or circle. Using SNPs from Dobermans diagnosed with this malady, scientists have recently determined that a gene on chromosome 7 may be responsible for some susceptibility to CCD.[40] Sixty percent of dogs with multiple compulsive behaviors have the allele thought to confer risk (*CDH2*), compared to 22% of control Dobermans. Such studies open the door for early intervention into compulsive disorders and perhaps even treatment.

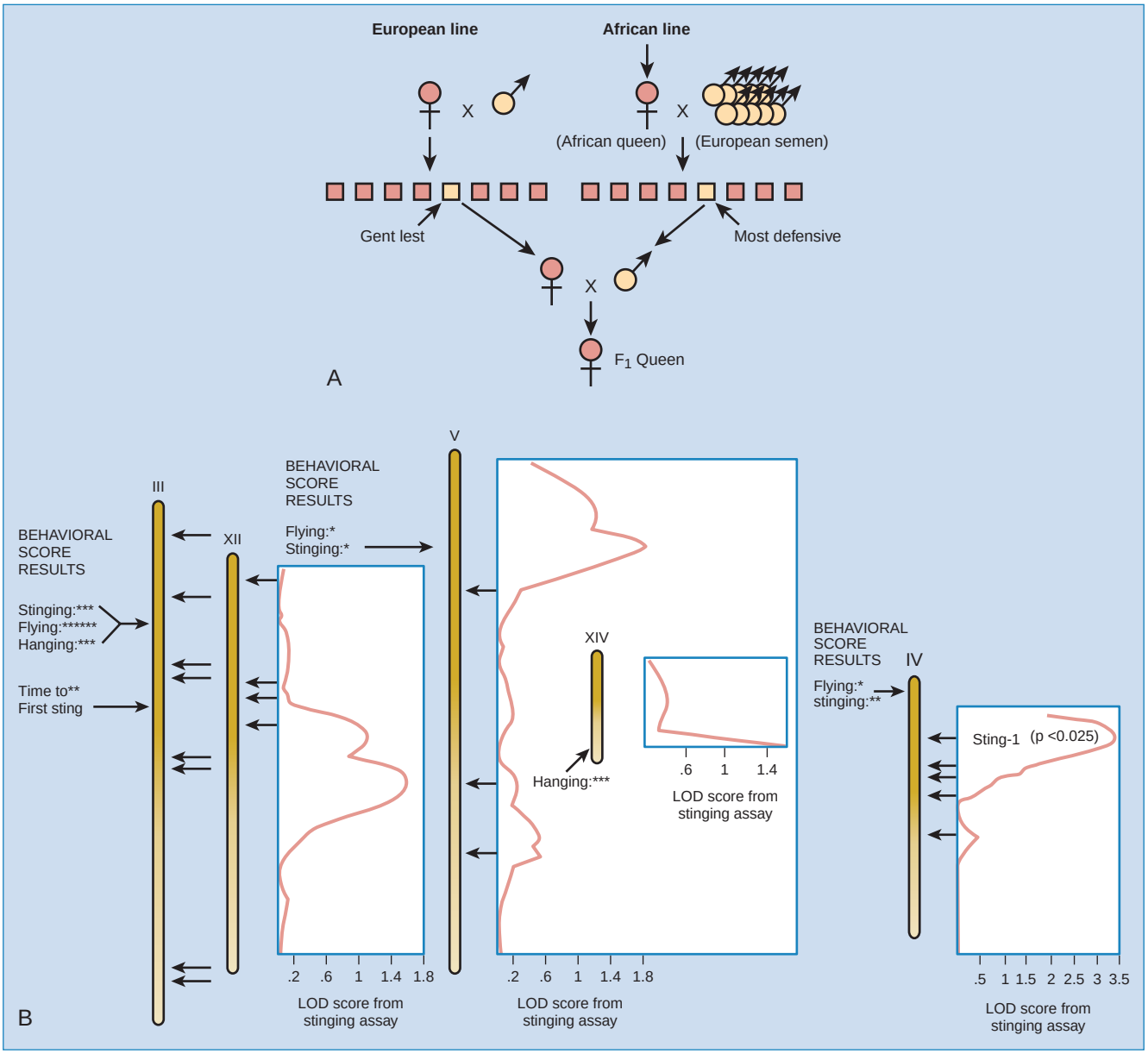

FIGURE 3.17

(A) Chromosomes and QTLs for stinging behavior in honeybees. (B) The crossing scheme used to map these behaviors, along with LOD analyses. The Roman numerals indicate chromosome number; each QTL for the behavior corresponds to a peak in the LOD analysis. *Adapted from Hunt et al. 1998. Genetics, Vol. 148, 1203–1213.*

Proximate Causes and Correlations: Genetic Dissections of Mechanisms Underlying Behavior

The basic goal of searching at the genetic level for correlations with behavior is to uncover the genes that "cause" the behavior. In other words, scientists can use a reductionistic approach to move from the organismic to the molecular and reveal the pathways by which a behavior is regulated. Generally, the first step is to identify candidate genes that are hypothesized to regulate the behavior. QTLs are revisited here because they can be used to develop candidate gene hypotheses. Other candidate gene approaches include microarrays (see page 99) of expressed sequence tags. Once candidate genes have been identified, experiments can determine if, indeed, that gene has a regulatory function for the behavior. Experimental studies establish the role of these genes in actually regulating phenotype.

BRUTE-FORCE APPROACHES TO FINDING CANDIDATE GENES

In some animals, such as fruit flies (*Drosophila melanogaster*), the roundworm (*Caenorhabditis elegans*), and house mice (*Mus musculus*), so much is known about the species' genetics that powerful mutagens, such as certain chemicals or radiation, can be applied to laboratory populations of the animal, and then animals of the next generation can be tested for "interesting" behavioral abnormalities. This technique has been used, for example, to find fruit flies whose biological clocks did not work properly. Once these mutants have been identified, backcrosses with "normal" laboratory strains can be used to map the location of the gene that causes the abnormality. This type of work takes many hours of concentrated laboratory effort because thousands of animals must be mated and tested, but this remains a powerful tool for identifying genes that code for proteins or regulatory elements that affect specific behaviors.

DISCUSSION POINT: CAUSE AND CORRELATION IN BEHAVIORAL GENETICS

We pointed out at the beginning of this chapter that single-gene mutants can give misleading information about genetic regulation of a behavior. A standard technique for producing mutants involves treating animals with either radiation or a chemical mutagen and then screening their offspring for behavioral abnormalities. Genetic investigation can then pinpoint the mutant gene that correlates with the abnormality. Is this proof that the gene "controls" the behavior? Why or why not?

FROM QTLS TO ASSESSING GENE EXPRESSION AND BEHAVIOR

For organisms with known linkage maps (e.g., nematodes, fruit flies, honeybees, mice,[41] humans), QTL analyses have provided intriguing windows into how variation in a small set of genes can explain much of the phenotypic variation in behavioral traits.[42] QTL analyses then open the door to identification of specific genes that are involved with a trait, and move the discussion from whole-organism approaches to looking at proximate causes at the level of the gene. Molecular techniques can pinpoint gene candidates for control of behavior. The exact location on the chromosome of a QTL location is not precisely known; the data show only that it is between two markers. If the distance between the two markers is not too large, molecular geneticists can overcome this problem by sequencing the chromosome from the two markers ("upstream" from one, "downstream" from the other). By comparing the DNA sequences with sequences of known genes, the scientist may determine the identity of the genes between the markers. The scientist can then form a hypothesis about which of the genes in the QTL region has the behavioral effect; this is a candidate gene hypothesis.

FIGURE 3.18
Junglefowl, the wild ancestor of domestic chickens.

A recent study of chickens[43] illustrates how interesting candidate genes are identified. Chickens are the domesticated descendants of junglefowl, a Southeast Asian bird species. Junglefowl (see Figure 3.18) are wilier and less manageable than their domestic relatives, in part because they are more aggressive. In this study, the investigators hypothesized a relationship between genes for growth, identified through QTL analyses, and domestication.

The investigators measured dominance and inspection of strangers (birds new to the social group). Differences among birds in these behaviors suggest that the arginine vasopressin receptor 1a (AVPR1a) gene may be involved in these social behaviors. Thus, AVPR1a is a candidate gene for social behavior in chickens. This is a fascinating discovery because vasopressin is the same hormone discussed in Chapter 2, Part II as

being important in pair-bonding and lifetime mating in voles. To verify the function of vasopressin in chicken social behavior, the investigators will now have to perform experiments directly addressing the effects of vasopressin on the behavior, but this finding is certainly highly suggestive. The candidate gene approach is only now coming into its own, in terms of being useful in behavioral investigations, but it holds extremely high promise in drawing links between genes and behavior and in allowing scientists to see similarities in behavioral regulation among species.

MICROARRAYS AND ASSESSMENTS OF GENE EXPRESSION

Are there are different patterns of gene expression between cells of animals with different behaviors? This question can be answered by looking at expressed sequence tags (ESTs). An EST is a tag based on a known functional gene sequence from an animal. An EST can be amplified—reproduced many times—and then be placed as part of a microarray (see Figure 3.19). A microarray holds each amplified EST in a small well, like an indentation on a microscope slide. A sample of tissue from an animal is then stimulated to produce proteins, which are labeled fluorescently. If a protein matches an EST, it binds, and the well becomes a fluorescent dot in the microarray, as shown in Figure 3.19. ESTs can then be compared between tissues, between animals, or between species. In the simplest analyses, the number of ESTs fluorescing is compared—for example, between sleeping and awake animals. But if details about the ESTs and which genes they derive from are known, this analysis can also provide clues about specific genes, leading to candidate gene hypotheses.

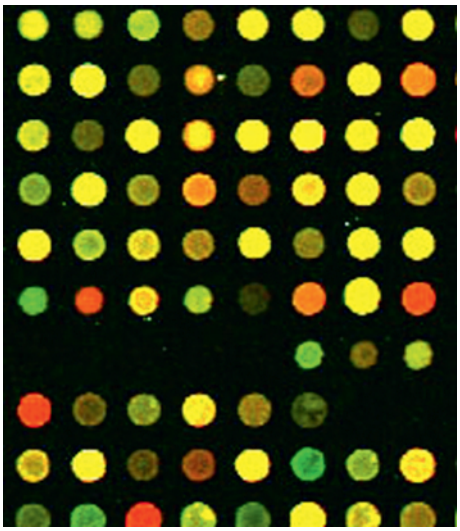

FIGURE 3.19
An EST microarray, showing strong expression of some ESTs and little or no expression of others.

RNA KNOCKOUTS

RNA knockouts are a relatively new tool in the exploration of genes and behavior. This technique involves synthesizing oligonucleotides that are complementary to the RNA products of genes thought to influence a behavior. When the synthetic oligonucleotide is introduced into the organism, it binds with the RNA from the target gene, effectively inactivating (or knocking out) that gene. Key to this approach is having a candidate gene with a hypothetical role in regulating a behavior.

SUMMARY

Behavioral genetics forms a critical component of nearly all behavioral studies. Understanding genetics establishes a thread that runs from the ultimate evolutionary causes of behavior to the behavior's proximate underpinnings, and knowing the genetic underpinnings of behavior is essential to understanding how behavior evolves. Behavioral genetics also helps in understanding the physiological and neurobiological systems that control behavior. While the approach of this chapter is genetics, most behavior is shaped by a combination of genetic and environmental factors. This chapter and the following two chapters—Chapter 4 on behavioral homeostasis and Chapter 5 on learning—work together to shape a picture of how behavior is controlled.

To understand the evolutionary roots of behavior (ultimate causes), behavioral geneticists often employ phylogeny. Genetics brings a broad range of tools to behavioral investigations. These tools are used to establish the phylogeny of animal groups, which can then be used to understand patterns of evolution for specific behaviors, such as nest construction by birds or colony defense by bees.

Peeling away the layers of genetic and physiological regulation of a behavior using behavioral genetics—starting with differences among species and moving down to the regulation of

gene expression—holds great promise for solving many of the mysteries of animal behavior. Single-gene effects on behavior are easily documented, but single-gene effects are relatively rare and may produce the erroneous notion that complex behavior is "controlled" by those genes. In fact, behavior is most often the result of a large number of genes acting together in a regulatory system. Quantitative genetics can be used to provide better explanations than single-gene models for most animal behavior traits. Studying heritability helps to unravel genetic and environmental influences on behavior. Molecular approaches to behavior genetics provide useful techniques in exploring the regulation of behavior.

Behavioral genetics is less a scientific discipline than a set of approaches to be applied to almost any behavioral question. Mastering the impressive array of behavioral genetic tools takes considerable work, but the reward is an ability to understand the relationship between ultimate and proximate causes, and to see the promise experimental genetics holds for unraveling complex scientific problems.

STUDY QUESTIONS

1. Food preference in a species of mouse has both innate (genetic) and experiential (learned) components. Design experiments that will test the following three hypotheses: (A) Learned information is used in preference to genetic information, when both are available; (B) there is a critical period for learning food preferences; and (C) the ability to learn food preferences has a high heritability. Make sure that the experimental designs include specification of sample sizes and controls.
2. The heritability of dominance behavior among males of a species of monkey is high (greater than 80%). Does this observation support a prediction that females use male dominance status in their choice of mates? Why or why not?
3. One of the central issues in behavioral genetics is how animals balance the use of genetically based and learned information. Under what circumstances might selection favor the use of inherited information? What circumstances favor the use of learned information? What are the general principles that determine the relative importance of learned and inherited information in shaping animal behavior? Remember to revisit this question after reading Chapter 5 on learning.
4. Without looking back in this chapter, define *heritability* and *additive genetic variation*. Why might additive genetic variation be more informative than total genetic variation? How does natural selection act on additive genetic variation?
5. If the heritability of a trait is high (close to 1.0), does this support a hypothesis that past selection on that trait has been high or low? Why?
6. Explain the candidate gene approach to discovering the regulatory pathways for a behavior. What are the advantages and drawbacks to making hypotheses about candidate genes?
7. How are microarray analyses useful in behavioral genetics?

Further Reading

Anholt, R.R.H., Mackay, T.F.C., 2009. Principles of Behavioral Genetics. Academic Press, San Diego, 334 pp.

Morris, D., 1969. The Naked Ape. Dell Publishing Co., New York.

Plomin, R., DeFries, J.C., McClearn, G.E., McGuffin, P., 2008. Behavioral Genetics. Worth, 560 pp.

Wilson, E.O., 1975. Sociobiology: The New Synthesis. Belknap Press of Harvard University Press, Cambridge, MA.

Notes

1. Tschirren, B., Bensch, S., 2010. Genetics of personalities: No simple answer for complex traits. Mol. Ecol. 19, 624–626.
2. Boake, C.R.B., Arnold, S.J., Breden, F., Meffert, L.M., Ritchie, M.G., Taylor, B.J., Wolf, J.B., Moore, A.J., 2002. Genetic tools for studying adaptation and the evolution of behavior. Am. Nat. 160 (Suppl. S.), S143–S159.

3. Ruttner, F., 1988. Biogeography and Taxonomy of Honeybees. Springer-Verlag, Berlin. Whitfield, C.W., Behura, S.K., Berlocher, S.H., Clark, A.G., Johnston, J.S., Sheppard, W.S., Smith, D.R., Suarez, A.V., Weaver, D., Tsutsui, N.D., 2006. Thrice out of Africa: Ancient and recent expansions of the honey bee. *Apis mellifera*. Science 314 (5799), 642–645.
4. Boake, C.R.B., Konigsberg, L., 1998. Inheritance of male courtship behavior, aggressive success, and body size in *Drosophila silvestris.* Evolution 52 (5), 1487–1492.
5. Cattaneo, E., Zuccato, C., Tartari, M., 2005. Normal huntingtin function: An alternative approach to Huntington's disease. Nat. Rev. Neurosci. 6 (12), 919–930.
6. Carhan, A., Allen, F., Armstrong, J.D., Goodwin, S.F., O'Dell, K.M.C., 2005. Female receptivity phenotype of icebox mutants caused by a mutation in the L1-type cell adhesion molecule neuroglian. Genes Brain and Behav. 4 (8), 449–465.
7. Crabbe, J.C., Phillips, T.J., 2003. Mother nature meets mother nurture. Nat. Neurosci. 6, 440–442.
8. Lehrman, D.S., 1953. A critique of Konrad Lorenz's theory of instinctive behavior. Q. Rev. Biol. 28, 337–363.
9. Rosenblatt, J.S. (1995). Daniel Sanford Lehrman: June 1, 1919–August 27, 1972. Biographies and Memories of the National Academy of Sciences 66, 227–245.
10. Shockley, W., 1992. Shockley on Eugenics and Race: The Application of Science to the Solution of Human Problems, preface by Jensen, A.R., Pearson, R. (Eds.), Scott-Townsend Publishers, Washington, D.C.
11. Wilson, E.O., 1975. Sociobiology: The New Synthesis. Belknap Press of Harvard University Press, Cambridge, MA.
12. Lewontin, R.C., Rose, S., Kamin, L.J., 1984. Not in our Genes: Biology, Ideology, and Human Nature. Pantheon Books, New York.
Kitcher, P., 1985. Vaulting Ambition: Sociobiology and the Quest for Human Nature. MIT Press, Cambridge, MA.
Hellman, H., 1998. Great Feuds in Science: Ten of the Liveliest Disputes Ever. Wiley, New York.
Gould, S. J., 1981. The Mismeasure of Man. W. W. Norton, New York.
13. Thornhill, R., Palmer, C.T., 2000. A Natural History of Rape: Biological Bases of Sexual Coercion. MIT Press, Cambridge, MA.
Thornhill, R., Palmer, C.T., Coyne, J.A., Berry, A., 2000. A natural history of rape: biological bases of sexual coercion. Nature 404, 121.
Ward, T., Siegert, R. 2002. Rape and evolutionary psychology: a critique of Thornhill and Palmer's theory. Aggress. Violent Beh. 7, 145–168.
14. Wilsson, E., Sundgren, P.E., 1997. The use of a behaviour test for selection of dogs for service and breeding. Heritability for tested parameters and effect of selection based on service dog characteristics. Appl. Anim. Behav. Sci. 54 (2–3), 235–241.
15. Hare, B., Brown, M., Williamson, C., Tomasello, M., 2002. The domestication of social cognition in dogs. Science 298 (5598), 1634–1636.
Koler-Matznick, J., 2002. The origin of the dog revisited. Anthrozoos 15 (2), 98–118.
16. Congdon, B., Platt, J.F., Martin, K., Friesen, V.L., 2000. Mechanisms of population differentiation in marbled murrelets: Historical versus contemporary processes. Evolution 54, 974–986.
17. Ossi, K., Kamilar, J.M., 2006. Environmental and phylogenetic correlates of Eulemur behavior and ecology (Primates: Lemuridae). Behav. Ecol. Sociobiol. 61 (1), 53–64.
18. Flint, J., 2003. Analysis of quantitative trait loci that influence animal behavior. J. Neurobiol. 54 (1), 46–77.
19. Deater-Deckard, K., Petrill, S.A., Thompson, L.A., 2007. Anger/frustration, task persistence, and conduct problems in childhood: A behavioral genetic analysis. J. Child Psychol. Psyc. 48 (1), 80–87.
20. Lee, P.L., Clayton, D.H., Griffiths, R., Page, R.D., 1996. Does behavior reflect phylogeny in swiftlets (Aves: Apodidae)? A test using cytochrome b mitochondrial DNA sequences. PNAS 93, 7091–7096.
21. Brady, S.G., Sipes, S., Pearson, A., Danforth, B.N., 2006. Recent and simultaneous origins of eusociality in halictid bees. Proc. R. Soc. B-Biol. Sci. 273 (1594), 1643–1649.
22. Johannessen, L.E., Slagsvold, T., Hansen, B.T., 2006. Effects of social rearing conditions on song structure and repertoire size: experimental evidence from the field. Anim. Behav. 72, 83–95.
23. Airey, D.C., Castillo-Juarez, H., Casella, G., Pollak, E.J., DeVoogd, T.J., 2000. Variation in the volume of zebra finch song control nuclei is heritable: Developmental and evolutionary implications. Proc. R. Soc. Lon., B-Biol. Sci., 267, 2099–2104.
24. Zhang,Y., Lawrance, S.K., Ryder, O.A., Zhang, Y., Isaza, R., 2000. Identification of monozygotic twin chimpanzees by microsatellite analysis. Am. J. Primatol. 52, 101–106.
25. Ross, K.G., Krieger, M.J.B., Shoemaker, D.D., 2003. Alternative genetic foundations for a key social polymorphism in fire ants. Genetics 165 (4), 1853–1867.
Ross, K.G., Vargo, E.L., Keller, L., 1996. Social evolution in a new environment: The case of introduced fire ants. Proc. Natl. Acad. Sci. U.S.A. 93, 3021–3025.
26. Suarez, A.V., Tsutsui, N.D., Holway, D.A., Case, T.J., 1999. Behavioral and genetic differentiation between native and introduced populations of the Argentine ant. Biol. Invasions 1, 43–53.
27. Yamamoto, D., Nakano, Y., 1999. Sexual behavior mutants revisited: Molecular and cellular basis of *Drosophila* mating. Cell. Mol. Life Sci. 56, 634–646.
28. Ruefenacht, S., Gebhardt-Henrich, S., Miyake, T., Gaillard, C., 2002. A behaviour test on German Shepherd dogs: Heritability of seven different traits. Appl. Anim. Behav. Sci. 79 (2), 113–132.

29. Meffert, L.M., Hicks, S.K., Regan, J.L., 2002. Nonadditive genetic effects in animal behavior. Am. Nat. 160 (Suppl. S DEC), S198–S213.
30. Ruefenacht, S., Gebhardt-Henrich, S., Miyake, T., Gaillard, C., 2002. A behaviour test on German Shepherd dogs: Heritability of seven different traits. Appl. Anim. Behav. Sci. 79 (2), 113–132.
31. Swallow, J.G., Carter, P.A., Garland Jr., T., 2004. Artificial selection for increased wheel-running behavior in house mice. Behav. Genet. 28, 227–237.
32. Cuervo, J., de Lope, F., Moller, A., 1996. The function of long tails in female barn swallows (*Hirundo rustica*): An experimental study. Behav. Ecol. 7, 132–136.
33. Nol, E., Cheng, K., Nichols, C., 1996. Heritability and phenotypic correlations of behaviour and dominance rank of Japanese quail. Anim. Behav. 52, 813–820.
34. Gleason, J.M., Nuzhdin, S.V., Ritchie, M.G., 2002. Quantitative trait loci affecting a courtship signal in *Drosophila melanogaster*. Heredity 89, 1–6.
35. Ruppell, O., Pankiw, T., Page, R.E., 2004. Pleiotropy, epistasis and new QTL: The genetic architecture of honey bee foraging behavior. J. Hered. 95 (6), 481–491.
36. Netto, W.J., Planta, D.J.U., 1997. Behavioural testing for aggression in the domestic dog. Appl. Anim. Behav. Sci. 52 (3–4), 243–263.
37. Guzman-Novoa, E., Hunt, G.J., Uribe, J.L., Smith, C., Arechavaleta-Velasco, M.E., 2002. Confirmation of QTL effects and evidence of genetic dominance of honeybee defensive behavior: Results of colony and individual behavioral assays. Behav. Genet. 32 (2), 95–102.
38. Hunt, G.J., Collins, A.M., Rivera, R., Page, R.E., Guzman-Novoa, E., 1999. Quantitative trait loci influencing honeybee alarm pheromone levels. J. Hered. 90 (5), 585–589.
39. Hunt, G.J., Guzmán-Novoa, E., Fondrk, M.K., Page Jr., R.E., 1998. Quantitative trait loci for honey bee stinging behavior and body size. Genetics 148, 1203–1213.
40. Dodman, N.H., Karlson, E.K., Moon-Fanelli, A., Galdzicka, M., Perloski, M., Shuster, L., Lindblad-Toh, K., Ginns, E.I., 2010. A canine chromosome 7 locus confers compulsive disorder susceptibility. Mol. Psychiatry 15, 8–10.
41. Gershenfeld, H.K., Neumann, P.E., Mathis, C., Crawley, J.N., Li, X.H., Paul, S.M., 1997. Mapping quantitative trait loci for open-field behavior in mice. Behav. Genet. 27 (3), 201–210.
42. Jordan, K.W., Morgan, T.J., Mackay, T.F.C., 2006. Quantitative trait loci for locomotor behavior in *Drosophila melanogaster*. Genetics 174 (1), 271–284.
43. Wiren, A., Gunnarsson, U., Andersson, L., Jensen, P., 2009. Domestication-related genetic effects on social behavior in chickens—Effects of genotype at a major growth quantitative trait locus. Poult. Sci. 88, 1162–1166.