Chapter 2: Genetic Causes of Behavior
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Introduction to Genetic Terms and Mendelian Traits

When we search for the proximate causes of behaviors, we naturally start with genes. When the fusion of sperm and egg produces a zygote, the stage is set for a cascade of developmental events that lead to the production of the phenotype. The central dogma of biology holds that DNA from alleles at a genetic locus is transcribed into RNA that is then translated into proteins at the ribosomes. Structural proteins are used to contract and thereby move cells. Enzymes run the cellular machinery. Cells in the developing zygote interact mechanically or chemically with adjacent cells. Cells produce "messengers" that facilitate the cell-cell communication at distance. Cell-cell and organ-mediated interactions cause a cascade of events that we call development. The embryo is gradually organized into a functioning animal with nerves and organ systems that begin to regulate behaviors. Those behaviors have functional or selective consequences. Individuals live, breed and die according to their phenotype, and underlying genotype. Gene frequencies change across generation and the species evolves.

The goal for this chapter is to develop an understanding of the genetic factors that govern the expression of morphological traits that are associated with behaviors. Techniques of classical genetics such as genetic crosses and pedigree analyses are now being combined with tools of molecular biology to identify specific genes that appear to govern the expression of complex behaviors. Before we begin this quest, we need to understand a few of the terms of genetics (distilled from Keller and Lloyd, 1992). In particular, we focus on those genetic processes that deal with genetic variation within and among individuals.

The Genotype and Phenotype

Genotype -- the sum total of all the alleles at all the loci in an organism. While precise and concise, this definition is not very useful. The genotype may also apply to genetic material at a single genetic locus and to describe the alleles that an individual possesses at a particular locus. The two alleles at a genetic locus come from the male and female parents and form the basic unit of genetic variation in an individual. The process of meiosis, Mendelian segregation, and recombination among all of the of genetic loci in each individual effectively makes each sexually produced individual unique (see Side Box 2.1. Mutation,

Segregation and Recombination). In contrast, an asexual organism is a genetic clone of its female parent. There are even mixtures of both asexual and sexual reproduction in the animal kingdom referred to as hybridogenetic mating systems, which we will treat in more detail in an upcoming chapter (4).

The genotype is **largely static** for an individual during its lifetime, except when a **mutation** occurs. A mutation is a lesion of DNA that changes the genetic material in one allele at a locus. If the mutation occurs in a somatic cell, there will be no consequence for transmission of genetic material across generations. However, if the mutation occurs in a cell of the germ line, which produces sperm or eggs for sexual reproduction, then the mutation can be transmitted across generations. Mutations are the ultimate source of all genetic variation.

Phenotype – The phenotype is the external expression of the genes, and the result of a gene's interaction with the environment. The expression of the phenotype includes mechanisms of development. Because the stage of development depends on age, **the phenotype can be highly labile**, or can change dramatically during its lifetime. The organism develops, it learns, it acclimates and its phenotype changes accordingly. For simplicity of analysis, we break the whole organism into **phenotypic traits** that are largely functional units. For example, the number of offspring that an animal produces in one bout of reproduction, termed fecundity, is a phenotypic trait related to reproduction. Likewise, the kind of parental care is a different phenotypic trait related to reproduction. However, phenotypic traits are often **correlated** with other phenotypic traits and such correlations arise from proximate mechanisms. For example, the number of offspring that a parent produces is related to the kind or quality of parental care that the parent can provide to the offspring. Usually we refer to such relationships as **fitness trade-offs** because an increase in one fitness trait (fecundity) has an impact on a correlated trait (quality of care). The source of fitness trade-offs is covered in Chapter 3. We can refer to these phenotypic correlations as genetic correlations, when the source of the coupling has some genetic basis. The proximate mechanisms that link traits used in the example of parental care and offspring number are related to energy and metabolism. Rearing large numbers of offspring requires more total energy to keep the level of care constant for each offspring. Alternatively, less energy (or lower quality care) is available for each offspring.

Environment -- the environment is anything external to the genetic material. For example, food availability in part determines body weight. In animals that lay eggs without parental care, the eggs are subjected to environmental variation in the form of temperature, hydration, or perhaps salinity. In animals that lay eggs and have extended parental care in a nest, the environment is a function of the parents and perhaps the other sib-mates in the nest. In animals with internal fertilization and some kind of gestation, the mother's physiology *per se* becomes an important component of the offspring's environment. In the case of a mother's environment, we refer to such influences as maternal effects. While we consider maternal effects in this chapter, an entire Chapter (16) is devoted to maternal (and paternal) effects on behavior. Regardless of whether or not an organism requires extended parental care as a juvenile, the environment still plays a major role in an individual's development. Of course, there is a second sense in which the environment interacts with the genotype and phenotype: the environment causes natural selection. While this natural selection acts on phenotypes within a generation (e.g., the parents, its effects are transmitted to the next generation (e.g., offspring) (treated in Chapter 3).

A simple expression describes the relationship between variation in a given phenotypic trait among individuals found in a single population:

$$\mathbf{P} = \mathbf{G} + \mathbf{E}, \qquad \textit{(Eqn 2.1)}$$

where **P**, **G**, and **E** are phenotypic, genotypic, and environmental variation in the trait. If the environmental variation is large, then little phenotypic variation arises due to the genetic sources of variation. Conversely, if genetic variation is large, then the phenotypic trait is largely determined by genetic factors. As we have seen in Chapter 1, *heritable variation* of some kind is central to Darwin's theory of natural selection, and evolutionary change will occur more rapidly for traits that are strongly determined by genetic factors. In this chapter, we will find out what kind of genetic variation is necessary for natural selection.

Side Box 2.1: Mutation, Segregation, and Recombination

The process of natural selection is often characterized as "blind" and this is largely because the source of all genetic variation is largely a stochastic process. The ultimate source of genetic variation is mutations; however, segregation, and recombination provide stochastic mechanisms for randomizing genetic variation in a population. While mutations alter DNA by changing base pairs, segregation and recombination do not alter the material content of DNA. Instead they provide powerful mechanisms for mixing up the DNA during sexual reproduction.

Mutation

The process of mutation is probabilistic. We describe this process in terms of the probability of a mutation arising in an individual during its lifetime, expressed on a per gene rate. Typical rates of mutation are between 1 in 10,000 (10^{-4}) and 1 in 1,000,000 (10^{-6}) for many organisms. Either this means that a long time must pass before a mutation occurs in a population, or the population must be very large. The population size must be in excess of 10^6 or one million members in order to see an average of one mutation per generation for a gene with a mutation rate of 10^{-6}. Most mutations are detrimental, and perhaps only 1 in 1,000 is beneficial. Thus, in this population of 1,000,000 we might have to wait for 1,000 years for a specific genetic locus to throw us a beneficial mutation. There are thousands of possible loci in an organism, approximately 30,000 loci in humans, so the waiting time for a beneficial mutation at any locus in the genome is less. Even if a mutation arises, there is no guarantee that natural selection will act on it.

The first random event that determines if a mutation will be acted upon by selection is meiosis. We can calculate the probability that a mutation makes it through meiosis and segregation. Assume a mutation occurs in the germ line of a diploid parent, which has 2 gene copies. The beneficial mutation has a 50% chance of being passed on to offspring and a 50% chance of not being passed on. With an organism that has 4 progeny, the probability that a single offspring will not get the beneficial mutation is **independent** of the other offspring receiving or not receiving the mutant allele. By using the laws of probability we can multiply successive independent events to compute the probability that none of the 4 progeny gets the beneficial mutation:

$$1/2 \times 1/2 \times 1/2 \times 1/2 = 1/16.$$

If the beneficial mutation is not passed on to the progeny, when it arises for the first time in a parent, then more time will be required for a new beneficial mutation to arise in the population. Only when it gets transmitted does selection have a chance of promoting its spread in the population.

Segregation

Genes are found on chromosomes. The first random event in sexual reproduction occurs during meiosis when homologous pairs of chromosomes line up on the equatorial plate of the cell in preparation for the cell division that reduces a diploid (2N) cell of the germ line to an haploid (N) gamete. During the process of segregation different homologues will randomly be distributed between the daughter cells. Gametes end up with very different chromosome complements. In sexual organisms, one chromosome in a pair is from the mother, the other is from the father. Given that the total number of chromosomes is C, then there are 2 (two types of chromosomes) raised to the power of C different gametes that could be produced from segregation of chromosomes (in humans that would be 2^{46}). Segregation can produce a vast number of different gametes.

Recombination

Recombination between homologous pairs of chromosomes during meiosis can generate even more gamete types. The chromosome is loaded with recombinational hotspots. Any given chromosome will typically recombine with its homologue at one or two points during a given meiotic event. The recombination rate on a given chromosome is a function of chromosome length. Short chromosomes recombine rarely. Long chromosomes recombine a lot. However, the recombination points for two different meiotic events can be different. Given that gametes such as sperm are generated by millions of different meiotic events, the number of potential gamete types from a single parent is vast. If one considers all possible parents in a modest-sized breeding population such as humans (5 billion), there are easily more potential recombination products than molecules in the known universe.

Components of Phenotypic and Genetic Variation

We can follow a roadmap from genotype to phenotype, but the route is rarely a one-to-one mapping between genes and phenotypic traits. Environment further obscures the relationship between genotype and phenotype. We have several hierarchical concepts that describe how genotype can be related to phenotypic traits. These additional terms describe different levels of genetic interaction and their effect on genotype and phenotype (the terms in brackets will be described below):

1. the effect of one allele on another allele at the same genetic locus (i.e., additive effect *versus* dominance – see Side Box 2.2),

2. the effect of one genetic locus on another locus (e.g., polygenic and epistasis),

3. the effect of a single gene or two or more phenotypic traits (e.g., pleiotropy),

4. the effect of two or more genes on a single phenotypic trait (e.g., additive effect *versus* epistasis),

5. the interaction between genetic factors and the environment (e.g, norm of reaction).

Pleiotropy -- a single gene that has an effect on the expression of two or more phenotypic traits is said to have a pleiotropic effect on the traits. For example, testosterone controls the development of what are referred to as **secondary sexual characteristics** (e.g., a male lion's mane), but testosterone also relates to behavioral traits like aggression. Thus, a gene that controls the levels of testosterone would have a pleiotropic effect on the expression of many morphological secondary sexual traits as well as many behavioral traits such as aggression. The concept of pleiotropy is intimately related to the concept of **trade-off** (Stearns 1976). Pleiotropy describes the proximate genetic source for many phenotypic trade-offs. If one gene controls the expression of two or more traits and those traits are related to a fitness trade-off, then we have identified the proximate source of the trade-off.

Polygenic -- if two or more genes are responsible for a single trait, the phenotypic trait is governed by polygenic factors (*poly* – many, genes). For example, growth rate is undoubtedly caused by a number of genes that act in a complex cascade. Body size, which is the result of a large number of genes, is polygenically determined. Genes that control growth hormone have a large effect on body size. Likewise, genes that control sex steroids like testosterone have some effect on body size, especially during maturation phases of growth. Undoubtedly, many other genes that influence metabolism have small, but measurable effects on size.

Additive effects within and between loci. The simplest additive genetic relationship occurs between two alleles at the same genetic locus. If two alleles are co-dominant, then the heterozygote is exactly intermediate in phenotype relative to the two homozygous types. In this idealized case, "the effect of substituting one allele for another is additive in its effect on phenotype". Just like alleles acting in an additive fashion at a single locus, polygenic loci can also interact in an additive fashion to produce a phenotype. If two or more genes have a simple effect on the phenotype they are generally thought of as having an additive effect. If a trait results from two or more genes then an additive relationship between them would lead to the simplest kind of polygenic inheritance. **Additive genetic variance** is what underlies the notion of heritability and is responsible for the similarity between parents and offspring. *Directional natural selection operates on additive genetic variation because additive genetic variation is largely responsible for the resemblance or heritability between parents and offspring* (see Side Box 2.2: Additive Genetic versus Dominance Variation).

Dominance. Interactions between **alleles** at a single locus are termed dominance interactions. For example, if an allele is recessive to another allele, then an individual that possesses a copy of the **dominant** allele and a copy of the **recessive** allele (e.g., **heterozygote**) will be phenotypically identical to an individual that possesses two copies of the dominant allele (e.g., **homozygous**). The "**recessive phenotype**" is only expressed if the individual is homozygous for two of the recessive alleles. The *rover* and *sitter* genotypes of *Drosophila* larvae, discussed

below, are a classic example of the effect of a dominant gene on behavior. If two alleles are co-dominant, then the heterozygote is intermediate between the two homozygous genotypes and the alleles are additive in their effect. While dominance variation can be a very large component of the total genetic variation of a phenotypic trait, dominance variation is entirely non-additive (see Side Box 2.2).

Epistasis. If two different genetic loci interact in any way that is **non-additive**, then they are said to act epistatically. Many pigmentation genes act in an epistatic fashion. Pigmentation genes form an extremely important component of animal signals, which are used in communication (Chapter 13). The simplest example of epistasis relates to genes that control pigmentation. This example illustrates how a dominance interaction between alleles at the same locus is conceptually similar to an epistatic interaction between two genetic loci. Consider a single locus such as the coat color of a popular breed of dog the Labrador retriever. The color yellow (*y*) is recessive to black (*b*). One copy of the black allele and the production of the black pigment overwhelms the expression of the yellow-coated phenotype and a heterozygous *yb* individual is black. The effect of the black pigment is non-additive, in that one copy completely masks the recessive yellow. A second coat color locus codes for production of brown pigment. The brown locus interacts in a non-additive fashion in that an individual with an allele at the brown locus can be brown regardless what pigment is expressed at the black locus. The brown locus overwhelms expression of the black locus in a non-additive fashion. In this case, brown interacts epistatically with the black locus. One hypothesis is that the gene for brown acts upstream of the black locus, short circuiting its action.

Genotype and Environment Interaction and Phenotypic plasticity. If the expression of a gene depends on the environment in any way, then the phenotype is said to be due to an interaction between the genotype and environment. This idea is central to behavior and we will explore it in great detail in upcoming chapters. For example, birds undoubtedly have genes for learning, and indeed some species of birds may differ in how they learn. A famous example of genotype environment interactions relates to how birds learned to open milk bottles in England. For years, milkmen left milk bottles on the porch with no problems. One day a single bird learned how to open the milk bottle top and drink milk. This trait was passed on to other birds by learning. Even different species of birds learned the task. The environment changed with the advent of a single bird that learned to open milk bottles. This teacher changed the environment. However, only some species of birds learned how to open milk bottles. Learning was presumably contingent on both the environment and on the genes for learning in each species. Both the presence and absence of milk bottles, and presence or absence of a tutor bird to teach naïve birds was necessary for learning this task.

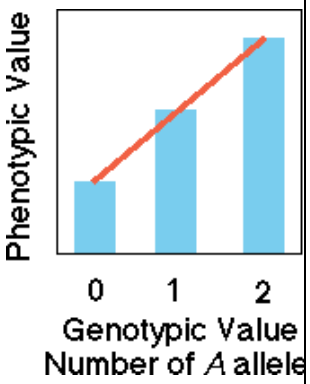
Phenotypic plasticity -- the expression of different phenotypes within a constant genetic background. There may be no genetic differences among individuals, but an environmental cue may cause development to proceed along different pathways. From a single genotype, many different phenotypes can be produced due to environmental differences.

Mendelian Traits

We will explore many of these issues of genetics through examples. I have chosen a diverse set of behavioral traits to introduce many concepts in animal behavior, which will be covered in greater detail in later chapters. Each example, illustrates one of the genetic terms discussed above. In many cases model laboratory organisms facilitate our study of the proximate causes of behavior because a large sample size is necessary to generate the statistical power that is required to assess the genetic bases of behavioral traits. Other genetic examples are drawn from natural populations. While it is more difficult to collect genetic data on animals in the wild, these animals provide answers to natural selection because we can follow them through their lives. Moreover, it is essential to study animals in the wild so that we can infer how their behavioral actions and reactions are reflected in the process of natural or sexual selection. Through long-term observations of animals in the wild, we are able to reconstruct present day patterns of selection (Lande, 1983) that may have been historically significant during the origin of behavioral traits. A first step in selection analyses is determining the genetic basis of behavioral traits. Classic genetic crosses and pedigree analyses are the standard tools of a behavioral geneticist.

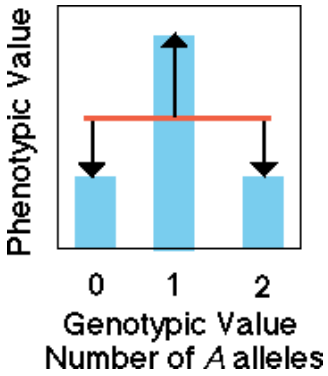
Side Box 2.2: Additive Genetic Variation versus Dominance Variation

What is the source of heritable variation? The resemblance between parents and offspring is entirely due to the additive effect that genes have on phenotype. Not all of the genetic variation expressed in an organism is additive. For example, dominance variation arises from the degree to which one allele at a locus alters the effect of the allele on the complementary chromosome. A useful way to think about additive genetic variation is to consider the proportion of the phenotype that can be predicted from a knowledge of the number of *A* versus *a* alleles that are present in the genotype -- or the genotypic value. Consider an *aa* individual to have zero *A* alleles, *Aa* has one *A* allele, and *AA* has two *A* alleles. Only in the idealized case when alleles at a genetic locus are co-dominant, are the genes said to act in a purely additive fashion. Dominance reduces the additive genetic variation, increasing the dominance variation at the same time.



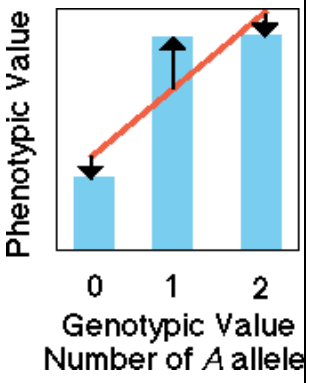
Co-dominant alleles

In the case of a co-dominant set of alleles, knowledge of genotype allows us to perfectly predict the value of the phenotype. A regression line (colored in red) of phenotypic value on the number of *A* alleles, or the genotypic value, yields a perfect predictive line. Adding one *A* allele from the baseline state of no *A* alleles (i.e., *aa* genotype), adds an increment to the phenotype (genotypic value 1 = *Aa*). The same increment is added when another *A* is added to create *AA*.



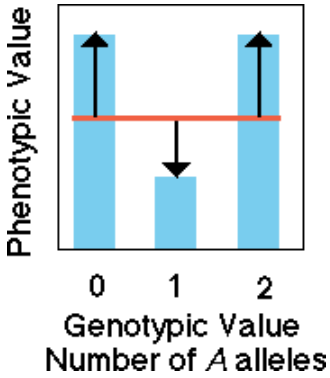
Overdominance

In the case of overdominance, knowledge of genotype does not allow us to confidently predict the value of the phenotype. There is no slope to the regression line so none of the variation is due to "additive genetic effects" of alleles. All variation in our model is due to error, which arises from dominance variation (black arrows). Consider two parents that are homozygous for alternative alleles (*aa* and *AA*). If behavior were strongly determined by an overdominant allele then the heterozygous progeny would not at all resemble the homozygous parents. The trait does not appear to be heritable.



Dominant and recessive alleles

In the case of a dominant *A* allele, knowledge of genotypic value allows us to only imperfectly predict the phenotypic value of an individual. Because heterozygotes and dominant homozygotes have the same phenotypic value, the fit between the regression (red line) of phenotypic value on genotypic value has error (black arrows). There is still a significant regression slope showing that some of the variation is due to additive genetic causes (red line) and some is due to dominance variation (black arrows).



Underdominance

In the case of underdominance, knowledge of genotype does not allow us to confidently predict the value of the phenotype. There is again no slope to the regression line so none of the variation is due to "additive genetic effects" of alleles. All variation in our model is due to error, which arises from dominance variation (black arrows). Consider two parents that are homozygous for alternative alleles (*aa* and *AA*). If behavior were strongly determined by an underdominant allele then the heterozygous progeny would not at all resemble the homozygous parents.

Behavioral Mutants in the Laboratory: *Roving* versus *Sitting* in *Drosophila* larvae

Many of the basic issues of Mendelian inheritance of behaviors are exemplified in the example of *rover* versus *sitter* larvae in *Drosophila*. Marie Sokolowski and her colleagues (Sokolowski 1985; de Belle and Sokolowski 1987) generated two strains of larvae and carried out some illuminating crosses. If you cross a pure *rover* type to a *rover* or a pure *sitter* type to a *sitter*, each of the ensuing strains is pure and homozygous for either the *rover* or *sitter* allele. Crossing *between* the pure parental lines (P0) produces progeny, which are referred to as the F1. In theory, if the phenotype rests entirely on a single gene with two alternative alleles (a dominant *rover* allele and a recessive *sitter* allele). The F1 cross should produce all *rovers*, no *sitters*. In practice, this cross does produce all *rovers*, suggesting that *sitter* is recessive to *rover* (or *rover* is dominant to *sitter*). The telling bit of evidence arises from a cross between F1 progeny, which should recover all 3 genotypes (see Punnett Square in Table 2.1.A), but only two phenotypes (see Table 2.1.B), owing to dominance of the *R* allele over the *S* allele. The offspring from this cross are *rovers* and *sitters* in nearly a 3:1 ratio -- exactly the pattern one expects if the F1 are heterozygous for *rover* and *sitter* alleles.

Table 2.1. A) Theoretical Results from F1 X F1 cross		**genotype of first parent**			**Table 2.1.B) Observed Phenotype in F2.**	**Hypothesized genotype**	**Predicted Ratio**
		R	*S*		**Rover**	*R*/*R*	1
genotye of	*R*	*R*/*R*	*R*/*S*		**Rover**	*R*/*S*	2
second parent	*S*	*R*/*S*	*S*/*S*		**Sitter**	*S*/*S*	1

Beak Size and Seed Preference in African Finches

Like the behavioral morphs of larval fruit flies, species of an African finch that inhabits Cameroon, *Pyrenestes ostrinus*, exhibits a discrete set of bill morphs with a simple Mendelian pattern of inheritance (Smith, 1993). In particular, a small-beaked (s) and large-beaked (L) form are syntopic and live in the same habitat, throughout their geographic range. The small- and large-beaked forms are often found building nests together. These two forms differ dramatically in feeding behavior and their preference for large versus small seeds. In the wild, the small-billed morph has a very strong preference for the seeds from a species of sedge that produces small seeds. The large-billed morph prefers the seeds from a large-seeded sedge species. Given their propensity to feed on seeds, the bird's common name is the seed-cracker. Thomas Smith initiated breeding studies on the *P. ostrinus* to determine the genetic basis of the behavioral and morphological differences between bill morphs. High levels of nest predation in the wild precluded the possibility of obtaining pedigrees from the wild. Smith circumvented this problem by importing breeding pairs for his study in a collaborative effort with the Riverbanks Zoo in South Carolina. To study the inheritance of beak morphology, which is associated with feeding behaviors, he first had to get the animals to breed in the laboratory.

<< Figure 2.1 Finch photo and pedigree >>

Finding the environmental factor that triggers reproduction in non-domesticated animals is often a daunting task. Many temperate birds are triggered to initiate reproduction with a change from short photoperiods to long photoperiods. The early attempts by the Riverbank Zoo to trigger reproduction by manipulating the photoperiod met with failure. However some tropical Finch aficionados suggested that they try an unusual environmental trigger. In the wild, the finches can rely on a reliable cue to breed, they experience two dry seasons and two wet seasons every year. One of the wet seasons is a little damper than the other. The workers at the Zoo played audiotapes with loud thunder and simultaneously drenched the aviaries with water. The finches, happy with the beginning of a simulated wet season, began to breed. This example, illustrates some of the challenges of non-model systems, but also the creative solutions used to solve practical problems in science.

Side Box 2.3: Random Mating and Gene Frequencies

The Hardy-Weinberg Theorem describes what happens to gene frequencies when no evolutionary forces act on phenotypes. This assumes that no selection, migration, mutation, or genetic drift alters the gene frequencies from generation to generation. Another important assumption is that individuals in the population breed randomly with respect to genotype and phenotype. In a randomly mating population gametes combine in proportion to the frequency of each gametic type taking part in the union. For a single locus with two alternative alleles, the *A* allele, which occurs at a frequency of *p*, and the *a* allele which occurs at frequency *q* (or 1-*p*), then the proportion of genotypes is given by multiplying the frequency of each gamete type:

Genotype	*AA*	*Aa*	*aA*	*aa*
frequency	$p \times p$	$p \times q$	$q \times p$	$q \times q$

or, summing up all genotypes, the more familiar: $p^2 + 2 \times p \times q + q^2 = 1$. These calculations describe how gametes pair up randomly during fertilization. Another expression of random mating occurs at the level of the phenotype. If the frequency of each phenotype is: p^2, $2pq$, and q^2, and phenotypes pair up randomly we would expect to see the following patterns of random mating. To simplify it further $S = p^2$ and $L=2pq+q^2$.

Random Mating Finches

Smith (1987) observed the following frequencies of mating in *Pyrenestes ostrinus*: $S_{males} \times S_{females} = 34$, $S_m \times L_f = 14$, $L_m \times S_f = 14$, $L_m \times L_f = 6$. A few simple computations are required to assess how the birds are breeding. The birds may mate **randomly**, **assortatively** by phenotype (like breeds with like) or **disassortatively** by type (birds seek out a more dissimilar partner). While we cannot distinguish between all the genotypic classes in *P. ostrinus* ($S = p^2$ and $L=2pq+q^2$), we can ask whether the phenotypes are breeding randomly. What frequency of mating would we expect by chance? We need to compute the frequencies of each genotype by sex.

freq small-billed males	(34+14)/(34+14+14+6) = 48/68 = **0.71**
freq of small-billed females	(34+14)/(34+14+14+6) = 48/68 = **0.71**
freq of large-billed males	(14+6)/(34+14+14+6) = 20/68 = **0.29**
freq of large-billed females	(14+6)/(34+14+14+6) = 20/68 = **0.29**

What is the probability that a small-billed male pairs randomly with a small-billed female? The probability that a small breeds with small at random, $S \times S$, is given by multiplying the frequency of each type:

$S \times S = 0.71 \times 0.71 =$ **0.54** and, we expect a total of $68 \times 0.54 =$ **34.3**.

By the same logic, we can compute our random expectations for the other three kinds of crosses to derive an expected number of crosses if the birds were randomly mating. By inspection alone we can see that the observed and expected random frequencies are nearly identical. We could carry out a formal test, the Chi-square, which is based on observed versus expected frequencies. The χ^2 test is described in Appendix 1.

Observed Female of the Pair

(Expected)

		S	*L*
Male of	*S*	34 (34.3)	14 (14)
the Pair	*L*	14 (14)	6 (5.7)

Mendelian Inheritance of Three Alleles

Formulae for genotype frequency are only slightly more complicated for 3 alleles:

Genotype	αlα	βlβ	γlγ	αlβ	βlγ	αlγ
frequency	p × p	q × q	r × r	2× p× q	2 × q× r	2 × p× r

The two allele and multi-allele Hardy-Weinberg Law really only implies that gametes achieve union randomly with respect to genotype. Given the observed *Ams* gene frequencies in isopods (see text), what is the frequency of mating phenotypes expected under random mating?

Over the years, Smith and his colleagues developed a relatively large pedigree from crosses: L × L, L × s, s × s. The best-fit genetic model for the control of beak size based on the genetic data in the pedigree, is a single Mendelian locus with an allele for the small beaked form (s) recessive to an allele for the large beaked form (L). (see Side Box 2.3: Hardy-Weinberg Law and Random Mating). A simple Mendelian gene causes large differences in morphology in the seed crackers, and this has cascading effects on a suite of foraging behaviors. This illustrates that a single gene can influence a large number of morphological and behavioral traits. In Chapter 3, "Adaptation and Selection", I describe how the gene for beak morphology influences survival in the wild, and in Chapter 6, Optimal Foraging, how it influences energy acquisition.

Molecular Tools of Behavioral Geneticists

The search for the genetic factors in natural populations and laboratory stocks depend largely on the pre-existence of genetically-based morphs. Examples include the case of the pedigree analysis of crosses between bill morphs in finches or in the genetic screen for alternative behaviors carried out in lab stocks of *Drosophila*. Many human genetic disorders are uncovered by analyses of pedigrees that are obtained from isolated populations where the genetic trait occurs at a relatively high frequency. When we see a maladaptive genetic disorder at a high frequency, genetic drift is the probable cause. In small isolated populations, a single copy of a genetic disorder can drift to high frequency when inbreeding occurs. Inbreeding is much more likely in a small population where **consanguineous** mating, or mating between related individuals, occurs at high frequency versus those in a large population where encountering a relative is quite unlikely. Behavioral geneticists use this fact to isolate and maintain laboratory stocks of interesting behavioral mutants.

Genetic sleuths refine their search for genetic factors underlying human behaviors by screening key families in which an affliction is prevalent. If researchers find a perfect match between transmission of a certain piece of DNA and transmission of the trait, they can map where the gene coding for the behavior is located on a genetic map of the human genome. This **marker gene** sits next the gene of interest. Behavioral genes are being discovered in humans at a rapid rate using inference from pedigrees and the transmission of specific regions of DNA.

Another important technique for isolating genes that control behavior entails a mutant screen. First the parental generation in a colony of animals is subjected to a mutagen, after which the progeny in the colony are screened for behavioral disorders. The researchers then search for the genetic basis by molecular methods that are similar to those described above in the analysis of human pedigrees. The next series of examples illustrates how molecular methods aid in the determination of genetic factors for behaviors. **<< Side Box 2.4 Gene maps in prep>>**

Mutation Analysis of a Gene for Parental Care in Mice: *fosB*

Brown et al (1996) have recently identified a gene in mice, *fosB*, which is essential for the correct expression of maternal behaviors. By chance, they induced a mutation in a single gene, *fosB*, that when its function was disabled appeared to extinguish nurturing behaviors in female mice. Evidence from their study suggests that a very simple neural pathway may be involved. Lesion experiments have shown that the hypothalmus in female mice is critical for nurturing behavior. By deleting a gene whose gene product is expressed in the hypothalmus, Brown et al (1996) have isolated a key genetic factor involved in nurturing. It appears that the gene products of *fosB* are expressed in the small part of the hypothalmus called the preoptic area of the brain. *Fos B* deficient mothers do not exhibit the following two nurturing behaviors:

1. they do not retrieve their young, despite normal maze running ability.

2. they do not nurse their young, despite normal mammary gland development.

By validating that *FosB* deficient moms are not incapacitated in basic organismal functions like spatial ability or hormone physiology, Brown et al have shown that *fos B* is important to nurturing *per se* and not merely a pleiotropic consequence of *fos B*'s effects on other non-nurturing traits. Indeed, *fosB* deficient mothers have normal expression of the reproductive hormones Estrogen and Progesterone, which change during the course of the reproductive cycle of the mother. In addition, the *fosB* deficient mothers also have normal olfactory abilities based on olfactory discrimination tests. *fosB* deficient mothers simply do not nurture their young, and there is little else wrong with their phenotype.

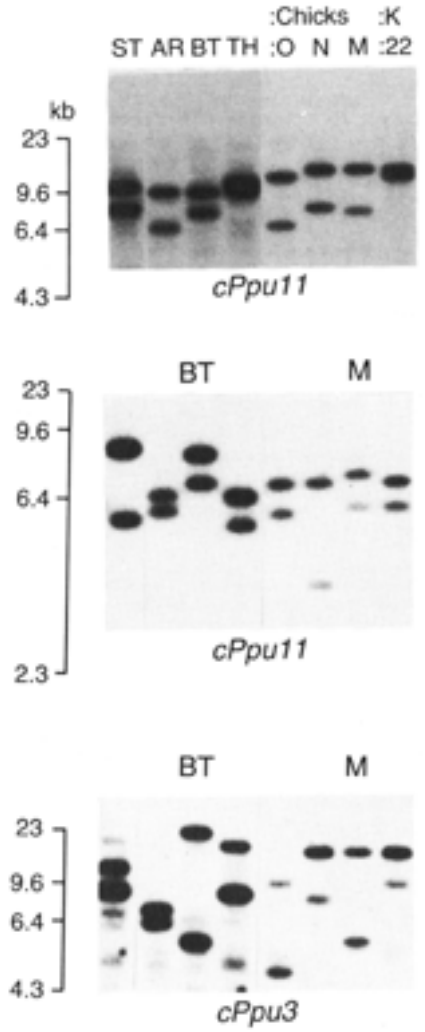
When normal female mice, intact virgins, and even males, are exposed to pups the expression of *fosB* genes are triggered in the preoptic area of the brain. Olfactory neurons in this neural pathway appear to be particularly important. The presence of *fosB* is *necessary* to induce normal parenting behavior, but it is not the only gene required -- it alone is not sufficient to elicit all aspects of parental care. Nevertheless, its the correct expression of *fosB* is crucial for the expression of normal nurturing. The *fosB* gene represents a crucial link to the chain of proximate factors that lead to a complex suite of parenting behaviors in mice, and perhaps many other mammals. While the *fosB* gene is necessary for normal nurturing behavior it is not sufficient. Therefore, *fosB* is not "the gene" for nurturing, it is however a key gene in the cascade of genes for nurturing behavior. Often claims are made in the popular press that "Scientists" have discovered the gene for behavior *X*. Invariably the "Scientists" have discovered a key gene in the cascade of gene action that gives rise to behavior *X*.

<Figure 2.2. on the normal expression of *fosB* in the preoptic area of mice brains, not shown>

Paternity Analysis and a Gene for Alternative Male Strategy in Ruffs

Molecular methods are also useful for determining pedigrees of animals which have bred in the wild. In such cases the mother is usually known with certainty because she laid the eggs. However, the female could have mated with a large number of putative sires. DNA paternity analysis has become standard in the determination of the identity of an offspring's sire.

Ruffs are a shore bird that breed and nest in northern Europe. Ruffs come in two plumage and behavior morphs. "Independent" male ruffs have a territory and defend females against other independents. The "territories" are very small and localized in "leks", which are areas where many males aggregate and display to attract visiting females. Non-territorial "satellite" males move among the independent males and obtain copulations from females on the independent's territory. Ruffs are fixed in their plumage color and behavior throughout their lives. Eighty-four percent of ruffs are independents, and sixteen percent are satellite males.



← **Figure 2.3.** Variation in minisatellite alleles in male ruffs (ST, AR, BT, TH) that are used to determine the sire of progeny (0, N, M) of the female (K:22). Allelic variation at three microsatellite loci are shown (cPpu11, cPpu11, cPpu3). Given the mothers known contribution to offspring, male BT is the only male that could be could contribute alleles at all three loci and he is the likely sire. (from Lank et al. 1995). The other sires are excluded because they lack a given allele that the real sire must have (i.e., an **exclusionary criteria**).

David Lank (1995) and his colleagues used molecular probes called "mini-satellites" to determine which male sired the chicks on ruff breeding grounds in Finland. By comparing the alleles in chicks, alleles in the mother and alleles in the putative sires, they were able to determine the father and reconstruct the father's morphology (Fig. 2.3). They also scored the morphology of the female parent's brother and father to determine the likely phenotype that the female would have expressed had she been male. Such genetic sleuthing is particularly important with sexually dimorphic traits which have a sex-limited expression (e.g., traits only seen in males or only seen in females). Females do not express the alternative male morphology, yet they might carry genes for the morphology and they pass on these genes to their male offspring. Lank and his colleagues then reared the field caught chicks to maturity when they could score the breeding morphology of

Figure 2.4. Genetic variation in plumage of male ruffs can be categorized into light (satellites) or dark plumage (independents) (photos by Lank).

the male progeny. They also reared an additional generation of chicks in captivity that were derived from the field-collected cohort of chicks. They were certain of the father's identity in captive bred birds and did not need to use molecular probes to determine paternity.

Because only two phenotypes are common in the ruff, a single genetic locus with two alleles must be controlled by a completely dominant allele and a recessive allele, otherwise we would expect to see three phenotypes. The data on ruff pedigrees can discriminate between several simple Mendelian patterns of inheritance. Mendelian genes are either found on sex chromosome or on autosomal chromosomes. Their genetic crosses rule out the possibility that the gene is a sex-linked dominant (Lank et al. 1995). Their pedigree data is consistent with an autosomal gene in which *Satellite* is dominant to the recessive allele for *Independent*.

While the example of male ruffs provides solid evidence of major genes that affect the phenotype, the sample size in these studies is still inadequate to test for the existence of other genetic loci that control development of male behaviors. Testing for the effects of two or more factors requires sample sizes in the thousands, particularly if those factors interact epistatically or in a non-additive fashion.

Epistatic Genes for Alternative Male Strategy and Sex Transformation in Marine Isopods

A final example of genes that have a major effect on behavior involves the genetic control of alternative male behaviors in a marine isopod, *Paracerceis sculpta*. Stephen Shuster and his colleagues have characterized three alternative male morphs in the marine isopod (Shuster and Wade, 1991, Shuster and Sassaman, 1997):

1. a large alpha male with elongate posterior appendages called uropods that they use to defend harems of females

2. a medium-sized beta male that can invade female harems by mimicking female behavior and morphology, and

3. a small-sized gamma male that invades female harems by virtue of its secretive behaviors.

In a large breeding study, based on hundreds of genetic crosses, Shuster has developed a genetic model that explains patterns of inheritance of the three morphs. Three alleles (α, β, γ) at the *Alternative male strategy* (*Ams*) locus provide a reasonable explanation of the general pattern of inheritance of the male morph. The three alleles have the following dominance relations:

1. β is dominant to both γ and α,

2. γ is dominant to α,

3. α is of course recessive to both β and γ.

The α allele occurs at a very high frequency in the population (93%) compared to either β (1%) or γ (6%). An alpha male phenotype must be homozygous (e.g, $\alpha|\alpha$) at the *Ams* locus because α is recessive to the other to *Ams* alleles. Gamma males can be either $\alpha|\gamma$ or $\gamma|\gamma$. However, the heterozygous form of gamma $\gamma|\alpha$ ($2 \times 0.06 \times 0.93$) is very common while the $\gamma|\gamma$ form is rare in natural populations. Likewise, beta males can be $\beta|\gamma$, $\beta|\alpha$, or $\beta|\beta$. Because α is the most common allele in the population, the $\beta|\alpha$ is the most common genotype for a beta male. As an exercise, compute the frequency of each male genotype assuming

Hardy-Weinberg gene frequencies (see Side Box 2.4) and convince yourself that α|α, β|α , and γ|α are the most common male genotypes in the isopod population.

Shuster and Sassaman (1997) found that a careful inspection of genetic crosses between females and the predominant male genotypes revealed a significant departure from a 50:50 sex ratio of the progeny. The existence of a second locus termed transformer (*Tfr-two alternative alleles, 1 and 2*) is required to adequately explain the aberrant sex ratio found in certain genetic crosses. The *Tfr* locus causes males to transform into females, or females to transform into males, as the embryo develops into the adult.

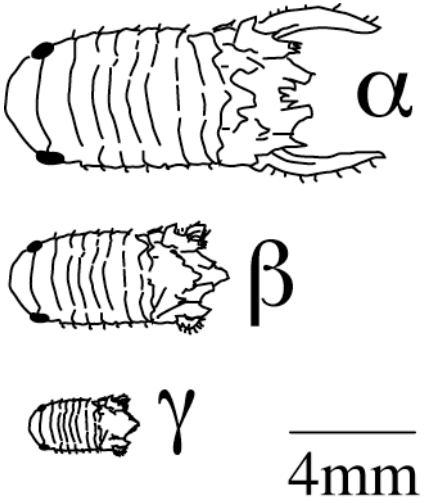


Figure 2.5. The three morphs of the marine isopod, *Paracerceis sculpta*. Drawing by sinervo, after Shuster (1989).

However, the direction of sex change depends on the genotype at the Ams locus. Homozygous alpha males transform from male to female if they bear at least one copy of the *Tfr* - 2 allele (e.g., genotypes 1|2 and 2|2). Alpha males with the 1|1 genotype at the *Tfr* locus are not sex-transformed. Conversely, beta males transform from female to male if they bear at least one copy of the *Tfr* - 1 allele (e.g., genotypes 1|1 and 1|2). Beta males with the 2|2 genotype at the *Tfr* locus are not sex-transformed. Gamma males transform from female to male if they are homozygous for the Tfr-1 allele, but not if they carry one or more copies of the Tfr-2 allele. The epistatic interaction between two loci governs the expression of male and female behaviors in this marine isopod. The gene for *Alternative male strategy* interacts in a very non-additive fashion with the gene for *Transformer*. The *Tfr-1* allele does not always transform females to males, but it interacts with alleles at the *AMS* locus to turn females into males or males into females. Epistasis leads to such non-linearity in gene action.

Table 2.2. The interaction between three alleles at the Alternative male strategy locus (*Ams*) and two alleles at the Transformer locus (*Tfr*) in a marine isopod, *Paracerceis sculpta*. See text for the dominance relations at the Ams locus. A plus symbol with arrow (+) indicates a sex transformation owing to the alleles at the Tfr locus. Alpha males that bear one or more copies of the *Tfr*-2 allele are transformed into females during embryogenesis (pink). Conversely, Beta males that bear one or two copies of the *Tfr*-1 allele are transformed from female to male (blue). The action of the *Tfr* gene can be accentuated by the presence of an extrachromosomal factor ECF (*). However, gamma males are only transformed from female to male if they are homozygous for *Tfr*. From Shuster and Sassaman (1997).

		Tfr genotype		
Ams genotype	SEX	1 | 1	1 | 2	2 | 2
α | α	M	–	–	–
	F	–	⇓* +	⇓ +
β | α	M	–	–	–
	F	⇑ +	⇑* +	–
γ | α	M	–	–	–
	F	⇑ +	–	–

Finally, we find an added complexity in this interesting genetic system. An extrachromosomal factor (ECF) accentuates the effect of the *Tfr* locus, but only in the *Tfr* heterozygotes of alpha and beta males. The exact nature of the ECF remains obscure but cytoplasmic effects that determine phenotype are common in the animal kingdom. They could be transmitted from mother to egg (as are many cell organelles such as mitochondria).

Polygenic Inheritance

Needless to say, analysis of the genetic control of behaviors becomes a daunting task for anything but the simplest genetic systems. As the number of genetic loci increases beyond three or four it becomes increasingly difficult to isolate the expression of a phenotypic trait to specific genes by using standard genetic crosses. The sample sizes required become far too large in practice. Consider two loci each of which has two alleles (A,a and B,b). Let us assume that alleles at both loci additively act in the following fashion. Whereas the allele *a* adds nothing to phenotype, the alternative allele *A* increments the phenotype by one unit. Likewise, allele *b* adds nothing to phenotype, the alternative allele *B* increments the phenotype by one unit. A Punnet square which describes the union of all possible gametic types yields 16 genotye combinations and five phenotype combinations (see Table 2.3).

Table 2.3. Punnett Square for the effect of two loci, which have purely additive effects yielding the following numerical values for phenotype:

	AB	**Ab**	**a B**	**a b**
AB	AABB = 4	AABb = 3	AaBB = 3	AaBb = 2
Ab	AABb = 3	AAbb = 2	AaBb = 2	Aabb = 1
a B	AaBB = 3	AaBb = 2	aaBB = 2	aaBb = 1
a b	AaBb = 2	Aabb = 1	aaBb = 1	aabb = 0

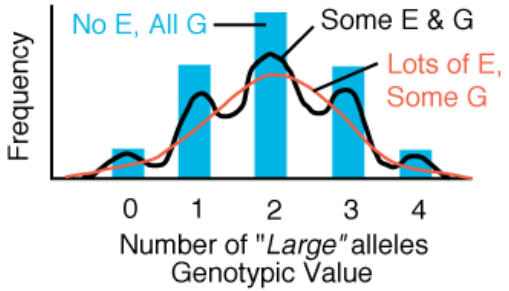


Figure 2.6. Phenotype distribution governed by 2 Mendelian loci that act additively. If the phenotype is not affected by environmental factors then 5 distinct modes are present. As phenotype becomes more and more affected by random environmental factors, the phenotype distribution becomes more normally distributed.

Side Box 2.5: Heritability

A central aspect of Darwin's theory of evolution includes a key statement: directional natural selection acts on traits that are heritably transmitted between parents and offspring.

Darwin's cousin, Sir Francis Galton coined the term for regression based on the observation that the regression line which predicts offspring phenotype from a parent's phenotype always has a slope less than one. Galton was referring to the pattern in which offspring from parents with both kinds of extreme values "regressed" towards the population mean (e.g., the slope of the line relating parents and offspring was always less than one, and the average offspring was closer to the mean than the parents, based on the line drawn through the points). The slope of the parent-offspring regression line is also known as **heritability**. The heritability for any phenotypic trait describes the proportion of the offspring's phenotype that we can predict from knowledge of the phenotype of both parents. The prediction is not exact and includes some error about the regression line because offspring phenotype includes environmental effects in addition to polygenic factors inherited from parents. Finally, another formulation of heritability is derived from Eqn. 2.1:

heritability = h^2 = G / P = G / (G + E).

Heritability is the proportion of phenotypic variation (P = G + E) that is due to genetic causes (G). Because phenotypic variation is larger than genetic variation, heritability be less than one.

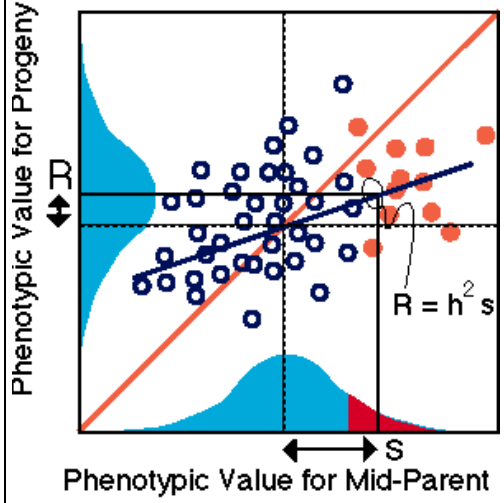


Figure 2.7. Heritability and the concept of regression.

Rather than isolate the action of single genes, quantitative genetic theory simplifies the problem and assumes that traits are due to many genes of small effect, each of which act additively to produce the phenotype (see Side Box 2.2). As the number of Mendelian factors increase, and as more of the phenotype is determined by environment, the distribution of many traits resembles a normal distribution (see Figure 2.6). The inheritance pattern of such polygenic traits is succinctly described by a simple parameter known as heritability (see Side Box 2.5. Heritability). The heritability reflects the proportion of a phenotypic trait that is due to the additive genetic effect of loci. The most straightforward way to estimate the heritability of a trait is to measure the resemblance or correlation between relatives such as parents and offspring, or the correlation between sibs. The statistical method used to measure a linear relationship is linear regression, which is described in Appendix 1.

A major caveat of the quantitative genetic approach to behavioral genetics is that it is quite difficult to build in epistatic effects such as those that are seen in alternative male morphs of isopods. This is because additive effects are easy to model. In contrast, epistatic models can generate hundreds of possible combinations for even a simple two or three locus system that interacts with another two or three locus system (e.g., see Table 2.2 on the *Tfr* and *Ams* gene of isopods).

It is possible for measured heritability to be confounded with environment (Falconer, 1981). For example, if parents and offspring share a common environmental factor, which makes them more similar by upbringing than genes alone heritability could be inflated. In principle, a regression based upon any related individuals can be used to estimate heritability. Sibs share the additive effects of alleles, much like parents resemble offspring because of the additive effect of alleles. We could use a correlation between sibs to predict h^2, however, sibs have an even greater tendency to share common environmental factors owing to their common rearing environment. The h^2 derived from sibs is likely to be inflated owing to shared environment. Sibs not only share additive effects of alleles and a common environment, but they also share another component of variation referred to as dominance variation that makes them resemble each other more so than they do their own parents. This added genetic complexity in sib relationships is discussed below.

Heritability of IQ

Few arguments in behavior and genetics are as contentious as those that rely on heritability estimates for IQ. Periodically, a popular book arises that argues for genetically based differences among racial or ethnic groups. These arguments invariably rely on estimates of the heritability of IQ derived from twin studies. In a sense, it is perplexing that something as well researched as IQ can remain so fiercely debated. For example, in a recent study Devlin et al (1997) amassed 212 separate analyses of familial resemblance, which comprised 50,740 distinct pairings of varying degrees of familial relations. Estimates of heritability were derived from correlations between: monozygotic twins, fraternal twins, siblings, parent and offspring, adoptive parents and offspring.

If IQ were determined by a large number of purely additive genes and did not include environmental influences, then the coefficient of relatedness could be used to predict IQ. Because twins share identical genes, a correlation between their IQ's should be close to the theoretical maximum of 1.0, which assumes no environmental influences on IQ, and only purely additive genetic influences. The correlation for identical twins reared together is 0.85, which suggests that at least part of IQ is environmental in origin reducing the value from its theoretical maximum of 1. However, the IQ of 0.75 for twins that were "separated-at-birth" and reared in different household environments provides a better estimate for IQ, and it suggests that the fraction of the variation that is due to a common rearing environment is 0.1. This still suggests a very strong genetic component to intelligence as indexed by IQ tests, but twins still share a common womb, which inflates the estimate.

However, the heritability estimate based on twins still confounds a number of genetic and environmental factors. Many popular arguments that rely on twin studies do not isolate the proportion of the correlation between relatives that is crucial to evolutionary arguments -- the additive genetic variation. The second component of genetic variation that is found between sibs does not contribute to evolutionary change -- dominance variation. This dominance component is often included in estimates of the heritability of IQ and when it is included we call it **broad sense heritability**, rather than the true estimate of **narrow sense heritability**, which is based on the additive genetic component.

Table 2.3. Correlations for IQ among various degrees of familial resemblance (table modified from Devlin et al. 1997). N.B. the * gives an example: While the coefficient of relatedness is 0.5 for each parent, if IQ were entirely due to additive genetic factors we could exactly predict IQ of offspring from parents so we have the following total quantity of information 0.5 + 0.5 = 1.0 (Li, 1965). The coefficient of relatedness ignores environment.

Familial relation	**Coefficient of Relatedness**	**Weighted Average Correlation**	**Why estimates for being reared together and apart differ.**
Identical twins reared together	1.00	0.85	The common rearing environment of each twin leads to resemblance in IQ that is not genetic. The environment is more different than the environment of twins reared together and who share a common household. Identical twins still share a common womb environment.
Identical twins reared apart	1.00	0.74	
Fraternal twins reared together	0.50	0.59	Fraternal twins share a common womb that inflates their resemblance. They are also reared together in the same household.
Siblings reared together	0.50	0.46	There are some environmental differences in the rearing environments. Common environmental differences in the rearing environments are eliminated, but they still share common womb as do all sibs. N.B. I suppose with the advent of new embryo implantation techniques it might be possible to eventually compare the IQ of sibs reared in different womb environments in a *Brave New World* (Aldous Huxley) style of comparison.
Siblings reared apart	0.50	0.24	
Mid-parent and child reared together	1.0*	0.50	The rearing environment that parents received from their own parents might be preserved and transmitted to their own children. However, the rearing environment could also be vastly different between generations (often referred to as the generation gap).
Single-parent and child reared together	0.50	0.41	This estimate is lower than that obtained for both parents , because a single parent is a less accurate predictor of a child's genetic background. However, this correlation still includes the possibility of common household environment in upbringing of parents and offspring. The correlation from a single parent and child reared apart eliminates common household environment in upbringing of parents and offspring.
Single-parent and child reared apart	0.50	0.24	
Adopting Parent and child	0.00	0.20	Resemblance must be largely due a common household environment in upbringing of parents and offspring. Pure cultural transmission of IQ.

To clarify these issues, Devlin et al (1997) used a technique called meta-analysis in which results from a large number of individual studies of familial resemblance were used to estimate various genetic and environmental influences on IQ. The model which best fit the data included an additive genetic factor, a factor for dominance variation, a factor for rearing household, and factor for pre-separation environment. The pre-separation environment, which has been neglected in previous twin studies, includes the common womb environment that twins would simultaneously share at or shortly after birth, but prior to separation by adoption. Devlin et al (1997) found that 20% of the variation in IQ was

explained by a common womb environment. While non-twin siblings share the same womb, they do not share it at the same time, but sequentially share the womb. Only 5% of the overall resemblance in IQ between ordinary siblings could be linked to this interesting maternal factor. Devlin et al's study shows that variation in IQ has more to do with development in the womb than previously thought. They suggest that many environmental agents are the likely causes of this environmental effect. For example, nutritional state, smoking, and alcohol consumption by the pregnant mother are all known to lower IQ of the progeny. Many toxic chemicals like PCBs (Polyclorinated BiPhenyls) lower IQ because they mimic effects of naturally occurring hormones. PCBs, which were commonly used in the manufacture of electronics, mimic the effect of the hormone thyroxine, which is critically required during fetal brain development. Individuals exposed to PCBs have an IQ that is 10 points lower (or more) than average.

From the results of Devlin et al (1997), it is clear that previous estimates based on twins have greatly overestimated the heritability of IQ (e.g., as high as 60-80%). The heritability estimate, which excludes maternal womb environment and common household environment, is 0.48.

Finally, many IQ studies do not make an important distinction between the total genetic variance in IQ and the additive genetic variance. Any estimate of IQ from full siblings confounds the additive genetic component of IQ with the genetic component in IQ that arises from dominance variation. All full siblings are expected to resemble one another a little more than other family members owing to the possibility that they inherit a similar dominance configuration from their parents (see Side Box 2.6). While full siblings share this additional genetic source of resemblance, it is not transmitted between parent and offspring. Thus, dominance variation is not an important source of genetic variation underlying directional change in intelligence. The amount of additive genetic variation for IQ is estimated at 0.34 (excluding dominance variation), which is still substantial but far lower than the amounts generally purported for this interesting human trait. Moreover, the identified environmental factors such as the womb environment (5-20%), and the common household environment (17%) total 22-37%, nearly the same as additive genetic factors. Improvement in human IQ could be brought about by pre-natal and post-natal intervention, and simply cleaning the environment.

Additive versus Dominance Variation

Relatives vary in the proportion of alleles that they share in common and the coefficient of relatedness is a measure of the probability that an allele is shared between relatives by descent. For example, the coefficient of relatedness is 0.5 for one parent and offspring, 0.5 for sibs, 0.25 for half sibs, and 1.0 for dizygotic twins. By sharing alleles, relatives end up sharing the additive effects of individual alleles. While the relationship for the proportion of shared alleles between a parent and an offspring is exact, the relationship between sibs is probabilistic. Offspring get exactly one half of their genes from a single parent. However, it is theoretically possible that full sibs could share none of their alleles, one of each pair of alleles, or both pairs of the alleles (Li, 1965). How could this be? First we can label alleles that parents could potentially give to their children: 1, 2 from their mother and 3, 4 from their father. Likewise, the situation where sibs share no genes occurs with probability one-quarter for any given gene. The most likely situation is when sibs share a single allele of a pair of alleles, which occurs with probability of one-half (they can share 1 allele 4 different ways, allele 1, allele 2, allele 3, or allele 4 in Figure 2.8). Finally, if offspring happen to share both copies of their alleles in common they also share any dominance relations between the two pairs of alleles that they inherit. This occurs with probability one quarter for the given gene.

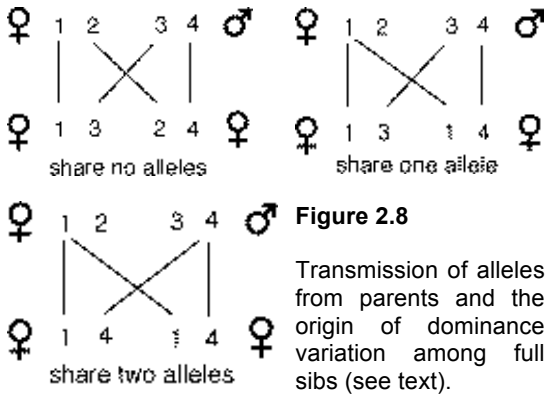


Figure 2.8

Transmission of alleles from parents and the origin of dominance variation among full sibs (see text).

If sibs happen to share pairs of alleles 1 and 4 (or any other set of two alleles), and 1 happens to be dominant to 4, then the sibs still inherit the additive effect of the genes as well as the dominance configuration of the two parents (the probability that sibs will share two alleles is ¼ so that the amount of dominance variation among full sibs is ¼ $V_{dominance}$ or ¼ of the total dominance variation in a given trait (Fisher 1918). Sibs still resemble each other largely because they share the additive effects of genes. However, sharing a similar dominance configuration also increases the resemblance between sibs relative to the resemblance between parents and offspring. Sibs resemble each other more so than they do their parents. This additional genetic component of variance is why some sibs appear to be identical twins. The additional dominance variation that sibs share (no other relationship shares this component of dominance variation) is also shared by twins. While full sibs share dominance variation, offspring do not share dominance variation with their parents, only additive genetic variance.

The final complication is that animal mating systems are rarely so simple as in the first three cases. It is likely that progeny will have different sires in a **polygamous** mating system. Depending on whether females are more promiscuous, referred to as **polyandry**, or whether males are more promiscuous, referred to as **polygyny**, or both sexes are promiscuous, progeny will have a lower probability of sharing alleles.

In this case, the relatedness will be lower and progeny will share far fewer alleles. The consequences of asymmetries in relatedness are the source of intense genetic conflict that has far-reaching consequences for mating system and social system dynamics, which is detailed in chapter 10.

Environmental Effects on Phenotype

I will end the discussion of genetics on the importance of environmental factors. Up to this point in our consideration of the genetic causes of behavior, we have treated the environment as if it were some random factor that obscures the genetic transmission of behavior. However, the environment can interact with genetic causes in interesting ways. In particular, the genotype can be relatively fixed (e.g., little variation among individuals), but the genetic machinery of development can still allow the phenotype to develop into alternative types. We have already seen how genetic differences among individuals can lead to alternative male phenotypes in marine isopods and the ruffed grouse. The environment per se can also trigger alternative developmental pathways that transform a juvenile into different morphologies, which have alternative behaviors (Smith-Gill 1983).

Condition-dependent strategies and alternative male types

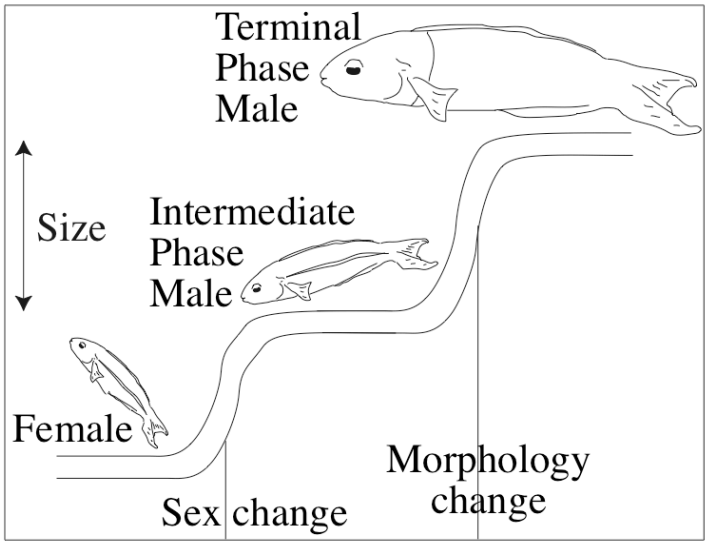
Many alternative male types are thought to be **condition-dependent strategies** in which an organism's internal physiological condition (e.g., nutrition, age, or body size) interacts with environmental factors (e.g., food availability, hatching date, etc.) to alter development of morphology and behavior. Differences in morphology and behavior are not governed by genetic factors such as alternative alleles. Bluegill sunfish have three alternative male phenotypes, however, the attempt to isolate alternative alleles that control such behaviors have not been successful (Gross, 1984; Gross, 1991). It is thought that when males mature into three different-sized types with different behaviors, the trigger for the three states is the overall body-size at maturation. Males in good condition may mature later, and grow into a large territorial male, that defends a small area in which females deposit their eggs. The male is also parental and takes care of the brood. Males that somewhat smaller, mature earlier, and develop into a female mimic that attempts to fertilize the female's eggs as the female attempts to oviposit them on a territorial holding males nest. Finally, the smallest male type is thought to mature rapidly, and at very small size. This sneaker male darts between the breeding pairs, and relies on confusion to obtain close access to the female. This sneaker male then squirts sperm while swimming between the territorial male and female. Evidence that these types are environmentally determined is provided by experimental manipulations of the availability of nest sites. If nesting sites are restricted, the frequency of medium and small-sized males increase, and the frequency of large parental males decrease (Gross, 1991)

Another case of condition-dependent strategies arises in the case of reef fish. For example, the **protogynous** hermaphrodite, the blue-headed wrasse, *Thalassoma bifasciatum*, are reproductive females during early life, intermediate phase (IP) males in mid-life, and terminal phase (TP) males during late-life when they grow to a sufficiently large size that

allows for nest defense (Warner & Hoffman 1980; Warner 1984). All individuals can be male or female, but social conditions dictate the change in behavior and morphology.

Condition-dependent strategies are also referred to as phenotypic plasticity. The phenotype of fixed genotypes can respond plastically to the environment. While the alternative phenotypes are not controlled by alternative alleles, the development of the phenotype is still governed by a cascade of events that are under some kind of genetic control. The distinction is important because presumably all individuals in a population are capable of developing into the alternative phenotypes, if their environment had been conducive to these alternative developmental pathways from the outset.

Figure 2.9. Sex change with age in the blue-head wrasse, *Thalasomma bifasciatum.* These changes in body form are governed by a complex endocnrine cascade, which is discussed in Chapters 7 and 8.



Alternative Larval Types in Spadefoot Toads

A clear example of alternative behaviors is seen in the development of larval amphibians. The interaction between environment and the behaviors governing energy acquisition are central to the growth and development of individuals. A basic dichotomy in feeding strategies entails the distinction between carnivory and omnivory. Carnivores largely feed on animals while ominvores tend to have a more broad diet consisting of animals, plants, and in some cases decaying plant and animal remains, which is referred to as detritus.

If you were to wander into the deserts of the southwest during dusk after a hot July day has been chilled by a torrential thunderstorm, you will find a small toad calling for mates. Clustered around ephemeral pools filled with water, you will find choruses of singing male spadefoot toads trying to attract females that are hopping towards the water to lay their eggs. Water is a scarce resource for the toads and they emerge with the first rains. Successful adult males clasp the back legs of females in a tight embrace, which is referred to as **amplexus**, and he stimulates the female to oviposit her eggs. The fertilized eggs are left behind in the pool to develop. As you might have already guessed, water does not last long in the glare of the summer desert sun. Tadpoles that hatch from egg masses must develop rapidly into the terrestrial adult of a toad form if they are to survive.

If a tadpole happens to hatch in a pond that is rich in fairy shrimp, a key resource (a.k.a, *Artemia salina* or sea monkeys), the tadpole might ingest enough shrimp and its development can be profoundly altered (Pfennig 1990, 1990a). Tadpoles that eat a lot of the shrimp are triggered to develop into a carnivore with a specialized keratinized tooth and large jaw musculature. The jaws are more efficient for feeding on shrimp. Carnivores patrol the pond in a solitary fashion searching for their next meal.

If however, the tadpoles end up in a pool with few or no shrimp they develop into an omnivore with a rasping jaw structure, and a huge gut that allows for efficient digestion of plant remains. Omnivores tend to swim in schools and the schooling behavior is thought to allow the group to stir up food more efficiently. Individuals are capable of transforming into the carnivorous morphology or the omnivorous morphology and behavior. They only need to ingest enough shrimp to develop into the carnivorous form.

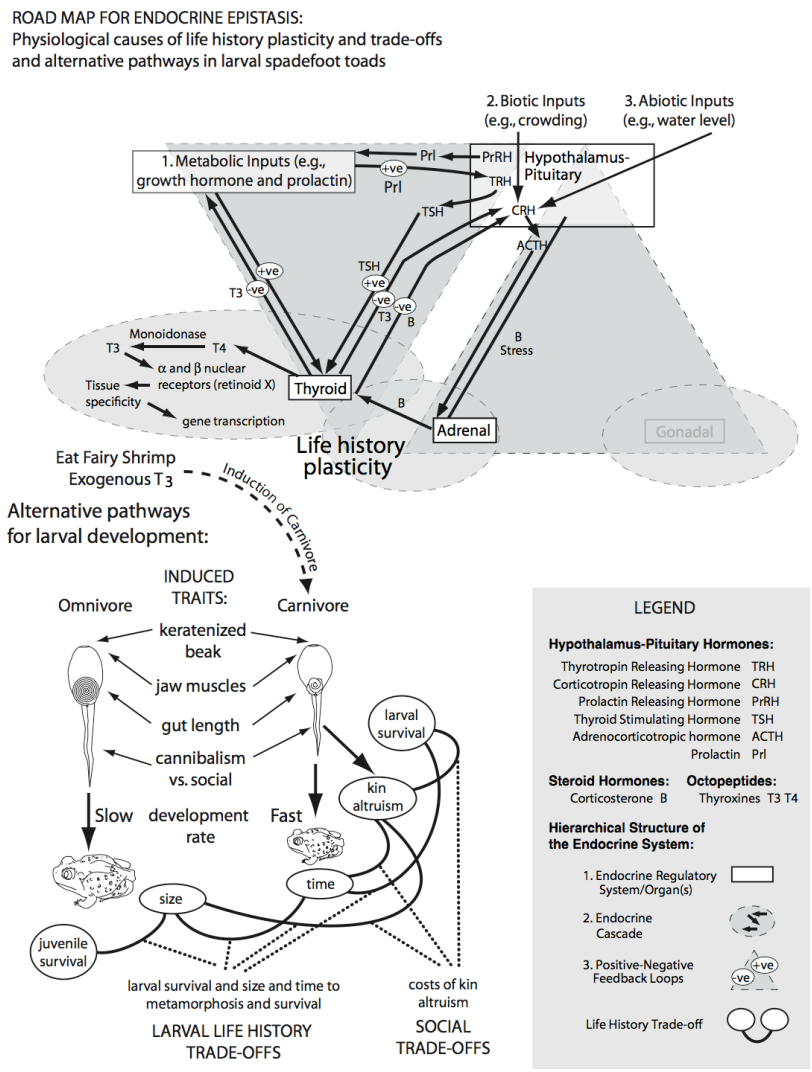
Side Box 2.6 — Endocrine control of life cycles in amphibians.

Hormones that regulate metamorphosis and migration in a typical pond-breeding amphibian (*Scaphipus multiplicatus*). In this species the entire regulatory system of larval growth has also been coopted to generate two distinct morphs of larval amphibians. The HP-prolactin system interacts with the HP-thyroid system to generate larval growth and metamorphic climax via a process of positive and negative regulatory feedback. Each endocrine system is hierarchically organized into: 1) endocrine glands, 2) endocrine glands trigger endocrine cascades of gene transcription, 3) two or more endocrine glands (and their endocrine cascades) interact to form endocrine axes, and 4) three endocrine axes comprise the fundamental unit of positive and negative endocrine regulation that generate organismal homeostasis or that trigger behavioural and physiological changes in life cycles.

In all vertebrates, metabolism is regulated by thyroxine, a small chain of eight amino acids called an octopeptide. In all vertebrates, a diverse class of protein hormones collectively called the growth hormone gene family regulate growth rate. In amphibians, a specific growth hormone called prolactin interacts with other growth hormones (Sawada et al. 2002) to regulate larval growth and inhibit metamorphosis (Denver 1997). High levels of prolactin inhibit metamorphosis, but enhance larval growth. At a critical larval size, prolactin secretion shuts down thereby releasing inhibition of thyroxine releasing hormones (TRH). Elevated TRH produced by the hypothalamus triggers release of (Thyroid stimulating hormone) TSH by the anterior pituitary. TSH simulates the thyroid to produce thyroxine, more specifically T_4. In specific tissues, an enzyme called monoiodinase converts T_4 to the more potent T_3. In cells of these tissues, T_3 binds to two classes of thyroxine receptors, a and b receptors, to trigger gene transcription, which reorganizes body form (i.e cell death and resorption of gills and tail fin, the triggering of melanization, reorganization of the adult skull and hyoid bones). Thus, the interaction of HP-prolactin-thyroid systems achieves metamorphic climax from larval to terrestrial.

In addition to governing amphibian metamorphosis, spadefoot toads exhibit plasticity with respect to a prey item, the fairy shrimp, which is either present or absent in their natal pond (Pfennig 1992). Two alternative larval strategies of omnivory versus carnivory are induced in spadefoot toads (Box 4.5), and the two strategies arise from plastic induction of the HP-thyroid axis. Fairy shrimp naturally contain high levels of throxine and thus, when larvae consume shrimp they ingest exogenous thyroxine, which precociously triggers development of a carnivorous morphology that is even more efficient at consuming shrimp.

The larval strategies of spade-foot toads are also associated with an antisocial behaviour of cannibalism versus social behaviour of schooling. Carnivorous tadpoles are more pre-disposed to exhibit cannibalistic behaviour, owing to large head size, stronger jaw muscles and highly-keratinized beak. Omnivores are more social and school. Kin-related effects are discussed in Chapter 4, Levels of Selection. The empty endocrine network (triangle) to the right of the genetic



cascade for thyroxine, reflects another network that is related to the water drive response, which interacts with the thyroxine network in the adult form as well as the gonads. I will discuss this water drive response, which is governed by the hormone prolactin, in the Chapter on migration and dispersal. Additional gene cascades that are not shown in this figure govern reproduction, which is discussed in Chapter 7, as well as other homeostatic and endocrine systems that impact behaviors. Endocrinology is the study of these gene cascades.

What is the key environmental factor in the shrimp? Shrimp are naturally loaded with the potent hormone thyroxine and shrimp also have a heavy does of iodine a key component of thyroxine (Pfennig 1992b). Thyroxine is a key metabolic hormone found in all vertebrates. In amphibians, thyroxine governs development of the tadpole larva, and it initiates the metamorphosis from tadpole larva to terrestrial form. By ingesting large amounts of this hormone, the tadpoles precociously develop structures that allow for a more carnivorous lifestyle, structures that are incidentally more reminiscent of the adult form. David Penning performed critical experiments to show that high levels of thyroxine precociously trigger development of the carnivorous tadpole morph.

Carnivores develop much more rapidly than the omnivore and carnivores metamorphose into toadlets in a shorter period of time. The omnivore must consume greater quantities of food to reach the size and stage required for metamorphosis to a terrestrial toadlet that can escape the desiccating pool. Carnivores have a tremendous time advantage in small ephemeral pools. Omnivores take longer to develop, but they tend to metamorphose at a slightly a larger size, and with greater fat reserves. Omnivores are successful in more permanent ponds. In contrast, the fairy shrimp is much more abundant in the smaller faster drying ponds. The plasticity in development of behavior and morphology is adaptive in that a female can leave progeny in a small fast-drying pond or a larger long-standing pond, and the tadpoles environment, presence or absence of shrimp determines the offspring's development.

Summary: Physiological epistasis and endocrine regulatory networks of genes

Sewall Wright (1969) considered physiological epistasis to be universal in genetic systems (Wade 2002, Sinervo and Svensson 2003). However, he theorized that genetic variation in epistatic networks destabilized organismal function, and he suggested that epistatic genetic variation is fixed in most species owing to such negative effects on fitness. Physiological epistasis is ubiquitous in the sender and receiver molecules of endocrine regulation, even if endocrine networks are fixed on a single type in the population.

Thus, endocrine networks of Side Box 2.6, are largely fixed for genetic variants in many species. However, key regulatory loci may harbor alternative alleles in some species, which generate alternative morphs that have a striking genetic basis (e.g., isopods, ruffs). In such species, expression of endocrine pathways is often altered by the morph loci. Morph loci like the *AMS* locus of isopods are thought to consist of supergenes or key regulatory genes of the endocrine system that organize suites of behavioural, morphological and life history traits (e.g., amphibian HP-thyroid axis). It is precisely for this reason that alternative morphs are of great interest to life history theory in general, and the evolution of behaviour in particular. A study of alternative morphs provides a window on the role of genetic and physiological epistasis in generating behavior (Sinervo and Calsbeek 2003). In Chapter 11 and 15, I will further illustrate physiological epistasis with respect to trade-offs in the design of the fundamental sexual morphs of all sexual species, males and females.

In the case of spade foot toads, the trigger for different morphs is not a genetic difference *per se*, but the trigger is pulled when the tadpole consumes enough shrimp, an environmental source of thyroxine. Therefore, the differences between genetic versus environmental control are really only related to the proximate control, a gene with alternative alleles, or an alternative environment. Both of these mechanisms act on fairly complex gene cascades to effectuate changes in behavior, morphology, and physiology.

Study Questions for Chapter 2

1.What environmental factors increase the similarity of sibs? What genetic factors are responsible for the similarity of parents and offspring? What additional genetic factors increase the resemblance between sibs (see Side Box 2.2 and Side Box 2.6)?

Which is important for evolutionary change and why?

2. Describe a mutation experiment that was used to isolate a gene for nurturing in mice. Why is it important to show that mice which are defective in this gene can perform non-nurturing tasks like maze running or olfaction tests? What kinds of genetic effects do these additional tests detect? Why are we interested in a lack of effect of a gene on other traits? (Hint what genetic effect confounds the interpretation of the results from a mutation experiment?

3. Why are heritability estimates for sibs raised in different households preferable to sibs raised in the same household? What additional confounding factors does this design not remove from the heritability estimates?

4. You overhear someone in the coffee shop citing the following evidence regarding heritability of IQ: "IQ must be very heritable, the correlation between fraternal twins reared by the same family is 0.59." "Hah!" the other combatant exclaims, "The correlation between an adopted child and their unrelated sib is 0.20." Who is right and why?

5. Compare and contrast genetic determination and condition-dependent determination of as a proximate causes of alternative male strategies.

6. Why are progeny on average half related, and why can progeny also be completely unrelated?

7. Briefly outline an endocrine cascade of genes for larval development in amphibians (diagram the thyroid system). Why is the environmental control of behavior and morphology similar to the genetic control of behavior and morphology? In what way are they different?