Chapter 3: Selection and Adaptation

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> It may metaphorically be said that natural selection is daily and hourly scrutinizing, throughout the world, the slightest variations; rejecting those that are bad, preserving and adding up all that are good; silently and insensibly working, whenever and wherever opportunity offers, at the improvement of each organic being in relation to its organic and inorganic conditions of life. We see nothing of these slow changes in progress, until the hand of time has marked the lapse of ages...
>
> Darwin, 1858

Introduction to Selection and Adaptation

Observing direct evidence of natural selection in the wild is difficult. However, long-term studies have provided us with a rare glimpse into this process that Charles Darwin viewed as too slow to record. Recent advances in computing have made it possible to investigate the process of selection by using brute-force statistical techniques. Understanding how natural selection acts upon genetic variation is central to understanding the evolution of behavior.

For the moment we will ignore the details, and focus on the effect of genes on phenotype, and the selective consequences of phenotype. We make this simplification because an understanding of genotype and phenotype is sufficient for a superficial understanding of the process of evolution by natural selection. Because the behavioral traits are dependent on the functioning of other phenotypic traits, we must also study how behavior evolves as a function of other phenotypic and morphological traits. Life history traits, which describe processes of birth and death, are central to understanding selection on behavior. Life history traits equate to fitness.

The analysis of genetic effects and the selective effects of behavioral traits illustrates the dichotomy between proximate and ultimate approaches to the analysis of behavior (as discussed in Chapter 1: History and Philosophy of Behavioral Studies). In this chapter, we will explore the two issues of proximate and ultimate causation. In terms of Tinbergen's four processes we focus on *causes*, in this case the most proximate of causes -- genes. We also focus on *function* or *adaptive value* as we look at phenotypic selection. The change in genetic factors as a function of the selective environment reflects the process of

adaptation. The first goal of this chapter is to develop an understanding of the process of natural and sexual selection. The second goal of the chapter is to develop an appreciation of the process of adaptation. I do not want to dissect selective explanations for all animal behaviors, but rather, provide a theoretical scaffolding on which to hang analyses of specific behavioral traits, which will be treated in greater detail in Chapters 4-11.

Natural Selection

Natural selection is the differential survival and/or reproduction of organisms as a function of their physical attributes. Because of their phenotypes, which are due to the amalgam of traits that make up an individual, some individuals do better than others. The concept of selection is central to Darwin's theory of evolution His concept forms the cornerstone of many theories in the field of animal behavior. Selection is defined as some sort of functional relationship between fitness and phenotype. We can easily describe fitness in terms of three kinds of functional relationships (Lande and Arnold, 1984):

1. directional selection -- the trait is linearly related to fitness,
2. stabilizing selection -- there is an optimal value for the trait of interest, and
3. disruptive selection -- individuals with the smallest and largest values of the trait have the highest fitness, and individuals with intermediate values are at a fitness disadvantage.

As we will see below each mode of selection alters the mean or variance of the phenotypic trait in a population or species. In the long term, directional selection has the most dramatic impact on the evolution of a species. Directional selection can lead to the formation of a new type from an existing type. This contrasts with the action of stabilizing selection which maintains the existing type without change in mean over long periods of time. Stabilizing selection eliminates the extremes in a distribution of phenotypes, leading to a refinement of the existing type. By eliminating individuals from the center of the distribution, disruptive selection favors the individuals in the tails or more extreme values of the phenotype. Disruptive selection can lead to the formation of two new types from a single existing type. The two types are formed from the extreme edges of the distribution.

Sexual Selection

Charles Darwin (1971) distinguished sexual selection as variance in the number of mates. Sexual selection acts to refine secondary sexual characters of the phenotype such as morphological differences between males and females, or differences between male types. **Primary sexual characters** are reflected in the basic differences between male and female reproductive genital systems and accessory glands. **Secondary sexual characters** are traits shaped by the action of sexual selection. Secondary sexual characters are often traits that aid in mate competition or mate attraction, like bright colors that signal an aggressive strategy to other males or elaborate plumage that is used as a signal to attract females. The action of sexual selection can take the same three modes that are discussed above for natural selection (direction, stabilizing, disruptive), but often the action of sexual selection promotes directional selection for ever increasing trait size or signal intensity.

Darwin viewed male sexual ornaments as a curious evolutionary puzzle that begged explanation. Natural selection tends to produce individuals that are well adapted to their environment. However, sexual selection does not adapt the individual to the general abiotic environment but rather adapts and enhances traits involved in the special social environment that governs mate acquisition. This process of sexual selection can produce individuals with elaborate ornaments. These structures are often energetically costly to develop, costly to maintain, and can lead to a direct survival cost for the individual. In this sense, sexual selection has the capacity to evolve maladaptive traits. Darwin's theory of sexual selection gave a plausible explanation for the origin of many splendid and often bizarre ornaments.

In his laboratory stocks of *Drosophila*, Bateman (1948) observed that usually a few males get most of the mate and many males get no mates. Drawing upon these observations by Bateman, Robert Trivers (1972) refined Darwin's theory of sexual selection with the following lemmas. Females are the limiting sex and invest more in their offspring than do males. Many females are unavailable for fertilization because they are caring for young or developing young. Because there is an excess of males, they tend to develop ornaments as a way to compete against other males or to attract females. This leads to sexual selection as a function of female choice or **intersexual selection**, in which females choose

males based upon elaborate ornamentation or male behaviors. Alternatively, male competition or **intrasexual selection** arises in which males compete for territory or access to females, or they compete on mating grounds where displays take place. Male-male competition can lead to intense battles for access to females where males use elaborate armaments (e.g., horns of many ungulates).

While the limiting sex tends to be female, this is not always the case in the animal kingdom. As we will see in chapters on mating systems, male pipefish are limiting because they brood offspring in a pouch. In this case females compete for mates, and in some species of pipefish, females are more brightly colored than males. It is often the exceptions that prove the rule in biology, and reverse sexual dimorphism in pipefish is a case in which females have become the sexually selected sex for ornamental traits.

In this chapter, I explore one model of female choice as a driving force behind sexual selection (Side Box 3.1: Runaway Sexual Selection). I discuss this theory to illustrate how sexual selection is a unique process relative to natural selection. Sir Ronald Fisher (1931) argued that sexual selection leads to a "runaway process". Fisher was a brilliant mathematician who added a great deal of new theory to the Neo-Darwinian synthesis. His capacity for intuitive arguments was unparalleled in evolutionary biology and his writings are a challenge to read. Such was the case with Fisher's intuitive argument of runaway process. Two theoreticians, Russel Lande (1981) and Mark Kirkpatrick (1982), independently derived a formal proof of runaway sexual selection, fifty years after Fisher's original verbal arguments. These derivations led to a renaissance in our thinking on sexual selection.

In the Kirkpatrick-Lande model, alleles for female choice led females to selectively choose males with elaborate traits. This "choice allele" is distinct from the "wild-type" female that has a more random pattern mate selection. Assortative mating tends to correlate alleles for choice and alleles for the elaborate trait, while at the same time leaving alleles for non-choosy females associating with males that display non-elaborate traits (e.g., the male wild type). The system evolves in a runaway and the male with the elaborate trait rises to high frequency in the population, even if it leads to detrimental effects on male fitness. This occurs because the elaborate male obtains all the female mates that are choosy, and half the female mates that express a random choice. The non-elaborate male only gets its mates from to pool of non-choosy females.

The theory of runaway sexual selection illustrates how genes for behavior and genes for morphology can become genetically correlated or linked. The linkage between genes is not a physical linkage where two loci lie in close proximity on the same chromosome. In the case of runaway sexual selection, the genes become inextricably linked by the strength of selection. The runaway model explains correlated evolution of female behavior and male ornaments. We are used to thinking of the process of natural selection as leading increasingly better-adapted individuals in the population through continued refinement of traits. Female choice is a striking example of maladaptive evolution. What is meant by maladaptive evolution? In the case of female choice and male ornamentation, average fitness of individuals in the population declines as the frequency and intensity of the bizarre male ornaments increase.

If we could compare the average fitness of individuals in a population that has not yet evolved elaborate sexual ornaments to a population, which is derived from the original stock, but has evolved ornamented males, we would find that many ornamented males die selective deaths. The average fitness of individuals in the ancestral population would be higher because fewer males die selective deaths compared to the number of males that die selective deaths in the sexually selected population.

Fewer males survive to maturity after sexual selection acts, than in previous generations. This decreased survival is referred to as the **genetic load** (Wallace 1970) of sexual selection, which builds a high frequency of female preference and a high frequency of the elaborate male trait. This is the sense in which runaway selection leads to maladaptive evolution; the average fitness of the population declines over time. As is often the case in evolutionary biology, there are many alternative theories of sexual selection. Runaway process is one of several competing theories to explain elaborate male ornaments. We will explore alternative theories of sexual selection in upcoming chapters on male competition and female choice. Behavioral traits can often lead to maladaptive evolution. The maladaptive end result arises because sexual selection or more generally social selection results in the evolution of social environments in ways that might be counter to natural selection.

Side Box 3.1: Runaway Sexual Selection

The simplest model for sexual selection, female choice and a male trait assumes a haploid organism (one set of chromosomes). Let us assume that females come in two types and they are genetically based. The male trait is also genetically based:

1. Choosy females prefer males with elaborate ornaments
2. Non-choosy females have no preference, and pick males with bright ornaments 1/2 of the time and males with more plain ornaments 1/2 of the time (Kirkpatrick, 1982).

Notice that the ornamented male has a fitness of 3/2 and the plain male has a fitness of ½ (Table 3.1). The ornamented male is favored by sexual selection. The next step in runaway is to consider what happens to loci for female choice and loci for male traits in their sons and daughters.

Origin of genetic correlations between female choice and male traits

A table can be used to contrast the differential success of the haploid creatures: Choosy females (**C**) choose Elaborate males (**E**) and reject non-elaborate males (**N**). Non-choosy females (+) choose **E** and **N** in a 50:50 ratio, with what would be expected under random mating (e.g., + females mate randomly). When females re given a choice of two males:

Table 3.1 Fitness of males. **Male Trait**

		Elaborate Male (E)	Normal male (N)
Female Choice	**Choosy (C)**	3/2	0
	Non-choosy (+)	1/2	1/2

The key step is to predict **gametes** produced by progeny in the next generation, which we do by predicting frequency of sons and daughters:

- 1/4 of the progeny have genes for the normal male ornament and the non-choosy female gene
- 1/2 of the progeny have genes for the elaborate ornament and the choosy female choice gene
- 1/4 of the progeny have genes for the elaborate male ornament and the non-choosy female gene
- None have genes for the choosy female and the normal male.

Sons and daughters have genes for the male trait, and both sons and daughters also carry a preponderance of the choosy gene. Based on the probability of choice, a male parent receives 1 share of his mates from choosy females, and 1/2 share of his mates from non-choosy females. Thus, from the point of view of the male genes and progeny, **66%** of the male elaborate trait genes in the population are associated with the female trait gene. In contrast, a mere **33%** of the elaborate trait genes are associated with the non-choosy female gene. In addition, all Normal males breed with non-choosy females and thus **100%** of the Normal male genes (all of them) are associated with non-choosy females (conversely **0%** of the Normal genes are associated with choosy females. Because the combination of elaborate male allele is combined with the alleles for choosy females in progeny, more offspring are produced with this combination. This occurs every generation and the choosy females and elaborate males both benefit and enjoy high fitness.

Choosy genes and elaborate male genes occur at a higher frequency than one would expect if the population were mating randomly. The central prediction of runaway process is that female choice and the male trait will become **genetically correlated** or the choosy gene becomes genetically linked to the non-choosy gene. It is more likely that you find a choosy gene associated with the male trait, or vice versa that you will find a non-choosy gene associated with the drab male gene.

If we continue this process, generation after generation, all of the daughters of the choosy females have both the choice gene and the male trait gene. This means that females will be producing sons and daughters with both choice and exaggerated trait genes together. Elaborate males have an advantage in subsequent generations and because choosy female genes are genetically correlated with elaborate male genes, female choice and the male trait spread together in a **Runaway Process**.

Genes for female choice and genes for male ornaments become linked in progeny. Such linkage by the process of **positive frequency dependent selection** leads to a runaway, because elaborate males who are chosen with high frequency produce lots of daughters who will be quite choosy. A critical prediction of Runaway process is that female choice should be genetically correlated with the genes for male choice. A key aspect of Fisherian runaway that I have ignored is that male ornaments will increase in frequency even if they are costly in terms of male survival.

A (very) brief introduction to frequency dependent selection

Runaway sexual selection provides us with our first example of **positive frequency dependent selection**. **Frequency dependent selection** is a special kind of selection, which is common for behavioral traits. Under frequency dependent selection the strength of selection depends on the frequency of a type in the population. In the case of positive frequency dependent selection, as a given type increases in frequency (e.g., positive change in frequency), the intensity of selection favoring that type increases. Thus, as elaborate males (and choosy females) become more prevalent in the population, the intensity of selection favoring the elaborate trait increases, thereby fueling even stronger frequency dependent selection in future generations. The role of frequency dependent selection is so critical to the evolution of behavioral traits that I spend three entire chapters on developing these concepts (Chapters 8, 9, 10) and discussing how the various genes for behaviors generate either positive or negative frequency dependent selection.

Below, I introduce the concept of **negative frequency dependent selection**. When frequency dependent selection is negative, as a type becomes more common, selection *against* that type intensifies. Negative frequency dependent selection is extremely common when predators are searching for prey, and they tend to pick out the more common type of prey, reducing the common prey type in frequency. The interactions between predators and prey and the subject of negative frequency dependent selection is further discussed in Chapters 13 and 14.

Artificial Selection on Polygenic Traits

The selection Differential and Response to Selection

Returning to the process of adaptation, evolution reflects a genetically based change in phenotypic traits over time. Humans have practiced artificial selection to "improve" cultivated plant and animal stocks for millennia and artificial selection has produced spectacular departures of cultivars from their original native varieties. The process of domestication leads to striking changes in behavioral and morphological traits. Darwin (1859) devoted an entire chapter in "*The Origin of Species*" to the subject of artificial selection. In his mind, artificial selection and the production of domesticated animals and plants was a powerful analogy for the action of natural selection.

Since Darwin's time, artificial selection has been practiced in laboratory populations in an attempt to simulate the slow process of natural selection, and serve as an experimental model of evolution. In an artificial selection experiment, the researcher typically applies a truncation point or threshold value (Fig. 3.1). Animals above or below this value are used to propagate a given experimental line, which are termed a high selected and low selected line respectively. A useful measure of the amount of selection during a given generation is the difference in the mean phenotype before and after selection, but before the next generation of progeny matures. The **selection differential** describes the strength of selection relative to the distribution or variance of the phenotypic trait (Falconer, 1981). The selection differential compares the mean of the population before or after selection (Appendix 2 gives the statistical derivation of the selection differential):

s = mean after selection - mean before selection. *Eqn 3.1*

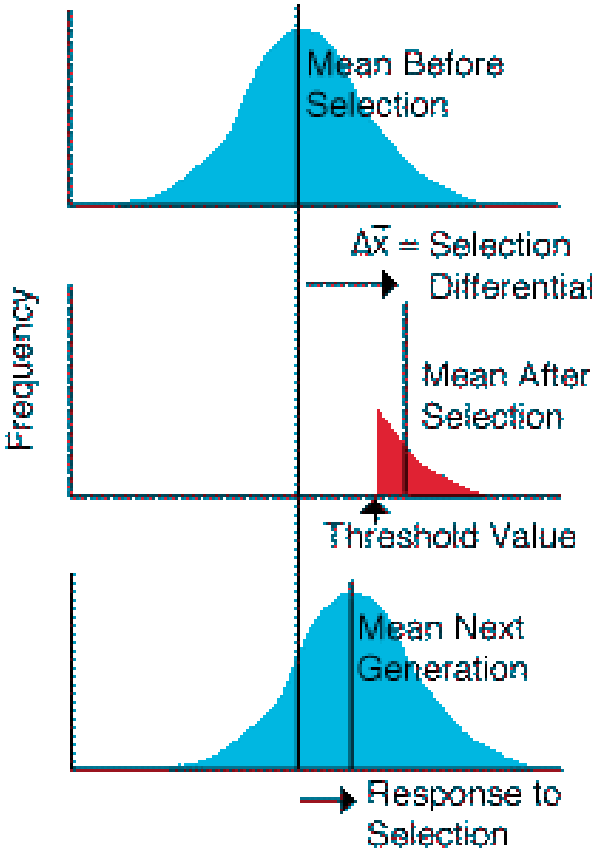
If the phenotypic distribution of the trait is due to a large number of polygenic loci, then the **response to selection** is a function of both the heritability of a trait (e.g., additive genetic variance) and the strength of selection on the trait (e.g., selection differential). With either low heritability or weak selection, response to selection is slow compared to strong heritability or strong selection. Response to selection is given by:

R=heritability × s = h^2 × s *Eqn 3.2*

The evolutionary response to natural selection is invariably less than the selection differential because the heritability of any trait is less than 1. The relationship between heritability and response to selection is explored in greater depth in Side Box 3.2. The concept of heritability is so crucial to understanding evolution that a widely used abbreviation is used as a short form, h^2. The term h^2 was coined by another architect of the Neo-Darwinian synthesis, Sewall Wright (1921) and it relates to concepts of path analysis, a widely used statistical method that is used in behavioral analysis (Appendix 1). A path coefficient like h^2 describes the proportion of variation (a squared quantity) that is due to a given cause, which in the case of h^2 is due to the additive effect of alleles.

Side Box 3.2. Heritability, Selection Differential, and Response to Selection

Figure 3.1. Schematic illustrating the design of an artificial selection experiment with **truncation selection** for large values of the phenotypic trait. Only individuals with values of the phenotypic trait greater than the **threshold value** are allowed to propagate the next generation of progeny. The change in means within a single generation caused by truncation selection is referred to as the **selection differential**. The change in means between generations is referred to as the **response to selection**.



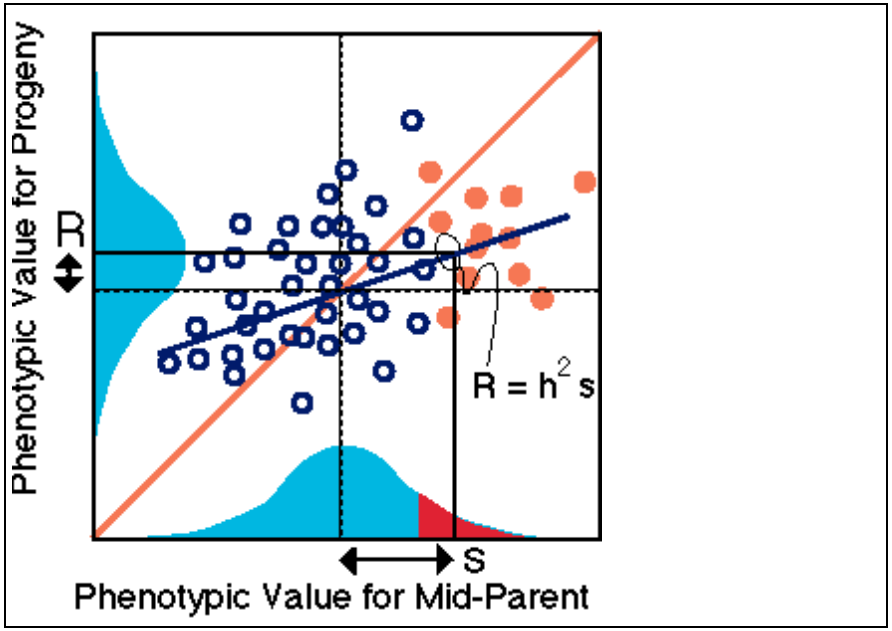


Figure 3.2. The effect of truncation selection across generations acts via heritable variation and we can visualize selection in terms of the relationship between parents and offspring. Assume that only parents with very large phenotypic values are allowed to breed. The selection differential, **s**, is the difference in mean of the selected parents versus to unselected parents (red vs. red+blue distributions on the horizontal axis). The response to selection, **R**, is the change in mean of the progeny distribution relative to parents.

Response to selection will always be less than the selection differential because, h^2, the slope of the line is less than one, or the proportion of phenotypic variation explained by additive genetic causes should be less than one. Another way to think about the relationship between R, h^2, and s is to predict R (the rise in the progeny generation) as a function of the s (the run), where rise over run is the slope (h^2). The rise (R) is a function of the run (s) times the slope (h^2) or $R = h^2 s$.

Artificial Selection and Effects on Genetic Variation

Carol Lynch (1980) selected for two strains of female mice that built either big nests or small nests. In high-selected lines, females in the upper 20th percentile of the distribution were chosen to breed. The converse was true for low-selected lines of nest builders. Control strains of mice were randomly bred from one generation to the next. She replicated each strain and kept them isolated. She was more successful in getting big nests compared to small nests. The leveling off in the efficiency of selection seen in the low line is evidence that the response to selection (R) was decreasing from one generation to the next.

Figure 3.3. Representative females at the end of Lynch's selection experiment for high and low-sized nest builders. Photo by L. Drickamer.

Lynch applied a constant selection differential to each generation thus, s in the equation $R=h^2s$ was constant. If nest size were the only trait under selection, then h^2 must have decreased with each generation in order to see a drop in R. Because h^2 is proportional to additive genetic variation, this selection experiment slowly eroded additive genetic variation. Low selected lines were becoming fixed for alleles at genetic loci that favored the production of small nests. Selection depletes additive genetic variation. Only mutation will restore the depleted additive genetic variation within any given line. Another reason for the leveling off of the response to selection could be counterbalancing selection against small nests, which perhaps arises from the effect of small nests on the probability of offspring survival. This phenomenon is referred to as **antagonistic pleiotropy**, in which the pleiotropic effect of genes (discussed in Chapter 2) for building small nests (favored by the researcher in one line) are antagonized by the lower survival of progeny. For an animal that thermoregulates, like the mouse, even in a controlled environment the neonates will require nesting material to maintain homeostasis. Either the mother or the progeny will have to supply additional energy to maintain the developing juveniles body temperature (Lynch et al 1988). Continued selection for small nests may have led to a reduction in juvenile survival. The converse limitation is not necessarily true for the high selected lines, and these lines do not yet show a clear sign of leveling off in the response to selection.

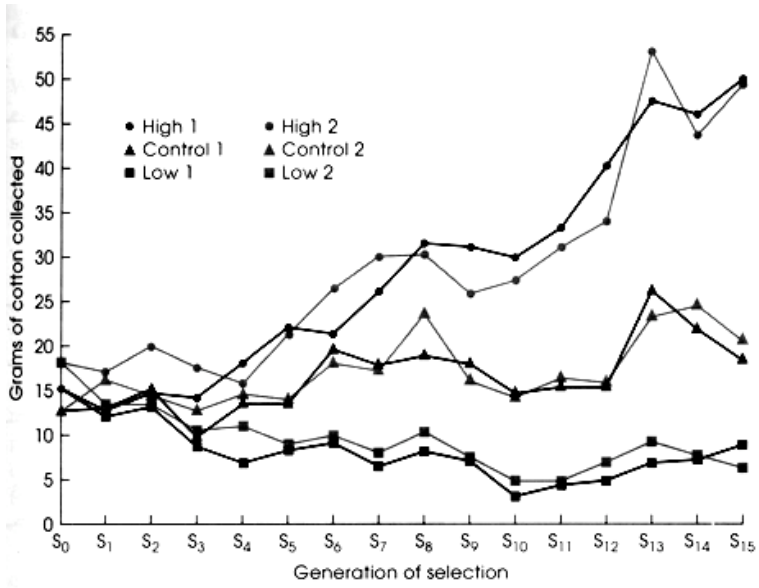
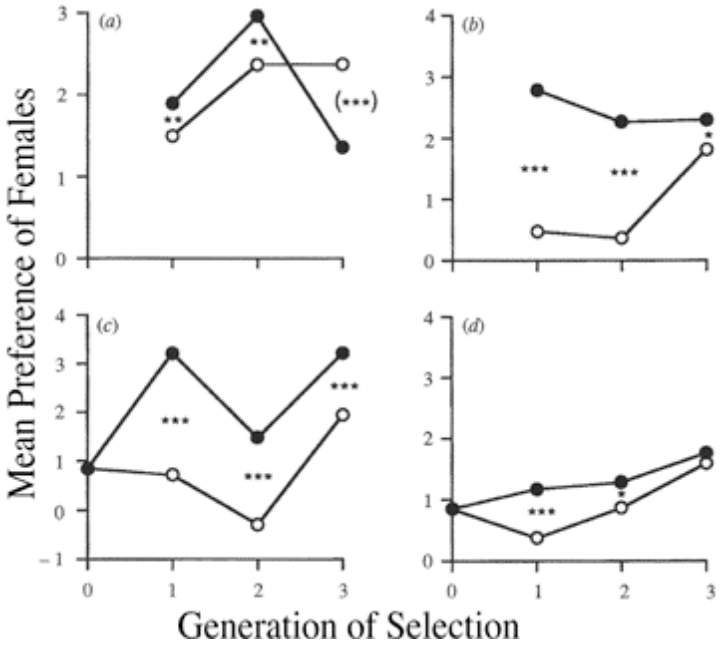


Figure 3.4. Response to 15 generations of artificial selection for large and small nests in two populations relative to control, random bred mice (Redrawn from Lynch 1980).

Correlated Artificial Selection for Female Choice and Male Color

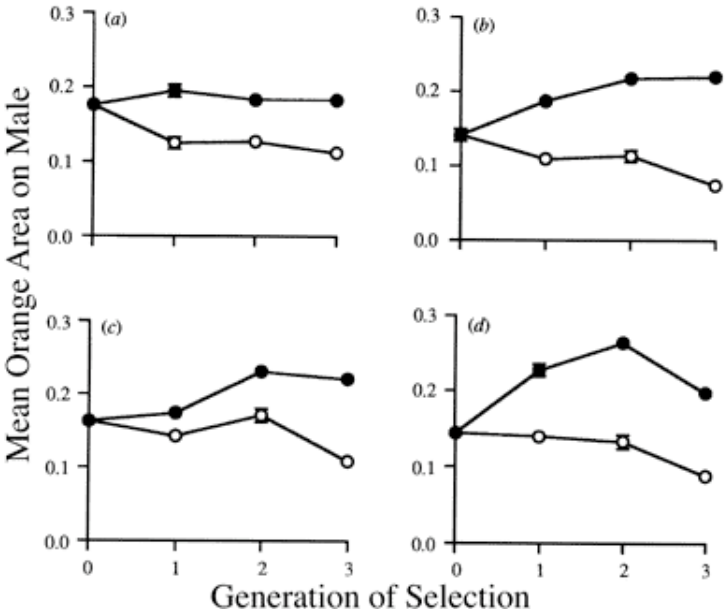
A key predictions regarding Fisherian runaway sexual selection is that genes for male coloration should become genetically correlated or linked to genes for female preference in natural populations. Anne Houde (1994) tested for the presence of a genetic correlation between male and female guppies, *Poecilia reticulata*. Houde used field collected males and females for her experiments and set up a high-selected line in which males with large amounts of orange were chosen as breeders for the next generation and she also set up a complementary low-selected line. Many of the genes for orange coloration appear to be Y-linked in guppies. Houde also scored the preference of females for males of varying orange coloration. She calculated the fraction of courtship displays by males that elicited a sexual response in the female.

Figure 3.5. Change in orange coloration of male guppies during three generations of artificial selection for high (filled circles) and low (open circles) amounts of orange. Differences between high and low lines are significant after the first generation.



Houde was successful in changing female preference by artificial selection on male coloration. While the differences between high and low male lines is maintained after three generations, the correlated changes in female preference that appear in the first generation appear to decay by the third generation in some of the lines. A parsimonious explanation for these patterns is that selection has eroded additive genetic variation in both male color and female preference. Coloration alleles that have become fixed by selection in males reduces the heritability for male coloration. A similar effect might be seen in female preference. However, a genetic correlation between male and female preference requires that male preference is heritable. In the absence of heritable variation in the male trait, the tug of the male trait on the female preference (due to the genetic correlation) would be attenuated.

Figure 3.6. Correlated change in mean preference of female guppies during three generations of artificial selection for high (filled circles) and low (open circles) amounts of orange in male guppies. Significant differences (** P <0.01, *** P<0.001) between high and low lines occur after the first generation. The difference appears to decay by the 3rd generation suggesting that the genetic correlation is breaking down.



Under conditions of artificial selection, heritability of male color should be reduced. While this may be an artifact of laboratory selection, large population size in nature may maintain high levels of heritability in the face of sexual selection. Houde's results demonstrate the existence of a genetic correlation between male traits and female preference. Similar correlations have been documented for sticklebacks (Bakker 1993) from correlations among relatives (e.g., sons and daughters), and from artificial selection on eye stalk length in male stalk-eyed flies and correlated changes in female preference (Wilkison and Reillo 1994).

In the chapters on sexual selection and mate choice, I will discuss many alternative models of sexual selection that have been developed over the years (e.g., collectively termed good genes hypotheses). Each of these models predicts that genes for female choice and male traits should be correlated. Even though her experiment does not discriminate between models, it is a splendid example of how correlations between behavioral and morphological traits can be elucidated with an artificial selection experiment.

Visualizing Natural and Sexual Selection in the Wild

While artificial selection is useful for documenting the genetic causes of behaviors and correlations between behavior and other traits, adaptational hypotheses are best tested in nature on animals that experience the force of natural and sexual selection. In his treatise on the *Natural Selection in the Wild*, John Endler (1986) considered natural selection to be a process that is inseparable from the genetic transmission of the successful traits. This would make the detection of natural selection a very difficult task because of the huge sample sizes required to assess the genetic contribution to behavioral traits (see chapter 2), particularly when combined with the large sample sizes required to measure selection on a phenotypic trait.

In recent years, other researchers have suggested that it might be informative to measure selection on the phenotype independent of the genetic transmission of the trait (Lande and Arnold, 1983). This simplification allows the researcher to focus on the measurement of the behavioral and morphological traits rather than worry about the details of genetics. Effort is focused on the assessment of how individuals survive and reproduce as a function of phenotype. Equations similar to the response to selection (Equation 2.3) can be used in conjunction with independently derived estimates of heritability such as those obtained by pedigree analysis or laboratory crosses (a generalized form of the equation for a selection differential is provided in Appendix 2). However, given the importance of environmental factors in confounding heritability estimates, the heritability of a behavioral trait measured in the laboratory may be quite different from heritability measured in natural populations, given that environments differ between the laboratory and field. In practice, however, when estimates are available from both the field and laboratory, the estimates are quite similar (Weigensberg and Roff, 1996).

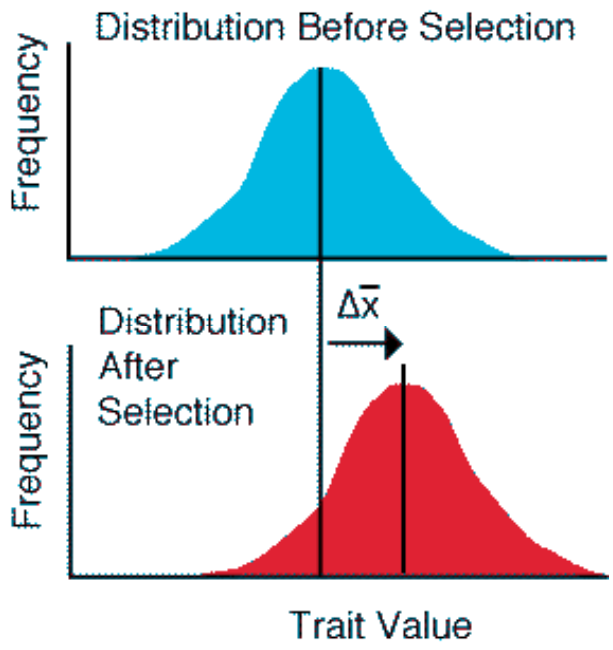
Despite these limitations, the combination of laboratory and field studies allows behaviorists to predict the evolutionary trajectory that a future population might experience, if the intensity of selection and the heritability remain constant (Lande and Arnold, 1983). If directional selection is maintained in the long term over many generations, it will lead to a change in the traits over time. This is true even if the magnitude of the selection differential is very small. All that is required is the presence of additive genetic variation in the trait over time, so that selection changes the population's mean. In the evolutionary long term, mutation (see Chapter 2) provides a constant input of new additive genetic variation.

Directional Selection

Directional selection is the easiest mode of selection to visualize. Directional selection is the natural analogue of artificial selection, which is practiced in the laboratory or cultivated field. We can compare the mean of the phenotypic frequency distribution before and after a selective episode (e.g., mortality or reproduction). If we see a shift in the mean for a trait (either up or down), a trait is under directional selection.

Directional selection also has a unique effect on the population -- it leads to an evolutionary response to selection that changes the mean of the trait from one generation to the next if the trait has a heritable component. Change in the mean across generations reflects the pattern of evolutionary change. What processes underlie this change. How do we relate change in distribution before and after selection to the process of natural selection? A simple statistical comparison of the covariation between a trait and fitness reveals the causal force of natural selection.

Figure 3.7. The action of directional selection on phenotype.



Episodes of selection: Reproductive Success of Male Bull Frogs

The lifetime of an organism can be partitioned into periods or events that describe episodes of selection. Selection rarely occurs in one single event. Selection can act at the various stages of an individual's life history, some selection episodes shape lifetime fitness more strongly than others. Natural selection typically acts via a few key components of the life history such as offspring size, juvenile mortality, time to maturity, fecundity, and patterns of adult mortality. Sexual selection acts via variance in the number of mates. In the examples of natural and sexual selection given below, we will see how selection acts on each of these life history traits. Understanding natural and sexual selection entails visualizing the relationship between a phenotypic trait and one of these components of the life history.

A linear regression describes directional selection and provides the best-fit linear relationship between the trait being selected and the measure of fitness (survival, number of mates, etc.). For traits such as number of mates, it is easy to understand how the linear relationship can be used as a predictor of fitness for a given trait. However, for traits like survival which take on fitness values of either 0 (dead) or 1 (alive), the linear regression describes how the probability of survival varies as a function of the trait of interest (see Appendix 1).

For example, Howard (1979) collected data on territorial male bullfrogs. His data was also re-analyzed by Arnold and Wade (1984) (Fig. 3.4.A.). Male bullfrogs congregate at the edges of ponds and call to attract to mates (Fig. 3.6). Howard measured the body size of adult males and assessed how many mates a male obtained as a function of his size. This would comprise one component of sexual selection that acts on male body size. Male bullfrogs also defend a stretch of shore along which females will deposit their eggs. In this arrangement, the male provides some paternal care in the form of protection from predators. Males of many other species of frogs even provide protection to the school of hatched tadpoles. The breeding season of frogs can be partitioned into relatively discrete episodes: 1) mate acquisition, 2) egg laying, and 3) egg development. The metamorphosed juveniles disperse and take several years to mature, at which point they will breed. A complete life cycle consists of development from egg to egg. While Howard's data set is remarkably useful for analyzing the activities around the pond, aspects such as juvenile growth and maturation are not amenable to measurement and thus, some natural selection is difficult to measure. The juveniles grow in the forest and are much more difficult to study.

With these caveats in mind, the fitness regression at each episode in the pond can be characterized. The fitness of the males at the pond is calculated as the product of fitness at each episodes. We use the symbol **W** to represent fitness as a **function** of the phenotypic trait. Howard measured the relationship between fitness and male body size at three episodes, all of which are positively related to fitness (Figure 3.8):

W(fitness at pond)=	W1(size)	×	W2(size)	×	W3(size)	*Eqn 2.3*
W(bullfrog male) =	Number of mates	×	Fecundity of mate	×	Survival of offspring	

The net result from this attempt to estimate the fitness of male bullfrogs suggests that larger size should be favored over evolutionary time. However, it is unclear whether all selection was assessed. Other episodes of selection may favor small body size and this selection could counterbalance the selection that favors large size. These effects were not measured in his study. Moreover, it is unclear whether the variation in male body size has a genetic basis. Much of the variation could be due to age, or perhaps to purely environmental sources of variation. Finally, the snap shot of a single season that was captured by Howard may not be representative of selection in the long term.

Setting aside these limitations, the selection revealed by Howard's data presents an interesting place to start asking more proximate questions regarding the size advantage of large bullfrogs. Why do larger bullfrogs get more mates? What kind of selection might be acting on choosy females? These questions will be answered in later chapters on female choice and communication. A short answer to these questions is that, larger frogs make deeper calls, that are more attractive to females. Elucidation of the proximate causes or mechanisms underlying sound production by males and sound reception by females will be explained in Chapter 13, Sensory Systems and Communication.

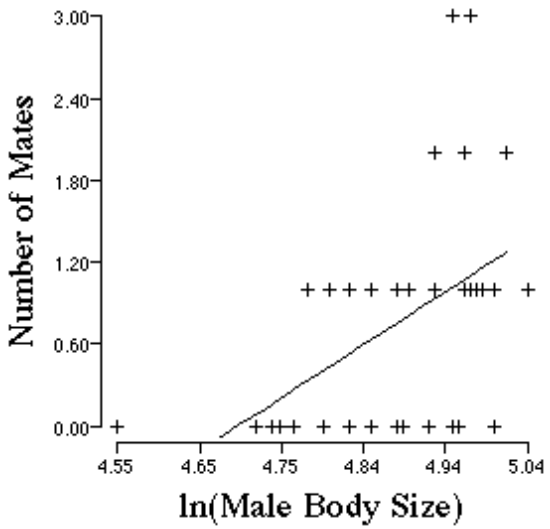


Figure 3.8. A. Episodes of selection in male bullfrogs. (Arnold and Wade 1984, data from Howard 1979). The first event (W1) is **mating**. Fitness was defined as number of females with which a male mated. The regression line is significant (P<0.05). We would expect chance alone to produce this strong a relationship less than 1 time in 20.

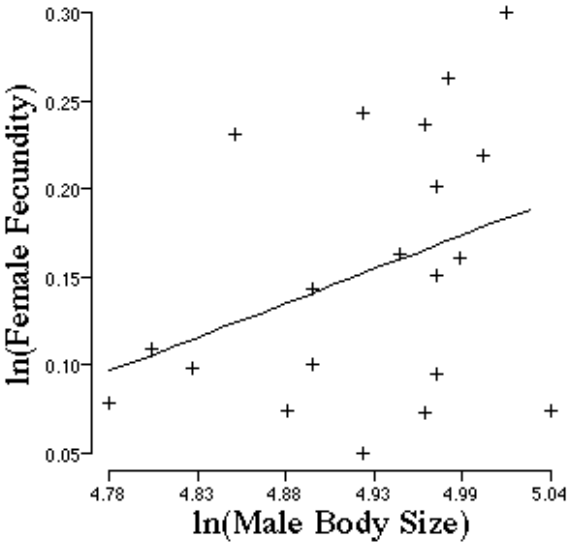


Figure 3.8.B. The second component was the **fecundity** of the females copulated by the male, or number of eggs laid by each female on his territory (W2, in this case, data values should be multiplied by ln(10000) to compute total eggs, the y-axis has been scaled by 10^{-5}). Notice, that this relationship is much weaker than W1. This relationship is not statistically significant (P>0.05), which means that a pattern as strong as the one observed would occur more frequently than 1 in 20.

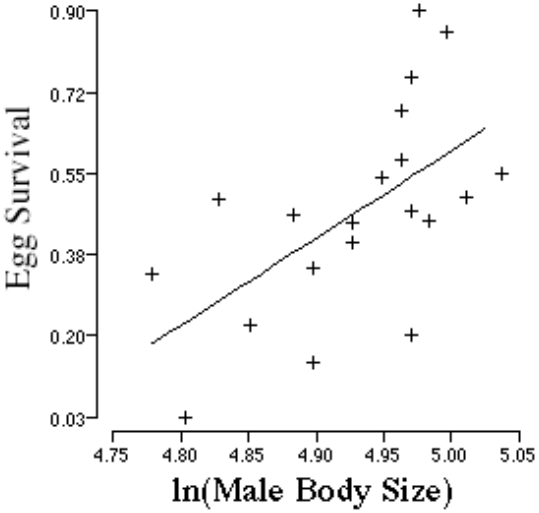


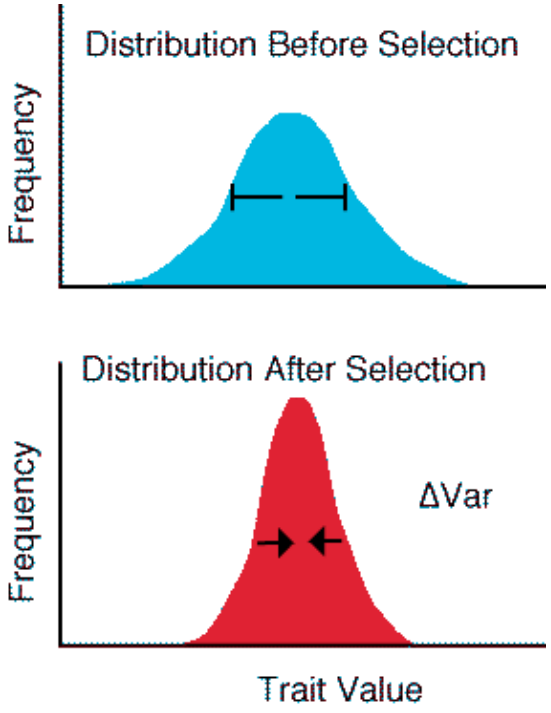
Figure 3.8.C. The final component was **probability of offspring survival**, or success with which eggs hatched into froglets (W3). Bullfrog dads may have good or bad territories from the point of view of hatching success. This territory quality might vary as a function of male body size. The regression line is significant (P<0.05).

The example of bullfrog territories exemplifies the concept of the **extended phenotype** (Dawkins 1976). The territory is not a phenotypic attribute of the male's body *per se*, but it is just as important for sexual selection as a physical attribute. A territory arises from many physical attributes related to aggression, discussed in Chapter 9, which females find attractive from the perspective of the survival of their progeny, a subject discussed in Chapter 10, Mate Choice and Sexual Selection.

Stabilizing Selection

Stabilizing selection tends to reduce the amount of variation in the phenotype distribution over an episode of selection. Individuals in the center of the phenotype distribution tend to be favored and have higher fitness than individuals in the "tails" of the distribution. A simple form of stabilizing selection might act on a single locus with two alleles where overdominant heterozygous individuals have an advantage relative to the two homozygous classes (see Side Box 2.1). If the heterozygotes have higher fitness, selection would tend to remove the extreme homozygous classes. Such traits are however, more often polygenic and genetic variation arises from many loci.

Figure 3.9. The phenotype can be stabilized by selection.



In contrast to the response to selection under directional selection, pure stabilizing selection does not change the mean of the phenotype distribution. Stabilizing selection reduces variation and favors individuals with an average phenotype over the extremes. This mode of selection is often referred to as optimizing selection. To describe a fitness surface in terms of stabilizing selection we need a quadratic or second order polynomial equation:

$$W = a + b \times x + c \times x^2 \qquad \text{Equation 3.1,}$$

where x is the value of the phenotypic trait and W is fitness, a, b, and c are three parameters of the quadratic equation). Quadratic regression is only a slightly more complicated form of linear regression, which is described in Appendix 1. Notice that the term $b \times x$ is the directional component. It is entirely possible to observed directional and stabilizing selection on a trait at the same time.

The examples of stabilizing selection that we will explore involve fundamental issues of life history as it relates to parental investment. The amount of parental care is a classic behavioral trait analyzed by behavioral ecologists in the wild. We will consider stabilizing selection on offspring size and offspring number in a vertebrate without parental care (a lizard), and two vertebrates with extended parental (humans and birds). In this exploration of selection and the life history, we will explore the concept of **life history trade-offs**. The notion of life history trade-offs is tightly related to the concept of **antagonistic pleiotropy** discussed above in the context of artificial selection experiments. These life history concepts are central to parental investment models that we will explore in subsequent chapters. As we shall see in the chapters on mating system and parental care, offspring number is a central trait influencing not only fitness of the parent (e.g., more offspring means more fitness), but also the amount of parental effort in the form of behavior and energy that could influence the parent's survival.

The examples discussed below illustrate how counterbalancing selection can constrain reproductive traits and result in the process of adaptation and lead to optimal combinations of traits. Trait **optimality** is one of the cornerstone concepts of the adaptational perspective. Classic paradigms developed by Lack (1947; 1954) and Williams (1966) provide the selective context for understanding the evolution of parental investment. David Lack (1947) is best known for his hypothesis that avian clutch

size was limited by the amount of care that parents could provide to offspring. The selective premium accrued by investing in additional offspring would be counter-balanced by the decreased fledgling success in large clutches owing to limitations on parental effort per offspring. The most successful nests would be those that produced an intermediate clutch size. Clutch size in birds should be under stabilizing or optimizing selection. This process reflects adaptation of the life history and behavioral traits that contribute to fitness.

Similarly, in organisms without parental care, Lack (1954) further suggested that production of large numbers of small offspring was balanced by the high survival of a few large offspring (Sinervo et al, 1992). A second paradigm of life history biology, **costs of reproductive allocation**, provides the context for understanding selection during adult phases of the life history. Williams (1966) refined Lack's ideas and considered parental costs of reproduction that arise from further investment in energy per offspring to offset deficiencies in offspring survival. Parents might have to forage further, or for longer periods of time during the day in order to satisfy the demands of the growing brood. Investment in current reproduction is expected to result in a cost to future reproductive success that should either lower survival of the parent, lower fecundity, or reduce growth rate and consequently affect body size and fecundity. Lack (1954) developed manipulations of clutch size to test for the causes of selection on parental effort and care.

Stabilizing Selection on Clutch Size in Birds

For over a decade, Lars Gustaffson and his colleagues have studied Lack's and William's hypotheses in the collared flycatcher, *Ficedula albicollis*, on the island of Gotland which is south of the Swedish mainland. Each spring they monitor a vast array of nest boxes. They tag each male and female and record the number of offspring that the female lays. They augment these studies of natural variation with experimental manipulations of clutch size (N = 320 manipulated nests). Results from their studies provide support both Lack's and Williams hypotheses.

Gustaffson and Sutherland (1988) found that the number of recruits produced by unmanipulated nests was higher than the number of recruits with eggs that were removed from or added to the nest. Parents that were induced to rear enlarged clutches produced lower quality offspring that had lower survival to maturity. In contrast, parents that were induced to produce smaller clutches could handle more offspring, but the number of recruits that they produced was reduced because of their initial smaller clutch size. Unmanipulated clutches also had the optimum number of offspring. In addition to observing stabilizing selection on the number of fledglings produced, they also found that the quality of the offspring at maturity was affected by the clutch size manipulation. In the next season when the female birds matured, if they had come from intermediate sized nests they produced the most fledglings in their own nests. Mature recruits produced fewer offspring at maturity if they came from nests with altered large clutch size. Lack's hypothesis concerning an optimal clutch size apparently holds for the collared flycatcher.

Finally, they also found that female parent's produced fewer eggs the next season if eggs were added to their nest. They produced more eggs the next season if they had eggs removed their nest. Thus, investment in a large clutch in one season results in a cost of reproduction that reduces the clutch size that the female parent produces the next season.

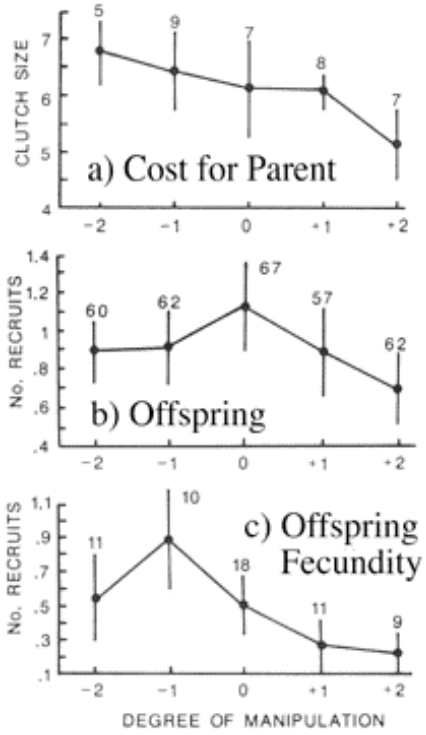


Figure. 3.10 Lack's tradeoff between offspring number and quality and William's tradeoff between current and future reproduction appear to operate in collared flycatchers. The effect of clutch size manipulation in collared flycatchers, *Ficedula albicollis* on a) fecundity of the female parent the next season, b) number of recruits in a nest that return to the breeding grounds the next spring, c) fecundity of the recruits when they return to breed. While selection on costs of reproduction that the female experiences in terms of reduced fecundity tends to favor the production of small clutches, the selection on offspring survival to recruitment and the number of offspring that offspring produce at maturity are both under significant stabilizing selection. From Gustaffson and Sutherland (1988).

Stabilizing Selection on Maternal Investment

Our discussion of parental investment began with birds, animals that are fairly advanced in terms of the duration of parental care. In phylogenetic terms, we consider parental care of eggs or juveniles to be a more derived condition, and lack of parental care to be the ancestral condition. Species with parental care evolved from an ancestor that presumably did not have advanced parental care. By examining organisms that typify the ancestral and derived states for parental care, we can see if there are common features of the life history that are subject to the action of stabilizing natural selection. These aspects of life history involve the fundamental selective constraints originally considered by Lack and Williams.

Many egg-laying reptiles have little or no parental care after laying their eggs. The side-blotched lizard is a useful system for investigating how parental investment influences offspring survival. Quantifying parental investment in egg-laying animals without parental care entails a measurement of energy content of egg production. Because the amount or mass of yolk in an egg reflects most of the direct energy invested by the female in her young, we can measure survival of the adult female and survival of her offspring as a function of egg mass to determine whether there is a net stabilizing selection on offspring size.

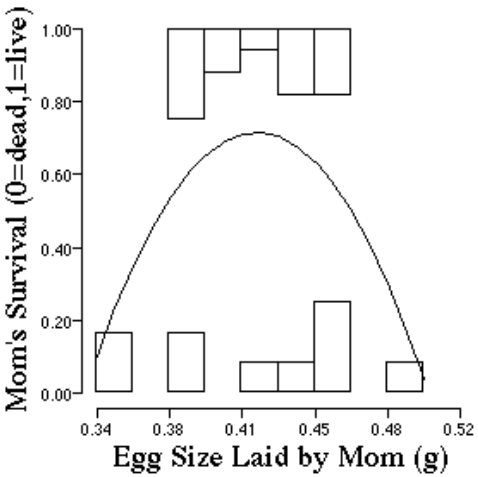


Figure 3.11. Survival of adult female lizards in nature as a function of investment egg size of individual offspring. The fitness of a female was either 0 (she died) or 1 (she survived). The histograms at the top of the graph shows the relative distribution of egg sizes laid by female parents that survived to produce another clutch of eggs. The histogram at the bottom that shows the distribution of those females that died shortly after laying their eggs. The curve describes the fitness surface for the probability of adult female survival as a function of the egg size that she laid.

In collaboration with a number of colleagues (Sinervo and Licht, 1991; Sinervo et al 1992; Sinervo and Doughty 1996), I have collected data on the survival of female lizards as a function of the quantity of yolk that a female parent puts into the egg. I measured the survival of these female parents to the production of summer clutches (Sinervo, 1994). One of the factors thought to influence patterns of adult mortality is related to **costs of reproduction**. If reproduction is costly, then heavy investment in current reproduction might be expected to lower survival or future reproductive success. From the fitness surface, which describes female survival as a function of her investment in individual offspring, it is clear that the distribution of survivors is much narrower than the distribution of females that died. Females that laid extremely large or small eggs had relatively low survival and selection was stabilizing. It appears that both production of very large eggs and very small eggs is costly in terms of survival.

Why should the production of small eggs be a liability? In these lizards females that produce small eggs also tend to lay many eggs or have large fecundity. In a separate series of experiments, Sinervo and DeNardo (1996) increased the clutch size of females and reduced egg mass, by implanting a pellet that released follicle stimulating hormone. Females with experimentally increased clutch size (and smaller eggs) had a reduced survival.

Natural selection favored females that laid intermediate-sized eggs. Why should the production of large eggs be a liability? Using the same technique of experimental clutch-size reduction, Sinervo and Licht reduced clutch size down to the smallest possible, a single egg. Females with reduced clutches experienced a slightly different problem in that they became egg bound at high frequency, and required a cesarean section to remove the obstructing eggs that were far too large to lay. Congdon and Gibbons (1987) have suggested that similar constraints limit adaptive evolution of clutch size and offspring size of turtles. All vertebrates must pass their eggs through an opening in the pelvic girdle.

These studies illustrate two points. First, there is an optimal offspring size that the female can produce. Second, experimentally altering the phenotype elucidates the causes of natural selection. In this case the optimizing selection on a female parent's survival results from two separate causes: too many eggs in a clutch, which may impact energetic

costs of reproduction or individual eggs that are too large for the pelvic girdle. Each of cause reflects a constraint of some kind.

Survival of adult females reflects a single episode of selection. Sinervo et al. (1992) show that there is also an optimal offspring size that ensures a juvenile lizard's survival to maturity. In this case, the optimal offspring size arises from a classic life history trade-off: fecundity selection that favors the production of small offspring is balanced by the survival selection that favors the survival of large offspring to maturity. Stabilizing selection on offspring size occurs during a number of separate life history episodes. Selection on maternal investment should have a genetic basis if the trait is to respond to natural selection. Indeed, egg size of the mother is positively correlated with egg size of a mature daughter. Given the results on heritability from this two year study we have also used selection estimates measured across additional years to correctly predict the response to selection on egg size (Sinervo 2000).

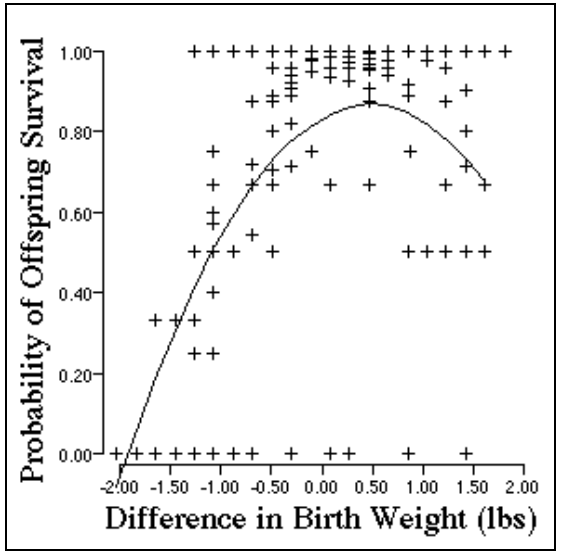
Selection on Human Birth Weight

In light of the constraints on maximum offspring size that arise from the pelvic girdle of lizards, we are prompted to ask whether such constraints operate on other vertebrates. Let's look at a problem more close to home. Humans invest an inordinate amount of energy into their young before they are born, as well as a large amount in extended parental care. The duration of parental care in humans after birth can last more than two decades. There is not much data to inform us how human parental investment after birth leads to offspring success. However, data is available on offspring size and problems during birth. The parturition problems experienced by lizards are a very general problem for all vertebrates that produce relatively large offspring. The problem with offspring size in humans is exacerbated by the large size of the cranium of the newborn relative to the size of the birth canal, which runs through the pelvic girdle.

Karn and Penrose (1958) collected data from hospitals on survival of offspring as a function of neonate size, and the duration of gestation (Schluter, 1988). Length of gestation confounds neonate size. Premature babies are usually smaller than babies born at full term. Thus, neonate size is expressed as the difference in size from the population mean, after removing the effect of factors such as length of gestation.

Two patterns emerge from the analysis of selection on human birth weight. First, there is significant stabilizing selection on neonate size. Small infants and large infants die during childbirth at a higher rate than average-sized infants. Second, there is also a directional component to selection. Notice that the optimal infant size is one-half of a pound higher than the average infant size in the population. The pattern of low survival of large offspring undoubtedly has different causes than the probability of low survival of small offspring. For example, small offspring may have had high mortality because of inadequate nutrition during gestation. Conversely, large offspring may have high mortality because of the large diameter of the cranium relative to the pelvic girdle and its effects on duration and difficulty of labor for the mother.

Figure 3.12. Probability of human infant survival after birth is affected by neonate size. Original data is from Karn and Penrose and reanalyzed by Schluter (1988). Data are standardized relative to average infant size at birth, which is located at 0.



The data that Karn and Penrose collected back in 1958 were collected at a time before the advent of modern techniques for neonate care. It would be interesting to know if the widespread use of cesarean sections and other medical techniques have altered selection on neonate size.

Additional data on the efficacy of techniques of intervention would provide experiments of a sort that could be used to pinpoint the causes of stabilizing selection, much like the techniques used in female lizards. Experimentation on humans cannot be conducted, but reference or control populations could be compared to observations of other populations that received different kinds of pre- or post-parturition treatment.

The evolution of litter size in primates

We have seen that the human species is subject to stabilizing selection for offspring size. In addition, the simple interpretation of the difference in optimum offspring size relative to average offspring size (0.5 lbs) would suggest that humans are also under some additional directional selection for large offspring size.

Has a functional ceiling in offspring size been reached in humans? Does the pelvic girdle limit the size and number of offspring in other primates? Humans and primates have a shared evolutionary history and limitations on parental investment might have constrained other primates during the evolutionary history of the group. This issue briefly touches upon the kind of historical and design constraints that Gould and Lewontin (1978) suggested are important to the process of evolution and natural selection.

Karn and Penrose's data reflects over 35,000 individual births. Is such data available on other primates? It would be incredibly difficult to collect this kind of data on free-ranging chimpanzees because we would have to census all existing chimpanzees found in nature. The data on mountain gorillas would be even more difficult to collect because this species has been dwindling in population size over recent years due to habitat fragmentation. Is there any other kind of data available on offspring size or litter size of primates that we could use instead of actual data on natural selection that describes survival or reproductive success in terms as a function of some trait?

There is a wealth of comparative data on neonate size, body size and litter size of primates that have been used to construct hypotheses on adaptation and constraint during the evolution of primate reproduction. The **comparative method** has a rich history in evolutionary and behavioral studies (Harvey and Pagel, 1991). In the comparative method, data is gathered on a large number of related species, the patterns for the species are graphed, and inferences are made regarding the process of adaptation. While such data is not as powerful as direct assessment of the process of natural selection, it provides us with insight into past processes that might have limited evolutionary change. In addition, we cannot observe natural and sexual selection on past events. We must rely on modern day inferences of selection if we are to understand the evolution of behavior. The comparative method is essential to make these kinds of inferences about historical patterns of selection.

Luetnegger (1976, 1979) used the comparative approach to investigate whether the evolution of large cranial size in primates may have constrained their reproductive biology. Many small species of primates tend to give birth to two offspring whereas all large species of primates give birth to a single progeny. This pattern presents us with a minor paradox. If fitness is increased by the number of progeny, and if a larger animal should be able to produce more offspring, then why don't large primates produce more than a single offspring?

The answer to this problem is certainly more complicated than fecundity. Perhaps it is important for larger primates to invest more in a single offspring given their ecology. If population size of primates is relatively stable or close to the carrying capacity of the ecosystem, a female from a large-bodied species might be expected to produce one very well provisioned offspring. A species that routinely experiences the force of competition is referred to as **K-selected**. K refers to the parameter for carrying capacity in the Lotka-Volterra equation describing population growth. On the other hand, an **r-selected** species is rarely at carrying capacity and production of a lot of small offspring is advantageous. The *r* refers to the **Malthusian parameter** or intrinsic rate of growth of a population. A population that is r-selected is often in a growing phase and an individual that produces lots of offspring has an advantage compared to a population that produces only a few offspring. Each individual offspring does not have to be very well provisioned. Thus an adaptive explanation for the production of lots of small offspring in small primates and only a single offspring in large primates is that they are r-selected and K-selected respectively.

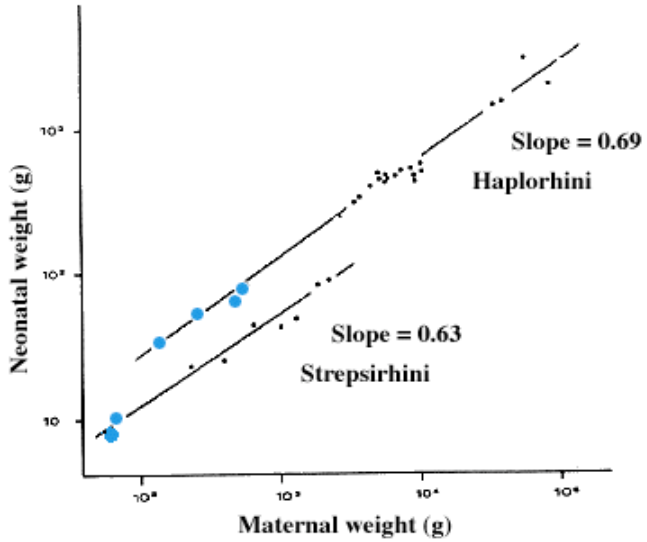
Luetenegger (1979) suggested a completely different explanation, which addressed the constraints on reproduction in primates. In the field of allometry, the relative size of various structures are compared to overall body size. In terms of primates, the evolution of large brain size has been an evolutionary trend that distinguishes *Homo sapiens* from other primates, and all of primates from other terrestrial vertebrates. Many marine mammals or cetacea have evolved relatively large brains that rival the human brain (Pagel and Harvey, 1989), but cetaceans, lacking hind limbs are freed from locomotor constraints of the pelvic girdle that are imposed on other terrestrial vertebrates. In principle, cetaceans should be less constrained in the evolution of brain size compared to humans. Human infants have relatively large heads relative to adult humans. This is an example of an allometric relationship.

To uncover allometric relationships the logarithmic function is first used to transform the x and y variables. On a log-log plot, the slope is used to infer the changes in relative size or shape as a function of body size. If a trait does not change relative to size across a range of body sizes, the slope of the line describing the relationship between body size and the trait is 1. For example, if we scale cardboard boxes up in size and plot the length of one side relative to the other, the slope on a log-log plot would be one. If we were to plot the volume of the card boxes relative to the length of one side, the slope on the log-log plot would be 3 because volume scales to the cubic power of linear dimensions. The surface area of cardboard used to construct the boxes would scale to the square of one side. The surface area divided by volume would scale to the 2/3 power. In this case surface per unit of volume does not keep up with increased size and there is relatively a smaller surface area per unit of volume in large cubes compared to small cubes.

When plotting neonate size against maternal body size of various species of primates, Luetenegger (1976) observed that neonate head size was relatively much larger for small-bodied primates compared to large-bodied primates. If the relative size of the pelvic girdle was fixed in size across these groups (i.e., pelvic girdles did not change shape relative to body size), then small-bodied primates may experience more difficulty during birth than large-bodied primates. Small-bodied primates show a much higher incidence of twinning, which is hypothesized to result from selection for more and smaller offspring. These small primates could not necessarily produce a single large offspring and twin instead.

While such hypotheses are difficult to prove without direct evidence on the fitness of a small primate that produced one versus two young, the example serves to illustrates Gould and Lewontin's point on alternative causes for evolutionary patterns. An adaptationist might speculate that small-bodied primates are selected to produce lots of small offspring because the optimal litter size is two young whereas a large primate is selected to produce a single large offspring. The argument based on architectural grounds contends that small primates are physically incapable of producing anything but two small young. These are two drastically different explanations of the same pattern -- one based on adaptational causes, the other based on constraints on organismal design.

Figure 3.13. Log-log plot for maternal weight as a function of neonate weight for two groups of primates. Notice that the slope of the relationship is nearly 2/3 or much less than 1 for each group (from Leutenegger 1979). Species that twin are highlighted in blue, the others given birth to one offspring.



When we consider animals that have a vastly different evolutionary history (amphibians, reptiles, birds, and mammals) we can draw conclusions with regards to natural selection and the life history trade-offs that involve offspring size or offspring number. While the parental investment trade-off operates on slightly different aspects of the

reproductive biology of each group, there is a common mode of selection that operates on the reproductive traits -- stabilizing or optimizing selection. Indeed many researchers have identified similar patterns in a wider array of taxa. These trade-offs can either be left unresolved and identified as simple stabilizing selection on the variance in offspring size or offspring number in the population, or the stabilizing selection can be further analyzed in terms of counterbalancing components of directional selection that act on different life history episodes. Regardless of whether or not the causes of stabilizing selection are elucidated, their impact on species is fairly clear. Stabilizing selection would act to maintain the constancy of a species over a long time frame. Moreover, if species had different optima for traits then stabilizing selection would tend to keep species differentiated.

The traits clutch size, offspring size, juvenile survival, adult survival, body size and time to maturity are the routes by which natural selection acts to shape behaviors. Lack's (1954) and Williams' (1966) principles represent the fundamental **selective constraints** that organisms experience. Any behavioral trait that is functionally related to a life history trait will be altered by the force of natural selection, if there is heritable variation for the behavior. Unfortunately, few studies of behavioral traits provide a comprehensive picture of lifetime reproductive success. Researchers can usually only get a small peek at the effect of a behavior on natural or sexual selection and the process of adaptation. In upcoming chapters we will analyze many more of these snapshots or episodes of selection that shape animal behaviors.

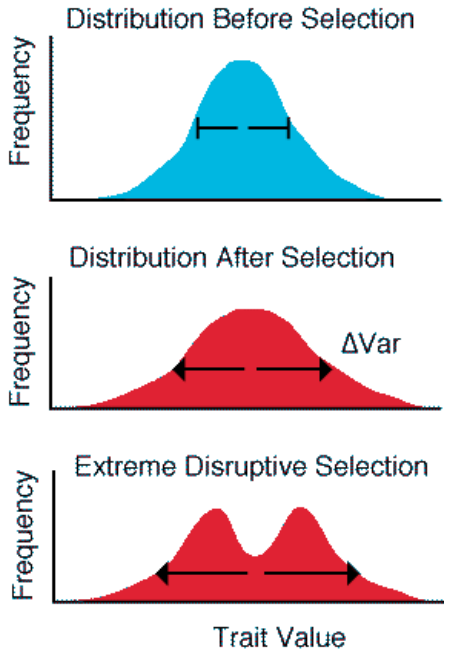
Finally, additional design constraints can limit the process of adaptation. These are **mechanistic or structural constraints** that limit organismal design (Maynard Smith et al 1985). Elucidating these design constraints, requires a deeper understanding of the proximate mechanisms of behavior, which we will uncover in future chapters.

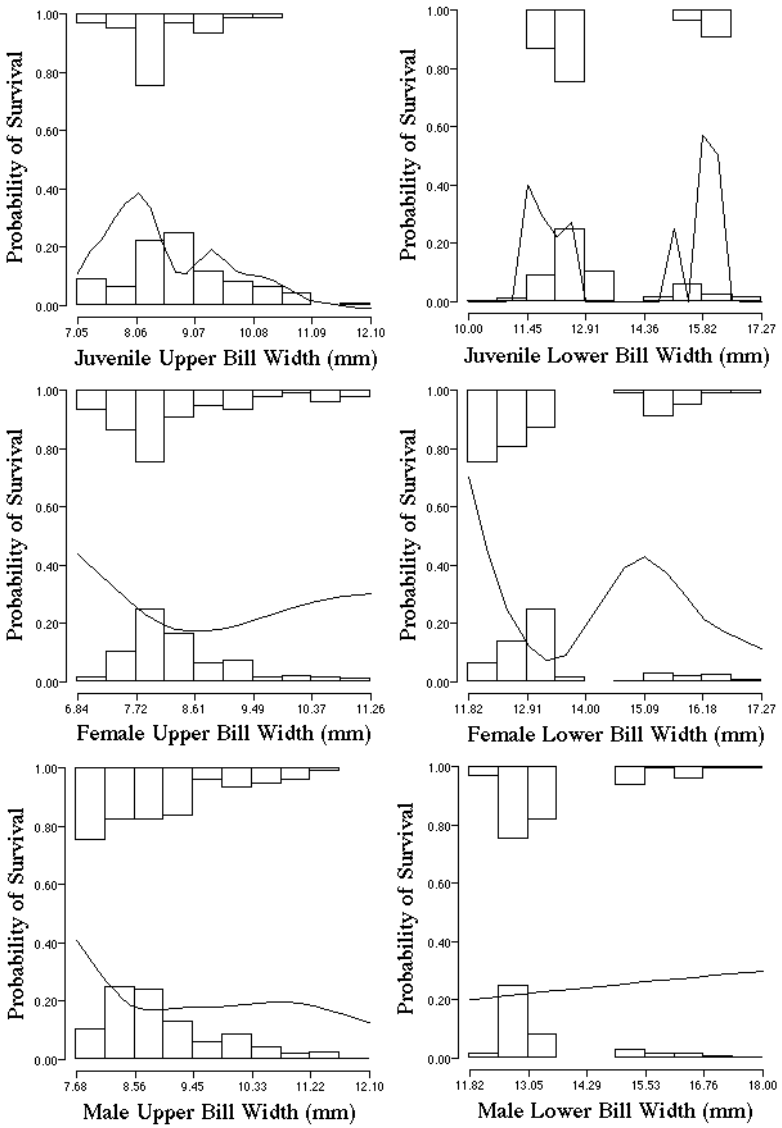
Disruptive Selection

Disruptive selection seems to be the rarest mode of selection (Kingsolver et al. 2001). Despite the scarcity of examples of disruptive selection, the process is thought to play a major role in speciation or the origin of new species (Templeton, 1982). The action of disruptive selection is more complicated than the action of directional selection in which a single agent of selection shapes a trait. Furthermore, the action of disruptive selection is likely to be more complicated than stabilizing selection which is sometimes composed of counterbalancing trade-offs. Disruptive selection favors individuals in the extremes of a phenotype distribution at the expense of those in the middle. A simple form of disruptive selection occurs on a single locus with two alleles when heterozygous individuals are at a disadvantage relative to the two homozygous classes (see Side Box 2.1, underdominance). In the case of such underdominance in fitness, selection favors homozygous classes.

If the action of disruptive selection is relatively weak it tends to increase the representation of individuals in the tails of the distribution. The distribution will appear to be more flat, with longer tails after selection compared to distribution before selection. If disruptive selection is strong, it will lead to two distinct modes that are separated by a fitness valley where selected phenotypes have been removed by selection.

Figure 3.12. The action of weak (middle panel) and strong (bottom) disruptive selection on a phenotype distribution (top).





In the long term, disruptive selection can create two distinct distributions from a single distribution. This kind of force is required for the origin of new species. Whereas directional selection can move the phenotype distribution of existing species in a different direction and stabilizing selection tends to hold the phenotype distribution in one place, disruptive selection creates new types.

Selection on bill morphs of the African Finch

Recall the pedigree analysis of the African Finch in which a simple Mendelian locus controlled bill morphology. Tom Smith (1987, 1993) has also studied juvenile and adult survival over a number of years to measure natural selection on morphology and feeding behavior of the finches. In the case of the Finches, the trait that is under selection is not a polygenic trait. Beak size is a relatively discrete Mendelian trait. However, there is still variation about each of the modes. The causes of such variation could be environmental, or perhaps due to the additive genetic effect of other loci which have much smaller effects on morphology and feeding behavior.

From Smith's data set on the survival of birds as a function of upper and lower bill width's it is clear that disruptive selection tends to eliminate the intermediate-sized birds in the population. However, selection also tends to eliminate the extreme tails of the distribution in at some age and sex classes (e.g., juveniles and adult females Fig. 3.13, top and middle panels respectively). Finally, selection on adult males appears to be much weaker than selection on adult females (bottom panels).

Given the simple Mendelian inheritance for beak size, it is clear that disruptive selection tends to maintain two distinct bill morphs by eliminating birds with intermediate-sized bills. Moreover, selection for the most extreme (e.g., largest and smallest) individuals in the population would tend to keep the bill size of African finches within a species typical range. Natural selection on beak size in seed cracking finches can be traced directly to feeding performance of the two morphs

Figure 3.13.A-F. Disruptive selection on bill morphs of the African Finch, *Pyrenestes ostrinus*. The fitness or survival of juveniles, adult females, and males was either 0 (dead) or 1 (survived) across one year. The histograms at the top of the graph gives the distribution of beak sizes for individuals that survived, relative to the histogram at the bottom, which gives the distribution of those individuals that died. The curve describes the probability of survival as a function beak size. Individuals with intermediate sized beaks sit in a fitness valley and have lower fitness than individuals with large or small beaks. While the pattern of disruptive selection is common, it is not observed to act simultaneously on both the upper and lower bills or for all age and sex classes.

on different sized seeds. The behavioral differences between morphs with regards to their feeding preferences can be traced directly to their survival in the wild. Both modes experience disruptive selection which refines the differences between morphs. In addition, each bill morph also experiences a form of optimizing selection, at least in juveniles and adult females. Tom Smith (1987, 1993) has captured a snapshot of the process of adaptation. Finch morphs have different seed preferences because it is highly adaptive for survival.

Selection on Correlated Characters

We cannot consider behavioral traits in isolation from the other morphological and physiological traits that make up what is referred to as the "**whole-organism**". Consider the selection on the upper and lower finch bills. The form of natural selection on males and female finches appears to be different, yet, males and females have very similar beaks. Why is this the case? Is it because a single gene controls the size of both the upper and lower beaks? Alternatively, is it because the action of the upper and lower bills must be functionally integrated and a bird with great disparity between upper and lower bills is selected against?

The analysis of selection on single traits, which is presented above (even the analysis of single traits during successive episodes), simplifies the action of natural selection. Natural selection usually acts simultaneously on many traits. If these traits are truly independent and natural selection on one trait does not affect natural selection on other trait, we can use the simple equations for response to selection on each trait to predict evolutionary change, $R=h^2s$.

However, if the traits that are under selection are joined by a common genetic cause such as pleiotropy, selection is interdependent. In this case, the correlation between the traits arises because of a mechanistic coupling that is due to a common genetic cause -- one gene controls the expression of two or more traits. When selection acts to change one trait, the other trait will change because the genetic response to selection involves the same genetic locus. When the response to artificial selection begins to level off (e.g., see selection on low nest size in mice), the hypothesis of **antagonistic pleiotropy** posits that the action of artificial selection influences not just nest size, but other traits such as pup mortality in those nest with too little material to keep the pups warm. In addition to the explanation of pleiotropy, genes that are physically linked or adjacent to one another on the chromosome behave as if they are one gene. While pleiotropy forms a relatively permanent coupling between traits, physical linkage between alleles that are located on different chromosomes can decay away owing to segregation (see Side Box 2.1). Alleles at different loci that are coupled by selection can become uncoupled by recombination between the two loci.

While natural selection on a single trait can simultaneously move two or more traits that are linked by pleiotropy or physical linkage, the reverse can also be true. Correlated natural selection can act on two or more traits that are functional related to produce suites of integrated traits. We have already seen how assortative mating can link genes for female choice and genes for male morphology during the process of runaway sexual selection (see Side Box 3.1). Natural selection can achieve a similar effect. The different origins for such correlations between traits which are collectively referred to as genetic correlations are addressed in the following examples of functional integration between behavior, physiology, and morphology. If traits are genetically correlated, then they tend to be jointly inherited. A genetic correlation can arise from two sources: **linkage** and **pleiotropy** (Falconer 1981). The role of linkage, the physical proximity of genes on a chromosome, in promoting genetic correlations are best studied with molecular tools, classical genetic crosses, and linkage studies. However, pleiotropy can be studied by manipulating a common endocrine mechanism that links traits. Life history trade-offs should be reflected in a negative genetic correlation between fitness traits. If proximate endocrine mechanisms form the basis of life history trade-offs then manipulations of the endocrine system should induce negative correlations between components of the life history that influence survival or reproductive success (Marler and Moore 1988; Ketterson et al. 1991, 1992; Sinervo and Licht 1991; Sinervo and DeNardo 1996).

Genetic correlations are derived from breeding studies or artificial selection experiments (Falconer 1981), and because statistical power and control over the environment are important concerns, genetic variation is usually measured under artificial, not natural conditions. Only in exceptional cases where long-term studies have acquired a large dataset is it possible to estimate genetic correlations in natural populations with

reasonably narrow confidence limits. It is not just for these pragmatic reasons that field biologists have resorted to endocrine manipulations as an experimental source of inference for trade-offs. Manipulations allow for cause-and-effect relations to be derived for natural selection on life history trade-offs (Sinervo and Basolo 1996; Sinervo and DeNardo 1996; Sinervo and Doughty 1996). Endocrine manipulations should alter allocation trade-offs by mechanisms similar to those initiated by genes.

Testosterone and trade-offs between Polygyny and Parent Care

Ketterson and her colleagues have used techniques of phenotypic engineering to change testosterone level in parental juncos and study associated life history trade-offs. Behavioral traits that lead to male and female parental care in birds are also under the influence of sexual selection. From the point of male fitness, investing energy in the rearing young represents one route by which a male might enhance his fitness. A male may increase his fitness by obtaining additional copulations from neighboring females. However, these females already have mates, and the male must either: usurp the neighboring male's territory, drive the other male out, or carry out other behaviors that might attract the neighbor's females. The tendency for birds to maintain long-term relationships with a single partner is referred to as monogamy. The tendency male to have more than a single mate is referred to as polygyny. The trade-off between monogamy and polygyny in birds arises from a simple hormonal differences among males that governs male behavior and aggression -- secretion of the sex steroid testosterone.

Testosterone is linked to aggression and territorial defense in a large number of vertebrates including birds. In addition, levels of testosterone in adult birds are also correlated with levels of parental care and the tendency to monogamy or polygamy. Species of birds that maintain high levels of testosterone during the entire reproductive season also tend to be quite polygamous. By maintaining high levels of T, these birds keep singing and courting additional females. These males may not the best parents however, as they usually leave the female to rear the young by herself (Wingfield et al. 1990).

To investigate these correlational pattern, Ketterson and her colleagues (reviewed in Ketterson and Nolan 1991,1992) picked a largely monogamous species, the dark-eyed Junco, in which males and females provide parental care. They implanted half of the males with T, and the other half with sham implants. As predicted, the T-implanted males sang more, ranged farther, and tried to court more females, all at the expense of parental care. The females of T-implanted males were left to carry the burden of care that normally is split between the male and the female. In the long run, such decreased care on the part of the male might be expected to cause the female to work harder and perhaps experience greater costs of reproduction. Ketterson and her colleagues found no evidence that the female parents had reduced survivorship.

If females do not show reduced fecundity, then why aren't T-levels higher in dark-eyed Junco males. T-implanted males did get more copulations than their sham-manipulated counterparts? Even though apparent success of T-implanted males was high, were they really more successful in siring young? To answer this question they turned into genetic sleuths and used a series of genetic probes, much in the same way that Lank et al (1995) determined paternity of ruffs. The probes that Ketterson et al used did not have the resolution determine the sire with certainty (Ketterson et al 2000). This is because of the large number of potential sires that could be the father in bird, which have high vagility. Birds have the potential for very long-range movement, and an actual sire might have come from off of their study site, where they did not sample tissues for genetic analyses.

Ketterson et al. (2000) did have resolution to exclude the most likely putative sire, her territorial partner, as the sire of her progeny. Their results were most informative with regards to success of T-implanted males. T-implanted males were more likely to sire additional offspring with females on their male neighbor's territory. These gains from what are referred to as **extra pair copulations** were nearly exactly offset by fitness losses to neighbors, which sired offspring with females on the T-implanted male's territory. In contrast, sham-implanted males were more likely to be the sole sire of the females on their territory and they experienced fewer fitness losses to neighboring rivals.

<< Figure on extra pair paternity >>

Prey Selection by Garter Snakes and Resistance to Anti-predator Tactics of their Prey

E.D. Brodie III and E. D. Brodie, Jr. have investigated the evolution of toxicity in the newt *Taricha granulosa* and resistance to this toxin by the garter snake *Thamnophis sirtalis*. *Tarchica granulosa* is a common newt on the west coast of North America, undoubtedly because it carries a potent poison, tetrodotoxin, in its skin. Tetrodotoxin is a neurotoxin that is also found in puffer fish. The newt possesses bright orange **warning coloration** located on its belly. When disturbed, the newt contracts the muscles in its back which arches its belly and exposes the orange. Most vertebrate predators take this as a warning and leave the newt alone. Anti-predatory strategies like warning colors are treated in Chapter 14.

Garter snakes, which are found in areas where the newt is quite common, have evolved the ability to detoxify the tetrodotoxin. The Brodie's reared neonate garter snakes and compared the resistance of sibs compared to non-relatives. They found a positive correlation for the levels of resistance to the newt toxin among sibships. Sibs in some families had high resistance, sibs from other families had low resistance. By demonstrating heritable variation for resistance to the newt toxin, the Brodie and Brodie (1990) showed that garter snakes have opportunity to respond to selection favoring resistance to newt toxin.

Moreover, populations of garter snakes differ greatly in their level of resistance to tetrodotoxin (Brodie and Brodie, 1991). Some populations have little resistance (Fig. 3.14) others have a lot of resistance. They have also studied the costs of evolving resistance that is expressed in terms of reduced locomotor performance, which is an example of a pleiotropic effect. Snakes that evolve high resistance, appear to crawl more slowly than low resistance populations. The cause of this slow crawling speed is related to the genes that protect the snakes from the tetrodotoxin. The tetrodotoxin works as a poison that attacks calcium channels of nerve cells. Snakes have evolved a calcium channel that is less susceptible to tetrodotoxin, but it makes the snakes much less behaviorally responsive and slows them down during locomotion.

This is extremely useful example as it introduces several concepts of evolution, but in particular the **evolutionary arms race** between a predatory snake and its toxic prey. Notice that both the newt prey and the predator have to have resistance, which arises at different locations in the calcium channel protein. A change in one combatant, which evolves more toxicity (and more resistance), is countered by a change in the other combatant such that it keeps up in the arms race. The example also introduces a key aspect that governs behaviors, the nervous system and the molecular action of calcium channels. Additional details on sodium channels activity are found in Side Box 3.3.

Hotspots and Coldspots
in the geographic mosaic of coevolution between garter snakes and newts
(Brodie, Ridenhour, Brodie. 2002. Evolution)

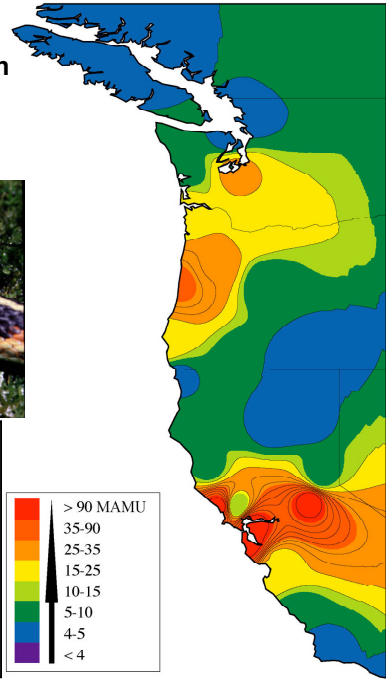
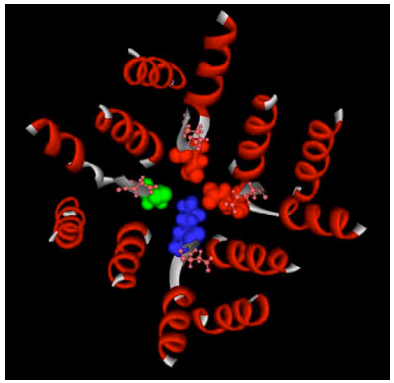


Figure 3.14. Variation in levels of TTX in the Newt, *Taricha granulosa,* in the western USA. *Thamnophis elegans* has evolved TTX resistance in 2 hotspots of newt toxicity. Gene sequence for the Na^{++} channel protein, Na_v, expressed in snake skeletal muscle, reveals that mutations in the outer pore of Na_v confers the snake TTX resistance.



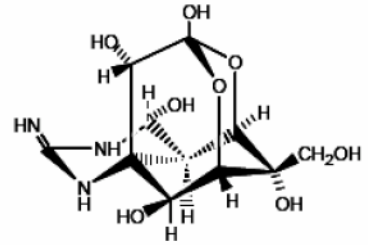


Figure 3.15. (Left) Protein structure for the Na^{++} channel protein Na_v. Pore proteins are colored blue, red, green. (Right) molecular structure of TTX.

Side Box 3.3. The mechanics of action potentials in nerve cells

Voltage-gated sodium channels (Na_v) are membrane-bound proteins that initiate action potentials in nerve and muscle cells. The channels open when the voltage, measured from the inside to the outside of the cell membrane, is depolarized by a few millivolts above the negative resting membrane potential. The open Na^{++} channel allows Na^{++} ions to flood a cell, depolarizing its membrane potential. The movement of sodium ions through the membrane comprises the rising phase of the action potential of a nerve cell. Rapid inactivation of the channel stops the flow of sodium ions through the pore. This step, along with the activation of the complementary voltage-gated K^+ channels, allows the membrane to repolarize and ends the action potential. Action potentials act as electrical messages that travel along the axons of nerve cells and the surface of muscle fibers. When the action potential reaches the end of the nerve cell it triggers release of neurotransmitters by neurons. If the neuron is connected to muscle, the muscle cell is triggered into action. If the neuron is connected to a neuron, neurotransmitters cross the gap junction to this neuron, triggering a drop in its action potential.

The actual sodium channel protein is formed by a 260 kDa α-subunit that is associated with different numbers of β subunits in the muscle (β1) versus central nervous system (β3 and β1 or β2). The α subunit forms the pore and the protein contains components required for other aspects of channel function including voltage-dependent activation and fast inactivation. The kinetics and voltage dependence of channel gating are modified by the β subunits, the α subunit contains all functionality of voltage dependence. A model of the two-dimensional folding pattern of the α subunit predicts that the protein consists of four homologous domains (DI-IV) composed of six α-helical transmembrane segments (S1-S6). Regions of the protein between segments S5 and S6 of all four domains reenter the membrane to form the outer pore of the channel through which sodium ions enter the cell. (see Figure 3.15).

<< diagram of nerves with gap junction, and muscle >>

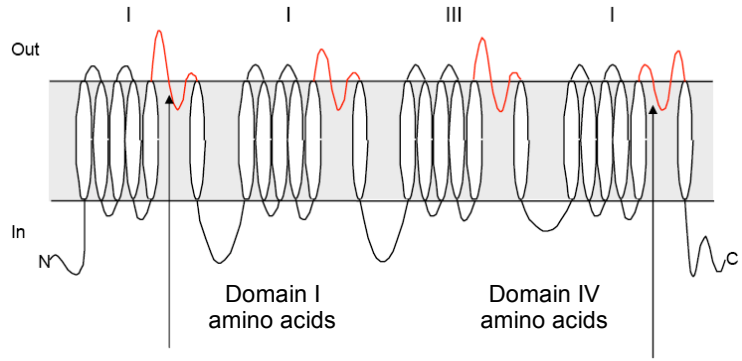


Figure 3.16. Secondary structure of voltage gated Na^{++} channels with orientation of membrane spanning segments. TTX resistance in the Newt arises from amino acid substitutions in domain I, but in domain IV in the snake.

Iso > Leu
Iso > Val
Gly > Ala
Asp > Asn
Gly > Val

The Brodies' studies parallels earlier work by Arnold (1980), who focused on the ability of snakes to feed on slugs. Arnold demonstrated that feeding on slugs versus feeding on frogs and fish runs in families. Behavioral preference for prey is heritable in *Thamnophis elegans* and can respond to the force of natural selection. Moreover, populations of slug-feeding snakes prefer slugs if the snake populations are located in coastal areas where slugs are a common resource. However, if the snakes come from inland areas where slugs are uncommon, the snakes prefer fish and frogs. Behavioral preferences for noxious prey like slugs have evolved in concert with the ability to ingest noxious or otherwise difficult prey. If a snake tries to feed on slugs the slug emits a sticky mucus which would make it difficult if not dangerous for a snake to consume the slug (Arnold, 1982). In the case of juvenile snakes, they can become completely trapped by the mucus secretions.

The examples from of snakes, newts, and slugs demonstrate that behavioral preferences appear to have evolved in response to the availability of prey. However, direct evidence of natural selection in these cases is lacking. Nevertheless, analysis of the correlations between behavioral traits that are expressed in a population (e.g., presence or absence of slugs) and the selective environment (e.g., presence or absence of prey) is a classic indirect testing for adaptation and inferring the historical action of natural selection.

Correlational selection on escape behavior and morphology

Functional interactions between behavior, physiology, and morphology may create suites of traits that are shaped by natural selection. Brodie (1992, 1993) has studied natural selection on escape behavior and color patterns of snakes to provide a direct test for the role of selection in developing functional integration among suites of traits. The dorsal patterning of snakes varies from stripped to blotchy. Snakes also vary in escape behavior. Some snakes flee at high speed in a direct line while others perform evasive reversals, first going in one direction, then rapidly changing directions, and then freezing in one place and relying on **crypsis** or **background matching** to avoid detection.

Correlational selection for particular combinations of traits may explain patterns of association between behavioral and morphological traits. Mark-recapture work in a natural population of the garter snake *Thamnophis ordinoides* has been used to study selection on both escape behavior and morphology. Brodie measured the escape behavior and scored the color pattern of a large number of neonates before he released them back into nature. Survival of the snakes varied as a function of both escape behavior and color pattern, but survival was a complex function of both of these traits (Fig. 3.17). Individuals with striped patterns that flee directly and those with spotted, or unmarked patterns that exhibit reversals, have high probability of survival (Brodie, 1992). In contrast, individuals with striped patterns that used reversals had low survival, as did snakes with spotted patterns that fled directly.

A snake must have a good match between its color pattern and it's escape behavior to survive. Such selection might lead to strong associations between the genes for escape behavior and color pattern because selection favors combinations of alleles at the separate loci that govern the distinctly different behavioral traits. Only snakes with a spotted pattern and that perform reversals survive well. Conversely, only snakes with stripes and that do not perform reversals have high survival and selection favors individuals that are fixed in the other alleles. Few snakes with reversals and stripes survive. There are likewise few that survive with no reversal and spots. This process leads to color pattern and escape behavior becoming **genetically correlated** in some natural populations of garter snakes (Brodie, 1993), even though the genes for these different traits are not on the same chromosome.

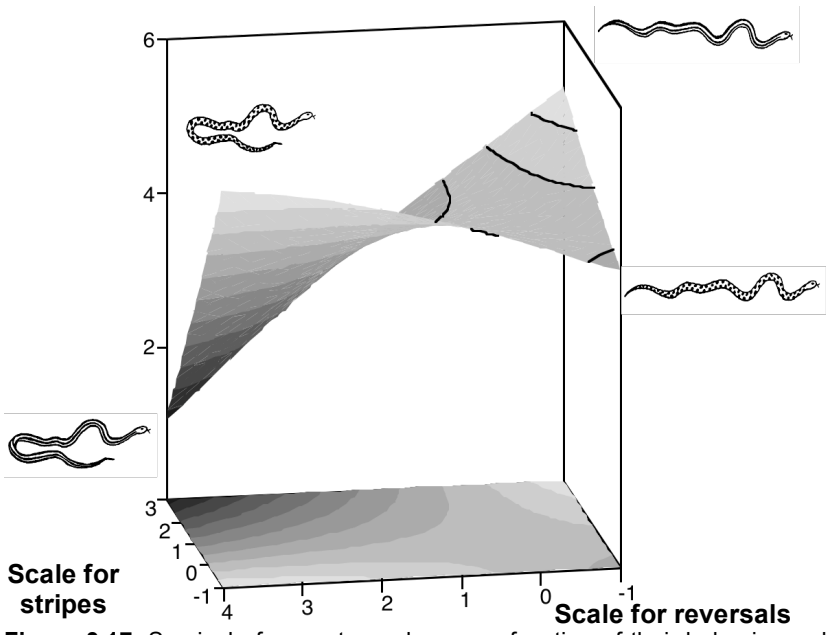


Figure 3.17. Survival of neonate snakes as a function of their behavior and morphology. Brodie scored the escape behaviors of snakes at birth and ranked them for number of reversals. If the snakes fled in a direct line, they received a score of -1. He also scored the pattern on the snakes: stripped versus blotches or no pattern. Many species of snakes exhibit the same pattern syndromes and it is likely that they also exhibit similar correlations with escape behavior.

Summary: the three causes of genetic correlations between traits

Genes can become coupled by the process of selection. This non-random coupling reflects the action of selection that can generate **linkage disequilibrium**, which is a non-random association of alleles at two loci. **Linkage equilibrium** is the multi-locus version of **Hardy-Weinberg equilibrium**.

If two loci are in *linkage equilibrium* the alleles at the two loci occur in individuals at a rate expected by chance. Thus, if two loci are in **linkage disequilibrium** this implies that some multi-locus genotypes are over represented while other combinations are under represented. In the case of sexual selection, a non-random association also forms between alleles for female choice (random versus preference for male trait) and the male trait (non-elaborate trait versus exaggerated trait). In the case of garter snakes, those with stripes and lack reversals survive, as do those with blotches and that reverse during escape, however, the other multi-locus genotype combinations do not survive and are under-represented compared to the association expected by chance.

The second source of a genetic correlation between two traits is **pleiotropy**, which is when a single gene controls the expression of two or more traits.

The final source of a genetic correlation between traits is physical linkage, which is the close proximity of two genes on the same chromosome. In the case of **physical linkage**, a second force is required to generate linkage disequilibrium and often that force is selection. Another way in which linkage disequilibrium can be built up between physically linked genes is by inbreeding, which is a special form of linkage called identity disequilibrium, named for the process of inbreeding, which is also called identity by descent (identity of alleles at the same locus, by descent). Physical linkage is useful in gene mapping studies when a researcher is interested in developing a genetic map of a behavioral trait (Chapter 2). The researcher looks for strong linkage disequilibrium between a marker and the trait of interest, in the context of a parent-progeny pedigree.

Whereas pleiotropy is a relatively permanent form of genetic correlation, the action of selection is erroneously thought to be less permanent in the sense that if selection is eliminated, the linkage disequilibrium of two unlinked genes will rapidly decay and the association will again reflect a random association of alleles at the two loci. We will see in the chapter on frequency dependent selection that the elimination of selection in the case of behavioral traits is actually quite unlikely. Stable social systems can generate a permanent linkage disequilibrium owing to frequency dependent selection.

Therefore behavior, in particular behaviors of signalers and receivers, can generate and create new combinations of traits. The signaler and receiver can be potential sexual partners in the case of sexual selection. Alternatively, the signalers and receivers can also be a predator and its potential prey in the case of garter snakes and their avian prey, or in the case of toxic newts and their snake predators. Behavioral traits are thought to be quite unique their propensity to **self-assemble** into new associations, compared to physiological or morphological traits.

Behavior versus Morphology as the Driver of Evolution

From the discussion of genetics and behavior it is clear that behavioral traits are seldom removed from other aspects of the phenotype such as morphology or behavior. This association is so pervasive that many evolutionary biologists have argued that behavior might play a major role in the diversification of organic forms (Mayr 1959, Brandon 1968, West-Eberhard 1989, Huey et al. 2003). Others have argued that the evidence for behavior as a driving force behind evolutionary change is scant (Plotkin 1988).

Ernst Mayr (1959) has argued that behavior drives evolutionary change for the following reasons. Behavior is often viewed as quite flexible. As we will see in upcoming chapters animals can learn tasks by trial and error. Moreover, a behavioral shift, whether due to genetic or environmental causes, exposes an organism to novel selective pressures. The selection then shapes evolutionary change in a number of morphological, physiological or ecological traits.

Consider the familiar example of runaway sexual selection in which genes underlying mate-choice can lead to the rapid evolution of bizarre and elaborate male ornaments. Does female behavior drive male morphology during runaway sexual selection or vice versa? Which came first, the evolution of escape behavior in garter snakes or color patterns? Did a mutation in the seed-crackers precede the behavioral adaptations

associated with foraging on different species of sedges? Was the behavioral flexibility in feeding on different sized seeds always present in the population, and did this facilitate the incorporation of a novel mutation for bill morphology.

These "chicken and egg" questions of precedence certainly merit further consideration. While we do not yet have all the information available to make this determination regarding the evolution of these behavior, the association between behaviors and morphology is striking enough to keep in the back of your mind as you read subsequent chapters such as those that treat sexual selection. Methods that are described in the chapter 18, the phylogenetic analysis of behavior, will allow us to date when a particular behavioral or morphological trait arose during the evolutionary history of a group. From such information we can determine whether behavioral trait arose before or after the evolutionary origin of other traits.

Study Questions for Chapter 3

1. After an artificial selection experiment on the frequency of wing beats during the courting rituals in flies, you reach an upper plateau (graph?). Give two reasons why wing beat frequency might not be able to continue increasing.

2. Two biologists observe male fighting behavior in red deer. One biologist suggests the reason for fighting is that dominant males control more females and have higher fitness. The other insists they fight because a seasonal increase in testosterone causes aggressiveness. Who is right? (a real thinker pulling in much of the material from chapter 1 -- proximate and ultimate causes, and chapter 3 -- selection, and even a bit on chapter 2 -- genes).

3. Name and explain three primary ways selection affects the differential fitness of individuals. Draw graphs to represent the effect of each functional relationship for selection on the mean and variance of a phenotypic distribution of a population.

4. How does selection on stripped vs. spotted snakes, and selection on reversing versus non-reversing escape tactics yield only two kinds of snakes:

- stripped – non-reversing snakes and
- spotted -- reversing snakes?

Assume one gene controls color, and another controls behavior. Assume that the gene that controls color has two alleles (stripped and spotted) and the gene that controls behavior has two alleles (non-reversing and reversing) shouldn't we see at least four kinds?

5. Compare and contrast natural selection and sexual selection. Is there a distinction between the two that is important?

6. In the runaway process of sexual selection, what trait is under selection in the males and what trait is under selection in the females? Which male traits are favored, and which female traits are favored? Why is runaway selection an example of maladaptive evolution?

7. What is linkage disequilibrium? What are three proximate sources of linkage disequilibrium. Which of these sources can generate permanent linkage disequilibrium and why?