Chapter 4: Levels of Selection
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Levels of Selection

The genetic makeup of a population is altered through an interaction with the ecology of the organism. We refer to this interaction as the process of natural and sexual selection. The fundamental premise of Darwinian selection is that natural selection acts on the individual, or more properly, differences in phenotype among individuals within a population. In recent years, a number of authors have argued that selection might act at a number of different levels and these levels of selection are loosely structured according to hierarchies of biological organization:

genes → individuals → kin → groups → species

We can arrange the five levels of selection along a continuum that describes degrees of genetic relationship. **Genes** are the fundamental unit by which we can define genetic relationships. If a gene is **identical by descent** to another gene, we are referring to a genealogical relationship -- at some point in the past, the two genes were derived from a single gene by a duplication event (e.g., meiosis in a sexual organisms). Dawkins (1976) has been the most vocal advocate that selection acts not on individuals *per se*, but on genes. The theory of **genic selection** is often referred to as the **selfish gene theory**.

Individual selection is next in the hierarchy and we have given individual selection extensive treatment in Chapter 3. **Kin selection** is a concept, which is closely related to individual selection. Kin are defined as closely related individuals, and it is thought that it might benefit an organism to aid kin in order to promote the **inclusive fitness of an individual**. Related individuals are likely to share many genes in common with a given individual. **Asexual clones** share all their genes. A sexual individual is comprised of genes from male and female parents. Half of the genes came from one parent and half from the other. **Kin** tend to share genes. The probability that two full sibs share genes at most genetic loci is ½. The probability that two half-sibs share genes at most genetic loci is ¼, because they only share one parent, not two as in the case of full sibs. The probability that cousins share alleles at most loci is 1/8.

Members of a group do not necessarily have to share genes, but the group lives or dies as a function of how their genes (and individuals) interact. **Group selection** is a more difficult concept to grasp, and it is

also the subject of an ongoing debate in evolutionary biology. Suffice it to say that selection at the level of groups occurs when a group of individuals produces more groups than other groups. Group selection is a process entirely analogous to individual selection, but it acts at the level of groups. Again the traits underlying group selection must be heritable if the species is to evolve by group selection. This is not a sufficient condition, but a necessary condition. A second necessary condition on group selection is that the timeframe over which group selection acts is just as rapid as the timeframe over which individual selection acts, otherwise individual selection can overwhelm the force of group selection.

Species selection acts at yet a higher level. Members of a species tend to share more genes than two individuals from a different species. Those members of a **clade** (e.g., a group of closely related species) that produce more species, or perhaps have greater longevity in the earth's history (e.g., lower extinction probability) are going to become overly represented on the planet in much the same way that one allele at a particular genetic locus spreads through a population of individuals and increases in frequency at the expense of alternative alleles.

Genic Selection

Is the unit of selection the gene?

Dawkins (1976, 1986) and other adherents to the theory of genic selection argue that because selection changes combinations of alleles at a genetic locus, the gene or genetic locus must be the fundamental unit of selection. At first glance, this argument seems imminently reasonable. For if natural selection acts to change allele frequencies at a genetic locus, then a purely reductionist's approach would reduce the problem to the lowest common denominator in all evolutionary theories -- the gene.

The gene is after all the fundamental unit of inheritance. I suppose an even purer reductionist might go further and say that a nucleic acid base pair was even closer to the true unit of inheritance, but that goes too far. While nucleic acids base pairs comprise the fundamental unit of genetic variation and information, nucleic acids do not form a functional unit. We also want a theory that links selection and inheritance with at least some aspect of the phenotype. Thus since a gene codes for a protein, the protein would be considered the smallest unit of the phenotype that comprises a functional unit. These philosophical issues require a bit of thought beyond this text, and I recommend a paper by Dawkins (1986).

Genic Selection, Selfish Gene, or Replicator Selection.

Despite the name, the theory of genic selection considers the allele as the fundamental unit of selection. The allele is the fundamental "**replicator**" because it gets copied during meiosis and mitosis. Accordingly, the term **replicator selection** has been synonymized with genic selection. The term selfish gene has also been applied to the theory, with reference to the notion that in the extreme case, the selfish gene is only concerned with propagating itself, perhaps even at the expense of the individual in which it resides (see t allele in mice below).

When thinking of the concept of genic selection, the selfish gene, or replicator selection, think in terms of the allele. Unfortunately, referring to these concepts with the terminology of allelic selection adds unneeded confusion to what are already difficult concepts. Realize that genic selection, selfish gene, and replicator selection refer to an allelic copy -- the fundamental replicator.

Is the unit of selection the individual organism?

The fundamental tenet of classic Darwinian selection is that the individual is the unit of selection. Let's explore the concept of individual selection as a review of previous chapters. We measure selection on individual traits and we tend to atomize the phenotype into its constituent traits. However, this focus on a phenotypic trait is really only a simplification of the real process of multivariate (many variables) selection. The individual trait is not the unit of selection. A researcher identifies a particularly trait that is under selection and tests to see whether the power of selection is statistically significant. In principle, we should analyze each and every trait in an organism and study the correlations between traits to completely describe the evolution of a species. The process of correlational selection, which was discussed in the previous Chapter on Selection, can couple two or more traits, and selection can couple the fate of the individual genes that control traits.

Brodie's study on garter snake morphology (striped vs. spotty) and behavior (straight slither vs. reversals) is a classic example of correlated selection. In the study of behavioral traits we must consider selection on morphological and physiological traits that might be functionally (e.g., selectively) or mechanistically correlated (e.g., by proximate mechanisms) with the behaviors. This approach to studying individual selection focuses on the whole organism and is described below.

t-alleles in Mice: a selfish gene

We will first consider a bonafide example of genic selection. Some populations of mice possess an interesting mutation that distorts the normal meiotic products of sperm that are produced by a male. This mutation is located *t*-locus of the mouse (Lewontin 1962). Let us denote the wild type allele at the *t*-locus +. Thus, we represent the homozygous wild type +/+. Let us denote the mutant allele at the *t*-locus: *t*. Thus, we denote an individual that is heterozygous at the *t*-locus *t*/+ and a homozygote *t*/*t*. The effect of the t-allele on the phenotype is as follows:

+/+ produce the normal wild type sperm.

t/+ produce normal sperm, however the ratio of sperm of the *t*-allele versus +-allele is distorted from 50:50 that one would expect by normal meiotic processes. These *t*/+ heterozygotes produce 85-95% *t*-allele sperm and only 5-15% +-allele sperm.

t/t homozygotes are sterile.

The process of genic selection promotes conditions for the origin of the *t*-allele and the spread of the *t*-allele. In a curious way, genic selection promotes high frequencies of the *t*-allele in the population but a high equilibrium *t*-allele frequency is counter-balanced by individual selection. As the frequency of the *t*-allele increases, more *t*/*t* individuals are produced, which are sterile.

1. The origin and spread of the *t*-allele

Because t/+ heterozygotes produce an overabundance of *t*-alleles, the *t*-allele is refereed to as a selfish gene relative to the +-allele. When such a **mutation** arises in a population it should spread rapidly. A male that carries one copy will produce 85-90% sons that also carry the *t*-allele. This is the classic notion of a selfish gene. By outcompeting other meiotic products the *t*-allele nearly guarantees it's successful transmission to the progeny, when it first arises or is at very low frequency in the population. Contrast this with the probability of spreading an allele under normal Mendelian segregation (see Side Box 2.1). The *t*-allele greatly enhances the chances that the mutation will spread after the mutant arises.

2. What limits the spread of the *t*-allele

The case of the *t*-allele is also useful for illustrating a selfish gene. The *t*-allele spreads and increases in frequency up to the point where a significant number of males in the population are in fact sterile. Thus, the fitness of individuals is sacrificed (e.g., sterility in some cases), as the *t*-allele marches up to very high frequencies. This leads to a considerable depression in the mean fitness of males in the population. Even females that happen to mate with such *t*-allele infested homozygotes produce no offspring. This last fact will become important in subsequent consideration of the *t*-allele when I discuss the possibility of group selection, below.

3. Equilibrium for the *t*-allele

The *t*-allele cannot spread beyond a certain frequency or the whole population will end up sterile. The *t*-allele needs a few of the wild type alleles (+-alleles) around in order to propagate. However, when two *t*-alleles end up in a single individual, the process of individual selection limits the spread of the allele.

Individual Selection and the Whole-organism Approach

The dichotomy between the whole-organism approach and genic selection appears to have roots in the 'How?' versus 'Why?' questions in evolutionary biology that were first discussed by Ernst Mayr (1961). Recall from the first chapter that I made a distinction between proximate mechanisms and ultimate mechanism. The concept of the gene as a unit of selection identifies the gene as *the source* for all proximate mechanistic processes that lead up to the phenotype. It also identifies the gene as the key element in the selective processes that lead to evolution

of the phenotype. The special kind of genic selection seen in the *t*-allele of mice is referred to as segregation distortion because the normal 50:50 ratio of gametic products is distorted at meiosis. These cases of genic selection are indisputable. However, Dawkins (1986) contends that all alleles, not just such cases of segregation distortion, are best understood from a genic selection perspective. In contrast, the **whole-organism approach** considers the atomization of phenotypic traits to the level of the gene unrealistic.

Let's start with the proximate effect of genes. From the discussion of genetic terms in chapter 2, we saw that an allele at a locus does not produce its effects on the phenotype in isolation. The dominance state on the other chromosome could modify the expression of the allele at the first chromosome. If the first gene were recessive and the second gene was dominant, then the first gene would have no effect on the phenotype of the organism. This is an example of a higher order interaction between alternative alleles that is difficult to reconcile if we were to consider the allele the unit of selection. Even the **fitness of the *t*-allele** is dependent on the allele on the other chromosome.

Moving up the causal cascade we could also consider the genetic locus as the unit of selection -- or rather the two alleles that comprise a genetic locus. Several problems arise as we move up to this level of selection. First, the expression of the phenotype as a function of this locus may be contingent on the interaction of this locus with other genetic loci in the organism -- epistasis may influence the outcome. Moreover, the expression of the gene and its effect on phenotype is also shaped by an interaction with the environment. Finally, a gene does not just affect one trait, but can affect many organismal traits through pleiotropy. Similarly, complete expression of a polygenic trait often requires the additive effect of many genes.

Proximate mechanisms as the source of correlations between traits

All of these genetic effects serve to link the various organismal traits together in a complicated web of genetic and mechanistic interactions. The end result of such interactions is seen as the **correlations between traits**. For example, the level of testosterone in an organism is correlated with aggression as well as with muscle mass. Testosterone actually interacts with a number of genes, and it turns on a suite of genes that build new muscle cells. The essence of the whole organism approach is to try to understand the source of correlations between organismal traits, and how these correlations govern the evolution of the species. For the moment, realize that the correlations between traits arise from the proximate mechanisms of genetics and the proximate mechanisms of development, physiology and learning. Such proximate mechanisms govern the forces of selection and they might constrain the direction that evolution can take. For example, is it possible to get an aggressive, testosterone-laden, care-giving male bird? Is aggression incompatible with parental care?

Natural and sexual selection as a correlating force between traits

Another source for the correlation between traits is the process of natural selection. Natural selection might favor specific alleles at a gene that controls a behavioral trait or at a gene that controls a morphological trait. Such loci are not linked by the mechanisms of genes, development, or physiology. The combinations of genes are linked by the process of selection, or more precisely by the agent of natural selection, a predator. The case of stripped versus spotted snakes (Brodie 1992, 1993) reflects an example where selection bundles up unlinked genes (e.g., genes that control behavior and genes that control morphology). Sexual selection bundles up the alleles for female choice and the allele for the male trait by a similar process. Thus, a correlation between the traits making up the organism also occurs by the process of individual selection.

Neither the interactions of alleles, genes, traits, nor effects of selection can be predicted from the structure of an allele. It is the existence of higher-level properties of genes and proximate mechanisms that present difficulties for theories of genic selection. We refer to such higher-level features of genes as **emergent properties**. The effect of genes cannot necessarily be reduced to a description of a single allele or even alleles at a locus, because the emergent properties of the genes arise from higher levels or organismal integration. Have we saturated the kinds of emergent properties of a gene or are there higher-level interactions to be found in kin and group selection? We explore these emergent properties of ensembles of genes in this chapter. Individuals in a social group have the ability to self-assemble into higher-level structures. These structures are built-up by more than just simple genic effects. Self-recognition behaviors can arise from interactions among many genes.

Kin selection

J. B. S. Haldane clarified the issues underlying the study of kin selection with his clever and illuminating statements regarding its operation (paraphrased from a purported impromptu lecture given by Haldane, in a pub in India):

> "I would gladly give up my life for two sibs, or 8 first cousins, ..."

Embodied in this statement is the notion of a genetic equivalence with an individual's entire genome and an equal number of shared alleles with related individuals. If 1/2 of the genes are shared with sibs, then it would take 2 sibs to make up for the total genetic material in an individual. Certainly, some shared alleles will be lacking, because of the probabilistic relationship of relatedness that sibs share (see Figure 2.8).

W. D. Hamilton captured the essence of kin selection in a simple equation that describes the concept of **inclusive fitness**. From the point of view of individual selection, the individual genes as well as the success of closely related individuals are important. A behavior that is deemed **altruistic** is one that helps another, but in some way costs the altruist. If the benefits of the altruistic act outweigh the costs, then selection will favor the behavior. The fitness of a behavior is determined by the direct effect of the behavior on the fitness of the individual, as well as the summation of indirect fitness effects across all kin that are affected, adjusting for the degree of relationship of the kin. Let's work through Haldane's impeccable logic for a "sib-sacrificial" behavioral act using Hamilton's equation. This argument assumes that the kin would have been eliminated unless Haldane intervened.

If Haldane were to sacrifice his own life, the effect of the self-sacrificial behavior on his fitness is **w = -1**. On the other hand, he saves two sibs, which yields **w = +1** for each sib. Each sib has a coefficient of relatedness, **r = 0.5**. Thus, the indirect effect of the behavior on his kin = **2 × 0.5 = 1.0**, and the net fitness of the behavior of saving two sibs at the cost of his own life is **w = -1 + 2 × 0.5 = 0**. Hamilton would just break even. If you were to throw in a first cousin (**r = 0.125**), he comes up positive in the fitness tally. Let's look at a more formal equation that Hamilton devised for inclusive fitness, which is expressed in terms of:

1. $\mathbf{w_{inclusive}}$ = inclusive fitness arising from a given behavior
2. $\mathbf{w_{direct}}$ = direct effect of the behavior on individual fitness
3. $\mathbf{w_{indirect,k}}$ = indirect effect of behavior on fitness of the kin of the individual
4. $\mathbf{r_k}$ = coefficient of relatedness with the *kth* kin.

Inclusive fitness is given by:

$$w_{inclusive} = w_{direct} + \sum_k w_{indirect,k} \times r_k \qquad \text{Eqn. 4.1}$$

If the direct effect, $\mathbf{w_{direct}}$, is deleterious to the individual, it is a fitness cost. The indirect fitness effect of the behavior on the *kth* relative, $\mathbf{w_{indirect,k}}$, (k = 1, 2, 3 … n relatives in the summation) must be a fitness benefit for the kin for the system to evolve the behavior. The break-even point would occur when we set $\mathbf{w}_{\text{inclusive}} = 0$, in the equation 4.1. The net fitness (right side of the equation) should be greater than zero:

$$0 < w_{direct} + \sum_k w_{indirect,k} \times r_k \qquad \text{Eqn. 4.2}$$

or upon rearranging,

$$-w_{direct} < \sum_k w_{indirect,k} \times r_k \qquad \text{Eqn. 4.3}$$

or as long as the **costs to individual < benefits to all kin**, the behavior will be favored by kin selection.

There is a final consideration, which Haldane did not discuss. This has to do with **reproductive value**. Intuitively, an old individual that gives up their life for kin is not strictly as great a sacrifice as a young individual that gives up their life for kin. Thus, the altruistic costs (and benefits) must take into account Fisher's (1930) concept of reproductive value. Reproductive value is a function describing age-dependent fitness that starts at zero, when an individual is born and climbs to a maximum when the individual reaches maturity. Thereafter, reproductive value declines until the age at which reproduction ceases. Reproductive value takes into account the probability of an individual surviving to maturity and the declining probability of survival and reproduction after maturity. If we take into account reproductive value then it is clear that an older senescent individual in a kin group should be much more likely to carry out self-sacrificial acts for kin that are very close to the age at maturity, and less likely to carry out kin altruistic acts directed to newborns.

Kin Selection and Haplodiploidy in Social Hymenoptera

Hamilton's statements regarding kin selection have a distinct genic selection flavor to them. A behavior can increase in frequency because it favors the fitness of genes *per se*. Regardless of whether the genes reside in an individual or a closely related kin is irrelevant from the viewpoint of genes. The most striking example of kin selection in the animal kingdom includes those social insects that develop into a sterile worker and allow their sister (the queen) to carry on all reproduction. This social system is common in the social hymenoptera like ants, wasps and bees. E. O. Wilson has suggested that such sociality has evolved on at least 11 separate occasions with this one order of insects. In contrast, only a few examples are found in all the other orders of insects. An unusual aspect of hymenopteran reproductive biology is related to the propensity with which this group of insects evolves kin altruism. Sex determination in the hymenoptera is governed by ploidy of the egg (n or 2n):

1. eggs that are fertilized develop into a female (2n),
2. eggs that are unfertilized develop into a male (n),

This form of sex determination, **haplodiploidy**, leads to very high coefficients of relatedness for females and males. For example, a male receives 100% of his genetic material from his mother. Males also produce genetically identical sperm. If a foundress or founding queen of a hive is fertilized by a single male, the daughters from the union share **0.75** of their genes on average, which is considerably higher than the typical **0.50** shared by sibs (see Side Box 4.2). Hamilton has argued that such a high coefficient of relatedness predisposes the hymenoptera to the evolution of kin based altruism.

The elegance of Hamilton's argument is only rivaled by its simplicity. Hamilton illustrated how a single proximate factor, haplodiploidy, was ultimately responsibility for an evolutionary predisposition to evolve one of the most advanced social systems found in the animal kingdom, eusociality. The evolution of **eusociality** as typified by hymenoptera is the most extreme form of altruism in which members of the colony sacrifice reproductive opportunities for the "common good of the colony". Haplodiploidy and the evolution of eusociality in hymenoptera exemplify the kind of explanation that we find satisfying in science because it spans a continuum from proximate to ultimate causation.

Side Box 4.1: Haplodiploid vs. Diploid Sex Determination

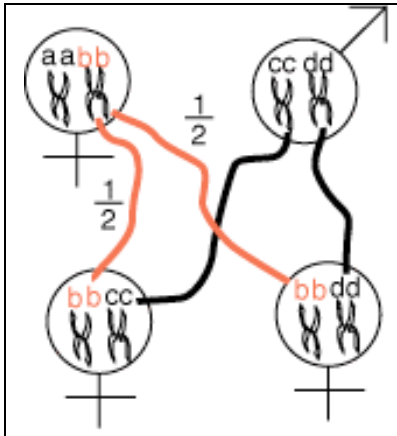


Figure 4.1. The probability of sharing genes in a typical sexual diploid. The same values of ½ apply to the male and female transmission of alleles to progeny.

Under mechanisms of diploid sex determination the probability that sibs will get the same copy of an allele is given by the probability of picking a specific allele (e.g., b) twice or 1/2 ×1/2 = 1/4. There are two possible ways in which this can happen from any given parent (each parent has two alleles) so we derive the probability of sharing alleles to be 2×1/2×1/2 = 1/2 from a specific parent. Each parent contributes half the genetic material to progeny so the probability of any gene being identical is 1/2×1/2 = 1/4 for any one parent. However, progeny have two parents so we multiply this by 2 to get the probability that sibs share alleles: 2×1/4 = 1/2. (see Side Box 2.4 for a second derivation of this same quantity.)

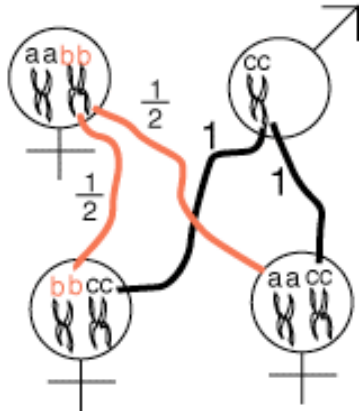


Figure 4.2. The probability of sharing genes in a sexual haplodiploid (male n, female 2n).

Under mechanisms of haplodiploid sex determination each offspring gets identical copies of half of their genes from their father so they share 1×1/2 =1/2 of their alleles from their father's side. They share the usual 1/4 from the maternal side (see above). The total probability of shared alleles is (1/2+1)/2 = 3/4, the average value of relatedness through the mother and the father. Thus, the coefficient of relatedness for sibs under haplodiploidy is substantially higher than it is under normal diploid sex determination.

While Hamilton's explanation appears to be an adequate explanation of the propensity for hymenoptera to evolve eusocial behaviors, it is not a comprehensive theory because it does not explain how eusociality evolves in other groups that do not have this peculiar form of sex determination. Eusociality is found in other social insects that do not have haplodiploidy such as termites, and even in some vertebrates such as the naked mole rat. While eusocial behavior is more rare without haplodiploid sex determination, how have these groups have evolved eusociality? High levels of genetic relatedness are readily achieved by the haplodiploid mating system and such relatedness appears to predispose the hymenoptera to eusociality. What other mechanisms might predispose a group of kin to high levels of relatedness. It has been hypothesized that genetic relatedness may contribute to the evolution of sociality in these other groups. High genetic relatedness cannot arise by the peculiarities of haplodiploidy, but may arise from inbreeding.

Kin Selection, Inbreeding, and Genetic Relatedness in Termites and Naked Mole Rats

Termites form colonies very similar to ants with a eusocial relationship between the queen and workers. Termites have a single, egg-laying queen in the termite colony that produces all the eggs, while workers collect food and warriors defend the colony. The eusocial system of termites might have been derived via kin selection if individuals breeding in the colony practiced brother-sister mating or mother-son mating. Genes become identical by descent across the colony by the process of consanguineous mating (see Side Box 4.2: Inbreeding). Consider sib-sib mating versus haplodiploidy. Without inbreeding sibs normally share 1/2 of their genes, but because of the probability of identity by descent, which is 0.25, the probability of shared genetic material is 0.75. This is only after a single generation. Repeated inbreeding events can lead to values for relatedness that rival or even exceed the 0.75 of hymenoptera.

If inbreeding is not a problem then inbreeding can readily promote the evolution of eusociality. In a species that has been inbred for a long time, deleterious recessive mutations would have been purged from the population long ago; therefore, an inbred species may be prone to evolve sociality. All members of a colony have nearly identical genetic backgrounds.

In addition to consanguineous mating, which promote inbreeding; the process of genetic drift can also produce high levels of inbreeding. Genetic drift is the random loss of alleles from a population owing to small population size. As more and more alleles are lost over time, a single allele can increase in frequency and come to predominate the population. In the extreme case, all copies of the allele are identical by descent. When colony size is small and colonies do not intermix the circumstances are primed for kin selection. Since both inbreeding and genetic drift act in concert under these circumstances, these two conditions greatly enhance the likelihood of evolving eusociality.

The population genetic structure of naked mole rats is fairly well characterized. There is next to no genetic variation within a colony of naked mole rats and they have a high degree of relatedness within a colony. In contrast, there is variation among colonies of naked mole rates. Inbreeding within colonies of naked mole rats could easily explain the low level of within versus between colony genetic variation. Moreover, Sherman (1998) has suggested that such inbreeding might also contribute to the evolution of eusociality in this group. If individuals in a naked mole rat colony interbreed, then the colony is likely to share a large number of genes that are identical by descent.

<< Naked mole rat photo >>

Side Box 4.2: Inbreeding

Inbreeding can be defined in many ways, but we generally refer to the simplest definition, which has to do with mating between related individuals. A more precise definition is to express **inbreeding** as the probability of the two alleles on complementary chromosomes being identical by descent. If these two copies of the allele (one from mother and one from father) are **identical by descent**, then they must have arisen in the not-so-remote past from a single strand of DNA.

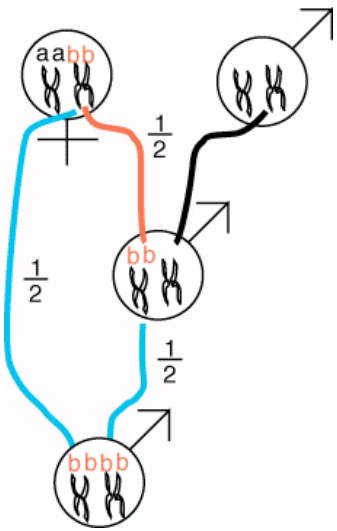


Figure 4.3. Mother-son mating.

Genes that have alleles identical by descent are readily generated from **consanguineous** matings, which is a mating among close relatives. Consider progeny fertilized from a mating between a mother and son. We can track one allele, *b*, from mother to son and from the mating of mother and son, which in a Greek tragedy is referred to as an Oedipal mating. Each chromosome is represented by two sister chromatids. Allele *b* is located on one of the mother's 2 chromosome pairs.

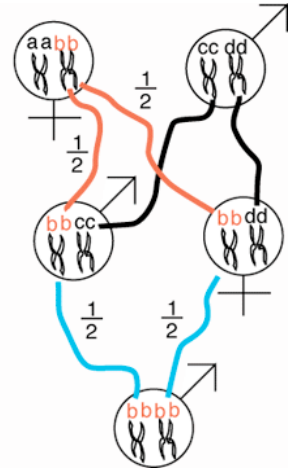
The son may receive 1 copy of allele *b* with probability of 1/2 (denoted by the red line). The blue lines denote the 1/2 probability that mom will pass on the *b* allele to a progeny sired by her son, who also contributes allele *b* with probability 1/2 via the second blue line. Because all events are mutually independent, we multiply probabilities to get the net probability that a child produced by an Oedipal mating possesses 2 *b* alleles:

½ × ½ × ½ = 1/8 (eqn 1).

Note that, the *a* allele could just as easily been transmitted to the son in the first place so the total probability that the alleles in the oedipal child are identical by descent can be achieved two ways, either with allele *a* or with allele *b* and the probability of inbreeding from an oedipal mating is:

2 × ½ × ½ × ½ = ¼ (eqn 2).

Figure 4.4. Brother-sister mating.



Of course, there is a 3/4 probability that the Oedipal child is not identical by descent. Let us consider a few special cases to assess the fitness consequences of other forms of inbreeding.

As a second example, let us consider the possibility of a sib-sib mating. We now have to keep track of the father's alleles because a grandchild can become inbred from one of the mom's alleles or one of the father's alleles. Again, we will track one allele, *bb*, from mother to son and from the mother to daughter. At the end we will multiply the probability of a single allele being identical by descent by four (all possible alleles that might be identical by descent) to determine the overall probability that any allele is identical by descent.

The mother gives the *b* allele to her son with probability of 1/2 and likewise the daughter receives the *b* allele with probability of 1/2 (both denoted by the red lines). Given that the daughter received the *b* allele, the daughter passes the allele on to her own progeny with probability 1/2 (blue line). Likewise, given that the son has the b allele he passes it on to his progeny with probability of 1/2. The probability that a grandchild received 2 copies of the *b* allele is:

½ × ½ × ½ × ½ = 1/16.

Finally, a grandchild could have received *a*, *b*, *c*, or *d* alleles by the same routes with the same probabilities thus, the probability of any two alleles at a single locus in the same individual being identical by descent is:

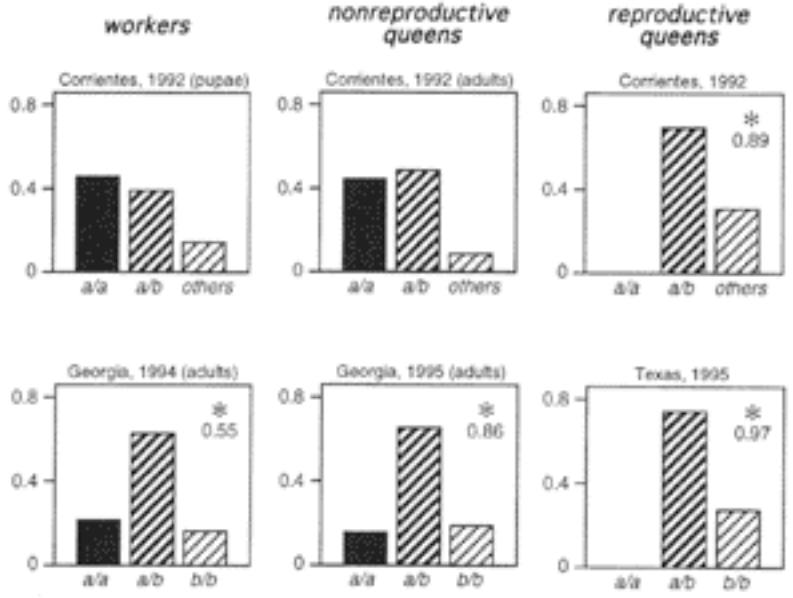
4 × ½ × ½ × ½ × ½ = ¼.

The same logic can be used to compute the probability of two cousins yielding a child that has genes identical by descent, though the length of the paths are a little bit longer. Indeed, the logic used above can be used to compute the **probability of inbreeding** or **identity by descent** for any **consanguineous mating**. The inbreeding in any set of pedigrees can be computed from similar **path diagrams** that chart the **genealogical relationships** among individuals.

Social Selection and Genes for Queen Determination in Ants

Perhaps one of the most interesting of all behaviors in the animal kingdom involves the evolution of sociality in which a colony divides labor between sterile workers that maintain the colony and fertile queens that produce all the individuals in a colony. The social hymenoptera form some of the largest and most complex societies in the animal kingdom. Ants and bees can have a number of fertile queens in a colony (**polygyne**) or a single fertile queen (**monogyne**). While the proximate mechanisms of queen determination are varied and can involve a suite of environmental factors (see chapter 14 on development and the environment). At least one case involves a genetic cause, which has been identified by Kenneth Ross and his colleagues (1996).

Fire ants have become a pest in North America since its invasion from South America. The particular species of fire ant, *Solenopsis invicta*, forms polygyne colonies with multiple queens. Egg-laying queens in multiple-queen colonies never possess a particular homozygous genotype (ala) at the *Pgm-3*, phosophoglucomutase-encoding gene, reproductive queens are always alb or blb genotypes. In contrast, sterile workers and non-reproductive queens can have all 3 genotypes.



What happens to all of the pre-reproductive queens with the ala genotype in these polygyne colonies? Why don't these females reach reproductive maturity? Pgm-3-ala queens do not make the transition from non-reproductive to reproductive status because workers kill these ala queens as they begin to mature and produce oocytes. The workers leave non-reproductive queens with the other two genotypes (alb, blb) to mature into reproductive queens. While the proximate cause of the developing ala queens demise is genetic, it involves the selective assassination behavior of workers. This reflects social selection.

Ross and his colleagues have not ruled out the possibility that another gene, which is tightly linked to the Pgm-3, determines the fate of the queen in multiple queen colonies. Not a single reproductive queen from polygyne colonies has been found to be homozygous for the Pgm-3-a allele despite a total of 4600 females that have been genotyped.

Further corroborative evidence that Pgm-3 is the actual gene responsible for selection is provided by patterns seen in monogyne colonies that only have a single queen. Reproductive queens in such single-queen colonies show a pattern of genotypes that is the same as workers and non-reproductive queens. The monogyne form of fire ant does not experience the same kind of worker selection that the polygyne form of fire ant experiences.

↑ **Figure 4.4.** Genotype proportions at the *Pgm-3* locus in introduced (Georgia) and native (Argentina) monogyne colonies.

← **Figure 4.5.** Genotype proportions at the *Pgm-3* locus in introduced (Georgia) and native (Argentina) polygyne colonies.

Finally, there is strong directional selection against the Pgm-3-a allele in polygyne colonies, yet it persists at high frequency in the species as whole. Selection against the Pgm-3-a allele in polygyne forms is opposed by gene flow from the monogyne form, in which the allele is common. Destruction of a particular genotype by members of the colony is a form of **social selection** that West-Eberhard (1981) suggested may play a major role in social evolution. Social selection leads to genetic differences between polygynous and monogynous forms of the fire ant.

Genic selection, and greenbeard tags

True altruism is defined to be a benefit from a donor to a receiver, which costs the donor of the beneficial act a fitness cost. Presumably, the receiver should reciprocate with altruistic behaviors at some future date in order for altruism to spread in a population. It is difficult to imagine how such self-less behavior can evolve in the context of cheaters that would take the benefit without ever giving in return. To get around this fundamental block to the evolution of social behavior, Hamilton envisioned a genetically based system of altruism that would be stable and thwart the action of cheaters, if altruistic behaviors could be directed to other bearers of the alleles. Dawkins coined the term **greenbeard** allele, based on Hamilton's theoretical musings, to describe an allele with the following simple behavioral properties:

1. the allele confers a unique signal
2. the allele confers recognition behavior of self (**autorecognition**) or non-self (**allorecognition**)
3. the allele also confers positive altruistic behavior on individuals bearing the same allele or conversely negative altruistic behavior on individuals bearing the non-self allele.

The concept of a greenbeard refers to any distinctive phenotypic attribute that could be used in allo or autorecognition Imagine humans with greenbeards that could recognize each other by the color of their greenbeard (Dawkins 1976), which is distinctive compared to normal beard colors. If animals simultaneously exhibited these two genetically based behaviors, then true altruism could evolve in which an individual carrying a gene for altruism might bear a cost that benefits a non-related individual that carries a similar gene for altruism.

The three behaviors imagined by Dawkins imply that the greenbeard allele has a pleiotropic effect on two behaviors or that three independent behaviors become linked by the effects of selection. In the first example that describes the fire ant, *Solenopsis invicta*, it appears that a single greenbeard gene has pleiotropic effects on the three behaviors.

A greenbeard gene in slime molds

In another example, a simple gene controls the fate of the social amoeba, *Dictyostelium discoideum* (Queller *et al.* 2003). High density induces individual amoebas to signal with cyclic AMP (adenine monophosphate). On receiving this signal, amoebas aggregate in the area of highest cyclic AMP concentration and begin to cooperatively build a fruiting body consisting of a stalk and reproductive spores. Some cells differentiate into stalk, a non-reproductive structure that raises the spores from the substrate. Other cells differentiate into the fruiting body that produce spores.

The *csA* gene, a cell surface adhesion protein, codes for a homophilic (self-loving) protein that is embedded in the cell membrane of an amoeba. The *csA* gene is intimately involved in aggregation behaviour. In laboratory experiments, the deletion of this single gene generates a mutant class of cheater cells that tend to become fruiting body, at the expense of non-mutant cells (wild type) that are pushed into the stalk. This reflects an altruistic act that arises in the context of mutant cheaters; a potential cost of carrying genes that might cause cells to differentiate into either stalk or spores. Only spores get fitness.

The advantage of cheaters is only realized in aggregations that are constructed as chimeras of mutant and wild-type amoebas. When allowed to self-aggregate on more natural substrates, the homophilic (e.g., self-loving) properties of the *csA* gene ensure that only self types gain access to the early stages of the fruiting body formation (e.g., wild types), thereby thwarting the cheater class that has a mutation at the *csA* gene (e.g., non-self), which is thus excluded from the entire fruiting body.

<< Full-page diagram of slime mould life cycle >>

Nepotistic greenbeards in fire ants

The case of polygene and monogyne queens in *S. invicta* colonies has elicited a great deal of interest because it is a case of a single gene that promotes a negative form of altruism. By killing off queens that bear the *aa* genotype of PGM, *ba* and *bb* workers are exerting a form of altruistic behavior that directly benefits the polygyne queens that bear the genotype *bb* and *ba*.

Laurent Keller has analyzed the proximate basis of such genic selection in a simple odor painting experiment. Queens that bear the vulnerable *aa* genotype, apparently express an odor in their cuticle that *b* workers find offensive (or lack an odor that protects), while the *bb* and *ba* queens are protected by some scent that is present in the cuticle. This cuticular difference between *bb*/*ba* and *aa* genotypes of queens can manipulated. The first step is to extract essence of *bb* cuticle from *bb* queens, and then painting *aa* queens with this extract. Queens with the *aa* genotype that are painted with this extract prior to maturation are rescued and protected from the murderous advances of the *bb* and *ba* workers.

This indicates that the *b* allele of *Solenopsis invicta* satisfied the two properties noted above, **allorecognition** and **negative or malevolent altruism** towards unrelated individuals. Are there examples of truly **benevolent altruism** in which an individual does something nice to another individual with which it shares alleles?

A genomic bluebeard in lizards and self-recognition behavior

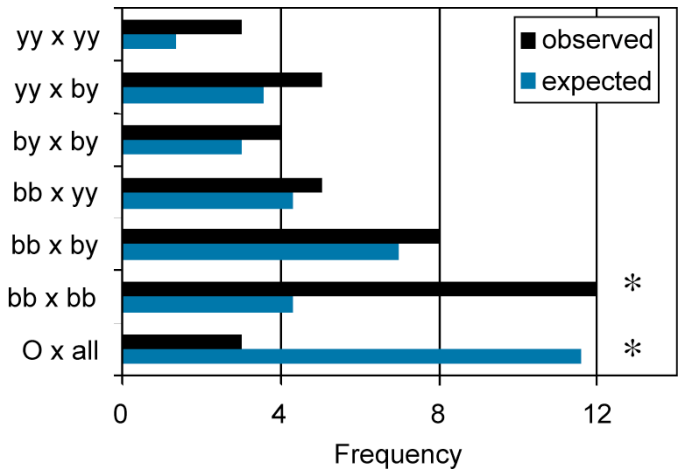
The evolution of altruism, cooperation, or any reciprocal behavior that decreases conflicts between neighbors has generated considerable theoretical interest given that it is at the root of sociality. The evolution of cooperative behavior is a balance between long-term benefits to donors from receivers, but enhanced risk of misdirected donation to cheaters. Theory suggests that the negative impacts of cheaters are diminished in small neighborhoods in which there is limited movement (e.g., dispersal). A simple solution is to evolve philopatric settlement (settle at the natal site) that enhances kin cooperation, but this has attendant fitness costs of inbreeding and kin competition, which will be discussed in Chapter 12 (Dispersal and Migration). Thus, dispersal and settlement is of prime importance for cooperation to evolve. Early theory suggested that high levels of dispersal are incompatible with the evolution of altruism, unless individuals can recognize genetically similar individuals. Recent theory suggests altruism and dispersal may not necessarily be incompatible behaviors, particularly if dispersal polymorphisms evolve (Van Baalen et al. 2006).

To test these ideas, Sinervo and Clobert (2003) examined dispersal behavior as a function of genetic similarity in a polymorphic vertebrate, the annual side-blotched lizard, *Uta stansburiana*. This lizard exhibits 6 color genotypes (*oo*, *bo*, *yo*, *bb, by*, *yy*) that serve as genetic markers for 3 male strategies. Color is controlled by the OBY locus, which has 3 alleles (*o, b*, *y*). Orange males (*oo*, *bo*, *yo*) usurp territory (O). Blue males (*bb*) are mate guarders (B). Yellow males (*by*, *yy*) are sneakers (Y). Male competition takes place among three different strategies: deceptive sneakers (Y) beat aggressive usurpers (O), cooperative mate-guarders (B) beat sneakers, and usurpers beat mate-guarders.

Sinervo and Clobert (2003) followed marked progeny whose natal site with respect to paternal and maternal territory was randomized for 10 generations, thereby removing natural correlations between genetically similar individuals that might arise from kin philopatry. DNA paternity with microsatellite loci was used to estimate the number of mature progeny (e.g., fitness). Microsatellite loci were also used to determine which male neighbors shared enough microsatellite alleles (referred to as genetic similarity) such that they could be sire-progeny or sibling pairs (e.g., relatedness=0.5). Maternal pedigrees were known with certainty, which allowed Sinervo and Clobert (2003) to determine whether genetically similar neighbors were in fact kin.

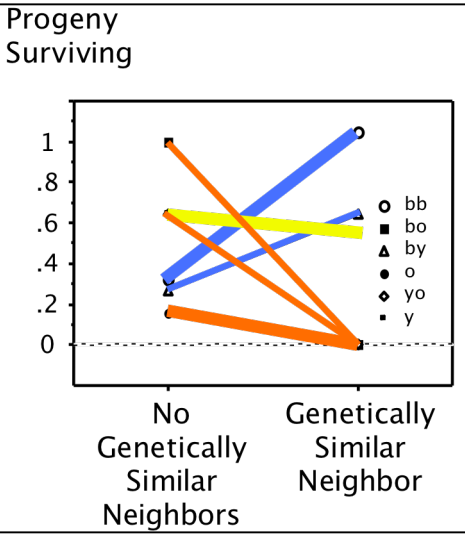
Genetically similar *bb* male neighbors were not related. Maternal and paternal pedigrees available for 38 pairs of genetically similar neighbors confirmed that none were kin (sibs, half-sibs, sire-progeny, or cousins). When B males find a genetically similar partner their fitness is tripled compared to males that lack a genetically similar partner. Patterns of B settlement are not merely due to selection at the individual level. Even though males gain an individual benefit, it is contingent upon presence of genetically similar neighbors with *b* alleles. Pedigree data indicate that kin selection or kin philopatry are not involved. Given that selection is not merely at the level of the individual or kin, the phenomenon reflects genic selection on a cooperative greenbeard linked to *b* alleles.

Side Box 4.3. A gene complex for greenbeard behaviors

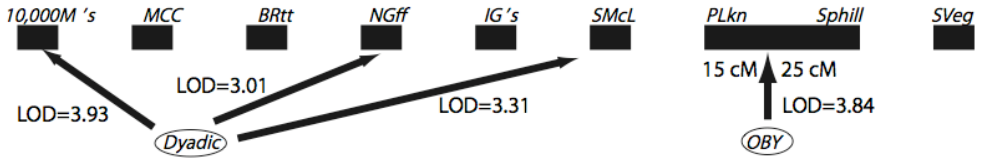


A. The behavior. In the face of experimental randomization of natal site, *bb* males settled beside genetically similar *bb* male neighbors at rates higher than expected given the frequency of male morphs. Observed association of O male genotypes (*oo*, *bo*, *yo*) with genetically similar neighbors occurred at rates significantly lower than expected. Frequency of other genotypes occurred at rates expected by chance.

B. The fitness consequences of the behavior. Correlational selection on recognition and settlement loci favors B males that settle next to genetically similar B males. O males are favored to settle next to genetically dissimilar males. Y is neutral for this behavior. Thus, genes for self-recognition and a settlement preference for self genotype neighbors should build up in the B strategy, while self-repulsion should build up in the O strategy. B only gets high fitness if it finds a genetically similar partner; O must avoid self genotypes.



C. Linkage maps of self-recognition loci. We mapped (Sinervo et al. 2006) loci for self-recognition, referred to as dyadic behavior. We scored behaviors in both sexes using a field pedigree. In males, we scored the propensity to settle beside genetically similar neighbors. In females, we scored the propensity to mate with genetically similar partners. We then determined which of the microsatellite markers was in physical linkage with genes responsible for self-recognition, using standard methods of gene mapping (Ott 1999). A primary linkage map must first be constructed for the microsatellite markers. Loci on separate blocks are unlinked (e.g., recombine with r=1/2) and are likely reside on different chromosomes or on the opposite arms of a large chromosome. The tendency of self-recognition behavior to be heritably transmitted to progeny is reflected in a marker locus that is transmitted in tight linkage. Loci with high LOD scores (Logarithm of Odds) sit on the same chromosome (e.g., physical linked) with the loci governing self-recognition (dyadic) behavior, or OBY color. Three loci mapped close to self-recognition, of the 9 microsatellite loci. The OBY locus mapped between 2 loci (15 centiMorgans [cM] from 1 locus, 25 cM from another). Therefore, genes that cause B male males to find and settle next to a genetically similar partner arise from the sharing of alleles at these 4 loci. We hypothesize that 1 locus must be related to a color preference for blue. We suspect that 1 locus is related to preference for males that have a self- similar pushup frequency: blue males spend a substantial fraction of the day directing push-ups to blue partners. The final locus may be related to genes for the major histocompatibility complex, the MHC, which is responsible for pheromone-based self-recognition behavior in vertebrates. The other loci (i.e., *MCC*, *BRtt*, *IG*, *SVeg*) that are not in physical linkage with self-recognition or color must sit next to a gene that helps B males cooperate. We suspect these loci are related to down-regulation of aggression in cooperative B males. However, alternative alleles that up-regulate aggression in O should also segregate at these **strategic loci**, named for their effect on male strategy.



Similarly, the *o* allele could be considered a greenbeard in that O males cooperate by dispersing away from other O males that carry *o* alleles, and thereby reduce direct competition. This spacing behavior would limit the fitness losses that are observed when males, which carry the *o* allele, settle next to a genetically similar male. Genetically similar groups of blue males form each generation through mutual attraction. Such groups of genetically similar *bb* males have higher fitness than pairs of *bb* males that are not genetically similar (which we call loners because they lack group behavior, even though they can have blue neighbors). Grouping behavior is a heritably transmitted each generation via the alleles for self-recognition and settlement preference that successful blue males pass on to their blue sons.

Finally, the OBY locus is not the only locus contributing to genetic similarity of morphs. An interaction between the morph locus and many other loci in the genome are involved in genetic similarity (see Side Box 4.1), and this similarity fuels the evolution of cooperation. This morphotypic level of selection may serve as a precursor for the evolutionary transition from selection at the level of the individual to true kin selection. Indeed, morphs are often associated with the evolution of eusocial behavior or other forms of kin selection. Thus, the *b* allele of *Uta stansburiana* satisfies the three properties of greenbeards noted above, (signal, recognition, donation). Blue males exhibit **allorecognition** and **positive altruism** towards unrelated, but genetically similar, individuals. This reflects **benevolent greenbeard altruism**, rather than the **malevolent greenbeard altruism** of fire ants.

In the case of cooperating male side-blotched lizards, it is clear that the *b* throat color allele is correlated with alleles at many loci across the genome conferring a genomic greenbeard. Dick Alexander envisioned that the factors contributing to cooperation via genetic similarity (e.g., kin selection or genic selection) should be distributed across the genome to be most effective. The lizards appear to reflect exactly this kind of greenbeard recognition, which has evolved between unrelated individuals. We are currently attempting to map the specific behaviors for self-recognition, as well as the factors that stabilize cooperation in B males, or allow O males to destabilize territorial relationships and act aggressively towards neighbors. We suspect that these loci are related to factors that enhance levels of plasma testosterone and other genes that enhance the ability of O males to take over territory. B males are down-regulated in these key performance traits to stabilize cooperation, while at the same time O males should be strongly up-regulated in these physiological traits.

Figure. 4.6. An orange homozygote (*oo* genotype), blue homozygote (*bb*) and a blue-yellow heterozygote (*by*) of male side-blotched lizards, *Uta stansburiana*, a species that gives me endless hours of amusement in the field.

If Dawkins had chosen another color in his hypothetical example of altruism, he might have presaged the bluebeard of cooperation between some blue-throated *Uta*. A salient difference between greenbeard recognition in *Uta* and Hamiltonian (1964) greenbeard supergenes envisioned by Dawkins (1976) is the genome-wide nature of the greenbeard in *Uta*. The *b* allele segregates at one key signal locus, but other self-recognition and color-recognition factors (Side Box 4.3) are linked to this signal by correlational selection on the benefits afforded by self-recognition and mutual cooperation in territorial partners (Sinervo et al. 2006) Correlational selection exists because all

components of signal-recognition-donation are simultaneously required to achieve cooperation, but these traits are harmful in other morphs like O that have aggressive behaviors, which destabilize cooperation. Hamilton (1964) did not consider the power of correlational selection in coupling suites of traits, thereby driving the evolution of coordinated interactions among the many loci required for complex traits like cooperation. The role of correlational selection in bundling the components for color-signal, self-recognition and self-attraction, plays a profound role in building the genome-wide greenbeard of *Uta*. Correlational selection on self-recognition systems should be general for many forms of evolutionary cooperation.

Even in kin altruism, sharing a fraction of genes is not sufficient for donation; specific genes that allow recognition of kin, exclude cheaters, and coordinate donations are required. A proximate explanation for kin altruism is not that kin share a fractional number of genes – rather kin altruists share key genes for signal, self-recognition and donation behavior. Our view is that cooperators need to share alleles at 3 key loci for signal, recognition, and donation. Given that all 3 loci are required to secure the fitness benefits of cooperation, correlational selection will build genetic correlations among these loci. Overall genetic similarity is thus a byproduct of correlational selection on these loci rather than a cause of cooperation.

Once key loci for cooperation exist, they cause genome-wide similarity to build by co-opting more loci into the gene complex. Adding more loci allows for the system to protect against the erosion of linkages due to **intragenomic conflict** (a subject that I treat in Chapter 11) and invasion by cheaters, as well as an added byproduct of spreading the long-term benefits of cooperation across the genome. Just as in individual selection where an altruist might pose the existential question:

> "Am I too young to die for my partner?"

genes that reside in the same genome as a greenbeard locus are in **conflict** with the action of an altruistic greenbeard that is self-sacrificial. However, if the greenbeard effect arises from many unlinked loci, nearly all loci would sit near one of the "distributed" greenbeard loci. In this case, these loci are now linked to the selective fate of the greenbeard complex. If a cooperative greenbeard is successful, linked genes can hitchhike to high fitness and thereby eliminating **intragenomic conflict**.

One reason we may not find many examples of greenbeards is that we typically look for simple examples with few genes (see references noted above for ants and slime molds). Many other examples with many shared genes are described as kin selection, even without a pedigree. Most studies to date, have looked for allele sharing as evidence of kin selection (e.g., paternity analysis), however, the pedigree data of lizards indicate that genetically similar males are not kin. An extensive pedigree is necessary to prove the action of kin selection or disprove the role of kin selection, in which case the social system reflects greenbeard cooperation. These cases of kin selection may arise from greenbeard correlational selection acting on either kin or unrelated pairs.

This process-based view of greenbeard correlational selection that acts on many signal-recognition-donation loci provides a promising avenue for investigating the evolution of cooperation with gene mapping studies of the genes that govern behavior.

Do other species harbor examples of cooperation arising from separate loci assembled in a co-adapted complex of greenbeard signals, recognition, and donation? A fruitful place to look is in the examples of kin altruism, which may harbor loci for signal, recognition and donation behavior that are under correlational selection.

Kin Selection and Cannibalism in Tiger Salamander Larvae

Kin selection does not just operate on organisms with eusocial lifestyles. It may operate whenever kin end up in close proximity. Cannibalism is a widespread trait in the animal kingdom. Where you find large monotypic stands of a species and when food becomes limiting, the best item on the menu might just be a conspecific. David Pfennig (1992) reared the larvae of the cannibalistic tiger salamander in various kin groups:

1. As a control, he reared unrelated individuals in the same tub
2. He reared half-sibs in the same tub (0.25 relatedness)
3. He also reared full sibs in the same tub (0.50 relatedness).

He then scored the probability of cannibalism and compared this among his experimental treatments. If the salamanders had sibs in their tubs the

probability of cannibalism was less than that for half sibs and the probability of cannibalism for half sibs was dramatically lower than tubs with unrelated individuals. These results suggest that: 1) the larvae have kin recognition, 2) kin recognition alters the behavior of cannibalism, and 3) presumably a tadpole that does not eat kin will be favored by kin selection by inclusive fitness. Pfennig has also found that morphs of the spade foot toad are likewise kin altruistic in their cannibalistic behaviors, which are described at the end of Chapter 2.

Pfennig also suggested that recognition of kin may arise from the distinct food ingested by aggregations of tadpoles. The tadpoles aggregate after hatching and a group of related tadpoles might stick around long enough to pick up a common set of odors that they share with related kin. I suggest that there are genes to be mapped that govern the propensity to be cannibalistic in tiger salamanders. Other genes for self-recognition, perhaps the MHC (see Side-Box 4.3), are likely to interact with the decision to be cannibalistic to non-kin (and kin alike). The MHC locus is a key gene involved in self-recognition and its role in mate preference is discussed extensively in Chapter 10.

<< photo of tiger salamanders and spade foot toads, each eating non-kin of their own species>>

Genic, Individual, and Kin Selection

In the example of *t*-alleles in mice we saw that genic selection, which favored the production of *t*-allele sperm over wild-type sperm, was counterbalanced by individual selection that favored homozygous wild type or heterozygous *t*-allele individuals over homozygous *t*-allele individuals. Is it also possible for genic selection to be counterbalanced by the action of kin selection? While we do not have concrete examples of such a process it is certainly easy enough to envision with a simple hypothetical example. Consider two parental birds that are rearing a brood of young (Williams and Williams, 1957).

Let's assume that a novel greedy type arises in a nest, which causes a chick to eat all the resources at the expense of the other nest mates. While the individual that houses the greedy allele fledges successfully, other nest mates waste away and die. The greedy gene has a selective advantage within the nest. One copy of this hypothetical allele is enough to ensure its short-term spread in the nest (albeit at the expense of its sib mates). However, nests with a greedy gene produce far fewer fledglings than those nests, which are comprised of pure wild type individuals. Kin groups, which lack a greedy gene, have a survival advantage relative to kin groups where greedy gene is present. Therefore, kin selection is likely to be held in a balance by the force of individual selection (Wade, 1980). The idea of a conflict between kin and individual selection is embodied in Hamilton's equation on inclusive fitness.

While this example involves individual selection and kin groups, it is a simple matter to extend the analogy to groups of individuals who are not genetically related. Can an altruistic gene spread in such groups or will greedy non-altruistic genes spread like wild fire? The answer to this question will require additional theoretical development, which is reserved for the chapters on Social Evolution. However, it is clear that the action of genic selection can be counterbalanced by the action of individual or kin selection, and the action of individual selection can be counterbalanced by the action of kin selection. The additional possibility of social selection (as seen in fire ants in which non-reproductive castes alter the fitness outcome of the reproductive caste) poses additional challenges for the study of levels of selection. Even though fire ant workers are non-reproductive their impact on the fitness of the *a*-allele within a polygynous colony is tremendous. Are there other emergent properties of genes, which act at the level of the group, besides those already noted for greenbeard self-recognition and self-assembly?

Group Selection and Self-limiting Behaviors

"Classic" group selection stems from arguments that Wynne-Edwards (1962) made in the 1960's. Wynne-Edwards was arguing for the evolution of population regulation by the mechanism of group selection, in which behaviors evolve because they benefit the group's existence. Charles Darwin was not immune to such conjectures as he wrote very similar statements in the Origin (1858). Wynne-Edwards argued that animals might evolve mechanisms that limit reproductive effort for the good of the species and the arguments are outlined as follows:

1. to avoid stripping the resource base and being exterminated, the individuals could evolve some kind of self-limiting behavior
2. difference in groups fitness, not individual fitness are what lead to the evolution of such self-limiting behaviors

3. the driving force underlying such group selection pressure is variation in rate of increase or extinction among groups
4. this behavior has a genetic basis (e.g., in the individuals that self-assemble into groups)
5. in the case of self-limiting behaviors, groups that evolve individual behaviors, which are self-limiting, will have a lower probability of extinction than groups, which lack self-limiting behavior.

The group selection advocated by Wynne-Edwards (1962) is a form of altruistic behavior in which individuals limit their own reproductive success to benefit the group. The key distinction in this case is that the members of the group are not related, or the argument might work as kin selection rather than group selection. The group selection argument requires a special kind of altruism towards non-kin to evolve -- true altruism not kin altruism.

The simple problem with group selection is that it can always be invaded by an individual "cheater" strategy (Williams 1966, Trivers 1971). An individual that does not live by the "group rules" and does not limit their own behavior will gain resources relative to other members of the group. This cheater can spread by virtue of its enhanced fitness.

Thus, true altruism is not an **evolutionary stable strategy** (ESS) because it can be invaded by a selfish morph. What is an evolutionary stable strategy or ESS? An ESS is a strategy that is so good in Darwin's struggle for existence that it is uninvadable by any (and all) mutant strategies that arise in the population. Because cheaters can invade altruism in the context of group selection, altruism is not stable over the long haul. The idea of an ESS originated with Maynard Smith and Price (1973), and we will use the concept over and over again in subsequent chapters. It is particularly useful in understanding the evolution of behavioral strategies that involve an interaction between two or more players -- an evolutionary game. When the fitness of an individual depends on the strategy or frequency with which alternative strategies occur in the population, the ESS concept is useful in the analysis of long-term evolutionary outcomes or the equilibrium states for a given social system.

In the case of group selection, those individuals that are playing the group strategy (e.g., self-limiting) will always do worse than the selfish individual that does not play by the rules. These cheater individuals will produce more progeny at the expense of the other members in the group that are self-limiting. Once the mutant arises, it spreads through the group rapidly. Another problem with group selection is the rapidity with which individuals reproduce compared to the turnover time involved in group selection. It takes far longer for groups to go extinct or for one group to overwhelm the selective advantage of individual selection.

Evolutionary biologists and behavioral ecologists do not say that group selection is a theoretical impossibility, but rather that the conditions required for group selection would rarely be met in natural populations (Maynard Smith, 1976; Wade, 1978; Wilson, 1983). Groups would have to be very small, and the selective advantage among groups must be enormous to overwhelm the ever-present force of individual selection. Interestingly, the *t*-allele in mice illustrates how group selection would work. Group selection is in conflict against the force of genic selection in the following example.

The *t*-allele revisited

Lewontin (1962) calculated that the *t*-allele should have a phenomenal advantage over the +-allele (wild type) but it would not completely eliminate it. The equilibrium or expected frequency based on the balance between genic selection, which favors the *t*-allele, and individual selection that removes homozygous sterile individuals from the breeding pool is $\hat{t}_{predicted}$ **= 0.70**. However, the observed frequency in populations was $\hat{t}_{observed}$ **= 0.36**. Why was there such a large discrepancy between expected and observed frequencies? N.B., the formula for the equilibrium frequency of an over-dominant allele is given in appendix 2.

The spread of the *t*-allele is in fact limited by group structure and the behavior of mice. Mice live in small **demes** with usually 2 males, and a few females. The *t*-allele should increase in frequency up to $\hat{t}_{predicted}$ **= 0.70**. With only two males in each group there is a really good possibility that some groups will have no fertile males and other groups will have fertile males.

Table 4.1. Possible group structures for the *t*-allele in male genotypes.

Fitness consequences of group structure	Male genotypes in the group
1. these groups breed true for +/+:	• +/+ and +/+ • *t*/*t* and +/+
2. these groups have a *t*-allele advantage:	• +/*t* and +/+ • +/*t* and +/*t* • +/*t* and *t*/*t*
3. this group is sterile and goes extinct:	• *t*/*t* and *t*/*t*

Groups that breed true for +/+ spawn off more groups that breed true and are more resistant to extinction relative to *t*-allele "infected" groups. The groups that have a *t*-allele will produce progeny with a greater incidence of the *t*-allele. Genetic drift, the random sampling of alleles might actually push some of the spawn of these groups over the top and they go to the *t*/*t* and *t*/*t* sterile state. Any sterile group goes extinct, even groups where the female is not sterile.

Thus, a group extinction process continually removes *t*-alleles from the population establishing the $\hat{t}_{observed}$ **= 0.36**, which is much lower than the $\hat{t}_{predicted}$ **= 0.70,** if genic selection and individual selection were the only forces at play (Lewontin 1962). Groups that breed true for the wild type +-allele, have a strong group advantage, even over the groups in which the *t*-allele has an advantage. Eventually the *t*-allele will increase in frequency in such *t*-allele infested groups to the point where the spawn of such groups fall into the sterile category. In the case of the *t*-allele, group selection balances out the effects of genic selection and it leads to a lower frequency of the *t*-allele than one would expect if genic selection were the only force acting on the population.

<diagram of how group selection acts to counterbalance the *t*-allele>

The force of sexual selection also plays a role in the *t*-allele example. Consider a female that carries one copy of the *t*-allele. She is selected to avoid heterozygous males that carry one *t*-allele, because of the ¼ probability of producing sterile sons. Amazingly, females have evolved this ability to discriminate mates in natural populations where the *t*-allele is prevalent. Thus, sexual selection plays a synergistic role in limiting the spread of the *t*-allele. If this behavior were a property of the *t*-allele *per se*, then it would be a form of genic mate choice in which *t*-allele females are self-recognizing and self types are repellent to them. This aspect of sexual selection and *t*-alleles has yet to be determined.

In case you think that *t*-allele are a rare phenomenon, it turns out that the there are many examples of *t*-alleles or analogues of segregation distortion in mammals and other vertebrates (Burt and Trivers 2006). This is, in retrospect, not surprising given that segregation can easily invade meiosis. Segregation distortion is a simple kind of cheater allele, which cheats on the fundamental cooperation present between maternally derived and paternally derived chromosomes at meiosis.

Artificial Selection for "Group Traits"

A final example will serve to illustrate how strong group selection must be to overwhelm the force of individual selection. Michael Wade (1977) raised lines of *Tribolium*, flour beetles, under conditions conducive to the group. Rather than propagate the lines by choosing the individuals, which fared best under high and low density, he propagated lines from those collective groups that produced the highest density colonies. This differs from a classic artificial selection experiment outlined in Chapter 3, in that one would choose those individual groups that performed the best in comparison to other groups. Wade also propagated some lines and allowed individual selection to predominate.

By choosing entire groups Wade was selecting a group that had the ensemble of genetic attributes that were conducive to self-limiting behaviors. The key behavior that Wade happened to select against in his group selection treatments was cannibalism or the tendency of individuals to feed on other members of the group (Wade, 1980). Perhaps this cannibalism gene reflects a genic altruism, which I have already speculated about in the example on tiger salamanders.

Over the long haul, Wade was successful in altering the "self-limiting capabilities" of the beetles and his lab experiments proved that group selection could alter the evolution of the species. But he was also careful to point out that there are very stringent conditions on the strength and efficacy of group selection. It helps to have small groups, rapid group generation time, and very strong group selection to overwhelm the force

of natural selection. Wade also described how such group selection is related to kin selection and cannibalism.

Levels Reconsidered

Convincing cases of genic selection for traits like segregation distorters appear to be quite plausible. Should we consider all behavioral traits subject to replicator selection? Tallying up the proportion of shared alleles, without regard to specific traits coded for by those alleles, as is the case for kin selection, has a genic selection flavor to the arguments. Concepts of kin selection are central to understanding issues of cooperation, which we will treat in greater detail in chapters on social evolution.

On the other hand, individual selection envisioned by Darwin (1859) has great explanatory power when considering suites of whole-organism traits. In such cases, the emergent properties of genic interactions argue for phenotypic selection as the appropriate level to adopt when considering the interaction between behavioral traits and the rest of the phenotype.

Whether or not we should reduce all the levels of selection to the replicator is indeed debatable or perhaps even a matter of personal taste. Perhaps an eclectic approach to the issue of level of selection might be the most appropriate. Use theories of individual selection as a default hypothesis, and only consider genic, kin, or group selection as alternative hypotheses when arguments based on natural or sexual selection are not a satisfactory explanation of behaviors.

A final puzzle to mull over. Consider groups of blue male lizards. This must reflect a form of group selection in that genic selection of a single gene, as classically envisioned by Hamilton and Dawkins, is not responsible for their formation. The collaboration of a large number of genes in the genome is required to create these groups of cooperators (Side Box 4.3). In a later chapter on Conflict (Chapter 11), I show that one blue male will altruistically give up his life to protect his partner.

The formation of these groups of genetically similar *bb* males is:

1. Highly heritable ($h^2 = 0.96$),
2. Groups form anew each generation, thus the force of group selection favoring blue males can keep pace with individual selection,
3. Groups of blue males that find a partner, which shares many genes (e.g., color, self-recognition, cooperation loci), have higher fitness than those loner blue males that lack a genetically similar partner,
4. Loner blue males also settle beside blue males, but these blue neighbors are not genetically similar to the loner. Therefore, they do not engage in the same group behaviors as observed in cooperative blue males that find a compatible (genetically similar) partner. Loner blue males have lower fitness because they lack the genetic factors that stabilize cooperation and are eliminated. N.B., many loner blue males will be produced, due to recombination between the suite of genes for B and the other two egoistical strategies of O and Y. These loners are constantly being purged from the population, much like sterile *tt* males. The loner B males reflect the **genetic load** of a cooperative B strategy, that segregates alleles with alternative egoistical strategies of O and Y.

Therefore, the case of the side-blotched lizard must be a case of group selection involving a large number of genic factors, that also are in conflict with individual interests (e.g., one male self-sacrifices to protect his buddy from orange neighbors). Are similar groups waiting to be discovered in the animal kingdom? I think so. We have already discovered a similar set of genes in the European common lizard that is strikingly similar to the side-blotched lizard in nearly every salient detail, including a greenbeard recognition based on belly coloration as well as 3 strategies: an orange aggressive strategy, a white cooperative strategy and a deceptive and cryptic yellow strategy (Sinervo et al. 2007).

Study Questions for Chapter 4

1. What are the five levels of selection and what is the continuum along which we rank the various levels of selection?

2. In the *t*-allele example, how is the force of genic selection balanced by the process of individual selection? How does the process of group selection favor non-t-allele mice?

3. Under what conditions will group selection overwhelm the force of individual selection? Discuss this in terms of Wynne-Edwards original proposal for explaining how self-limiting reproductive behaviors might evolve in a population.

4. What is the appropriate level of selection to consider for behavioral traits and why? Pick the most appropriate level or levels and explain your choice.

5. Why are social hymenoptera predisposed to "altruism" more so than most other organisms (see also Side Box)?

6. What is the whole-organism approach and what is its most persuasive argument regarding genic selection.

7. Describe Pfennig's experiment on kin selection in tiger salamanders in terms of the balance between kin and individual selection.

8. What are the 3 attributes of a Hamiltonian greenbeard. What do we call a nasty greenbeard? What do we call a nice greenbeard?

9. What are the conditions required for group selection to operate, and perhaps overwhelm the force of individual (and genic) selection?