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Social Selection

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Individuals sometimes give up their own resources to benefit their neighbors. Such altruistic traits posed a difficulty for the original Darwinian formulation of natural selection, which emphasized the spread of individually advantageous characters. So how have altruistic traits become common in some populations? This is an important question because a purely individualistic world would look very different from the one that we see. With no altruism, there would be no multicellularity with specialized nonreproductive tissues, no social insects with specialized worker castes, and nothing at all like complex human societies.

In this chapter, I discuss three processes that can promote altruism and the evolution of social cooperation. I start with kin selection, in which an individual may give up some of its own reproduction to aid relatives, or an individual may coordinate its behavior with phenotypically similar neighbors to promote the good of the group. Altruism toward kin helps to explain patterns of parasite virulence, sex ratios, and complex sociality with division of labor between different individuals.

Some groups build up a high level of social cohesion in spite of little relatedness and low opportunity for kin selection. In the second section, I discuss how repression of competition can be a powerful force integrating the interests of individuals. With no opportunity to compete against neighbors, an individual can only increase its own success by increasing the success of the whole group. Meiosis provides the classic example, in which the strict control of chromosomal segregation into gametes prevents competition between different chromosomes. It is only through such repression of competition between chromosomes that the genome developed into a highly integrated and cohesive unit.

In the third section, I turn to another key theme in the history of life—the evolutionary innovations of cooperative symbioses between different species or different kinds of genomes. The first genomes near the origin of life probably evolved by biochemical synergism between different replicating molecules; eukaryotic cells arose by symbioses between different species; and lichens, mycorrhizal–plant systems, and many other symbioses have contributed greatly to the complexity of modern life. The evolution of symbioses concerns the same social tensions between conflict and cooperation as the more familiar problems from kin groups and animal societies.

Cooperative symbioses may evolve by positive feedback between partners. In such synergistic relations, one party gives up some of its resources to enhance the success of its partner, and the partner does the same. The vast majority of cooperative symbioses arose as biochemical synergisms between organisms without complex behavioral flexibility. By contrast, the exchange of benefits between partners with the capacity for memory and the potential for strategy leads to issues of cheating, detection of cheaters, and strategic assessment of partner behavior and quality. Such problems of reciprocal altruism (Trivers 1971) pervade many aspects of vertebrate sociality, in which individuals remember particular partners and their past behaviors, and individuals can assess the complex strategies of others and form their own strategies in response. I do not cover behavioral reciprocity in this chapter, in order to focus on the more genetically relevant aspects of social selection that fit the themes of this volume.

My three topics of kin selection, repression of competition, and synergistic symbiosis all play fundamental roles in the evolution of genetic systems.

KIN SELECTION AND CORRELATED BEHAVIORS

Two different kinds of problems often arise when studying altruistic behavior in social interactions (Frank 1997b, 1998). First, an individual may give up some of its own reproduction to help kin increase their reproduction. For example, a young bird may remain with its parents and help raise its siblings rather than leave to set up its own nest and reproduce directly. This is the classic problem of altruism and kin selection.

Second, an individual may live in a group and face a tension between selfish and cooperative behaviors. For example, an individual may gain by selfishly grabbing a larger share of local resources but at a cost to the efficient use of those resources by the group. This trade-off between selfish gains for the individual and prudent benefits for all arises in problems such as the evolution of sex ratios and parasite virulence. Cooperation increases with the correlation in behaviors between group members. Correlation in behavior can arise for various reasons, of which kinship is often the most important.

The history of these subjects has turned on how to calculate when an altruistic behavior toward kin will increase, or how to calculate the optimum mixture of selfish and cooperative behavior in groups that share a common resource. The following sections give a sense of those calculations. I simplify the mathematics to emphasize the essential concepts. After developing the concepts, I illustrate the main ideas with a few examples.

Hamilton's Rule for Kin Selection

How can we determine whether a trait for self-sacrifice spreads in a population? Hamilton (1964a, b, 1970) took a population genetics approach by calculating when the allele frequency for an altruistic character increases. Suppose, for example, that an individual gives up its own opportunity to reproduce and instead uses its resources to help its sister. Do genes that reduce individual reproduction in this way increase or decrease over time?

Hamilton's calculation proceeds roughly as follows. The altruistic individual gave up the chance to make C babies and instead helped its sister to make an extra B babies. We need some measure of exchange to figure out how to weight the loss of the individual's own offspring and the gain in its sister's offspring. Our ultimate scale is change in allele frequency.

What effect does a loss of C babies by an individual have on the allele frequency of altruistic traits? This depends on the difference between the frequency of those alleles carried by the individual, q, and the allele frequency in the population, $\bar{q}$. Let us write this difference as $\delta = q - \bar{q}$. If the individual has the same allele frequency as occurs in the population, $\delta = 0$, then no matter how many babies this individual makes, there will be no consequence for the population allele frequency. In general, loss of C babies by our individual has allele frequency consequences in proportion to $-\delta C$, where the minus sign arises because this term represents a loss in reproduction.

What effect does a gain of an extra B babies by the individual's sister have on allele frequency change? If our focal individual has a allele frequency deviation δ, then on average the sister has a deviation $r\delta$, where r is the coefficient of relatedness of the focal individual to its sister (Box 23.1). Thus, a gain of an extra B babies by the individual's sister has allele frequency consequences in proportion to $r\delta B$.

We get the total effect on allele frequency change by combining the two terms, $r\delta B - \delta C = \delta (rB - C)$. If the total effect is greater than zero, then the frequency of alleles causing the altruistic behavior increases, and altruism spreads in the population. This gives us the famous result

$$rB - C > 0 \qquad (23.1)$$

known as Hamilton's rule for the spread of an altruistic behavior, where B is the benefit of the behavior directed toward kin related by r, and C is the cost of the behavior.

For example, an individual is typically related to its mother by $r = 1/2$. Thus an individual would be favored to forgo reproduction and stay with its mother if, for every lost baby of its own, $C = 1$, the altruistic individual added more than two offspring to its mother's reproduction, $B > 2$.

In this problem, individuals exchange direct transmission of genes for transmission by indirect routes via the extra reproduction of kin. The coefficient of relatedness, r, is the exchange rate that scales direct and indirect reproduction to obtain the ultimate

BOX 23.1. Coefficients of Relatedness

There is a vast literature on coefficients of relatedness in social interactions (Frank 1998). The two different kinds of social problems—self-sacrifice and correlated behaviors—are rarely distinguished in a clear way, causing the literature on this topic to be confusing and difficult to read. I will not attempt to review the literature or provide technical details here. Rather, I will outline the main concepts of relatedness in an intuitive way, using simple equations. Mastery of the subject requires deeper study, but the points here highlight the essential differences between the two types of social problems for which relatedness has been used.

The study of relatedness can be developed most naturally through the basic equation of linear regression. Simple regression predicts the value of a variable given a measurement on another variable. For example, we may predict the amount of rain given a measurement of cloudiness, or we may predict weight given a measurement of height.

The basic linear regression equation is

$$y = a + bx + \varepsilon,$$

where x is a value we measure, and y is the value we wish to predict given the measurement on x. In regression analysis, we predict y given a measurement of x as $\hat{y} \mid x = a + bx$. From regression theory, $a = \bar{y} - b\bar{x}$ and $b = \text{cov}(y,x) / \text{var}(x)$, where b is called the regression coefficient, and overbars denote average values.

We can use linear regression to predict expected values of y whenever we have paired observations (x, y). The values of a and b come from minimizing the average distance between the actual values of y and the predicted values, $\hat{y}$. No assumptions are required about the distributions of x and y; for example, they do not have to be normally distributed. Requirement of normality arises only in tests of statistical significance, not in developing predicted values of y that minimize the distance between prediction and observation.

The regression coefficient, b, provides a measure of conditional information about the variable we wish to predict. We can see this by rewriting the basic regression equation. First, define the deviation of x from its average value as $\delta = x - \bar{x}$. Then, using the above details about the standard form of regression, we can rearrange the terms as

$$\text{E}(y - \bar{y} \mid \delta) = b\delta,$$

which can be read as: the expected deviation of y from its average value, $\bar{y}$, given the deviation, δ, of the predictor variable x from its average value, equals the regression coefficient multiplied by the deviation of the predictor from its average, $b\delta$. In other words, the regression coefficient tells us how much y is expected to deviate from its average given a certain deviation of x from its average.

Now let us take the first kin selection problem of self-sacrifice, in which we need to measure allele frequency deviations from the population average. Suppose the average frequency of an altruistic allele is $\bar{q}$, and the frequency of the allele in the actor who may behave in a self-sacrificing way is q. What information about allele frequency in recipients of the altruistic act is contained in the fact that the actor has allele frequency deviation $\delta = q - \bar{q}$? Suppose the allele frequency in recipients is q'. Then the expected allele frequency deviation in recipients is

$$\text{E}(q' - \bar{q} \mid \delta) = r\delta,$$

BOX 23.1. *(cont.)*

where r is the relatedness coefficient, which is the regression coefficient from standard linear regression

$$r = \frac{\mathrm{cov}(q',q)}{\mathrm{var}(q)}.$$

There are more general ways of analyzing such problems that avoid assuming a single locus controls self-sacrifice. For example, one can formulate the theory in terms of breeding value from quantitative genetics (Frank 1997b, 1998). But in the more general theory, the basic use of regression remains the same. For self-sacrifice, relatedness measures the conditional prediction about genetic deviation of social partners from the population average given the deviation of the actor from that average. This provides the scaling needed to measure gains and losses in allele frequency in different classes of individuals.

The second type of problem concerns correlated behaviors in groups where all members express a behavior. This is more of a game-like situation in which behaviors are strategies that determine payoffs to individuals. The question is how an individual should adjust its behavior, such as its sex ratio, in order to maximize its own direct payoff.

Consider a group game, such as the tragedy of the commons or the sex ratio. Here, our focal individual has a behavior y, the actor's group has average behavior z which includes the contribution of the actor, and the population average is $\bar{z}$. Let the focal individual's deviation from the population average be $\delta = y - \bar{z}$. Then

$$\mathrm{E}(z - \bar{z} \mid \delta) = r\delta,$$

that is, the expected deviation of social partners from the population average, given the actor's deviation, is $r\delta$. Here, r measures conditional information about the behavior of social partners given the actor's own behavior. In this case, the regression coefficient is

$$r = \frac{\mathrm{cov}(z,y)}{\mathrm{var}(y)},$$

the regression (slope) of average group phenotype on actor phenotype. This gives r entirely in terms of phenotypes, which is what we need if we are interested in the immediate payoff to an actor when playing a game in which partners have correlated strategies. In evolutionary analysis, we are more interested in what the actor transmits to progeny, so we may choose to focus on the genetically transmitted value (breeding value) for the behavior, g, where g is roughly the expected contribution of the actor to the value of y in progeny (Frank 1998). We can do the analysis using g in place of y, giving

$$r = \frac{\mathrm{cov}(z,g)}{\mathrm{var}(g)},$$

which is the slope of partners' phenotype on actor's genotype. Partners will often have correlated behaviors because they are genealogical relatives. But genetic relatedness is not required, only an association between partner phenotype and the breeding value of the actor.

In the first model of self-sacrifice, the proper measure of relatedness is the regression of recipient genotype on actor genotype. This provides a measure of information

(continued)

BOX 23.1. *(cont.)*

about the genetic value transmitted by the recipient given the actor's genetic value (or allele frequency), allowing one to measure the total gains and losses in the transmission of genetic value. In the second model of correlated behavior, the proper measure of relatedness is the regression of the partners' phenotype on the focal individual's genetic value. This provides a measure of how partners' behaviors affect the transmission of the focal individual's genetic value.

In more complex social situations, the particular trait of an individual can affect reproduction by different kinds of recipient individuals. For those complex situations, it is often best to consider how the trait of an actor affects the fitness of the recipients in different classes. For example, a helper that remains with its parent may affect the fitness of its parent and of its siblings of different ages in the extended family.

If we would like to study the evolution of helper behavior, we must follow the effect of the helper's phenotype on the transmission of genetic value for the helping trait in the different classes of recipients. One can usually use the method for self-sacrifice discussed above, but it is often more natural conceptually and mathematically to formulate the problem in terms of the direct fitness method (Taylor & Frank 1996). With that method, the flow of effects goes from the actor's phenotype to the transmitted genetic value of the different classes of recipients. Consequently, the direct fitness coefficient of relatedness measures the regression of actor phenotype on the recipients' transmitted genetic value (Frank 1998).

The general conclusion is that relatedness measures for studying social evolution take on different forms of regression coefficients according to the flow of effects in particular analyses.

consequences for allele frequency change. The coefficient r can be thought of as a measure for the fidelity of transmission of genetic information via different pathways of direct and indirect reproduction—an extended form of the standard heritability coefficient of quantitative genetics (Frank 1997b, 1998).

Partitions and Scaling

In the previous section, I considered how an altruistic behavior may increase the transmission of genes in nondescendant lineages. Hamilton's rule for that problem partitions the total effect of a behavior on allele frequency change into two components: the effect, C, on the actor's reproduction and the effect, B, on the recipient's reproduction. The coefficient r provides the proper scaling so that rB and C give allele frequency effects on the same scale.

Whenever we wish to partition the total effect of some behavior into direct and indirect components, we will end up with three factors. First, we must measure the direct effect on the scale of interest. Second, we need the indirect effect, usually measured on some different scale. Third, we must render the indirect effect on the same scale as the direct effect so that we can obtain the total effect. For example, we used C for the direct effect, B for the indirect effect, and r for scale translation.

Given the general structure of partitioning into direct and indirect effects, we should not be surprised to find that different partitions all end up looking like Hamilton's rule. The next section provides an example of another partition that looks exactly like our first Hamilton's rule, yet has a very different meaning.

Correlated Behaviors and Cooperation in Social Groups

Consider an individual that interacts with a partner or with a small number of others in an isolated group. Each individual faces the essential tension of sociality. On the one hand, it can act selfishly to grab a larger share of the limited resources in its group, but selfish behavior causes inefficient use of local resources and lowers the total output of the group.

On the other hand, an individual can act altruistically, taking a smaller share of local resources and raising the total success of the group.

In this case, we are concerned only with how changes in an individual's behavior affect its own success. Let the individual's level of cooperation be y, such that larger values mean better cooperation with neighbors and smaller values mean greater selfishness. Let the average value of y in our focal individual's group be z, and let the average level in the population be $\bar{z}$. Thus, our focal individual's difference from the population average is $\delta = y - \bar{z}$. Higher δ means our focal individual is more cooperative than average and gives up a greater share of personal gain. The direct loss in success caused by cooperative behavior is $-\delta C$. Here, δ is the deviation from average phenotype and C scales between phenotype and a measure of success such as the number of offspring.

The group's deviation from the population average can be measured as $r\delta = z - \bar{z}$, where r is the slope of group behavior, z, on individual behavior, y. Here r measures phenotypic similarity—from an individual's point of view, it is a measure of information about the behavior of social partners (Box 23.1). When individuals in the group all act in the same way, then $r = 1$. When there is no association between individuals in a group of size N, then $r = 1/N$, because our focal individual is identical to itself and contributes a part $1/N$ to the group average.

The higher the group's level of altruism, z, the greater the benefit to our focal individual for living in a cooperative and efficient group. The expected group deviation from the population average is $r\delta$. Let the benefit per unit deviation be B, so the total benefit from group-level altruism is $r\delta B$. Thus, the total gain to an individual for increasing y, its level of altruism, is $r\delta B - \delta C$, which is positive when $rB - C > 0$.

This is the same expression as Hamilton's rule, but this expression must be interpreted differently from the prior result about allele frequency. Here, C is the direct cost of the individual's own altruism on its success. The term B is the indirect effect of the group's altruism and efficiency on our focal individual's success. The scale in this case is the individual's own success.

For each unit change in the individual's own phenotype, the group phenotype changes by r. Thus, I like to think of r as a measure of information about social partners. If r is high, then individuals acting altruistically have a high chance of associating with similar, altruistic partners. If r is low, then altruistic individuals often associate at random and will often have selfish partners that take advantage of them.

Loosely speaking, if one knows that partners will behave similarly to oneself, then acting altruistically and promoting group efficiency provide a direct benefit to oneself. By contrast, if one has little information about partners, then altruistic behavior will often be taken advantage of by selfish partners.

It is easy to be misled by the identical $rB - C > 0$ form of the expressions for the increase in altruism under self-sacrifice and under correlated behaviors (Frank 1997b, 1998). Whenever we partition total effects into two components measured on different scales, we end up with results that have the same $rB - C$ structure. In the two cases, the terms have different meanings. The first applies to allele frequency change when an actor behaves toward a neighbor, with the term r translating allele frequency deviations between actor and recipient. The second applies to an individual's success when actors and neighbors both act mutually in a social game of cooperation and selfishness, with the term r translating between individual- and group-level deviations from the average level of altruism.

We can use the second model for any sort of mutual interaction between individuals, including interactions between different species (Frank 1994b, 1998). Most often, however, the phenotypic similarities measured by r will arise from genetic similarity. By contrast, the first model makes reasonable sense only when applied to the behavior of individuals toward genetically similar recipients of the same species.

Applications to Self-Sacrifice

Suppose an offspring gives up its own opportunity to reproduce and instead helps its parents to raise more of its siblings. The most extreme case arises in sterile workers of social insects, but there are many examples in birds and mammals in which offspring spend at least part of their adult life aiding their parents. This is a clear case of the first model: self-sacrifice for a genetic relative.

Our theory tells us that we need to evaluate $rB - C > 0$ to determine when selection favors sacrifice of direct reproduction to help parents. At first glance, this seems to make a clear, simple,

and testable prediction about whether or not offspring will stay to help their parents. We do indeed have the right pieces, because our partition identifies the three key factors: direct reproduction, C, indirect reproduction, B, and scale translation, r. But quantitative evaluation can be difficult.

Consider a young bird that may either try to reproduce on its own or remain in its natal territory to help the current breeders (Emlen 1984; Brown 1987). How do we estimate loss in direct reproduction, C? This cost depends on demographic opportunities to obtain a territory, a mate, sufficient food to raise progeny, and the bird's vigilance to defend against predators. In addition, individual vigor and competitiveness vary, so we must account for each individual's particular attributes. And we must compare the value of an offspring raised alone with the value of an offspring raised on the natal territory, taking account of such issues as the potential for offspring to inherit their parents' territory.

In studying such problems, observers sometimes try to measure all possible factors needed to tally $rB - C$ and determine whether the balance for a young individual tips toward helping or going it alone. In my opinion, while the theory is valuable in calling attention to the relevant issues, there is little hope of measuring all factors that contribute to costs and benefits. Thus, consistency checks that seek to match the sign of $rB - C$ with individual behavior in a particular setting can be difficult to interpret.

I prefer simple comparative hypotheses and tests that respect the general, abstract nature of the theory. Comparative use of the theory can explain much of observed behavior, without trying to explain more than we are really able to do. For example, the greater the value of r, the more likely an individual will remain on its natal territory as a helper rather than try to reproduce on its own. The more severe the competition for establishing new territories or taking over existing ones, the more likely an individual will help rather than try to reproduce.

Applications to Correlated Behavior in Groups

The "tragedy of the commons" problem captures the tension between individual selfishness and group efficiency (Hardin 1993; Frank 1995b). Suppose each group has a common, renewable resource. The more each individual takes from the common pool, the greater its success. However, greater exploitation of local resources lowers total yield, so group productivity rises if individuals restrain their selfish tendencies. For example, a group of parasites may share a common host. The faster a parasite consumes resources and reproduces, the greater its share of the host. Rapid consumption may, however, overexploit the host, reducing host vigor or survival and lowering the total yield of the parasite group. In this tension between individual success and sustainable yield, parasite virulence may be shaped by a tragedy of the commons (Frank 1996).

A simple model for this problem can be written as

$$w(y,z) = \frac{y}{z}(1 - z) \qquad (23.2)$$

where an individual's fitness, w, depends on its selfish tendency to grab local resources, y, and the average selfish tendency in the local group, z (Frank 1994c, 1995b). The term y/z is the relative success of an individual within its group; for example, $y/z = 2$ means that the individual gets twice the average share. The term $1 - z$ is group productivity—the greater the average selfishness, z, the lower the group productivity.

Here the trait y is selfishness, so we may say that altruism increases as y declines. We know by the general theory of partitioning effects that altruism increases (y declines) when $rB - C > 0$, that altruism decreases when $rB - C < 0$, and that the system comes to equilibrium when $rB - C = 0$. So all we need to do is solve $rB - C = 0$ to determine what level of altruism tends to evolve in this situation. However, it is not obvious from looking at Equation 23.2 how to determine costs and benefits. Remember, in this situation we focus on an individual and measure the direct cost to its fitness of becoming more altruistic, the benefit to the focal individual from living in a group that shares its altruistic tendency, and the measure r that gives the translation between our individual's level of altruism and the tendency of its neighbors also to be altruistic.

There is a simple technique to extract the cost and benefit terms (Frank 1995b, 1998; Taylor and Frank 1996). The cost is the change in fitness, w, with respect to change in individual behavior, y, holding group behavior z constant. In mathematical terms this is $\partial w/\partial y = -C$, where the minus sign arises because costs enter into the total in a negative way. The benefit term is the change in w with respect z, holding y constant, $\partial w/\partial z = B$.

What we have done is step through the mathematical expression for the total change in fitness,

w, with respect to individual behavior, y, which can be expanded as

$$\frac{dw}{dy} = \frac{\partial w}{\partial y} + \frac{\partial w}{\partial z}\frac{dz}{dy} \qquad (23.3)$$
$$= -C + Br$$

where $r = dz/dy$ shows that the relation between group behavior and individual behavior is simply given by the slope from the derivative.

From all this we want the equilibrium behavior to which the population settles. At equilibrium, small changes in behavior do not increase fitness, otherwise the population would continue to evolve; thus we want $\delta w/\delta y = 0$, which is equivalent to $rB - C = 0$. At this equilibrium point all individuals have converged to the same, optimal behavior, so we evaluate the condition at $y = z = z^*$. Checking our calculus book to get B and C by applying Equation 23.3 to Equation 23.2, and solving $dw/dy = 0$, gives the equilibrium $z^* = 1 - r$ (Frank 1994c, 1995b). As the similarity in behavior within groups, r, rises, individuals lower their selfish tendencies, z^*.

I went through the steps in some detail, but the overall approach to understanding social selection is very simple. From the fitness expression in Equation 23.2, we take the slope of fitness on individual behavior in Equation 23.3 and solve at the equilibrium where the slope is zero.

Now let us analyze a sex ratio problem, focusing on the biology rather than the method. Sex ratio has been the most important problem for developing and testing the theory of social selection in group-structured populations (Hamilton 1967; Charnov 1982; Godfray & Werren 1996).

Suppose that several mated female insects (foundresses) land in a patch of resources and lay their eggs. The progeny emerge and mate among themselves. The males die, and the mated females fly off to find a new patch in which to lay their eggs. The problem concerns the ratio of sons and daughters produced by foundresses. To express the sex ratio in a consistent way, I use the number of males divided by the total number of progeny—the frequency of males. Making sons is a selfish act because males do not contribute to group productivity. Instead they compete in the mating arena with the sons of other foundresses for the local resource, which is unmated females. Making daughters is an altruistic act because it increases the pool of the local resource available for mating by males.

Let a focal foundress's sex ratio be y and the average sex ratio in a group be z. Our focal foundress's relative share of matings through sons is y/z, and the pool of available females is in proportion to $1 - z$, so total success through sons is proportional to $(y/z)(1 - z)$. Success through daughters is proportional to $1 - y$, the fraction of progeny that are female, assuming females do not compete in the local patch for food or space but instead compete after mating and dispersal.

Putting the terms together, fitness is

$$w(y,z) = \frac{y}{z}(1 - z) + 1 - y. \qquad (23.4)$$

Setting the derivative in Equation 23.2 to zero and evaluating at the equilibrium condition $y = z$ gives the optimal sex ratio as $z^* = (1/2)(1 - r)$ (Frank 1986). Note the similarity to the model for the tragedy of the commons, both in the expression for the male component of fitness and in the result.

If there are n foundresses in each patch, and a female's sex ratio is uncorrelated with her neighbors' sex ratios, then $r = 1/n$ and $z^* = (n - 1)/2n$, which is Hamilton's (1967) famous result for sex ratio under local mate competition. The result here based on r is more general because it shows how the sex ratio evolves when neighboring foundresses have correlated sex ratio behavior, perhaps because the foundresses are genetically related and sex ratio behavior is influenced by genotype.

Summary

In this section, I discussed two distinct processes. In the first case, individuals sacrifice their own reproduction to aid nondescendant genetic relatives. Hamilton's rule, $rB - C > 0$, partitions the consequences of self-sacrifice into a cost in direct reproduction, a benefit in the reproduction of relatives, and a scaling factor r that measures the genetic relatedness between altruist and recipient. The factor r can also be thought of as a scaling for the heritability of traits via nondescendant lineages compared with the heritability in direct reproduction—a measure of information about the transmission fidelity of characters via different pathways.

In the second case, individuals face a tension between their share of group resources and the efficiency of the group. Members of the group interact symmetrically, each having its own behavior that affects its success and the success of the group.

The consequences of each individual's behavior on its own reproduction can be partitioned into three terms. The first is a cost that measures the direct effects of the behavior on the individual's success, holding constant the group-level efficiency. The second is an indirect benefit that measures the effects of the average behavior in the group on group efficiency, holding constant individual behavior. The third is a scaling r that measures the similarity between an individual's behavior and the behavior of its neighbors—a measure of information about the behavior of social partners. Thus, r measures the extent to which a more cooperative individual tends to have more cooperative partners. In this case, the partners are not required to be genetic relatives, because payoff is measured entirely in terms of effects on our focal individual's total success.

The three terms of the second case can be combined into an expression for the increase in cooperative behavior, $rB - C > 0$. The similarity to Hamilton's rule for self-sacrifice is obvious, and indeed those of us who have developed this expression have called it a form of Hamilton's rule (Queller 1992; Taylor & Frank 1996; Frank 1997b, 1998). However, the two distinct processes of social selection—self-sacrifice for genetic relatives and cooperation in groups—give rise to similar expressions because the best method of analysis partitions success into costs, benefits, and a scaling factor, r. The similarity is both instructive and misleading; it is important to recognize the distinct biological interactions in the two cases and the different interpretations of r (Frank 1997b, 1998).

OTHER SOCIAL PROCESSES

I discussed the concepts of kin selection and correlated behaviors at length because they are the major forces of social selection. In the remainder of this chapter, I briefly introduce two additional topics. These topics build on the foundation of natural selection and kin selection to show how additional processes have shaped social interactions. I continue to use the word "social" in the broadest way, to cover all aspects of evolutionary change that deal with the tension between conflict and cooperation. Thus, social selection is important for understanding how different genetical elements came to be integrated into genomes, and how different cells became integrated into complex multicellular organisms.

REPRESSION OF COMPETITION

It would not make sense to speak of the genetic relatedness between genes on the Y chromosome and genes on chromosome 7 of the human genome. Those different genes are different kinds of things, almost like different species.

The genes on different chromosomes are integrated into a cohesive, cooperative group, yet they may also conflict over transmission to the next generation. For example, genes on the Y chromosome pass to the next generation only through sons, whereas those on chromosome 7 pass equally to sons and daughters. A Y chromosome that biased the sex ratio toward sons would increase in frequency. The genes on chromosome 7 in the same genome with the biasing Y would end up in sons. If a male bias developed in the population, sons would have lower fitness than daughters because the excess of males would be competing for relatively few females, and each male would on average be the father of less than one brood. So chromosome 7 would lose in transmission as the Y gained from causing a male bias.

In general, a chromosome can gain by increasing its transmission to offspring above the standard Mendelian fraction of one half (Lyttle 1991). Such segregation bias often imposes a fitness cost on the entire group of genes in the genome, either by sex ratio distortion or because the "driving" chromosome typically carries deleterious effects. Thus, the integration of the genome into a cohesive unit requires repression of the selfish transmission gains by subunits of the genome. The standard Mendelian segregation ratio of one half probably depends on mutual suppression of drive between chromosomes, repressing the potential for internal competition within the genome. With repression of the opportunities for gain against neighboring genetic elements, the only way that parts of the genome can increase their own success is by increasing the success of the entire group.

The Mendelian segregation ratio of one half for chromosomes is sometimes called fair meiosis, to emphasize that each chromosome has an equal or fair chance of being transmitted. Leigh (1971, 1977) was perhaps the first to emphasize that fair meiosis may have arisen to repress competition in the genome, thus integrating the reproductive interests of genomic subsets into a cohesive unit. Leigh (1977, p. 4543) expressed this idea rather colorfully when he said that the many genes of the genome repress biases of

individual chromosomes "as if we had to do with a parliament of the genes, which so regulated itself as to prevent 'cabals of a few' conspiring for their own 'selfish profit' at the expense of the 'commonwealth.'"

Leigh (1977) noted that alignment of individual and group interests shifts selection to the group level. However, meiosis was the only compelling case known at that time. Without further examples, there was no reason to emphasize repression of internal competition as an important force in social evolution. From the conceptual point of view, it may have been clear that repression of internal competition could be important, but not clear how natural selection would favor such internal repression.

Alexander and Borgia (1978) joined Leigh in emphasizing the potentially great potency of internal repression in shaping interests and conflicts in the hierarchy of life. From this, Alexander (1979, 1987) developed his theory of human social structure. In that theory, intense group-against-group competition dominated the success of humans and thus shaped societies according to their group efficiencies in conflicts. Efficiency, best achieved by aligning the interests of the individual with the group, favored in the most successful groups those laws that partially restricted the opportunities for reproductive dominance. For example, Alexander (1987) argues that socially imposed monogamy levels reproductive opportunities, particularly among young men at the age of maximal sexual competition. Those young men are the most competitive and divisive individuals within societies, and also the pool of warriors on which the group depends for its protection and expansion.

In the late 1970s, the concept of internal repression remained limited to meiosis and perhaps some aspects of human social structure. The concept could not gain attention as a potentially important process in the history of life without further examples. In the 1980s, three independent lineages of thought developed on social insects, cellular competition in metazoans, and domestication of symbionts. These different subjects would eventually contribute to a fuller understanding of the conceptual issues and biological significance of internal repression of competition (Frank 2003). Here, I briefly summarize only one of those subjects: cellular competition in metazoans.

Many multicellular animals are differentiated into tissues that predominantly contribute to gametes and tissues that are primarily nonreproductive. This germ–soma distinction creates the potential for reproductive conflict when cells are not genetically identical. Genetically distinct cellular lineages can raise their fitness by gaining preferential access to the germline. This biasing can increase in frequency even if it partly reduces the overall success of the group.

One way to control renegade cell lineages is with policing traits that enforce a germ–soma split early in development (Buss 1987). This split prevents reproductive bias between lineages during subsequent development. Once the potential for bias has been restricted, a cell lineage can improve its own fitness only by increasing the fitness of the individual. This is another example of how reproductive fairness acts as an integrating force in the formation of units.

Maynard Smith (1988) agreed with Buss's logic about the potential for cell lineage competition, but he argued that metazoans solved their problems of cell lineage competition by passing through a single-celled stage in each generation. When an individual develops from a single cell, all variation among subsequent cell lineages must arise by de novo mutation. In Maynard Smith's view, such mutations must be sufficiently rare that the genetic relatedness among cells is essentially perfect. Thus, the soma sacrifice reproduction as an altruistic act in favor of their genetically identical germline neighbors. Buss recognized the need for de novo mutations within an individual and argued that these would be sufficiently common to favor significant cell lineage competition and policing. Arguments on this topic continue (Michod and Roze 2001).

In summary, Leigh (1971, 1977) may have been the first to emphasize how repression of internal competition aligns individual and group interests. However, meiosis provided the only good example at that time, so the idea did not lead immediately to new insight. Alexander (1979, 1987) used the idea and the example of meiosis as the foundation for his novel theories about human social evolution. Buss's (1987) argument followed on the role of cellular competition and repression in the evolution of metazoans.

Buss stimulated Maynard Smith (1988) to consider how social groups became integrated over evolutionary history. Maynard Smith disagreed with Buss's particular argument about the importance of the germ–soma separation in metazoans. But in

considering the general issues, Maynard Smith had in hand several possible examples, including meiosis and genomic integration, limited cellular competition in metazoans, and the social insects. From these examples, Maynard Smith (1988, pp. 229–230) restated the essential concept in a concise and very general way:

> One can recognize in the evolution of life several revolutions in the way in which genetic information is organized. In each of these revolutions, there has been a conflict between selection at several levels. The achievement of individuality at the higher level has required that the disruptive effects of selection at the lower level be suppressed.

Disruptive effects may be repressed by high relatedness and kin selection, which favors self-restraint, or by repression of competition among unrelated or distantly related members of a group. Together, kin selection and repression of competition define the key evolutionary processes that have driven the major revolutions in the organization of genetic information (Maynard Smith & Szathmáry 1995).

SYNERGISTIC SYMBIOSIS

Gene products act within complex biochemical networks. From a social perspective of conflict and cooperation, biochemical networks usually pose no difficulties when the genes reside within a single genome. Each gene gains or loses in transmission along with its genomic neighbors—the group is bound by its common timing of replication and its common pathways of transmission. Adaptation has to do with engineering of biochemical networks for increased performance.

The smooth integration of genomes into biochemical networks obscures a great evolutionary puzzle. The earliest replicating molecules in the history of life probably did not live in integrated genomes with synchronous replication and common pathways of transmission (Maynard Smith & Szathmáry 1995). How did those individual replicators evolve to make complex, well-integrated biochemical networks? Put another way, how did those different species of early replicators come to act synergistically in symbiotic biochemical networks? In modern organisms, how do genes that reside in different species evolve synergistic symbioses?

The general problem of synergistic symbiosis can be studied by focusing on the joint evolution of two genetic loci (Frank 1995c, 1997c). The two loci may be in the same genome or in different species. I divide aspects of social selection into two parts. The first occurs when symbiont and partner have mutually beneficial effects on each other—a positive synergism between loci. The use of "locus" to describe partner and symbiont may seem a bit strange; it would seem more natural to say "a positive synergism between species." I use "locus" to emphasize that the symbiont and partner could be two different replicating molecules (genes or chromosomes) in a primitive genome or an insect and its bacterial symbiont.

The second part of social selection concerns various processes that bind together the reproductive interests of the two loci. The most obvious form of binding is physical, in which two separate replicators are joined together chemically to form a longer chromosome. The joined pair of loci may always be transmitted together, in which case their reproductive interests are completely aligned and they form a single evolutionary unit, as if they were a single locus. Or the loci may be shuffled occasionally by recombination, in which case they "codisperse" with a probability of one minus the recombination fraction. I have used standard genetical language, but physical binding might just as well cause a host locus and a symbiotic bacterial locus to codisperse, with shuffling defined by a parameter analogous to recombination.

Physical binding is easy to understand, but other types of association between pairs of loci have similar evolutionary consequences. Reproductive synchrony prevents competition and binds reproductive interests via the common timing of replication. Reproductive entrainment among chromosomes is certainly one of the outstanding features of mitosis and meiosis. These orderly cellular processes are derived conditions from the primitive state of scramble competition among a pool of unconstrained replicators.

Loci that have a positive synergism on reproductive success can develop statistical correlations between genetic variation at the loci (Frank 1994b). These correlations can arise even when there is limited codispersal. Such conclusions are well known in standard Mendelian population genetics. A pair of loci on separate chromosomes, recombining freely, will develop a statistical association when there is a positive or negative interaction between loci (epistasis). This statistical association is called

linkage disequilibrium. Thus, synergism creates associations between loci, and statistical association may have consequences similar to physical linkage.

This discussion emphasizes that symbiotic genetics shares many properties with standard, Mendelian genetics. But a generalization is required, removing the standard assumptions of meiotic reproductive synchrony and rigid patterns of codispersal.

Many models of cooperative symbiosis start with the assumption that each individual donates a fraction of its energy to aid partners. For example, hypercycle models assume mutual enhancement of replication by separate species of replicators and then study the conditions under which complex genomes can evolve (Eigen & Schuster 1979; Maynard Smith & Szathmáry 1995). Models for the origin of chromosomes start with the assumption of positive synergism between separate replicators and then ask when selection favors those separate replicators to become biochemically linked on chromosomes (Maynard Smith & Szathmáry 1993).

I studied the prior step in the evolution of cooperative symbiosis (Frank 1994b, 1995c): How do different loci first evolve to aid partner loci? This step must be passed before one can invoke synergism to study hypercycles, genomic integration, and the evolution of chromosomes. I emphasized the early evolution of genetic systems, but the models apply to any kind of cooperative mutualism with behaviorally inflexible traits (e.g., biochemical mutualism).

Two processes influence the origin of synergistic traits. First, both partners must have a minimal level of expression for their mutualistic trait. Second, pairs that develop positive synergism must be associated in space so that benefits conferred to a partner are returned to the initial donor. These spatial associations have two components: selection creates spatial association (linkage disequilibrium) in trait values between symbiotic partners (Frank, 1994b), and the benefits of cooperation, returned from partners, must be provided to relatives of the original donor (Hamilton 1972; Wilson 1980).

The initial level of trait expression and the spatial associations determine threshold trait values that are required for the origin and evolution of synergistic symbiosis (Figure 23.1A). Locus 1 has a trait, T_1, that enhances the reproductive rate of species 2 but reduces its own fitness. Likewise, locus 2 has a trait, T_2, that enhances the reproduction of locus 1 at a cost to itself. Larger values of T provide more benefit to the partner at a higher cost to the donor. When both loci have low trait values, as would be expected when the partners first meet, selection pressure continually pushes the traits to lower values. If, however, the pair of traits is above a threshold upon first meeting, then cooperation can increase because of synergistic feedback (Frank 1995c). Statistical association between loci increases the probability that a particular group will have a pair of symbionts above the threshold.

An example of how the benefit:cost ratio affects cooperative evolution is shown in Figure 23.1B. The benefit:cost ratio defines a scaling for the positive effect an individual has on its partner relative to its own cost. In this example, both partners start with the same trait value, T. If the benefit:cost ratio

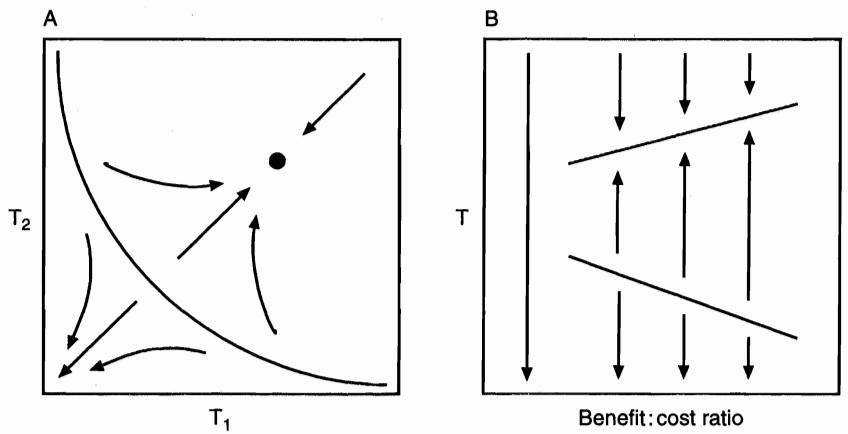

FIGURE 23.1. The threshold model for the evolution of synergistic symbiosis. For details see text. Reprinted from Frank (1997c).

is low, then selection reduces trait values from any starting point. As benefits increase relative to costs, the potential for positive feedback increases: lower trait values are needed to get over the initial threshold, and the traits evolve to higher equilibrium values.

This threshold is a key step in the origin of synergistic traits and cooperative symbiosis. Once the threshold is passed, symbionts may evolve through an irreversible stage, leading to an obligate relationship in which neither partner can live alone (Frank 1995c; Maynard Smith & Szathmáry 1995). By this process, genes with different phylogenetic histories can become integrated into complex biochemical networks.

DISCUSSION AND FUTURE DIRECTIONS

Genes act in biochemical networks. These social aggregations of genes define the environment in which reproductive interests play out in terms of replication and transmission—and in terms of conflict and cooperation. In this way, social selection dominates the evolutionary history of genetic systems.

At first glance, I may seem to be overstating the case. In the normal Mendelian system of classical population genetics, the rigid rules of replication, segregation, and transmission bind the interests of most genomic components into a unit that shares a common interest. The biochemical network can be studied from an engineering perspective of function without regard to social phenomena. However, the Mendelian condition derives from a social history of conflict and cooperation, in which replicating molecules developed systems of cotransmission, replicative synchrony, and reproductive fairness. The social processes of kin selection, repression of competition, and synergistic symbiosis melded the Mendelian genome into a cohesive unit.

Even modern Mendelian genomes fail to achieve complete unity (Werren et al. 1988; Hurst et al. 1996). Transposons violate the strict controls on replication and transmission. Uniparentally inherited mitochondria and other symbiont genomes often distort sex ratios. Chromosomes may gain transmission advantage by violating the normally fair segregation ratios. Each case shows the conflicting and common interests of different genomic components—the normally repressed social processes seething just below the surface of Mendelian rigidity.

Bacteria and viruses do not follows Mendel's rules. Their genetic systems remain more openly social, probably more like the primitive condition of genetic systems. For example, plasmids sometimes cotransmit with their bacterial hosts' genomes, joining plasmid and chromosome into a cooperative group. At other times, plasmids transmit horizontally as parasites (Levin & Lenski 1983). Multiple plasmids in a bacterial cell can be related as genetic kin; biochemical synergisms between different plasmids or between host and plasmid may favor combinations of genes to form more stable associations. Reproductive synchrony of plasmid and host genomes represses competition and tends to align the interests of genomic components.

Viral genetics also varies in non-Mendelian ways. For example, when multiple RNA viruses infect host cells, then there are multiple copies of each viral gene. Some viral genomes lose part of their genes through deletions. Those defective genomes can be copied when they coinfect with full genomes that provide the needed viral gene products (Holland 1990). The shortened genomes often replicate faster than the full genomes, probably because there is less RNA to copy. Thus, the infected cell produces proportionately more of the defective viruses than the wild-type viruses. This scenario matches the tragedy of the commons. When viruses coinfect with correlated genomes, then full genomes tend to go with full genomes, and defective genomes tend to end up with defective partners. The correlation between partners determines whether defective genomes match with full genomes sufficiently often to increase—in other words, the coefficient of relatedness plays a key role in the social evolution of viral genomes.

Other viruses have multipartite genomes, in which different components of the genome are packaged into separate particles (Matthews 1991). Viral success requires coinfection by all the different genomic pieces. This is a synergistic symbiosis in which the genes have become obligately entwined into cohesive biochemical networks, yet genomic components retain separate identities and interact as mutualistic symbionts.

Social selection also continues to be a dominant force in the more familiar types of sociality. But the powerful role of social selection in the history of

life and in the evolution of genetic systems is sometimes overlooked.

SUGGESTIONS FOR FURTHER READING

Frank (1998) provides detailed summaries and references on kin selection and inclusive fitness along with methods to solve problems of sex ratios, dispersal, and altruism. Maynard Smith and Szathmáry (1995) emphasize the role played by repression of competition in shaping the cohesiveness of social groupings and the creation of new evolutionary units in the history of life. The ongoing conflicts within groupings at the genomic level may have important consequences for several aspects of genomic organization (Hurst et al. 1996). Conflict within groups also influences parasite virulence, which Frank (1996) framed within the general theory of kin selection and life history evolution. Many aspects of evolutionary units, genome evolution, and transitions in the history of life depend on the tension between conflict and cooperation among symbionts (Frank 1997).

Frank SA 1996 Models of parasite virulence. Q. Rev. Biol. 71:37–78.

Frank SA 1997 Models of symbiosis. Am. Nat. 150:S80–S99.

Frank SA 1998 Foundations of Social Evolution. Princeton Univ. Press.

Hurst LD, Atlan A & BO Bengtsson 1996 Genetic conflicts. Q. Rev. Biol. 71:317–364.

Maynard Smith J & E Szathmáry 1995 The major transitions in evolution. Freeman.

Acknowledgments National Science Foundation grant DEB–0089741 and National Institutes of Health grant AI24424 support my research. I used material from Frank (1997c) and Frank (2003) with permission from the original sources.

Suggestions for future reading

Frank, S. A. 1996. Models of parasite virulence. Quarterly Review of Biology 71:37-78.

Frank, S. A. 1997. Models of symbiosis. American Naturalist 150:S80-S99.

Frank, S. A. 1998. Foundations of Social Evolution. Princeton University Press, Princeton, New Jersey.

Hurst, L. D., Atlan, A., and Bengtsson, B. O. 1996. Genetic conflicts. Quarterly Review of Biology 71:317-364.

Maynard Smith, J., and E. Szathmáry. 1995. The major transitions in evolution. Freeman, San Francisco.

References

Alexander, R. D. 1979. Darwinism and human affairs. Univ. of Washington Press, Seattle.

Alexander, R. D. 1987. The biology of moral systems. Aldine de Gruyter, New York.

Alexander, R. D., and G. Borgia. 1978. Group selection, altruism, and the levels of organization of life. Ann. Rev. Ecol. Syst. 9:449–474.

Brown, J. L. 1987. Helping and Communal Breeding in Birds. Princeton University Press, Princeton, New Jersey.

Buss, L. W. 1987. The evolution of individuality. Princeton Univ. Press, Princeton, NJ.

Charnov, E. L. 1982. The Theory of Sex Allocation. Princeton University Press, Princeton, New Jersey.

Eigen, M., and P. Schuster. 1979. The hypercycle: a principle of natural self-organization. Springer–Verlag, New York.

Emlen, S. T. 1984. Cooperative breeding in birds and mammals. In Krebs, J. R.; Davies, N. B. (ed), Behavioural Ecology: An Evolutionary Approach, 2nd ed., pages 305-339. Blackwell Scientific Publications, Oxford.

Frank, S. A. 1986. Hierarchical selection theory and sex ratios I. General solutions for structured populations. Theoretical Population Biology 29:312-342.

Frank, S. A. 1994a. Genetics of mutualism: the evolution of altruism between species. Journal of Theoretical Biology 170:393-400.

Frank, S. A. 1994b. Kin selection and virulence in the evolution of protocells and parasites. Proceedings of the Royal Society of London B 258:153-161.

Frank, S. A. 1995a. Mutual policing and repression of competition in the evolution of cooperative groups. Nature 377:520-522.

Frank, S. A. 1995b. The origin of synergistic symbiosis. Journal of Theoretical Biology 176:403-410.

Frank, S. A. 1996. Models of parasite virulence. Quarterly Review of Biology 71:37-78.

Frank, S. A. 1997a. The Price equation, Fisher's fundamental theorem, kin selection, and causal analysis. Evolution 51:1712-1729.

Frank, S. A. 1997b. Models of symbiosis. American Naturalist 150:S80-S99.

Frank, S. A. 1998. Foundations of Social Evolution. Princeton University Press, Princeton, New Jersey.

Frank, S. A. 2003. Repression of competition and the evolution of cooperation. Evolution 57:693-705.

Godfray, H. C. J. and Werren, J. H. 1996. Recent developments in sex ratio studies. Trends in Ecology and Evolution 11:59-63.

Hamilton, W. D. 1964a. The genetical evolution of social behaviour. I. Journal of Theoretical Biology 7:1-16.

Hamilton, W. D. 1964b. The genetical evolution of social behaviour. II. Journal of Theoretical Biology 7:17-52.

Hamilton, W. D. 1967. Extraordinary sex ratios. Science 156:477-488.

Hamilton, W. D. 1970. Selfish and spiteful behaviour in an evolutionary model. Nature 228:1218-1220.

Hamilton, W. D. 1972. Altruism and related phenomena, mainly in social insects. Annual Review of Ecology and Systematics 3:193-232.

Hardin, G. 1993. Living within Limits: Ecology, Economics, and Population Taboos. Oxford University Press, Oxford.

Holland, J. J. 1990. Defective viral genomes. In Virology, Fields, B. N., ed, 2nd ed, pp. 151-165. Raven Press, New York.

Hurst, L. D., Atlan, A., and Bengtsson, B. O. 1996. Genetic conflicts. Quarterly Review of Biology 71:317-364.

Leigh, Jr., E. G. 1971. Adaptation and diversity. Freeman, Cooper, San Francisco.

Leigh, Jr., E. G. 1977. How does selection reconcile individual advantage with the good of the group? Proc. Natl. Acad. Sci. USA 74:4542–4546.

Levin, B. R. and Lenski, R. E. 1983. Coevolution in bacteria and their viruses and plasmids. In Futuyma, D. J.; Slatkin, M. (ed), Coevolution, pages 99-127. Sinauer Associates, Sunderland, Massachusetts.

Lyttle, T. W. 1991. Segregation distorters. Annual Review of Genetics 25:511-557.

Matthews, R. E. F. 1991. Plant Virology, 3rd ed. Academic Press, New York.

Maynard Smith, J. 1988. Evolutionary progress and levels of selection. Pp. 219-230 *in* M. H. Nitecki, ed. Evolutionary progress. Univ. of Chicago Press, Chicago.

Maynard Smith, J., and E. Szathmáry. 1993. The origin of chromosomes I. Selection for linkage. Journal of Theoretical Biology 164:437-446.

Maynard Smith, J., and E. Szathmáry. 1995. The major transitions in evolution. Freeman, San Francisco.

Michod, R. E., and D. Roze. 2001. Cooperation and conflict in the evolution of multicellularity. Heredity 86:1–7.

Queller, D. C. 1992. A general model for kin selection. Evolution 46:376-380.

Taylor, P. D.; Frank, S. A. 1996. How to make a kin selection model. Journal of Theoretical Biology 180:27-37.

Trivers, R. 1971. The evolution of reciprocal altruism. Quarterly Review of Biology 46:35-57.

Werren, J. H., Nur, U., and Wu, C.-I. 1988. Selfish genetic elements. Trends in Ecology and Evolution 3:297-302.

Wilson, D. S. 1980. The natural selection of populations and communities. Benjamin/Cummings, Menlo Park, California.