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LONG ARTICLE

# The Altruism Paradox: A Consequence of Mistaken Genetic Modeling

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**Abstract** The theoretical heuristic of assuming distinct alleles (or genotypes) for alternative phenotypes is the foundation of the paradigm of evolutionary explanation we call the Modern Synthesis. In modeling the evolution of sociality, the heuristic has been to set altruism and selfishness as alternative phenotypes under distinct genotypes, which has been dubbed the "phenotypic gambit." The prevalence of the altruistic genotype that is of lower evolutionary fitness relative to the alternative genotype for non-altruistic behavior in populations is the basis of the "paradox of altruism." I show in this article that the assumption of contrasting genotypes for altruism and selfishness in our "phenotypic gambit" is inconsistent with the empirical data when viewed in the light of today's post-Mendelian understanding of gene expression. I demonstrate that however nuanced and sophisticated the models may have become today, they are still rooted in that fundamentally problematic assumption. I then offer a genetic conception of altruism that best fits the field data.

**Keywords** Altruism · Phenotypic gambit · Phenotypic plasticity · Social evolution

## Introduction

Alger and Weibull (2012, p. 42) have suggested that "when evolution operates at the level of behavior rules rather than directly on acts, as is usually assumed, the level of cooperation generally violates Hamilton's rule at the behavioral level." By "evolution operating directly on acts" they mean "when a trait is an action always to be taken," as opposed to the situation in which the trait expresses one behavior or another contingent upon some exogenous factor. My argument in this article is that, whereas our current models generally fall under the former category, as Alger and Weibull (2012) concur, the empirical data overwhelmingly suggest the latter to be the real state of affairs in nature. In their recent paper, "The Evolution of Eusociality," Nowak et al. (2010) proposed a model consistent with the latter conception, and were severely critical of Hamilton's theory. In the rancorous debate that ensued, van Veelen et al. (2010) urged a return to basics, rigor in analysis, and empirical testing of theories and assumptions. We see all the more reason why, in the following discussion.

The "paradox of altruism" is arguably the most enduring riddle in evolutionary biology. In extant conceptualization, it fits what William (1981, p. 164) describes as "the classic problem … of a mechanism by which a behavior can evolve (genetically) even though it lowers the fitness of the individual engaging in this behavior." But why is that a problem? Darwin's ([1859] 1998) theory of natural selection predicts, via mathematical models pursuant to the Modern Synthesis, that a fitter (genetic) trait would increase in frequency in a population while the less fit alternative would be eliminated. Thus, the paradox arises because we suppose distinct genetic factors for altruism and selfishness in a situation where the less fit "altruistic allele" is persistent, contrary to the Darwinian theoretical prediction that it should perish. It is none other than this conception that gives occasion to theories such as kin selection, group selection, evolutionarily stable strategies (ESS), etc., all of which came about on account of the perceived paradox of a flourishing maladaptive altruistic trait. In this article I point out that the most fundamental

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assumption under which we model the evolution of altruism is itself problematic; correcting it could dissipate the paradox and consequently obviate the need for special mechanisms for the evolution of altruism.

## The Phenotypic Gambit

Grafen (1984) has noted that our modeling of the evolution of behavioral phenotypes involves a "phenotypic gambit"—that there is a haploid locus at which each distinct phenotype is represented by a distinct allele. Grafen raises some concerns as well as some hopes about the phenotypic gambit. He explains that due to the dearth of our knowledge regarding the exact relationships between genotypes and phenotypes of behavioral traits, building population-genetic models for such traits would be quite impossible without making such a gambit. Grafen indicates however, that "taken literally the gambit is usually false" (1984, p. 64), and that "it is a leap of faith" we take (p. 65). However, the phenotypic gambit as I see it is likely false only if we think of it as a phenotypic "gamble," as Grafen's discussion seems to suggest. If under our phenotypic gambit we want to imagine the single haploid locus as representing the totality of a complex behavior such as altruism, then it is most certainly false. Grafen (1984) explains, however, that we nevertheless need to conceptualize it in that manner on grounds of theoretical expedience. I think not, for the following reason.

Darwin ([1859] 1998, pp. 112, 160) describes evolution by natural selection as happening through a cumulative series of small, imperceptible changes. If every one of these small changes has to be heritable, as Darwin insists, then they will each have to be inscribed by some heritable factor, which in today's understanding will be a single mutation or nucleotide substitution. When we adopted Mendelian particulate genetics under the Modern Synthesis, as opposed to blending inheritance, we essentially quantized heredity, and consequently, the evolutionary process. Thus, our phenotypic gambit should be seen as a "zoom-in" on just a single one of the hundreds or perhaps thousands of discrete but imperceptible heritable steps by which a trait evolves. Each discrete step should coincide with a single nucleotide substitution. Think of this evolutionary step as one of the series of infinitesimal changes that we imagine in calculus to collectively constitute a curve. In trying to understand how a curve of any shape comes about, we isolate one of the infinitesimal steps that make up the curve and describe it to represent the manner in which the curve is formed. Similarly, an evolutionary model describes the manner of evolutionary change, and it necessarily has to describe the character of the steps that constitute the change.

The models in which we make the phenotypic gambit therefore describe the conditions under which such an evolutionary step in a particular direction might occur. Our models should be conceived as suggesting, consistent with Darwin ([1859] 1998, pp. 112–113), that if such conditions persist, more of such steps would occur, and the cumulative effect would be a visible phenotypic change. Viewed in this sense—i.e., a phenotype as a collection of advantageous heritable variations, one of which our evolutionary model describes—rather than the totality of the phenotype, our phenotypic gambit would more likely be true than false. In fact, any trait whose evolution cannot be simplified into this format is likely non-Darwinian. Therefore, when we make our phenotypic gambit that depicts a single haploid locus, we are actually dealing with just a "quantum bit" of evolutionary change, a long series of which ultimately yields the phenotype whose evolution we are modeling.

Unfortunately, our population-genetic models are often not clear on this point; in fact, they are generally misleading. Evolution by natural selection is actually a two-dimensional progression. The first dimension is the "calculus analogy" I have discussed above. It is the gradual accumulation of favorable heritable variations (Darwin [1859] 1998, pp. 137, 141), which leads to the intensification, magnification, actualization or ultimate manifestation of the trait. The other dimension of evolutionary change is the spread or penetration of the advantageous variation in the population, which is the gradual supplanting of the individuals without the favorable variation by those who have that variation (p. 154), leading to the ultimate fixation of the trait. However, when we define and model evolution under the Modern Synthesis simply as a change in gene frequencies, where the genes/alleles are assumed to represent complete behavioral phenotypes/strategies, we reduce a two-dimensional progression into one. For example, we generally purport to model the evolution of cooperation by describing the conditions under which one full-fledged and well-formed complex strategy may successfully invade another. The models thus collapse the numerous steps involved in the intensification process into a single step or a single mutation and then describe how it spreads though the population to eventual fixation. As Grafen (1984, p. 64) puts it, we proceed "as if enough mutation occurred to allow each strategy to invade."

However, Darwin ([1859] 1998, p. 160) placed the essence of natural selection in the intensification process, saying that "natural selection acts exclusively by the preservation and accumulation of variations which are beneficial." He adds further that "it seems as improbable that any part should have been suddenly produced perfect" (p. 66), and that we should expect "monstrosities" (sudden

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major deviations in structure) to be rare (p. 121). This is why any evolutionary model that supposes an entire complex trait to be represented by a single haploid locus is quite un-Darwinian. It would be as if the sequential nucleotide substitutions that occurred at numerous loci all happened in one flash to create the phenotype. Grafen (1984) is thus right in saying that the phenotypic gambit is likely false, but I do not agree with him that it is only by such a false simplification (what he calls a caricature) that we are able to model the evolution of behavior by natural selection. As the discussion above has shown, this particular problem is fixed if we re-cast our conception of the phenotypic gambit according to the "calculus analogy" I have described above.

## The Phenotypic Gambit and Altruism

An additional difficulty arises from the way we frame the phenotypic gambit for modeling the evolution of sociality. It is abundantly clear from the literature (see the next section) that in modeling the evolution of altruism, most models take selfishness to be the alternative phenotype with which the altruistic phenotype vies for evolutionary ascendency. As a consequence, our "phenotypic gambit" has been to assume corresponding competing alleles for the two phenotypes. This is another problematic aspect of our modeling assumption, as it is starkly contrary to the field data on altruism, properly interpreted in the light of today's understanding of gene expression. There have been concerns raised over this state of affairs (West-Eberhard 2003).

In fact, the empirical data presented here will show consistently that the altruistic and selfish phenotypes are plastic expressions of a single genotype under alternative environmental circumstances. If the altruistic and selfish phenotypes are thus tied to a single genotype, they cannot be competing alternatives in populations as today's models cast them. Had we recognized this empirical fact as the fundamental genetic principle in altruism, and had we had that empirical reality reflected in our genetic models rather than the contrasting allele assumption, the theoretical landscape of the evolution of altruism would look much different, and certainly much less complicated and problematic than it is today.

Some behavioral ecologists tend to be quite sensitive to discussions of genes and behavior because of the associated issue of genetic determinism. However, supposing separate genes for two contrasting phenotypes does not necessarily entail genetic determinism (Okasha 2009). It is one thing to acknowledge that we make an assumption that is "literally false," and defend it on grounds of theoretical expedience (Grafen 1984), or explain what it should not be misconstrued for (Okasha 2009). It is another to deny altogether, as some are inclined to do, that our standard models make such an assumption, or to charge that this kind of criticism sets up a "straw man." People who hold this latter view contradict the preeminent scholars on this subject, most of whom will be quoted below as clearly espousing the contrasting allele view for altruism and selfishness. In fact, the next section is devoted to proving the prevalence of the genotypic dichotomy assumption, which would not have been necessary here, were it not for the tendency in some quarters to deny that our standard evolutionary models hinge on such an assumption, or to suggest that there are "nuances" in the assumption that make it reasonable.

## Extant Genetic View

In his review of Sober and Wilson (1998), Maynard-Smith (1998, p. 639) notes that "there are two kinds of individual[s]: altruists, who benefit others at a cost to themselves; and non-altruists who do not. A field example of these two distinct individuals is given by Okasha (2009), who writes: "To see this, imagine that some members of a group of Vervet monkeys give alarm calls when they see predators, but others do not." In this example, those individuals who call the alarm are the altruists, and those who do not call the alarm are selfish. This binary conception of phenotype as well as genotype (Van Veelen et al. 2012, p. 68) is standard in the modeling of altruism. The preferred terms in game-theoretic models are "cooperators" and "defectors."

The critical question is: what is the genetic relationship between such altruistic and selfish individuals (or strategies) in a population? Is it as described by William (1981): "differences among phenotypes are causally associated with genotypic differences (in other words) genetic differences underlie phenotypic differences"? This is one of the least explored empirical and theoretical questions of evolutionary research. Grafen (1984, p. 65), suggests that even though the behavioral ecologist relies on population genetics, "our method is designed to avoid doing genetics." Hence, according to him, the behavioral ecologist takes a leap of faith, and goes with a "phenotypic gambit" that there is an allele for one phenotype and a contrasting allele for the other. Knowing the exact genetic details may indeed be impossible, but we should not altogether ignore even the low-hanging fruits of empirical research that suggest what the broad genetic relationships may be. Our phenotypic gambit becomes a true "caricature of reality" (Grafen 1984; Gardner et al. 2011) only if it contains assumptions that are contrary to the empirical facts at hand. It is the source of what West-Eberhard (2003, p. 3) observed as the "gap between the conclusions of the genetical theory of the origin and spread of a new trait and the observed nature of the trait being explained." The

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gambit of assuming contrasting alleles for contrasting phenotypes as applied to altruism implies that the altruist and non-altruist are distinguishable genetically by the possession (or lack thereof), of "a gene for altruism." In other words, we assume a genetic basis for the contrasting behaviors of altruism and selfishness.

In explaining kin selection, Okasha (2009), for one, invites us to "imagine a gene which causes its bearer to behave altruistically towards other organisms." He suggests that "organisms without the gene are selfish," and then goes on to say that "the altruists will be at a fitness disadvantage, so we should expect the altruistic gene to be eliminated from the population." This renders most concisely the problem of altruism as conceived by evolutionary biologists today, and is posted under "Biological Altruism" in the *Stanford Encyclopedia of Philosophy*. That is, an altruistic gene that codes for the weaker altruistic phenotype thrives in competition against a selfish gene that codes for the fitter selfish phenotype—hence, a paradox. It would be hard to imagine any nuance or semantic spin that could be put on such statements to mean anything other than that we suppose a gene for altruism and a separate gene for selfishness. I am aware of no other basis ever adduced for the paradox of altruism. From the angle of group selection, Wilson and Wilson (2007, p. 329) render the problem exactly the same way, as they write: "a heritable trait that increases the fitness of others in a group (or the group as a whole) at the expense of the individual possessing the trait will decline in frequency within the group."

Okasha (2009) vehemently denies that "genetic determinism" is entailed in this kind of genetic supposition in evolutionary models. He writes: "Kin selection theory does not deny the truism that all traits are affected by both genes and environment." I make no charge of the kind of genetic determinism Okasha denies. In fact, it is true that some models do offer a scenario in which "having the cooperative genotype only implies a certain probability of expressing it" (van Veelen et al. 2012, p. 68). However, such models (often called conditional altruism models) do not set this scenario as something contrary to the contrasting genotypes view. In fact, they often do so while at the same time maintaining separate genotypes for the two phenotypes, which is actually my worry.

Consider, for example, Trivers' (1971, p. 36) reciprocal altruism model, which hinges upon conditional expression of the altruistic genotype. Yet, he sets the following as the genetic assumption in the model: "Assume that the altruistic behavior of an altruist is controlled by an allele (dominant or recessive), $a_2$, at a given locus and that (for simplicity) there is only one alternative allele, $a_1$, at that locus and that it does not lead to altruistic behavior." (Other examples of models that claim an altruistic gene that is non-deterministic but distinct from the selfish gene include Alger and Weibull 2012; Hamilton 1964; Queller 1985; van Veelen et al. 2012.)

Those models aside, straightforward declarations of the "contrasting genotypes" supposition are also ubiquitous in the literature. On how kin selection explains altruism, Curry (2006, p. 683) writes: "Well, genes for altruism can spread if they help copies of themselves that reside in other individuals." Bowles (2006, p. 1569) supposes in his group selection model that "(**A**) individuals are bearers of a hypothetical "altruistic allele"; those without the allele (**N**s) do not behave altruistically." In Haldane's (1932, p. 208) model, aa is the recessive character that causes altruistic behavior. Similarly, Rousset and Roze (2007, p. 2321) engage in a very elaborate mathematical analysis of the possible evolutionary outcome of a "helping allele (H0)" versus a cheating allele (H1)." Sober (1984, p. 184) supposes an altruistic trait "A–one that causes individuals with the trait to benefit others at their own expense." In one of the most recent papers on the subject, Gardner et al. (2011, pp. 1029–1030) give one of the most sophisticated analyses of the major altruistic models to date; in setting up the assumptions for the analysis, they write:

> We assume an infinite population of haploid individuals engaged in two-player games. A single locus controls the cooperation phenotype, with a proportion p of individuals carrying an allele A which encodes the cooperator strategy, and the remaining 1 − p carrying an allele a, which encodes the non-cooperator strategy.

This common supposition of altruism and selfishness as genetic allelomorphs in extant models emanates from the fundamental population-genetic template for modeling evolution through gene frequency changes as shown in Table 1 (from Halliburton 2004, p. 133).

In modeling the evolution of altruism, extant models build upon this template, in which they usually assume altruism and selfishness to be the contrasting alleles $A_1$ and $A_2$—the phenotypic gambit. This is why I have referred to this approach by extant evolutionary models of altruism as the Altruism Selfishness Allelomorphism (ASA) models. The alternative I propose is what the empirical data suggest, i.e., altruism and selfishness as alternative phenotypes of a single plastic genotype, hence, the Altruism Selfishness Plasticity (ASP) model. In the latter, we cannot

**Table 1** Allele frequencies chart (from Halliburton 2004, p. 133)

| Genotype | $A_1A_1$ | $A_1A_2$ | $A_2A_2$ |
|---|---|---|---|
| Frequency | $P^2$ | 2pq | $q^2$ |
| Fitness | $w_{11}$ | $w_{12}$ | $w_{22}$ |

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represent altruism and selfishness separately as $A_1$ and $A_2$ in Table 1, since they are of one and the same genotype, as the empirical data suggest. The consequence of ASA is that the distinct "altruistic allele" would be of lower evolutionary fitness and therefore ought to decline in frequency. This was what led Hamilton (1964, p. 16), who clearly held that ASA conception of altruism, to declare that the existing mathematical models did not allow for the evolution of sociality (p. 1), and consequently some mechanism was needed that would offset the apparent decline of the altruism allele.

Like Hamilton and his inclusive fitness hypothesis, most of our extant models of altruism (including those which cautiously talk only of phenotypes) are driven by the need to provide some countervailing mechanism to this theoretically predicted attenuation of altruistic allele frequencies in populations. From the group-selectionist camp, Wilson and Wilson (2007, p. 329) assert that "something more than natural selection within single groups is required to explain how altruism and other group-advantageous traits evolve by natural selection"; the group selection answer is that the "within-group" disadvantage of the altruist is counteracted by the between-group advantage of the group with altruists. In the parlance of game-theoretic/ESS models, we may read statements such as this from Taylor and Nowak (2007, p. 2281) that "Cooperation is always vulnerable to exploitation by defectors; hence, the evolution of cooperation requires specific mechanisms, which allow natural selection to favor cooperation over defection" (see also Allen et al. 2012 and Nowak 2012). This is exactly the problem Hamilton (1964) pointed out five decades earlier, and such statements are driven by the thought of a distinct altruistic allele (gene/genotype) that is in danger of being overrun by a distinct selfish allele. In Dawkins' (1976/1989, p. 184) metaphor, "cheat genes" are spreading through the population while "sucker genes" are driven to extinction. Wilson (2005, p. 159) summarizes the problem thus: "How might such a behavior evolve if the genes promoting it are at such a disadvantage in competition with genes that oppose it?"

Nevertheless, the less-fit altruistic allele is prevalent in natural populations, and this presents an anomaly to explain. Suggested mechanisms of how this could have come about include: (1) altruists associating exclusively with other altruists (Maynard-Smith 1998; Sober and Wilson 1998); and (2) conditional deployment of the altruistic behavior, i.e., only towards genetic relatives (Hamilton 1964), or towards other altruists (Trivers 1971). Dawkins (1976/1989, p. 89) simplifies the concept for a popular science audience with the "green beard" metaphor, in which we are to imagine altruists identifying other altruists by a characteristic green beard. For group selection models, Godfrey-Smith (2009, p. 174) explains that if social groups are formed randomly, "the **A** (altruistic) type is lost regardless of the details." However, the altruist can be maintained, he explains further, "if groups are formed in a way that 'clumps' the two types, so like tends to interact with like [and] the benefits of having '**A**s' around tend to fall mainly on other **A**s" (p. 174). For ESS game-theoretic models, Burton et al. (2012, p. 55) state: "one general answer is that interactions need to be assortative, so that individuals carrying genes coding for cooperation interact, on average, more often with cooperating individuals than individuals carrying genes coding for defection." Similarly, "clustering" is called for in the latest ESS models using "evolutionary graph theory" so that "the benefits of cooperation are received mostly by other cooperators" (Allen et al. 2012 and references therein). If these are the "nuances" suggested in the latest models, they do not renounce the contrasting genotypes assumption. Rather, they suggest how cooperation may still occur under that assumption.

All the safeguards that are sought by extant (ASA based) models with a view to exclude the selfish individuals in the population from benefiting from altruistic acts stem from the supposition that the two phenotypes are of rival genotypes. However, all these exclusionary mechanisms would not be necessary if such models had a genetic conception of altruism that is consistent with the field data, in which the selfish individuals also carry and transmit the altruistic gene, as the ASP model will show to be the case. This is why we cannot take the matter of the genetic relationship between the contrasting phenotypes in such populations lightly. For if it is indeed the case that such phenotypes are plastic expressions of a single genotype, then the exclusionary mechanisms which have been the focus of the evolutionary modeling of altruism in the last five decades are actually not necessary.

If we subscribe to phenotypic plasticity as the genetic basis of altruism and selfishness, then the phenotypically selfish individuals in the population can also transmit the altruistic gene and need not be excluded from receiving altruistic acts. This is the case with the selfish queens in eusocial organisms, as we shall see later. As I shall also show later, even in non-eusocial altruistic societies, such as human, baboon, or ground squirrel societies, selfish individuals have the capacity to reproduce altruistic individuals, and can even perform altruism themselves given the right social circumstances. Conditional altruism models do not see it this way, because they generally consider altruistic and selfish individuals to be genotypically different.

It is therefore very critical to evolutionary modeling to ascertain whether it is indeed by virtue of a genotypic difference that some individuals in a population behave altruistically while others behave selfishly. If we say the worker bee is altruistic and the queen bee is selfish; if a

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ground squirrel that sounds the alarm is altruistic and the adjacent squirrel that does not is selfish; and if also by altruism we mean the vampire bat that shares blood with a roost mate, while the bat that refuses to share we call selfish; if these are the criteria by which we designate altruists and non-altruists in populations—and in fact, these are some of the most compelling cases of the altruism we model—then it would be quite wrong to adopt a modeling assumption that there is an allele for altruism and a contrasting one for selfishness. For as we shall see below, there is enough in the field data to establish that the altruistic and selfish traits are of the same genotype rather than separate. Our "phenotypic gambit" in modeling the evolution of altruism right now is blatantly contrary to this empirical reality. In the sections that follow, I present an analysis of the empirical data, which shows that the altruistic and selfish phenotypes are not of separate genotypes.

## Genes and Environment

The interplay of genes and the environment in shaping phenotypes is not new in science today. We now know also that there are many genes that would not express phenotypically unless triggered by some environmental cue. In such cases, two individuals carrying the same gene could nevertheless differ phenotypically with respect to that genotype due to differences in their environmental experiences. Figure 1 (from Agrawal 2001) shows two clones of the water flea *Daphnia lumholtzi*. The individual on the left with the spiny helmet and longer tail spine was raised in an environment in which chemical cues from a predacious fish were introduced. The other clone (on the right) was the control. The experiment demonstrates that any individual from this species can assume either phenotype depending on whether it is growing in an environment with predators or in one without predators. Hence, the two phenotypes, even though once thought to be separate species, actually do not differ with respect to the genotype for helmet.

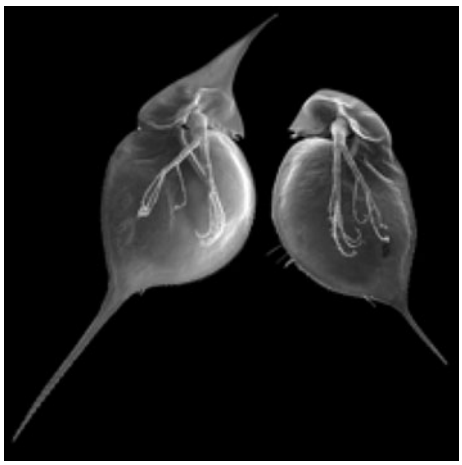

**Fig. 1** Clones of *D. lumholtzi* (from Agrawal 2001)

The property of a given genotype to produce different phenotypes in response to distinct environmental conditions defines *phenotypic plasticity* (Pigliucci 2001, p. 1). In the case of *Daphnia* it is helmets that are expressed in response to the presence of a predatory fish. There are many other examples of phenotypic features that are expressed only under certain environmental cues. Other such cues include: parasites (Moore 1995), diet (Greene 1989; Pfennig and Murphy 2000); predators (Lively 1986; Agrawal 2001); competition (Harvey et al. 2000); population density (Deno and Roderick 1992); temperature (Morreale et al. 1982; Roff 1986). In all of these examples, as in *Daphnia*, a single genotype expresses one phenotype or another, depending on the presence or absence of specific environmental cues. Clearly, any evolutionary model that assumes a genotypic difference between such dimorphic phenotypes would simply be incorrect. For example, in Table 1 it would be wrong to designate helmets as allele $A_1$ and non-helmets as allele $A_2$. It would also be fundamentally wrong to even conceive of the two phenotypes as competing evolutionary alternatives. In the sections that follow, I will try to persuade the reader that the field data suggest that we model the evolution of the two phenotypes of altruism and selfishness as we would do for helmets and non-helmets in *Daphnia*.

How could we talk of a declining altruistic allele against a fitter selfish allele in the polyembryonic wasp (*Copidosoma floridanun*), for example, in which clones from a single embryo differentiate into altruistic soldiers who do not reproduce but defend the selfish ones who reproduce (Donnell et al. 2004)? Other such cases of clones differentiating into altruistic and selfish individuals have been reported in gall aphids (Ito 1989; Abbot et al. 2001). It is clear in these cases that altruism and selfishness are indeed plastic expressions *of* a single genotype, since the two phenotypes are expressed by different individuals of the same clone. Should there be any inclination to think that these are obscure anecdotal examples, consider some of the best-known examples of altruism in the sections below.

## The Hymenoptera/Daphnia Parallel

Let us start by examining the detailed empirical observations of altruism as expressed in the social hymenoptera. In a honey bee colony, for example, there are three castes consisting of a queen who does nothing but reproduce; a few hundred males called drones who also do not do much other than wait for an opportunity to mate with a queen;

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and thousands of non-reproductive females called workers, who toil all their lives taking care of the colony, including foraging and possibly laying down their lives when this is necessary in order to defend the colony. The reproductive queen has been designated as selfish, while the non-reproductive workers are traditionally viewed as the altruists. In fact, Shanahan (2004) regards the behavior of the worker castes of the social insects as the epitome of altruism.

So, are there distinct alleles/genotypes for altruism and selfishness in eusocial populations as we suppose in our models of altruism? It has been known and documented since the 1830s that "a fertilized honeybee egg, which would normally yield a worker bee, will give rise to a queen bee if the ensuing larva is fed 'royal jelly'" (Prete 1990, p. 273). Detailed modern studies have revealed further that whether a bee larva is raised a queen or a worker begins with the type of honeycomb cell into which the egg is laid (Winston 1987). The workers will rear a larva as a queen if it is in a queen cell, by feeding it royal jelly. On the other hand, they will rear it as a worker if it is in a worker cell, by feeding it worker food. The eggs and early larval stages are totipotent (i.e., can develop into different functional entities). According to Winston (1987, p. 66), an egg or larva less than 3 days old that is moved from a worker cell into a queen cell will be fed royal jelly by the nursing workers and it will consequently develop into a queen. Conversely, an egg or larva transferred from a queen cell into a worker cell will be fed worker food and will consequently develop into a worker. This is a very powerful indication that there is no genetic basis for initial placement of an egg in a queen cell or a worker cell, and whether a bee becomes a reproductive (selfish) queen or a non-reproductive worker (altruist) is determined by an environmental stimulus (i.e., diet) rather than genotype.

We now know the specific genes in the honeybee whose differential expression results in the selfish queen and the altruistic workers (Evans and Wheeler 1999); they are plastic genes that are common to both the selfish and altruistic castes. Patel et al. (2007) have detailed the signaling pathways by which different diet regimes activate or depress generic genes to yield different honeybee castes. In fact, inter-caste individuals (i.e., individuals with both queen and worker features) have been artificially created by the experimental manipulation of larval diet (Wilkinson 1984, p. 68). Therefore, the observation that each female honeybee has the potential to develop into a queen or a worker suggests both phenotypes are expressed by the same genotype in response to different environmental stimuli (e.g., diet regimes) rather than separate genotypes coding for the two phenotypes.

In other examples, experimental studies indicate that in the eusocial wasps (Vespidae) differences in nutrition during larval development are often the basis of caste determination (O'Donnell 1998). In other species of social insects, it has been demonstrated that individuals can make a transition between altruistic and selfish behavior through experimental manipulation of their environments (Field et al. 2006). Thus, the genetics of altruism in these cases is very much like that of helmets and non-helmets in *Daphnia*, and therefore an unmistakable case of *phenotypic plasticity*, which is defined as "the environmentally sensitive production of alternative phenotypes by given genotypes" (DeWitt and Scheiner 2004, p. 2). This should be quite obvious in the social insects. West-Eberhard (1986) lists the queen-worker dimorphism in the social insects as one of the examples of alternative phenotypes that are produced by genes borne by all individuals of the population. Wilson (2008, p. 18) has also come to the understanding that "the different roles of the reproductive mother and her non-reproductive offspring are not genetically determined." Rather, "as the evidence from primitively eusocial species has shown, they represent different phenotypes of the same recently modified genome."

A genetic switching mechanism triggers such alternative phenotypes depending on the developmental stage or some environmental stimulus. Unless one rejects this entire catalogue of empirical data, it would be wrong to designate altruism and selfishness as the allelomorphs $A_1$ and $A_2$ in Table 1, as the ASA models do. Also, it is clear that, with this kind of genetics, the altruistic behavior is at no fitness disadvantage relative to the selfish behavior in the social insects. If so, why are evolutionary biologists, as cited above, jumping through hoops to provide circuitous explanations as to how an altruistic gene of lower evolutionary fitness can be sustainable against a selfish alternative, when that is totally not the issue?

It all stems, as I explained above, from the modeling assumption that is inherent in our population genetic models of evolution. Consider the genetic assumptions in the most widely accepted explanations of altruism today, which emanated from the icons of evolutionary biology in the 20th century. Maynard-Smith (1964), like Haldane (1932, p. 208) and Trivers (1971) above, assumed altruism to be caused by a Mendelian recessive character aa as opposed to the characters AA and Aa for the non-altruistic condition. Hamilton (1963, p. 354) supposes "a pair of genes g and G such that G tends to cause some kind of altruistic behavior and g is null. He also points out (1964, p. 16) that in modeling his inclusive fitness hypothesis, he imagined "model organisms, whose (altruistic) behavior is determined strictly by genotype." These seminal works clearly assume a genotypic dichotomy between the altruistic and selfish phenotypes, which in turn precipitates the concern over a declining altruistic allele and consequently the paradox of altruism and a scramble for explanations

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and auxiliary hypotheses. From these scholars emanated inclusive fitness, kin selection, reciprocal altruism, and the ESS models. Of course, we have seen the same concern raised by the proponents of group selection cited above. These models have gotten ever more complex over the years, but the underlying genetic assumption remains.

## Altruistic Expression and Social Cues

In the above section, honeybee society was used to demonstrate how the altruistic and selfish phenotypes in the social insects are determined by different environmental cues rather than genetic differences. Now I turn to the non-eusocial social organisms. Starting with vampire bats, let us examine the relative efficacies of the ASA and ASP models in explaining reciprocal altruism, for which Trivers (1971) provided an explanation based on the ASA assumption. Vampire bats roost in dark places by day and go out at night to feed. For some species the diet is exclusively blood, usually from other mammals. There are occasions when some individuals will find very little to eat while others will be more fortunate. Researchers have observed that the hungry individuals would often solicit some food from the individuals that are better fed. Sometimes an individual would oblige and regurgitate some blood to a soliciting individual, while on other occasions individuals have also been observed to steadfastly refuse to share food with a soliciting individual. It is traditionally held that those individuals observed to obligingly share their food with soliciting individuals are altruistic while those that refuse to share are selfish. Then under the altruism/selfishness allelomorphism (ASA) models we have to assume that the individuals that share blood carry the altruistic allele whilst those that refuse to share are under the influence of the selfish allele.

Closer observations reveal, however, that whether a vampire bat shares blood or not in any situation would be determined largely by the circumstances at the time, such as whether the solicitor has given the actor blood before (Wilkinson 1984), or whether the solicitor is judged likely to give blood to the donor when he is in need. If so, we could suppose that the vampire bat that is seen today sharing blood with a neighbor and judged to be doing so under the expression of an "altruistic allele," could on another occasion be seen steadfastly refusing to give blood to a bat that is starving, possibly because the then solicitor may have refused to share previously. Hence, the bat that is characterized as the altruist today would be the selfish individual on some other occasion. Since organisms are not known to change genotypes in that manner, the difference between sharing then (altruism) and refusing to share now (selfishness) is not a matter of genes but largely the circumstances of the (social) environment. Thus the social environment, like chemical cues in *Daphnia* and diet in the honeybee, serves as a cue for the conditional expression of altruism and selfishness as dimorphic behavioral phenotypes.

Among Belding's ground squirrels, mostly adult females make alarm calls, and the frequency of the calls has been observed to correlate with the presence of relatives (Sherman 1977). Thus, the alarm-calling behavior seems to be conditional, depending upon the presence of relatives. That is exactly what Hamilton (1964) suggested would enhance *inclusive fitness*, and many would celebrate this as a triumph for kin selection. Let me just point out here before I proceed with the current chain of thought, that evidence of kin-motivated altruism here and there does not establish kinship as necessary or sufficient for altruism. The point with this example is that the fact indicating that the same individual can behave altruistically (i.e., call the alarm) at one instance and selfishly (i.e., refuse to call the alarm) at another, undermines the underlying genetic assumption upon which kin selection is established. Rather than two separate genotypes causing the two phenotypes as the architects of kin selection, Hamilton (1964) and Maynard-Smith (1964), suppose, the behavior suggests a dimorphic phenotypic expression of a single genotype. Hence, it is consistent with the ASP model and contrary to the ASA models.

Among olive baboons (*Papio anubis*), Packer (1977) reports that an adult male will give aid to a soliciting troupe member based on whether he has received help from the solicitor before or whether the solicitor is deemed capable of giving meaningful help when it is needed. Thus an adult male is more likely to deny aid to soliciting juveniles and females during fights. It has similarly been reported in vervet monkeys (*Cercopithecus aethiops*) that whether an individual responds to a solicitation or not depends on whether it has previously received grooming (or aid) from the solicitor, in addition to other social considerations (Seyfarth and Cheney 1984). If giving aid is "altruism" and refusing to help is "selfishness," then it is evident here that external factors, rather than genotype, determine whether an individual behaves altruistically or selfishly.

A pattern thus seems to emerge from the key examples of altruism analyzed here: that an individual will respond altruistically only when certain environmental circumstances are present, and would respond selfishly if those environmental cues were lacking. It is no different from the arctic fox expressing white fur in the winter and brown in the summer. It is important to note that no evidence has yet been presented to date that demonstrates that under the same set of environmental circumstances only certain individuals (i.e., those who carry the altruistic allele) are capable of reacting altruistically, while others will always refuse to assist because they lack the altruistic gene. In

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other words, there is no empirical evidence of such an altruistic allele that serves as the underlying distinction between the altruistic and selfish phenotypes. To prove genotypic dichotomy we need to demonstrate, for example, that certain honeybees will always mature into workers (altruists) irrespective of diet or any other external factor; that certain members of a vampire bat colony will always share food even when the solicitor is one who has consistently refused to share; that from one external circumstance to another, only certain individuals will consistently call the alarm while others would never call the alarm under any circumstance. The ASA models presume these tests to be met. In reality there is no basis for such a presumption.

What is clear and consistent from the studies cited here is the association between certain environmental cues and the expression of the altruistic phenotype, while other circumstances trigger a selfish response. For example, a baboon gives aid (altruistic) under one circumstance and denies aid (selfish) under another. That strongly suggests a plastic behavioral response of a single genotype to different (social) environmental circumstances. Thus, as in the *Daphnia* example, there cannot be separate genotypes for altruism and selfishness, since each individual in the population has the capacity to express both phenotypes.

## Reproductive Altruism and the Social Environment

Reduced fecundity in deference to others has often been cited among the examples of altruistic expression (Shanahan 2004; Okasha 2009). Reproductive suppression (or even exclusion) of subordinate females and males is a common feature of animal social organizations. In these cases of altruism it becomes ever more preposterous to imagine that a genotypic difference could be causing the behavioral difference between the individuals who reproduce and those who do not. Observations indicate very strongly that in such societies the "altruistic" behavior is imposed by external circumstances rather than by specific genotypes. In social mammals for example, it is often the dominant female or male that prevents the others from breeding, through a variety of schemes, including physical deterrence from mating. In the naked mole rat (*Heterocephalus glaber*), pheromones given off by the dominant female act on the hormonal systems of subordinate females to render them infertile (Faulks et al. 1991). Those pheromones are analogous to chemical cues from predacious fish in the case of *Daphnia*, and it is they rather than genotype that elicit the non-reproductive altruistic behavior in the mole rat. In meerkat societies the reproductive efforts of subordinate females are deterred and disrupted by the dominant female (Young and Clutton-Brock 2006). In the case of helper birds, individuals are forced to assume the non-reproductive (helper) position by external circumstances such as demography, rank, and availability of nest cites (Rabenold 1985) rather than the dictates of some "altruistic gene" in the helper. Yet these are all frequently cited examples of altruism, whose sustainability we are confounded by as we attempt to explain it by making a "phenotypic gambit" that the two phenotypes are supported by contrasting genotypes.

In most social situations, it is where an individual ranks in the social structure that determines whether it reproduces or not. In hyena and wolf packs for example, only the alpha male and female breed and the rest of the pack we must call altruists. However, upon the death of the alpha female, as observed in the naked mole rat (*Heterocephalus glaber*) by Lacey and Sherman (1991), any of the non-reproductive (altruistic) females can undergo some hormonal changes and ascend to the role of the reproductive (selfish) female. A similar observation has been made with the termite, *Zootermopsis nevadensis*, in which a replacement is drawn from amongst the workers upon the death of the king or queen (Johns et al. 2009). This means a phenotypic transformation of an altruistic worker into a selfish king or queen. Recall the sex change behavior of the marine goby *Coryphopterus personatus* (Allsop and West 2004) from the literature of phenotypic plasticity. In this case also, the same individual can be non-reproductive (altruistic) in one social circumstance and become reproductive (selfish) when the circumstances change. Such transitions between the altruistic and selfish phenotypes by individuals belie the assumption of an underlying genotypic dichotomy between the phenotypes in current models.

Gadagkar (1997, p. 28) notes that a social organism would assume a subordinate role not because of any altruistic reasons but because it is the best of the available alternatives. In the social wasp *Ropalidia marginata*, Gadagkar (1997, p. 72) reports that individual wasps can act as queens or workers in response to the opportunities available. He observed further that often a worker would later drive its mother (the queen) out and become the queen. The change in status or phenotypic behavior from worker to queen has also been reported in other social insects (Field et al. 2006). As Queller (2006, p. 42) observes in the eusocial insects, "workers are not leaping at every opportunity to be altruistic, they are coerced." Coercion as a trigger of altruism, in the absence of which an individual would rather remain selfish, is indicative of the plastic phenotypic deployment of a common genotype. Wenseleers and Ratnieks (2006) also conclude from studies of ten social insect species that "it is mainly social sanctions" that keep individuals altruistic where they would otherwise have behaved selfishly. Emlen and Wrege (1992) report that in the white-fronted bee-eater (*Meropsis*

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*bullockoides*), young males are forced by older nest-owning males into helper status by harassment and disruptions of their attempts to set up their own nests. Such "altruistic" helpers can change their status to "selfish" reproductive nest owners whenever the opportunities arise in the future. In fact, in meerkat societies, as Young and Clutton-Brock (2006) report, not only are our designated altruists (the subordinate females) able to express the selfish phenotype by reproducing when they get the opportunity, they are able to match the selfishness of the dominant female by murdering the infants of other mothers.

In all these examples, it is remarkably consistent that the altruistic and selfish phenotypes are determined by environmental circumstances rather than genotype. One very crucial observation is that individuals are often able to make transitions between the two phenotypes in response to changes in their social environment. These facts are clearly, inconsistent with the notion of two separate genotypes for altruistic and selfish individuals as assumed by extant genetic models of altruism.

One possible objection, which space does not allow me to discuss in detail here, is that some may think conditional altruism extenuates this criticism. In the usual conception of conditional altruism, an individual who is genetically an "altruist" is able to withhold altruistic behavior under certain circumstances. But the models often do not characterize such situations in which an altruist withholds altruism as selfish behavior, i.e., they do not see the altruist to be expressing the same phenotype as the selfish individuals. Also, they certainly do not think the genetically selfish individuals exhibit conditional selfishness. So those models still try to maintain two genotypically distinct individuals in the population, which is contrary to the empirical evidence.

## Concluding Remarks

In the foregoing discussion, I have tried to bring the conceptions and assumptions in our evolutionary modeling of sociality closer to reality. No evolutionary biologist thinks that a single mutation underlies a complex trait such as altruism. Yet, we feel compelled to model it as if that were the case. I have shown a way out of that. Second, we have generally assumed in our evolutionary models that altruism and selfishness are competing evolutionary alternatives under distinct genotypes. This I have also shown to be blatantly contrary to the empirical data. It is another "caricature of reality" we need to wean our models off. We have an epistemic responsibility to keep our theoretical assumptions consistent with well-confirmed empirical evidence. Theoretical expediency should never supersede the empirical evidence.

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