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Kin Selection–Mutation Balance: A Model for the Origin, Maintenance, and Consequences of Social Cheating

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ABSTRACT: Social conflict, in the form of intraspecific selfish “cheating,” has been observed in a number of natural systems. However, a formal, evolutionary genetic theory of social cheating that provides an explanatory, predictive framework for these observations is lacking. Here we derive the kin selection–mutation balance, which provides an evolutionary null hypothesis for the statics and dynamics of cheating. When social interactions have linear fitness effects and Hamilton’s rule is satisfied, selection is never strong enough to eliminate recurrent cheater mutants from a population, but cheater lineages are transient and do not invade. Instead, cheating lineages are eliminated by kin selection but are constantly reintroduced by mutation, maintaining a stable equilibrium frequency of cheaters. The presence of cheaters at equilibrium creates a “cheater load” that selects for mechanisms of cheater control, such as policing. We find that increasing relatedness reduces the cheater load more efficiently than does policing the costs and benefits of cooperation. Our results provide new insight into the effects of genetic systems, mating systems, ecology, and patterns of sex-limited expression on social evolution. We offer an explanation for the widespread cheater/altruist polymorphism found in nature and suggest that the common fear of conflict-induced social collapse is unwarranted.

Keywords: kin selection, cheating, conflict, mutation, load, policing.

Introduction

Intraspecific cooperation and altruism are prevalent in nature, with examples spanning the continuum of biological complexity from unicellular prokaryotes and eukaryotes to plants and animals. Altruism is believed to play an essential role in the evolutionary transitions that brought about the origin of the genome, multicellularity, and societies (Buss 1987; Maynard Smith and Szathmary 1998; Michod 1999), making altruism a fundamental and ubiquitous feature of life. Yet explaining the existence of al-

truism has posed a challenge to evolutionary theory. The problem is that altruists always decline in frequency within groups as they are outcompeted by selfish “cheaters,” who reap the benefits of altruism from others but do not pay the costs (Haldane 1932; Wright 1945). A solution to this problem is formulated by kin selection theory, which shows that selection within groups favoring cheaters can be counteracted by selection among groups favoring altruists (Darwin 1859; Hamilton 1975; Wade 1980, 1985; Queller 1992). The response to among-group selection against cheaters exceeds the opposing response to within-group selection favoring cheaters when Hamilton’s rule, $br - c > 0$, is satisfied (Hamilton 1975; Wade 1980), where b is the benefit to recipients of altruism, r is the relatedness of altruists to recipients, and c is the fitness cost of acting altruistically (Hamilton 1963, 1964). The condition for the invasion and spread of cheaters, $br - c < 0$, is the inverse of Hamilton’s rule (Wade and Breden 1980). Because Hamilton’s additive linear model does not permit internal equilibria (Michod 1982), a polymorphism with coexistence of cheaters and cooperators is not possible.

Contrary to these theoretical predictions, natural populations are commonly polymorphic for cooperative and cheating phenotypes. For example, cheater/cooperator polymorphisms have been observed in nature in the slime mold *Dictyostelium discoideum* (Buss 1982; Strassmann et al. 2000), quorum-sensing systems of pathogenic bacteria (Diggle et al. 2007; Sandoz et al. 2007), nitrogen-fixing bacteria of the genus *Bradyrhizobium* (Sachs et al. 2010), endomycorrhizal fungi (Denison et al. 2003), clinical isolates of the opportunistic pathogen *Pseudomonas aeruginosa* (Lee et al. 2005), cancerous cells in multicellular organisms (Buss 1987), and egg-laying workers and queen-biased development (i.e., “royal cheats”; Hughes and Boomsma 2008) in the eusocial Hymenoptera (Starr 1984; Ratnieks and Visscher 1989; Wenseleers and Ratnieks 2006).

The effort to bridge the gap between theory and data has been hampered by the absence of a rigorous, formal population genetic model of cheating. In classical population genetics, there are two primary mechanisms that

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maintain polymorphism in natural populations (Lewontin 1974): frequency-dependent selection and recurrent mutation. By analogy, there are two approaches to reconciling theory with data in social evolution. In the first, one could alter the underlying model of social interactions substantially by making fitness nonlinear, thus abandoning the standard form of Hamilton's rule (Smith et al. 2010). Like frequency dependence in classical population genetics, nonlinear social interactions can maintain a stable polymorphism of cooperators and cheaters (Doebeli et al. 2004; Smith et al. 2010). Nonlinear frequency-dependent social interactions arise when the social benefit contributed by an altruist depends on the number of altruists in a group. For example, if doubling the number of altruists in a group always exactly doubles the total benefit of altruism, then social interactions are said to be linear. However, if the total benefit is more than doubled (or less than doubled) or if it is doubled only when altruists are at a specific frequency within the group, then social interactions are nonlinear. A disadvantage of this approach is that the number of possible nonlinear fitness functions is vast, making it difficult to extract general predictions. Further, this model does not provide a satisfactory evolutionary null hypothesis (*sensu* Lynch 2007), because selection is required to actively maintain the cheater/altruist polymorphism.

Here we suggest a simple but overlooked alternative explanation for the statics and dynamics of social conflict in nature. Specifically, we propose taking the canonical model of social evolution introduced by Hamilton (1964) and adding a previously neglected but fundamental evolutionary force: mutation. In this case, polymorphism is maintained by recurrent mutation despite linear, purifying kin selection. Mutation is a necessary, ubiquitous, and inexorable evolutionary force that has been widely neglected in social-evolution theory (although see Michod 1996, 1997, 1999; Michod and Roze 2000, 2001). Mutation is the ultimate source of genetic variation and serves as a central parameter in the modern study of genetic variation at the molecular level, where sequencing technology is providing an ever-increasing supply of data. Thus, a mutational theory of social evolution provides both a systematic theory of cheating and allows theoretical extension to address patterns of gene sequence variation (Cruickshank and Wade 2008; Linskayer and Wade 2009; Van Dyken and Wade 2010), bringing the population genetics of social evolution into the postgenome era.

The use of Hamilton's additive linear model with recurrent mutation as an evolutionary null model for the maintenance of polymorphism in social traits parallels the use of purifying selection with recurrent mutation as an evolutionary null model for nonsocial traits (Kimura 1983; Lynch 2007). By "evolutionary null," we mean a hypothesis

whereby adaptive evolution is not responsible for actively maintaining cheaters or creating the patterns of social conflict observed in nature. This is not necessarily a claim about nature but rather an empirical tool for distinguishing among alternative hypotheses, whereby the most parsimonious (i.e., nonadaptive) explanation is taken as the null and can be rejected by data.

We show that the steady influx of cheater mutants into populations is balanced at equilibrium by their removal by kin selection in a process that we call the "kin selection–mutation balance." We analyze this process for different genetic systems, mating systems, patterns of sex-limited expression, and scales of density regulation, and we investigate its long-term evolutionary consequences.

Results

The Model

Consider a population with discrete, nonoverlapping generations where two alleles, *A* and *a*, segregate at a locus that controls a cooperative trait. The population is subdivided into cooperative groups, wherein interactions occur at random with respect to genotype (i.e., indiscriminate, whole-group sociality). These groups are equivalent to "trait groups" (Wilson 1975). We consider haploid, diploid, and haplodiploid populations and assume that there is random mating (nonrandom mating and dominance will be considered elsewhere [J. D. Van Dyken and M. J. Wade, unpublished manuscript]). In diploids and haplodiploids, homozygous *AA* individuals are "altruists," *aa* homozygotes are socially defective "cheaters," and *Aa* heterozygotes express the altruist and cheater phenotypes with probability 1/2 each (i.e., the alleles act additively). Cheaters are socially defective individuals that gain the benefits of altruism received from altruists within their group but do not suffer a cost to individual fitness because they do not express the social trait. In haploids, *A* individuals are altruists and *a* individuals are cheaters.

Altruists suffer a relative decrease in fitness by an amount *c*, but they increase the fitness of each individual *k* with which they interact by an amount *b_k*. When altruists form a sterile caste, as in the eusocial Hymenoptera, the direct fitness cost *c* can be interpreted as the probability that an individual possessing an altruist genotype becomes a sterile worker (which then has a direct fitness of 0). In this case, a colony fixed for an altruism allele will be composed of some proportion *c* of sterile workers. The total gross benefit of altruism received by an individual in a group is $Nb_k = b$, where *N* is the number of individuals in a group, unless expression of altruism is sex limited, in which case the total benefit is proportional to the number of the altruistic sex in a group. (Here, we are assuming

that benefit accrues to the performer as well as to other group members. If it is assumed that it does not, then the equations below are only modestly altered; see Van Dyken 2010.) Costs and benefits are measured with respect to a reference allele, in this case the *a* allele, such that the fitness costs and benefits of the *AA* homozygotes are equal to the difference between its absolute costs and benefits and those of *aa* homozygotes ($c = c_{AA} - c_{aa}$; $b_k = b_{k,AA} - b_{k,aa}$). This allows us to investigate mutant alleles expressing an arbitrary degree of social defect rather than simply pure altruists and pure cheaters. For example, we can investigate cheaters that provide less benefit to the group ($b_k > 0$) or cheaters that incur lower costs ($c > 0$). Both cases are particularly important in tests of the theory using DNA sequence data, where mutational effect sizes may vary among nucleotides within a sequence.

Allele Frequency Recursion

We use the hierarchical Price equation (Price 1972; Hamilton 1975; Wade 1985; Rice 2004) and Hamilton's linear fitness model (Hamilton 1964; Wilson 1975; Wade 1978, 1979; Wade and McCauley 1980) to derive an expression for the change in frequency of a cheater allele under kin selection (Van Dyken 2010). In the simplest case of equal ploidy and expression in both sexes (which we relax below), it can be shown that

$$\Delta q = -\frac{bV_b - cV_t}{\bar{W}} \quad (1)$$

(app. A), where q is the global frequency of the *a* "cheater" allele, V_b is the variance of mean frequency of the *a* allele among groups, V_t is the variance of allele *a* among individuals over the global population, and \bar{W} is the mean fitness of the metapopulation.

Note that cheaters are selected against ($\Delta q < 0$) when $bV_b > cV_t$, which is a restatement of Hamilton's rule with r equal to V_b/V_t , the fraction of the total genetic variation that is among groups (Wade 1980). When Hamilton's rule is satisfied, cheaters are equivalent to deleterious mutants, even though they are favored within groups. If $bV_b < cV_t$, then cheating is net advantageous (after summing selection within and among groups) and will spread throughout the population.

Equation (1) applies equally to haploids and diploids under the current assumptions, although the value of the variance components depends on the genetic system (Wright 1951, 1965). For diploids,

$$V_b = pqf_{st}, \quad (2a)$$

$$V_t = \frac{pq(1 + f_{it})}{2}, \quad (2b)$$

where $p = (1 - q)$ is the frequency of the altruism allele *A*; f_{it} is the total inbreeding coefficient, which accounts for both population subdivision (f_{st}) and nonrandom mating within groups (f_{is}). These components are related by the equation $1 - f_{it} = (1 - f_{st})(1 - f_{is})$ (Wright 1951). While we assume throughout that there is random mating ($f_{is} = 0$, so that $f_{it} = f_{st}$), this general form is preferable because it allows us to link the variance components of haploids and diploids. This is done by noting that $f_{is} = f_{it} = 1$ in haploids (Hamilton 1972). Substituting into equations (2a) and (2b), we obtain the haploid variances,

$$V_b = pqf_{st}, \quad (2c)$$

$$V_t = pq. \quad (2d)$$

For all genetic systems, the average relatedness within groups (i.e., the regression coefficient of relatedness) is expressed as

$$r = \frac{V_b}{V_t} \quad (3)$$

(Crow and Kimura 1970; Hamilton 1975; Michod and Hamilton 1980). In haplodiploids (see below and app. B), the diploid sex obeys equations (2a) and (2b) while the haploid sex obeys equations (2c) and (2d).

Importantly, identity coefficients (f_{st} and r) are not influenced by allele frequency (Wright 1951, 1965; Rousset 2004). This allows us to solve for the equilibrium allele frequency across many different metapopulation genetic structures. In addition, identity coefficients will typically be independent of mutation rate, as long as the mutation rate is small relative to the rate of migration (Wright 1951, 1965; Rousset 2004). When mutation is sufficiently high, it can contribute to an increase in variance within groups, which drives a decline in values of f_{st} and r . Here we limit our explicit attention to the common case in which dispersal is much higher than mutation, although our model is general enough to apply to situations of low dispersal and high mutation.

With recurrent mutation, the *A* allele is mutated to *a* at a rate μ , and *a* is mutated to *A* at a rate ν . It is reasonable to assume that spontaneous mutations are more likely to knock out or reduce cooperative allele function than to improve it, such that the likelihood of creating an altruistic allele by mutation, ν , is negligible. This is justified by natural and laboratory populations, where cheaters typically arise when cooperation alleles sustain loss-of-function or diminution-of-function mutations (Foster et al. 2007). Indeed, deleting or otherwise impairing "cooperation" genes is a common method for creating cheater strains for use in social-evolution experiments (reviewed in Foster et al. 2007). Adding mutation to equation (1) gives us the expression for change in frequency of a cheater allele under

selection and mutation for the additive, linear model with equal ploidy and expression in both sexes (these latter two assumptions will be relaxed for haplodiploidy below):

$$\Delta q = -\frac{bV_b - cV_c}{\bar{W}} + p\mu. \quad (4)$$

Kin Selection–Mutation Balance

Haploid Populations. From equations (2c), (2d), and (3), we find that for haploids, $r = f_{st}$. Substituting into equation (4) and rearranging, we find the change in frequency of the cheater allele under kin selection and mutation to be

$$\Delta q = -\frac{pq(br - c)}{\bar{W}} + p\mu. \quad (5)$$

Setting equation (5) equal to 0 and assuming weak selection, such that $\bar{W} \sim 1$, we can solve for the equilibrium frequency of cheaters at the kin selection–mutation balance for haploids as

$$\hat{q} \cong \frac{\mu}{br - c}. \quad (6)$$

Diploid Populations. Substituting equations (2a) and (2b) into equation (4), we find that

$$\Delta q = -\frac{pq[2bf_{st} - c(1 + f_{st})]}{2\bar{W}} + p\mu. \quad (7)$$

From equations (2c), (2d), and (3), the relatedness in diploid populations with no inbreeding ($f_{is} = 0$) is $r = 2f_{st}/(1 + f_{st})$. Substituting r into equation (7) and rearranging, we find that

$$\Delta q = -\frac{pq(1 + f_{st})(br - c)}{2\bar{W}} + p\mu. \quad (8)$$

Assuming that $\bar{W} \sim 1$, setting $\Delta q = 0$ in equation (8), and solving for q , we find the equilibrium frequency of cheater alleles to be

$$\hat{q} \cong \frac{2\mu}{(1 + f_{st})(br - c)}. \quad (9)$$

This is the kin selection–mutation balance for diploids. The factor 2 in the numerator reflects the fact that the cheating mutation has been assumed to act additively ($h = 1/2$). When there is no population structure ($f_{st} = r = 0$), equation (9) retrieves the mutation–selection balance for an additively acting locus on a direct-effect mutation (Muller 1950), and when $b = 0$, it retrieves the mutation–selection balance for direct-effect loci in struc-

ured populations under hard selection (Whitlock 2002). Note, too, that for $f_{st} < 1$, the equilibrium frequency of cheaters is always greater for diploids than for haploids because of the factor $2/(1 + f_{st})$ in equation (9).

Haplodiploid Populations. In haplodiploid species, social traits are commonly expressed by only a single sex, typically females (reviewed in Linksvayer and Wade 2005). In the eusocial Hymenoptera (including ants and some bees and wasps), there is a division of labor between fully fertile (i.e., “queen”) and nonreproductive helper (i.e., “worker”) females, while all males are reproductive (Anderson et al. 2008; Schwander et al. 2010). We focus especially on this case of female-limited expression of sib care because the eusocial Hymenoptera are well-established model systems for studying social evolution. In this case, the cost of helping is experienced only by worker females, and the benefit accrues to fully fertile sibling males and females. Mutations occur in both sexes but are expressed only in females.

With haplodiploidy, allele frequency change is measured separately in males and females and then averaged, weighting males by a factor of 1/3 and females by 2/3, which is the “reproductive value” of each sex, defined as the asymptotic proportion of genes in future generations contributed by each sex (Crow and Kimura 1970; Taylor 1988). In appendix B, we show that the allele frequency recursion under the present assumptions is given by

$$\Delta q = -\frac{pq(1 + f_{st,f})[bs_f r - (2/3)c]}{2\bar{W}} + p\mu, \quad (10)$$

where $f_{st,f}$ is the value of f_{st} taken among females in the metapopulation and $r = (1/3)r_{fm} + (2/3)r_{ff}$, where $r_{fm} = \text{Cov}_b(p_f, p_m)/V_c(p_f)$ is the regression coefficient of relatedness of females to males and $r_{ff} = V_b(p_f)/V_c(p_f)$ is the regression coefficient of relatedness among females. The value s_f is the total proportion of females in a group ($s_f = N_f/N$). This includes both fully fertile and sterile females (i.e., both queens and workers) and is not to be confused with the operational sex ratio, which generates Fisherian sex ratio selection (Fisher 1958), opposing a deviation from the optimal sex ratio. For simplicity, our model does not correct for deviations from an equal reproductive sex ratio, which can be done with methods discussed by Taylor (1988) and Grafen (1986). We assume that the sex ratio is at its equilibrium and is determined by factors extrinsic to our model. Equation (10) shows that the inclusive fitness effect is greater for higher s_f (a female-biased sex ratio), which simply means that the colony has higher viability when there are more female altruists contributing to group benefit.

From equation (10), we find the kin selection–mutation balance to be

$$\hat{q} \cong \frac{2\mu}{(1 + f_{st,r})[bs_r r - (2/3)c]}. \quad (11)$$

Note the similarity to equation (9), with $f_{st,r}$ here instead of f_{st} and the factors s_r modifying the total benefit and $2/3$ modifying the cost; all three differences reflect the effect of limiting the expression of social behavior to females, which weakens selection (Wade 1998) and inflates the frequency of cheaters relative to expression in both sexes.

Comparison of Genetic Systems at Kin Selection–Mutation Balance

Ever since Hamilton (1964) suggested that haplodiploidy promotes the evolution of altruism, there has been intense interest in determining how genetic system affects social evolution (e.g., Reeve 1993; Reeve and Shellman-Reeve 1997; Wade 2001; Linksvayer and Wade 2005). In order to compare the equilibrium frequency of cheaters among the three genetic systems modeled above, we first compare the case where social groups are composed of siblings in monogamous, single-mother (queen) families (fig. 1A). In this case, relatedness differs between genetic systems. In haplodiploids, following Grafen (1986) and Bourke and Franks (1995), the two regression coefficients of relatedness for full sibs are $r_{fm} = 1/2$ and $r_{ff} = 3/4$, so that $r = 2/3$ for haplodiploids under the current assumptions. Relatedness is more straightforward in haploids and diploids, with $r = 1$ (we assume asexual haploids) and $r = 1/2$, respectively. Also, with these assumptions and applying equations (2a), (2b), and (3), we find that $f_{st} = 1/3$ in diploids, and in haplodiploids among females we find that $f_{st,r} = 3/5$. In order to compare diploidy and haplodiploidy on an equal footing (i.e., to not confound genetic system with sex-limited expression), we also consider diploidy with female-limited expression of altruism. Analytical results for this case are given in appendix B and are plotted in figure 1.

Figure 1A shows that the expected frequency of cheaters is lowest for haploids and highest for diploids with female-limited expression of altruism. Interestingly, haplodiploidy with female-limited expression, as found in the eusocial Hymenoptera, leads to a greater frequency of cheaters than diploidy with expression in both sexes. This is a consequence of relaxed selection on cheating mutations due to female-limited social expression (Wade 1998; Linksvayer and Wade 2009; Van Dyken and Wade 2010). This has important implications, as most cases of extreme diploid sociality (e.g., naked mole rats and termites) have both male and female expressions of altruism (Reeve and Shellman-Reeve 1997), while in haplodiploids altruism is typically female limited (Linksvayer and Wade 2005). This

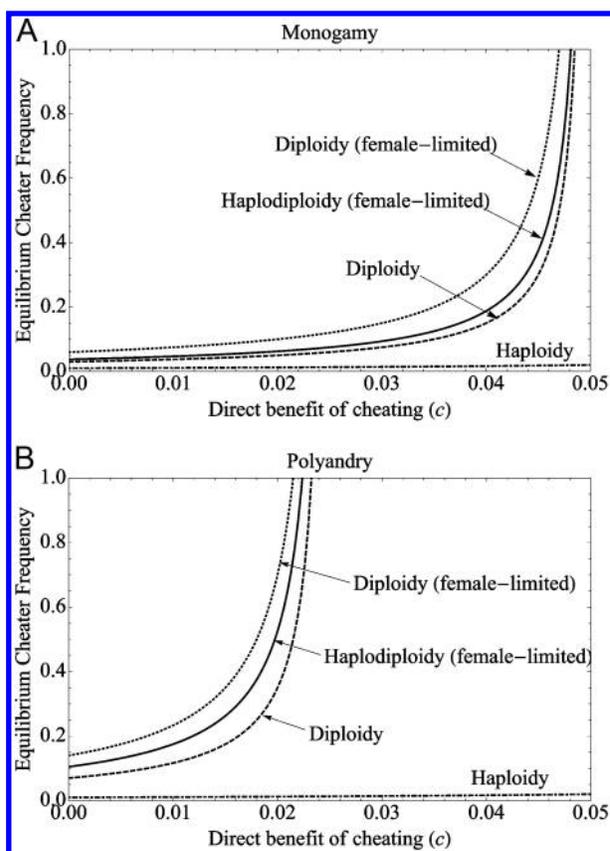


Figure 1: Kin selection–mutation balance as a function of cheater benefit (altruist cost) c for different genetic systems. Parameter values: $b = 0.1$ and $\mu = 10^{-3}$. In A, social groups are families with single-mated foundresses, while in B, foundresses are multiply mated. While not shown here, haploidy has a vertical asymptote at $c = 0.1$ (i.e., where $c = b$). Vertical asymptotes indicate where the inclusive fitness effect is equal to 0.

clearly demonstrates how an advantage of haplodiploidy for the evolution of altruism is largely negated by sex-limited gene expression.

Polyandry (i.e., multiple mating by foundresses) greatly increases the frequency of cheaters at equilibrium (fig. 1B). When hymenopteran queens mate multiply, relatedness of females within a colony decreases rapidly with the number of sires, from $r_{ff} = 3/4$ to $r_{ff} = 1/4$, while relatedness of females to males remains unchanged at a value of $r_{fm} = 1/2$. The total relatedness in our model for haplodiploids under multiple mating, then, is $r = 1/3$. The relatedness in a diploid family with a single, multiply mated foundress is $r = 1/4$. Likewise, f_{st} values are altered accordingly for each of these cases.

The kin selection–mutation balance draws an analogy between cheating and deleterious mutation at nonsocial loci. Without mutation, the condition for an increase in

frequency of the altruism allele in haploids and diploids is $br - c > 0$, the familiar Hamilton's rule (Hamilton 1964). In haplodiploids with female-limited expression of altruism, this condition becomes $bs_r r - (2/3)c > 0$. If these conditions hold, then there will be no cheaters in a population when mutation is absent. If $br - c < 0$, then the right-hand side of equations (5), (8), and (10) will be positive and cheating will be favored by natural selection ($\Delta q > 0$). By analogy with the classic recursion of allele frequency for alleles with direct effects on viability (Haldane 1932), we see that $br - c$ or $bs_r r - (2/3)c$, also called the "inclusive fitness effect" of the altruism allele (Hamilton 1964; Michod 1982), is equivalent to the net selection coefficient (summed across both levels of selection) favoring the altruism allele or, equivalently, to that opposing the cheating allele. Thus, if Hamilton's rule is satisfied, then cheaters are simply deleterious mutants opposed by net selection (see also Wade and Breden 1980).

Hamilton's Rule with Mutation

Equations (6), (9), and (11) demonstrate that despite satisfying Hamilton's rule (i.e., $br - c > 0$), cooperative alleles may nevertheless be rare in the population (see also fig. 1). As the inclusive fitness effect ($br - c$ or $bs_r r - (2/3)c$) approaches 0, the cheater allele approaches fixation in the population because of recurrent mutation pressure. Hamilton's rule, then, does not preclude the existence of cheaters in an otherwise cooperative population, nor does it predict the frequency of cooperators or cheaters in a population. When cheaters arise at a constant rate via recurrent mutation, the inclusive fitness effect must be sufficiently greater than 0 for cooperation to be common at equilibrium.

We suggest a corollary to Hamilton's rule that accounts for mutation. Cooperation will be "common" in a population when $\hat{q} < 1 - \varepsilon$, where ε is an arbitrary cutoff frequency of cooperators such that the frequency of cooperators exceeds this cutoff when

$$br - c > \frac{\mu}{1 - \varepsilon}, \quad (12a)$$

$$br - c > \frac{2\mu}{(1 + f_{st})(1 - \varepsilon)}, \quad (12b)$$

$$bs_r r - \frac{2}{3}c > \frac{2\mu}{(1 + f_{st,r})(1 - \varepsilon)}, \quad (12c)$$

for haploids, diploids, and haplodiploids, respectively.

These conditions predict the evolutionary stability of cooperation in the face of recurrent cheating mutations. Note that these conditions are always more stringent than Hamilton's rule. If explaining the prevalence or stability

of cooperation/altruism within a species is desirable, equations (12) provide the appropriate reformulation of Hamilton's rule.

Note that equations (12) cannot be satisfied if $\varepsilon = 1$. This demonstrates that at equilibrium, kin selection is never strong enough to eliminate all selfish cheaters from a population. Yet the introduction of cheaters into a population by mutation implies neither the destabilization of cooperation nor the spread of cheaters throughout the global population, as is frequently assumed (see Haldane 1932; Maynard Smith 1964; Hamilton 1971; West et al. 2006a, 2006b).

The Cheater Load

The cheater load is the reduction in mean individual (or equivalently, mean group) fitness due to the presence of cheating mutants (Travisano and Velicer 2004). Even though cheaters may not spread to fixation, their ineluctable presence via recurrent mutation generates important long-term evolutionary consequences.

The mean fitness in a population in the current model at equilibrium is $\bar{W} = 1 + (b - c)(1 - \hat{q})$ for haploids and diploids and $\bar{W} = 1 + [bs_r r - (2/3)c](1 - \hat{q})$ for haplodiploids. As long as $b > c$, which is required for the cooperative trait to persist, the mean fitness is maximal when the cooperative allele is fixed ($q = 0$), demonstrating that the presence of cheaters causes a cheater load that reduces mean fitness by the amount $(b - c)\hat{q}$ (or by $[bs_r r - (2/3)c]\hat{q}$ for haplodiploids).

The cheater load is a function of both the reduction in mean cost and the loss of benefits, because cheaters neither bear the costs nor contribute benefits. The mean fitness in the population is not explicitly dependent on population structure (f_{st} and r do not appear in the expression for \bar{W}), although it is implicitly dependent through the effect of population structure on equilibrium cheater frequency, \hat{q} (eqq. [6], [9], [11]).

The cheater load can be quantified, by analogy to other types of genetic load (Crow 1970; Charlesworth and Charlesworth 1998), as the difference between the maximum attainable mean fitness and the observed mean fitness, $L = \bar{W}_{\max} - \bar{W}$. Substituting \bar{W} and equations (6), (9), and (11) into L , we find

$$L_{\text{haploid}} = \frac{\mu(b - c)}{br - c}, \quad (13a)$$

$$L_{\text{diploid}} = \frac{2\mu(b - c)}{(1 + f_{st})(br - c)}, \quad (13b)$$

$$L_{\text{haplodiploid}} = \frac{2\mu[bs_r r - (2/3)c]}{(1 + f_{st,r})[bs_r r - (2/3)c]}. \quad (13c)$$

When $b = 0$, equations (13) retrieve the genetic load for direct-effect genes in structured populations under hard selection (Whitlock 2002). When $b = f_{st} = r = 0$, these equations reduce to the classic result for genetic load on direct-effect genes in unstructured populations (Muller 1950; Werren 1993), as expected (although our haplodiploid model accounts for female-limited expression, while the previous results did not).

The ratio $(b - c)/(br - c)$ in equations (13) is the ratio of the group-selection gradient to the total-selection gradient. Whenever $r < 1$, this ratio is greater than 1, increasing the load. This can be viewed as a consequence of the additional mean fitness-reducing effect of selection within groups that generates a conflict between levels of selection (Michod 1999). The apparent dependence of the load on absolute fitness can be removed by defining the parameter $\lambda = c/b$, which then leads to

$$L_{\text{haploid}} = \frac{\mu(1 - \lambda)}{r - \lambda}, \quad (14a)$$

$$L_{\text{diploid}} = \frac{2\mu(1 - \lambda)}{(1 + f_{st})(r - \lambda)}, \quad (14b)$$

$$L_{\text{haplodiploid}} = \frac{2\mu[s_f - (2/3)\lambda]}{(1 + f_{st,r})[s_f r - (2/3)\lambda]}, \quad (14c)$$

under the constraint that $r > \lambda$, which is the condition for Hamilton's rule to hold (Hamilton 1963, 1964).

Like the mutation load on direct-effect genes (Muller 1950), the cheater load does not depend on the value of the fitness effect of mutations. However, unlike the classic result, the mutation load on social loci depends on the relative magnitudes of direct and indirect effects, through λ , and on the population structure, through f_{st} and r (fig. 2). When $f_{st} = r = 1$, there is no levels-of-selection conflict to further reduce mean fitness. Mutations that arise are localized within clonal groups of mutants, so that selection is equivalent to selection among "individuals," which is why equations (14) retrieve the classic results for individual-level traits when $r = 1$. Whenever $r < 1$, the mutation load is always greater for social loci than for nonsocial loci because of the conflict between the levels of selection.

The Targets of Load Modification

Because the cheater load reduces fitness, we might expect modifiers of the cheater load to arise and be favored by selection. Mutual policing is one such mechanism of cheater control (Ratnieks 1988; Frank 1995; Travisano and Velicer 2004; Brandvain and Wade 2007). The canonical example of policing is the enforcement of worker sterility in the eusocial Hymenoptera (Ratnieks 1988), where unfertilized "sterile" workers can cheat by laying eggs that

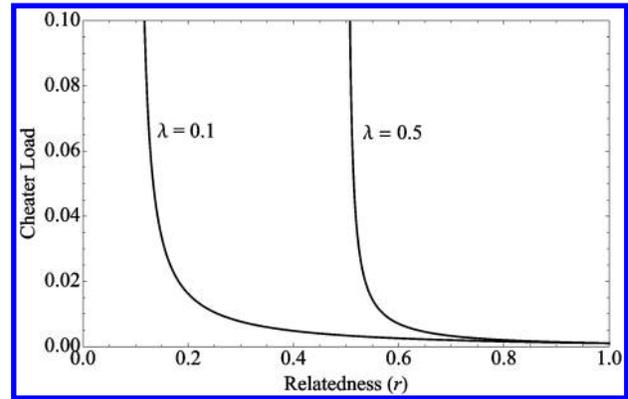


Figure 2: Cheater load as a function of relatedness for diploids, with parameter values $\lambda = 0.1$ or 0.5 , where $\lambda = c/b$ is the cost/benefit ratio, and $\mu = 10^{-3}$. The curves for other genetic systems are omitted for clarity, but the relationship between genetic systems in terms of relative magnitude of the cheater load follows that for the equilibrium cheater frequency in figure 1. Note that the cheater load decreases as relatedness increases.

develop into sons (arrhenotoky) or daughters (thelytoky). This is thought to reduce the colony-beneficial productivity of the cheater, as such cheaters have been found to spend less time foraging or in colony defense, both of which cause a reduction in colony fitness (West-Eberhard 1975; Cole 1986; Hartmann et al. 2003; Dampney et al. 2004; Dijkstra and Boomsma 2006).

In general, for a given mutation rate, increasing relatedness (r) and reducing the ratio of costs and benefits (λ) are the only ways to reduce the cheater load. The effect on the cheater load of a small change in r and λ is found by taking the partial derivative of load with respect to r and λ , respectively. For haploids, we have

$$\frac{\partial L}{\partial r} = -\frac{\mu(1 - \lambda)}{(1 - r)^2}, \quad (15a)$$

$$\frac{\partial L}{\partial \lambda} = \frac{\mu}{1 - r}. \quad (15b)$$

Equations (15) show that the load is reduced by increasing r and/or by lowering λ and that increasing relatedness has a much larger effect on load reduction than does modification of costs and benefits, which is true for all values of r and λ that satisfy Hamilton's rule.

Modifiers of relatedness might act to affect the number of individuals founding a new group, the number of males inseminating a female that founds a colony, the migration rate among groups, or kin recognition. Alternatively, microbes may secrete anticompetitor toxins that prevent the invasion of a clonal group by outsiders ("xenophobia"; see Travisano and Velicer 2004). Modifiers of λ include po-

licing behavior, which diminishes the detrimental effect that cheaters have on group fitness (Ratnieks 1988; Frank 1995; Travisano and Velicer 2004). According to equations (15), policing should evolve only when there are constraints (genetic, developmental, ecological, or phylogenetic) that prevent modification of relatedness.

Kin Competition

Kin competition (i.e., soft selection) occurs when local density regulation diminishes the expression of differential group productivity, resulting in a diminished response to group selection (Van Dyken 2010; see app. A). Kin competition has been shown to be a potentially severe constraint on the evolution of altruism (Grafen 1984; Wade 1985; Kelly 1992, 1994; Taylor 1992; Wilson et al. 1992; Frank 1998). We would like to know how kin competition affects the kin selection–mutation balance and the cheater load. Following Van Dyken (2010; see app. A), we find that the equilibrium frequency of cheaters under an arbitrary intensity of kin competition α is

$$\hat{q}_{\text{haploid}} = \frac{\mu}{br - c - \alpha r(b - c)}, \quad (16a)$$

$$\hat{q}_{\text{diploid}} = \frac{2\mu}{(1 + f_{st})[br - c - \alpha r(b - c)]}, \quad (16b)$$

$$\hat{q}_{\text{haplodiploid}} = \frac{2\mu}{(1 + f_{st,f})\{bs_f r - (2/3)c - \alpha r[bs_f - (2/3)c]\}}. \quad (16c)$$

When $\alpha = 0$, there is no kin competition (the efficacy of kin selection is not diminished by local density regulation; i.e., there is “hard selection”), and when $\alpha = 1$, there is strict kin competition (“soft selection”; Wallace 1968; Wade 1985; Whitlock 2002; Van Dyken 2010). Figure 3 shows that increasing the degree of kin competition inflates the equilibrium frequency of cheaters in a population. Importantly, for all parameter values, there exists some degree of kin competition above which cheaters will fix in the population.

While greater kin competition monotonically increases cheater frequency, its affect on the cheater load is more complicated, revealing some interesting properties of kin competition. The mean fitness with an arbitrary degree of kin competition α is $\bar{W} = 1 + (1 - \alpha)(b - c)(1 - \hat{q})$ for haploids and diploids and $\bar{W} = 1 + (1 - \alpha)[bs_f - (2/3)c](1 - \hat{q})$ for haplodiploids with female-limited altruism expression. The maximum fitness under kin competition occurs when $\hat{q} = 0$, giving $\bar{W}_{\text{max}} = 1 + (1 - \alpha)(b - c)$ for haploids and diploids and $\bar{W}_{\text{max}} = 1 + (1 - \alpha)[bs_f - (2/3)c]$ for haplodiploids with female-limited

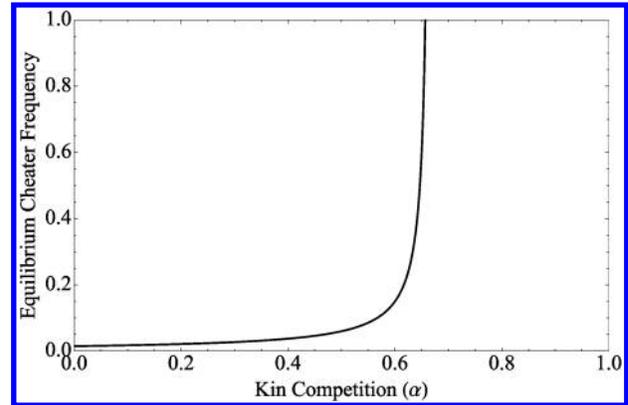


Figure 3: Effect of kin competition (i.e., soft selection) on the equilibrium frequency of cheaters at kin selection mutation balance. Parameter values are $b = 0.4$, $c = 0.1$, $r = 0.5$, and $\mu = 10^{-3}$. This curve represents the effect for diploids, which is qualitatively the same for all other genetic systems.

altruism. Applying \hat{q} and α (eqq. [16]) to the mean fitness and solving for $L = \bar{W}_{\text{max}} - \bar{W}$, we find

$$L_{\text{haploid}} = \frac{\mu(1 - \alpha)(b - c)}{br - c - \alpha r(b - c)}, \quad (17a)$$

$$L_{\text{diploid}} = \frac{2\mu(1 - \alpha)(b - c)}{(1 + f_{st})[br - c - \alpha r(b - c)]}, \quad (17b)$$

$$L_{\text{haplodiploid}} = \frac{2\mu(1 - \alpha)[bv_f - (2/3)c]}{(1 + f_{st,f})\{bs_f r - (2/3)c - \alpha r[bs_f - (2/3)c]\}}. \quad (17c)$$

It is seen immediately from equations (17) that with strict kin competition ($\alpha = 1$), the cheater load will be 0. The load is experienced as an increased death rate relative to birth rate in a population, but by the definition of strict kin competition, all local groups are maintained at a constant carrying capacity, so that cheaters (or any other deleterious mutations) do not cause a net increase in selective deaths (Wallace 1968; Whitlock 2002; Van Dyken 2010). Although cheaters are more abundant, their effect on mean fitness is diminished. Differently put, with strict kin competition, selection is blind to the social effects of traits (Van Dyken 2010), so that the loss of group benefits owing to cheaters has no (negative) effect on mean fitness.

Figure 4 shows a unimodal relationship between cheater load and degree of soft selection. The cheater load increases with increasing soft selection until the cheating allele fixes in the population, at which point the load decreases as $(1 - \alpha)(b - c)$. Figure 4 compares the cheater load under kin competition with the cheater load under strict hard selection with an equivalent frequency of cheaters (fig. 4,

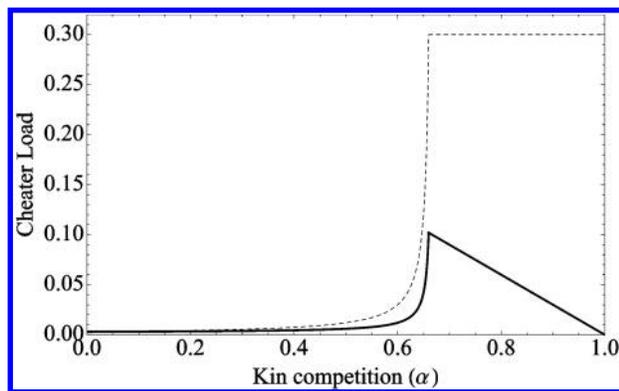


Figure 4: Cheater load under kin competition (i.e., soft selection) is shown by the solid line. The maximum occurs at the value of α where cheaters fix because of soft selection. The dashed line represents the predicted cheater load if the population represented by the solid line were experiencing hard selection. While the cheater load increases initially with soft selection, it does not increase as much as would be predicted by its effect on cheater frequency (*dashed line*). With strict kin competition, there is no load. Parameter values are $b = 0.4$, $c = 0.1$, $r = 0.5$, and $\mu = 10^{-3}$.

dashed line). While kin competition increases the cheater load by increasing the frequency of cheaters, the resulting load is not as great as it would be with the same cheater frequency under hard selection.

Discussion

The current perception of cheaters as a perpetual threat to complex social systems has a long history in evolutionary studies. In the earliest genetic model of the evolution of altruism, Haldane (1932, p. 207) showed how alleles for “socially valuable but individually disadvantageous characters” (i.e., altruistic traits) could increase in frequency to fixation when a population was genetically structured into groups of relatives. He argued, however, that selfish cheaters or “reverse mutations” would occur and spread, concluding that, because a system of altruism was invulnerable to selfish alleles, “it [is] difficult to suppose that many genes for absolute altruism are common in man” (p. 210). Similarly, Maynard Smith (1964, p. 1146) argued that all social systems are invulnerable to selfish or cheater alleles and that “any plausible model of group selection must explain why they do not spread.” More recently, West et al. (2006a, p. R482) summarize the common narrative that because altruistic populations are vulnerable to invasion by cheaters, “we would not expect altruistic behaviours to be maintained in a population—put formally, altruism should not be evolutionarily stable.” Consideration of mutation has generated further confusion. Using inclusive fitness reasoning, West et al. (2006b, p. 606) argue that “mutation can produce

‘unrelated’ cheater lineages, which could spread through a population.”

We have demonstrated that these fears of social collapse in the face of recurrent cheater mutations are typically unwarranted (but for counterexamples, see Michod 1996, 1997, 1999; Michod and Roze 2000, 2001). Specifically, cooperation is evolutionarily stable, despite recurrent cheating mutations, when the condition of a modified form of Hamilton’s rule is satisfied (eqq. [12]). The perceived threat posed by cheaters arises only when selection is viewed as a process occurring at a single level of organization (specifically, within groups). By explicitly modeling selection of recurrent cheating mutations within and among groups, we have shown that the within-group advantage of cheating, which is the basis of most arguments identifying cheating as a problem (e.g., Haldane 1932; Maynard Smith 1964; West et al. 2006a, 2006b), can be overcome by selection among groups opposing cheating. The strength of among-group selection is weakened by factors that reduce relatedness (e.g., high dispersal rate, multiple foundresses, polyandry) and/or increase kin competition. Proper conceptualization of the levels of selection influencing cheating evolution prevents the view that cheaters are a general problem to the stability of cooperative groups and generates predictions for when cheating will and will not be a legitimate threat to social cohesion.

While cheaters do not necessarily undermine the evolutionary persistence of cooperation, the maintenance of cheating mutants at equilibrium nonetheless has long-term evolutionary consequences. By generating a cheater load (i.e., a reduction in mean fitness due to the presence of cheaters), recurrent cheating mutation creates selection for load modifiers, such as worker policing or modification of relatedness through monogamy or limited dispersal. Theories of policing have been developed without explicitly considering the mechanism by which cheaters originate or are maintained in the population (Frank 1995; Brandvain and Wade 2007; Wenseleers et al. 2004). Without this information, it is impossible to quantify the strength of selection favoring policing, which is central to understanding when policing will or will not evolve. The kin selection–mutation balance provides a quantitative prediction for the strength of selection favoring policing that may be useful in explaining or predicting patterns of policing in nature. It also provides a mechanism whereby cheaters are maintained in the population over a long enough evolutionary time, without spreading to fixation, to allow selection for policing.

How many cheaters should we expect to find in nature? For the simplest single-locus case, as modeled here, we can take the rate of deleterious mutation to be on the order of 10^{-6} to 10^{-7} per gene per generation (Kibota and Lynch 1996; Haag-Liautard et al. 2007). There are few

estimates of the value of the inclusive fitness effect, but an average estimate of 0.01 has been calculated for developmental cheaters in the social bacterium *Myxococcus xanthus* (smith et al. 2010). This fits with the magnitude of beneficial effects found for nonsocial loci (Perfeito et al. 2007). Given these rough estimates, we would expect to find one cheater for every 10,000–100,000 individuals. This amounts to a large number of cheaters at equilibrium in microbial populations, where densities can be of order 10^8 cells mL^{-1} , and in eusocial insects, where colonies can contain millions of individuals (Hölldobler and Wilson 1990). Of course, population structure prevents mutants from being uniformly distributed among groups, so the actual cheater count in any given group in nature will be higher or lower than this value. With multiple loci (higher μ) and/or weaker selection, the equilibrium frequency of cheaters will be much higher. Clearly, more estimates of the mutation rate μ , selection coefficients (e.g., $br - c$), and local population regulation α are needed for social traits before we can forecast the expected number of cheaters in any system. Our theory shows that cheaters will be present in all social systems and that their frequency depends on the strength of selection, the population genetic structure, and the ecology of local density regulation.

The mathematical models developed here are simple but nonetheless incorporate a great deal of biologically realistic complexity. We have accounted for multiple genetic systems (haploidy, diploidy, haplodiploidy), patterns of sex-limited expression (female-limited expression), mating systems (monogamy, polyandry), and the effects of ecology through the spatial scale of density regulation. Future models should add overlapping generations, multiple loci, dominance, and frequency-dependent selection in order to encompass a more complete range of biological complexity. While we have contrasted frequency dependence and recurrent mutation as processes that maintain cheaters in populations, these phenomena are not mutually exclusive: mutation will always introduce a constant stream of cheaters into populations, which will contribute to variation even when cheaters are maintained by nonlinear frequency dependence. In general, more theoretical work is necessary for the full development of a mutational theory of kin selection, which will be important in generating novel explanatory and predictive theories for social evolution, particularly at the molecular level.

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APPENDIX A

Derivation of Allele Frequency Recursion

Following the procedure in appendix A of Van Dyken (2010), equation (1) can be derived by use of a standard n -ploid population genetic model with Hamilton's linear fitness function. We present the derivation for diploids, which is readily extended to haploids. Consider a diploid population with alleles A and a in frequency p and q , respectively, segregating at a single biallelic locus controlling altruism (see "The Model"). Let p_{ij} be the frequency of allele A in individual j in group i , with the values $(0, 1/2, 1)$; p_i be the mean frequency of allele A in group i , and p be the mean frequency of A in the global population. Expressing the altruistic trait decreases the fitness of the altruist j by an amount c_j but increases the fitness of each group member by an average amount b_k . Assuming that Hardy-Weinberg proportions obtain, on average, within groups, the mean and genotypic fitnesses in group i are

$$\bar{W}_i = p_i^2 W_{AA,i} + 2p_i(1 - p_i)W_{Aa,i} + (1 - p_i)^2 W_{aa,i} \quad (\text{A1a})$$

$$W_{i,AA} = 1 + b_k N_{\text{alt},i} - c, \quad (\text{A1b})$$

$$W_{i,Aa} = 1 + b_k N_{\text{alt},i} - hc, \quad (\text{A1c})$$

$$W_{i,aa} = 1 + b_k N_{\text{alt},i}, \quad (\text{A1d})$$

where $N_{\text{alt},i} = N_i[p_i^2 + 2p_i(1 - p_i)h]$ is the number of altruists in group i (Wilson 1975; Wade 1978, 1979, 1980). This fitness model is for "whole-group" altruism (Pepper 2000), where altruists receive the benefit of their altruistic actions. "Others-group" altruism can be modeled by making a simple substitution to this fitness model (see app. A of Van Dyken 2010).

The multilevel Price equation (Price 1972; Hamilton 1975; Wade 1985; Rice 2004) can be used to generate an allele frequency recursion for our model. We can write the Price equation as

$$\bar{W}\Delta p = \text{Cov}(w_i, p_i) + E(\text{Cov}(w_{ij}, p_{ij})), \quad (\text{A2})$$

where the first term is the change in allele frequency due to selection among groups and the second term is change due to selection within groups. We can rewrite this equation into its regression coefficient form (Wade 1985), which, assuming additivity ($h = 1/2$), gives

$$\overline{W}\Delta p = \beta_{w_i, p_i} V_b + \beta_{w_{ij}, p_{ij}} V_w, \quad (\text{A3})$$

where β_{w_i, p_i} and $\beta_{w_{ij}, p_{ij}}$ are the partial regression coefficients of group and individual allele frequency on group and individual fitness, respectively (Frank 1998; Rice 2004), V_b is the variance in allele frequency among all groups, and V_w is the average within-group variance in individual allele frequency (i.e., $V_w = E(\text{Var}_w(p_{ij}))$). Values for these variance components are given for neutral alleles by Wright (1951, 1965). The among-group partial regression coefficient β_{w_i, p_i} can be found by taking the derivative of equation (A1a) with respect to p_i . The within-group partial regression coefficient $\beta_{w_{ij}, p_{ij}}$ is the difference in the marginal within-group fitness of each allele (Rice 2004). This can be written as $\beta_{w_{ij}, p_{ij}} = (W_{A,i}^* - W_{a,i}^*)$, where $W_{A,i}^* = p_i W_{AA,i} + (1 - p_i) W_{Aa,i}$ and $W_{a,i}^* = (1 - p_i) W_{aa,i} + p_i W_{Aa,i}$ are the marginal fitnesses of the *A* and *a* alleles, respectively, within group *i*. Thus, we find that $\beta_{w_i, p_i} = (b - c)$ and $\beta_{w_{ij}, p_{ij}} = -c$. Substituting into equation (A3), further substituting $1 - q$ for p , and then simplifying gives equation (1).

Kin competition can be modeled by modifying the multilevel Price equation as follows:

$$\overline{W}\Delta p = (1 - \alpha) \text{Cov}(w_i, p_i) + E(\text{Cov}(w_{ij}, p_{ij})) \quad (\text{A4})$$

(Van Dyken 2010). Here, α is the intensity of kin competition. Following the same procedure as above but using equation (A4) instead of equation (A2) leads to equations (16) and (17).

APPENDIX B

Haplodiploid and Diplodiploid Models with Female-Limited Altruism Expression

If a population is structured into different classes of individuals, such as different sexes, and if classes differ in ploidy, then allele frequency change must be computed separately for each class. In haplodiploids, each sex composes a class, where one sex (typically males) is haploid and the other (typically females) is diploid. The allele frequency change in each sex is weighted by the reproductive value of the sex. In haplodiploids, males are weighted by 1/3 and females by 2/3 (Taylor 1988). Thus, the total allele frequency change is given by

$$\Delta p = \frac{1}{3} \Delta p_m + \frac{2}{3} \Delta p_f, \quad (\text{B1})$$

where for each sex *l* the change is

$$\overline{W}_l \Delta p_l = \text{Cov}(w_{li}, p_{li}) + E(\text{Cov}(w_{lij}, p_{lij})). \quad (\text{B2})$$

Assuming that only females (the diploid sex) express al-

truism, we can write the following genotypic fitnesses for females and males:

$$\begin{aligned} W_{fi,AA} &= 1 + b_k N_{alt,i} - c, \\ W_{fi,Aa} &= 1 + b_k N_{alt,i} - hc, \\ W_{fi,aa} &= 1 + b_k N_{alt,i}, \\ W_{mi,A} &= 1 + b_k N_{alt,i}, \\ W_{mi,a} &= 1 + b_k N_{alt,i}. \end{aligned} \quad (\text{B3})$$

Assuming that selection is weak, Hardy-Weinberg proportions hold approximately and allele frequency is approximately equivalent in males and females after reproduction. With these assumptions, combining equations (B1)–(B3) and doing some algebra gives

$$\overline{W}\Delta p = bs_f \left(\frac{2}{3} V_{b,f} + \frac{1}{3} \text{Cov}(p_{fi}, p_{mi}) \right) - c \frac{2}{3} V_{t,f}, \quad (\text{B4})$$

where s_f is the proportion of females in a group ($s_f = N_f/N$), which appears because only females are altruists. Using the relations of equations (2a), (2b), and (3) and noting that $V_{b,f}/V_{t,f} = r_{ff}$ (the regression relatedness among females) and $(\text{Cov}(p_{fi}, p_{mi}))/V_{t,f} = r_{fm}$ (the regression relatedness of females to males), we obtain equation (10).

To find the recursion for female-limited expression of altruism in diploids, we follow the same procedure as above but let both sexes be diploid and weight the contribution of each sex by 1/2 (i.e., the reproductive value of each sex in diploids). After some algebra, we find that

$$\Delta p = \frac{pq(1 + f_{st,f})[bs_f r - (1/2)c]}{2\overline{W}}. \quad (\text{B5})$$

This requires the fact that there are no relatedness asymmetries in diploids, so that females are equally related to sisters and brothers.

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