



UvA-DARE (Digital Academic Repository)

Evolution of altruism: Exploring adaptive landscapes

Spichtig, M.

Publication date
2013

[Link to publication](#)

Citation for published version (APA):

Spichtig, M. (2013). *Evolution of altruism: Exploring adaptive landscapes*. [Thesis, fully internal, Universiteit van Amsterdam].

General rights

It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations

If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: <https://uba.uva.nl/en/contact>, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

The evolution of altruism as a sexually selected handicap

Mathias Spichtig · Martijn Egas · Maurice W. Sabelis

Abstract It is claimed that sexual selection may favor altruism as a handicap. Here, we challenge these claims by developing a model for sexual selection acting on an altruistic handicap trait. By means of simulations we investigate if altruism can evolve as a sexually selected handicap trait despite the fact that competition for mating opportunities may cause altruists to benefit their competitors. We study two scenarios in which individuals with different fitness benefit from altruistic acts. In the first scenario, the benefits from altruism accrue to individuals with high fitness, whereas in the second they are the same for all individuals independent of their fitness. Simulations show that the evolution of altruism is hampered when all recipients benefit equally, but promoted when fitter recipients benefit more. Thus, dependent on how altruism benefits recipients with different fitness, sexual selection can promote altruism as a handicap.

1 Introduction

Altruists incur a fitness cost to benefit others, leading to a conundrum for the evolution of altruism: how can altruistic traits evolve when they reduce the fitness of altruists? Current theories focus on mechanisms involving kin selection, group selection and reciprocity (Lehmann and Keller 2006; Nowak 2006; West et al. 2007a,b). Considered from the perspective of sexual selection, however, a different mechanism emerges. Altruists may gain mating chances when the fitness costs they incur represent a handicap that is serious enough to be an honest signal of high fitness. This has led several authors to propose that altruism as a handicap could be favored by sexual selection (Zahavi 1977, 1995; Tesson 1995; Zahavi and Zahavi 1997; Miller 2000, 2007; Walker 2008; for a critical view see Driscoll 2006).

Various lines of empirical evidence support a role for sexual selection in the evolution of altruism. In birds, for instance, altruism may be expressed in the form of risky

M. Spichtig, M. Egas, M.W. Sabelis
Institute for Biodiversity and Ecosystem Dynamics (IBED), University of Amsterdam,
P. O. Box 94248, 1090 GE Amsterdam, The Netherlands

alarm calls or food sharing and the evidence for interpreting this as a handicap is discussed by several authors (Zahavi and Zahavi 1997; Wright et al. 2001; Doutrelant and Covas 2007; Scheid et al. 2008). With respect to humans, Gurven (2003) proposed that altruism can be a costly signal and thus a handicap. Recently, Griskevicius et al. (2007), Philips et al. (2008), and Barclay (2010) report evidence that humans consider altruistic behavior to be an attractive trait of mating partners. They also interpret altruistic behaviors in humans as handicap traits subject to sexual selection.

Despite this empirical evidence and repeated verbal claims for altruism as a handicap under sexual selection, there is still no theoretical work to substantiate this claim. On the one hand there are models of sexual selection on handicap traits (Grafen 1990a,b; Houle and Kondrashov 2001; for a review see Kokko et al. 2006). On the other hand there are models of the evolution of altruism that do not consider sexual selection (e.g., Lehmann and Keller 2006; Nowak 2006; West et al. 2007a,b), perhaps because the existing empirical evidence for altruism as a sexually selected handicap was perceived as meager (see Clutton-Brock 2009). Gintis et al. (2001) and Lotem et al. (2003) provide theory on the evolution of altruism as a costly signal, but they only consider individual selection, not sexual selection.

Here, we challenge the verbal claims for altruism as a sexually selected handicap. To do so, we extend the model of Gintis et al. (2001) by including sexual selection acting on the handicap trait. The aim of our study is to investigate if altruism can evolve as a sexually selected handicap trait despite the fact that competition for mating opportunities may cause altruists to benefit their competitors. We study two scenarios in which individuals with different fitness benefit from altruistic acts. In the first scenario, the benefits from altruism accrue to individuals with high fitness. In the second scenario, equivalent to that assumed by Gintis et al. (2001), the benefits from altruism are the same for all individuals independent of their fitness. By the aid of numerical simulations, we explore how the social impact of altruism affects its evolution and whether a preference for altruists implies a preference for high fitness individuals.

2 Theoretical setting

Following Gintis et al. (2001), we consider two dimorphic traits – the quality trait indicating either ‘high fitness’ or ‘low fitness’ and the sender trait indicating either ‘sender’ or ‘non-sender’. Only the sender trait can be perceived by others. In the model of Gintis et al. (2001), the population may evolve to a state where high fitness individuals are senders and low fitness individuals are non-senders. This population state of ‘honest signaling’ can be achieved when sending a signal comes at a cost and this cost is too large for low fitness individuals, but not for high fitness individuals (Gintis et al. 2001; Getty 2006). Costly signaling and honest signaling are thus synonymous terms, in that the former refers to cause while the latter refers to effect. In our model it is the sender who incurs a cost of sending and thereby carries a handicap.

In contrast to the model of Gintis et al. (2001) where traits are culturally inherited, our model is of the population genetic type (*sensu* Pen and Weissing 2000) where traits are genetically inherited from two parents. Importantly, such a model type includes direct sexual selection on the genetically determined sender trait, but also indirect selection on the genetically determined quality trait. In addition, there is a third genetic trait determining preferences for either morph of the sender trait. This preference gives

rise to discrimination among mating partners, thereby creating sexual selection. As a consequence of these modifications, the frequency of high fitness individuals can change during evolution in our model, whereas it is fixed in the model originally formulated by Gintis et al. (2001). In the main analysis presented in this article (formal representation in Appendix 1-1), we ignore costs of choosiness, a factor typically considered in sexual selection models. This is because – as shown in Appendix 1-2 – extending the model by including costs of choosiness did not alter the main conclusions. Also, to avoid the complications of the paradox of the lek (Kirkpatrick and Ryan 1991), we allow for high mutation rates at the locus that determines the fitness value.

Following Gintis et al. (2001), we assume that senders can have a social impact: they increase (through pro-social impacts) or decrease (through anti-social impacts) the fitness of all others in the population (i.e., they provide a public good or a public bad, respectively). Senders are altruists if they incur a sending cost (the cost of altruism) whereas others benefit (through pro-social impact). Assuming that social impact and fitness differences concern the same components of fitness, altruism can affect the fitness differences that the handicap trait may inform about. If this assumption would not hold (e.g., when fitness differences concern males and males direct altruistic acts towards females, e.g., nuptial gift), altruism can only have marginal effects on such fitness differences. Using this assumption, we provide an analysis to show how social handicap traits can evolve by sexual selection, even when an altruistic male promotes the fitness of all individuals including its competitors, rather than only the fitness of potential mating partners. We emphasize that social handicaps can lead to benefitting competitors which is not the case with non-altruistic handicaps (e.g., the ‘peacock tail’).

3 Model description

We model an infinite population of haploid hermaphrodites that evolves in discrete non-overlapping generations (see Kokko [2007] for a comparison of models with haploid or diploid genetics). Their life history is as follows: The haploid adults produce haploid (anisogamic) gametes that fuse with other gametes, thereby creating diploid zygotes. The zygotes meiotically segregate into the haploid adults of the next generation. For simplicity, mutation occurs only at segregation. The quality trait and the sender trait, which are determined by three loci, influence the number of eggs produced by the individual. The production of sperm is assumed unlimited. The preference trait, determined by a fourth locus, influences the choice of the partner that fertilizes the eggs.

The model consists of a system of difference equations (see Appendix 1-1) whereby each equation determines a genotype frequency. In this way, bookkeeping of genotype frequencies is performed for all possible genotypes that emerge given the four loci. The quality trait is determined by di-allelic locus q . Individuals carrying allele q_h express the ‘high-quality’ morph of this trait. Individuals carrying allele q_l express the ‘low-quality’ morph. Mutation transforms allele q_h to allele q_l at rate μ_{qh} and allele q_l to allele q_h at rate μ_{ql} . We assume asymmetric (high) mutation ($\mu_{qh} \neq \mu_{ql}$) to maintain non-marginal frequencies of these alleles even under opposing selection: in this way we prevent the paradox of the lek. Locus q is pleiotropic as the locus co-determines the morph of the sender trait together with di-allelic loci p_h and p_l . A carrier of allele q_h

expresses the ‘sender’ morph if it carries allele (+) at locus p_h . The ‘non-sender’ morph is expressed if it carries allele (-). For carriers of allele q_l , the sender phenotype is analogously determined by locus p_l . This three-loci genotype allows that information about the (unperceivable) quality trait can be obtained from the (perceivable) sender trait whereby the quality of this information depends on the genotype frequency distribution at these loci. For example, fixation of genotype $\{p_h, p_l\} = \{(+), (-)\}$ means that the mating partner carries the high-quality allele q_h if a sender is selected as partner and the partner carries the low-quality allele q_l otherwise. Such fixation corresponds to honest signaling as defined by Gintis et al. (2001). At loci p_h and p_l , mutation swaps the allele at rate $\mu_p (> 0)$. The preference trait is determined by locus x . The four loci $\{q, p_h, p_l, x\}$ are unlinked and hence freely recombine.

3.1 Quality trait, sender trait, and their effects on fitness

By selecting a mating partner, a (haploid) parent selects the gametes that fuse with the own gametes into a zygote. To emphasize the perspective of the selecting parent, by eggs we refer to the gametes of the selecting parent (and by sperm to the gametes of the selected parent). All individuals obtain the same share from a resource w and convert their share into eggs. The quality trait determines the rate of conversion. The contribution of eggs by a non-sender is $w_{n-S,l} = l (> 0)$ if it is of low-quality and $w_{n-S,h} = h = k l$ (hence, $k = \frac{h}{l}$) if it is of high-quality. From their share of the resource, high-quality individuals produce more eggs as we assume $k > 1$. Consequently, k constitutes the advantage in the conversion rate that high-quality individuals have over low-quality individuals.

Expressing the sender morph comes at cost $c (> 0)$, which – in addition to the quality trait – influences the number of eggs generated by senders. We distinguish two types of cost. The cost of sending is either paid in terms of resource (*quality-biased*) or in terms of eggs (*quality-unbiased*; i.e., an alternative resource for which the transformation into eggs is independent of locus q). Under *quality-biased* costs, the number of eggs from a low-quality sender is $w_{S,l} = l - c$ and the number of eggs from a high-quality sender is $w_{S,h} = h - k c$. The cost is therefore higher for high-quality senders, i.e., they sacrifice more eggs than low-quality senders. Under *quality-unbiased* costs, the costs of sending are independent of the quality trait, i.e., $w_{S,l} = l - c$ and $w_{S,h} = h - c$. We assume $w_{n-S,h} > w_{S,h} > w_{n-S,l} > w_{S,l} > 0$ (exceptions to be announced later). Hence, high-quality individuals produce more eggs than low-quality individuals. Furthermore, non-senders produce more eggs than senders of the same quality (i.e., same conversion rate).

In our model, sending a signal can have a social impact. Senders can increase the number of eggs generated by the population (pro-social impact). We specify the social impact each individual obtains as $r c s$ whereby r is the multiplication factor and s is the frequency of senders (i.e., the sending cost, c , is multiplied by r and every individual in the population gets the same share). The impact is pro-social if $r > 0$ (anti-social impact, $r < 0$, is ignored in this study). Sending has no social impact if $r = 0$. Senders are altruists whenever the impact is pro-social and the sending cost is then the cost of altruism.

As for the cost of sending, we make a distinction between the type of social impact that is additional resource (*quality-biased*) and the type that is additional eggs (*quality-unbiased*). We assume that the resource obtained through *quality-biased* social impact has the same conversion rate as the w -resource. Under this type, the total contribution of eggs by an individual with genotype i is $\omega_i = w_i + r c s$ if the genotype expresses low-quality and $\omega_i = w_i + k r c s$ if the genotype expresses high-quality. The benefit obtained from the pro-social impact is therefore higher for high-quality individuals. Under *quality-unbiased* social impact, the total contribution of eggs is $\omega_i = w_i + r c s$ for all genotypes and therefore independent of quality. Combining sending cost types (*quality-biased*, *quality-unbiased*) and social impact types (*quality-biased*, *quality-unbiased*) there are four scenarios that are further referred to by the following notation: {*quality-biased* costs, *quality-biased* impact}, {*quality-biased* costs, *quality-unbiased* impact}, {*quality-unbiased* costs, *quality-biased* impact}, {*quality-unbiased* costs, *quality-unbiased* impact}.

3.2 Preference trait

For each egg, a mating partner is selected. Individuals never reject the opportunity to fertilize the egg. This is assumed for simplicity because rejection would result in an alternative form of sexual selection. Individuals can perceive the signal trait morph of potential partners but not their quality. The selection of mating partners is determined by the allele at locus x . Two types of alleles can occur, i.e., type $x_{S,a}$ and type $x_{n-S,a}$. For the fraction a of its eggs, a carrier of allele $x_{S,a}$ ($x_{n-S,a}$) selects a sender (non-sender) as partner. For the other eggs, the partner is selected randomly from the population (random mating). Hence, individuals with an allele of type $x_{S,a}$ ($x_{n-S,a}$) express a preference for senders (non-senders). Allele x_0 is phenotypically identical to alleles $x_{S,0}$ and $x_{n-S,0}$ and individuals with this allele always mate randomly. Locus x contains a finite number, n_x , of alleles. At rate $\mu_x (> 0)$, mutation transforms the inherited x -allele with equal chance into any of the other alleles. As with loci q , p_h , and p_l , we thus assume that mutation results in discrete predetermined changes at locus x (alternative mutation regimes could consider the continuum underlying the definition of the x -alleles). This mutation regime results in a fixed number of genotypes and, more importantly, individuals with a preference for senders and individuals with a preference for non-senders are (due to mutation) maintained in the population (if locus x contains both allele types).

In any given generation, a fraction $e-S$ of eggs is fertilized through preference for senders, a fraction $e-n-S$ of eggs is fertilized through preference for non-senders, and for the remaining fraction of eggs ($1 - e-S - e-n-S$) mating is random. The formula for the expression of the preference for senders (non-senders) is given by $e-S = \frac{1}{\omega} \sum_{i \in \Phi} \phi_i \omega_i \alpha_i$ (by $e-n-S = \frac{1}{\omega} \sum_{i \in \Phi} \phi_i \omega_i \beta_i$), whereby $\alpha_i = a_i$ ($\beta_i = 0$) if genotype i contains allele $x_{S,ai}$ and $\alpha_i = 0$ ($\beta_i = a_i$) if it contains allele $x_{n-S,ai}$.

4 Simulations

We provide an analysis of the model through numerical simulations with the following initial conditions and criteria for characterizing end states of the simulations.

4.1 Initial states of simulations

In simulations, the populations are initialized by the following procedure. The frequency of one genotype (the initial genotype) is set to $\phi_i = 1$. The individuals of this population mate in order to produce the initial generation. We assume that mutation is recurrent and most alleles are faithfully inherited: $0.02 \geq \mu_{q_h}, \mu_{q_l}, \mu_p, \mu_x > 0$. In order to relax the paradox of the lek, we assume allele q_h to be more prone to mutation than allele q_l : $\mu_{q_h} > \mu_{q_l}$. This is assumed because q_h functions better than q_l and thus mutation more readily deteriorates q_h than q_l . Because the higher mutation rate makes the inheritance of q_h less faithful, we initiate simulations by setting genotypes with this allele to a high frequency. It should be noted that all genotypes are retained in the population as a consequence of mutation.

To start the simulations, we use one of three initial genotypes. These genotypes all have alleles for high quality and for not sending when being of low quality, but differ in sending when of high quality and in preference for senders (*Send*), for non-senders (*non-Send*), or for none (i.e., random mating: *Rand*). The initial genotype used for *Send*, therefore, represent high-quality senders that prefer senders: $i = \{q_h, p_h = (+), p_l = (-), x_{S,0.95}\}$. The initial genotype used for *non-Send* represents high-quality non-senders that prefer non-senders: $i = \{q_h, (-), (-), x_{n-S,0.95}\}$. The initial genotype used for *Rand* represent high-quality non-senders that always mate randomly: $i = \{q_h, (-), (-), x_0\}$. Thus, in initial state *Send* senders are abundant and preferred, whereas in initial states *Rand* and *non-Send* they are rare and not the preferred mating partners.

4.2 End states of simulations

Simulations are run until – for each genotype – the difference in frequency between consecutive generations is less than 10^{-9} . We provisionally refer to the final population state as ‘equilibrium’ although we realize more is needed to prove that it is an equilibrium. By equilibrium we only imply that all genotypes are present ($\phi_j > 0$) but none is invading because the response to selection is negligibly small. For two simulations that differ only in the initial state, we say the same equilibrium is attained if the frequency of senders, s^* , differs less than 10^{-7} between them. We refer to them as alternative equilibria, otherwise.

We consider a change in condition to promote the evolution of altruism if there are more initial states from which an equilibrium with sender dominance is reached. We use the term ‘sender dominance’ if the equilibrium frequency of senders $s^* > 0.90$ and ‘sender subdominance’ if $s^* > 0.6$.

5 Results

Given that we assume two types of sending costs (*quality-biased cost*, *quality-unbiased cost*) and social impact (*quality-biased impact*, *quality-unbiased impact*), there are four scenarios for which we perform simulations. These four scenarios are $\{\textit{quality-biased cost}, \textit{quality-biased impact}\}$, $\{\textit{quality-biased cost}, \textit{quality-unbiased impact}\}$, $\{\textit{quality-unbiased cost}, \textit{quality-biased impact}\}$, $\{\textit{quality-unbiased cost}, \textit{quality-unbiased impact}\}$. For each scenario, we explore how the multiplication factor (r) affects the frequency of senders (altruists) and the prevalence of high-quality individuals at equilibrium. Before dealing with equilibria, however, we first show how these are reached from the initial states.

5.1 Trajectories of high quality partner acquisition and preferences

Handicap traits are expected to be preferred when they correlate with fitness. Indeed, our simulations show this, for example when starting from initial state *Rand*. For *quality-unbiased costs* and without social impact ($r = 0$) we first observe an increase in the fraction of pairings that are formed by selecting a sender as mating partner (e - S in Fig. 1-1a). This is because the fraction of high-quality individuals among senders exceeds that among non-senders ($S_{gh} > n$ - S_{gh} in Fig. 1-1a): more high-quality partners are acquired by selecting senders than by selecting non-senders. Ultimately these fractions

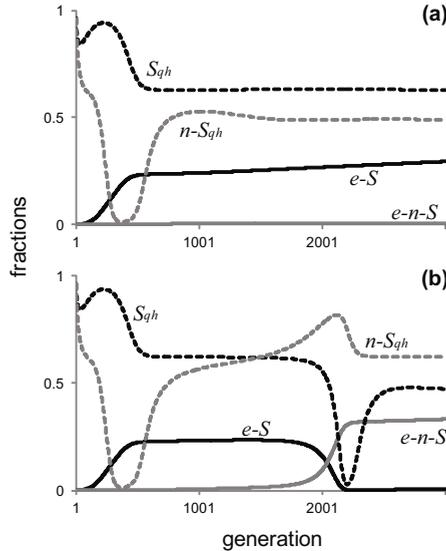


Fig. 1-1 Shown are the initial generations of two simulations with preference trait alleles $\{x_{S,0.95}, x_{S,0.05}, x_0, x_{n-S,0.05}, x_{n-S,0.95}\}$, with *quality-unbiased cost* (panel **a**), with *quality-biased cost* (panel **b**). Both simulations apply to the case without social impact ($r = 0$) and they start from initial state *Rand*. Both panels show the fraction of pairings that are formed by selecting senders, e - S (black continuous line), the fraction of pairings that are formed by selecting non-senders, e - n - S (gray continuous line), the fraction of high quality individuals among senders, S_{gh} (broken black line), and the fraction of high quality individuals among non-senders n - S_{gh} (broken gray line). Parameters: $r = 0$, $c = 0.02$, $\mu_{gh} = 0.02$, $\mu_{ql} = 0.001$, $u_{ph} = u_{pl} = 10^{-4}$, $u_x = 10^{-4}$, $h = 1.0$, $l = 0.9$, $k = hl^{-1}$.

reach the equilibrium state shown in Fig. 1-2b. For *quality-biased costs* and without social impact ($r = 0$) we again first observe an increase in the fraction of pairings that are formed by selecting a sender as mating partner ($e-S$ in Fig. 1-1b) and again the fraction of high-quality individuals among senders exceeds that among non-senders ($S_{gh} > n-S_{gh}$ in Fig. 1-1b). However, this holds only for the first 1480 generations. Thereafter, the fraction of high-quality individuals among non-senders exceeds that among senders ($n-S_{gh} > S_{gh}$ in Fig. 1-1b). In these subsequent generations we observe an increase in the fraction of pairings that are formed by selecting a non-sender as mating partner ($e-n-S$ in Fig. 1-1b). This goes at the expense of the fraction of pairings that are formed by selecting a sender as mating partner ($e-S$ in Fig. 1-1b). Ultimately these fractions reach the equilibrium state shown in Fig. 1-2a. In both scenarios, individuals produce more offspring if they express a preference that gives the highest chance of acquiring a high-quality partner. This mechanism results in a straight approach towards an equilibrium with sender dominance for the case of *quality-unbiased costs* (Fig. 1-1a). However, for the case of *quality-biased costs* it first approaches sender dominance and then switches to approach an equilibrium with non-sender dominance (Fig. 1-1b).

5.2 Effects of the multiplication factor on the equilibrium frequency of the sender trait

Simulations of all four scenarios always reached an equilibrium state for r equal to 0, 1, 10, 100, 1000 and 10000, and with the preference trait locus x alleles $\{x_{S,0.95}, x_{S,0.05}, x_0, x_{n-S,0.05}, x_{n-S,0.95}\}$ (all other parameters given in Fig. 1-2). For the equilibrium frequencies of senders (s), we distinguish three types of equilibria (Fig. 1-2): equilibria with sender dominance ($s \sim 1$), equilibria with non-sender dominance ($s \sim 0$), and equilibria without a dominant (sender trait) morph ($s \sim 0.5$). From initial states *Send* and *Rand*, equilibria with sender dominance are reached for several combinations of r -values and scenarios (Fig. 1-2) but equilibria with sender dominance are not reached from initial state *non-Send*. Note that when a high-quality partner is acquired, the offspring can inherit the high-quality allele q_h from this partner. If senders are dominant ($s \sim 1$), practically all low-quality individuals are senders (altruists) and selecting a sender (rather than a random partner) does not quite help in finding a high-quality partner. Then, the sender morph does not function as an honest handicap trait signal for the quality trait.

The average costs of sending are higher under *quality-biased cost* (since high-quality senders incur cost $k c$) than under *quality-unbiased cost* (since high-quality senders incur cost c). To evaluate this difference we ran additional simulations. For the two scenarios with *quality-unbiased cost*, we increased the cost of sending from c to $k c$, *i.e.*, the value incurred by high-quality senders under *quality-biased cost*. The results were practically the same as those shown in Fig. 1-2b,d (and also Fig. 1-4b,d that will be discussed later) (data not shown). Thus, the higher average cost of sending under *quality-biased cost* (as opposed to *quality-unbiased cost*) cannot explain the results in Fig. 1-2 (and Fig. 1-4 to be discussed later).

5.3 The preference trait at equilibrium

At equilibria with sender dominance the frequencies of the x -alleles decrease in the following order: $x_{S,0.95}$, $x_{S,0.05}$, x_0 , $x_{n-S,0.05}$, $x_{n-S,0.95}$. At equilibria with non-sender dominance the frequencies decrease in the reverse order. The five frequencies are practically equal at the equilibria without a dominant morph.

That sexual selection drives evolution towards dominance of a single morph, is particularly clear at equilibria with sender dominance because senders produce less eggs (i.e., the gametes for which they choose mating partners) than non-senders of the same quality. That equilibria with sender dominance are reached (Fig. 1-2), is taken to indicate evolution of altruism by sexual selection. At equilibria without a dominant morph mating is not strictly random (about 60% of the eggs are born to a random pair of partners). However, as both preferences are expressed equally frequently, sexual selection favors no morph over another at such equilibria.

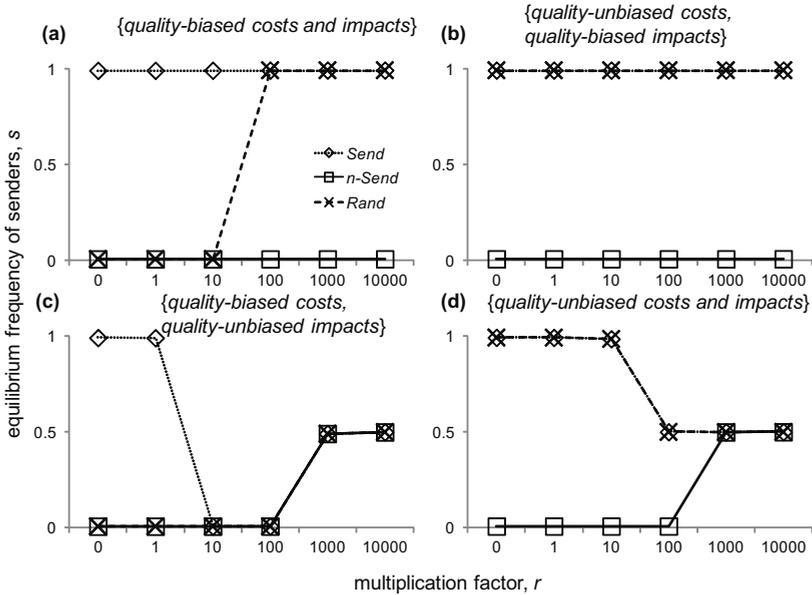


Fig. 1-2 Shown are equilibrium fractions of senders, s , as a function of the multiplication factor, r , from simulations for the four cost/impact scenarios (corresponding to four panels): (a) costs of sending and social impacts are resource (i.e., the cost is higher for high-quality senders and high-quality individuals benefit more from the altruistic impact as high-quality individuals convert the resource into more eggs) in scenario {quality-biased cost, quality-biased impact}, (b) costs are eggs (i.e., costs are the same for all senders) and impacts are resource in scenario {quality-unbiased cost, quality-biased impact}, (c) costs are resource and impacts are eggs (i.e., all individuals benefit the same from these impacts) in scenario {quality-biased cost, quality-unbiased impact}, and (d) costs and impacts are eggs in scenario {quality-unbiased cost, quality-unbiased impact}. The s -values of the simulations starting from initial states *Send*, *Rand*, and *n-Send* are indicated with diamond (dotted lines), cross (continuous lines), and square symbols (dashed lines); symbols (and lines) overlap whenever the same equilibrium is attained (whenever the same equilibria are attained between two r -values). The simulations are done with alleles $\{x_{S,0.95}, x_{S,0.05}, x_0, x_{n-S,0.05}, x_{n-S,0.95}\}$ at mating preference locus x and the other parameters are $\mu_{qh} = 0.02$, $\mu_{ql} = 0.001$, $u_p = 10^{-4}$, $u_x = 10^{-4}$, $h = 1.0$, $l = 0.9$, $k = l^{-1}$, $c = 0.02$.

5.4 Effects of social impacts on morph dominance at equilibrium

We first consider simulations with *quality-biased social impact*, i.e., high-quality individuals benefit more. In this case, the system evolves to sender dominance from initial state *Send*, whereas it evolves to non-sender dominance from initial state *non-Send* (Fig. 1-2a,b). Evolution from initial state *Rand* depends on the type of sending costs, however (compare Fig. 1-2a and 1-2b). For *quality-biased cost*, equilibria with non-sender dominance arise for $r \leq 10$ and equilibria with sender dominance for $r > 10$ (Fig. 1-2a). For *quality-unbiased cost*, equilibria with sender dominance are obtained for all r -values (Fig. 1-2b). The effect of the cost type (*quality-biased cost*, *quality-unbiased cost*) therefore emerges only for $r \leq 10$ (compare Fig. 1-2a and 1-2b) and increasing r (and thereby the effectiveness of altruism) promotes the evolution of altruism only under scenario $\{\textit{quality-biased cost}, \textit{quality-biased impact}\}$.

It is important to note that equilibria with sender dominance (and $r > 0$) are reached from initial state *Rand*: a population of high-quality non-senders that mate randomly. Because senders arise by mutation, are therefore rare in the initial state *Rand* and experience a fitness cost for expressing the sender trait, they must have been favored by sexual selection to become the dominant morph. To illustrate such evolution, the frequencies of the four genotypes at loci $\{p_h, p_l\}$ (which, together with locus q , determine the sender morph) in the initial generations from initial state *Rand* are shown in Fig. 1-3a for the simulation of Fig. 1-2a with $r = 100$. The depicted evolution is representative for the dynamics leading from *Rand* to an equilibrium with sender dominance. The honest signaler genotype ($\{p_h, p_l\} = \{+, -\}$) has initially the steepest increase (Fig. 1-3a). Genotype $\{+, +\}$ supplants the honest signaler genotype as the most common genotype in the following (Fig. 1-3a; alternatively, this be inferred from $s \sim 1$ at equilibrium [Fig. 1-2a]). The attractiveness of senders (the frequency of allele $x_{S,0.95}$) increases over the depicted generations (data not shown). The results of Fig. 1-3a imply that this attractiveness can compensate for the sending costs. For high-quality senders, the compensation is thereby effective at lower attractiveness than for low-quality senders.

We now consider simulations with *quality-unbiased social impact*, i.e., benefits are independent of the quality of the individuals. Here, equilibria with a predominant morph arise for $r \leq 100$ (Fig. 1-2c,d), whereas those without a dominant morph arise for higher r -values ($r \geq 1000$ in Fig. 1-2c and $r \geq 100$ in Fig. 1-2d). Equilibria without a dominant morph arise due to high *quality-unbiased* social impacts (i.e., high r) (compare Fig. 1-2c,d with 1-2a,b). For *quality-biased cost* senders dominate if $r \leq 1$ and the initial state is *Send* (Fig. 1-2c) whereas for *quality-unbiased cost* senders dominate if $r \leq 10$ and the initial genotype states are *Send* or *Rand* (Fig. 1-2d). For these ranges of r -values, equilibria with (non-)sender dominance evolve from the same initial states as in simulations with identical cost type and with *quality-biased social impact* (compare Fig. 1-2c with 1-2a and 1-2d with 1-2b). Hence, sending cost type is the sole determinant of the initial states from which these equilibria evolve which is explained by the low impacts, $r c s$, at low r -values. Various effects of the social impact type on the equilibrium can be found for $r \geq 10$ (compare Fig. 1-2c with 1-2a) respectively for $r \geq 100$ (compare Fig. 1-2d with 1-2b). Particularly, in contrast to simulations with *quality-biased impact* (Figs. 2a,b), no equilibria with sender dominance arise for these multiplication factors in simulations with *quality-unbiased impact* (Figs. 2c,d). It is important to note that – for *quality-unbiased social*

impact – increasing r (the effectiveness of altruism) negatively affects the evolution of altruism through sexual selection, whereas – for *quality-biased social impact* – the effect was positive.

5.5 Effects of social impacts on the prevalence of high-quality individuals

How social impacts affect the equilibrium fraction of high-quality individuals (i.e., the frequency of allele q_h), is shown in Fig. 1-4, using the same simulations as shown in Fig. 1-2.

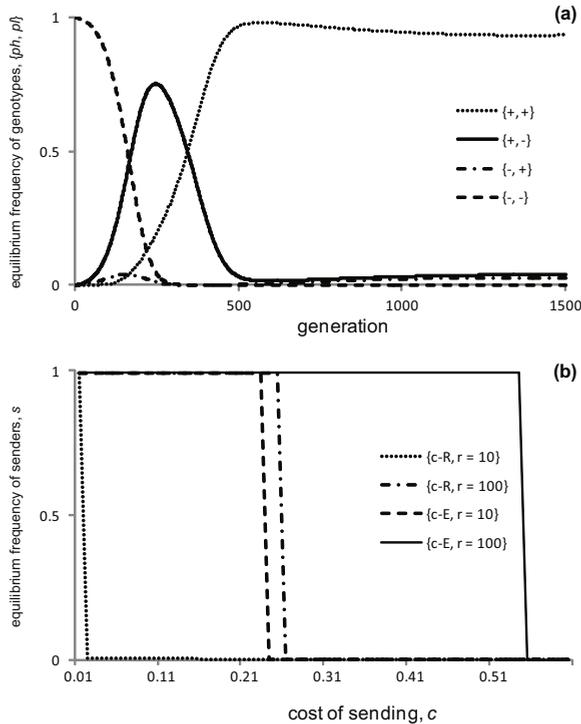


Fig. 1-3 Shown are, in panel (a), the evolution of the frequencies of the four genotypes at loci $\{p_h, p_l\}$ in the initial generations of the simulation starting from initial state *Rand* in Fig. 1-1a at $r = 100$. The frequencies of genotypes $\{+, +\}$, $\{+, -\}$, $\{-, +\}$, and $\{-, -\}$ are indicated with a dotted line, a continuous line, an irregularly broken line, and a regularly broken line. Genotype $\{+, -\}$ is the honest signaler genotype and genotype $\{+, +\}$ is the genotype that reaches near fixation at equilibrium (with $s \sim 1$, see Fig. 1-1a). Shown are, in panel (b), equilibrium frequencies of senders, s , as a function of the sending cost, c , from simulations starting from initial state *Rand* (the parameters [except from c] are those from Figs. 1a,b at $r = \{10, 100\}$). Results from simulations with conditions $\{quality\text{-biased cost}, r = 10\}$, $\{quality\text{-biased cost}, r = 100\}$, $\{quality\text{-unbiased cost}, r = 10\}$, and $\{quality\text{-unbiased cost}, r = 100\}$ are show with a dotted line, an irregularly dashed line, a regularly dashed line, and a continuous line. The range of c -values is chosen such that all four conditions, an equilibrium with sender dominance is attained at the lowest value ($c = 0.01$) and an equilibrium with non-sender dominance is attained at the highest value ($c = 0.6$).

In all simulations with *quality-biased social impacts* (Figs. 1-4a,b) fractions of high-quality individuals are close to 0.62 and thus hardly affected by r or s (see Fig. 1-2a,b). Because the product $r c s$ determines social impact size and s differs for each equilibrium, the prevalence of high-quality individuals cannot depend on social impact. High-quality individuals clearly prevail in the population at equilibrium which is probably because they lay $(k - 1)$ more eggs than low-quality individuals for each ‘unit’ of social impact. Moreover, for $r = 0$, high-quality non-senders also lay $(k - 1)$ more eggs than low-quality non-senders and high-quality senders lay more than low-quality senders, albeit not exactly $(k - 1)$ more eggs due to sending costs. Thus, whether senders or non-senders prevail at equilibrium, high-quality individuals maintain higher relative fitness for all social impact levels and this may explain why they prevail.

In simulations with *quality-unbiased social impacts* the equilibrium fractions of high-quality individuals tend to decrease with increasing r (Fig. 1-4c,d). The frequency of high-quality individuals is lower the higher r , the higher s (see Fig. 1-2c,d) and hence, the higher the social impact ($r c s$) (Fig. 1-4c,d). With *quality-unbiased social impacts*, the differences in the number of eggs produced by the individuals remain (as $|\omega_i - \omega_j| = |w_i - w_j|$ whereby $w_i, w_j \in \{w_{S,h}, w_{S,l}, w_{n-S,h}, w_{n-S,l}\}$) but every individual (independent of phenotype) produces more eggs ($\omega_i = w_i + r c s$) because of the social impact. Hence, the fitness of high-quality individuals (to be precise, the difference in the number of eggs produced relative to that of low-quality individuals) decreases with increasing pro-social impacts (i.e., increasing r and s). Since the fitness of these individuals is determined by allele q_h , the frequency of this allele decreases in concert (Fig. 1-4c,d).

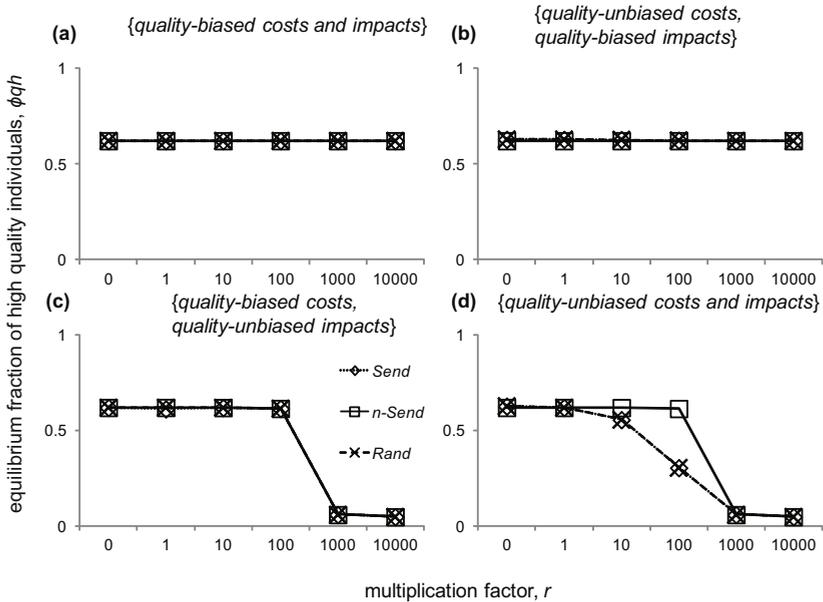


Fig. 1-4 Equilibrium fractions of high-quality individuals (i.e., the equilibrium frequency of allele q_h) are shown as a function of the multiplication factor, r , for each of the four scenarios considered in Fig. 1-2.

Apart from the allele at locus q , also the cost of sending creates w -fitness differences. For both types of social impact (*quality-unbiased impact* and *quality-biased impact*) the relative cost of sending decreases with increasing social impact: the prices remain the same while everybody gets richer (fitter). For the same reason, the relative cost difference between high-quality senders and low-quality senders under *quality-biased cost* decreases with increasing impact. In simulations with *quality-biased impact*, equilibria with sender dominance are reached from initial state *Rand* only for $r \geq 100$ with *quality-biased cost* (Fig. 1-4a) and for all r -values with *quality-unbiased cost* (Fig. 1-4b). We infer from this finding that in simulations with $\{\textit{quality-biased cost, quality-biased impact}\}$, the higher sending cost for high-quality senders (i.e., the difference between *quality-biased cost* and *quality-unbiased cost*) impedes the evolution to this equilibrium for $r \leq 10$. For higher r -values, the social impact sufficiently decreases the relative cost difference to overcome this impeding effect.

Sufficiently high sending costs (of both types) should impede the evolution towards equilibrium with sender dominance. Using the conditions of Fig. 1-2a,b at $r = \{10, 100\}$, the effects of r on this impeding effect is investigated in simulations starting from initial state *Rand*: the equilibrium frequency of senders, s , is shown as a function of sending cost c in Fig. 1-3b. For each cost type (*quality-biased cost* and *quality-unbiased cost*), the transition from $s \sim 1$ to $s \sim 0$ is at a higher c -value with the higher factor $r = 100$ (Fig. 1-3b). We thus find a promoting effect of r on the evolution of sender dominance for *quality-biased cost* and (in contrast to Fig. 1-2) also for *quality-unbiased cost*. Note that without pro-social impact, low-quality non-senders generate more eggs than high-quality senders ($w_{S,h} < w_{n-S,l}$) for a considerable fraction of the simulations in Fig. 1-3b. Due to the impact of type *quality-biased impact*, high-quality senders might still generate more eggs than low-quality non-senders (i.e., $\omega_{S,h} > \omega_{n-S,l}$) under these conditions.

Since high *quality-unbiased social impact* (i.e., high r) give rise to equilibria without a dominant morph (Fig. 1-2c,d), we infer that the relative differences in the number of eggs produced between the four phenotypes become marginal (i.e., $\omega_{S,h} \sim \omega_{S,l} \sim \omega_{n-S,h} \sim \omega_{n-S,l} \sim r c s$). If so, selection of mating partners does not matter. This does not apply to *quality-biased social impacts*, because high-quality individuals produce more eggs due to the social impact (i.e., $\omega_{S,h} \sim \omega_{n-S,h} \sim k r c s > \omega_{S,l} \sim \omega_{n-S,l} \sim r c s$) and hence equilibria without a dominant morph do not arise even for very high social impacts.

6 Discussion

Our simulations show that sexual selection can drive the evolution of altruism, but only under a special set of conditions. First, the initial composition of the population matters (Fig. 1-2). From an initial state where senders are preferred as mating partners (*Send*) or where mating is random (*Rand*), equilibria with sender dominance can be reached, but not from an initial state where non-senders are preferred (*non-Send*). Second, the type of social impact matters (Fig. 1-2a,b vs. 1-2c,d). If altruism is directed towards high-quality individuals (as is the case under *quality-biased impact*) and r sufficiently high, equilibria with sender dominance are reached (Fig. 1-2a,b and 1-3b), but much less so if altruism is directed to all individuals equally (as is the case under *quality-unbiased impact*) (Fig. 1-1c,d). Moreover, under *quality-biased impact*, frequencies of high-quality alleles (q_h) are maintained at a high level, but under *quality-*

unbiased impact, these frequencies decline (Fig. 1-4). Clearly, in our model, acquiring mating partners of high-quality drives sexual selection for altruism.

Some of the assumptions need scrutiny. We assumed sending costs to be fixed and thus independent of the actual mating opportunities. However, altruism as a form of courtship behavior needs to be expressed only whenever there is a mating partner offering a fertilizable egg. If so, costs to the sender increase with the frequency of altruists in the population (because we modeled altruism in terms of gifts that increase the number of eggs). When fixed, the costs relative to the number of laid eggs (in our model, the sending cost decreases the number of eggs of the sender while altruism increases the number of eggs for all individuals) decreases and this promotes the effect of r on the evolution of altruism. However, when increasing with the number of altruists, it is not clear whether the relative costs decrease because the impact of altruism should also increase with the increased expression of altruism. Therefore, relative sending costs that increase (rather than decrease) with r represent an alternative scenario to be considered.

A second assumption in our model is that pro-social impacts influence fertility and thereby all fitness differences arising from the quality trait and the sending costs. However, this assumption may not hold if fitness differences concern males that direct altruistic acts only towards females, or if fitness differences concern viability and the social impact improves immediate fertility. Indeed, if the model were changed such that the quality trait locus q , the sending cost, and the altruistic impacts all concern viability (instead of fertility), we expect that increasing the *quality-unbiased social impact* will magnify differences in relative fitness (between high-quality non-senders, high-quality senders, low-quality non-senders, and low-quality senders). However, further simulations are needed to verify this.

A third assumption in our model is that costs of choosiness are ignored. To evaluate its importance we provide an extended version of the model in Appendix 1-2. The cost of choosiness is incorporated as a restriction on the number of potential mating partners that an individual may encounter, and if the preferred partner type is not among them, then the egg is not fertilized and dies. Using this representation of the costs of choosiness simulations revealed that such a cost could prevent the evolution of preference for the type that is initially rare (e.g., for partner sample sizes that are small enough no equilibria with sender dominance emerge from initial state *Rand*). Other than this effect, the simulations did not reveal any major deviations from the conclusions of the original version presented in this article. Note that the cost of choosiness constitutes a risk that depends on the fraction of altruists in the population, yet is unaffected by the social impact of the altruists. The cost of choosiness thus represents an example of a fitness element that is not influenced by the social impact, as discussed in the preceding paragraph.

Apart from the assumptions also some of the predictions need scrutiny. We modeled altruism as a handicap trait because altruists may favor their competitors, but one may ask whether the altruism that evolved in our simulations represents a genuine handicap. The idea of altruism evolving as a handicap trait is that only carriers of the high-quality allele q_h signal their high fitness by acting altruistically. In that case, given a preference for altruists, more high-quality partners would be acquired. Under *quality-unbiased social impact*, the fitness superiority due to the high-quality allele q_h is undermined, thereby reducing the advantage and attractiveness of high-quality partners, as well as the incentive for acting altruistically. However, under *quality-biased*

social impact, fitness superiority due to the high-quality allele q_h is maintained or even increased. This indeed seems to point at the evolution of altruism as a genuine handicap, but there is a pitfall here. In equilibria with sender dominance, nearly all low-quality individuals act altruistically. This is in conflict with the idea of a handicap trait and altruistic behavior is not an honest signal of quality (fitness) at these equilibria. Honesty – represented by the honest signaler genotype $\{p_h, p_l\} = \{+, -\}$ in the model – temporarily dominates as behavior in the course of evolution from the initial state to an equilibrium with sender dominance (Fig. 1-3a). Due to the increasing attractiveness of senders (altruists) during these evolutionary trajectories (because a preference for senders successfully invades), the low-quality individuals eventually fare better by incurring the sending costs (losing eggs), resulting in honesty as a transient phenomenon that had faded by the time the equilibrium was reached.

Whereas handicaps usually serve to signal superiority to others, altruism as a sexually selected handicap may not only signal this, but also provide a benefit to others, including males that are competitors. By taking these effects on others into account, our model makes the following two predictions. First, if all individuals benefit equally, such altruism does not easily evolve and, if it does, the prevalence of high-quality individuals goes down. Second, if high-quality recipients benefit relatively more, then altruism can evolve more readily by sexual selection.

While there is evidence that in humans females prefer altruistic males (Griskevicius et al. 2007; Philips et al. 2008; Barclay 2010), there is as yet no empirical evidence to test the above two predictions. However, some relevant insight comes from organisational psychology, a field of empirical research on how individuals behave towards others in the network of a hierarchical organization. It appears to be a general outcome that humans frequently flatter superiors and work harder for them whereas they ignore or even kick inferiors (Vonk 1998a,b; Tal-Or 2010; although, ‘alms giving’ [i.e., donating to the poor] is probably the epitome of genuine altruism). In our model there is no social hierarchy, but only differences in quality of mating partners. Nevertheless, we propose that selective altruistic behavior is an efficient way to make good use of the differences in quality among the recipients. Our model suggests such selective altruistic behavior to evolve by sexual selection. Whether women also prefer men pleasing their superiors more than others, however, remains to be seen and requires the scrutiny from empiricists in the behavioral and social sciences. If anything, our model yields the conditions for altruism to evolve by sexual selection and, in particular, highlight the importance of analyzing the preferences of altruists, thereby generating questions such as: do individuals differ in their benefit from altruism and who benefits more than others? How fit are recipients relative to others? Do females prefer males directing their altruism towards the more fit recipients?

Acknowledgements We thank Susanne Schindler (Imperial College, Silwood Park, UK) for comments on an earlier version of the manuscript.

Appendix 1-1

Formal representation of the model

We model the evolution of four unlinked haploid loci that evolve in discrete non-overlapping generations in an infinite population of hermaphrodites. The set of genotypes defined by the four loci is finite as each locus has a finite number of alleles and Φ represents this set. By ϕ_i , we denote the frequency of genotype i in the focal generation. We refer to the egg-donating parent as mother and to the sperm-donating parent as father. Being a hermaphrodite, an individual is the mother of part of its offspring and the father of the other part. Individuals of genotype i lay ω_i eggs (i.e., the offspring that reproduce in the next generation) and $\bar{\omega} = \sum_{i \in \Phi} \phi_i \omega_i$ is the average number of eggs.

For each egg, the mother selects a father and the opportunity to fertilize an egg is never rejected. For the (genetically determined) fraction a_m of eggs, the mothers with genotype m decide on which male is going to be the father according to a preference (for details see model section) and for the other eggs, these mothers select the father for their offspring randomly from the population (random mating). The preference is genetically determined for either of the two morphs of the trait ‘sender’. The probability that a father with the preferred morph of genotype m carries genotype f is given by $\sigma_{m,f}$. The probability that an offspring of a mother with genotype m and of a father with genotype f carries genotype i is given by $\tau_{m,f,i}$. The frequency of genotype i in the next generation, ϕ'_i , is given by a summation over maternal (m) and paternal (f) genotypes:

$$\phi'_i = \frac{1}{\bar{\omega}} \sum_{m,f \in \Phi} \phi_m \omega_m (a_m \sigma_{m,f} + (1 - a_m) \phi_f) \tau_{m,f,i}$$

The preference for a mating partner is genetically determined whereby certain genotypes determine a preference for the morph sender and the other genotypes determine a preference for the other morph non-sender. The set of genotypes expressing the sender morph denoted by Φ_S and the set of genotypes expressing the non-sender morph is denoted by Φ_{n-S} . The frequency of senders is $s = \sum_{i \in \Phi_S} \phi_i$. Whenever a preference for

senders is expressed, then the father has genotype g with probability $\sigma_{j,g} = \phi_g s^{-1}$ if $g \in \Phi_S$ and with probability $\sigma_{j,g} = 0$ if $g \in \Phi_{n-S}$. For the expression of other preference, these probabilities are defined by analogy as $\sigma_{j,g} = \phi_g (1 - s)^{-1}$ if $g \in \Phi_{n-S}$ and $\sigma_{j,g} = 0$ if $g \in \Phi_S$.

Each locus is subject to mutation with a locus-dependent mutation rate and each allele mutates into any of the other alleles of the locus with a fixed probability. The fraction, $\tau_{j,g,i}$, of offspring with genotype i from a mother with genotype j and a father with genotype g is consequently fixed. As the four loci are unlinked, an offspring inherits each allele with equal probability either from the mother or from the father.

Appendix 1-2

Costs of choosiness

In an extended version of the model, we restrict individuals expressing a preference to select mating partners from a random sample of n individuals. If this sample contains individuals with the preferred type, then one of these individuals is randomly selected as a partner. If this sample contains no such individual, a cost of choosiness arises because the egg is not fertilized and ultimately dies. Egg mortality occurs at a probability $(1 - s)^n$ when the preferred type is sender and at $(1 - [1 - s])^n = s^n$ when the preferred type is non-sender. Due to the cost of choosiness, the number of (viable) eggs generated by an individual with genotype i is therefore given by

$$\omega_i = (w_i + \kappa r c s) (1 - a_i [1 - y]^n),$$

where w_i is the number of eggs produced by individuals with genotype i , $\kappa r c s$ is the number of additional eggs produced due to the social impact (r and c as in main text and κ equals k or 1 to represent the quality bias of the social impact), a_i is the fraction of eggs for which individuals with genotype i express the preference, and y is the frequency of the preferred type. Note that the risk of losing an egg due to choosiness decreases with increasing n . For $n \Rightarrow \infty$, this risk vanishes (as $1 > s > 0$) and we retrieve the model version considered in the main text.

We repeated the simulations of Fig. 1-2 using $n = 10$. In these simulations, equilibria with sender dominance ($s \sim 1$) are obtained from initial state *Send* and equilibria with non-sender dominance c are obtained from initial states *Rand* and *non-Send* (Fig. 1-A1). In state *Send*, non-senders are rare and in the latter two states, senders are rare. Our cost of choosiness thus prevents the evolution of a preference for the (initially) rare type in the simulations as is expected because this cost increases with rarity of the preferred type. Furthermore, equilibria ‘without a dominant morph’ ($s \sim 0.5$) (as in Fig. 1-2c,d) do not emerge in the simulations with $n = 10$. Because the cost of choosiness, either $(1 - s)^n$ or s^n , is unaffected by the social impact of altruism, preferring the rare type remains too costly and the initially dominant type remains dominant independent of the social impact (compare Fig. 1-A1 with Fig. 1-2). We found qualitative differences in outcome when costs of choosiness were taken into account: equilibria with non-sender dominance do not occur from initial state *Send*, equilibria with sender dominance do not occur from initial state *Rand* and equilibria without a dominant morph do not occur from all initial states. Hence, the initial composition of the population still matters to the outcome, but are independent of social impact and ensure a dominant morph. These differences are a consequence of the fact that preferring the rare type is too costly and therefore evolution of this preference is prevented. Since these effects are straightforward consequences of the introduced cost of choosiness, the extended model does not add much to the other insights gleaned from the model discussed in the main text.

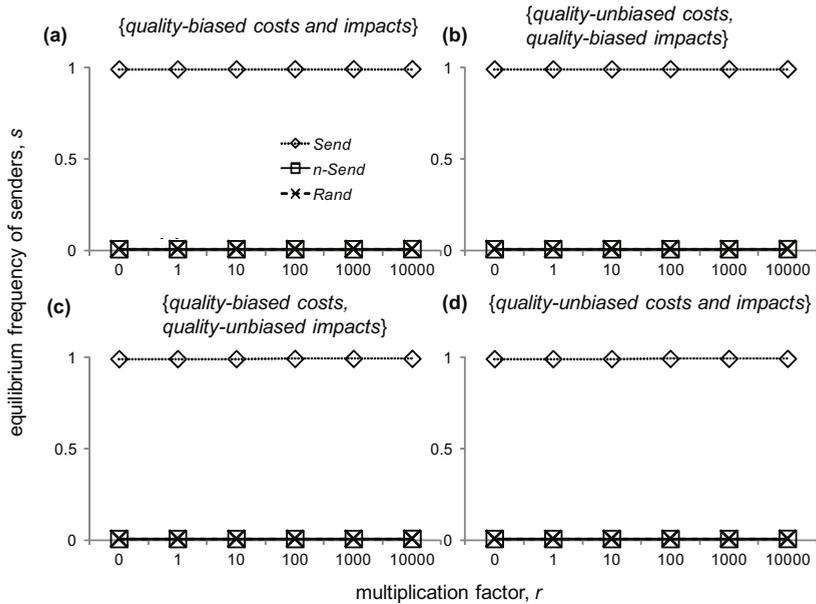


Fig. 1-A1 Equilibrium fractions of senders, s , as a function of the multiplication factor, r , from simulations for the four cost/impact scenarios (corresponding to four panels) using the settings from Fig. 1-2 but adding a cost of choosiness ($n = 10$). (a) costs of sending and social impacts are resource (i.e., the cost is higher for high-quality senders and high-quality individuals benefit more from the altruistic impact as high-quality individuals convert the resource into more eggs) in scenario {*quality-biased cost, quality-biased impact*}, (b) costs are eggs (i.e., costs are the same for all senders) and impacts are resource in scenario {*quality-unbiased cost, quality-biased impact*}, (c) costs are resource and impacts are eggs (i.e., all individuals benefit the same from these impacts) in scenario {*quality-biased cost, quality-unbiased impact*}, and (d) costs and impacts are eggs in scenario {*quality-unbiased cost, quality-unbiased impact*}. The s -values of the simulations starting from initial states *Send*, *Rand*, and *n-Send* are indicated with diamond (dotted lines), cross (continuous lines), and square symbols (dashed lines); symbols (and lines) overlap whenever the same equilibrium is attained (whenever the same equilibria are attained between two r -values).

References

- Barclay P (2010) Altruism as a courtship display: Some effects of third-party generosity on audience perceptions. *British Journal of Psychology* 101:123-135
- Clutton-Brock T (2009) Cooperation between non-kin in animal Societies. *Nature* 462:51-57
- Doutrelant C, Covas R (2007) Helping has signalling characteristics in a cooperatively breeding bird. *Animal Behaviour* 74:739-747
- Driscoll C (2006) The Bowerbirds and the Bees: Miller on Art, Altruism, and Sexual Selection. *Philosophical Psychology* 19:507-526
- Getty T (2006) Sexually selected signals are not similar to sports handicaps. *TREE* 21:83-88
- Gintis H, Smith EA, Bowles S (2001) Costly signaling and cooperation. *Journal of Theoretical Biology* 213:103-119
- Grafen A (1990a) Biological signals as handicaps. *Journal of Theoretical Biology* 144:517-546.
- Grafen A (1990b) Sexual selection unhandicapped by the Fisher process. *Journal of Theoretical Biology* 144:473-516
- Griskevicius V, Tybur JM, Sundie JM, Cialdini RB, Miller GF, Kenrick DT (2007) Blatant benevolence and conspicuous consumption: when romantic motives elicit strategic costly signals. *Journal of Personality and Social Psychology* 93:85-102

- Curven M (2004) To Give and to Give Not: The behavioral ecology of human food transfers. *Behavioral and Brain Sciences* 27:543-583
- Houlé D, Kondrashov AS (2001) Coevolution of costly mate choice and condition-dependent display of good genes. *Proceedings of Royal Society of London B* 269:97-104
- Kirkpatrick M, Ryan MJ (1991) The evolution of mating preferences and the paradox of the lek. *Nature* 350: 33-38
- Kokko H (2007) *Modelling for field biologists*. Cambridge University Press. Cambridge, UK
- Kokko H, Jennions MD, Brooks R (2006) Unifying and testing models of sexual selection. *Annual Review of Ecology, Evolution, and Systematics* 37:43-66
- Lehmann L, Keller L (2006) The evolution of cooperation and altruism: a general framework and classification of models. *Journal of Evolutionary Biology* 19:1365-1378
- Lotem A, Fishman MA, Stone L (2003) From reciprocity to unconditional altruism through signalling benefits. *Proceeding of the Royal Society London B* 270:199-205
- Miller GF (2000) *The mating mind*. Heinemann, London
- Miller GF (2007) Sexual selection for moral virtues. *Quarterly Review of Biology* 82:97-125
- Nowak MA (2006) Five rules for the evolution of cooperation. *Science* 314:1560-1565
- Pen I, Weissing FJ (2000) Sexual selection and the sex ratio: An ESS analysis. *Selection* 1:59-69
- Phillips T, Barnard C, Ferguson E, Reader T (2008) Do humans prefer altruistic mates? Testing a link between sexual selection and altruism towards non-relatives. *British Journal of Psychology* 99:555-572
- Scheid C, Schmidt J, Noe R (2008) Distinct patterns of food offering and co-feeding in rooks. *Animal Behaviour* 76:1701-1707
- Tal-Or N (2010) Indirect ingratiation: Pleasing people by associating them with successful others and by praising their associates. *Human Communication Research* 36:163-189
- Tessman I (1995) Human Altruism as a Courtship Display. *Oikos* 74:157-158
- Vonk R. (1998) Effects of Cooperative and Competitive Outcome Dependency on Attention and Impression Preferences. *Journal of Experimental Social Psychology* 34:265-288
- Vonk R. (1998) The slime effect: Suspicion and dislike of likeable behavior towards superiors. *Journal of Personality and Social Psychology* 74:849-864
- Walker T. (2008) Could sexual selection have made us psychological altruists? *Studies in History and Philosophy of Biological and Biomedical Sciences* 39:153-162
- West SA, Griffin AS, Gardner A (2007) Evolutionary explanations for cooperation. *Current Biology* 17:R661-R672
- West SA, Griffin AS, Gardner A (2007) Social semantics: altruism, cooperation, mutualism, strong reciprocity and group selection. *Journal of Evolutionary Biology* 20:415-432
- Wright J, Berg E, de Kort SR, Khazin V, Maklakov AA (2001) Cooperative sentinel behaviour in the Arabian babbler. *Animal Behaviour* 62:973-979
- Zahavi A (1977) Reliability in communication systems and the evolution of altruism. In B Stonehouse & C Perrins (Eds.). *Evolutionary ecology* (pp. 253–260). MacMillan Press, London
- Zahavi A (1995) Altruism as a handicap – the limitations of kin selection and reciprocity. *Journal of Avian Biology* 26:1-3
- Zahavi A, Zahavi A (1997) *The handicap principle*. Oxford University Press, New York