Abstract

Background: The evolutionary origin of strong altruism (where the altruist pays an absolute cost in terms of fitness) towards non-kin has never been satisfactorily explained since no mechanism (except genetic drift) seems to be able to overcome the fitness disadvantage of the individual who practiced altruism in the first place.

Results: Here we consider a multilocus, single-generation random group model and demonstrate that with low, but realistic levels of recombination and social heterosis (selecting for allelic diversity within groups) altruism can evolve without invoking kin selection, because sampling effects in the formation of temporary groups and selection for complementary haplotypes generate nonrandom associations between alleles at polymorphic loci.

Conclusion: By letting altruism get off the ground, selection on other genes favourably interferes with the eventual fate of the altruistic trait due to genetic hitchhiking.

Background

More than thirty years ago Hamilton [1] and Wilson [2] independently discussed the evolution of altruism – cooperative behaviours that decrease the fitness of the individuals that perform them—assuming a structured population divided into small temporary groups; a new view of group selection termed ‘trait-group’ or ‘structured-deme’ models [2,3]. A single heritable trait (allele A) stimulates its bearer to provide a group benefit, and its eventual spread is conditional on the greater productivity of altruistic groups. Depending on whether or not the recipients of the group benefit include the actor itself, the effects were named ‘whole-group’ or ‘other-only’ trait, respectively [4]. Whereas other-only traits are clearly altruistic because involve an absolute cost to the actor, whole-group behaviours generate confusion in the definition of altruism as they may be mutually beneficial to both the actor and the recipients [4,5]. Wilson [3,6] had previously coined the terms ‘strong altruism’ for those traits involving an absolute cost to the actor (i.e. other-only traits count always as strongly altruistic), and ‘weak altruism’ when the cost to the actor is only relative.

Contrarily to weakly altruistic traits which can increase in frequency when groups are randomly formed each generation [7], the evolution of strong altruism requires positive assortment for the benefits of altruism to fall preferentially on other altruists [1,6,8,9]. This can be


understood in terms of Hamilton's rule [10] of kin selection which states that the condition for an altruistic trait to increase in the next generation is

\[ rb > c \]  

(1)

where \( r \) is the coefficient of relatedness between actor and recipient, \( b \) is the fitness benefit (offspring gain) provided to the recipient, and \( c \) is the reproductive cost to the actor for providing benefits. Under random assortment other group members are a random sample of the global population minus the actor, and relatedness in this case is [4,11,12]

\[ r^o = \frac{-1}{N-1} \]  

(2)

(superscript \( o \) stands for other-only relatedness, and \( N \) is total population size), which makes it clear that condition (1) cannot be satisfied because kin selection requires a sufficiently high degree of relatedness (\( r > 0 \)) for altruism to evolve. The conclusion that strong altruism does not progress in this model has been recently challenged [13], but the authors assumed multigenerational groups that were initially randomly formed, which just lag the necessary positive assortment among altruists to the next generations. If, however, we stick to Hamilton's [1] original structured-deme model of single-generation, randomly formed groups it still remains undisputed as it has ever been since his formal proof that strong altruism cannot evolve.

No special process has to be invoked to create a structured-deme population as it can simply arise from random distributions of genotypes across patchy resources; a quite common process in many animals, particularly insects. Genetic variation among finite groups creates environmental heterogeneity unrelated to resource heterogeneity [14,15], and there are numerous empirical examples (see especially Table 2 in [16]) showing that there is a positive association between productivity (offspring number) and the levels of genetic variation within a resource. Thus, competition is known to be stronger in 'pure cultures' when compared with genetically diverse groups; and various behaviours may provide a whole-group effect as, e.g., access to resources that are unavailable to solitary individuals, or increased protection to parasites or predators. A recent elaboration on this old theme shows that allelic diversity can be potentially maintained at many loci as it is a positive trait in and on itself [16]. Therefore, differential productivity among groups will not only depend on the frequency of cooperators in a group but also on the level of genetic variation at other loci. Predictions from single-gene theory can be very misleading if it is assumed that in Hamilton's [1] original model haploid individuals are endowed with genomes that code for a range of trait-groups subject to selection, mutation, and recombination. Multilocus systems introduce additional issues such several alternative (meta)stable states [17,18] and the Hill-Robertson effect describing that linkage between loci under selection will reduce the overall effectiveness of selection in finite populations [19,20]. Structured-deme models unavoidably introduce linkage disequilibria that interfere with selection [21], but this effect has never been incorporated in the life cycle interactions that take place between a small number of individuals in a group to study the fate of altruistic traits in a multilocus context.

In this work, we focus on the evolution of altruistic versus selfish alleles and of polymorphisms that show a positive association between productivity and within-group genetic variation to study the fate of altruism in a multilocus context assuming the structured-deme model of single-generation, randomly formed groups originally used by Hamilton [1]. The fate of altruism will be studied by means of computer simulations and mathematical analyses of a relatively simple case. We show that although the altruistic trait is being selected against, when it is genetically linked to productivity enhancing loci altruism can invade. It should be stressed from the very beginning that the model is not producing any assortment mechanism and that it has nothing to do with kin recognition: what happens is that there is a synergy between hitchhiking and group selection that keeps allelic diversity.

**Methods and Results**

**Multilocus model and computer simulations**

Figure 1 illustrates the 'mating pool' mode of reproduction on which our model is based. A haploid finite population of size \( N = m \times n \) is randomly subdivided into \( m \) groups with \( n \) individuals per group. Offspring genotypes at each generation are sampled from a common population and randomly subdivided into temporary groups where individuals representing a finite sample from the pooled distribution reproduce proportional to their fitness. Productivity enhancing (offspring number) genes when in genetically diverse groups coded by \( g = 1 \), \( U \), \( G \) loci were stored as lists on a single chromosome. The haploid population was initially polymorphic for \( i = 3 \) alleles at equal frequencies. With random grouping the distributions of the different compositions of groups for each of the \( i \) alleles will be binomial with parameters \((p_i, n)\), where \( n \) is the group size. Assuming that allele 1 is the fittest one, within-group selection at the \( g \)th locus was modelled as...
where $y$ is a constant for the proportional increase of group's fitness as a function of allele diversity $a$ (i.e. number of alleles in a group); and $z$ models the benefits of allele diversity upon fitness. Therefore, without genetic diversity increasing group's fitness directional selection drives allele 1 to fixation whenever $f_s$ or $f_d < 1$. We set $y = 0.5$, $z = 1$, and $f_s = f_d = 0.95$. Allelic diversity can easily persist in this model under a wide range of parameter values [16,21], and there are up to $3^G$ possible segregating haplotypes.

After an initial period of 500 generations to allow the population to reach a quasi-equilibrium state under selection and free recombination between adjacent loci, the focal strongly altruistic locus was introduced in the centre of the set of those whole-group beneficial loci with alleles sampled from a binomial distribution with $p = 0.05$ and $q = 0.95$ for the $A$- and $S$-types, respectively. Selection at the...
With order to add of altruism is In other words, the critical for altruism to evolve is [1] mutation was introduced whenever a locus increases in the whole-group beneficial loci [21], and with multiple loci the probability of allele fixation is 0% of altruistic members in a group of size n, and k is the units of fitness given by an A-type in order to add K units to the joint fitness of his (n - 1) companions. The degree of positive assortment (F) required for altruism to evolve is [1] \[
\frac{K}{k} = \frac{1}{F}.
\] (5)

In other words, the critical c/b ratio for positive selection of altruism is k/K.

With N = 240 and n = 4 as assumed in most simulations (but see below) only 60 groups are formed, and the hypergeometric probability (i.e., sampling is without replacement) of randomly forming homogeneous groups of altruists with initial p = 0.05 is 3.67 × 10⁻⁶. Random group formation events will result in relatedness (r²) values which fluctuate around −1/(N - 1) for the altruistic locus. With k = 0.1 and K = 0.2 as assumed here strong altruism is expected to be lost a few generations after its introduction into the population since homogeneous groups of altruists will be surely absent.

With multiple loci the probability of allele fixation increases in the whole-group beneficial loci [21], and mutation was introduced whenever a locus g was fixed for one allele. This is, however, a relatively minor problem with less than G = 40 loci or so [21], and the only point to introducing mutation was to keep constant the 'effective' number of segregating G loci in the population.

We simulated repeated introductions of the A allele into the population whenever the S-type reached fixation. The lists of loci could undergo a recombination process following a stochastic multilocus method [22] with recombination frequency between adjacent loci denoted by R (no interference was assumed). In each generation, the sequence of events was mating pool formation, mutation, selection, recombination, and subdivision. We assumed multiplicativity of selective effects. Roulette selection operator, in which the chance of a chromosome getting selected is proportional to its fitness, was used. Selected chromosomes were randomly paired for recombination and randomly assigned to a group. Up to seven computer-simulation trials with different random number seeds were run for each combination of parameter values, and each trial was run for up to 50,000 generations. By following the fate of the A-type variant introduced repeatedly into the population, until it is fixed or lost, we can estimate the mean times to fixation or loss, respectively. The simulations were implemented in MATLAB version 7.2 [23]. An analytical treatment of the model in a simplified situation with group size n = 2 and no recombination is given after the numerical results.

**Numerical results**

In order to numerically demonstrate how strong altruism can evolve in single-generation, randomly formed groups, consider a focal locus with two alleles (A: altruistic; S: selfish types) embedded in a multilocus genome containing g = 1, U, G selected loci stored as lists on a single chromosome with whole-group beneficial traits in quasi-equilibrium state under selection (see above). Group members interact for one generation and because of behaviours individuals perform during such single-generation associations, group members affect each other fitness and may also enjoy certain individual-level benefits expressed by groups composed of genetically different individuals before the population is pooled back again and randomly forms new groups in the next generation. We have chosen this model primarily because it explicitly incorporates the key characteristics of the Hill-Robertson effect – random sampling and selection at multiple (independent) loci. Here we explore the extent to which recombination influences the evolution of an altruistic allele surrounded by other loci subject to selection.

The population size was fixed to N = 240 individuals. This introduces high stochasticity (but see below for the effect of both population size and group size), but parameters in the c/b ratio were chosen as to make it very unlikely that the A-type could be fixed by genetic drift in the single locus case (ultimate loss of the altruistic allele was constantly observed after > 2.5 × 10⁵ introductions into the population with n = 4). An initial survey of a range of number of loci and values for the proportional increase of group’s fitness as a function of allele diversity at locus g was made, and here we present summary results with group size n = 4; G = 16, 32, 48, 64 loci. As expected, the numerical outcomes show that the mean persistence time of the altruistic A-type is influenced by recombination between adjacent linked loci (Figure 2a). When embedded in a chromosome, or chromosome fragment, with tight linkage R ≤ 0.001 (notice that an R value of 10⁻³ between adjacent loci would correspond to a region approximately (G + 1)× 100 × 10⁻³ centimorgans in length and roughly equivalent to a chromosome with 1,000 genes and total map distance 100 centimorgans), the altruistic allele could easily persist in the population for quite a long time because the genome eventually crystallized in a few segregating haplotypes. This effect was
Figure 2 (see legend on next page)
already clear with only \(G = 8\) loci, a situation that allows tracking the haplotype dynamics and the eventual trapping of the A-type by complementary segregating haplotypes (Figure 2b).

The results can be understood as follows. Deterministic haplotype dynamics assuming linkage equilibrium at generation \(t_0\) guarantees that any A-type mutant introduced into the population will be damned to eventual loss no matter how many G loci are segregating in the population (see analytical treatment below). Variance in linkage disequilibrium is generated directly by random drift, and the A-type can be kept in the population when statistically associated (hitchhike) with haplotypes that remain segregating at quasi-equilibrium state. Stochasticity in this model is obviously a function of haplotype space that a given population size can explore. Since there are up to \(3^G\) possible segregating haplotypes for the G loci, no real population will eventually behave according to the deterministic dynamics and hitchhiking effects related to the action of social heterosis may prevent the altruistic allele from going quickly extinct. With low recombination neighbouring genes tend to be inherited together, and those three segregating haplotypes in the sampled simulation (Figure 2b) can be thought of as alleles of a whole-group effect supergene, with all group members experiencing a net benefit from genetic diversity. Relatedness in this case is [13] \(r^w = 1/n\) (superscript \(w\) stands for whole-group relatedness) and is obviously positive. This does not, however, invalidate our claim that strong altruism can invade without kin selection because relatedness at the focal locus still remains \(r^w = 1/(N-1)\), so there is no kin selection effect involved. The role of group size \(n\) can also be easily visualized. A large group size will tend to have low across-group variance in genetic diversity, which eventually drives haplotype [1211S2112] in Figure 2b to fixation since we have assumed that at each gth locus allele 1 is the fittest one (eq. 3).

Recombination breaks down the statistical associations between alleles at linked sites (i.e. detaches \(r^w\) from \(r^w\)) but, as we might expect, the hitchhiking effect is most marked when there are many segregating loci. With intermediate linkage (\(R = 0.005\)) fixation of strong altruism was a sporadic outcome for \(G = 32\) and \(G = 48\) loci, and happened with a relatively high probability for \(G = 64\) loci (Figure 3). In these cases it was computationally unfeasible to keep track of all segregating haplotypes in the population, but the results clearly suggest that the eventually successful A-allele was locked around a haplotype region that went to fixation or, alternatively, to various regions that happened to reach a high enough frequency (see below). Finally, swift loss of the A-type was always the final outcome with \(R \geq 0.10\).

**Analytical dynamics of whole-group genes**

In order to understand the genetic hitchhiking effect responsible for the spread of strong altruism in our group selection model, it is important to first understand the dynamics of whole-group genes since linkage disequilibria can also be generated by deterministic forces. To clarify matters, we deal here with the simplest situation where the \(g = 1\), \(\cup\), G loci with whole-group beneficial traits have two alleles each (e.g., \(1^1\) and \(2^1\); \(1^2\) and \(2^2\) for a 2-locus genome). For each pair, allele 1 is the fittest one as assumed in Methods. This creates up to \(2^G\) possible haplotypes. We set group size to \(n = 2\) and assume no recombination (\(R = 0\)). It is then possible to easily follow the dynamics of all segregating haplotypes in the population through time. Assuming that the benefits of allele diversity upon fitness are linear (\(z = 1\) in eq. 3), the payoff matrix for the gth locus is

\[
P_g = \begin{pmatrix}
  1^g & 2^g \\
  w_{11} & 1 + y \\
  w_{21} & w_{22} \\
\end{pmatrix}
\]

(6)
where element $w_{ij}$ is the fitness of individual $i$ (1g, 2g) in a group with individual $j$. With $G$ loci and multiplicativity of selective effects the payoff matrix is simply

$$\Gamma = P_1 \otimes P_2 \otimes \cdots \otimes P_G$$

(7)

where $\otimes$ is the Kronecker tensor product. For $G = 2$ this gives the matrix

$$\begin{pmatrix}
1^{112} & 1^{122} & 2^{112} & 2^{122} \\
1^{112} & 1 & (1+y) & (1+y) \times (1+y)^2 \\
1^{122} & f_d(1+y) & f_s & f_d(1+y)^2 & f_s(1+y) \\
2^{112} & f_d(1+y) & f_d(1+y)^2 & f_s & f_s(1+y) \\
2^{122} & f_d(1+y)^2 & f_s & f_d(1+y) & f_s^2 \\
\end{pmatrix}$$

(8)

It is clear that group mean is

$$\Psi = \frac{1}{2}(\Gamma + \Gamma^T)$$

(9)

where superscript $^T$ signifies matrix transposition. Now we have to generate the appropriate matrix of haplotypes, which can be done as follows. At generation $t_0$ segregating alleles have frequencies $p_0^g$ and $1 - p_0^g$ for locus $g$, and linkage disequilibrium is absent ($D = 0$). Assume for simplicity $G = 2$ loci; then the $4 \times 4$ haplotype matrix is obtained in two steps. First, we multiply the allele frequency vectors to obtain the haplotype frequencies

$$h = \left[ p_0^1 (1 - p_0^1) \right] \otimes \left[ p_0^2 (1 - p_0^2) \right]$$

(10)

and second, we multiply the resulting $h$ vector

$$v = h \otimes h$$

(11)

This row vector can now be rearranged by noting that its first 2$^G$ elements are the corresponding random group frequencies of haplotype $1^{112}$ in a group with haplotype $1^{112}, 1^{212};$ the second 2$^G$ elements the corresponding frequencies of haplotype $1^{122}$ in a group with haplotype $1^{112}, 2^{122};$ etc. After rearranging this vector we obtain

Figure 3
Fixation of strong altruism in Hamilton's random group model. With $G = 64$ loci and $R = 0.005$ the probability of fixation of the strongly altruistic $A$-type estimated after 8,652 independent introductions was $5.78 \times 10^{-4}$. The plot shows a sample simulation where the $A$-type reached fixation at generation 8,088 after 330 prior unsuccessful introductions into the population. Time to fixation was 241 generations.
the suitable matrix of haplotypes (H). Since \( R = 0 \), the
recurrence relations for the haplotype frequencies are

\[
\bar{w}_i = \sum_j H_{i,j} \Gamma_{i,j} \Psi_{i,j} \tag{12}
\]

where

\[
\bar{w} = \sum_{i,j} H_{i,j} \Gamma_{i,j} \Psi_{i,j}
\]

is the average fitness, \( h_i \) is the corresponding row for the
ith haplotype (individual), and \( \cdot \Gamma \cdot \cdot \cdot \) denotes element-by-
element multiplication of the corresponding \( i, j \) elements in
the three matrices (also known as a Hadamard product).

Letting \( f_s = f_d = 0.95 \) in payoff matrix (6), Figure 4 shows
the haplotype dynamics for different values of the propor-
tional increase of group's fitness \( y \) as a function of allele
diversity.

It is clear that with the assumed starting conditions the
deterministic short-term evolution drives the population
to end up with only coupling haplotypes at equilibrium state. We can also define ESS (Evolutionary Stable Strategy)
conditions for the payoff matrix (8) by noting that the
game theoretical model is isomorphic to a trait group
model with group size \( n = 2 \). Inspection of (8), by appli-
cation of standard game theory [24] and dynamics [25],
reveals that there are only two ESSs, corresponding to
couple coupling \{11^12, 2^12^2\} and repulsion \{1^12^2,
2^11^2\} haplotypes. All other equilibria are unstable. This
holds if \( f_s < f_d \gamma < 1; \gamma > (1 - f_d) / f_s \). The domains of attraction
of the two ESSs are not equal, hence from initial linkage
equilibrium the system converges to the coupling ESS
(Figure 4). It is straightforward to show that by increasing
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The preceding analysis can explain some previous numerical
results [26]. Computer trials using bi-allelic loci with
all haplotypes initially at equal frequencies and finite
population size \( N = 120 \), with group size set to \( n = 2 \) and
parameter values \( f_s = f_d = 0.95 \) and \( y = 0.2 \), had found that
coupling haplotypes were the most common ones left in
the population assuming no recombination, but in 42.8%
of the trials with \( G = 2 \) loci, and 56.5% with \( G = 3 \) loci, the
fittest haplotype was lost from the population. There was,
however, strong selection for allelic diversity at each locus
to be maintained. This was most evident when repulsion
haplotypes were the only ones left, as they remained seg-
regating in the population at equilibrium state. Finally, it
should be stated here that by letting \( f_s = 1 \) in payoff matrix
(6) the system converges to the standard heterotic case
[17,18,27].

**Fate of the altruistic allele in the analytical dynamics**

It is straightforward to incorporate the altruistic locus in
the former analytical treatment for the whole-group
genes. The payoff matrix is now (eq. 4)

\[
P^a: \begin{bmatrix} S & A \\ S & 1 & (1 + K) \\ A & (1 - k)(1 - k + K) \end{bmatrix}
\tag{13}
\]

where \( S \) and \( A \) stand for the selfish and (strongly) altru-

"should be maintained in the context of this document or dataset"
Figure 4
Deterministic haplotype dynamics for the whole-group loci without recombination. At generation $t_0$ all segregating alleles were assumed to be at equal frequencies, and linkage disequilibrium was absent. (a), (b). Two-loci haplotypes, with $y$ values as given at the top of each panel and haplotypes as indicated. (c). Eight-loci haplotypes.
an important way. As the number of loci increases, it remains true that the resident, altruistic pair of haplotypes can be invaded by two appropriate haplotypes only; all the other selfish haplotypes are repelled.

Deterministic haplotype dynamics assuming linkage equilibrium at generation $t_0$ also shows that any $A$-type mutant introduced into the population is damned to eventual loss no matter how many $G$ loci are segregating in the population. The only possibility for the $A$-type to be kept in the population is to statistically associate itself (hitchhike) with haplotypes that remain segregating at quasi-equilibrium state.

In finite populations allelic diversity can be lost when the fittest haplotype is present since its eventual fixation can occur [26], a result which strongly depends on the number of $G$ loci initially introduced in the population and the strength of selection [21]. This can be understood by comparing Figures 4b and 4c, where the equilibrium frequency of the fittest haplotype is higher as the number of loci increase. Because in the model for whole-group genes there is also strong selection to maintain allelic diversity it is, therefore, not surprising that a mutant $A$-type could eventually hitchhike a haplotype that is complementary to those already present in the population. The interaction between linkage, selection, and sampling in structured-deme models is precisely the phenomenon that can be described by the ‘Hill-Robertson’ effect [19] or genetic hitchhiking [20].

We can now envisage the effects of group size $n$ and population size $N$ on the eventual fate of allele $A$ under a given set of conditions. Increasing group size decreases the across-group variance in allelic diversity at the $G$ loci, which in turns increases the within-group selection and reduces the likelihood of polymorphism since the fittest allele $A^k$ at the $g$th locus goes to fixation (eqs. 3 and 6). Hence, hitchhiking may not be possible and the eventual dynamics of the $A$-type converges to the single locus case. In other words, the initial spread of altruism in this model is conditional on a large enough among-group genetic variance and will not happen in genetically homogeneous settings. Simulations with $N = 240$, $G \geq 16$, and parameter values as above indicate that group size $n \geq 16$ is large enough as to dramatically reduce or even prevent the eventual trapping of the $A$-type by complementary segregating haplotypes when $R = 0$.

The effect of population size $N$ is relatively more complex since stochasticity in this model is obviously a function of the strength of selection at each locus and the haplotype space that a given population can explore. For instance, in simulations with $G = 8$ (haplotype space $2 \times 3^8 = 13,122$; including the focal altruistic locus), $n = 4$, $R = 0$ and strong selection quasi-stable polymorphism of the $A$-type was not detected when $N \geq 500$ since haplotype evolution tends to approach the deterministic situation; but was a frequent outcome with $G \geq 32$ loci even when $N = 2,000$. On the other hand, reducing the strength of selection by 60% (i.e., $y = 0.2$, $k = 0.04$ and $K = 0.08$) allowed to increase population size to $N = 500$ as to qualitatively obtain similar results for $G = 8$ as those obtained with strong selection and $N = 240$. In summary, the hitchhiking effect is robust for large population sizes as long as group size $n$ is small enough, so that among-group allelic diversity at the $G$ loci can be selectively kept.

In the simulations with $G = 64$ loci and $R = 0.005$ we detected a relatively high probability of fixation of strong altruism ($5.78 \times 10^{-4}$). Figure 5 also shows a sample simulation where a snapshot of segregating haplotypes right after fixation of the $A$-type was obtained. As expected from the analytical treatment above, it is clear that the successful $A$-allele was locked around a complementary region that reached a high enough frequency because it allowed group members to experience a net benefit from genetic diversity at the $G$ loci.

**Discussion and Conclusion**

We acknowledge that our structured-deme model assumes strong selection in the collections of individuals who influence one another’s fitness, mainly to prevent waiting times before the eventual spread of the altruistic trait to becoming computationally unmanageable. The important point, however, is to understand that linkage, selection, and sampling will necessarily interact in multilocus structured-deme models, and the interference effect will extend to all loci in a block depending on the recombination value. With tight linkage a few selected loci may be enough to trap the altruistic allele in a quasi-equilibrium polymorphic state. With moderate recombination fixation of strong altruism can occur. Our results do pose a serious challenge to the universally accepted view that kin selection is the key component to explain altruistic behaviours that impose an absolute fitness cost to the actor and, to some extent, might be relevant to understand the origin (as opposed to the maintenance) of eusociality [28,29]. No matter how stable can the long-term evolution of altruism be (i.e., resident altruistic haplotypes can always be invaded by appropriate, and only the appropriate, haplotypes carrying the selfish allele; see above), the results suggest that genetic hitchhiking is a basic ingredient for the evolution of cooperation considering the crucial problem faced by altruistic behaviours in structured-deme models: their initial establishment when rare [30]. As forcefully stressed by Field [31], we should avoid here the ‘inverse genetic fallacy’;namely, the inappropriate attribution of mechanisms that may be sustaining cooperation to the explanation of its origin. This is clearly the
case here when contrasting with the consequences that positive assortment (i.e., altruists settle with altruists) can have on the maintenance of altruism.

Positive assortment increases the relatedness at the focal altruistic locus and, depending on the linkage disequilibrium between that locus and nearby $G$ loci it also increases the relatedness at those loci. This, in turn, decreases the within-group variance and the likelihood of genetic hitchhiking since the fittest allele at the $g$th whole-group locus can become fixed more easily. We thus have a powerful mechanism to explain the evolutionary origin of altruism, but unless linkage disequilibria tend to be reduced its long-term maintenance (by kin selection) can be compromised if group-beneficial effects exerted by social heterosis and random grouping are stronger than those exerted by the focal locus under positive assortment. As long as the formation of groups remains random, there is no chance to cast our results in terms of kin selection: thus, kin selection and social heterosis are in this sense opposing mechanisms to help the initial spread of strong altruism. We are not considering the chance spread of just any
maladaptive allele; rather, the necessary synergy between hitchhiking and (strong) group selection that keeps allelic diversity.

In the same vein that multivariate selection theory provides a framework to predict the direct and indirect effects of selection on a suite of complex traits [32] and illustrates the empirical shortfalls of focusing on a single character, we hypothesize that multilocus approaches to social evolution [33] will eventually demonstrate that cooperation can evolve without assuming interactions between relatives: the condition will be that indirect effects of selection on linked loci should be greater than the direct response to selection at the focal altruistic locus. Even if Hamilton’s condition (1) applies, the effects discussed in this paper may help the initial spread of altruists.

**Authors’ contributions**
Both authors contributed equally to this manuscript. Both authors read and approved the final manuscript.

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**References**
1. Hamilton WD: Social epistemology. In Sociobiology: A treatise on the new synthesis. Edited by: Wilson DS. Cambridge, Cambridge Univ Press; 1975. 59:1-52.
2. Wilson DS: Altruistic cooperation. Proc Nat Acad Sci USA 1975, 72:355-358.
3. Wilson DS: Structured demes and trait-group variation. Am Nat 1979, 113:605-610.
4. Pepper JW: Relatedness in trait group models of social evolution. Am Nat 2000, 206:355-368.
5. West SA, Griffin AS, Gardner A: Social semantics: altruism, cooperation, mutualism, strong reciporcity and group selection. J Evol Biol 2007, 20:415-432.
6. Wilson DS: Weak altruism, strong group selection. Oikos 1990, 59:135-140.
7. Uyenoyama M, Feldman MW: Theories of kin and group selection: a population genetics perspective. Theor Pop Biol 1980, 17:380-414.
8. Grafen A: Models of r and d. Nature 1980, 284:494-495.
9. Nunney L: Group selection, altruism, and structured-deme models. Am Nat 1985, 126:212-230.
10. Hamilton WD: The genetical evolution of social behaviour, I & II. J Theor Biol 1964, 7:1-52.
11. Hamilton WD: Selweless and altruistic behaviour in some extreme models. In Man and Beast: Comparative Social Behavior Edited by: Eisenberg JF, Dillen WS. Washington, Smithsonian Press; 1971:57-91.
12. Grafen A: A geometric view of relatedness. In Oxford Surveys in Evolutionary Biology Volume 2. Edited by: Dawkins M, Ridley M. Oxford: Oxford Univ Press; 1985:28-89.
13. Fletcher JA, Zwick M: Strong altruism can evolve in randomly formed groups. J Theor Biol 2004, 228:303-313.
14. Hoffmann AA, Nielsen KM: The effect of resource subdivision on genetic variation in Drosophila. Am Nat 1985, 125:421-430.
15. Santos M: Resource subdivision and the advantage of genotypic diversity in Drosophila. Heredity 1997, 78:302-310.
16. Nason P, Kapheim KM: Social heterosis and the maintenance of genetic diversity. J Evol Biol 2007, 20:2253-2265.
17. Stabbin M: On treating the chromosome as the unit of selection. Genetics 1972, 72:157-168.
18. Yamazaki T: The effects of overdominance on linkage in a multilocus system. Genetics 1977, 86:227-236.
19. Hill WG, Robertson A: The effect of linkage on the limits to artificial selection. Genet Res 1946, 8:269-274.
20. Barton NH: Genetic hitchhiking. Phil Trans R Soc Lond 2000, 355:1553-1562.
21. Santos M: ‘Social heterosis’ as a process that maintains genetic variation — a comment. J Evol Biol 2008, 21:625-630.
22. Fraser A, Burnell D: Computer Models in Genetics New York: McGraw-Hill; 1970.
23. The MathWorks Inc: Matlab, version 7.2 The Language of Technical Computing 2005 [http://www.mathworks.com].
24. Maynard Smith J: Evolution and the Theory of Games Cambridge: Cambridge Univ Press; 1982.
25. Hofbauer J, Sigmund KE: The Theory of Evolution and Dynamical Systems Cambridge, Cambridge Univ Press; 1988.
26. Nason P, Kapheim KM: Social heterosis and the maintenance of genetic diversity at the genome level. J Evol Biol 2008, 21:631-635.
27. Franklin I, Lewontin R: Is the gene the unit of selection? Genetics 1970, 65:707-734.
28. Wilson EO, Hölldobler B: Eusociality: origin and consequences. Proc Natl Acad Sci USA 2005, 102:13367-13371.
29. Wilson DS, Wilson EO: Rethinking the theoretical foundation of sociobiology. Q Rev Biol 2007, 82:327-348.
30. Charlesworth B: A note on the evolution of altruism in structured demes. Am Nat 1979, 113:601-605.
31. Field AJ: Altruistically Inclined? The Behavioral Sciences, Evolutionary Theory, and the Origins of Reciprocity Ann Arbor, The Univ of Michigan Press; 2004.
32. Lande R: Quantitative genetic analysis of multivariate evolution, applied to brain: body size allometry. Evolution 1979, 33:402-416.
33. Gardner A, West SA, Barton NH: The relation between multilocus population genetics and social evolution. Am Nat 2007, 169:207-226.