



## Relatedness in Trait Group Models of Social Evolution

JOHN W. PEPPER\*

*Santa Fe Institute, 1399 Hyde Park Road, Santa Fe, NM 87501, U.S.A.*

*(Received on 10 January 2000, Accepted in revised form on 20 June 2000)*

Genetic relatedness is a central concept in the study of social evolution. Though originally defined in terms of genealogy, the modern version of relatedness accommodates genetic similarity of any origin. This paper examines relatedness in group structured modes, in which a trait affects the fitness of all group members. Such traits can be divided into two types, based on whether their group fitness effects encompass all group members including the actor (“whole-group traits”), or only group members other than the actor (“other-only traits”). Both trait types are common in nature as well as in theoretical models, but they have rarely been distinguished clearly. The average relatedness of recipients to actors differs for the two trait types within the same population and even the same individual, leading to different selection pressures and evolutionary outcomes. Total relatedness in group-structured models can be partitioned into two components: structural relatedness due to the size and number of groups in the population, and assortative relatedness due to the distribution of genotypes among groups. Each component differs for whole-group vs. other-only traits, both in terms of their values and the factors that influence them. Some key differences include: positive relatedness requires positive assortment for other-only but not for whole-group traits; negative relatedness is possible for other-only but not whole-group traits; relatedness depends on average group size for whole-group but not other-only traits, and non-random assortment into groups affects relatedness more strongly for other-only than whole-group traits. Recognizing the distinction between these trait types resolves some apparent contradictions in the literature, and clarifies the limits of some previous results.

© 2000 Academic Press

### Introduction

The phrase “social evolution” refers to situations in which an individual’s genotype affects the fitness of other individuals as well as itself. A classic example is an altruistic trait, which confers a cost on the actor and a benefit to other individuals. One very general tool for studying social evolution is the concept of inclusive fitness. This approach partitions fitness effects into two components: one affecting only the actor and the other affecting other individuals, with the second

component weighted by relatedness to the actor. A social trait is selectively favored when it satisfies Hamilton’s rule:  $rb - c > 0$ , where  $b$  is the fitness effect on recipients of the act,  $-c$  is the fitness effect on the actor, and  $r$  is the relatedness of the recipients to actor (Hamilton, 1964). The sign of  $r$  indicates what kind of social effects are selected for (beneficial when  $r$  is positive, detrimental when  $r$  is negative), and its magnitude indicates how important social effects are in determining what evolves.

The concept of relatedness has itself evolved over the years. It was originally conceived as the expected proportion of genes identical between

\* E-mail: [jjpepper@santafe.edu](mailto:jjpepper@santafe.edu)

two individuals due to recent common descent, as calculated from a pedigree (Hamilton, 1964). Later, Hamilton (1970, 1972) redefined relatedness in a more general way as a statistical measure of genetic similarity regardless of its source. The coefficient of relatedness ( $r$ ) was redefined as the regression slope of recipient genotype on actor genotype. Here a “recipient” is any individual whose fitness is affected by the social effects of the trait, and “genotype” is defined either as the frequency of a given allele (as in population genetics), or as the “breeding value” of a phenotypic trait (as in quantitative genetics). Instead of recent common descent, this coefficient measures the extent to which actors are either more similar to recipients than to average members of the population (positive  $r$ ), or less similar (negative  $r$ ).

The two concepts of relatedness are sometimes distinguished by using the terms “relationship” to denote the original narrower notion of common ancestry, and “relatedness” to denote statistical similarity regardless of its source (Grafen, 1985, p. 41). Under certain simplifying assumptions the two definitions coincide, but when they do not it is the modern statistical definition rather than the original genealogical version that makes inclusive fitness theory work (Hamilton, 1975; Grafen, 1991). In this paper, the term “relatedness” will mean genetic similarity as measured by the regression coefficient.

This paper addresses relatedness in the context of subdivided or group-structured populations. In particular, it compares two kinds of social traits that have each been modeled many times, but are rarely distinguished. These two trait types differ in terms of the factors that influence relatedness, the values that relatedness can take, and the kinds of social effects that can evolve, and failing to distinguish between them has led to confusion in the past.

### Relatedness in Group-structured Models

Relatedness was originally framed in terms of interactions between specific individuals, but it has been extended by Hamilton (1975) and others to contexts in which a population is divided into groups, and the trait in question affects the fitness of all members of the actor’s group. Because these

models define group membership in terms of the fitness effects of a particular trait, they are often referred to as “trait group” models (Wilson, 1975). For simplicity, it is generally assumed that all recipients receive an equal share in the trait’s “social” fitness effects. Whether a trait with a given individual cost and total group benefit is positively selected depends on the average relatedness of recipients to actors. Hereafter, the term “relatedness” will be used as shorthand for average relatedness of recipients to actors.

Depending on the nature of the trait in question, the recipients of the group fitness effect may or may not include the actor itself. In other words, a given trait may provide a benefit to all group members except the actor, or to all group members including the actor. I will refer to these two types as “other-only” and “whole-group” traits, respectively. Both situations are frequently encountered in nature (Table 1) and both have been treated many times in theoretical models (Table 2). The distinction between whole-group and other-only traits is rarely noted, but it has important implications both for how relatedness is calculated, and for how it is influenced by various factors.

### Calculating Relatedness

The earliest models of group-structured populations were whole-group models (Table 2). However, Hamilton developed the regression coefficient of relatedness in the context of other-only traits (Hamilton, 1964), and continued to use other-only traits in his models of group-structured populations (Hamilton, 1975). Some later formulations of relatedness were also first applied to other-only traits (here termed “Covariance ratios”; Table 3). At least two formulas for the relatedness coefficient were developed in the context of whole-group traits: the “Proportion of variance” formula, and the “Subjective difference” formula for single-locus two-allele haploid systems (Table 3).

Each of these formulas was originally developed in the context of one of the two trait types, but most can be applied to either trait type simply by defining recipients appropriately. The exception is the “Proportion of variance”

TABLE 1  
*Proposed empirical examples of traits with other-only and whole-group fitness effects*

Trait	Reference
<i>Whole-group traits</i>	
Predator inspection in fish	Dugatkin (1990)
Quorum sensing in bacteria	Brookfield (1998)
Virus "cheater" strains*	Turner & Chao (1999)
Improvement in food supply	
Prudent predation	Gilpin (1975), Hemptinne & Dixon (1997)
Parasite avirulence	Frank (1996a), Miralles <i>et al.</i> (1997)
Group provisioning	Rissing <i>et al.</i> (1989)
Bacterial exoenzymes	Jones <i>et al.</i> (1993)
Social alliances and coalitions	
Dolphins	Connor <i>et al.</i> (1992)
Lions	Packer & Pusey (1982)
Primates	Bradley (1999)
Social engineering	
Policing	Frank (1995b, 1996b)
Punishment	Boyd & Richerson (1992), Clutton-Brock & Parker (1995)
Within-individual conflict*†	Hurst <i>et al.</i> (1996)
"Petite" mitochondria	Albert <i>et al.</i> (1996)
Cytoplasmic sex determinants	Hurst (1993)
Segregation distorters	van Boven & Weissing (1999)
Transposons	Zeyl & Bell (1996)
<i>Other-only traits</i>	
Cannibalism*	McCauley & Wade (1980)
Infanticide*	Breden & Hausfater (1990)
Alarm calling	Sherman (1977), Hoogland (1983)
Food calling	Wilson (1977a), Evans & Marler (1994)
Food sharing	Wilkinson (1984)
Reduced lifespan	Kirchner & Roy (1999)
Sterility	
Eusocial colonies	Bourke & Franks (1995), Seeley (1995)
Cell differentiation in metazoans	Buss (1987)
Bacterial fruiting body production	Velicer <i>et al.</i> (2000)
Suicidal aid	
Brain worm	Wilson (1997b)
Honeybee sting	Seeley (1995)
Distastefulness to predators	Hamilton (1964), Edmunds (1974)
Apoptosis in yeast	Frohlich & Madeo (2000)
Bacterial anticompeteritor toxins	Chao & Levin (1981), Riley & Gordon (1992)

\* Here the label refers to the selfish rather than the cooperative form of the trait.

† Here the entities analogous to individuals are subunits of the organism, and the organism plays the role of a group.

formula, which applies only to whole-group traits. The other formulas in Table 3 can be rewritten in a general form by replacing either  $G_W$  (average genotype of all group members including actor) or  $G_O$  (average genotype of other group members) with the more general notation  $G_R$  (average genotype of recipients). To apply the generalized formula correctly one must use the appropriate definition of  $G_R$  for the trait at hand; either  $G_O$  for other-only or  $G_W$  for whole-group traits.

The relationship between  $G_O$  and  $G_W$  depends on actor genotype and group size. For whole-group traits, the average genotype of recipients includes the contribution of both self and others, weighted by their relative frequency among recipients. For an actor in a group of size  $n$ , self makes up  $1/n$  of the total set of recipients, with others making up the remaining  $(n - 1)/n$ . Thus, for whole-group traits the average genotype of recipients can be represented as the weighted

TABLE 2  
*A chronological list of some inclusive fitness models of group-structured populations based on whole-group and other-only traits*

Reference	Whole-group trait	Other-only trait	Both trait types
Haldane (1932)	X		
Wright (1945)	X		
Williams & Williams (1957)	X		
Hamilton (1964)		X	
Wright (1969; p. 127)	X		
Hamilton (1970)		X	
Eshel (1972)	X		
Boorman & Levitt (1973)	X		
Levin & Kilmer (1974)	X		
Charnov & Krebs (1975)	X		
Hamilton (1975)		X	
Wilson (1975)		X	
Cohen & Eshel (1976)	X		
Matessi & Jayakar (1976)	X		
Wilson (1977a)			X
Bell (1978)		X	
Wilson (1979a)		X	
Wilson (1980)		X	
Grafen (1984)		X	
Nunney (1985)	X		
Breden (1990)	X		
Wilson (1990)		X	
Taylor (1992)	X		
Wilson <i>et al.</i> (1992)	X		
Frank (1996b)	X		
Taylor & Frank (1996)	X		
Frank (1997)	X		
Michod (1997)	X		
Wilson & Dugatkin (1997)	X		
Day & Taylor (1998)	X		
Pepper & Smuts (2000)			X

average of the genotypes of self and others:

$$G_w = \frac{G_A + (n-1)G_O}{n}. \quad (1)$$

Because the average genotype of recipients differs for whole-group vs. other-only traits, relatedness differs for the two trait types within the same population, and even within the same individual. For whole-group traits the average relatedness of recipients to actor is an average over self and others, weighted by the frequency of each among the recipients. Relatedness to self is always 1, and we can let  $r^o$  denote the average relatedness of other group members to the actor. Then for other-only traits, average relatedness of recipients to actors is simply  $r^o$ . For whole-group

traits in a population with uniform group size  $n$ , it is

$$r^w = \frac{1 + (n-1)r^o}{n}. \quad (2)$$

When group sizes vary, the relationship between  $r^w$  and  $r^o$  is complicated by the distribution of genotypes among groups of varying size, as described below (Fig. 3). However, relatedness is always at least as high for whole-group as for other-only traits of the same individual. Rearranging eqn (2) shows that under uniform group size relatedness is greater for whole-group than other-only traits by  $(1 - r^o)/n$ , which is non-zero when  $r^o < 1$ , or whenever there is genetic variation within groups.

TABLE 3  
Some formulations for the relatedness coefficient

Description	Formula	References
Regression coefficient	$\beta(G_O, G_A)$	Hamilton (1972)
or	$\frac{Cov(G_A, G_O)}{Var(G_A)}$	
Covariance ratio A	$\frac{Cov(G_A, P_O)}{Cov(G_A, P_A)}$	Orlove & Wood (1978), Queller (1985)
Covariance ratio B	$\frac{Cov(P_A, G_O)}{Cov(G_A, P_A)}$	Michod & Hamilton (1980), Seger (1981), Queller (1985)
Proportion of variance	$\frac{Var(G_W)}{Var(G_A)}$	Breden (1990), Frank (1995a, 1997)
Subjective difference	$Ave_x(G_W) - Ave_y(G_W)$	Wilson (1977a), Wilson <i>et al.</i> (1992), Kelly (1992), Taylor (1992)

*Abbrev:*  $G_A$  = genotype of actor (defined as either allele frequency or breeding value);  $G_W$  = average genotype of actor's whole group;  $G_O$  = average genotype of other group members excluding actor;  $P_A$  = phenotype of actor;  $P_O$  = average phenotype of other group members excluding actor;  $\beta$  = regression coefficient;  $Cov$  = covariance;  $Var$  = variance;  $Ave_x$  = average across all type x actors;  $Ave_y$  = average across all type y actors.

The relationship between whole-group and other-only relatedness can be used to derive a modified "Proportion of variance" formula (Table 3) for other-only traits. If we assume uniform group size, we can substitute the "Proportion of variance" formula for  $r^w$  into eqn (2) and rearrange, yielding

$$r^o = \left( \frac{n}{n-1} * \frac{Var(G_W)}{Var(G_A)} \right) - \frac{1}{n-1}. \quad (3)$$

If group size varies, however, there is no fixed relationship between the ratio of variances and other-only relatedness. (See Fig. 3 for an example in which the variance ratio changes while other-only relatedness remains constant.)

### Population Structure as a Source of Relatedness

Relatedness in group-structured models can be influenced by many processes, including patterns of group formation and extinction, migration between groups, reproduction, offspring dispersal, and death. However, the resulting pattern of relatedness that exists at any given time can be decomposed into just two components. The first is population structure, defined as population

size and group sizes. The second is the pattern of assortment, or how genotypes are distributed among groups. It is useful to partition the coefficient of relatedness into a sum of two values corresponding to these components. Relatedness due to population structure is "structural relatedness", or  $r_s = E(r)$ , defined as the expected relatedness of recipients to actors for a given population structure under the assumption that individuals assort into groups randomly with respect to genotype.

### CALCULATING STRUCTURAL RELATEDNESS

To calculate structural relatedness one needs to know expected relatedness to both self and other group members, and the frequency of self vs. others among recipients. Relatedness to self is always one. Relatedness of the rest of the global population to an actor is  $-1/(N-1)$ , where  $N$  = global population size (Hamilton, 1971; for derivation see Appendix A). Under random assortment, other group members are a random sample of global population minus the actor, so that expected relatedness to other group members equals relatedness to the rest of the global population. Thus, structural relatedness for

other-only traits is

$$r_s^o = \frac{-1}{N-1}. \quad (4)$$

To find structural relatedness for whole-group traits we can substitute eqn (4) into eqn (2) and average across group sizes. The result can be expressed in terms of either average group size or number of groups:

$$r_s^w = \frac{N - \bar{n}}{\bar{n}(N-1)} = \frac{g-1}{N-1}, \quad (5)$$

where  $N$  is the global population size,  $\bar{n}$  the average group size, and  $g$  the number of groups in the global population (for derivation see Appendix B).

Comparing eqns (4) and (5) shows that within the same population, structural relatedness is greater for whole-group than other-only traits by  $g/(N-1)$ . Thus, relatedness is expected to differ most for the two trait types when groups are small (Fig. 2). Note that structural relatedness is completely determined by population size and average group size, and is unaffected by allele frequency or variance in group size.

#### EFFECTS OF GROUP SIZE

Equations (4) and (5) show that group size affects relatedness for whole-group but not other-only traits. For example, in an infinite population structural relatedness is zero for other-only traits but is  $1/\bar{n}$  for whole-group traits (Fig. 1; Appendix B). This is because the relative contribution of actors to the average recipient genotype is always zero for other-only traits, but varies with group size for whole-group traits, making up  $1/n$  of other group genotype, where  $n$  is the group size. As group size falls the influence of actor's genotype on average recipient genotype increases, and therefore so does the expected relatedness of recipients to actors.

#### EFFECTS OF POPULATION SIZE

Population size affects relatedness for both trait types, but in slightly different ways. In an infinite population relatedness to the rest of the population is zero. Under random assort-

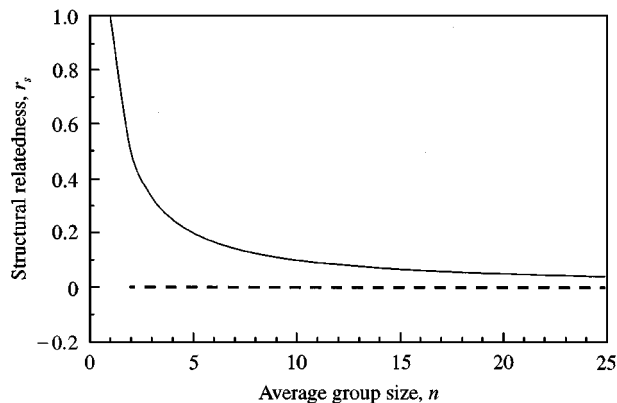


FIG. 1. Effects of group size on structural relatedness ( $r_s$ ) in infinite populations. For other-only traits  $r_s = 0$ , (---); and for whole-group traits  $r_s = 1/\bar{n}$  (—).

ment, other group members are a random sample of the global population, so that expected relatedness to the rest of the group is also zero. (That is, the actor is no more or less similar to other group members than to an average member of the global population.) In contrast, in a finite population the average genotype of the rest of the population depends on the actor's genotype. Any deviation of actor's genotype from the global average  $G_W$  is offset by a deviation with the opposite sign by the rest of the population. In other words, the sign of  $G_A - G_W$  is opposite that of  $G_O - G_W$ . This is illustrated by rearranging eqn (1):

$$(G_A - G_W) = -(G_O - G_W)(n-1). \quad (6)$$

If we think of the global population as a single group, eqn (6) shows that actor genotype is negatively correlated with the average genotype of the rest of the global population, so that relatedness is negative. Thus, under random assortment, expected relatedness to the rest of the group is also negative.

Population size affects relatedness more strongly for other-only than for whole-group traits in the same population. This is because others make up a larger proportion of recipients for other-only than for whole-group traits (1 vs.  $(n-1)/n$ ). The effect of population size on whole-group traits diminishes as groups get smaller, vanishing at  $n=1$  because the frequency of

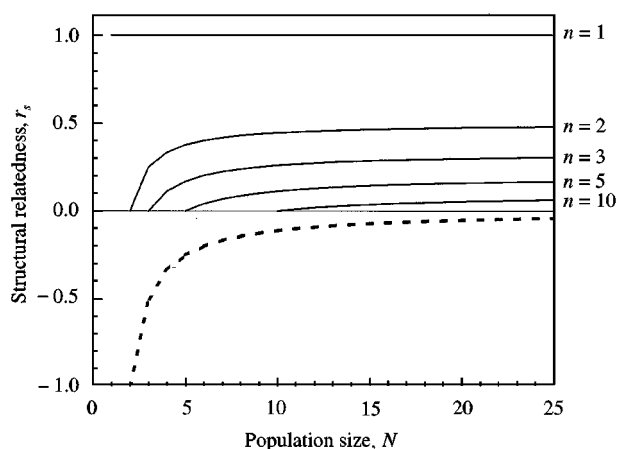


FIG. 2. Effect of population size on structural relatedness for whole-group traits (—) and other-only traits (---) for various average group sizes ( $n$ ). For equations see eqns (4) and (5). The extreme left end of each line, although included for completeness, does not actually correspond to a population divided into multiple groups in which the trait can be expressed. (Note that solitary individuals are treated differently for the two trait types. For the whole-group traits the actor is the only recipient, so that relatedness is 1. For other-only traits there is no recipient and relatedness is undefined. Thus, for other-only traits, the effective  $N$  for calculating relatedness is the number of non-solitary individuals.)

others among recipients decreases to zero as group size decreases to one (Fig. 2).

Population size has little effect on relatedness unless populations are quite small, i.e. well under 100 individuals (Fig. 2). This condition is unlikely to be met when populations include an entire species or deme. However, for Hamilton's rule to apply correctly under local competition, relatedness must be calculated relative to the local competitive neighborhood rather than the entire interbreeding population (Queller, 1994). In such cases, the relevant population size may be small enough to affect the outcome.

#### Assortment as a Source of Relatedness

Relatedness in group-structured populations depends not only on the size and number of groups, but also on how individuals are distributed among those groups—specifically whether they are distributed randomly with respect to genotype. When genetically similar individuals tend to be in the same groups the population is said to exhibit positive assortment, and when

dissimilar individuals tend to be together more often than expected, the population is said to exhibit negative assortment. [Equivalent terms include “assortation” and “dissortation” (Hamilton, 1971, 1975), and “underdispersion” and “overdispersion” (Bell, 1978)].

#### MEASURING ASSORTMENT

To measure assortment, Hamilton (1975) assumed other-only fitness effects and infinite population size, and defined assortment as the regression of recipient genotype on actor genotype. This of course is also the coefficient of relatedness. As eqn (4) shows, under these assumptions structural relatedness is zero, so that non-zero relatedness can indeed only be due to assortment. How can relatedness due to assortment be measured in a more general way that also applies to finite populations and to whole-group traits? By definition, relatedness in group-structured models is influenced only by population structure and assortment. We can therefore define relatedness due to assortment as all relatedness not accounted for by population structure:

$$r_a \equiv r - r_s, \quad (7)$$

where  $r_a$  is the relatedness due to assortment,  $r$  the total relatedness, and  $r_s$  the structural relatedness. In other words,  $r_a$  is the deviation of the relatedness coefficient from its expected value under random assortment. This measure subsumes Hamilton's (1975) measure of assortment as a special case for  $r_s = 0$ . Its expected sign is zero when assortment is random, and it is positive when group members are more similar than expected and negative when they are less similar than expected.

Under this definition, what constitutes non-random assortment actually differs for the two trait types in the same population at the same time. For other-only traits, assortment can be non-zero only if other group members are more (or less) similar to actors than expected. For whole-group traits, however, assortment can also be non-zero if self makes up a larger (or smaller) proportion of recipients than expected. This occurs if the genotype of interest tends to occur in smaller (or larger) groups than would be expected

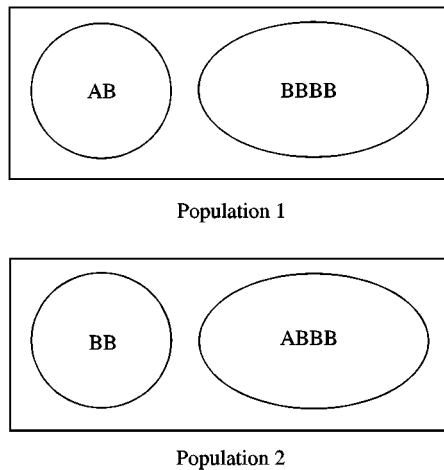


FIG. 3. An illustration of how distribution with respect to group size can contribute to assortment for whole-group but not other-only traits. Each letter represents a single-locus haploid individual of type A or B that is part of a group ( $\circ$ ) within a population ( $\square$ ). For other-only traits expected relatedness under random assortment is  $r_s^o = -0.2$  [eqn (4)]. In both populations total relatedness  $r^o = -0.2$ , and assortative relatedness is  $r_a^o = r^o - r_s^o = 0$ . This lack of assortative relatedness is expected, because there are no other A's for the single A individual to assort with non-randomly. In contrast, for a whole-group trait relatedness is higher in Population 1 ( $r^w = 0.4$ ) than in Population 2 ( $r^w = 0.1$ ). In both populations, expected relatedness is  $r_s^w = 0.2$  [eqn (5)]. Thus, for the whole-group trait assortment is positive in Population 1 ( $r_a^w = r^w - r_s^w = 0.2$ ) and negative in Population 2 ( $r_a^w = r^w - r_s^w = -0.1$ ). To express this result in terms of the "Proportion of variance" formula (Table 3), the genetic variance between groups is greater than expected under random assortment in Population 1, and less than expected in Population 2.

if distributed randomly. A bias toward smaller groups generates positive assortment, and a bias toward larger groups generates negative assortment. Thus, for whole-group but not other-only traits, even a single individual of a given type can assort either positively or negatively with itself (Fig. 3).

#### ASSORTMENT HAS A GREATER IMPACT ON OTHER-ONLY THAN WHOLE-GROUP TRAITS

Assortment affects relatedness more strongly for other-only than whole-group traits. For any given population with uniform group size  $n$ , the difference is a factor of  $(n - 1)/n$  (Appendix C). This reflects the fact that for other-only traits all recipients are subject to the effects of assortment,

while for whole-group traits size only non-self recipients are subject to the effects of assortment, and non-self individuals make up  $(n - 1)/n$  of the recipients.

Assortative relatedness also has a wider range of potential values for other-only than for whole-group traits. Consider first the range of positive assortment. Under maximal positive assortment each group consists of genetically identical individuals. Recipients are therefore identical to actors, resulting in relatedness of 1 for both trait types. Thus, for both trait types the upper bound on  $r$  is 1, and by the definition of  $r_a$ , the upper bound on  $r_a$  is  $1 - r_s$ . Because  $r_s$  is greater for whole-group than other-only traits in the same population, maximal assortative relatedness is greater for other-only than whole-group traits.

Assortative relatedness can also range lower for other-only than whole-group traits. Under extreme negative assortment each group has the same average genotype. For whole-group traits this means that recipient genotype always equals the global average genotype, so that relatedness is zero. Thus, the lower bound on  $r^w$  is 0, and the lower bound on assortative relatedness is  $r_a^w = 0 - r_s^w = -r_s^w$ . In contrast, for other-only traits structural relatedness cannot be greater than zero, so total relatedness under negative assortment is always negative. How negative it can be depends on group size. This can be seen by analogy with eqn (A.5) in Appendix A, which states that relatedness of the rest of the population to an actor is  $r = -1/(N - 1)$ , where  $N$  is the population size. Under maximum negative assortment every group has the same average genotype, so that each group plays the role of a population in eqn (A.5). For uniform group size  $n$ , relatedness of the rest of the group to actor takes the same value in each group of  $r^o = -1/(n - 1)$ . Thus, the lower bound on  $r^o$  is  $-1/(n - 1)$ , and the lower bound on assortative relatedness under uniform group size is  $r_a^o = r^o - r_s^o = -1/(n - 1) - r_s^o$ .

Of course, these bounds on  $r_a$  are theoretical limits, subject to the constraints that the actual distribution of genotypes in a given population permits assortment of individuals into groups of genetically identical individuals for maximal  $r_a$ , and groups that each have the same average genotype for minimal  $r_a$ . The bounds on average

TABLE 4  
*Range of possible values for relatedness and its components in group-structured populations. Some inequalities assume that group size is uniform*

Trait type	Structural relatedness	Assortative relatedness	Total relatedness
Other-only	$-1 < r_s^o \leq 0$	$-1/(n-1) - r_s^o \leq r_a^o \leq 1 - r_s^o$	$-1/(n-1) \leq r^o \leq 1$
Whole-group	$0 < r_s^w \leq 1$	$-r_s^w \leq r_a^w < 1 - r_s^w$	$0 \leq r^w \leq 1$

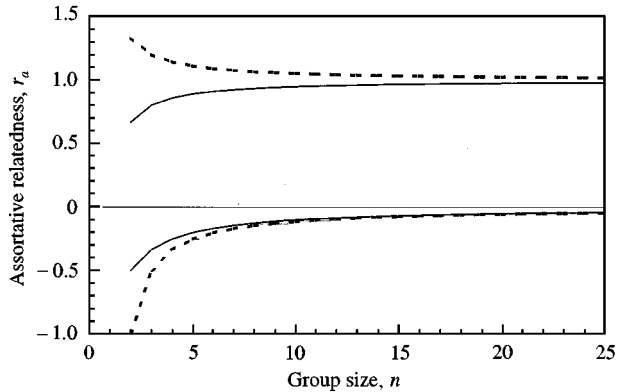


FIG. 4. The potential range of assortative relatedness ( $r_a$ ) as a function of group size. Dashed lines indicate upper and lower bounds for other-only traits, and solid lines indicate upper and lower bounds for whole-group traits. Lower bounds assume infinite population size, and upper bounds assume the minimal population size of twice the group size.

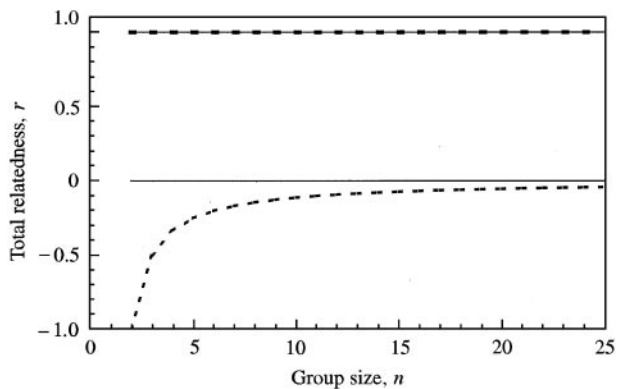


FIG. 5. The bounds on total relatedness as a function of group size for whole-group traits (—) and other-only traits (---). Lower bounds assume extreme negative assortment and upper bounds represent extreme positive assortment. Inequalities are from Table 4.

relatedness and its components are summarized in Table 4 and illustrated in Figs 4 and 5. A crucial difference between the two trait types is that total relatedness can be negative only for other-

only traits (Fig. 5). Although negative relatedness is possible for other-only traits under any population structure, it falls significantly below zero only when the population is quite small (Fig. 2), or there is strong negative assortment among small groups (Fig. 4).

### Discussion

To summarize, the relatedness of recipients to actors differs even for whole-group vs. other-only traits of the same actor. This is an example of the more general phenomenon that relatedness can differ among traits or loci in the same individual when genetic similarity is caused by anything other than common ancestry in an outbred population (Grafen, 1991; Queller, 1996). Relatedness is generally higher for whole-group traits, especially in small groups, in small populations, and under negative assortment. To clarify the sources of these differences we can partition relatedness into structural relatedness due to group size and population size, and assortative relatedness due to departures from random group composition. Each component is subject to different influences and has different bounds for other-only vs. whole-group traits. As a result the two trait types differ in their evolutionary responses to population size, group size, and assortment pattern.

These differences have received surprisingly little attention, perhaps because most studies of selection in group-structured populations have considered only one trait type (Table 2). These studies do not always stress that the critical quantity with regard to inclusive fitness effects is relatedness between actor and recipients of group fitness effects. Many phrases used to describe recipients, such as “group members” or “group of interactants”, are ambiguous as to whether or not

TABLE 5

*Some statements that are valid for one trait type but not the other. The term "relatedness" refers to average relatedness of recipients to actors in group-structured populations*

	Other-only traits	Whole-group traits
Relatedness is zero in randomly assorting infinite populations	True	False
Relatedness is zero only when all groups have the same average genotype	False	True
Relatedness can be negative	True	False
Relatedness can be positive without positive assortment	False	True
Relatedness falls with increasing group size, all else being equal	False	True
Relatedness equals the proportion of total genetic variance that falls into the between-group component	False	True
Traits conferring a within-group disadvantage can spread under random assortment	False	True

they include actors. As a result, the literature contains various statements about relatedness that can appear contradictory. A clear distinction between whole-group and other-only traits helps to reconcile these apparent conflicts (Table 5).

Some previous findings based on consideration of only one of the two trait types are valid for both, but lead to predictably different conclusions for the two types. For example, Hamilton (1970) showed that negative relatedness is necessary for the evolution of spiteful behaviors, which lower the absolute fitness of both actor and recipients. The fact that average relatedness cannot be negative for whole-group traits thus has implications for the kinds of social traits that can evolve. Whole-group traits cannot evolve to be spiteful, and can be selected for only if they benefit the groups they reside in. In contrast, other-only traits need not provide a group benefit to be favored, and can evolve to be spiteful.

Other findings are only valid for one trait type. For example, Breden (1990) showed that Hamilton's rule can be derived from a covariance model, leading to an equation for allele frequency change in terms of costs, benefits, and relatedness. However, this analysis relies on the "proportion of variance" formulation of relatedness (Table 3), and is therefore valid only for whole-group traits. Other authors have also used this formulation of the relatedness coefficient without explicitly noting that it applies only to whole-group traits (Frank, 1995a, 1997).

Another example of conclusions that are valid for only one trait type concerns the role of non-random assortment. Some authors have stated

that when assortment is random, relatedness is zero and there is therefore no effect of grouping on evolutionary outcomes (Grafen, 1984; Nunney, 1985; Linhart, 1999), while others have claimed that grouping patterns can change evolutionary outcomes even under random assortment (Wilson, 1990). The first statement is true for other-only traits, while the second is true for whole-group traits. In a similar vein, Hammerstein (1995) criticized Frank's (1995b) model of policing on the grounds that when interactions involve non-kin, i.e. when assortment is random, relatedness will be zero, making policing unlikely to evolve. This argument would be correct for other-only traits, but the policing trait that Frank modeled is a whole-group trait, for which relatedness in large populations is actually  $1/n$  rather than zero (Frank, 1996b).

Even some widely held assumptions are true of one trait type but not the other. For example, the quality of being either altruistic or not is a fixed characteristic of whole-group but not other-only traits. This point requires a brief digression into terminology because two definitions of the term "altruism" are in common usage. These have been distinguished as "weak" vs. "strong" altruism (Wilson, 1979b, 1990). For a trait to be weakly altruistic requires only that it confers an individual cost, so that it reduces the actor's fitness relative to the rest of its group. In contrast a trait is strongly altruistic only if the net effect of its individual cost and group benefit is a reduction in the actor's direct fitness. Previous authors have assumed that a given trait is either strongly altruistic or not, independent of its social context. This assumption is valid for other-only traits,

which are always strongly altruistic because they confer only costs on the actor. It is not valid for whole-group traits, however, if the total group benefit is fixed. Whole-group traits are strongly altruistic when the actor's share of the group benefit is less than the individual cost. This depends partly on group size, so that the same whole-group trait may be strongly altruistic in large groups but not in small groups (Pepper & Smuts, 2000). Strong altruism is therefore not a characteristic of the trait alone, but is codetermined by population structure.

The concept of relatedness in evolutionary biology still lives an odd double life. On the one hand, the primary literature is in complete agreement that the more general concept of relatedness as genetic similarity is the correct predictor of evolutionary outcomes, rather than the narrower idea of relatedness through common descent. In the words of Hamilton (1975, pp. 140–141), “[K]inship should be considered just one way of getting positive regression of genotype in the recipient.... Thus the inclusive fitness concept is more general than ‘kin selection.’” The modern concept of relatedness accommodates both structural and assortative components. Among sources of assortative relatedness, common descent is clearly predominant in nature. However, “the special role of relatives is a powerful result of the theory, not a restricting assumption” (Grafen, 1991, p. 9). On the other hand, textbooks on evolution continue to mention only the older and narrower concept of genealogical relatedness (Ridley, 1996; Bell, 1997; Hartl & Clark, 1997; Futuyma, 1998; Maynard Smith, 1998; but see Frank, 1998). Although it reflects the emphasis placed on kinship in empirical research, this restricted view of relatedness obscures the full meaning and utility of this powerful concept.

I thank Barbara Smuts for many valuable discussions. Thanks also to Stephen Frank, Michael Lachmann, Barbara Smuts, David Wilson and two anonymous reviewers for helpful comments on earlier drafts.

#### REFERENCES

- ALBERT, B., GODELLE, B., ATLAN, A., DEPAEPE, R. & GOUYON, P. H. (1996). Dynamics of plant mitochondrial

- genome: model of a three-level selection process. *Genetics* **144**, 396–382.
- BELL, G. (1979). Group selection in structured populations. *Am. Nat.* **112**, 389–399.
- BELL, G. (1997). *Selection: The Mechanism of Evolution*. New York: Chapman & Hall.
- BOORMAN, S. A. & LEVITT, P. R. (1973). Group selection on the boundary of a stable population. *Theor. Pop. Biol.* **4**, 85–128.
- BOURKE, A. F. G. & FRANKS, N. R. (1995). *Social Evolution in Ants*. Princeton: Princeton University Press.
- BOYD, R. & RICHERSON, P. (1992). Punishment allows the evolution of cooperation (or anything else) in sizable groups. *Ethology and Sociobiology* **13**, 171–195.
- BRADLEY, B. J. (1999). Levels of selection, altruism, and primate behavior. *Quart. Rev. Biol.* **74**, 171–194.
- BREDEN, F. (1990). Partitioning of covariance as a method for studying kin selection. *Trends Ecol. Evol.* **5**, 224–228.
- BREDEN, F. & HAUSFATER, G. (1990). Selection within and between social groups for infanticide. *Am. Nat.* **136**, 673–688.
- BROOKFIELD, J. F. Y. (1998). Quorum sensing and group selection. *Evolution* **52**, 1263–1269.
- BUSS, L. W. (1987). *The Evolution of Individuality*. Princeton: Princeton University Press.
- CHAO, L. & LEVIN, B. R. (1981). Structured habitats and the evolution of anticompetitor toxins in bacteria. *Proc. Natl. Acad. Sci. U.S.A.* **78**, 6324–6328.
- CHARNOV, E. L. & KREBS, J. R. (1975). The evolution of alarm calls: altruism or manipulation? *Am. Nat.* **109**, 107–112.
- CLUTTON-BROCK, T. H. & PARKER, G. A. (1995). Punishment in animal societies. *Nature* **373**, 209–216.
- COHEN, D. & ESHEL, I. (1976). On the founder effect and the evolution of altruistic traits. *Theor. Pop. Biol.* **10**, 276–302.
- CONNOR, R. C., SMOLKER, R. & RICHARDS, A. F. (1992). Two levels of alliance formation among male bottlenose dolphins (*Tursiops* sp.). *Proc. Natl. Acad. Sci. U.S.A.* **89**, 987–990.
- DAY, T. & TAYLOR, P. D. (1998). The evolution of temporal patterns of selfishness, altruism, and group cohesion. *Am. Nat.* **152**, 102–113.
- DUGATKIN, L. A. (1990). N-person games and the evolution of cooperation: a model based on predator inspection behavior in fish. *J. theor. Biol.* **142**, 123–135.
- EDMUNDS, M. (1974). *Defence in Animals. A Survey of Anti-predator Defences*. Essex: Longman, Harlow.
- ESHEL, I. (1972). On the neighbor effect and the evolution of altruistic traits. *Theor. Pop. Biol.* **3**, 258–277.
- EVANS, C. S. & MARLER, P. (1994). Food calling and audience effects in male chickens, *Gallus gallus*: their relationships to food availability, courtship and social facilitation. *Anim. Behav.* **47**, 1159–1170.
- FRANK, S. A. (1995a). George Price's contributions to evolutionary genetics. *J. theor. Biol.* **175**, 373–388.
- FRANK, S. A. (1995b). Mutual policing and repression of competition in the evolution of cooperative groups. *Nature* **377**, 520–522.
- FRANK, S. A. (1996a). Models of parasite virulence. *Quart. Rev. Biol.* **71**, 37–78.
- FRANK, S. A. (1996b). Policing and group cohesion when resources vary. *Anim. Behav.* **52**, 1163–1169.
- FRANK, S. A. (1997). Models of symbiosis. *Am. Nat.* **150**, S80–S99.

- FRANK, S. A. (1998). *Foundations of Social Evolution*. Princeton, NJ: Princeton University Press.
- FROHLICH, K. & MADEO, F. (2000). Apoptosis in yeast—a monocellular organism exhibits altruistic behaviour. *FEBS Lett.* **473**, 6–9.
- FUTUYMA, D. J. (1998). *Evolutionary Biology*, 3rd Edn. Sunderland, MA: Sinauer Associates Inc.
- GILPIN, M. E. (1975). *Group Selection in Predator–Prey Communities*. Princeton, NJ: Princeton University Press.
- GRAFEN, A. (1984). Natural selection, kin selection and group selection. In: *Behavioural Ecology: An Evolutionary Approach* (Krebs, J. R. & Davies, N. B., eds), pp. 62–84. Sunderland, MA: Sinauer Associates Inc.
- GRAFEN, A. (1985). A geometric view of relatedness. In: *Oxford Surveys in Evolutionary Biology*, Vol. 2 (Dawkins, R. & Ridley, M., eds), pp. 28–29. Oxford: Oxford University Press.
- GRAFEN, A. (1991). Modelling in behavioural ecology. In: *Behavioural Ecology* (Krebs, J. R. & Davies, N. B., eds), 3rd Edn. pp. 3–31. Oxford: Blackwell Scientific.
- HALDANE, J. B. S. (1932). *The Causes of Evolution*. New York: Harper & Brothers.
- HAMILTON, W. D. (1964). The genetical evolution of social behaviour. I & II. *J. theor. Biol.* **7**, 1–52.
- HAMILTON, W. D. (1970). Selfish and spiteful behaviour in an evolutionary model. *Nature* **228**, 1218–1220.
- HAMILTON, W. D. (1971). Selection of selfish and altruistic behaviour in some extreme models. In: *Man and Beast: Comparative Social Behavior* (Eisenberg, J. F. & Dillon, W. S., eds), pp. 57–91. Washington, DC: Smithsonian Press.
- HAMILTON, W. D. (1972). Altruism and related phenomena, mainly in social insects. *Ann. Rev. Ecol. Syst.* **3**, 193–232.
- HAMILTON, W. D. (1975). Innate social aptitudes of man: an approach from evolutionary genetics. In: *Biosocial Anthropology* (Fox, R., ed), pp. 133–155. London: Malaby Press.
- HAMMERSTEIN, P. (1995). A twofold tragedy unfolds. *Nature* **377**, 478.
- HARTL, D. L. & CLARK, A. G. (1997). *Principles of Population Genetics*, 3rd Edn. Sunderland MA: Sinauer Associates Inc.
- HEMPTINNE, J. L. & DIXON, A. F. G. (1997). Are aphidophagous ladybirds (*Coccinellidae*) prudent predators? *Biol. Agric. Hort.* **15**, 151–159.
- HOOGLAND, J. L. (1983). Nepotism and alarm calling in the black-tailed prairie dog, *Cynomys ludovicianus*. *Anim. Behav.* **31**, 472–479.
- HURST, L. D. (1993). The incidences, mechanisms and evolution of cytoplasmic sex-ratio distorters in animals. *Biol. Rev. Cambridge Philos. Soc.* **68**, 121–194.
- HURST, L. D., ATLAN, A. & BENGTTSSON, B. O. (1996). Genetic conflicts. *Quart. Rev. Biol.* **71**, 317–364.
- JONES, S., YU, B., BAINTON, N. J., BIRDSALL, M., BYCROFT, B. W., CHABRA, S. R., COX, A. J. R., GOLBY, P., REEVES, P. J., STEPHENS, S., WINSON, M. K., SALMOND, G. P. C., STEWART, G. S. A. B. & WILLIAMS, P. (1993). The *lux* autoinducer regulates the production of exoenzyme determinants in *Erwinia carotovora* and *Pseudomonas aeruginosa*. *EMBO J.* **12**, 2477–2482.
- KELLY, J. K. (1992). Kin selection in density regulated populations. *J. theor. Biol.* **157**, 447–461.
- KIRCHNER, J. W. & ROY, B. A. (1999). The evolutionary advantages of dying young: epidemiological implications of longevity in metapopulations. *Am. Nat.* **154**, 140–159.
- LEVIN, B. R. & KILMER, W. L. (1974). Interdemic selection and the evolution of altruism: a computer simulation study. *Evolution* **28**, 527–545.
- LINHART, Y. B. (1999). Mosaic landscapes, family structure and evolution. *Trends Ecol. Evol.* **14**, 376–377.
- MATESSI, C. & JAYAKAR, S. D. (1976). Conditions for the evolution of altruism under Darwinian selection. *Theor. Pop. Biol.* **9**, 360–387.
- MAYNARD SMITH, J. (1998). *Evolutionary Genetics*, 2nd Edn. Oxford: Oxford University Press.
- MCCAULEY, D. E. & WADE, M. J. (1980). Group selection: the genetic and demographic bias for the phenotypic differentiation of small populations of *Tribolium castaneum*. *Evolution* **34**, 813–821.
- MICHOD, R. E. (1997). Evolution of the individual. *Am. Nat.* **150**, S5–S21.
- MICHOD, R. E. & HAMILTON, W. D. (1980). Coefficients of relatedness in sociobiology. *Nature* **288**, 694–697.
- MIRALLES, R., MOYA, A. & ELENA, S. F. (1997). Is group selection a factor in modulating the virulence of RNA viruses? *Genet. Res.* **69**, 165–172.
- NUNNEY, L. (1985). Group selection, altruism, and structured-deme models. *Am. Nat.* **126**, 212–230.
- ORLOVE, M. J. & WOOD, C. L. (1978). Coefficients of relationship and coefficients of relatedness in kin selection: a covariance form for the rho formula. *J. theor. Biol.* **73**, 679–686.
- PACKER, C. & PUSEY, A. E. (1982). Cooperation and competition within coalitions of male lions: kin selection or game theory? *Nature* **296**, 740–742.
- PEPPER, J. W. & SMUTS, B. B. (2000). The evolution of cooperation in an ecological context: an agent-based model. In: *Dynamics in Human and Primate Societies: Agent-Based Modeling of Social and Spatial Processes* (Kohler, T. A. & Gumerman, G. J., eds). Oxford: Oxford University Press.
- QUELLER, D. C. (1985). Kinship, reciprocity and synergism in the evolution of social behavior. *Nature* **318**, 366–367.
- QUELLER, D. C. (1994). Genetic relatedness in viscous populations. *Evol. Ecol.* **8**, 70–73.
- QUELLER, D. C. (1996). The measurement and meaning of inclusive fitness. *Anim. Behav.* **51**, 229–232.
- RIDLEY, M. (1996). *Evolution*, 2nd Edn. Cambridge, MA: Blackwell Science Inc.
- RILEY, M. A. & GORDON, D. M. (1992). A survey of Col plasmids in natural isolates of *Escherichia coli* and an investigation into the stability of Col-plasmid lineages. *J. Gen. Microbiol.* **138**, 1345–1352.
- RISSING, S. W., POLLOCK, G. B., HIGGINS, M. R., HAGEN, R. H. & SMITH, D. R. (1989). Foraging specialization without relatedness or dominance among co-founding ant queens. *Nature* **338**, 420–422.
- SEELEY, T. D. (1995). *The Wisdom of the Hive: The Social Physiology of Honeybee Colonies*. Cambridge: Harvard University Press.
- SEGER, J. (1981). Kinship and covariance. *J. theor. Biol.* **91**, 191–213.
- SHERMAN, P. W. (1977). Nepotism and the evolution of alarm calling. *Science* **197**, 1246–1253.
- TAYLOR, P. D. (1992). Altruism in viscous populations—an inclusive fitness model. *Evol. Ecol.* **6**, 352–356.
- TAYLOR, P. D. & FRANK, S. (1996). How to make a kin selection model. *J. theor. Biol.* **180**, 27–37.

- TURNER, P. E. & CHAO, L. (1999). Prisoner's dilemma in an RNA virus. *Nature* **398**, 441–443.
- VAN BOVEN, M. & WEISSING, F. J. (1999). Segregation distortion in a deme structured population: opposing demands of gene, individual and group selection. *J. Evol. Biol.* **12**, 80–93.
- VELICER, G. J., KROOS, L. & LENSKI, R. E. 2000. Developmental cheating in the social bacterium *Myxococcus xanthus*. *Nature* **404**, 598–601.
- WILKINSON, G. (1984). Reciprocal food sharing in vampire bats. *Nature* **308**, 181–184.
- WILLIAMS, G. C. & WILLIAMS, D. C. (1957). Natural selection of individually harmful social adaptations among sibs with special reference to social insects. *Evol.* **11**, 32–39.
- WILSON, D. S. (1975). A theory of group selection. *Proc. Natl. Acad. Sci. U.S.A.* **72**, 143–146.
- WILSON, D. S. (1977a). Structured demes and the evolution of group advantageous traits. *Am. Nat.* **111**, 157–185.
- WILSON, D. S. (1977b). How nepotistic is the brain worm? *Behav. Ecol. Sociobiol.* **2**, 421–425.
- WILSON, D. S. (1979a). Coevolution in structured demes. *Proc. Natl. Acad. Sci. U.S.A.* **76**, 2084–2087.
- WILSON, D. S. (1979b). Structured demes and trait-group variation. *Am. Nat.* **113**, 606–610.
- WILSON, D. S. (1980). *The Natural Selection of Populations and Communities*. Menlo Park, CA: Benjamin Cummings.
- WILSON, D. S. (1990). Weak altruism, strong selection. *Oikos* **59**, 135–140.
- WILSON, D. S. & DUGATKIN, L. A. (1997). Group selection and assortative interactions. *Am. Nat.* **149**, 336–351.
- WILSON, D. S., POLLOCK, G. B. & DUGATKIN, L. A. (1992). Can altruism evolve in purely viscous populations? *Evol. Ecol.* **6**, 331–341.
- WRIGHT, S. (1945). Tempo and mode in evolution: a critical review. *Ecology* **26**, 415–419.
- WRIGHT, S. (1969). *Evolution and the Genetics of Populations. Vol. 2; The Theory of Gene Frequencies*. Chicago: University of Chicago Press.
- ZEYL, C. & BELL, G. (1996). Symbiotic DNA in eukaryotic genomes. *Trends Ecol. Evol.* **11**, 10–15.

## APPENDIX A

### Average Relatedness of the Rest of the Population to an Actor

We can express the average genotype of the rest of the population, excluding the actor, in terms of the genotypes of the global population and the actor. The genotype of the total population is an average of the contributions of actor and other individuals, weighted by their relative frequencies:

$$G_P = \frac{G_A + (N - 1)G_O}{N}, \quad (\text{A.1})$$

where  $G_P$ ,  $G_A$ , and  $G_O$  are the average genotypes of the global population, the actor, and the rest of the population excluding the actor, respectively,

and  $N$  is the global population size. This can be rearranged as

$$G_O = G_P - \frac{G_A - G_P}{N - 1}. \quad (\text{A.2})$$

The general formula for the regression coefficient is

$$\beta(y, x) = \frac{\sum(x - \bar{x})(y - \bar{y})}{\sum(x - \bar{x})^2}. \quad (\text{A.3})$$

To calculate relatedness we substitute  $G_A$  for  $x$  and  $G_O$  for  $y$ . However, averaging across all possible actors in the population,  $\overline{G_A} = G_P$  and  $\overline{G_O} = G_P$ . Thus, the average relatedness of the rest of the global population to the actor is

$$r = \beta(G_O, G_A) = \frac{\sum(G_A - G_P)(G_O - G_P)}{\sum(G_A - G_P)^2}. \quad (\text{A.4})$$

Substituting eqn (A.2) into eqn (A.4) and simplifying yields

$$r = \frac{-1}{N - 1}. \quad (\text{A.5})$$

## APPENDIX B

### Whole-group Structural Relatedness under Mixed Group Sizes

To find structural relatedness for whole-group traits under uniform group size  $n$  we substitute eqn (4) into eqn (2), giving

$$r_s^w = \frac{1}{n} - \frac{n - 1}{n(N - 1)}. \quad (\text{B.1})$$

For mixed group sizes, we assume that genotypes are distributed randomly with respect to group size. The probability of an individual appearing in a given group is then proportional to the group's size. Average relatedness is a weighted average of the right-hand side of eqn (B.1) across groups, with group size as the weighting factor. This is equivalent to averaging across individuals. Let the total population consist of  $N$  individuals indexed by  $i$ , divided into  $g$  groups,

with  $n_i$  representing the size of the group containing the  $i$ -th individual. Then whole-group structural relatedness is

$$r_s^w = \frac{1}{N} \sum_{i=1}^N \left( \frac{1}{n_i} - \frac{n_i - 1}{n_i(N - 1)} \right). \quad (\text{B.2})$$

This can be rearranged as

$$r_s^w = \frac{\sum_{i=1}^N (1/n_i) - (1/(N - 1)) \sum_{i=1}^N (n_i - 1)/n_i}{N}. \quad (\text{B.3})$$

We next substitute identities for the two summations in eqn (B.3). The first identity is

$$\sum_{i=1}^N \frac{1}{n_i} = \sum_{j=1}^g \sum_{i=1}^{n_i} \frac{1}{n_i} = \sum_{j=1}^g 1 = g, \quad (\text{B.4})$$

where  $i$  indexes individuals and  $j$  indexes groups. Using eqn (B.4), the second identity is

$$\sum_{i=1}^N \frac{n_i - 1}{n_i} = N - \sum_{i=1}^N \frac{1}{n_i} = N - g. \quad (\text{B.5})$$

Substituting eqns (B.4) and (B.5) into eqn (B.3) and simplifying yields

$$r_s^w = \frac{g - 1}{N - 1}. \quad (\text{B.6})$$

To put this result in terms of group size rather than number of groups, we can substitute  $N/\bar{n}$  for  $g$ , yielding

$$r_s^w = \frac{N - \bar{n}}{\bar{n}(N - 1)}. \quad (\text{B.7})$$

This can be rewritten as

$$r_s^w = \frac{1}{n} - \frac{\bar{n} - 1}{\bar{n}(N - 1)}, \quad (\text{B.8})$$

showing that as population size approaches infinity,  $r_s^w$  approaches  $1/\bar{n}$ . This was shown by Frank (1996b) for uniform group size.

## APPENDIX C

### Assortative Relatedness for Whole-group vs. Other-only Traits

Let the superscripts  $r^w$  and  $r^o$  indicate relatedness for whole-group and other-only traits, respectively, the subscripts  $r_s$  and  $r_a$  represent relatedness due to population structure and assortment, respectively, with  $r$  with no subscript indicating total relatedness. By the definition of assortative relatedness given in eqn (7), assortative relatedness for whole-group traits is

$$r_a^w = r^w - r_s^w. \quad (\text{C.1})$$

Under the assumption of uniform group size, from eqn (2) we have

$$r^w = \frac{1}{n} + \frac{(n - 1)r_s^o}{n}. \quad (\text{C.2})$$

Because eqn (C.2) is not based on any assumptions about assortment, it also holds under random assortment. Therefore, it is a spatial case of eqn (C.2) that

$$r_s^w = \frac{1}{n} + \frac{(n - 1)r_s^o}{n}. \quad (\text{C.3})$$

Substituting eqns (C.2) and (C.3) into eqn (C.1) yields

$$r_a^w = \frac{(n - 1)}{n} r_a^o. \quad (\text{C.4})$$