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A simple rule for the evolution of contingent cooperation in large groups

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Humans cooperate in large groups of unrelated individuals, and many authors have argued that such cooperation is sustained by contingent reward and punishment. However, such sanctioning systems can also stabilize a wide range of behaviours, including mutually deleterious behaviours. Moreover, it is very likely that large-scale cooperation is derived in the human lineage. Thus, understanding the evolution of mutually beneficial cooperative behaviour requires knowledge of when strategies that support such behaviour can increase when rare. Here, we derive a simple formula that gives the relatedness necessary for contingent cooperation in n -person iterated games to increase when rare. This rule applies to a wide range of pay-off functions and assumes that the strategies supporting cooperation are based on the presence of a threshold fraction of cooperators. This rule suggests that modest levels of relatedness are sufficient for invasion by strategies that make cooperation contingent on previous cooperation by a small fraction of group members. In contrast, only high levels of relatedness allow the invasion by strategies that require near universal cooperation. In order to derive this formula, we introduce a novel methodology for studying evolution in group structured populations including local and global group-size regulation and fluctuations in group size.

1. Introduction

Unlike other mammals, humans cooperate in large groups of unrelated individuals. Examples include warfare, the construction of roads, canals and other capital facilities, and risk buffering behaviours such as food sharing and mutual aid. It seems likely that our ability to cooperate played a crucial role in the rapid growth and spread of human populations over the past 50 000 years [1,2]. Beginning with Trivers's seminal paper [3], many authors have argued that human cooperation is explained by reciprocity and other forms of contingent behaviour. Because people can recognize a sizable number of individuals and remember their previous behaviour, selection leads to a psychology in which the behaviour of actors is contingent on the previous behaviour of others. Individuals help only those who have helped them in the past, or punish those who do not cooperate in mutually beneficial activities. If, in the long run, benefits of sustained cooperation exceed the short-term benefits of defection, then contingent strategies supporting cooperation can be evolutionarily stable. Such equilibria can explain the persistence of cooperation among unrelated individuals.

However, showing that cooperation can persist is not enough. Under plausible conditions, contingent strategies can stabilize virtually any behaviour including non-adaptive and maladaptive behaviours [4,5]. A complete explanation must explain why contingent cooperation is a likely evolutionary outcome. Moreover, contingent cooperation, especially in sizable groups, appears to be very rare among primates [6], and thus it is very likely that the ancestral condition in the human lineage is non-cooperative. This means it is not enough to explain the stability of contingent cooperation [7–10]; we must also explain how contingent strategies supporting cooperation can increase when rare. This is problematic because such strategies are altruistic when rare. Because other group members are unconditional defectors, rare contingent cooperators pay the cost of cooperation

and benefit others, but do not gain any long run benefit. In a similar way, strategies that punish contingent on others punishing, must punish or make a costly signal of intent to punish in order to determine how many punishers there are in the group.

For reciprocity among pairs, kinship provides an easy solution to this problem. If interactions are repeated many times, the benefits to reciprocity can be very large. This means that rare reciprocators can increase even if they have only a small chance of interacting with another reciprocator, and thus even low levels of relatedness can allow reciprocating strategies to increase [11]. Since population structure often leads to low but positive background levels of relatedness, there is a plausible explanation for the evolution of pairwise reciprocity.

It is not clear whether relatedness can play a similar role in the evolution of contingent cooperation in larger groups. Boyd and co-workers [8,12] have presented models which suggest that the effect of relatedness diminishes rapidly with group size. However, these models assumed that groups are formed by sampling individuals with a constant relatedness to each other. Basic models of population structure are not consistent with this assumption because the biological processes that generate relatedness lead to interdependencies, so that knowing that two individuals share a gene by common descent increases the probability that other members of the group also share that gene by common descent. For a given relatedness, this increases the likelihood that groups will contain enough cooperators to sustain cooperation. As a result, existing work underestimates the possibility that contingent cooperation can increase when rare as a result of assortment due to population structure [13,14].

Here, we derive a rule (5.7) that gives the relatedness necessary for contingent cooperation in n -person iterated games to increase when rare. This rule applies to a wide range of payoff functions, but requires that the strategies supporting cooperation are based on a threshold. Such strategies are common in the literature [5,7,12,15,16]. For example, in the iterated public goods game, a plausible strategy is to cooperate during the first period, and then cooperate if at least a fraction θ of the n individuals in the group cooperate. The derivation of this rule also assumes that groups are very large, that relatedness is low and generated by an elastic island model population structure [14], or by budding viscosity population structure [17] (propagule dispersal with group competition [18], two-level Fisher–Wright [13]). We will present numerical results which suggest that this rule provides also useful estimates when some of these assumptions are relaxed and the demographic parameters are in the biologically relevant range, including levels of relatedness in the range from 2% to about 10%. In order to derive this rule in §5, we will, in §3 and §4, introduce a novel methodology for studying evolution in group structured populations including local and global group-size regulation and fluctuations in group size. This methodology is also useful for studying other problems and provides new insights about how migration and local regulation affect the evolution of cooperation and altruism.

2. The model

Individuals live in groups of a size that may fluctuate, but is usually close to a common value n . During a life cycle, they interact T times, and in each interaction they can express either a cooperative behaviour A or a non-cooperative behaviour N. Let δv_x be the incremental effect of an interaction on

the fitness of an individual expressing A given that a fraction x of the individuals in the group express A. By fitness, we mean the expected number of adult offspring of an individual. Here, $\delta \geq 0$ is a constant that gives the strength of selection and that we will always suppose to be small (weak selection). The cooperative behaviour may also affect the fitness of individuals in the group that do not cooperate; let $\delta v'_x$ be the incremental effect of an interaction on the fitness of an individual not expressing A given that a fraction x of the individuals in the group express A. Non-cooperators neither produce benefits nor experience any personal cost, so that $v'_0 = 0$. For technical reasons, we also assume, without restricting the applicability of the model, that v_x and v'_x are piecewise continuous and always continuous from the right. And we suppose that $v_0 = \lim_{x \rightarrow 0} v_x < 0$, meaning that social interaction reduces the fitness of an individual behaving cooperatively in a group in which few others behave cooperatively.

There are two heritable strategies. Cooperators express behaviour A during the first interaction and continue to express A during future interactions if the fraction of individuals in the group expressing A during the previous interaction is greater than or equal to θ . This means if the fraction of cooperators in the group is at least θ , cooperators behave cooperatively during all T interactions. We assume that $v_\theta > 0$ so that such sustained cooperation is mutually beneficial to the cooperators. Defectors never express the cooperative behaviour.

When cooperators are rare and groups are formed at random, virtually all cooperators are in groups without any other cooperators. Thus, cooperation cannot increase because cooperators experience a reduction in fitness in the first round compared to defectors, and thereafter the two types behave identically and receive no pay-off.

Cooperators can increase when rare only if groups are formed assortatively so that there is some chance that they benefit from long-term cooperation. This means that relatedness in the groups is key to the evolution of cooperation. However, knowing the coefficient of relatedness within groups (R) alone is not, in principle, enough to determine whether cooperation can increase because the fitness functions that we are considering are nonlinear functions of the frequency of cooperators in a group. To calculate the expected fitness of rare cooperators, the entire probability distribution of frequencies is required [13,14,19]. This distribution depends on the population structure.

Here, we assume non-overlapping generations and that groups are linked by migration so that each generation each individual migrates with probability m . And we assume one of the following two kinds of population structure.

- (1) Groups form an island model with group size elasticity. This population structure was introduced in [14] under the assumption of purely local regulation. Here, we will extend it to include also global regulation and call it ‘islands with local and global regulation’ (ILGR). The population structure and the relevant results will be summarized in §3. More detail and self-contained derivations that extend the results from [14] are provided in the electronic supplementary material. (This differs from the inelastic island model [20] which assumes completely fixed group sizes and thus cannot accommodate average fitness different from 1. Other approaches to the effects of group elasticity on the evolution of cooperation can be found, for instance, in [21–23] and references therein.)

- (2) Groups compete among themselves for the production of new groups in the next generation. This is the ‘budding viscosity’ population structure of Gardner & West [17], called ‘propagule dispersal with group competition’ in [18], and ‘two-level Fisher–Wright’ (2IFW) in [13]. The idea is that cooperators, at a cost to themselves, help their group in its competition with other groups.

After discussing these population structures in the next section, we will indicate why we conjecture that the results and methods that we use should apply to a broader class of population structures.

3. Population structures: general results

First, we describe ILGR and summarize the main results derived in the electronic supplementary material. In ILGR, the population consists of g groups with a common carrying capacity n_0 . We will assume that g and n_0 are large. In the absence of selection, when all the groups have n_0 individuals, the individuals have fitness 1. When the total population size differs from $gn_0 = N_0$, fitnesses are modified through global regulation, and when the size of a group differs from n_0 , fitnesses of its members are also modified through local regulation. We model these effects, including also selection, by setting the absolute fitness (expected number of adult offspring) of a focal individual of type $*$ (there are two types, A and N) as

$$w^*(x, s, S) = H(s, S) + \delta h(s)v_x^*, \quad (3.1)$$

where x is the fraction of types A in the focal’s group, $s = n/n_0$ is the scaled size of the focal’s group, assumed to currently have size n , and $S = N/N_0$ is the scaled size of the complete population, assumed to currently have size N . The pay-off $h(s)v_x^*$ indicates how the fitness of the focal individual is modified by the behaviour of types A and N in its group, and we assume that $v_x^N = 0$, $h(s)$ is differentiable and $h(1) = 1$. We assume that $H(s, S)$ is strictly decreasing in s and in S , is continuously differentiable, takes the value 1 at $(s, S) = (1, 1)$ and that its partial derivatives at this point, $\partial H(s, S)/\partial s = -\lambda$ and $\partial H(s, S)/\partial S = -\lambda_g$ satisfy $\lambda \geq 0$, $\lambda_g \geq 0$, $\lambda + \lambda_g \leq 1$. These assumptions mean that $H(s, S)$ describes local regulation, with strength λ , towards group size n_0 , and global regulation, with strength λ_g , towards average group size n_0 . Note that in the absence of selection (when $\delta = 0$), all the individuals have fitness given by $H(s, S)$, and that if s and S are close to 1, then $H(s, S) \approx 1 - \lambda(s - 1) - \lambda_g(S - 1)$, so that $(s, S) = (1, 1)$ is a stable equilibrium. The fitness (3.1) is the expected number of adult offspring of each individual, and a full specification of the model must include the choice of the offspring distribution with this mean (e.g. Poisson).

The model assumes non-overlapping generations, and random migration at rate m after reproduction in each generation, meaning that with probability m each individual born in this generation leaves its group once it reaches adulthood and relocates in a randomly chosen group. Since larger groups produce more migrants, migration as well as local regulation drive groups towards the average size n_0 . The comparison between these two forces, quantified by m and λ , is crucial in the results described below. Selection will be assumed to be weak, meaning that δ is positive but small (the precise conditions are discussed in §5 of the electronic supplementary material). This implies that regulation and

migration act faster than selection and drive the system to a quasi-equilibrium in which the distribution of group sizes varies little over time (at any time $s = 1 + O(\delta)$), while the fraction p of types A changes at a rate of order δ , and therefore may change substantially in the long time-scale $1/\delta$.

In the electronic supplementary material, we study how in quasi-equilibrium p changes over one generation. We show that

$$\Delta p = \delta pqF(p) + o(\delta) \quad (3.2)$$

and

$$F(p) = \int_0^1 \text{beta}(x|lp + 1, lq)(v_x^A + Q\bar{v}_x)dx - \int_0^1 \text{beta}(x|lp, lq + 1)(v_x^N + Q\bar{v}_x)dx, \quad (3.3)$$

where $q = 1 - p$, $\text{beta}(x|\alpha, \beta)$ is the probability density of a beta distribution with parameters α and β , $l = (1/R) - 1$ (recall that R is group relatedness), $\bar{v}_x = xv_x^A + (1 - x)v_x^N$, and

$$Q = \frac{-1}{1 + 2m_e/\lambda}, \quad (\text{ILGR}), \quad (3.4)$$

with $m_e = m((1 - m/2)/(1 - m)^2)$ (which is close to m when m is small). The condition for p to increase is $F(p) > 0$, and in particular, by taking the limit $p \rightarrow 0$, we obtain the condition for types A to proliferate when rare as

$$\int_0^1 \text{beta}(x|1, l)(v_x^A + Q\bar{v}_x)dx > 0. \quad (3.5)$$

Table 1 and figure 1 provide support for the conclusions summarized above, based on numerical simulations. In our simulations, group sizes were chosen in the range from 20 to 320 and offspring distributions were Poisson. This last assumption implies relative variability of group sizes from generation to generation of the order of $\sqrt{n_0}/n_0$, which could be as large as $\sqrt{20}/20 \approx 22\%$. Such conditions proved to be compatible with the theoretical approximate beta distribution of x , required in the derivation of (3.3), provided we used for relatedness in the groups the empirical value of $F_{\text{st}} = ((\text{Var}(x))/pq)$, so that $l = (1/F_{\text{st}}) - 1$. This is natural since, when the frequency of cooperators in the population is p , the distribution of x is approximately beta with parameters pl and ql , which has mean p and variance $((pq)/(1 + l))$, implying that the empirical F_{st} should be $1/(1 + l)$. As expected, the agreement with the beta distribution, the value of Q and the predicted evolution of p improve with increasing n_0 . But agreement is still very good even for the smaller values of n_0 .

The quantity Q is an important population parameter that, in the ILGR setting, measures the relative strength of local regulation and migration in keeping group sizes close to n_0 . Its appearance in the formulae above can be explained qualitatively as follows. Observing the type of a focal individual tells us something about the composition of its group, not only at the present time, but also in the recent past, since the lineage of the focal must have been in the group for a time of order $1/m$ generations. This implies that the average pay-off \bar{v}_x in the recent past correlates with the type of the focal. Hence, the current size of the group also correlates with the focal’s type through a term of order $\delta\bar{v}_x$. And this affects the fitness of the focal individual through a term of the same order, as a result of group regulation. The computation

Table 1. Simulation results. Evolution in a finite ILGR population simulation with 10^6 groups was simulated for 1100 time periods. Initially, frequency of type A was p_0 and groups were formed at random. Population regulation was given by the logistic $H(S, s) = r_0((1 - a)(1 - S) + a(1 - s))$, $h(s) = 1$ and $\delta = 0.01$. There were three pay-off functions that gave the incremental effect of social behaviour on the expected number of offspring of types A and N in a group in which As had frequency x . Linear: $v_x^A = x - 0.0625$ and $v_x^N = x$. Threshold: for $x < 0.2$, $v_x^A = -0.0625$ and $v_x^N = x$, and for $x \geq 0.2$, $v_x^A = 1 - 0.00625$ and $v_x^N = 1$. Iterated Public Goods Game (IPG): for $x < 0.2$, $v_x^A = (x - 0.0625)/9$ and $v_x^N = x/9$, and for $x \geq 0.2$, $v_x^A = x - 0.0625$ and $v_x^N = x$. The observed value of Q is the average of values in the interval [100, 1100] calculated using (3.3) in the electronic supplementary material. The first 100 time periods were ignored to allow the population to reach demographic quasi-equilibrium. The predicted value of Q is given by (3.4). The observed moments of the distribution of x were calculated using the empirical distribution of x at $t = 1100$ and the predicted ones were calculated using the beta(p/q) distribution for the values of p and $l = 1/F_{xt} - 1$ calculated from the empirical distribution of x at $t = 1100$. This is the beta distribution that has the first two moments identical to the observed ones, and for these reason those first two moments are omitted from the table. The observed value of p_{1100} was calculated from the distribution of x at time $t = 1100$, and the predicted value was calculated by iterating the recursion for p_t given in (3.2) and (3.3), where the betas are calculated using $l = 1/F_{xt} - 1$ for period t , and Q is given by (3.4), beginning at time 100 with the value of p_{100} calculated from the simulation at time 100.

population parameters		moments of distribution of x												
n_0	m	r_0	a	p_0	pay-off function	Q	3rd	4th	5th	6th	7th	p_{1100}		
80	0.05	1	0.5	0.1	linear	pred.	-0.822	0.00313	0.00101	0.000388	0.000167	7.90×10^{-5}	0.0697	
					thresh.	obs.	-0.816	0.00319	0.00104	0.000402	0.000174	8.30×10^{-5}	0.0700	
					IPG	pred.	-0.822	0.0439	0.0211	0.0111	0.00621	0.00368	0.283	
	320	0.05	1	0.5	0.1	linear	obs.	-0.821	0.0444	0.0214	0.0113	0.00641	0.00383	0.283
						thresh.	pred.	-0.822	0.00327	0.00106	0.000404	0.000173	8.12×10^{-5}	0.0714
						IPG	obs.	-0.819	0.00338	0.00111	0.000427	0.000186	8.93×10^{-5}	0.0730
20		0.05	1	0.5	0.1	linear	pred.	-0.822	0.00535	7.31×10^{-5}	1.17×10^{-5}	2.13×10^{-6}	4.40×10^{-7}	0.0570
						thresh.	obs.	-0.826	0.00553	7.66×10^{-5}	1.24×10^{-5}	2.31×10^{-6}	4.77×10^{-7}	0.0568
						IPG	pred.	-0.822	0.0145	0.00416	0.00128	0.00417	0.000144	0.221
	20	0.05	1	0.5	0.1	linear	obs.	-0.822	0.0146	0.00428	0.00131	0.00428	0.000148	0.221
						thresh.	pred.	-0.822	0.000885	0.000132	2.27×10^{-5}	4.39×10^{-6}	9.44×10^{-7}	0.0710
						IPG	obs.	-0.822	0.000911	0.000137	2.39×10^{-5}	4.71×10^{-6}	1.03×10^{-6}	0.0717
20		0.05	1	0.5	0.1	linear	pred.	-0.822	0.0345	0.0234	0.0172	0.0133	0.0107	0.133
						thresh.	obs.	-0.798	0.0352	0.0241	0.0179	0.0140	0.0114	0.131
						IPG	pred.	-0.822	0.170	0.131	0.107	0.0901	0.0771	0.366
	20	0.05	1	0.5	0.1	linear	obs.	-0.801	0.171	0.133	0.109	0.0923	0.0803	0.397
						thresh.	pred.	-0.822	0.0180	0.0118	0.00846	0.00641	0.00510	0.0740
						IPG	obs.	-0.797	0.0184	0.0121	0.00881	0.00681	0.00543	0.0787

(Continued.)

Table 1. (Continued.)

population parameters		moments of distribution of x										
n_0	m	r_0	a	p_0	pay-off function	Q	3rd	4th	5th	6th	7th	P_{1100}
40	0.05	1	1	0.1	linear	pred.	0.00784	0.00381	0.00211	0.00127	0.000822	0.0771
						obs.	0.00808	0.00396	0.00221	0.00135	0.000885	0.0740
40	0.1	1	0.5	0.1	linear	pred.	0.0515	0.0300	0.0191	0.0130	0.00926	0.261
						obs.	0.0525	0.0308	0.0198	0.0136	0.00978	0.250
40	0.1	1	0.5	0.1	linear	pred.	0.00480	0.00170	0.000700	0.000320	0.000160	0.0842
						obs.	0.00475	0.00165	0.000670	0.000305	0.000152	0.0861
80	0.05	0.5	0.5	0.1	thresh.	pred.	0.0908	0.0512	0.0308	0.0196	0.0129	0.380
						obs.	0.0908	0.0511	0.0308	0.0196	0.0131	0.384
80	0.05	0.5	0.5	0.1	linear	pred.	0.00393	0.00132	0.000513	0.000225	0.000108	0.0802
						obs.	0.00408	0.00138	0.000546	0.000243	0.000118	0.0805
80	0.05	1	0.05	0.1	thresh.	pred.	0.0794	0.0431	0.0249	0.0152	0.00978	0.361
						obs.	0.0801	0.0435	0.0254	0.0156	0.0101	0.367
80	0.05	1	0.05	0.1	linear	pred.	0.00774	0.00279	0.00116	0.000531	0.000265	0.120
						obs.	0.00800	0.00291	0.00122	0.000566	0.000285	0.120
80	0.05	1	0.5	0.9	thresh.	pred.	0.172	0.110	0.0740	0.0515	0.0414	0.504
						obs.	0.174	0.112	0.0755	0.0529	0.0383	0.507
80	0.05	1	0.5	0.9	linear	pred.	0.668	0.600	0.544	0.497	0.458	0.858
						obs.	0.669	0.602	0.547	0.501	0.462	0.860
80	0.05	1	0.5	0.9	thresh.	pred.	0.856	0.821	0.790	0.762	0.738	0.942
						obs.	0.856	0.822	0.791	0.763	0.740	0.943

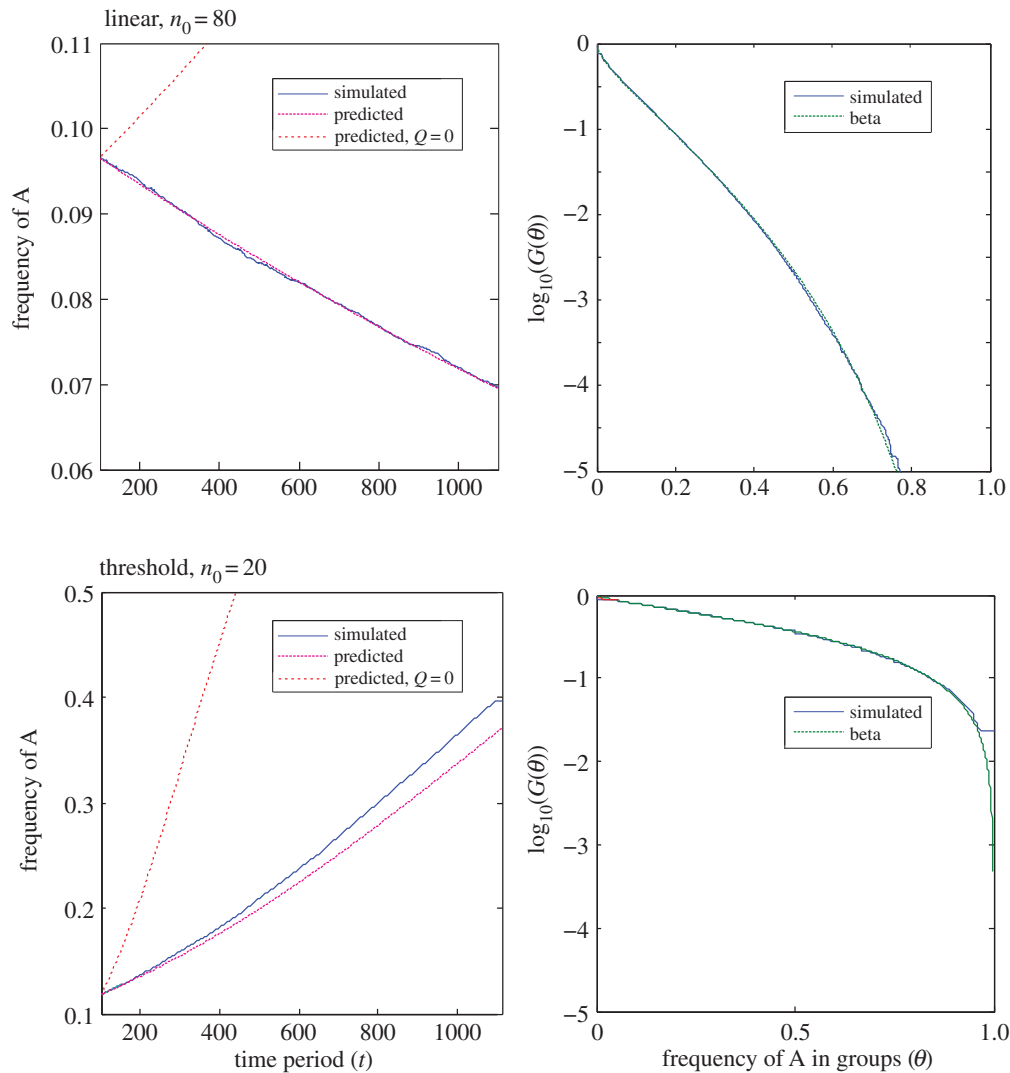


Figure 1. Simulation and predicted ILGR results compared for two models. In the top panel, carrying capacity was $n_0 = 80$ and the pay-off functions were linear ($v_x^A = x - 0.0625$ and $v_x^N = x$) and in the lower panel $n_0 = 20$ and pay-offs were a threshold function of the frequency of A in groups (for $x < 0.2$, $v_x^A = -0.0625$ and $v_x^N = 0$, and for $x \geq 0.2$, $v_x^A = 1 - 0.0625$ and $v_x^N = 1$). The functions $H(s, S)$ and $h(s)$ are as in table 1, with $r_0 = 1$, $a = 0.5$, $m = 0.05$ and $p_0 = 0.1$. The left panels compare the simulated trajectory of p_t , the frequency of A in the population, with the frequency calculated by iterating the recursion for p_t given in (3.2) and (3.3), where the betas are calculated using $l = 1/F_{st} - 1$ for period t , and Q is given by (3.4), beginning at time 100 (to allow for demographic quasi-equilibrium) with p_{100} equal to the value calculated from the simulation at time 100. The $Q = 0$ line was produced in the same way, but with $Q = 0$, to contrast with the proper prediction and highlight the need for Q . The right-hand panels compare the probability that the frequency of x in a group is greater than or equal to θ (the upper tail, $G(\theta)$). The observed values are calculated from the simulated distribution of x in the population at the end of the simulation ($t = 1100$) and the predicted values are derived using the beta(lp, lq) distribution for the values of p and $l = 1/F_{st} - 1$ calculated from the empirical distribution of x at $t = 1100$.

of this effect in the electronic supplementary material leads to the term $Q\bar{v}_x$ that appears in (3.3) and (3.5). Thus Q is an ecological parameter that tells us how much a small change in average fitness of group members in the past affects their current fitness by affecting group size. Note that λ_g is absent in (3.3) and (3.4). Global regulation affects all the groups in the same way and therefore does not produce correlations between group composition or focal type and group size. Global regulation does play an important role, though, in restraining average group size variability as p changes (see §5 of the electronic supplementary material).

To elaborate further on the intuitive meaning of Q in ILGR, we assume now that types A are cooperators that provide some costly benefit to the members of the group, in the sense that \bar{v}_x increases with x and $v_x^A < v_x^N$. When $\lambda \ll m$, groups with more cooperators are driven to the typical size n_0 mostly by migration, i.e. by producing more emigrants. The average fitness of members of such groups remains

larger than 1 in quasi-equilibrium. The frequency of cooperators may increase or decrease in time, depending on how the average values of v_x^A and v_x^N compare, and this is precisely what (3.2) and (3.3) entail in this case, as $Q \approx 0$. We call this regime the ‘Hamilton regime’. But when λ is comparable to m , a high level of cooperation in a group increases the size of the group to an equilibrium slightly (meaning order δ) above n_0 , in which local regulation reduces the effects of further cooperation. In the extreme case $m \ll \lambda$, groups with more cooperators are larger, but are in an equilibrium produced primarily by group regulation, in which its members have average fitness 1. Cooperators are then selected against as, in groups with both types, their fitness must be less than 1, while that of non-cooperators must be larger than 1. Again, this is precisely what (3.2) and (3.3) entail, as now $Q \approx -1$ and hence $F(p) < 0$. We call this regime the ‘crowded regime’. (For more on the intuition in this paragraph, see [14].)

We turn now to the population structure 2IFW. We provide a brief description of the model and the results from [13,14] that will be needed and refer the reader to these papers for further details. In 2IFW, generations are again non-overlapping, and the number of groups in each generation is again fixed as g . Groups have exactly the same size n . In each generation, each group selects independently a group from the previous generation to be its parent group, with probabilities proportional to the average fitness of members of each group in the previous generation. The membership in each group in the new generation is determined then by each member of the group selecting independently a member of the parent group with probabilities proportional to the fitnesses of these individuals. Once reproduction has occurred in this fashion and all the new g groups have been created, a fraction m of the individuals, chosen at random, is removed from their groups and relocated at random, preserving the size n of the groups. We suppose that there are two types, A and N, that the relative fitnesses of the individuals depend on the fraction x of types A in the group, being given by $w_{\text{rel}}^*(x) = 1 + \delta v_x^*$, and that offspring inherit the type of the parent. (The absolute fitnesses are simply the relative ones divided by the average value of the relative ones in the whole population. This average value has the form $1 + O(\delta)$, and therefore the absolute fitnesses have the form $w^*(x) = w_0 - \delta c 1_A + \delta b x + O(\delta^2)$, where w_0 is a common value for all individuals in the population in each generation.) In [13], we studied invasion in this setting, under strong or weak selection. In the relevant case for us here, in which groups are large, migration m low and selection is weak, we showed that the condition for invasion to occur is precisely (3.5), with $Q = 0$ (see display (3.3), [13]). We also explained in [14] that not only (3.5), but also the more general (3.2), (3.3) holds for 2IFW, with $Q = 0$.

Intuition about why the same beta distribution turns out to be relevant in the case of ILGR and 2IFW is as follows [14]. When one tracks the lineages of the members of a group back in time, in these two distinct settings, one obtains the same coalescent process, and that determines the distribution of x , which is known to be beta with parameters lp and lq [24]. Even when groups are smaller and group size fluctuations are relevant, we observe the beta distributions in computer simulations as good approximations (even with n_0 as small as 10). And even in simulations of population structures in which groups split, or groups become extinct at a low rate and recolonized, we have observed the betas. This can be explained by the fact that the relevant time-scale of the coalescent is given by the typical time $1/m$ needed for a lineage to exit the group. When m is small, this time-scale is much longer than the time-scale at which groups fluctuate in size and we obtain the same kind of coalescent, with rates of coalescence of lineages given by averages over the time-scales of the fluctuations in size.

Intuition about why $Q = 0$ in the 2IFW case is as follows. Information about the type $*$ of the focal individual affects our knowledge of its current fitness w in two ways. One is $* \rightarrow x \rightarrow w$; the first arrow is mediated by relatedness R , and this is the basis of Hamilton's work. The other is $* \rightarrow [\bar{w}]_{\text{rp}} \rightarrow n \rightarrow w$, where $[\bar{w}]_{\text{rp}}$ is the average fitness in the group in the recent past. In ILGR, n is elastic and this channel is important and is mediated by Q . In population structures that have a fixed group size, this channel is absent, or equivalently,

$$Q = 0 \quad (\text{population structures with fixed group size}). \quad (3.6)$$

A number of previous readers of this paper felt tempted to take a limit $\lambda \rightarrow \infty$ in ILGR, in order to produce a model with 'infinite rigidity' meaning fixed group sizes, and were puzzled by the fact that the right-hand side of (3.4) is then converging to -1 rather than to 0. In fact, if one violates the assumed conditions of ILGR, which require $\lambda < 1$, and considers the case of very large λ , one runs into a situation in which $(s, S) = (1, 1)$ becomes an unstable fixed point, and our analysis of ILGR, including (3.4), does not apply.

The two channels discussed above lead to a decomposition $F(p) = F_{\text{Hamilton}}(p) + F_{\text{extra-Hamilton}}(p)$, with the first term corresponding to the first channel and the second term to the second channel and including the factor Q . The discussion above and in [14], and the computation of $F_{\text{extra-Hamilton}}(p)$ in the electronic supplementary material suggest that (3.2) and (3.3) should apply to a broader class of population structures, including the possibilities of groups splitting or becoming extinct and being recolonized. The fashion in which Q relates to population parameters (migration rate, regulation rates, rates of group extinction, etc.) must be population-structure-dependent (in the same way that the relatedness R is). But we conjecture that (3.2) and (3.3) will apply quite broadly, with R and Q fully summarizing the role of the population structure in affecting the direction and speed of selection.

4. Linear public goods game: comparison with some of the related literature

In the case of a linear public goods game, $v_x^A = -c + bx$, $v_x^N = bx$, $\bar{v}_x = (b - c)x$, and (3.2) and (3.3) become (see the electronic supplementary material)

$$\Delta p = \delta p q (-c + (b + Q(b - c))R) + o(\delta). \quad (4.1)$$

The condition for types A to increase in frequency is then $R(b + Q(b - c)) > c$. This is deceptively similar to the condition in display (16) of Gardner & West [17], with their parameter a in place of our $-Q$ (see pages 1711–1713 in that paper for background on that condition and its relation with their display (10)). But the meaning of $-Q$ and a are completely different, as one can see from the fact that their population structures have a fixed size and therefore the quantity that we call Q is 0 in their setting (see (3.6)). Moreover, our δb and δc are (Hamilton's) fitness costs and benefits (when the population is in quasi-equilibrium, and up to errors of order δ^2), while theirs are vital rates that relate only indirectly to fitnesses. We explain these two claims next.

In our setting, consider first ILGR with $h(s) = 1$. In this case, the absolute fitness (3.1) is given by $w^*(x, s, S) = H(s, S) - \delta c 1_A + \delta b x$, where the symbol 1_A takes the value 1 when $*$ is A and 0 when $*$ is N. This means that the behaviour of each type A increases the absolute fitness of all members of its group (self-included) by $\delta b/n$, and additionally decreases its own fitness by δc . In the case of ILGR with arbitrary $h(s)$, we know from the electronic supplementary material that in quasi-equilibrium $s = 1 + O(\delta)$, and therefore also $h(s) = 1 + O(\delta)$, so that (3.1) becomes $w^*(x, s, S) = H(s, S) - \delta c 1_A + \delta b x + O(\delta^2)$, justifying again our claim above. Similarly, in the case of 2IFW, we again have absolute fitnesses $w^*(x) = w_0 - \delta c 1_A + \delta b x + O(\delta^2)$, justifying our claim.

The fact that the parameters c and b that appear in [17,20], and related models are typically not fitness costs and benefits is well known and is discussed in detail in [18], but since it is a pivotal issue in our discussion, we explain it again in the context of Taylor [20] (similar analysis applies to the more elaborate models of [17], which extend the model of Taylor [20]). The population structure introduced in [20] has fixed-size groups. In each generation, adults produce a very large (ideally infinite) number of offspring, given (in our notation, and with L as a large number) by $f^*(x) = L(1 - \delta c 1_A + \delta b x)$. A fraction m of the juveniles disperses to randomly chosen groups. Competition among the juveniles in each group eliminates most of them and leaves exactly n of them in each group. They grow to adulthood and start the next cycle. A computation of the absolute fitness (e.g. [18]) gives $w^*(x) = 1 - \delta c 1_A + \delta(b - (b - c))x + O(\delta^2) = 1 - \delta c 1_A + \delta c x + O(\delta^2)$. This means that the behaviour of each type A increases the absolute fitness of all members of its group (self-included) by $\delta c/n$, and additionally decreases its own fitness by δc . The parameter b has no effect on fitness. This is very different from the situation in our setting, as discussed above. In [20], $Q = 0$, since groups have fixed size, but types A will be eliminated by selection whenever $R < 1$, as $\Delta p = \delta p q(-c + cR) + o(\delta) < 0$.

In [14], we had referred to the regime of ILGR in which Q is close to -1 , as the ‘Taylor regime’ because of similarities between the results of Taylor [20] discussed above, and what happens in our setting, in this regime. (In our case, we also have $\Delta p = \delta p q(-c + cR) + o(\delta) < 0$, from (4.1), but note that the cancellation of b occurs not at the level of the computation of fitness components, but only at the level of the computation of Δp .) We were aware of the important differences stressed above, but considered that name as appropriate if the differences were also kept in mind. Remarks by the referees convinced us that that name would rather produce confusion, and we changed it here to ‘crowded regime’. In this regime, types A find themselves in groups in which the effect of their ancestors in the same group increased the group density (made it crowded), so that local regulation now cancels the fitness effect that they still have at the current time on their group members. The cancellation that occurs here happens through the effects of past types A on the local ecology (crowding), while in [20] the cancellation is in the computation of the effects of the behaviour of the current types A on fitness.

The referees recommended that we compare our approach with that introduced in [21], and further applied in [22]. There are several distinctions to make. First, Rousset & Ronce [21] analyse the evolution in time not of p , but of a weighted average of reproductive values of the types A and N. The direction in which this quantity changes is also an indication of the direction in which p varies, but its analysis is, in principle, harder. In [21], the quantity denoted by S plays a role similar to our $F(p)$, giving the direction of selection. In displays (23), (24) and (25) of that paper, they provide a partition $S = S_f + S_{Pr}$ that is worth comparing with our partition $F(p) = F_{\text{Hamilton}}(p) + F_{\text{extra-Hamilton}}(p)$. In addition to being partitions of different quantities, their S_{Pr} reflects future effects on the fate of the types A and N, due to distinct reproductive values of offspring in different groups in the next generation. In contrast, our $F_{\text{extra-Hamilton}}(p)$ reflects the effects from the past actions of types A and N on their current differences in absolute fitnesses. In other words, we are considering different objects, and partitioning them in ways that are conceptually different. Second, in [21,22], the assumption of additive gene

action is made, which restricts the pay-off function to that of the linear public goods game. (Technically, this assumption is made as the assumption that fitness functions are differentiable, an important restriction that we discuss in detail in [25].) As our main interest in this paper is in iterated games with behaviour contingent on threshold number of participants, gene action across individuals is non-additive. For instance, the fitness effects of having 20% of types A in a group are not necessarily twice that of having 10% of types A in the group. Therefore, we needed to develop a methodology that would not require additive gene action. And this flexibility in our methodology (v_x^* is arbitrary in (3.3)) is indeed one of its qualities. Third, and perhaps even more important in the context of the comparison with [21,22], Lehmann *et al.* [22, p. 1142] concede that in situations in which group size is variable, they cannot compute S_{Pr} explicitly, and rather analyse only its sign. One of the most relevant contributions in the current paper is the explicit computation of both terms in $F(p)$, with the extra-Hamilton one yielding the factor Q , that we explicitly computed as (3.4) in the ILGR population structure that includes group size variability as a fundamental ingredient. The very simple expression that we obtained for Q clarifies the competitive effects of local group size regulation and migration in a quantitative and transparent fashion.

5. Invasion in iterated games

In this section, we will apply (3.5) to the pay-offs discussed in §2. This means that $v_x^A = v_x$, if $x < \theta$ and $v_x^A = T v_x$, if $x \geq \theta$; $v_x^N = v_x'$, if $x < \theta$ and $v_x^N = T v_x'$, if $x \geq \theta$. Applying these to (3.5), the condition for invasion is

$$\int_0^\theta \text{beta}(x|1, l) \hat{v}_x dx + T \int_\theta^1 \text{beta}(x|1, l) \hat{v}_x dx > 0, \quad (5.1)$$

where $\hat{v}_x = v_x + Q(xv_x + (1-x)v_x')$. The integrals in the above equation are relatively simple, since $\text{beta}(x|1, l) = l(1-x)^{l-1}$. However, instead of integrating, we will exploit the fact that when relatedness R is low, the exponent $l-1 = (1/R) - 2$ is large (the situation to keep in mind is $R \leq 10\%$, which implies $l-1 \geq 8$), so that this density function decreases rapidly with x . This means that the integrals put much more weight on the values of \hat{v}_x when x is close to the left end of the integration interval than on the values when x is further to the right. To use this observation, care has to be taken with the normalization when we restrict the distribution to an interval. For this purpose, we define $\langle \hat{v}_x \rangle_a^b$ as the average value of \hat{v}_x with respect to the beta distribution conditioned to being in the interval $[a, b]$. The conditional probability density, properly normalized, is $[(1-a)^l - (1-b)^l]^{-1} l(1-x)^{l-1}$, and the steep decrease of $(1-x)^l$ as x grows, implies that $\langle \hat{v}_x \rangle_a^b \approx \hat{v}_a$. (The rigorous statement is $\lim_{l \rightarrow \infty} \langle \hat{v}_x \rangle_a^b = \hat{v}_a$.) Motivated by these observations, we rewrite (5.1) as

$$[1 - (1-\theta)^l] \langle \hat{v}_x \rangle_0^\theta + T(1-\theta)^l \langle \hat{v}_x \rangle_\theta^1 > 0, \quad (5.2)$$

and then use the approximation just discussed, and the fact that $\hat{v}_0 = v_0 + Qv_0' = v_0$, to replace it with the approximate condition

$$[1 - (1-\theta)^l] v_0 + T(1-\theta)^l \hat{v}_\theta > 0. \quad (5.3)$$

We now assume, as we did in §2, that $v_0 < 0$. Then (5.3) implies that invasion requires $\hat{v}_\theta > 0$, and T sufficiently large:

$$\hat{v}_\theta = v_\theta + Q[\theta v_\theta + (1-\theta)v_\theta'] > 0 \quad (5.4)$$

Table 2. Simulation results. In all simulation, there were $g = 100\,000$ groups each with carrying capacity n_0 that exchanged migrants with all other groups at a rate m . The global carrying capacity was n_0g . As described in the text, the local share of population regulation was a , so $a = 1$ means that regulation only depended on group size. The initial frequency of A in all simulations was set to 0.001 and then the simulation was run for t_{sim} generations. Fitnesses in a step fitness function were given by $v_0 = -0.5$, $v_\theta = 0.7$ and $v'_\theta = 1$, and $\delta = 0.001$. Averages of F_{st} and group size n were taken over the last $t_{\text{sim}} - 50$ generations, to allow for quasi-equilibrium. This average value of F_{st} was used to compute $l = 1/F_{\text{st}} - 1$ that was used in two ways. (i) To calculate the predicted critical values of T using (5.7), labelled T_{sf} (for 'simple formula'). (ii) Using (3.3), to numerically calculate the value of T for which $F(0.001) = 0$. This value is labelled T_β . Finally, the observed average fitness difference between A and N types over the last $t_{\text{sim}} - 50$ generations were fit to a straight line using linear regression and the intersection with the x -axis is reported as the observed critical value of T , labelled T_0 . Negative values of T indicate that A cannot invade for any positive value of T because the population is in the crowded regime.

t_{sim}	θ	m	r_0	a	n_0	n	F_{st}	T_{sf}	T_β	T_0
1000	0.2	0.05	0.1	1	80	76	0.116	9.0	7.4	7.7
100	0.2	0.1	0.5	0.1	80	80	0.063	25.0	23.9	29.6
1000	0.2	0.05	0.5	1	80	79	0.114	-37.8	-41.1	-32.4
100	0.2	0.1	0.1	1	80	77	0.066	27.3	26.7	35.3
100	0.2	0.1	0.1	0.5	80	78	0.064	23.4	22.6	26.9
1000	0.2	0.1	0.1	0.5	80	78	0.064	23.4	22.4	26.3
1000	0.2	0.1	0.5	0	80	80	0.062	19.8	18.9	22.9
100	0.2	0.1	0.05	1	80	77	0.064	23.4	20.8	24.8
100	0.1	0.1	0.1	0	200	200	0.027	31.1	28.2	42.7
100	0.15	0.1	0.1	1	120	116	0.044	39.9	39.2	48.2

and

$$T > \frac{-v_0}{\hat{v}_\theta} \tilde{T}, \quad \text{where} \quad \tilde{T} = (1 - \theta)^{-1} - 1. \quad (5.5)$$

When the pay-offs are given, (5.4) becomes a condition on Q . It is interesting to observe that (5.4) can be satisfied even with a negative v_θ , provided that $Q < 0$ and v'_θ is sufficiently negative. This situation characterizes spiteful behaviour by the cooperators, in which at a cost to themselves the cooperators harm the non-cooperators. We defer a detailed analysis of this case, observing only that in this situation, the more negative Q is, the better for the spread of cooperators. We assume from now on, as we did in §2, that $v_\theta > 0$. If $v'_\theta < v_\theta$, then when $Q = 0$, we have $\hat{v}_\theta = v_\theta > 0$ so that (5.4) is satisfied, and when $Q \in [-1, 0)$, we have $\hat{v}_\theta > v_\theta(1 + Q)$, and (5.4) is also satisfied. And if $v'_\theta \geq v_\theta$, then the square bracket in (5.4) is positive and this condition becomes

$$Q > Q_c = \frac{-v_\theta}{\theta v_\theta + (1 - \theta)v'_\theta}. \quad (5.6)$$

In summary, assuming $v_0 < 0$ and $v_\theta > 0$, invasion happens when

$$(v'_\theta < v_\theta \text{ or } Q > Q_c) \text{ and } T > \frac{-v_0}{\hat{v}_\theta} \tilde{T}. \quad (5.7)$$

The impossibility of invasion when $v_\theta \leq v'_\theta$ and $Q \leq Q_c$ extends the crowded regime. And when $v'_\theta < v_\theta$, or $Q > Q_c$, the condition on T provides intuition on when the underlying game, the threshold θ and the relatedness R as well as Q (those two being the only inputs determined by the population structure) allow for invasion. The effect of Q in this inequality is restricted to the presence of \hat{v}_θ , rather than v_θ there. To understand its effect, observe (see (5.4)) that \hat{v}_θ is a linear function of Q in the interval $[Q_c, 0]$, which takes the value 0 at Q_c and the value v_θ at 0. This means that if Q is close to Q_c (only possible if $v_\theta \leq v'_\theta$), then T will have to be very large for cooperation to invade.

But if Q is far from Q_c , the effect of Q on the order of magnitude of the needed T is small. For instance, if $Q \in [0.1Q_c, 0]$, then $\hat{v}_\theta \in [0.9v_\theta, v_\theta]$, and having \hat{v}_θ in (5.5) instead of v_θ , amounts at most to a factor of about 1.1 in the needed T .

If v_0 and \hat{v}_θ are of similar order of magnitude, then the order of magnitude of T will be given by the factor \tilde{T} , which does not depend at all on the underlying game that is iterated. This factor depends on the population structure only through the level of relatedness R , which provides $l = 1/R - 1$ and on the threshold θ . It is also very sensitive to these two inputs, as the following examples show. To explore the effect that θ has on \tilde{T} , suppose that $R = 0.07$, which yields $l = 13.29$. Then with $\theta = 0.2$, we have $\tilde{T} = 18$, with $\theta = 0.25$ we have $\tilde{T} = 45$, with $\theta = 0.4$ we have $\tilde{T} = 887$, and with $\theta = 0.5$ we have $\tilde{T} = 10\,015$. And to explore the effect of R on \tilde{T} , suppose that $\theta = 0.1$. Then with $R = 0.02$, we have $l = 49$ and $\tilde{T} = 174$, with $R = 0.025$ we have $l = 39$ and $\tilde{T} = 60$, with $R = 0.03$ we have $l = 32.33$ and $\tilde{T} = 29$, and with $R = 0.04$ we have $l = 24$ and $\tilde{T} = 12$. When $\theta \ll 1$, additional intuition on the dependence of \tilde{T} on R and θ can be obtained from the approximation $\tilde{T} \approx e^{\theta l}$. For arbitrary θ , this becomes an inequality, $\tilde{T} \geq e^{\theta l}$. The exponential form of the dependence of \tilde{T} on θl explains the strong sensitivity to the values of R and θ illustrated by the numerical examples above. This is the reason why invasion is relatively easy when θ is low and very hard when θ is large.

In the electronic supplementary material, we computed the integrals in (5.1) for two kinds of pay-off functions: step functions and linear public goods games. And we used the resulting detailed formulae to analyse the conditions under which the approximation in (5.3) and (5.7) is good.

We have performed numerical simulations, covering a range of parameters (table 2). In these, (5.7) provides a value of T that is reasonably close to the empirical results. One should keep in mind that when p is very low (invasion conditions), drift is a powerful force competing with selection and adding randomness to the evolution. This noise is reflected in the lesser accuracy of the predictions of the critical

T as compared to the predictions in table 1, even when we used the full theory (3.3) based on the beta distribution. Considering the biological reality of drift as a source of significant noise when an invading gene still has low frequency (e.g. [24], ch. 4), the level of agreement in table 2 is reassuring of the value of the theory even in these extreme conditions.

6. Discussion

Inequality (5.3) and the more detailed (5.7) give simple approximations for the conditions necessary for contingent cooperative strategies to increase when rare. They do not depend on the form of the underlying fitness function, but do depend on various assumptions. (i) Contingent strategies that support cooperation lead individuals to continue cooperating if the number of cooperators exceeds a threshold. (ii) The assortment necessary for cooperative strategies to increase when rare results from an elastic island model population structure [14], (§4), or from groups competing for the production of new groups [13,17,18], or from other population structures for which (3.5) holds, with a constant Q that depends on the population structure. We conjecture that this is the case for many population structures (see §3 and [14]). (iii) The derivation of (5.3) and (5.7) also depend on the assumption that groups are very large, migration rates are low, and relatedness in groups is also low. However, numerical calculations suggest that they also give useful approximations in a wide and reasonable range of parameters.

The simple rule given by (5.7) provides a number of insights. It shows the relative importance of the population structure (through R and Q), of the pay-offs, of the threshold θ and of the number of iterations T .

It suggests that the evolution of contingent cooperation is very sensitive to relatedness (R) and to the threshold number of iterations necessary for cooperation to persist (θ). As long as $|Q| \ll |Q_c|$ and the fitness parameters v_0 and v_θ are of comparable order, the order of magnitude of the threshold number of interactions necessary for contingent cooperation to increase will be mainly determined by \tilde{T} which depends only on θ and R . When θ is large, under realistic levels of relatedness, invasion will require unreasonably large numbers of iterations. For instance, if $R \leq 0.1$ and $\theta \geq 0.65$, then $\tilde{T} \geq (1 - 0.65)^{-((1/0.1)-1)} - 1 = 12\,686$. On the other hand, when θ is small, invasion can occur at very low levels of relatedness. For instance, if relatedness is 0.02, and $\theta = 0.1$, then $\tilde{T} = 173$.

This sensitive dependence of the required level of relatedness on θ suggests that the high levels of cooperation

observed in humans are more likely to have evolved by contingent punishment than by contingent cooperation. Costly contingent punishment that persists at a low threshold θ can invade much more easily than costly contingent cooperation that persists only at a high threshold θ . But even a small fraction of punishers in a group can induce massive group cooperation. In the model presented in [12], individuals punish non-cooperators if enough other individuals in the group are also willing to punish non-cooperators. Because even a modest fraction of punishers can motivate others to cooperate, such contingent punishment strategies can increase when rare at relatively low levels of relatedness and still stabilize cooperation at a high level. Strategies that continue cooperating even when only a small fraction of others cooperate typically reach a stable polymorphic equilibrium in which the population displays a mix of cooperative and non-cooperative strategies [7,8]. Strategies that tolerate more defectors achieve lower frequencies of cooperators at equilibrium. Thus contingent strategies that behave altruistically when a small fraction of the group also behave altruistically can support ongoing cooperation, but will produce equilibria in which most individuals in the group do not contribute. Cooperation of this kind is observed. For example, in the United States, public radio is supported by voluntary contributions by a small fraction of listeners—most free ride. However, such strategies cannot support the widespread cooperation observed in many contexts. For example, virtually all Turkana men participate in warfare, even though the Turkana lack formal coercive institutions [26]. Our result is consistent with the idea that such widespread cooperation is supported by coordinated punishment of non-cooperators, by individuals that are willing to persistently punish non-cooperators at a cost to themselves, provided a small threshold number of punishers is achieved in the group.

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