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Evolution of emotional contagion in group-living animals

Wataru Nakahashi*, Hisashi Ohtsuki

School of Advanced Sciences, SOKENDAI (The Graduate University for Advanced Studies), Shonan Village, Hayama, Kanagawa 240-0193, Japan

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ABSTRACT

Emotional contagion refers to an instantaneous matching of an emotional state between a subject and an object. It is believed to form one of the bases of empathy and it causes consistent group behavior in many animals. However, how this emotional process relates to group size remains unclear. Individuals with the ability of emotional contagion can instantaneously copy the emotion of another group member and can take relevant behavior driven by this emotion, but this would entail both cost and benefit to them because the behavior can be either appropriate or inappropriate depending on the situation. For example, emotional contagion may help them escape from a predator but sometimes induce mass panic. We theoretically study how these two aspects of emotional contagion affect its evolution in group-living animals. We consider a situation where an environmental cue sometimes indicates a serious event and individuals have to make a decision whether to react to them. We show that, as the group size increases, individuals with the ability of emotional contagion would evolutionarily weaken their sensitivity to environmental cues. We also show that a larger group yields a larger benefit to them through such evolutionary change. However, larger group size prevents the invasion of mutants with the ability of emotional contagion into the population of residents who react to environmental cues independently of other group members. These results provide important suggestions on the evolutionary relationship between emotional contagion and group living.

1. Introduction

In many animals, strong emotion manifested by an individual triggers similar emotion and associated behavior in other individuals around him/her, which is called emotional contagion. This emotional process is considered to form one of the bases of empathy and contribute to various social behavior (de Waal, 2008, 2012; Panksepp and Panksepp, 2013). However, why such an emotional process evolved in many animals is a big mystery because of its cost. Here, by cost we refer not only to the developmental and neurophysiological cost to support and maintain such ability, but also to the cost of enhancing and/or suppressing neural and physiological activity in copying other's (often negative) emotion, such as increasing blood pressure or decreasing activity level (freezing). Nakahashi and Ohtsuki (2015) constructed a mathematical model to investigate conditions for emotional contagion to evolve, and showed that copying other's emotion is more adaptive than reacting independently when the environmental similarity between individuals is larger. However, their model assumed an interaction between two individuals only, so the condition for emotional contagion to evolve in group-living animals remains unclear. Since emotional contagion is biased toward in-group members (de Waal, 2012), there can be an evolutionary relationship between emotional contagion and group living. Therefore, how emotional contagion affects group size and how the group size affects the evolution of emotional contagion still remain an important but unsolved problem.

Why many animals form co-living groups and what affects their group size are important problems in evolutionary biology. Various merits of group formation have been proposed; for example, to keep body temperature, to cooperatively hunt preys, to resist predators, to struggle against other individuals, to find mating partners, to divide the labor, and so on (Nakahashi and Horiuchi, 2012; Nakahashi and Feldman, 2014; Nakahashi, 2016). When we study the relationship between emotional contagion and group formation, we have to consider the merit of information transmission within a group, because individuals may learn environmental information from others via emotional contagion. However, although some studies considered social learning within a group (Aoki and Nakahashi, 2008; Nakahashi et al., 2012; Ohtsuki et al., 2017), they did not study emotional contagion because the time scale of emo-

* Corresponding author.
E-mail address: w.nakahashi@gmail.com (W. Nakahashi).

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tional contagion is completely different from that of social learning assumed in these studies. In the previous studies, information is often assumed to be transmitted intergenerationally. However, information transmission occurs instantaneously, usually within seconds, through emotional contagion. Therefore, we need to develop a new framework to study the evolutionary relationship between emotional contagion and group formation.

Provided that there is no conflict of interest among group members, beneficial information to a group member should also be beneficial to other members. Since the probability that at least one member obtains beneficial information may increase as the group size increases, one may naively expect that individuals with the ability of emotional contagion obtain a larger benefit in a larger group. However, the actual situation is not so simple because individuals are error-prone. As the group size increases, the probability that at least one member obtains wrong information may also increase, which can cause harm in a group, by inducing a mass panic, for example. In fact, some breeds of chickens are too sensitive to sounds or stimuli and sometimes show panic behavior, which causes hurt (Abe et al., 2013). Therefore, it is unclear whether a larger group is beneficial for individuals with the ability of emotional contagion.

How to select information is an important problem for individuals. Although every individual should increase the probability of obtaining beneficial information and decrease that of wrong information, there is always a tradeoff between them. That is, when individuals attempt to obtain more information to receive more benefits, they inevitably obtain more wrong information at the same time. Therefore, there may be an optimum amount of information that an individual collects. The situation becomes more complex if individuals belong to a group and learn information from others via emotional contagion. There, the best strategy of information collection in each individual depends on those in other group members, and vice versa, so we need to consider a game-theoretic situation.

In this paper, we study the evolution of emotional contagion by using mathematical models. Since sensitivity to environmental information can affect the fitness of individuals, we also consider the evolution of sensitivity. We seek for the condition under which the fitness of individuals with the ability of emotional contagion is higher than that of individuals who always react independently of others (independent reaction). We also focus on the effect of group size on the evolution of emotional contagion and sensitivity.

2. Model

2.1. Overview

We suppose that individuals live in an environment where two kinds of events may happen, labeled as *trivial* and *serious*, and that the appropriate reaction to one event is inappropriate to the other. When a *serious* event happens, individuals should have strong emotion and react to it immediately, but when the event is *trivial*, they should ignore it. For example, if an individual finds a predator, he/she should have the emotion of fear and run away quickly driven by that emotion, but if it is a harmless animal or even just breeze, he/she should not react to it to avoid unnecessary cost.

We assume that if an individual takes an appropriate reaction to the event that happened (either *trivial* or *serious*), he/she suffers no fitness loss. In contrast, an actor incurs some fitness cost when he/she behaves in an inappropriate way. Although inappropriate reactions to serious events may be more costly than those to trivial ones, serious events may less frequently happen than trivial ones. Therefore, the product of event frequency and the cost of each inappropriate reaction may be in a similar order between these two. By abusing terms, we call this product (frequency times cost of single inappropriate reaction) the "cost of events" hereafter. In particular, the cost of trivial events is normalized to unity and that of serious ones is set as c (>0), i.e., the latter is c times as large as the former.

Individuals recognize each event via an environmental cue, but they sometimes mistakenly recognize it due to recognition errors and/or environmental noise. For example, they may mistake a predator for a harmless animal, or vice versa, if those animals look similar. To model uncertainty in environmental cues, we assume for simplicity that an environmental cue is represented by a real number z and that it distributes in a one-dimensional space. In particular, we assume that trivial and serious events always send cues $z=0$ and $z=1$, respectively, but each individual recognizes them with normally distributed variance σ^2 due to recognition errors and noise. In other words, σ^2 indicates cognitive ambiguity between these cues. Note that values $z=0$ and $z=1$ do not have any special meanings here, but they are merely results of non-dimensionalization of model parameters. In particular, the distance between the positions of those two cues (which is 1) and the magnitude of noise (σ) are on a comparable scale.

Suppose that an individual has perceived a cue, y, which contains some noise in it. Then this individual has to infer if the original cue was $z=1$ (serious) and the noise made it look y, or if the original cue was $z=0$ (trivial) and the noise made it look y. If the individual believes the former, he/she takes an appropriate action for a serious event (for example, escaping behavior), and if he/she believes the latter he/she takes an appropriate action for a trivial event (for example, ignoring it). We, however, model decision making by individuals in a simpler and more realistic way. In particular, we assume that each individual has a rule of thumb parameterized by a threshold value, x; he/she takes an action for a serious event (escaping behavior) if the recognized cue y is larger than the threshold x, and he/she takes an action for a trivial event (ignoring the cue) if the recognized cue y is smaller than x. In other words, one's x represents his/her "insensitivity" to environmental cues. We believe that this threshold model is appropriate for a wide range of animals because it does not require sophisticated ability in inference. Rather, our approach merely assumes that individuals can have different sensitivity to environmental cues, which can be physiologically realized by having different numbers/types of receptors or by having different neural connections. Hence it is natural to assume that the threshold x is genetically encoded.

With these assumptions, the probability that an individual with threshold x takes an *appropriate* reaction to a serious event (i.e. escaping the danger) is

$$p=\int_{y=x}^{\infty}\frac{1}{\sqrt{2\pi\sigma^2}}\exp\left[-\frac{(y-1)^2}{2\sigma^2}\right]dy \tag{1}$$

and that he/she shows an *inappropriate* reaction to a trivial event (i.e. escaping from nothing) is

$$q=\int_{y=x}^{\infty}\frac{1}{\sqrt{2\pi\sigma^2}}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy. \tag{2}$$

In the following we will mainly consider the evolution of this continuous trait, x. Note that, q is the probability of committing a false positive error (type I error; "escaping from nothing"), the cost of which (times the frequency of trivial cues) is assumed to be 1. In contrast, $1-p$ is the probability of committing a false negative one (type II error; "ignoring the danger"), the cost of which (time the frequency of serious cues) is assumed to be c. Obviously there is a trade-off between these two errors; if individuals attempt to decrease type I error by reducing their sensitivity, type II error necessarily increases, and vice versa, so there should be an optimal threshold x.

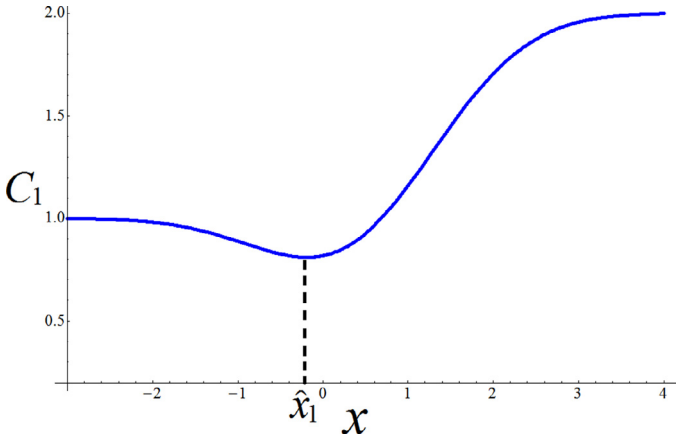

Fig. 1. The effect of insensitivity, x, on the total cost, C_1, is shown. Parameters are $\sigma^2 = 1$ and $c = 2$.

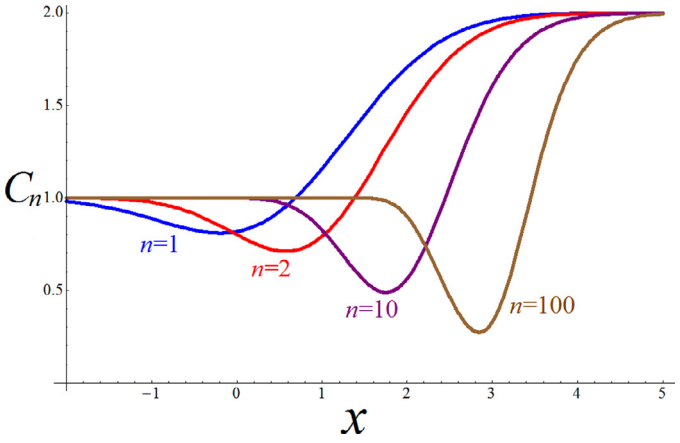

Fig. 2. The effect of insensitivity, x, and the group size, n, on the total cost, C_n, is shown. Parameters are $\sigma^2 = 1$ and $c = 2$.

2.2. Solitary animals

First, let us consider the decision strategy of solitary animals. For a solitary individual its group size is $n = 1$, so there are no interactions with others. According to the argument above, it suffers the cost of false positives by q and that of false negatives by $c(1-p)$. Hence its total cost is

$$C_1 = c(1-p) + q, \tag{3}$$

where p and q are given by (1) and (2), which are dependent on x. Fig. 1 shows how C_1 depends on x. From (1) and (2), we have

$$\frac{dp}{dx} = -\frac{1}{\sqrt{2\pi\sigma^2}}\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right] \tag{4}$$

and

$$\frac{dq}{dx} = -\frac{1}{\sqrt{2\pi\sigma^2}}\exp\left(-\frac{x^2}{2\sigma^2}\right), \tag{5}$$

so that

$$\frac{dC_1}{dx} = \frac{1}{\sqrt{2\pi\sigma^2}}\left\{c\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right] - \exp\left(-\frac{x^2}{2\sigma^2}\right)\right\}. \tag{6}$$

Therefore, C_1 is minimized when

$$c\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right] = \exp\left(-\frac{x^2}{2\sigma^2}\right), \tag{7}$$

i.e., the optimal insensitivity is

$$\hat{x}_1 = \frac{1}{2} - \sigma^2 \log c, \tag{8}$$

which should be favored by natural selection. Therefore, solitary animals evolve to be more sensitive to environmental cues when the cues are more ambiguous (large σ^2) provided the cost of serious events is higher than that of trivial events ($c > 1$), especially when serious events are very risky (large c). This makes sense, because it is of the utmost importance that one should not commit a false positive (i.e. mistakenly ignoring the danger). When $c = 1$, that is when trivial and serious events are equally risky, the evolutionary optimum exists at $\hat{x}_1 = 1/2$. This also makes sense because 1/2 lies exactly halfway between $z = 0$ (a cue of a trivial event) and $z = 1$ (a cue of a serious event).

2.3. Group-living animals with emotional contagion

Next, let us consider group-living animals with the ability of emotional contagion. Here, the group size is n and we assume that everyone can observe all the other group members. Emotional contagion in such a group is modeled in the following way. Suppose that at least one individual in the group takes a reaction appropriate for a serious cue (for example, escaping behavior) induced by strong emotion, such as fear. Then all the other individuals in the group that have the ability of emotional contagion immediately copy that strong emotion through emotional contagion and take reactions appropriate for a serious cue, too. For example, when an individual suddenly runs away, the other individuals with the ability of emotional contagion immediately follow him/her. An important assumption here is that a reaction appropriate for a trivial cue (such as neglecting the cue) is not copied through emotional contagion. For example, when an individual neglects an environmental cue and stays calm, that behavior is not copied via emotional contagion, because staying calm is associated with no strong emotional representation and hence it does not affect anyone. In this respect, our modeling of emotional contagion is clearly different from behavioral mimicry. In behavioral mimicry, individuals copy any behavior of others irrespective of their emotional states. In contrast, our emotional contagion model assumes that only reactions to serious cues are copied, because such reactions are induced by strong emotion.

Let us consider the evolution of sensitivity to environmental cues in a group of size n. Suppose that all n individuals have the ability of emotional contagion and that they have the same (in)sensitivity to environmental cues, x. The probability that no group members recognize a serious event (false negative) is $(1-p)^n$ (where p is given by (1)). This can be understood as follows. If at least one member correctly recognizes the serious event, the whole group can take an appropriate action through emotional contagion and a false negative is avoided. Because $1-p$ is the probability that a single individual fails to react to a serious event, its nth power gives us the probability of a false negative. Regarding false positives, if at least one group member mistakes a trivial event for a serious one then this false positive propagates to the whole group through emotional contagion. The probability that such a false positive does not occur equals the probability of no one committing a false positive, which is $(1-q)^n$ (where q is given by (2)) and therefore the probability of committing a false positive is $1-(1-q)^n$. In sum the total cost for each group member is calculated as

$$C_n = c(1-p)^n + 1 - (1-q)^n, \tag{9}$$

which is a function of x. Fig. 2 shows how C_n depends on x and n.

Because the cost (9) depends on insensitivity x, and because x is subject to evolution, we are interested in its evolutionary consequence. It is natural to believe that the insensitivity in animals

living in a group of size n should evolve toward the direction to reduce the total cost, (9). As shown in Appendix A, the total cost C_n is minimized at $x = \hat{x}_n$, where $\hat{x}_n$ is given as the solution to

$$x = \hat{x}_1 + (n-1)\sigma^2 \log \frac{1-q}{1-p}. \tag{10}$$

Note $\hat{x}_1$ is the optimal insensitivity by a solitary individual derived in (8). Also note that p and q in (10) are functions of x, so (10) contains x in both sides and therefore $\hat{x}_n$ is given only implicitly there. Calculations in Appendix A show that there exists a unique x that satisfies (10) because $(1-q)/(1-p)$ is monotone decreasing in x. Moreover, it is shown that the optimal insensitivity $\hat{x}_n$ increases as n increases. This makes intuitive sense; as the group size n increases, each single individual needs to be less sensitive to counterbalance increased sensitivity of the whole group due to emotional contagion. We have confirmed by a formal invasion analysis that $\hat{x}_n$ is indeed favored by natural selection (see Appendix B).

Next we study the advantage of group living and emotional contagion. Denoting the minimized total cost as $\hat{C}_n$, let us consider how $\hat{C}_n$ depends on n. Our goal here is to demonstrate $\hat{C}_n < \hat{C}_{n-1}$ for $n \geq 2$. Its proof consists of two steps, as shown below.

In the first step, consider a group composed of $n-1$ members with the optimal insensitivity $\hat{x}_{n-1}$ for a group of size $n-1$, and one "deviant" member with an arbitrary insensitivity x. Then, the cost that each member in this group suffers is calculated as

$$C_n(\hat{x}_{n-1}, x) = c(1-p)\left(1-\hat{p}_{n-1}\right)^{n-1} + 1 - (1-q)\left(1-\hat{q}_{n-1}\right)^{n-1}, \tag{11}$$

(where $\hat{p}_{n-1}$ and $\hat{q}_{n-1}$ are values of p and q (see (1) and (2)) evaluated at $\hat{x}_{n-1}$) which is minimized when the deviant member has insensitivity

$$\tilde{x} = \hat{x}_1 + (n-1)\sigma^2 \log \frac{1-\hat{q}_{n-1}}{1-\hat{p}_{n-1}}. \tag{12}$$

Since $C_n(\hat{x}_{n-1}, x) = \hat{C}_{n-1}$ when $x = \infty$ (if the deviant member has the ability of emotional contagion but has infinite insensitivity in reacting to environmental cues by itself, this group looks as if there were just $n-1$ individuals with the ability of emotional contagion), we have $C_n(\hat{x}_{n-1}, \tilde{x}) < \hat{C}_{n-1}$. In other words, a group can decrease the cost by adding one member with appropriate sensitivity.

All that remains is to show $\hat{C}_n < C_n(\hat{x}_{n-1}, \tilde{x})$ to complete the proof of $\hat{C}_n < \hat{C}_{n-1}$. In the second step, we consider a group composed of $n-2$ members with fixed insensitivities $x_{F1}, x_{F2}, \cdots, x_{Fn-2}$ and two members with arbitrary insensitivity x_A and x_B. Then, the cost that each of the group members suffers is

$$C_n = c(1-p_A)(1-p_B)\prod_{i=1}^{n-2}(1-p_{Fi}) + 1 - (1-q_A)(1-q_B)\prod_{i=1}^{n-2}(1-q_{Fi}). \tag{13}$$

Let us obtain the optimal combination $(\hat{x}_A, \hat{x}_B)$ that minimizes this cost. When x_B is fixed, the cost is minimized at

$$\tilde{x}_A(x_B) = \frac{1}{2} - \sigma^2 \log c' + \sigma^2 \log \frac{1-q_B}{1-p_B}, \tag{14}$$

and when x_A is fixed, it is at

$$\tilde{x}_B(x_A) = \frac{1}{2} - \sigma^2 \log c' + \sigma^2 \log \frac{1-q_A}{1-p_A}, \tag{15}$$

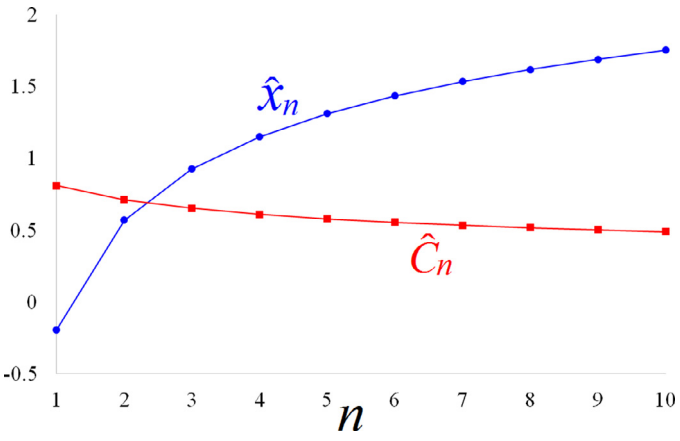

Fig. 3. The effect of the group size, n, on the optimal insensitivity, $\hat{x}_n$, and the minimized total cost, $\hat{C}_n$, is shown. Parameters are $\sigma^2 = 1$ and $c = 2$.

where

$$c' = c\prod_{i=1}^{n-2}(1-p_{Fi}) \Big/ \prod_{i=1}^{n-2}(1-q_{Fi}). \tag{16}$$

The optimal combination $(\hat{x}_A, \hat{x}_B)$ should be at a crossing point of $x_A = \tilde{x}_A(x_B)$ and $x_B = \tilde{x}_B(x_A)$, and because of the symmetry, there exists a unique crossing point where $x_A = x_B$ is satisfied (the uniqueness is proved in Appendix C). Therefore, the cost is minimized when every combination of two members has the same insensitivity, i.e., everyone has the same insensitivity. This minimal cost is $\hat{C}_n$, which is, by definition, smaller than $C_n(\hat{x}_{n-1}, \tilde{x})$. This completes the proof.

To sum up the analysis in this subsection, as shown in Fig. 3, the optimal insensitivity $\hat{x}_n$ that minimizes C_n increases as the group size n increases, and $\hat{x}_n$ is favored by natural selection. This is because the group-level sensitivity to environmental cues increases exponentially with the group size and individual insensitivity counters this effect. Moreover, the minimized total cost $\hat{C}_n$ decreases as n increases (see Fig. 3). In other words, group-living animals with emotional contagion evolve to be less sensitive to environmental cues when the group size is larger, and a larger group is more adaptive for them provided that they have emotional contagion with appropriate sensitivity.

2.4. Invasion analysis and evolution of emotional contagion

In the model above, it is assumed that all individuals innately show emotional contagion. Here, we consider an alternative (probably ancestral) strategy, independent reaction (IR), and study the evolution of emotional contagion strategy (EC). Individuals with IR decide their behavior independently of other group members. This is quite in contrast to individuals with EC strategy who react to others' emotional representation.

Because IR individuals behave as solitary ones even if they belong to a group of multiple individuals, their insensitivity should evolve to the same level of solitary ones;

$$x_{IR} = \hat{x}_1 = \frac{1}{2} - \sigma^2 \log c \tag{17}$$

(the same as (8)) and then they suffer the cost

$$C_{IR} = \hat{C}_1, \tag{18}$$

which is independent of strategies of other group members. Here, the probability that they react appropriately to a serious event (i.e.

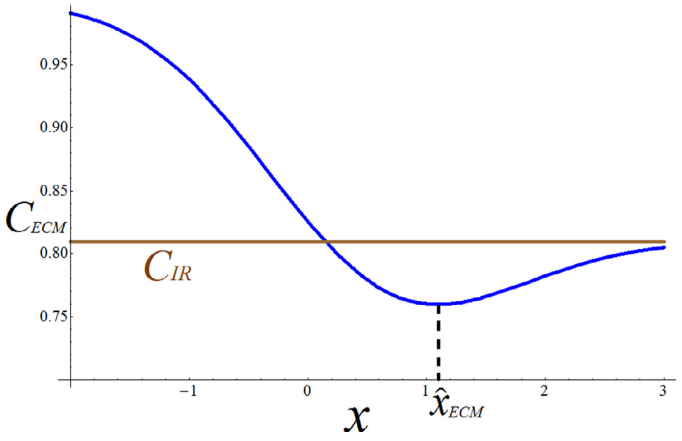

Fig. 4. The effect of insensitivity of an EC-mutant, x, on his/her total cost, C_{ECM}, is shown. C_{IR} is also shown for comparison. Parameters are $\sigma^2 = 1$, $c = 2$, and $n = 2$.

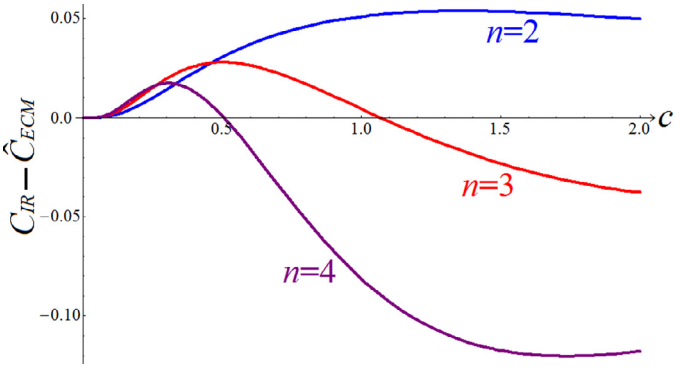

Fig. 5. The effect of the cost of serious events, c, on the difference in the total costs between IR-residents and an EC-mutant with the optimal insensitivity, $C_{IR} - \hat{C}_{ECM}$, is shown. When $C_{IR} - \hat{C}_{ECM}$ is positive, the EC-mutant can invade an IR population. Parameter is $\sigma^2 = 1$.

escaping danger) is

$$p_{IR} = \hat{p}_1 = \int_{y=\hat{x}_1}^{\infty} \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left[-\frac{(y-1)^2}{2\sigma^2}\right] dy \tag{19}$$

and that they show an inappropriate reaction to a trivial event (i.e. escaping from nothing) is

$$q_{IR} = \hat{q}_1 = \int_{y=\hat{x}_1}^{\infty} \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left(-\frac{y^2}{2\sigma^2}\right) dy. \tag{20}$$

In this subsection we will study evolutionary competition between IR and EC strategies.

Given that all IR individuals use the optimal threshold x_{IR}, let us first consider the invasion of IR into an EC population. For that purpose let us assume that the resident population uses EC and that individuals there live in a group of size n ($n \geq 2$). In such a group, EC individuals adopt the evolutionarily-selected optimal insensitivity $\hat{x}_n$, so that the cost they suffer is $\hat{C}_n$. In contrast, a typical IR mutant belongs to a group of size n where all the other $n-1$ members are EC individuals, and the cost that such an IR mutant suffers is C_{IR}. Because $C_{IR} = \hat{C}_1 > \hat{C}_n$ holds for all $n \geq 2$ (we saw this in the previous subsection), we conclude that IR-mutants cannot invade the EC population. Note that the existence of IR-mutants does not affect the (average) fitness (cost) of EC-residents because the frequency of IR-mutants in the population in this invasion analysis is assumed to be ignorable. Moreover, since the fitness of IR-mutants is independent of strategies of other group members, the conclusion that IR-mutants cannot invade the EC population still holds even if we consider the invasion of multiple mutants into a group due to, for example, positive assortment among mutant individuals.

Next, let us consider the opposite direction; invasion of EC into an IR population. When the group size is n (≥ 2), we can assume that a typical EC-mutant (with arbitrary insensitivity x) coexists with $n-1$ IR-residents in a group (later we will relax this assumption by considering some genetic relatedness between members in the same group). The cost that such an EC-mutant suffers is

$$C_{ECM} = c(1-p)(1-p_{IR})^{n-1} + 1 - (1-q)(1-q_{IR})^{n-1}. \tag{21}$$

Note that C_{ECM} is a function of x, possibly written as $C_{ECM} = C_{ECM}(x)$, because p and q are functions of x (see (1) and (2)). As shown in Fig. 4 and Appendix D, this cost is minimized when he/she has the optimal insensitivity,

$$\hat{x}_{ECM} = x_{IR} + (n-1)\sigma^2 \log\frac{1-q_{IR}}{1-p_{IR}}. \tag{22}$$

In the following we will consider whether this "best" EC-mutant with the optimal insensitivity $\hat{x}_{ECM}$ can invade the resident IR-population. The invasion conditions derived below should therefore be interpreted as the invasion potential of EC strategies into an IR-population at their best; if the EC strategy with the fine-tuned value of insensitivity $\hat{x}_{ECM}$ cannot invade the IR-population, then any other EC strategies with arbitrary insensitivity x cannot invade IR, either. Since IR-residents always suffer the cost C_{IR}, the "best" EC mutants can invade the population of IR when $\hat{C}_{ECM} < C_{IR}$, where $\hat{C}_{ECM} = C_{ECM}(\hat{x}_{ECM})$ is the cost that such mutants pay.

For $n = 2$, $\hat{C}_{ECM} < C_{IR}$ is always satisfied because $\hat{C}_{ECM} < C_{ECM}(\infty) = C_{IR}$ holds. Hence EC invades IR.

For $n \geq 3$, the invasion condition of EC into an IR population can be obtained only numerically, not analytically. Nevertheless we can derive its necessary condition. A straightforward calculation shows that

$$\begin{aligned} C_{IR} - C_{ECM} &= c(1-p_{IR}) + q_{IR} - [c(1-p)(1-p_{IR})^{n-1} + 1 \\ &\quad -(1-q)(1-q_{IR})^{n-1}] \\ &= c(1-p_{IR})\left[1-(1-p)(1-p_{IR})^{n-2}\right] \\ &\quad -(1-q_{IR})\left[1-(1-q)(1-q_{IR})^{n-2}\right] \\ &\leq c(1-p_{IR}) - (1-q_{IR})\left[1-(1-q_{IR})^{n-2}\right] \end{aligned} \tag{23}$$

holds, where we used $p \leq 1$ and $q \geq 0$ to show the last inequality. Therefore, a necessary condition is that the last line in (23) is positive. However, for large n, the last line in (23) approaches

$$c(1-p_{IR}) - (1-q_{IR}), \tag{24}$$

which is confirmed to be negative by a straightforward calculation. Therefore, EC-mutants cannot invade an IR population when n is large enough. Moreover, as shown in Appendix E, we can also prove that EC-mutants cannot invade an IR population when c is large enough. In other words, if IR is ancestral, a large group size and high cost of serious events prevent the invasion (initial evolution) of EC. Fig. 5 shows when $\hat{C}_{ECM} < C_{IR}$, that is, when EC can invade an IR population, for a given group size $n \geq 3$ and a given cost of committing a false negative error, c. We find that EC strategy can invade an IR population only when n and c are small.

In this model, we assume that EC individuals necessarily copy strong emotion when at least one group member shows it. If this strong assumption is relaxed to consider weaker emotional contagion, for example, if EC individuals observe only proportion r of other group members, the "effective" group size could decrease to $1 + r(n-1)$, so that EC-mutants may more easily invade an IR population, but the conclusion that a larger group size prevents the invasion of EC still holds.

So far, our invasion analysis has assumed that mutants are so rare that each mutant is surrounded by $n-1$ resident individuals in its group. Next we consider what if this assumption is vio-

lated. More specifically, we consider the effect of genetic relatedness within groups and assume that some groups comprise more than one mutant individuals. There are various ways to implement this assumption, but here we employ the distribution of mutants predicted by Wright's infinite island model. For simplicity we assume haploid genetics. We further assume that individuals in the same group interact with each other, but reproductive competition occurs globally in the whole population; we do not assume any local competition over reproduction. Emotional contagion occurs in the same way between relatives and between non-relatives in our model.

Consider the invasion condition of EC into an IR population (because invasion of the opposite direction has already been proved impossible in the argument above). The island model has a single parameter m ($0 < m \leq 1$) that determines the rate of migration. The smaller m is, the larger the average genetic relatedness R within groups. In Appendix F we derive how mutants are distributed over groups of size n for a given migration rate, m. When $m < 1$ (migration is not global), relatedness R becomes positive. As a result, some groups contain more than one mutants and hence the model deviates from the conventional situation that each EC-mutant is surrounded by $n-1$ IR-residents. For a smaller m, relatedness R is higher, and groups containing at least one EC-mutant tend to contain multiple EC-mutants. In the extreme case of $m \downarrow 0$, relatedness becomes maximal ($R \uparrow 1$), and the population consists of groups of n IR-residents and groups of n EC-mutants only. In this case, invasion success of EC-mutants is equivalent to that a group of EC individuals is more successful than a group of IR individuals.

Fig. 6A shows the costs of EC-mutants (with insensitivity x) and IR-residents for various values of average genetic relatedness R. Fig. 6B similarly shows the effect of group size n. EC-mutants with insensitivity x can invade an IR-population when $C_{ECM} < C_{IR}$. We find that although higher relatedness makes the invasion of EC-mutants easier, the invasion becomes more difficult as the group size increases.

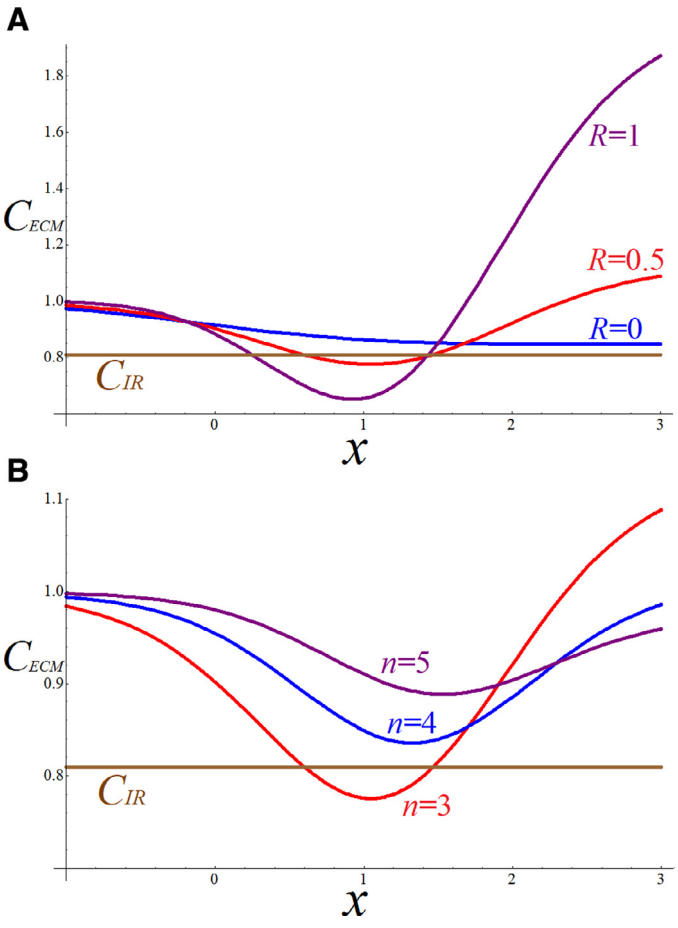

Fig. 6. (A) The effect of average genetic relatedness, R, on the total cost of an EC-mutant with insensitivity x, C_{ECM}, is shown. C_{IR} is also shown for comparison. (B) The effect of the group size, n, is similarly shown. Parameters are $\sigma^2 = 1$, $c = 2$, and $n = 3$ in (A) and $R = 0.5$ in (B).

3. Discussion

Since many animals form a group, it is important to know the merit of group living. In this study, we have considered the merit yielded by emotional contagion among group members, which has not been well studied before, although emotional contagion is frequently observed in group-living animals. Emotional contagion may help group members to escape from predators, to find food resource, and/or to struggle against outgroup individuals, which are frequently proposed merits of group living. However, emotional contagion may also induce harm to groups, such as via panic. Therefore, the relationship between emotional contagion and group living is not a simple problem.

We have considered a situation where individuals attempt to react appropriately to serious and trivial events that are indicated by environmental cues but cannot perfectly distinguish them. If individuals decrease the sensitivity to environmental cues, the probability of unnecessary reactions to trivial events (i.e. false positives) may decrease, but the chance that they fail to correctly react to serious events (i.e. false negatives) may increase. We have firstly considered solitary animals and shown that there is optimal sensitivity, which becomes higher as the cost of serious events increases, or as the cues become more ambiguous provided that the serious event is more risky than the trivial event. In the animal kingdom, physically weak animals (herbivores, subordinate males) tend to be more cautious than physically strong animals (carnivores, alpha males), and individuals pay larger attention to environments when the situation is unclear, which may be explained by our result.

Next, we have considered a group of individuals with the ability of emotional contagion, who copy the emotion of other group members that show unusual behavior induced by strong emotion. Here we have assumed for simplicity that when at least one group member shows strong emotion, all other group members copy it by emotional contagion. Empirical studies have shown that group living, or familiarity, among individuals is a key to the occurrence of emotional contagion (Langford et al., 2006; Gonzalez-Liencres et al., 2014). We have shown that a larger group favors lower sensitivity, which leads to higher fitness of individuals. This could explain why many group-living animals decrease the level of individual vigilance as the group size increases (Powell, 1974; Lima, 1995; Roberts, 1996).

Last, we have considered the evolution of emotional contagion by comparing the fitness of two types of individuals, those with emotional contagion (EC) and those who always react independently of others (IR). We have shown that IR-mutants cannot invade an EC population because EC individuals with appropriate sensitivity can form a group where every member suffers smaller cost than IR. We have, however, also shown that even if EC-mutants have appropriate sensitivity, they cannot invade an IR population if the group size or the cost of serious events is large. These results imply that large group size prevents the initial evolution of emotional contagion, but once it evolved, individuals suffer a smaller cost when they form a larger group. This conclusion is unchanged even if we consider weaker emotional contagion or genetic relatedness within groups, although these factors make the invasion of EC-mutants into an IR population easier. We have assumed there that emotional contagion occurs in the same manner

between relatives and between non-relatives. This is because it has been shown in rats that not close kinship but shared social experience such as being cagemates is a predictor of pro-social behavior induced by emotional contagion and empathy (Bartal et al., 2014).

Since many factors affect group formation, our results do not perfectly explain the origin of groups but provide many suggestions on the merit of group formation. For example, our model confirms a widely accepted view that group formation is adaptive for preys because the probability that at least one group member recognizes a predator increases (many-eyes hypothesis; Lima, 1990). However, our model also reveals a demerit of a large group that the probability that at least one group member erroneously reacts to safety signals also increases. To reduce the demerit, individuals should weaken their sensitivity to environmental stimuli. Some breeds of chickens often show panic behavior due to their high sensitivity, probably because they are raised in a larger group than the natural group of smaller size.

Our model also provides important suggestions on collective intelligence. When there are two options of which one is correct and the other is wrong, if the group follows the majority rule and each individual is more likely to select the correct one, the probability for the group to reach the correct decision increases as the group size increases, which is known as the Condorcet's jury theorem (Sekiguchi, 2016). Although our model similarly assumes two candidate behaviors (react or ignore) of which one is correct, each individual does not follow the majority rule in our model but rather copies unusual reactions of others via emotional contagion. Nevertheless, the probability of correct decision increases as the group size increases, provided that each individual weakens their sensitivity to environmental cues to reduce the probability of wrong reactions. Although employing majority rule greatly improves the accuracy of group-decision making, we believe that emotional contagion is much less cognitively demanding and therefore it should work as an effective strategy to take advantage of collective intelligence.

The model assumptions of this study are somewhat different from those of Nakahashi and Ohtsuki (2015) where adaptive significance of emotional contagion in dyadic interactions was studied in comparison with independently reacting individuals and those who adopt behavioral mimicry. Nakahashi and Ohtsuki (2015) modeled variety of behavior by one-dimensional real values, whereas our current study considered discrete actions, such as whether or not to react to environmental cues. In other words, appropriate emotion does not necessarily yield the best behavior in the previous model, which is not considered in this study. If this factor is included in the present model, emotional contagion may be less likely to evolve when the behavior induced by strong emotion differs largely from the best behavior. Moreover, Nakahashi and Ohtsuki (2015) assumed that an individual who recognized an environmental cue always reacts appropriately but the appropriate behavior differs between individuals. They found that emotional contagion is more likely to be adaptive than independent reaction when the appropriate behavior is more similar between individuals. In the current model, reactions are error-prone but the appropriate behavior (emotion) is the same among all group members. If we instead assume that the appropriate behavior (emotion) may differ among group members, emotional contagion is less likely to evolve because copying other's emotion would be less useful.

In conclusion, when individuals have to make a decision of whether to react to an environmental cue that potentially indicates serious events, they evolve to be more sensitive to the cue when the event is more risky. When individuals with emotional contagion form a group, their sensitivity to environmental cues evolve to be weak as the group size increases, and a larger group yields larger benefit to them. When individuals form a group but react to environmental cues independently of other group members, larger group size prevents the invasion of individuals with emotional contagion, but once every member adopts emotional contagion, individuals who react independently of others cannot invade. From these results we predict that large group size should not favor the evolution of emotional contagion, but that emotional contagion, once evolved, should favor the evolution of larger group size.

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Appendix A

From (4), (5), and (9), we have

$$\frac{dC_n}{dx}=\frac{n}{\sqrt{2\pi\sigma^2}}\left\{c(1-p)^{n-1}\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right]-(1-q)^{n-1}\exp\left(-\frac{x^2}{2\sigma^2}\right)\right\}, \tag{A.1}$$

so that C_n is minimized when

$$c(1-p)^{n-1}\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right]=(1-q)^{n-1}\exp\left(-\frac{x^2}{2\sigma^2}\right), \tag{A.2}$$

i.e., when

$$\begin{aligned}x&=\frac{1}{2}-\sigma^2\log c+(n-1)\sigma^2\log\frac{1-q}{1-p}\\&=\hat{x}_1+(n-1)\sigma^2\log\frac{1-q}{1-p}\end{aligned} \tag{A.3}$$

Since the line $x-\hat{x}_1$ is monotone increasing and the curve $(n-1)\sigma^2\log[(1-q)/(1-p)]$ is monotone decreasing (see below) in x, there exists a unique x that satisfies (A.3), which we denote as $\hat{x}_n$. Then, since $\log[(1-q)/(1-p)]$ is positive for all x because $p>q$, $\hat{x}_n$ increases as n increases. Note that $(1-q)/(1-p)$ is monotone decreasing in x because

$$\begin{aligned}&\frac{d}{dx}\left(\frac{1-q}{1-p}\right)\\&=\frac{1}{(1-p)^2\sqrt{2\pi\sigma^2}}\left\{(1-p)\exp\left(-\frac{x^2}{2\sigma^2}\right)\right.\\&\quad\left.-(1-q)\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right]\right\}\\&=\frac{1}{2\pi\sigma^2(1-p)^2}\int_{y=-\infty}^{x}\left\{\exp\left[-\frac{(y-1)^2}{2\sigma^2}-\frac{x^2}{2\sigma^2}\right]\right.\\&\quad\left.-\exp\left[-\frac{y^2}{2\sigma^2}-\frac{(x-1)^2}{2\sigma^2}\right]\right\}dy\\&=\frac{1}{2\pi\sigma^2(1-p)^2}\int_{y=-\infty}^{x}\exp\left(-\frac{x^2+y^2+1}{2\sigma^2}\right)\\&\quad\times\left[\exp\left(\frac{y}{\sigma^2}\right)-\exp\left(\frac{x}{\sigma^2}\right)\right]dy\\&<0\end{aligned} \tag{A.4}$$

Appendix B

Let us consider the evolution of x in an EC population using the invasibility analysis. Denote the insensitivity of residents as x_R and their reaction probabilities to serious and trivial events as p_R and q_R, respectively. Then, residents suffer the cost

$$C_R=c(1-p_R)^n+1-(1-q_R)^n, \tag{B.1}$$

and a mutant with insensitivity x suffers the cost

$$C_M = c(1-p)(1-p_R)^{n-1} + 1 - (1-q)(1-q_R)^{n-1}. \tag{B.2}$$

Therefore, when

$$C_R - C_M = c(p_R - p)(1-p_R)^{n-1} - (q_R - q)(1-q_R)^{n-1} \tag{B.3}$$

is positive, the mutant can invade. Since $C_M = C_R$ when $x = x_R$ and

$$\left.\frac{dC_M}{dx}\right|_{x=x_R} = \frac{1}{\sqrt{2\pi\sigma^2}}\left\{c(1-p_R)^{n-1}\exp\left[-\frac{(x_R-1)^2}{2\sigma^2}\right] - (1-q_R)^{n-1}\exp\left(-\frac{x_R^2}{2\sigma^2}\right)\right\}, \tag{B.4}$$

a mutant with insensitivity $x_R + \delta$ $(0 < \delta \ll 1)$ can invade when

$$c(1-p_R)^{n-1}\exp\left[-\frac{(x_R-1)^2}{2\sigma^2}\right] < (1-q_R)^{n-1}\exp\left(-\frac{x_R^2}{2\sigma^2}\right) \tag{B.5}$$

i.e., when

$$x_R < \hat{x}_1 + (n-1)\sigma^2 \log\frac{1-q_R}{1-p_R} \tag{B.6}$$

and that with $x_R - \delta$ $(0 < \delta \ll 1)$ can invade when these inequalities are reversed. From Appendix A, (B.6) is satisfied when $x_R < \hat{x}_n$, and the reverse is satisfied when $x_R > \hat{x}_n$, suggesting that insensitivity evolutionarily converges to $\hat{x}_n$. Note that once every resident adopts $\hat{x}_n$, no mutants with a slightly different insensitivity can invade because

$$\begin{aligned}\left.\frac{d^2C_M}{dx^2}\right|_{x=\hat{x}_n} &= \frac{1}{\sqrt{2\pi\sigma^2}}\left\{-2c\left(\hat{x}_n-1\right)\left(1-\hat{p}_n\right)^{n-1}\exp\left[-\frac{\left(\hat{x}_n-1\right)^2}{2\sigma^2}\right] + 2\hat{x}_n\left(1-\hat{q}_n\right)^{n-1}\exp\left(-\frac{\hat{x}_n^2}{2\sigma^2}\right)\right\} \\ &= \frac{2c\left(1-\hat{p}_n\right)^{n-1}}{\sqrt{2\pi\sigma^2}}\exp\left[-\frac{\left(\hat{x}_n-1\right)^2}{2\sigma^2}\right] > 0\end{aligned} \tag{B.7}$$

where $\hat{p}_n$ and $\hat{q}_n$ are reaction probabilities of individuals with insensitivity $\hat{x}_n$ to serious and trivial events, respectively.

Appendix C

The uniqueness of the crossing point of $x_A = \tilde{x}_A(x_B)$ and $x_B = \tilde{x}_B(x_A)$ can be proved as follows. From (14), we have

$$\tilde{x}_A'(x_B) = f(x_B) - f(x_B - 1) \tag{C.1}$$

where

$$f(x_B) = \sigma^2\exp\left(-\frac{x_B^2}{2\sigma^2}\right) \Big/ \int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy. \tag{C.2}$$

Since

$$f'(x_B) = -\frac{\exp\left(-\frac{x_B^2}{2\sigma^2}\right)\left[\sigma^2\exp\left(-\frac{x_B^2}{2\sigma^2}\right) + x_B\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy\right]}{\left[\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy\right]^2}, \tag{C.3}$$

$f'(x_B) < 0$ is always satisfied because

$$\begin{aligned}x_B\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy &> \int_{y=-\infty}^{x_B} y\exp\left(-\frac{y^2}{2\sigma^2}\right)dy \\ &= -\sigma^2\exp\left(-\frac{x_B^2}{2\sigma^2}\right)\end{aligned} \tag{C.4}$$

Moreover, we can show $f'(x_B) > -1$ as follows. Denoting

$$\begin{aligned}g(x_B) = &\left[\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy\right]^2 \\ &-\exp\left(-\frac{x_B^2}{2\sigma^2}\right)\left[\sigma^2\exp\left(-\frac{x_B^2}{2\sigma^2}\right) + x_B\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy\right],\end{aligned} \tag{C.5}$$

$f'(x_B) > -1$ is equivalent to $g(x_B) > 0$. Since $g(-\infty) = 0$, $g(x_B) > 0$ is satisfied if $g'(x_B) > 0$ i.e., if

$$h(x_B) = x_B\exp\left(-\frac{x_B^2}{2\sigma^2}\right) + \left(1 + \frac{x_B^2}{\sigma^2}\right)\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy > 0, \tag{C.6}$$

which is always satisfied because $h(-\infty) = 0$ and

$$h'(x_B) = \exp\left(-\frac{x_B^2}{2\sigma^2}\right) + \frac{x_B}{\sigma^2}\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy > 0. \tag{C.7}$$

Note that from (C.4),

$$\lim_{x_B\to-\infty} x_B\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy = 0 \tag{C.8}$$

and

$$\lim_{x_B\to-\infty} x_B^2\int_{y=-\infty}^{x_B}\exp\left(-\frac{y^2}{2\sigma^2}\right)dy = 0. \tag{C.9}$$

To sum up, we have $-1 < f'(x_B) < 0$, i.e., $-1 < \tilde{x}_A'(x_B) < 0$. Therefore, because of the symmetry, $x_A = \tilde{x}_A(x_B)$ and $x_B = \tilde{x}_B(x_A)$ have a unique crossing point where $x_A = x_B$ is satisfied.

Appendix D

From (4), (5), and (21), we have

$$\frac{dC_{ECM}}{dx} = \frac{1}{\sqrt{2\pi\sigma^2}}\left\{c(1-p_{IR})^{n-1}\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right] - (1-q_{IR})^{n-1}\exp\left(-\frac{x^2}{2\sigma^2}\right)\right\}, \tag{D.1}$$

so that C_{ECM} is minimized when

$$c(1-p_{IR})^{n-1}\exp\left[-\frac{(x-1)^2}{2\sigma^2}\right] = (1-q_{IR})^{n-1}\exp\left(-\frac{x^2}{2\sigma^2}\right), \tag{D.2}$$

i.e., when

$$\begin{aligned}x &= \tfrac{1}{2} - \sigma^2\log c + (n-1)\sigma^2\log\tfrac{1-q_{IR}}{1-p_{IR}} \\ &= x_{IR} + (n-1)\sigma^2\log\tfrac{1-q_{IR}}{1-p_{IR}}\end{aligned} \tag{D.3}$$

Appendix E

Using the notation of Appendix C, the condition for the last line of (23) to be negative, i.e. $c(1-p_{IR}) - (1-q_{IR})[1-(1-q_{IR})^{n-2}] < 0$, can be rewritten as

$$f(x_{IR}-1) > f(x_{IR})/\left[1-(1-q_{IR})^{n-2}\right], \tag{E.1}$$

i.e.,

$$(1-q_{IR})^{n-2} < 1 - f(x_{IR})/f(x_{IR}-1), \tag{E.2}$$

where we used the fact

$$c = \exp\left(-\frac{x_{IR}^2}{2\sigma^2}\right)/\exp\left[-\frac{(x_{IR}-1)^2}{2\sigma^2}\right]. \tag{E.3}$$

Since

$$\lim_{x_{IR}\to-\infty} f(x_{IR})/x_{IR} = -1, \tag{E.4}$$

and (C.8), when $n \geq 3$, we have

$$\lim_{x_{IR}\to-\infty} [1 - f(x_{IR})/f(x_{IR}-1)] = 0, \tag{E.5}$$

$$\lim_{x_{IR}\to-\infty} (1-q_{IR})^{n-2} = 0, \tag{E.6}$$

and

$$\lim_{x_{IR}\to-\infty} (1-q_{IR})^{n-2}/[1 - f(x_{IR})/f(x_{IR}-1)] = 0. \tag{E.7}$$

Therefore, EC-mutants cannot invade an IR population when x_{IR} is small enough, i.e., when c is large enough.

Appendix F

Suppose that the global frequency of mutants in the population is $\varepsilon\,(\ll 1)$. To derive how mutants are distributed over groups of size n, let $\phi_i\,(0 \leq i \leq n)$ be the proportion of groups that contain i mutants and $n-i$ residents. According to the assumption of the Wright's infinite island model that proportion $1-m$ of genetic contribution comes from the original group and the rest comes from a global gene pool, the equilibrium values of $\phi_i\,(0 \leq i \leq n)$ satisfy

$$\phi_i = \sum_{j=0}^{n} \phi_j \binom{n}{i} \left[(1-m)\frac{j}{n} + m\varepsilon\right]^i \left[(1-m)\frac{n-j}{n} + m(1-\varepsilon)\right]^{n-i} \tag{F.1}$$

which can formally solved, up to the first order of ε, as

$$\phi_0 = 1 - O(\varepsilon) \tag{F.2}$$

and

$$\phi_i = \psi_i\varepsilon + o(\varepsilon)\ (1 \leq i \leq n) \tag{F.3}$$

where $O(\varepsilon)$ and $o(\varepsilon)$ are Landau's symbols, and ψ_i does not include ε. Note that the global frequency of mutants, up to order ε, is

$$\sum_{i=1}^{n} \frac{i}{n}\phi_i = \left(\sum_{i=1}^{n} \frac{i}{n}\psi_i\right)\varepsilon, \tag{F.4}$$

which must be equal to ε, and therefore $\sum_{i=1}^{n} \frac{i}{n}\psi_i = 1$ must hold as a consistency condition. For example, for $n=3$ we have

$$\psi_1 = \frac{9m\left(4m^4 - 20m^3 + 39m^2 - 25m - 7\right)}{4m^5 - 20m^4 + 34m^3 - 4m^2 - 34m - 7} \tag{F.5}$$

$$\psi_2 = \frac{-9(m-1)^2 m\left(2m^2 - 6m + 7\right)}{4m^5 - 20m^4 + 34m^3 - 4m^2 - 34m - 7} \tag{F.6}$$

$$\psi_3 = \frac{(m-1)^3\left(4m^2 - 8m + 7\right)}{4m^5 - 20m^4 + 34m^3 - 4m^2 - 34m - 7}, \tag{F.7}$$

and it is not difficult to see that they satisfy the consistency condition. For large n, expressions of ψ_i's are very complicated, so we do not explicitly write them here. Average relatedness within groups is calculated as

$$R = \sum_{i=1}^{n} \frac{i-1}{n-1}\psi_i, \tag{F.8}$$

(Eq. (11) in Mullon et al. (2016)), which is a monotone decreasing function of m, ranging from $R=1$ ($m=0$) to $R=0$ ($m=1$).

Let $C_{ECM}^{(i)}$ be the cost that EC-mutants in a group of i EC-mutants and $n-i$ IR-residents suffer. Remember that it is a function of insensitivity x of EC individuals. Similarly to (21), it is given as

$$C_{ECM}^{(i)} = c(1-p)^i(1-p_{IR})^{n-i} + 1 - (1-q)^i(1-q_{IR})^{n-i}. \tag{F.9}$$

Then, the average cost that EC-mutants suffer, C_{ECM}, is the weighted average of $C_{ECM}^{(i)}$, calculated as

$$C_{ECM} = \sum_{i=1}^{n} C_{ECM}^{(i)} \frac{i}{n}\psi_i. \tag{F.10}$$

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