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ORIGINAL PAPER

Evolution of signal emission by uninfested plants to help nearby infested relatives

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Abstract Herbivory by arthropods often induces the emission of plant volatiles, which attract natural enemies of the herbivores. This induced emission of volatiles is considered to be a strategy of plants to effectively defend against herbivores by employing bodyguards. Recent empirical research has revealed that these volatiles can also affect neighboring undamaged plants and cause them to emit volatiles secondarily. Provided that signal emission imposes some cost on plants, the evolutionary advantage to undamaged plants in the emission of such secondary signals is unclear. We hypothesized that plants have evolved to emit a secondary signal to help nearby relatives by promoting the recruitment of natural enemies, whereby they increase inclusive fitness. We constructed a simulation model to evaluate this hypothesis. Our simulations suggest that a secondary signal evolves if the following five conditions are met: the cost of the signal is low; the potential risk of infestation is high; the attractiveness of the signal to natural enemies is highly positively correlated with the local density of the signal chemical; dispersal of offspring is spatially restricted, causing population viscosity, and; sufficient vacant space is available, allowing the population to be elastic.

Keywords Altruism · Eavesdropping · HIPV · Kin selection · SOS signal · Talking plants · Viscous population · Elasticity

Introduction

Herbivory by arthropods often induces the systemic release of volatile chemicals by the infested plant. These volatiles are called ‘‘herbivore-induced plant volatiles’’ (HIPV), and they attract natural enemies of herbivores such as predators or parasitoids (Takabayashi and Dicke 1996; Dicke and Vet 1999; Sabelis et al. 1999, 2002). HIPV benefits both the plants and the natural enemies of herbivores because it helps the natural enemies find prey,

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and this promotes the survival of the infested plants. Thus, HIPV realizes a close mutualistic relationship between the first and third trophic levels. From the viewpoint of plants, emission of HIPV is a defense strategy, and natural enemies are their bodyguards. In this sense, HIPV is sometimes referred to as an ''SOS signal''. Emission of such volatile chemicals has been reported in various plant species, including lima bean, cucumber, tomato, corn, and cabbage (for review, see Dicke 1999; Dicke and Vet 1999). Some authors have used mathematical models to theoretically assess conditions under which SOS systems are evolutionarily stable (Sabelis and De Jong 1988; Godfray 1995).

Prior experiments suggest that undamaged plants can release volatiles when exposed to HIPV emitted by other infested individuals, as though the receivers were imitating the behavior of the senders (Dicke et al. 1990; Bruin et al. 1992; Birkett et al. 2000). Experiments also suggest that an undamaged plant whose rhizosphere is exposed to water-soluble info-chemicals exuded from the rhizosphere of an infested plant can release volatiles from its aerial parts (Chamberlain et al. 2001; Dicke and Dijkman 2001; Guerrieri et al. 2002). Thus, even undamaged plants can release signals in the presence of infested neighbors. If this trait has been established by natural selection, its cause is a challenging puzzle. Here we propose a hypothesis to address the evolution of such a secondarily released signal. Other hypotheses are discussed elsewhere (Kobayashi and Yamamura 2003; Kobayashi et al. 2006; see also Discussion).

Our hypothesis relies on two assumptions. First, we assume that the effectiveness of signals in calling predators is positively correlated with the local number of signalers. In other words, a signaling plant with signaling neighbors can attract predators more effectively than a plant without signaling neighbors can. This assumption would be plausible, for example, if signals from the neighbors act as guideposts, leading predators to the targets. It is likely that a dense signal from multiple individuals is easier to find than a less dense signal from a single individual. The second assumption is that spatially neighboring plants are genetically related. This would occur in ''viscous'' populations, in which genes are not well mixed because of spatially limited dispersal of offspring (Hamilton 1964).

From the first assumption, an uninfested plant near infested individuals can promote the recovery of its neighbors by signaling together. The second assumption leads to the improvement of the indirect fitness of the uninfested plant. The inclusive fitness is the sum of the direct and indirect fitness. Therefore, from the theory of kin selection, plants can evolve to emit a signal in the presence of infested neighbors to improve their inclusive fitness. We assume that the genetic correlation between neighboring plants is caused by population viscosity. Therefore, the problem essentially involves the evolution of altruism in viscous populations, which is an elaborately explored topic in evolutionary ecology (for review, see e.g. Queller 1992; West et al. 2002; see also below). In the next section, we describe a computer simulation model to confirm the validity of this hypothesis.

Although neighboring individuals tend to be genetic relatives in viscous populations, at the same time, they are competitors for local space. Therefore, the altruistic behavior benefiting neighbors inevitably has two antagonistic effects. First, it increases the indirect fitness of the actor by benefiting its relatives, and second, it decreases the direct fitness of the actor by benefiting its competitors. Most initial studies on altruism in viscous populations failed to realize the evolution of altruism (Taylor 1992a; Wilson et al. 1992). This was because the effect of increasing local competition counterbalanced the effect of benefiting relatives (see also van Baalen and Rand 1998). We can state this problem in a different way by supposing two discrete strategies, altruism and selfishness, as follows. Population viscosity makes each strategy form spatial clusters. Since altruists benefit neighbors, the clusters of altruistic individuals can produce more offspring than those of

selfish individuals; however, they cannot force increased offspring out of the cluster because of the low dispersal propensity. Thus, in clusters of altruists, competition is more intense than in those of selfish individuals, and this negative effect can cancel out the positive effect of the increased number of offspring (Taylor 1992a, b; Wilson et al. 1992; see also a detailed analysis on the cancellation by Gardner and West 2006).

However, some succeeding works have successfully found situations in which population viscosity can drive the evolution of altruism. Taylor and Irwin showed that generation overlap can favor altruism (Taylor and Irwin 2000; Irwin and Taylor 2001). Wilson et al. (1992) suggested that ''budding viscosity'', in which dispersal is of groups rather than of individuals, can favor altruism, and this suggestion was confirmed numerically by Goodnight (1992) and analytically by Gardner and West (2006). Taylor (1992b) considered ''elastic populations'', in which there are some vacant sites and the population size can grow with the fecundity of individuals. He showed that altruism can evolve in an elastic viscous population because vacant sites buffer competition for space (see also Mitteldorf and Wilson 2000). In other words, altruists can enjoy the benefit of increased offspring by pushing the offspring into nearby vacant sites. Yamamura et al. (2004) also observed the same effect of vacancies in their model of the evolution of inter-specific mutualism in a viscous population. Studies of Prisoner's Dilemma games in structured populations adopted slightly different selection criteria from those of Wilson et al. (1992) and Taylor (1992a, b) to enable the spread of altruism: For example, in the two-dimensional lattice model of Nowak and May (1992), each local site is replaced by the type with the highest ''score'' among the immediate neighbors, so that there is no chance for other types to occupy the site. In this deterministic model, the ''cooperator'' can spread in the population of the ''defector'', when clusters of the cooperator initially have certain shapes; the cooperator and the defector can even coexist, forming complex and interesting spatial patterns. Nowak et al. (1994) considered a more realistic model in which new site occupants are stochastically determined according to the scores of neighbors, and they showed that the cooperator can still spread in the population of the defector. However, this is because each individual plays Prisoner's Dilemma games not only with its neighbors but also with itself, from which the cooperator can always gain a significant amount of benefit. Therefore, the net effect of a cooperator's behavior on its score may be positive, which means the behavior is not really ''altruistic'' but rather ''mutually beneficial'' (West et al. 2007). Indeed, they clearly state that the cooperator cannot be maintained without self-interaction in a purely stochastic model (see Nowak et al. 1994, pp. 4879, right column). Nakamaru et al. (1997, 1998) considered an iterated Prisoner's Dilemma game on lattice-structured space. In their model, the death of individuals is unsynchronized and a dead individual is immediately replaced by one of immediate neighbors. Prisoner's Dilemma games are played between immediate neighbors. Since a dying individual cannot leave any offspring to compete for the current site, competition for space does not occur between individuals playing the same game; thus, the evolution of cooperation is promoted. See e.g. Doebeli and Knowlton (1998), Killingback et al. (1999), Koella (2000), and Tainaka and Itoh (2002) for other examples of spatially explicit Prisoner's Dilemma games. Importantly, although these studies adopted various selection criteria, the results do not contradict Taylor (1992a) and Wilson et al. (1992).

We show that the counterbalance between the effect of increasing competition and that of benefiting relatives also occurs in our model. We also show that this problem is overcome by assuming population elasticity (Taylor 1992b; Mitteldorf and Wilson 2000). Our primary aim in this study was to show that a secondary signal evolves by kin selection. At the same time, however, our hypothesis is an interesting application of the theory of

altruism in viscous populations. Our model is quite different from previous models of the evolution of altruism, especially in terms of population structure and the mode of interaction. Therefore, our results corroborate the effectiveness of viscosity and elasticity in the evolution of altruism.

Model

We consider an asexual plant population with a square lattice structure. Major notation is summarized in Table 1. In many lattice models in evolutionary ecology, each cell is assumed to have at most one individual. In this study, we instead assume that each cell is a patch with N sites, and each site can have at most one plant individual, but may also be empty. Each generation consists of five steps: attack, recovery, reproduction, seed dispersal, and competition for space.

Every generation starts with the attack step in which each individual plant is attacked by herbivores with probability h. In reality, herbivores may be likely to migrate from an infested plant to adjacent plants. It is known that secondary signaling can evolve without kin selection under such spatial effect (Kobayashi and Yamamura 2003; Kobayashi et al. 2006; see also Discussion). In the present model, we exclude this spatial effect regarding herbivore attack, because our aim is to investigate whether and when secondary signaling can evolve purely by kin selection. We assume two phenotypes: primary and secondary signalers. Primary signalers signal only when infested, while secondary signalers signal not only when infested, but also when the number of infested patch mates is larger than a specific threshold n^*, where $1 \leq n^* \leq N-1$. Each signaling individual incurs the cost S of the signal.

The next step is recovery. Each patch is visited by predators with probability $V(n_s)$, which is an increasing function of the number of signaling individuals n_s in the patch. Since patches with no signaling individuals have no infested individuals, they are irrelevant to recovery; therefore, $V(n_s)$ is defined only for $N \geq n_s \geq 1$ and not for $n_s = 0$. For simplicity, we assume that $V(n_s)$ is a linear function with the minimum value $V(1) = r$ and the maximum value $V(N) = R$, where $1 \geq R > r \geq 0$. These assumptions lead to the following function form:

$$V(n_s) = (R - r)\frac{n_s - 1}{N - 1} + r, \tag{1}$$

Table 1 Notation

N	Number of sites per patch
H	Probability of being attacked by herbivores
n_s	Local number of signaling individuals
n^*	Signaling threshold of secondary signaling
S	Cost of signaling
d	Cost of damage
$V(n_s)$	Recovery probability in a patch with n_s signaling individuals
R	Maximum recovery probability
r	Minimum recovery probability
m	Migration rate
ε	Elasticity

If a patch obtains some predators, all infested individuals in the patch recover. Unlucky individuals that cannot recover in this step incur the cost d of damage, while those that recover do not incur any cost due to damage. Note that the recovery probability takes only the maximum (R) and minimum (r) values under $N = 2$.

The recovery step is followed by asexual reproduction. The number of seeds (fecundity) produced by a plant reduces according to the costs that the plant incurred during the attack and recovery steps. Without loss of generality, we assume that the baseline fecundity for a plant without any costs is given by 1. Accordingly, the fecundity of a plant that incurred the signaling cost but not the damage cost is given by $1 - S$, while the fecundity of a plant that incurred both the signaling and damage costs is given by $1 - S - d$. Note that infested individuals always signal regardless of their phenotypes, so that the damage cost (d) is always accompanied by the signal cost (S), but not vice versa.

As mentioned above, secondary signalers emit a signal in the presence of infested neighbors, even when they are not themselves infested. They incur the cost of signal emission (S), while their infested patch mates can obtain the benefit of recovery. Thus, the secondary and primary signalers are altruistic and selfish, respectively, if the signal cost is positive ($S > 0$). On the other hand, if the cost is negative ($S < 0$), secondary signaling is beneficial to both the signaler and the patch mates; therefore, secondary signaling is selfish or ''mutually beneficial'' (West et al. 2007). In this case, the primary signalers are ''spiteful'' (Hamilton 1970; Gardner and West 2004), since they are refusing to help neighbors at the expense of a cost ($-S$).

In the step of seed dispersal, the fraction $1 - m$ of seeds remains on the native patch, while the other is equally dispersed to each of the four adjacent patches in the two dimensional grid; that is, each neighboring patch receives the fraction $m/4$ of seeds from the focal patch.

The last step is competition for habitat. All parents die and new owners of sites are chosen at random from the seeds in the local patch by competition. We let A and B denote the numbers of seeds of secondary and primary signalers, respectively, in a specific patch. A and B include not only native seeds, but also immigrants from neighboring patches. We assume that the probability that a site is occupied by a plant increases with the local seed number ($A + B$), but in a saturating manner. We also assume that the probability is zero when the seed number is zero. To suffice these assumptions, we chose a simple saturating function $(A + B)/(A + B + \varepsilon)$ as the probability of site occupation, where ε is a so-called ''half-saturation constant'', i.e. the number of seeds that renders the probability of occupation 0.5. Moreover, we assumed that the conditional probability that an occupied site is owned by a phenotype is proportional to the fraction of that type in the local seed pool. Thus, in the next generation, each site is occupied by a secondary and primary signaler with probabilities

$$\left(\frac{A}{A+B}\right)\frac{A+B}{A+B+\varepsilon} = \frac{A}{A+B+\varepsilon}, \tag{2a}$$

and

$$\left(\frac{B}{A+B}\right)\frac{A+B}{A+B+\varepsilon} = \frac{B}{A+B+\varepsilon}, \tag{2b}$$

respectively. A site becomes vacant if it is not occupied by any phenotype, which occurs with probability

$$1 - \frac{A + B}{A + B + \varepsilon} = \frac{\varepsilon}{A + B + \varepsilon}. \quad (2c)$$

According to Taylor (1992b), the population is said to be elastic when the probability of site occupation is less than one (see Taylor 1992b, pp. 300, left column); therefore, in our model, the population is elastic when $\varepsilon > 0$. Indeed, as long as $\varepsilon > 0$, there should be some vacant sites, and the number of occupied sites in the population should be positively correlated with the number of seeds, realizing population elasticity (Taylor 1992b; Mitteldorf and Wilson 2000). Taylor (1992b) defined population elasticity as the derivative of $\ln(q)$ with respect to $\ln(x)$, where q is the probability of site occupation and x is the local seed number. According to this definition, in our model, elasticity is given by $\varepsilon/(A + B + \varepsilon)$, which is exactly equivalent to the probability of vacancy (Eq. 2c). This probability is positively correlated with ε unless the number of seeds is zero; therefore, we refer to ε as elasticity for simplicity hereafter. The situation $\varepsilon > 0$ occurs for example when seeds have to struggle against not only other seeds but also against a harsh environment to occupy a site. After the establishment of the new generation, the cycle begins again with the attack step.

In most simulations, we initially let each site be occupied at random by a secondary or primary signaler, each with a probability of 0.5. Thus, there are initially no vacant sites. The effect of the initial frequency is briefly discussed in the Discussion section. In all simulations, the lattice includes 50×50 patches with $2{,}500 \times N$ individuals. We assumed a periodic boundary condition to eliminate the effects of edges, so that the lattice had a torus-like form.

Results

Figure 1 shows the dynamics of the number of secondary signalers in the population for several simulations with different values of the signal cost (S), where elasticity (ε) is 0, so that there are no vacant sites. Note that secondary signalers quickly decrease in number and go extinct under positive signal costs ($S > 0$). We experimentally ran some simulations under negative costs ($S < 0$), which implies that plants obtain some additional benefits by signaling. As soon as the signal cost (S) becomes slightly lower than zero, secondary signalers quickly increase to be fixed in the population. These results suggest the counterbalance between the positive effect of benefiting relatives and the negative effect of intensifying competition. That is, the secondary signal can evolve only if it is directly beneficial to the signaler itself. However, it should also be noted that secondary signalers go extinct even when the signal cost is zero ($S = 0$) (Fig. 1), suggesting that the effect of competition may slightly exceed the counterbalance.

Figure 2 shows the effects of the signal cost (S) (2a, b) and elasticity (ε) (2c) on the numbers of individuals of both phenotypes after a long time (3.0×10^4 generations). Each bar shows an average over ten simulations. When we increased or decreased the parameter values, transitions occurred between three phases: the phase in which primary signalers tend to be fixed (hereafter referred to as the *PS* phase), the phase in which secondary signalers tend to be fixed (*SS* phase), and the phase in which both phenotypes are not viable and the population goes extinct (*V* phase). Whether and when such a transition occurs

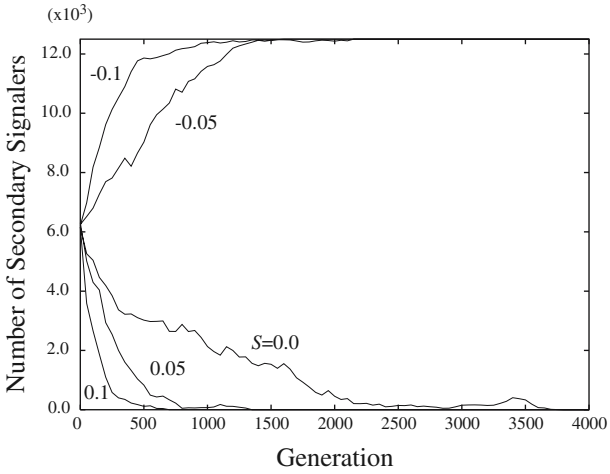

Fig. 1 Dynamics of the number of secondary signalers in the population for several simulations with different values of the signal cost (S). Parameter values are $h = 0.6$, $d = 0.7$, $\varepsilon = 0.0$, $m = 0.1$, $n^* = 3$, $R = 1.0$, $r = 0.2$, and $N = 5$. Numbers beside the lines represent the values of S

depended on the values of parameters other than the focal parameter, but the direction of transition was generally invariable.

We sometimes observed a narrow transient phase between an *SS* phase and a *PS* phase. In this transient phase, fixation requires many generations, and both phenotypes can be fixed. This is because the selective difference between the primary and secondary signalers is very small, and the fate of the population is mainly determined by random genetic drift.

Figure 2a clearly shows the counterbalance between the two antagonistic effects in an inelastic population ($\varepsilon = 0$). The phase transition from an *SS* phase to a *PS* phase occurs at a signal cost (S) slightly smaller than zero, suggesting that the effect of intensifying competition slightly exceeds the effect of benefiting relatives. Remember that the secondary signalers are selfish and the primary signalers are spiteful when the signal cost is negative ($S < 0$), as mentioned above. Figure 2a suggests that a spiteful strategy can evolve in our model, though the cost of spite ($-S$) must be quite small ($-S < 0.015$ in Fig. 2a). Thus, the exact counterbalance, which was found in Taylor's (1992a) model and analyzed more in detail by Gardner and West (2006), might not be an absolute principle in viscous populations, as pointed out by Mitteldorf and Wilson (2000) (see Discussion).

The dilemma caused by antagonistic effects in viscosity is successfully overcome under population elasticity. The transition from an *SS* phase to a *PS* phase occurs at a positive signal cost ($S > 0$) in elastic populations ($\varepsilon > 0$) (Fig. 2b). This is because the clusters of secondary signalers force many seeds into vacant sites, and quickly spread into sparse areas compared to clusters of primary signalers, as in Mitteldorf and Wilson's (2000) model. Since individuals of both phenotypes release a signal when infested, signal costs that are too high inevitably lead to a decrease in the average fitness of the population. Thus, the population loses viability and goes extinct when the cost increases beyond a threshold; in other words, the phase transition from a *PS* phase to a *V* phase occurs (Fig. 2b). We can confirm the effect of elasticity from Fig. 2c. Secondary signalers are favored in a highly elastic population (high ε), in which many vacant sites are maintained.

Phase transitions also occurred when we changed the values of parameters other than the signal cost (S) and elasticity (ε), and the results are shown in Fig. 3 (see Discussion).

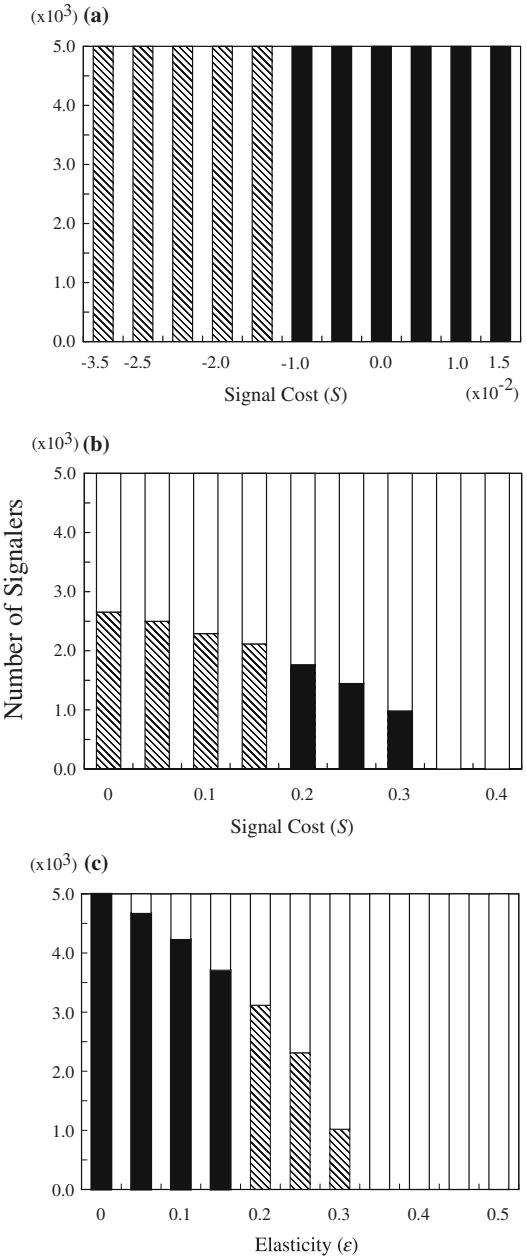

Fig. 2 Effects of the signal cost (S) (3a and 3b) and elasticity (ε) (3c) on the numbers of secondary and primary signalers after a long time (3.0×10^4 generations). Each bar shows an average over ten simulations. Black bars show the number of primary signalers; striped bars show the number of secondary signalers. $d = 0.5$, $h = 0.5$, $R = 0.8$, $r = 0.2$, $n^* = 1$, $N = 2$. Other parameter values are (**a**) $\varepsilon = 0.0$, $m = 0.2$, (**b**) $\varepsilon = 0.3$, $m = 0.2$, (**c**) $S = 0.1$, and $m = 0.1$

Discussion

The evolution of altruism in viscous populations was first suggested by Hamilton (1964), first systematically investigated by Taylor (1992a) and Wilson et al. (1992), and has been elaborately explored by various modelers (Taylor 1992b; Goodnight 1992; Nowak and May 1992; Queller 1992, 1994; Nowak et al. 1994; Nakamaru et al. 1997, 1998; Doebeli

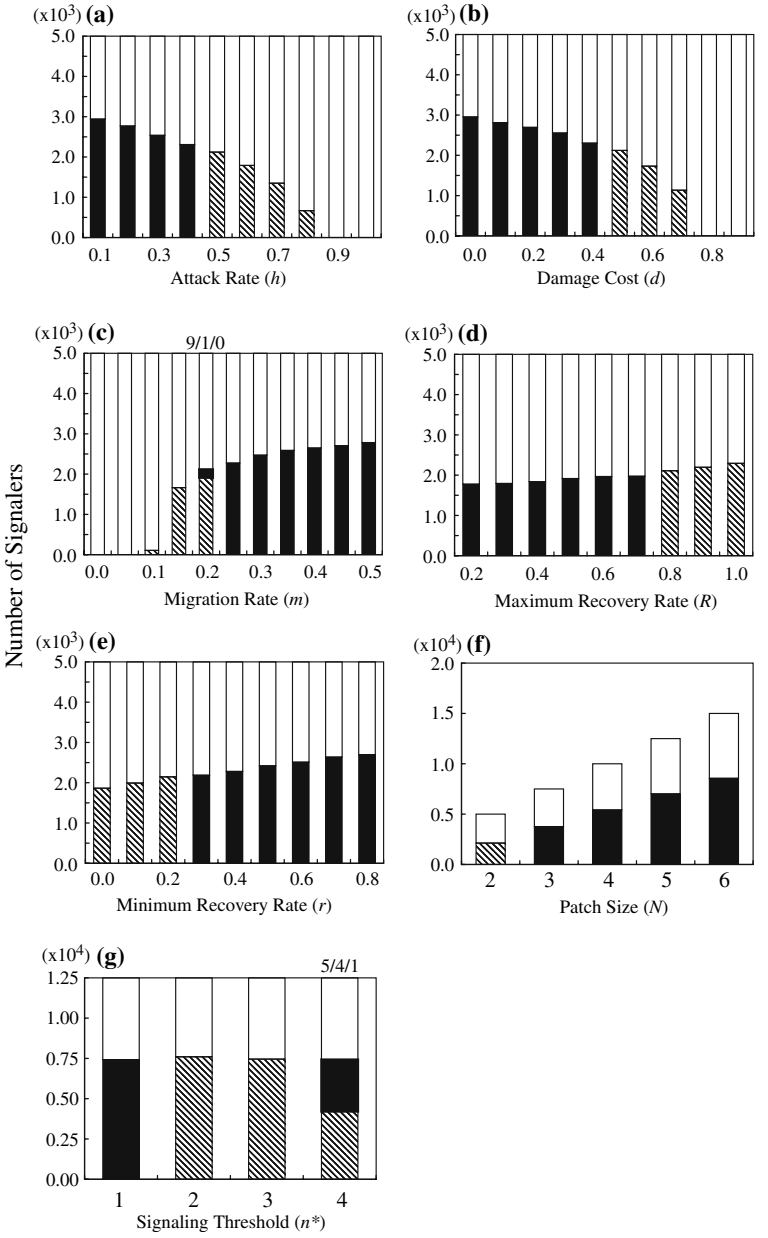

Fig. 3 Effects of (**a**) the attack rate (h), (**b**) the damage cost (d), (**c**) the migration rate (m), (**d**) the maximum recovery rate (R), (**e**) the minimum recovery rate (r), (**f**) the patch size (N), and (**g**) the signaling threshold (n^*) on the number of secondary and primary signalers after a long time (3.0×10^4 generations). Each bar shows an average over ten simulations. Black bars show the number of primary signalers; striped bars show the number of secondary signalers. Three digits ($i/j/k$) on some bars mean that the simulations ended up with fixation by the secondary signaler i times, with fixation by the primary signaler j times, and with a polymorphic state k times. Default parameter values are $S = 0.15$, $\varepsilon = 0.2$, $h = 0.5$, $d = 0.5$, $m = 0.2$, $R = 0.8$, $r = 0.2$, $N = 2$, and $n^* = 1$. In panel (g), $N = 5$, $S = 0.02$, and $r = 0.1$

and Knowlton 1998; van Baalen and Rand 1998; Killingback et al. 1999; Koella 2000; Mitteldorf and Wilson 2000; Tainaka and Itoh 2002; Yamamura et al. 2004; Gardner and West 2006). Aided by computer simulations or special approximation techniques applicable to lattice models (Matsuda et al. 1992), these studies have yielded valuable information. Based on this knowledge, we investigated the evolution of secondary signal emission by plants, which is a prevalent topic in chemical ecology. By analyzing a simulation model, we have shown that plants can evolve to emit costly signals to help infested neighbors in a viscous population. Population elasticity is essential to this evolution. Indeed, in a viscous population, neighbors are likely to be genetic relatives, but they are competitors for space at the same time. Therefore, for secondary signalers to spread in the population, there must be vacant space to buffer the competition, as expected from prior models of evolution of altruism in elastic viscous populations (Taylor 1992b; Mitteldorf and Wilson 2000; Yamamura et al. 2004). Otherwise, the effect of benefiting relatives is counterbalanced by the effect of intensifying competition, and altruism cannot evolve, as shown by prior analytical works (Taylor 1992a, b; van Baalen and Rand 1998; Gardner and West 2006) and numerical works (Wilson et al. 1992; Mitteldorf and Wilson 2000).

Thus, a secondary signal is favored in highly elastic populations according to our model. The most significant point is that the population can grow in size by cooperation. That is, in order for altruism to evolve, the population must be prevented by some outer stress from reaching its carrying capacity. Without outer stress such as predation, starvation, aridity, or competition for resources with other species, the species is spoiled and cannot develop altruistic traits. However, environments that are too severe will drive the population to extinction. Thus, altruistic behavior may arise particularly on the verge of population or species extinction. It is noteworthy that vacant sites may not be literally vacant. Instead, they may be occupied by some other species that do not benefit from the altruistic behavior. In this case, the individuals of one species are the common enemies of another species. Interspecific competition may buffer intraspecific competition, promoting altruism. This possibility should be investigated in future work.

Mitteldorf and Wilson (2000) numerically showed that the exact counterbalance can break under ''strong selection'', where the benefit and cost of altruism are not small, and altruism can be favored. On the other hand, spite can be favored in our model. The reason for this is not clear from the present numerical analyses. We suspect that variation in fecundity among individuals and/or among patches due to stochastic herbivore attack may be responsible for the spread of spite, because this fecundity variation is the most conspicuous difference between our model and Taylor's one. Such variation might affect the relatedness among interacting individuals and break the counterbalance. However, the effect of fecundity variation on the evolution of spite or altruism should be investigated more in detail in future works, using simpler models. Despite these, note that the counterbalance roughly holds in our model; i.e., the cost ($-S$) must be very small for the evolution of spite.

Figures 2 and 3 show how phase transition occurs when various parameters are changed. Note that transition occurs as ($V\rightarrow$) $SS \rightarrow PS$ with increasing population size in most cases. This is because there are many vacant sites in a small population, so that secondary signalers, which can exploit vacant areas more efficiently than primary signalers, fare better. However, phase transition occurs as $PS \rightarrow SS$ with increasing the population size, when the maximum recovery probability (R) is changed (Fig. 3d). Although the vacant space reduces with increasing the maximum recovery probability, the effectiveness of the signal also increases; probably, this latter effect overcomes the former effect, changing the order of phase transition.

The effect of the signaling threshold (n^*) is a little complicated. As it increases, the expected number of relatives that a secondary signaler can help increases, so that the secondary signaling may become favored. However, at the same time, a secondary signaler rarely emit a secondary signal when the signaling threshold (n^*) is large; thus, the selective difference between the two strategies reduces. These two effects are clearly shown in Fig. 3g.

In all the simulations that we conducted above, we assumed that the initial frequency of the secondary signaler is 0.5. One may wonder how the results are affected by the initial frequency, because it is relevant to the invasibility of a strategy to the other. Part of the results of such analyses is shown in Fig. 4. In Fig. 4a, b (4c, d), all parameter values are the same as in Fig. 2a (2b), except for the initial frequency of the secondary signaler; the initial frequency is 0.1 in Fig. 4a, c, while 0.9 in Fig. 4b, d. The boundary between the *SS* phase and the *PS* phase becomes less clear than when the initial frequency is 0.5. This is because a rare phenotype is likely to be lost by random drift, if selection is nearly neutral. However, the figure shows that our major conclusion is not qualitatively affected by the initial frequency; i.e., the secondary signaler becomes more favored as the signal cost becomes lower, and also, elasticity enables the evolution of the secondary signaling under positive signal costs. Of course, if the initial frequency is even closer to 0 or 1, the rare phenotype is

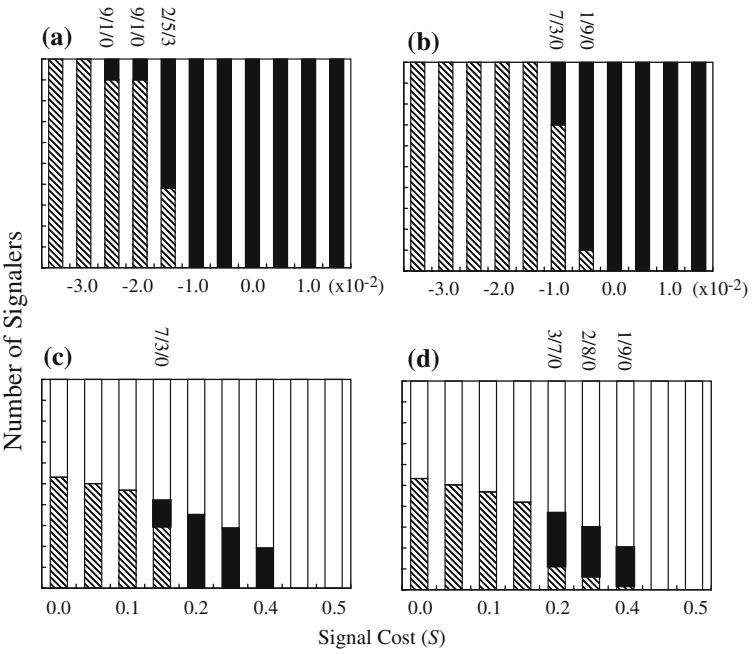

Fig. 4 Effects of the signal cost (S) on the number of the secondary and primary signalers after a long time (3.0×10^4 generations). Each bar shows an average over ten simulations. Black bars show the number of primary signalers; striped bars show the number of secondary signalers. Three digits ($i/j/k$) on some bars mean that the simulations ended up with fixation by the secondary signaler i times, with fixation by the primary signaler j times, and with a polymorphic state k times. The initial frequency of the secondary signaler is 0.1 in (a) and (c), and 0.9 in (b) and (d). The population is inelastic ($\varepsilon = 0$) in (**a**) and (**b**), and elastic ($\varepsilon = 0.3$) in (**c**) and (**d**). Other parameter values are $d = 0.5$, $h = 0.5$, $R = 0.8$, $r = 0.2$, $n^* = 1$, $N = 2$, and $m = 0.2$

almost always lost by random drift and it becomes very difficult to evaluate the selective advantage of a phenotype over the other.

Here, we hypothesized that kin selection is the evolutionary cause of signal emission in the presence of infested neighbors. We call this the ''kin-selection hypothesis''. There are at least two more hypotheses that can explain the evolution of signal emission of secondary signals. One of these is the ''pre-attack extermination hypothesis'', which was proposed by Kobayashi and Yamamura (2003). According to the pre-attack extermination hypothesis, signal emission by uninfested plants is a strategy to reduce the risk of future attacks by herbivores. Kobayashi and Yamamura (2003) assumed that plants with infested neighbors are more likely to be attacked in the near future than those without. They also assumed that uninfested plants can contribute to the extermination of herbivores infesting neighbors by emitting signals. From these assumptions, they hypothesized that plants have evolved to emit secondary signals to eliminate the herbivores infesting neighboring plants, and thereby reduce future risk. They investigated conditions under which such a strategy can evolve using a game-theory model. The other hypothesis is the ''pre-attack protection hypothesis'' (Kobayashi et al. 2006). The pre-attack protection hypothesis considers that the function of secondary signals is to attract and keep predators prior to herbivore attacks (Kobayashi et al. 2006). If predators cannot respond quickly to a signal, it would be too late to signal after the occurrence of herbivory. In this case, signal emission prior to attacks would be useful to reduce risk.

Importantly, a secondary signaler obtains direct benefits under the pre-attack extermination hypothesis and the pre-attack protection hypothesis, where secondary signaling is not considered altruistic. Although the three hypotheses, i.e., the kin-selection hypothesis, the pre-attack extermination hypothesis, and the pre-attack protection hypothesis, all assume different mechanisms, they are not exclusive. They can work together to promote the evolution of signal emission by uninfested plants in the presence of infested neighbors. In other words, the direct benefits from pre-attack extermination and pre-attack protection can partly compensate or even outweigh the immediate cost of secondary signaling, promoting the evolution of secondary signaling that helps neighboring relatives. It would be interesting to investigate such a complex situation in future works from the viewpoint of the kin selection theory.

There is also a non-evolutionary way of explaining secondary emission of volatiles. Volatiles emitted by infested individuals may adhere to the surface of leaves of neighboring uninfested individuals and gradually re-diffuse into the air (Choh et al. 2004). In this case, the emission of volatiles by uninfested plants may be a trivial physical phenomenon, rather than a strategy per se. However, it is also known that plants can emit signals from aerial parts when their rhizospheres are exposed to the chemicals exuded from the rhizospheres of other infested plants. The explanation by adhesion would not be applicable to such plant–plant communication through the rhizosphere. Furthermore, the surfaces of plant leaves may be under selection, and even adhesion and re-diffusion of volatiles can be a strategy of plants. In this case, the above-mentioned evolutionary hypotheses would be applicable.

Recent studies have shown that chemical communication is possible even between heterospecific plants (e.g., Karban et al. 2000; Karban 2001; Karban and Maron 2002; Karban et al. 2003). Although the kin-selection hypothesis assumes interaction between conspecifics, a similar logic would be applicable to interaction between heterospecifics. Yamamura et al. (2004) showed that interspecific mutualism can evolve in a viscous and elastic population (see also, Doebeli and Knowlton 1998). Therefore, viscosity with

elasticity may also promote the evolution of secondary signals between heterospecifics, although further modeling is required to confirm this.

Our evolutionary and physiological understanding of chemical communication in plants is still far from sufficient. We need more empirical investigations to elucidate how plants communicate, how widespread such a phenomenon is in the plant kingdom, and what results from it. We hope that evolutionary models will stimulate further development of this research area.

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