

REPORT

Community structure and the evolution of aposematic coloration

Abstract

Studies on the evolution of aposematic coloration (prey coloration advertising for unpalatability) have mainly focused on predator psychology in simplified single-prey species systems. We chose, instead, to model population dynamics on the community level. We studied the invasion by an aposematic phenotype in the presence and absence of another prey species. The single-prey and two-prey models differed in two major ways. First, with two prey species the invasion was possible only with a weak aposematic signal, whereas with a single prey species there was no such an upper limit for signal strength. Second, with a single prey species, increase of the aposematic phenotype always resulted in rapid extinction of the predator. Resource value and growth rate of the alternative prey species affected the invasion. These results suggest that community structure is an important determinant of the conditions for invasion of aposematism, and may have contributed to its initial evolution.

Keywords

Aposematism, multi-prey model, population dynamics, predation.

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INTRODUCTION

Aposematism is an anti-predator defence, combining a warning signal, usually a bright coloration, with unpalatability (Cott 1940; Edmunds 1974). The evolution of aposematism has largely remained an intriguing paradox (Guilford 1988, 1990; Endler 1991). An aposematic coloration always attracts the attention of predators but only when aposematic individuals have become common will predators effectively learn to associate the coloration with the unpalatability of the aposematic prey. Therefore, the initially rare aposematic individuals are assumed to have less protection due to their increased detectability than the non-aposematic individuals of the population, making it difficult to understand how the strategy has invaded many taxa. The candidate hypotheses for overcoming the initial disadvantage of aposematic coloration include kin-selection, benefits from prey gregariousness, neophobia by predators and gradual change of prey coloration (see Guilford 1990 and Endler 1991 for reviews). Hitherto, the studies about the evolution of aposematism have mainly focused on predator psychology, often being directly or indirectly related to predator learning (e.g. Gittleman & Harvey 1980; Wiklund & Järvi 1982; Sillén-Tullberg & Bryant 1983; Roper & Redston 1987; Guilford 1990; Alatalo & Mappes 1996; Gamberale & Tullberg 1996, 1998; Yachi & Higashi 1998; Lindström *et al.* 1999).

However, invasion of a prey population by an aposematic phenotype can also be approached in the context of population ecology.

The invasion by an aposematic phenotype has mainly been studied with laboratory experiments and theoretically in the simplest possible systems, consisting only of a predator and one prey species, into which the aposematic phenotype enters. Results from such studies have then been extrapolated to conclusions about evolutionary phenomena on the community level. However, little is truly known about aposematism on the community level. For example, it is unknown how community structure affects the invasion of aposematism. There is a need to fill this gap of knowledge as it might increase our understanding of the evolution of aposematism and affect the interpretation of the results from previous studies. In fact, integration of knowledge about evolution and ecology of community and population levels has generally been scarce (Abrams 1996, 2000).

To achieve this, we have chosen to approach the evolution of aposematism by studying population dynamics on the community level. We modelled a community of one predator species and either one or two prey species. We call the prey species that is present both in the single-prey species and the two-prey species model 'the focal prey species'. We call the alternative prey species only present in the two-prey species model 'the reference species'. In the

two-prey species model, the predator species preys on both prey species, but if necessary, it can survive on one prey species only. The coexistence of these three species is assumed to have continued long enough for the community population dynamics to represent their typical behaviour. Then, in the population of the focal species, a rare aposematic phenotype appears. We compare the invasion success of the aposematic phenotype between the two-prey species model and the single-prey species model. The single-prey species model is otherwise similar to the two-prey species model, but the community structure is simpler, consisting only of the predator and the focal prey species. Our main interest is not in the probability of invasion by the aposematic phenotype as such, but rather in the effect of community structure on this probability.

Depending on the conditions, the newly introduced rare aposematic phenotype may increase in the population or it may die out. The conditions under which the aposematic phenotype will increase, as well as the change in these conditions when the reference species is excluded from the community, are of the uppermost interest to us. To our knowledge, no previous study on aposematism has included an alternative prey species in the predator–prey system.

COMMUNITY MODEL OF THE EVOLUTION OF APOSEMATISM

We used a discrete-time community model to study the evolution of aposematism. In the model with two prey and one predator species, the two prey species are assumed not to compete directly with each other. The density dependence in the prey dynamics is described applying the Ricker form. Thus, before the introduction of the aposematic phenotype into the community the population model can be given as (Nicholson & Bailey 1935)

$$R(t+1) = R(t) \exp(r_R(1 - R(t)) - R_P(t)) \quad (1)$$

$$C(t+1) = C(t) \exp(r_C(1 - C(t)) - C_P(t)) \quad (2)$$

$$P(t+1) = b_R R(t)(1 - \exp(-R_P(t))) + b_C C(t)(1 - \exp(-C_P(t))), \quad (3)$$

where $R(t)$, $C(t)$ and $P(t)$, respectively, denote at time t ($t = 0, 1, 2, \dots$) the population sizes of the reference prey species, the focal prey species (consisting of non-aposematic phenotypes only) and the predator, respectively. Parameters b_R and b_C are the resource values of the two prey species for the predator, describing how the consumption of a given prey type translates to the predator population size. $R_P(t)$ and $C_P(t)$ are the numbers of reference and focal species eaten at time t , respectively. The functional responses to predation are given as

$$R_P(t) = c_R P(t) / (1 + c_C b_C C(t)) \quad (4)$$

$$C_P(t) = c_C P(t) / (1 + c_R b_R R(t)), \quad (5)$$

where c_R and c_C are parameters describing prey coloration and are hereafter called detectabilities of the phenotypes. When detectability has its maximum value of 1, none of the prey of the given type passes unnoticed by the predator, whereas the crypsis of the prey increases with decreasing values of detectability. Thus according to eqns 4 and 5, increasing the abundance of the predators and the detectability of the prey will increase the predation pressure on a given prey type. On the other hand, predation on a given prey is decreased by increasing abundance, resource value or detectability of the other prey type. When the resource values and population sizes of the two prey species are equal then the detectabilities of the species will determine the division of the predation pressure.

Next, a novel aposematic phenotype A with the resource value of b_A , is introduced in the population of the focal prey species (cf. Yachi & Higashi 1998; Lindström *et al.* 1999). Our model is based on the assumption that the two phenotypes of the focal species compete equally for resources. Furthermore, the predators can distinguish between the aposematic and the non-aposematic phenotype. The aposematic phenotype may appear in the population through genetic or behavioural alterations, or it may immigrate from another population. The former could take place for example, through one mutation, or several mutations in linked genes, or because of a change of host plant by a herbivore, making it inedible (Harvey & Paxton 1981; Guilford 1988; Endler 1991).

We assume that no avoidance learning by predators takes place before $t = 101$, because before that moment a phenotype with a combination of unprofitability and conspicuousness effective enough to elicit avoidance by predators does not exist. Thus at $t = 101$, such an aposematic phenotype is introduced in the population of the focal species. Alternatively, the genetic or behavioural changes leading to the introduction have started earlier, but the distastefulness is not strong enough, the number of the distasteful individuals is not high enough, or the predators cannot distinguish them before that moment. The latter implies that if the aposematism is a result of several, subsequent mutations in linked genes, this event need not necessarily be a rapid chain of events. At $t = 101$ predators become able to tell apart the aposematic phenotype from the non-aposematic phenotype. This is based on a difference in coloration or odour, or in the case of a host plant change, even the association with the new plant might contribute to it. Whatever the route of introduction of the aposematic phenotype is, the ability of the predator to distinguish it from the non-aposematic phenotype is the prerequisite for effective avoidance learning without the problem of auto-mimicry (e.g. Guilford 1994).

The community model now becomes

$$R(t+1) = R(t) \exp(r_R(1 - R(t)) - R_P(t)) \quad (6)$$

$$C(t+1) = C(t) \exp(r_C(1 - C(t) - A(t)) - C_P(t)) \quad (7)$$

$$A(t+1) = A(t) \exp(r_A(1 - C(t) - A(t)) - A_P(t)) \quad (8)$$

$$P(t+1) = b_R R(t)(1 - \exp(-R_P(t))) \\ + b_C C(t)(1 - \exp(-C_P(t))) \\ + b_A A(t)(1 - \exp(-A_P(t))). \quad (9)$$

We expect that the introduction of the aposematic phenotype into the community may modify the interactions between the prey species and the predator. To incorporate this into our model we proceed as follows. Following the logic presented above about the predation pressure, we assume that the number of the aposematic phenotypes encountered by a predator at time t is given as

$$A_e(t) = c_A P(t) / (1 + c_R b_R R(t) + c_C b_C C(t)). \quad (10)$$

The predators may learn to avoid the unpalatable aposematic phenotype and thus, each aposematic prey a predator encounters need not become eaten. Therefore we assume that the predation pressure on the aposematic phenotype is given as

$$A_P(t) = A_e(t) / (1 + (4 \times c_A A(t))^3). \quad (11)$$

This equation incorporates the aposematic protection through learning by predators to avoid the aposematic phenotype. It assumes that the detectability c_A translates into the memorability of the aposematic signal. Based on this equation, the number of eaten aposematic prey is an increasing function of the number of encountered aposematic prey and a decreasing function of detectability (i.e. memorability) and abundance of the aposematic prey. Figure 1 illustrates the nonlinear relationship between encountered and eaten aposematic prey. When predators encounter a small number of aposematic prey, all or almost all of them are eaten, because due to the low encounter rate, individual predators learn little or not at all and furthermore, only a small fraction of the predator population encounters aposematic prey. Thus, when the number of encountered aposematic prey is low, the number of eaten aposematic prey increases with the number of encountered aposematic prey. However, with increasing encounters the relationship inflects and with even higher numbers of encounters the aversion becomes strong and the number of eaten aposematic prey approaches zero. Note, that eqn 11 is not the learning function of an individual predator, but rather it describes the avoidance response of a population of predators that show avoidance learning towards aposematic prey under varying prey and predator densities. As already mentioned, no data exist on the response of a predator population on aposematic prey, and therefore we have based

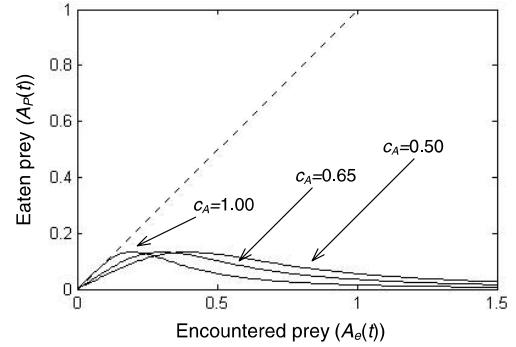


Figure 1 The effect of signal strength (c_A) on the relationship between eaten aposematic prey for three different values of c_A . This relationship is determined by eqn 11. The dashed line illustrates the situation when all encountered prey are eaten. Note that the curves are drawn under the simplifying assumption that $A_e(t) = c_A \times A(t)$ (cf. eqns 10 and 11).

this function on the following two arguments. First, we assume for obvious reasons that aposematism sometimes can invade a prey population and also, that the associative learning by predators has an important role here. Second, the function corresponds to the generally accepted pattern of learning in which the warning coloration is beneficial to the aposematic phenotype only above a certain threshold abundance (Guilford 1988; Yachi & Higashi 1998) and the predator learns to avoid more readily a conspicuous than a cryptic coloration (Gittleman & Harvey 1980; Roper & Redston 1987; Lindström *et al.* 1999; Speed 2000).

The number of individuals protected by aposematism, $A_s(t)$ (i.e. the aposematic individuals that are encountered but not eaten) is given as

$$A_s(t) = A_e(t) - A_P(t). \quad (12)$$

Because of the introduction of the aposematic phenotype, eqns 4 and 5, which give the number of eaten individuals of the reference prey and non-aposematic phenotype of the focal species become

$$R_P(t) = c_R P(t) / (1 + c_C b_C C(t) \\ + c_A b_A A(t)(1 - A_s(t)/A_e(t)) \\ + A_s(t) c_R (b_A/b_R) R(t) / (R(t) + C(t))) \quad (13)$$

$$C_P(t) = c_C P(t) / (1 + c_R b_R R(t) \\ + c_A b_A A(t)(1 - A_s(t)/A_e(t)) \\ + A_s(t) c_C (b_A/b_C) C(t) / (R(t) + C(t))). \quad (14)$$

Thus, as before the introduction of the aposematic phenotype, the predation pressure is directed more towards phenotypes with high abundance, high resource value and high detectability. However, a proportion of the predation

pressure corresponding to the number of prey protected by aposematism, $A_i(t)$, is re-directed to the reference species and the non-aposematic individuals of the focal species. This additional predation pressure caused by the aposematism is divided between these two prey types according to their relative densities, and modified by their detectabilities as well as their relative resource values.

Note that in all the predation equations (eqns 11, 13 and 14) the predation pressure on a given prey type is decreased by the abundance of the other prey types, instead of being directly increased by the abundance of any given prey type itself. If the predation pressure on a prey type were increased by its own abundance, it would result in frequency-dependent selection, favouring rare prey types. This, in turn, would have forcefully facilitated the invasion by the initially rare aposematic prey under any conditions. However, assuming such frequency-dependent predation would not be parsimonious and would give the aposematic phenotype an unjustified benefit. Instead, the strength of the avoidance response that the predator population shows towards the aposematic prey depends on the probability of encounters between the predators and the aposematic prey, which is determined by the density of the aposematic prey as well as on the abundance of alternative prey types (see eqns 10 and 11).

We carried out the simulations as follows. In all the simulations, the initial population sizes at time $t = 1$ were random numbers between 0 and 1. First, in the absence of an aposematic phenotype, the community dynamics were simulated for 100 iterations to remove the effect of the initial transient. Then we introduced the aposematic phenotype into the focal prey population by mutating the small proportion of 10^{-6} of the non-aposematic individuals to aposematic individuals. After the introduction we ran the simulation for another 1000 iterations. We scored the ability of the mutant aposematic phenotype to invade the population. According to our definition an invasion took place when the proportion of the aposematic phenotype exceeded the proportion of the non-aposematic phenotype in the population. The results are illustrated graphically as bifurcation diagrams. These are based on solutions from iteration 1000 to iteration 1100, which are plotted against the given values of the parameters of interest. No predator evolution was assumed to take place.

We compared this two-prey species model with a single-prey species model. The single-prey species model was otherwise similar to the two-prey model, but the reference species was excluded. The equations for the single-prey species model were thus acquired by replacing the abundance of the reference species $R(t)$, and the number of eaten reference species $R_p(t)$, with zeros in the equations for the predator and focal species given above.

RESULTS

We found that in certain conditions the novel aposematic phenotype with a higher detectability than the non-aposematic phenotype increased and invaded the population in our model community. The reference species played an important role in determining the conditions and consequences of the invasion.

First, we set the prey parameters of the two-prey species model in the absence of the aposematic phenotype such that the prey species would provide large enough resource for predator sustenance. For the survival of the predator population and thus, for the existence of the model community of the three species, resources for the predators had to exceed a minimum level. Given the detectabilities of $c_C = c_R = 0.50$ and growth rates of $r_C = r_A = 3.0$ and $r_R = 1.9$ for the prey species, a resource value of the reference species of $b_R \geq 1.7$ was required for the predators to survive when $b_C = 2.0$. With these settings both prey species survived also.

Next, we studied the invasion of aposematism. After the first 100 iterations, a distinct aposematic phenotype was introduced in the community. Again, we set the detectabilities to $c_C = c_R = 0.50$ and the growth rates to $r_C = r_A = 3.0$ and $r_R = 1.9$ for the prey species. The resource values were $b_C = b_A = 2.0$ and $b_R = 3.0$. (These values were also used below if not stated otherwise.) We varied the detectability of the aposematic phenotype from $c_A = 0.50$ to $c_A = 1.00$ between simulations. With these settings the aposematic phenotype invaded the population of the focal species within 1000 iterations after the introduction when its detectability was $c_A \leq 0.66$ (Fig. 2). Thus, the invasion by a more easily detected phenotype was possible within certain limits in the model system. Further, this upper limit depended on the resource value of the non-aposematic phenotype of the focal species, such that for example $b_C > 4.0$ enabled the invasion of a phenotype with the higher value of detectability $c_A = 0.80$. Similarly, decreasing the detectability of the non-aposematic phenotype from $c_C = 0.50$ to $c_C = 0.45$ decreased the highest detectability of the aposematic phenotype, allowing invasion to the lower value of $c_A = 0.58$.

Keeping the other settings unchanged, but fixing the detectability of the aposematic phenotype to $c_A = 0.65$, and instead, varying the resource value of the reference species (b_R), showed the importance of the existence of an alternative prey to the invasion. The aposematic phenotype could not invade the population within 1000 iterations when $b_R < 2.5$ (Fig. 3) because of too high predation on the focal species to allow the initial increase of the aposematic phenotype. At the other extreme, for $b_R > 5.8$ the predator received so much of its resources from the reference species that there was not enough predation on the focal species for an effective associative learning to take place. Consequently,

the density of the aposematic phenotype increased too slowly for the invasion to take place within the 1000 iterations (Fig. 3).

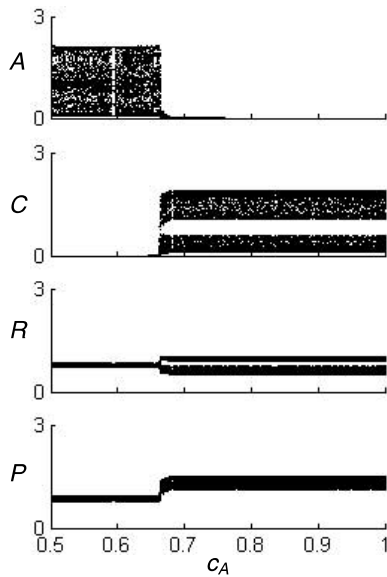


Figure 2 Bifurcation diagram for the aposematic (A) and the non-aposematic (C) phenotype of the focal prey species, the reference prey species (R) and the predator (P) when the detectability of the aposematic phenotype (c_A) was increased from 0.50 to 1.00.

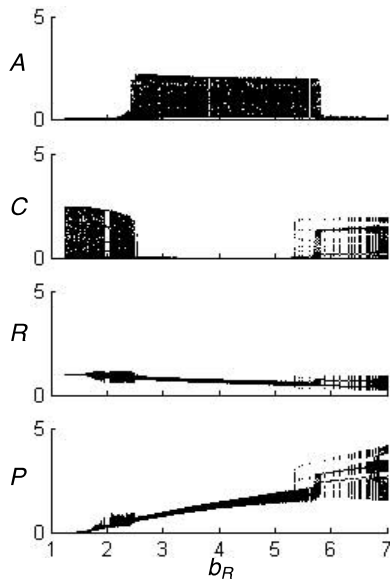


Figure 3 Bifurcation diagram for the aposematic (A) and the non-aposematic (C) phenotype of the focal prey species, the reference prey species (R) and the predator (P) as a function of resource value b_R .

When we again fixed the resource value of the reference species at $b_R = 3.0$ and decreased the resource value of the focal species to $b_C = b_A = 0.5$, a more conspicuous aposematic phenotype could not invade the population, but an invasion was possible only for $c_A = 0.50$. This suggests that invasion by aposematism is unlikely when the focal species initially has a low predation rate, for example because of its low profitability for the predator.

Also, varying the growth rate of the reference species (r_R) and keeping the other parameters constant demonstrated the important role of the reference species for the invasion of the aposematic phenotype. An invasion by the aposematic phenotype could not take place when $r_R < 1.7$ (Fig. 4). Too low values of growth rate of the reference species resulted in too high predation on the focal species for the aposematic phenotype to increase, this time because of the low replacement rate of the reference population. On the other hand, with values of r_R close to 3.0 and higher, the fluctuation of the reference species population became so high that it had a negative effect on the predator population, such that it occasionally became extinct. With values of r_R high enough it always became extinct within the 1000 iterations (Fig. 4).

Excluding the reference species from the system had a dramatic effect on the conditions and consequences of the invasion of the aposematic phenotype. Because the predator did not have any alternative resources in the single-prey species model, a higher resource value of the focal species than in the two-prey species model was required. The

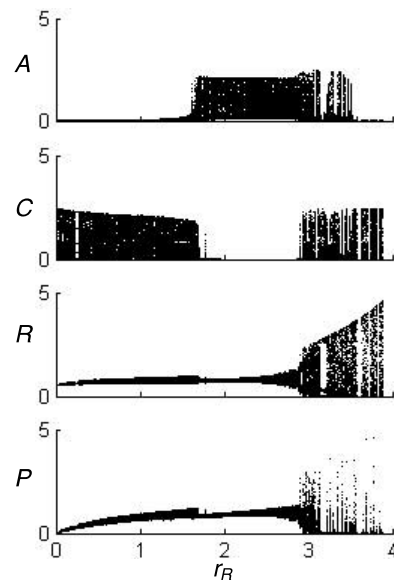


Figure 4 Bifurcation diagram for the aposematic (A) and the non-aposematic (C) phenotype of the focal prey species, the reference prey species (R) and the predator (P) as a function of growth rate r_R .

resource value of the focal species, b_C , needed to be 3.5 or higher for the predator to survive. Invasion of the aposematic phenotype was possible when $b_C \geq 3.6$. An essential difference between the models was that in the single-prey species model there was no upper limit to the detectability of the aposematic phenotype, c_A , for the invasion to take place. Instead, the invasion occurred with any value of c_A between $0.50 \leq c_A \leq 1.00$. Another difference between the models was that when the invasion took place, it was always relatively rapid, taking less than 100 iterations. Furthermore, the invasion always resulted in fast extinction of the predator, leaving a low density of the non-aposematic phenotype left in the focal population. Increasing the resource value of the aposematic phenotype, b_A , together with b_C did not change these events.

DISCUSSION

Our results suggest that an alternative prey species affects the conditions and consequences of the invasion of an aposematic phenotype. In the two-prey species model the reference prey turned out to have an important role in determining the conditions of the invasion of the aposematic phenotype. An invasion was possible only when the reference prey provided a stable and plentiful enough resource for the predator. Otherwise, the predation pressure on the focal species was so intense that it prevented the aposematic phenotype from reaching a high enough density for the predators to learn to avoid it. However, at a certain point, increasing the resource value of the reference prey slowed down the invasion, implying that too plentiful alternative prey might result in a too weak avoidance response and decrease the probability of a successful invasion by the aposematic phenotype.

There were two major differences in the invasion of the aposematic phenotype between the two-prey species model and the single-prey species model. Because the focal prey as well as the predator and its response to prey were equal in these models, the observed differences are purely the result of the difference in the community structure. First, in the two-prey species model there was an upper limit of detectability of the aposematic phenotype for the invasion. This suggests that in the presence of alternative prey, the evolution of aposematism is likely to be gradual (cf. Leimar *et al.* 1986; Mallet & Singer 1987; Yachi & Higashi 1998; but see Lindström *et al.* 1999). Thus at first, an aposematic phenotype with a relatively weak aposematic signal invades a prey population. Then, a generalization of prey coloration by the predator may allow the selection for stronger and stronger aposematic signals, gradually giving better protection (Gamberale & Tullberg 1996). In the single-prey species model invasion was also possible with high values of detectability of the aposematic phenotype. Second, in

contrast to the two-prey species model, in the single-prey species model the invasion always resulted in rapid extinction of the predator, leaving a small proportion of the non-aposematic phenotype in the prey population. With the extinction of the predator in the single-prey species model the benefit of aposematism disappears and therefore, it is possible that aposematism is transient in this case. Note, that a study focusing on a predator's ability to learn to avoid aposematic prey would not reveal whether or not the assumed conditions of invasion of an aposematic phenotype were sufficient for predator population sustenance.

Interestingly, our study suggests that an aposematic phenotype is unlikely to invade a prey population with a low predation rate, because the avoidance response by predators is not strong enough. This also implies, that if a non-aposematic population is already defended by distastefulness, the weak avoidance response will probably prevent it from becoming conspicuous.

The nature of the aposematic defence may differ between the two community structures. In the two-prey species community it is enough that the aposematic defence turns the predation pressure to the alternative prey and, thus, the edibility of the aposematic prey in relation to the edibility of the alternative prey species should be of importance. On the other hand, in the single-prey species community the aposematic defence must be strong enough to decrease the predation rate in absolute terms. As our model shows, this may have dramatic effects on the predator population.

It is known that community structure may have evolutionary effects (Abrams 2000 and references therein). Our results suggest that the behaviour of the predator does not necessarily constitute sufficient conditions for the evolution of aposematism but also the community structure and ecology of other prey species may affect these conditions. Thus, empirical and theoretical studies on community ecology of aposematic species are needed to increase our understanding of the evolution of aposematism.

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