SIMULATION OF COEVOLUTION IN BATESIAN MIMICRY

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ABSTRACT

Coevolution may be defined as an evolutionary change in a trait of one species in response to a change in a trait of a second species, followed by an evolutionary response by the second species to the changes in the first. Yet, more complex evolutionary relationships are known to occur, such as Batesian mimicry, where three or more separate species interact along their evolutionary history. Do these complex interactions involve coevolution? In order to explore possible answers to this question, we built an agent based simulation model in which we monitored the evolution of the characteristics of individuals in a Batesian mimicry system consisting of predators, noxious prey or toxic models, non-noxious prey mimicking the toxic model, and non-mimic non-noxious prey or alternate prey. The evolutionary game consisted in Predators evolving genetic mechanisms for avoiding preying on Toxic Models, in Toxic Models evolving anti-predator toxins and signals for Predators, and in Mimics evolving mimic anti-predator signals, taking as a modeling reference the case of the evolution of harmless look-alikes of venomous coral snakes is taken as a reference. Results showed that a likely evolutionary scenario for Batesian mimicry is a mutual selective pressure between Predators and Toxic Models (i.e. Predators and Models coevolve), a selective pressure acting from Predators on Mimics, and a "dilution effect" exerted by Mimics and Alternative Prey upon the Predator-Model interaction. Alternative genetic mechanisms by Predators to avoid toxins in Models (resistance), slows Predator-Model coevolution. The simulations provide for the following testable predictions: Mimics will not coevolve with Predators nor Models but evolves in response of selective pressure from Predators without affecting the evolution of Predators nor Models. Predators and Models have to affect each others population significantly in order for a Batesian ring to get established. Deterrents in Models have to be very toxic against Predators in order to allow the establishment of a Batesian mimicry ring.

Key words: Batesian, mimicry, population dynamics, toxins

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INTRODUCTION

Coevolution, as defined by Ehrlich and Raven (1964) and elaborated by Janzen (1980) is a process by which two organisms develop a close association over evolutionary time, by means of a series of reciprocal steps. Ehrlich and Raven developed this theory using the tight relationship between some butterflies and their host plants, but since then the theory of co-evolution has been generally accepted as an explanation for the evolution of associations between many pairs of groups of organisms (Gilbert 1983, Dettner and Liepert 1994). Coevolution implies that each member of the association directly influences the evolution of the other organisms. Thus, in a plant/herbivore system, not only does the herbivore influence the evolution of the plant, but the plant also influences the evolution of the herbivore. It has, however, proved difficult to find evidence for coevolution (Jermy 1984, 1993). There are also a number of predictions associated with coevolution; the strongest of these is that the phylogeny of the taxa involved should be parallel. In the few cases studied, however, parallel phylogenies have not been found; in fact the phylogenies of the taxa have been shown to be distinctly un-parallel (Miller 1986, Brown 1987, Brown and Henriques 1991)

One of the more interesting cases of putative coevolution are the relations between organisms in a Batesian mimicry system. Here, at least two broad categories of evolutionary interacting systems can be visualized: In one category of systems, predators learn to recognize toxic prey, and models and mimics may coevolve. Here, the genetic evolution of the predator, relevant to the Batesian mimicry ring, is more related to its discrimination abilities between models and mimics than to its abilities in learning. The condition for these systems to work is that the toxic mimic does not kill the predator, allowing it to learn. In the second category of systems, predators do not have to learn to recognize toxic models but acquire their innate discriminatory abilities through genetic evolution. In these cases, the evolution of at least three different species may be entangled: Predator, toxic prey which the predator should avoid, and non-toxic prey organism which mimics the model to avoid predation. If in this Batesian mimicry system coevolution is involved, then Predators, Toxic Models and Mimics should influence the evolution of each other. There is a theoretical possibility that because of the complexity of interactions in this Batesian mimicry ring, coevolution may not be possible.

Empirical evidence against coevolution in systems where it was previously thought to occur does not imply that it is not theoretically possible. Models of coevolution considered two species systems, such as plant/herbivore (Levin 1983, Matsuda and Abrams

1994) or predator /prey (Marrow *et al.* 1992, Marrow and Cannings 1993, Van der Laan and Hogeweg 1985), show the possible character of coevolutionary systems. In the case of Batesian mimicry, models have emphasized on specific parts of the interactions, allowing for a better understanding of the problem (Holling 1965, Emlen 1968, Nur 1970, Estabrook and Jesperson 1974, Matthew 1977, Luedeman *et al.* 1981, Owen and Owen 1984, Turner 1984, Turner, Kearney and Exton 1984, Getty 1985, Huheey 1988, Speed 1993, Yamauchi 1993, Turner and Speed 1996).

Simple models, however, may fail in grasping relevant natural processes (Leaven et al. 1997). Simulation of the behavior of each individual in a large population of individuals is now possible, thanks to advances in computing power. These "agent-based" simulations allow for the study of properties that emerge from complex webs of interactions. It is imaginable that the complex webs of interactions in a Batesian mimicry ring might show such emergent behavior. One such model, Biodynamica, has shown that when using agent-based simulations, former intractable problems in biological evolution become accessible to theoretical analysis (Jaffe 1996, 1998, 1999, 2000, 2001, Ochoa and Jaffe 1999), providing experimentally testable predictions that have been validated so far (Jaffe *et al.* 1997, Rincones *et al.* 2001, Cabrera *et al.* 2001). Here, using this agent-based simulation approach, we want to explore whether coevolution is theoretically possible in a more complex simulation of a Batesian mimicry system. In order to build the simulation, the case of the evolution of harmless look-alikes of venomous coral snakes is taken as a reference. The red, yellow (or white) and black ringed markings on the mimics and on poisonous snakes induce avoidance behavior in predators (which have similar generation times than snakes), even without prior experience to these snakes (Smith 1975), and this avoidance behavior is lost when dangerous models are absent (Pfenning *et al.* 2001).

THE MODEL

The numerical simulation model "Biodynamica" used here (available at http://atta.labb.usb.ve/klaus/klaus.htm) is an agent based model programmed with Visual Basic. It constitutes a population of agents, growing, reproducing and interacting in virtual space simulating a changing environment (see also Jaffe 1996, 1998, 1999, 2000, 2001, Jaffe et. al. 1997). The agents were of four different types, representing four different species of animals (Predators, Toxic Models, Mimics and Alternative non-noxious prey). Each individual agent possessed a specific genotype, defined by alleles of up to 17 different genes, each coding for a given phenotype (Table 1). The agents could evolve over time, with each time step representing one reproductive cycle. The genetic composition of the population of agents was monitored continuously. We could thus visualize the dynamic properties of a complex interacting assemblage of genes evolving together in a population

of organisms of four different species. This scenario simulates the putative simultaneous evolution of various traits, in up to four different interacting species, where each suffered genetic drift and adaptations. The simulation tries to get as close as possible to evolution in a natural environment.

Two sets of experiments were run. In the first set, agents were modeled as simple as possible. Here, all agents were defined as belonging to one of 3 types of species (Predator, Model, Mimic), each living for a maximum of 5 time steps, reproducing after the age equivalent to one type step by producing 6 offspring's in each reproductive cycle, if the organism was female and if it was fertilized by a male. Organisms were diploid and bisexual (i.e we simulated male and female agents). In this set of experiments, the optimal population for each of the 3 different species was 400 organisms. When the actual population of agents surpassed this threshold, random selection culled the population exponentially in regard to population number so as to get close to 400 individuals per species. At each time step, each predator met with either a mimic or a model, chosen randomly. If the model or mimic possessed allele Nr. 5 of their corresponding gene for signaling, and the predator possessed the allele Nr. 5 of the gene for toxic model recognition, no predation occurred. Otherwise the predator killed the model or mimic. If the agents representing models possessed the allele for toxicity, the predator was killed in the interaction. Demonstration file "Species1" in Biodynamica reproduce this simulation scenario.

In the second set of experiment, the number of individuals in the populations of each species could vary in accordance to the degree of predation or poisoning suffered. Here, four different species were modeled: Predators, Models, Mimics and Alternative prey. The initial population had a random distribution of genetic characters (alleles) in each of the 17 different genes or loci of the genome of agents listed in Table 1.

In both cases, the initial population was then subjected to a five-step transformation algorithm (see below) at each time step. The exact genetic composition of the multi-species population was plotted at each time step, with the actual number of surviving individuals. Thus we could assess when and if each population reached a maximum population size and when it fixed the adaptive alleles (see below). Repetition of this kind of simulation, with different random initialization, and different combination of species (see below) provided the basis for calculating the allelic frequency distribution at equilibrium for each species. External parameters were constant throughout each simulation and were: optimal size of the total population of agents = 2000, initial size of the population of each species = 400,

optimal clutch sizes, and optimal age for reproduction for each species varied (in the second set of experiments only) between species so as to achieve an equilibrium situation where non of the 4 different species went extinct.

The five transformation steps consisted of the following:

Mating: Females mated with the first male randomly encountered. Females not finding an adult male of its species, in up to 90 attempts in a random search at each time step among all individuals in the population, did not reproduce during that time step.

Reproduction: Mated individuals produced offspring according to their phenotypically fixed clutch size (see Table 1), transmitting their genes to the offspring according to Mendelian rules for bisexual-diploid reproduction with uniform recombination. That is, each parent provided half of the genes to the newborn; and individuals had two copies of different alleles for each gene respectively.

Variation: Randomly selected genes mutated, changing their allelic value randomly, according to a probability (mutation probability) given by the allele in the gene "mutation"(see Table 1).

Phenotypic expression: A single, randomly chosen allele, i.e., only one of the two alleles at each locus was expressed phenotypically. That is, the value of this allele was used to assign the corresponding phenotypic characteristic of the individual as given in Table 1. This method produces values intermediate to that of simulations of gene dominance and recessive genes (Jaffe 2000), and avoids the arbitrary *a priori* assignment of dominance/recessive status to alleles.

The final clutch size of individuals was calculated based on the allelic characteristic of the gene coding for clutch size, and the minimal age for initiating reproduction, using a normal distribution, with maximal clutch sizes being genetically predetermined and occurring at an optimal age of reproduction. The size of the clutch of newborns affected the probabilities of survival of the future adult, so that individuals born in clutches larger than optimal decreased their probability of survival exponentially.

Selection: Individuals were excluded from the population when their age exceeded their genetically prefixed life span, when randomly selected by density dependent criteria which increased exponentially after optimal population sizes had been reached, and when they possessed not-resistant alleles to biocides 1 and 2 with probabilities which changed randomly each time step from 0 to 0.9. Biocides 1 and 2 acted differentially on Prey, Models, Mimics and Alternative Prey so as to achieve an equilibrium situation where non of these four populations went extinct. Predators that found no palatable prey died by starvation.

In the more complex simulations we explored the dynamic interactions between organisms of four different species evolving their allelic configurations independently: Predator could feed on Toxic Models, Mimics or Alternative Prey; Toxic models were able to evolve anti-predator toxins and an aposematic warning signal independently; Mimics developed signals similar to those of Models for its defense but with no actual noxious mechanisms; and Alternative Prey which did not evolve any kind of anti-predator strategy. The complexity of the simulated agents was necessary in order to achieve differential mortality and reproduction so as to achieve populations where non of the four type of organisms went extinct. In simpler simulations, this could not be achieved, unless the population size for each of the type of agents was fixed artificially, as was done in the first set of experiments. In the second set of experiments, each organism interacted with another organisms, randomly chosen from among the whole population of organisms, so that the probability of encounter with any of them was directly related to the relative abundance of these organisms.

Among the genes present (see Table 1), Predators possessed a gene that could evolve an allele for detecting the noxious prey (allele Nr 5 of gene l). Toxic Models had a gene (t) that could evolve increasing degrees of toxicity and another gene (s) that could evolve an allele that served as an advertising signal (allele Nr. 5 of gene s); and Mimics possessed gene m that could evolve an allele (Nr. 5) that could mimic allele Nr 5 of s. Predators could, through the adaptive emergence of the right allele in l, recognize Models possessing allele 5 of s, if they had evolved their toxins, avoiding preying on them. Predators possessing the allele 5 of l, did not prey upon Toxic Models possessing the allele 5 of s. Predators expressing phenotypically other alleles of l or encountering Models not possessing allele 5 of gene s predated the Model. In such a case we simulated two different situations:
- Extreme interactions in which Predators killed Models and themselves;
- Variable interactions in which Predators reduced the fitness of Models according to the allele of toxicity in gene t ranging from 0 to 100 % and Predators reduced their fitness according to their allele of a resistance gene (r) ranging also from 0 to 100%. The fitness reduction was calculated as r-t.

Mimics could possess allele 5 of gene m which had the same effect on Predators as allele 5 of gene s. Encounters between Predators and Mimics were always fatal for Mimics, except when Mimics had allele 5 of gene m and Predators had allele 5 of gene l. In some simulations, successful predation of Mimics and Alternative Prey by Predators increased

the fitness of Predators by a factor of 0.05 to 0.5, according to the allele in gene p (positive fitness increase) of Predators. Predators encountering Alternative Prey always killed it.

Simulation results were used to compute correlations between variables. Here, correlations appearing after a large number of simulations indicate covariance between the correlated variables. Making one variable constant at different values and studying the evolution of a given set of variables, served to test the relevance of a given parameter in determining the values of others.

RESULTS

Figure 1 shows an example of the evolutionary process of a community of organisms consisting of Predators, Toxic Models and Mimics, using the simplified version of the simulations (first set of experiments described in methods). The number of Toxic Models having the adaptive allele of s (allele 5) for signaling, the number of Predators having the adaptive allele of l for signal recognition, and the number of Mimics possessing the adaptive allele of the mimic gene m, increases continuously until eventually displacing all other alleles in the population. As shown in Figure 1, populations of Mimics often increase the frequency of their adaptive allele, expressing the aposematic mimic signal faster than do populations of Toxic Models increase the frequency of their aposematic signal. But basically, all three organisms evolve their adaptive allele concomitantly.

Figure 2 shows an example of the evolutionary process of a community of organisms consisting of Predators, Models and Mimics, in which the Models have to evolve not only an aposematic signal, but also an allele for toxicity (allele 10 of t; resistance gene r of predators was fixed at 10), using the simplified version of the simulations. As evident from the figure, none of the adaptive alleles becomes established and fixes into the population. There are exceptional runs (less than one in 50) where sufficient Model organisms fixed alleles for toxicity and aposematic signals at the same time, allowing the increase in frequency of the adaptive alleles of the Predators and thus of the other organisms of the community. This difficulty in simulating the evolutionary establishment of a Batesian mimicry ring, was largely due to the fact that the effect of predation and toxicity did not affected the total population of the affected organisms (prey and predator respectively), as they had to be maintained constant.

Therefore, simulations using the second set of experiments with more genetically complex agents were performed. Here, the predators could decimate the models, and toxic models could wipe out the whole population of predators. Varying randomly the complete set of external parameters and varying also the composition of the initial populations of organisms, including alternative prey which buffered the effect of predation, led to the

eventual establishment of equilibrium situations, where all different types of organisms survived during a given period of time. These simulations showed that the speed at which the adaptive alleles are fixed may vary depending on the following factors: Complexity of genetic composition of the species (i.e. number of genes subjected to selection, not shown but see Jaffe 1998); relative numbers of Predators and Models with regard to Mimics and Alternative Preyy; strength of the interaction, i.e. if Toxic Models kill the Predator or only lowers their fitness, if the Predator increase their fitness from feeding on Mimics and/or Alternative Prey; frequency of changes in the environment (i.e. appearance of additional selective pressures: see Biocides 1 and 2 in Table 1). We chose the parameter values so that genetic drift was evidenced but did not dominate the simulations. That is, with 500 time steps and a total population of 2000 individuals, the adaptive alleles of gene l, s and m were fixed in the respective populations with a probability > 0.92. The other outcomes of the simulations were the extinction of one or more of the populations, and the elimination of the adaptive allele without reappearing through mutation during the time period studied (normally 80 time steps).

We analyzed quantitatively the proportion reached by each adaptive allele in its population of genes, in simulations modeling extreme interactions between Predator and Toxic Model (predation on the Model always killed the Model and Pedator). These proportions, after 15, 30 or 60 time steps, are presented in Table 2. This table shows the average values of the allelic frequencies derived from 300 simulations in which the number of organisms in each population is approximately the proportion given in the table. Results show that the proportion of adaptive alleles of the genes l and s was independent of the presence or absence of Mimic organisms; but the proportion of adaptive alleles of gene m increased when the populations mix included Toxic Models. The results also show a "dilution effect", in that simulations with high proportions of Mimics + Alternative Prey in relation to the number of Predators and Model individuals, gave lower proportions of adaptive alleles of genes l, s and m. That is, the lower the proportion of predators in the population mix, the lower the selective pressure, the slower the fixing of adaptive alleles. In simulations with variable interactions between Predator and Model, where Predators had variable resistance to the toxin of the Models, and the Models variable toxicity to Predators, each determined by a gene which was itself subjected to the five step selection process in the simulations, the speed of fixing of adaptive alleles of genes l, s and m slowed down as compared to simulations with extreme Predator-Model interactions (experiment 11 vs 10, Table 2). The final outcome of the evolutionary process, however, although slower, was similar to that of experiments modeling extreme Predator-Model interactions, as shown by results of simulations modeling variable Predator-Model interactions after 60 time steps

(experiment 12, Table 2). An important difference is that Mimics had a higher proportion of adapted alleles m relative to s and l, compared to other simulations (e.g. experiments 10 & 11 in Table 2).

A Spearman's rank coefficient test applied to 56 simulations after 60 time steps in which the resistance of Predators (r), the toxicity of Models (t) and the fitness of Predators after preying upon Mimics or Alternative Prey could increase according to alleles of gene p, showed the following: The amount of adaptive l, s and m alleles correlated positively and highly significantly with each other (Table 3). The amount of resistance (given by gene r) in the Predator population correlated negatively with the amount of toxicity (given by gene t) of the Model population. The amount of r correlated positively with m and t negatively with m, indicating that when populations where optimizing r and/or t, they were affecting the evolutionary dynamics of the Mimic. This effect was due to the fact that high r and/or low t values increased populations of Predators, which in turn accelerated the emergence of the protecting allele of m. Increasing the fitness benefits of Predators through adaptation of p did not affect the evolution of the other alleles tested.

The likelihood of alleles coding for a signal indicating toxicity to a prey, being fixed in a given population, was independent of the presence of the organism mimicking this signal, so long as the amount of mimics do not significantly reduce the likelihood of encounters between the predator and the noxious prey. Adaptive pressure seems to act from the predator to the toxic prey and from the predator to the mimic (see Table 2, experiments 1 and 6, without Predators, compared to the other experiments with Predators); Adaptive pressure also acts from the Toxic Model to the Predator population by inducing genetic adaptation of the signal by the predator (see experiments 2 and 7, without Toxic Models compared to other experiments with Models). Thus, coevolution is likely to occur between two of the three components of the system, i.e. the Predator and Model. The Model affects the evolution of the Mimic (experiments 2 and 7 compared with 4 and 9) but the Mimic does not affect adaptation of either the Model or the Predator (experiments 3 and 8 compared with 4 and 9), unless the mimics outnumber other available prey. The amount of Mimic, of course, caused a decrease in Alternative Prey by shifting predatory pressure to the Alternative Prey.

DISCUSSION

The evolutionary interactions between model and mimic have been the topic of much discussion. Brower and Brower (1972) coined the term *advergence* for the process whereby mimic and model are involved in an arms race (Dawkins and Krebs 1979, Huheey

1988) which the mimic wins by evolving towards the model faster than the model can evolve away. Turner (1984) modeled the gradual advergence of a Batesian mimic to its toxic model, showing that the mimic did indeed evolve towards the model faster than the model could evolve away from the mimic. Turner suggested that this was because "the advantage of being a mimic is considerably greater than the disadvantage of being a model". Our model, confirmed that the "advantage of being a mimic is considerably greater than the disadvantage of being a model", as populations of mimics evolved their signal faster than populations of models. However, our model differs from that of Turner (1984) as s and m have only one value that is aposematic/mimetic and it shows that there is not really an arms race at all between the model and the mimic, because the model is not, generally speaking, affected by the presence of the mimic. Thus the mimic evolves in response to the model but not *vice versa.* This possible lack of an arms race looks to us analogous to that alluded to by Dawkins (1988) albeit in a different context - that of the origins of eusociality, whereby there is no counter-selection by the juveniles in response to parental manipulation.

Our simulations showed that in order for a Batesian ring of species to get established, there has to be a strong selection pressure due to predation on the model population, and a strong selection pressure, due to toxicity of the model population on predators. We showed that the evolutionary dynamics of gene l was not affected by the presence or absence of mimics but that the dynamics of the evolution of gene m was affected by toxic models, as shown in the correlations and in the comparisons of the actual values of the simulations. In addition, when alternative prey was present (experiments 5 and 10 in Table 2) mimics were less affected compared to situations where alternative prey was absent (experiments 4 and 9). Mimics evolved anti-predator signals in population mixtures with small populations of toxic models or even in the absence of them due to a small amount of predators possessing the adapted allele of gene l (experiments 2 and 7). This result is congruent with previous findings that mimics in imperfect Batesian mimicry rings still gain an advantage by resembling models compared to the predation levels on the alternate prey and the mimic will then be under a greater selection pressure to evolve defensive signals (Hetz and Slobodchikoff, 1988).

Our simulations showed that the role of alternative prey is to dilute the interactions slowing the speed of adaptations but not changing their final outcome (experiments 4 and 9 compared with 5 and 10 in Table 2). This is logical if we assume that it is the ability of the predator to discriminate between model and mimic that governs the evolution of the latter. Thus if there is no alternative prey, predators will be under a greater selection pressure to distinguish between model and mimic (Hertz and Slobodchikoff 1988, Huheey 1988). A

similar effect can be expected from situations with multiple prey or predators, assuming that these do not exert opposing selective pressures. This should not be confused with the one of diffuse coevolution (Fox 1988). Diffuse coevolution implies that many species on the same or different trophic levels may simultaneously exert selective pressures on one another and be affected by changes in other component members. Since Alternative Prey does not evolve any anti-predator characteristics and does not compete with Toxic Models or Mimics in our model, it cannot exert a selective pressure on any of these elements. We propose the term dilution of selection pressure for such a situation. This dilution effect has been experimentally evidenced by studying the effect of large mimic populations, or the absence of alternate prey, on the survival of the model (Brower et al. 1964, 1967, Cook et al. 1969, Dill 1975, Nonacs 1985, Slododchikoff 1987)

Comparing experiments 4 and 9 with 5 and 10 (Table 2), we find that the mimic does not affect the evolution of the predator's capacity for genetic adaptation for recognizing toxic models (gene *l*). This may be explained by the life dinner principle (Krebs and Dawkins 1984, Dawkins 1989). That is, it can be argued that the predator in our computer model is manipulated by the mimic, in that by avoiding preying on the mimic the predator is losing out on a meal and thus not increasing its fitness. For the predator not to be manipulated by the mimic it must evolve "discrimination genes" in order to discriminate between the mimic and the toxic prey or model; but genes for "good mimetism" by mimics are more likely to be passed down through generations than genes for "discrimination" by predators because if the mimic fails to deceive the predator he loses his life, whereas if the predator fails to recognize a mimic as a non-toxic prey he merely loses a dinner. This was observed by Howse and Allen (1994) in a reply to the paper by Dittrich *et al.* (1988) on hoverflies that mimic wasps as a protection against pigeons. Howse and Allen (1994) noted that the pigeons showed very little evidence of discrimination between "intermediate" hoverfly mimics (as opposed to "very good" or "very bad" mimics) - although it must be taken into account that the pigeons powers of discrimination are not necessarily comparable to our own, thus a "good mimic" in pigeon terms is not necessarily a "good mimic" in human terms (Dittrich *et al*. 1988).

Table 3 shows an interesting negative correlation between genes *t* and *m* and a positive correlation between *r* and *m*, suggesting that the less toxic the model and/or the more resistant the prey the less alleles for mimetism to evolve. These are not direct correlations due to the fact that only *l* affects *m* in our model. Thus, these correlations are due to the intermediate effect of *r* and *t* on the amount of predators and adapted alleles *l*, which in turn affect the evolutionary dynamics of *m*.

This model may have many limitations. One of them is that natural interactions often involve two or more predators. Here we simulated one predator population acting on various prey populations. The effect of multiple selective pressures on the evolution of genetic traits has been studied elsewhere (Jaffe et al, 1997 for example), where it was shown that the use of multiple pesticides slowed down the emergence of genetic resistance in diploid sexual organisms, but did not avoid it. Thus, we may reasonably assume that the emergence of genetic resistance to multiple pesticides is analogous to the emergence of anti-predator signals by multiple preys.

The interaction between the four species modeled was dependent on the probability of the predator encountering any of the other organisms. Coevolution between predator and the toxic model occurred with very high probability. The evolution of the model and the mimic were dependent on the relative amount of predators. Coevolution between the mimic and the model may occur only when the amount of mimics diluted the selective pressure of the predator upon model; and thus only when populations of mimics largely outnumbered those of models. These results suggest that the likely evolutionary scenario for Batesian mimicry is a mutual selective pressure between the predator and the model, a selective pressure acting from the model to the mimic, and a "dilution effect" exerted by the mimic and any other prey upon the predator-model interaction. Alternative genetic mechanisms of the predator to avoid toxins in the prey (resistance) slow and/or interfere with the coevolutionary process.

One important finding here is that the more options are available to avoid the noxious effects of predation on toxic prey, the slower the fixation of any specific gene coding for a given anti-predator strategy. Thus a prediction of our simulations, which could be used to eventually falsify our model, is that in demonstrated cases of Batesian mimicry, no alternatives to avoid intoxication (such as resistance or tolerance) exist for the predator. That is, potential predators of models in Batesian mimics should not possess biochemical mechanisms to tolerate the toxicity in the model organism. Or viewed from the opposite perspective, models of Batesian mimic systems should have potent toxins against their potential predators. This prediction is contrary to what is commonly assumed in Batesian mimicry, i.e. toxins should allow survival of the predator to allow for learning, and thus should be rather mild. However, observational learning for example, common among vertebrates and invertebrates, seems to be faster than associative learning (Fiorito and Scotto, 1992), and does not require low toxicity of models in order for predators to learn to recognize toxic prey.

Table 1: Genes defining the phenotype of the simulated organisms

Gene	Alleles	Phenotypic expression coded
species	1-4	Type of species. This gene never mutated. Alleles defining the species were assigned to agents at the beginning of the simulation. These were Predator, Model, Mimic or Alternative Prey.
life	0-10	The value of the allele codes for maximum life span ranging from 0 to 10 time steps. For example allele 3 codes for maximum life span of 3 time steps which was reached by the organism if no other phenomenon killed it before reaching that age
clutch	0-10	Clutch size ranging from 0 to 10 offspring per reproductive cycle per female
repro-f	0-5	Minimum age for the start of female reproduction ranging from 0 to 5 time steps
sex	1-2	Sex, either female (allele 1) or male (allele 2)
repro-m	0-5	Minimum age for male reproduction ranging from 0 to 5 time steps
mutation	0-10	Mutation probability coding for mutation rates from 0.2 (allele 0) to 10^{-7} (allele 10) mutations per gene per offspring following the formula: $p = 0.2 \wedge (v/2 +1)$, where v is the value of the allele.
sex-det	0-1	Sex determination mechanism, either random or weighted. If weighted then gene sex-rat was expressed
sex-rat	0-10	Sex ratio of newborns. Allele 0 produced only females, allele 10 only males, values from 1 to 9 produced both males and females in increasing proportions of males
resistance1	0-10	Resistance to biocide 1. Only allele 0 gave resistance to the organism possessing it. This gene, together with the gene for resistance2, served to regulate differential mortality between species and thus served to control the population sizes of the various species, avoiding the take over of one population by another.
resistance2	0-10	Idem as resistance1 but for biocide 2.
neutral	1-100	Neutral gene. It was used for monitoring genetic drift. It had no effect on the phenotype.
l	0-10	Allele 5 in Predators allowed to recognize Toxic Prey possessing allele 5 in gene *s*. All other alleles do not allowed recognition of Toxic Models. This gene represents the genetic recognition of aposematic signal.
s	0-10	Allele 5 in Models allowed Predators to recognize Models as toxic. All other values did not allow the recognition of Toxic Models. The gene serves to simulate the presence of aposematic signals in Models
m	0-10	Allele 5 in Mimic prey allowed avoidance of predation. All other values did not repel Predators. The gene simulates the presence of aposematic signal in mimics.
t	0-10	Degree of toxicity of Models when ingested by Predators. Allele 0 is non-toxic and allele 10 is 100% toxic, killing any Predator *
r	0-10	Degree of resistance of Predators when ingesting toxic Models. Allele 0 is non-resistant and allele 10 is 100% resistant *

* Predator was killed when $r - t <= 0$

Table 2: Results of simulations (mean values or frequencies from 50 simulation for each data point after 60 time steps) exploring the effect of the presence and absence of mimics (M) in the development of noxious prey (T) – predator (P) coevolution, in the presence or absence of alternative prey (A). Simulations where one of the sub-populations went extinct were eliminated from this analysis.

| Experiment	Proportion (%) of organisms in total population				Proportion (%) of adapted alleles respect to all alleles of the gene			
	P	T	M	A	l	s	m	P-T interaction
after 15 tsteps:								
1	0	33	33	33	-	9 ±03	8 ±03	none
2	39	0	31	30	11 ±06	-	13 ±08	extreme
3	34	33	0	32	18 ±09	17 ±10	-	extreme
4	36	32	32	0	17 ±09	17 ±10	16 ±08	extreme
5	25	25	25	25	14 ±06	13 ±06	14 ±05	extreme
after 30 tsteps:								
6	0	33	33	34	-	10 ±04	9 ±04	extreme
7	39	0	31	30	10 ±06	-	20 ±09	extreme
8	38	35	0	27	41 ±27	42 ±27	-	extreme
9	33	34	33	0	42 ±25	40 ±25	40 ±18	extreme
10	26	25	25	24	23 ±13	24 ±15	21 ±11	extreme
11	19	30	26	25	11 ±05	11 ± 04	14 ±06	variable
after 60 tsteps								
12	26	31	22	21	21 ±12	21 ±10	40 ±18	variable
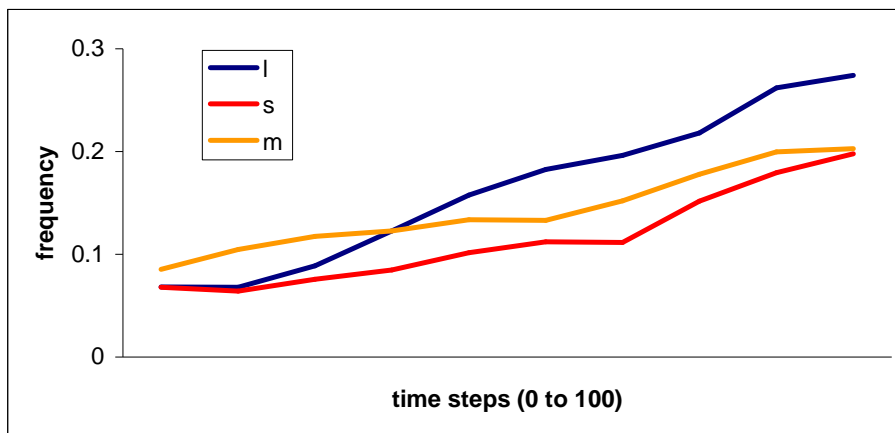
Figure 1: Frequency of occurrence of alleles *l* allowing recognition of toxic prey by predators (blue); alleles *s* signaling toxicity in toxic models (red); and alleles *m* mimicking the aposematic signal in mimics (orange). The figure represents the average of 10 simulations with 100 time steps each.
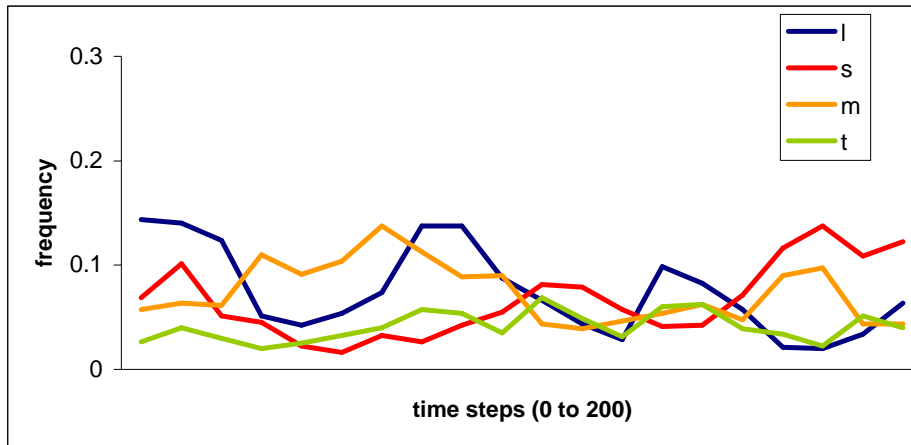

Figure 2: As in Figure 1 but toxic mimics had to acquire toxicity by evolving the corresponding allele of *t* for toxicity whose frequency is indicated by the green line.
The figure represents a single simulation with 200 time steps. More examples can be produced using Demo "Species1" from Biodynamica available at http://atta.labb.usb.ve/klaus/klaus.htm

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