The origin of mimicry
Deception or merely coincidence?

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Abstract. One of the most remarkable phenomena in nature is mimicry, in which one species (the mimic) evolves to imitate the phenotype of another species (the model). Several reasons for the origin of mimicry have been proposed, but no definitive conclusion has been found yet. In this paper, we test several of these hypotheses through an agent based co-evolutionary model. In particular, we consider two possible alternatives: (1) Deception, in which mimics evolve to imitate the phenotype of models that predators avoid to eat, and (2) Coincidence, in which models evolve a warning color to avoid predation, which coincidentally benefits the mimics. Our agent-based simulation shows that both these hypotheses are plausible origins for mimicry, but also that once a mimicry situation has been established through coincidence, mimics will take advantage of the possibility for deception as well.

1 Introduction

One of the most remarkable phenomena in nature is mimicry, in which one species (*mimic* animals) imitates the phenotype of another species (*model* animals). Typically, the effect is called mimicry when the model species are dangerous to predators. In this case, the mimic species benefits from mimicry when predators mistake the mimic animals for model animals. Depending on characteristics of the mimic species, the model species may benefit (Müllerian mimicry, [11]) or suffer (Batesian mimicry, [1]) from the presence of mimic animals. In this paper, we investigate two possible hypotheses for the origin of mimicry through an agent-based co-evolutionary model.

The pioneer in mimicry research was Bates [1]. Bates found that there are poisonous animals with very bright colors, and camouflaged animals which were not poisonous. Even though the brightly colored animals are more easily detected by predators, they were also identified as dangerous by these predators. This so-called *aposematism* effect became more remarkable when Bates found animals with similar colors and shapes as the toxic animals that were not toxic. This type of mimicry is called Batesian mimicry, in which non-toxic animals imitate the phenotype of toxic animals. This effect has been found in butterflies [11, 1], snakes [12], and various other animals [9].

In Batesian mimicry, mimic animals are not toxic. As a result, whenever a mimic animal is eaten or tasted by a predator and found to be harmless, this

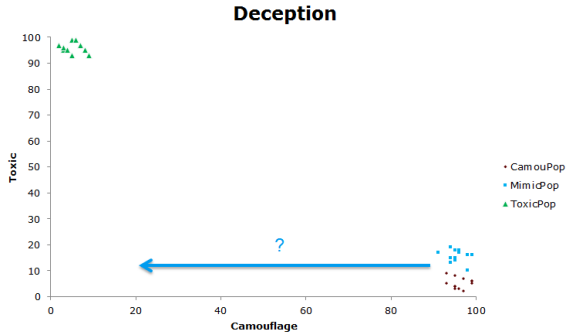

Fig. 1. In deception, the mimic and camouflaged animals start with the same characteristics. The hypothesis states that the mimicry group will move toward the place with low toxicity and camouflage: the mimicry place, since this place has the lowest evolutionary cost.

gives positive feedback to the predator to eat similar animals. This results in model animals being eaten more. Hence, the more mimic animals exist in the habitat of the model, the lower the survival chance of the model. This results in a negative or parasitical effect on the model. Müllerian mimicry [11], on the other hand, involves two species of animals that are both toxic to a certain degree, and therefore both contribute to this anti-predation mechanism.

In this paper, we investigate two possible hypotheses for the origin of Batesian mimicry. A common assumption in the literature is that the mimic animals deliberately deceive their predators by imitating model animals [7, 12]. That is, mimic animals evolve to have the same phenotype as the model animals because this lowers predation. However, mimicry may also come about through coincidence. That is, model animals may evolve a phenotype that allows predators to distinguish them, and which happens to be the phenotype of the mimic animals.

These two hypotheses will be tested through an agent-based co-evolution model. Agent-based modeling has proven its usefulness as a research tool to investigate how behavioral patterns may emerge from the interactions between individuals (cf. [4, 5]). Among others, agent-based models have been used to explain fighting in crowds [8], the evolution of cooperation and punishment [2, 13], and the evolution of language [3]. In this paper, we use agent-based modeling to test two hypotheses on the origins of mimicry. We will elaborate on the hypotheses in the next two sections.

1.1 Deception hypothesis

The deception hypothesis reflects the typical assumption about mimicry. According to the deception hypothesis, mimic animals evolve to have a phenotype that is as similar as possible to the phenotype of model animals. This benefits mimic animals because they are mistaken for animals that are dangerous to

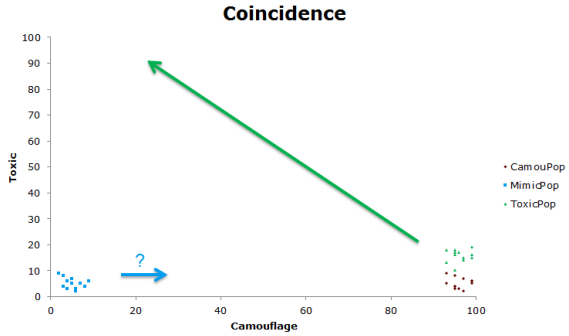

Fig. 2. In coincidence, the toxic model animals and the camouflaged animals start with the same characteristics. The mimicry group is already in place. The toxic population now gets more toxic, and moves toward the lesser camouflaged site.

predators. Therefore, if model animals are less camouflaged, the mimic animals are expected to evolve a lower level of camouflage as well.

This idea is represented graphically in Figure 1. The figure shows three types of prey: model animals (ToxicPop), mimic animals (MimicPop), and a control population of camouflaged animals (CamouPop). Only the model animals are dangerous to the predators, as shown by a high level of toxicity, while both the control and mimic animals start out with high levels of camouflage. The deception hypothesis predicts that, over time, the mimic animals take advantage of the eating behavior of predators and evolve lower levels of camouflage (blue arrow in Figure 1), since being camouflaged has a negative influence on their chance to reproduce. That is, the deception hypothesis describes a process of *speciation*, where one population of prey splits into a population of mimic and a population of control animals.

1.2 Coincidence hypothesis

In contrast with the deception hypothesis, the coincidence hypothesis describes a small role for the mimic animals. The coincidence hypothesis describes the situation in which the mimic animals do not change their phenotype, but that the model animals evolve a distinct phenotype, which happens to be the same as the phenotype of the mimic animals. Note that in this situation, there is an important role for the behavior of the predators. Predators learn to create a discriminatory line between the model animals and the control population, which drives the selective pressure for model animals to evolve a phenotype that is distinct from that of the control animals. The coincidence hypothesis states that the mimic animals may happen to be on the model animal side of this line, and therefore experience a coincidental benefit.

The coincidence hypothesis is described graphically in Figure 2. The figure shows three types of prey: model animals (ToxicPop), mimic animals (Mim-

icPop), and a control population of camouflaged animals (CamouPop). All these populations start out with low levels of toxicity, and are therefore harmless for the predators. In addition, the mimic animals start out with low camouflage, while the control and model animals have high levels of camouflage. The coincidence hypothesis predicts that when the model animals evolve higher levels of toxicity, they will also decrease their level of camouflage (green arrow in Figure 2). In addition, the coincidence hypothesis predicts that the mimic population would not increase its camouflage to more closely resemble the model animals (blue arrow in Figure 2). Note that the coincidence hypothesis also describes a process of *speciation*, but in this case, one population of prey splits into a population of model and a population of control animals.

The idea behind the coincidence hypothesis is that there are relatively few mimic animals who already have a distinctive color because of pre-adaptation [7]. The model animals experience selective pressure towards the phenotype of the mimic animals because of the relative low population sizes of the mimic animals compared to the population size of the control animals.

1.3 Structure of the paper

The remainder of this paper is set up as follows. In Section 2, we will discuss the simulation model, first in general terms and then in more depth. We present our simulation results in Section 3. Section 4 will discuss the results and provides directions for future research.

2 Model

While mimicry is defined in terms of the evolutionary behavior of prey animals, mimicry also depends on the behavior of predator animals. As a result, there are three different ways to study mimicry [10]:

- The evolutionary dynamics way, which studies the evolution of the prey but ignores the behavior of the predators [6];
- The receiver psychology way, which focuses on the behavior of the predators, but tends to ignore the evolution of the prey [7]; and
- The traditional natural historical way, which analyzes the behavior of both predator and prey. In these kinds of research, the co-evolution between predators and prey is studied [1].

In this paper, we follow the traditional natural historical way by explicitly modeling both the evolution of prey animals and the behavior of predator animals. To study this co-evolution, we construct an agent-based model that models individual prey and predator animals. In Section 2.1, we first give a general explanation of our model. A more technical discussion of the model can be found in Section 2.2.

2.1 Model description

Our model of mimicry investigates the co-evolution of predator and prey animals. Prey animals are further subdivided into three separate populations, which we will call the *toxic*, *camouflaged*, and the *mimic* populations.

Predator agents perform two actions: eating prey and reproducing. A predator consists of a neural network that determines whether a predator will eat a prey that it encounters. This network is evolved, which means that predators do not learn over their lifetime, but instead inherit their decision function from their parent. At each time-step of the model, the predator encounters a number of prey. For each encountered prey, the predator decides whether or not to eat the prey, depending on the prey's phenotype. Eating non-toxic prey increases evolutionary fitness, while eating toxic prey decreases fitness. Reproduction occurs by selecting the agent with the highest fitness from a random sub-set of predators as the parent. The child inherits all characteristics of this parent, subject to a small probability of mutation, which will be elaborated on in Section 2.2.

Prey are defined by three characteristics: camouflage, toxicity, and pattern. A prey's camouflage determines the probability of being detected, so that a higher camouflage lowers the probability of being encountered by a predator. A prey's pattern, on the other hand, does not influence the probability of being encountered. A prey's phenotype consists of its camouflage and pattern. That is, both camouflage and pattern are observable characteristics, while toxicity is a characteristic that cannot be observed by predators.

Prey reproduce by selecting two parents with the highest fitness from a random subset of the population. The fitness of a prey is determined by the number of times it is eaten by a predator. In addition, both toxicity and camouflage decrease a prey's fitness.

To investigate mimicry, prey animals are subdivided into three separate populations of constant size that reproduce independently. The first population, called the *toxic* prey, has a small genetic drift toward higher toxicity. This population is meant to simulate model animals. Similarly, the *camouflaged* prey experience a small genetic drift towards higher camouflage, and are meant as a control population. The third, *mimic* population does not experience any genetic drift.

2.2 Model details

In this section we look at the model in more detail. In particular, we take a closer look at the eating behavior and knowledge of the predator, the mechanism of reproduction, and the setup of different parameters.

Eating behavior of predators During every time step of the model, each predator encounters a fixed number $Y_{encountered}$ of randomly selected prey animals (see also Algorithm 1). For each of these encounters, the camouflage of the prey animal determines the probability with which the prey is found, so that

Algorithm 1 Eating behavior of predators.

```
let C_y camouflage of prey y, scaled [-50, 50]
let P_y pattern of prey y, scaled [-50, 50]
let NN(X, Y) neural network function of predators
for each encountered prey y do
    if random(100) > C_y then                          ▷ Prey y is found
        if NN(P_y, C_y) > random(1) then
            Eat prey y
        end if
    end if
end for
```

prey with a high camouflage are more likely to hide from the predators. If the predator finds the prey, it can choose whether or not to eat the prey.

The predator uses a simple feed-forward neural network to propagate the phenotype of the found prey, which results in a decision on whether or not to eat a prey. This neural network consists of two input nodes, which represent the prey's camouflage and pattern; three hidden nodes; and one output node that controls the predator's eating decision. The output node is implemented as a probability between 0 and 1, so that there is a low probability that predators will try to eat prey that they believe to be dangerous.

Each node is connected to all nodes in the next layer. This results in 6 synapses from the input nodes to the hidden nodes and 3 from the hidden nodes to the output node. The total number of synapses (weights) is thus 9 per predator. After each synapse round, an activation function is applied to scale the values between 0 and 1. The activation function used is the sigmoid function.

Reproduction The mechanism with which animals reproduce is different for predators than it is for prey. However, for the selection of the parents, both types use tournament selection.

Predators reproduce asexually, so that every child has a single parent. A child inherits the neural network from its parent, subject to a low probability of mutation. When a weight mutates, a value between -0.25 and 0.25 is added to it. The value is then cut to the domain between -2 and 2. Because of the survival of the fittest principle, the best predators evolve and anticipate on the changes within the prey. This way of learning can be seen as a random search.

Prey, on the other hand, reproduces sexually. The two parents with the highest fitness are chosen with the tournament selection, and the child is a combination of these parents. Each child has camouflage, toxicity, and pattern that is the mean of the corresponding characteristic of its two parents. The values of the prey characteristics have a value between 0 and 100. Each characteristic has a low probability of mutation, in which case a random number between -10 and 10 is added to it. If the new value exceeds the borders of 0 or 100, it is cut off at that value. In the case of genetic drift, the `Genetic Drift` parameter is added to the mutation value, giving more chance for an increasing mutation.

Table 1. Parameter settings used in the simulation runs.

Parameter	Predators	Toxic	Camouflaged	Mimic
Population size	10	300	300	30
Genetic drift	-	3	3	0
Prey encounters ($Y_{encountered}$)	3	-		
Mutation rate	20	2		
Tournament size	3	10		
Lifespan	3	5		
Chance-being-found	101	-		
Camouflage disadvantage	-	3.0		
Toxicity disadvantage	-	0.2		

For both predators and prey, reproduction occurs in generations. After each generation, all animals in the old generation die and are replaced by an identical number of new individuals. This means that there are no animals older than other animals, and that all animals die at the same moment after a predefined number of time-steps. This number of time-steps differs between predators and prey (see the lifespan parameter in Table 1) to reflect differences in learning. Note that prey animals do not die due to being eaten by a predator, but only die when their generation dies. Instead, the fitness of a prey decreases when it is eaten by a predator, reducing the chance for reproduction.

Parameters settings and fitness Within our simulation, the number of predators is fixed, as well as the number of prey within each subpopulation. In every run of the model, there are fewer predators than prey, corresponding to the real world.

The predators have a lower life span than preys, to reflect that they learn faster than the rate at which prey evolves. The prey becomes older, which makes the difference in fitness between preys which are eaten and that are not eaten bigger.

Each time-step, a predator encounters the number of prey divided by the number of predators. This is multiplied by $Y_{encountered}$ to make the selective pressure higher. From the point of view of the prey, it has $Y_{encountered}$ encounters with predators.

In our model, each individual prey and predator represents a group of animals. For this reason, prey does not die when it is eaten. Instead, the fitness of a prey animal y is determined by the number of times it is 'eaten' (E_y). In our model, we assume both toxicity and camouflage to be detrimental to fitness. The toxicity (T_y) and camouflage (C_y) of prey y are multiplied by the toxic disadvantage (TD) and camouflage disadvantage (CD) parameters respectively. For example, a prey with a toxicity of 80 and toxicity disadvantage 0.2 will experi-

ence a 16 point penalty to its fitness. The fitness of a prey is updated according to

$$F_y = -(T_y \cdot TD) - (C_y \cdot CD) - (500 \cdot E_y). \quad (1)$$

Note that the most detrimental effect to the fitness of a prey is to be eaten. In addition, a prey's fitness is a non-positive number, with 0 being the highest possible value.

The fitness of a predator is determined by what prey it eats, according to the following formula

$$F_r = \sum_{y \in Eat_r} (T_y - 60). \quad (2)$$

The formula shows that the fitness of the predator r (F_r) depends on the sum of the toxicity of all the prey it has eaten (Eat_r). Toxicity values are reduced by 60, so that predators increase their fitness whenever they eat a prey with toxicity lower than 60, and decrease their fitness otherwise.

3 Results

We used the model outlined in Section 2 to perform simulation runs, the results of which are discussed in this section. The results are divided in four different sections. In Section 3.1, we discuss the deception hypothesis. In Section 3.2, we investigate the coincidence hypothesis. For both these hypotheses, we show results from 100 runs of 14,000 time steps each. After this, in Section 3.3, we will discuss an individual run. Lastly, in Section 3.4 the difference between model animals and mimic animals in the different hypotheses will be discussed.

3.1 Deception hypothesis

For the deception hypothesis (also see Figure 1), the mimic and camouflaged populations start out with high camouflage. The toxic population starts at its final position with high toxicity and little camouflage (that is, brightly colored and toxic animals). The deception hypothesis predicts that the mimic population would evolve to decrease its camouflage while maintaining low toxicity.

Figure 3 shows the average camouflage of the three prey populations across 100 runs. The figure shows that the mimic population indeed decreases its level of camouflage over time. The camouflaged population also initially reduces its level of camouflage, but later returns to high camouflage levels. This can be explained by the genetic drift of the camouflaged population. However, the larger population size of the camouflaged prey also slows down the evolutionary process. In addition, predators find and eat more of the less camouflaged individuals than the more camouflaged individuals, which gives additional selective pressure to increase camouflage.

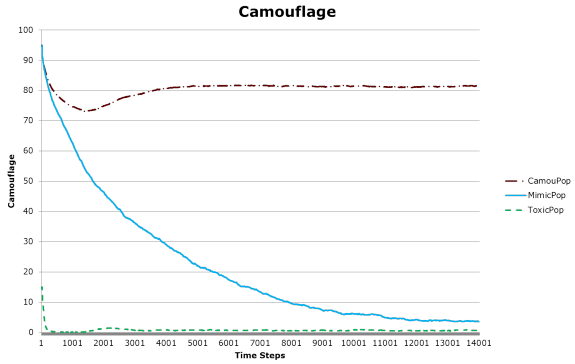

Fig. 3. The average camouflage value for each of the three prey populations across 100 runs for the deception hypothesis. The mimic and camouflaged populations start out with the same high camouflage, while the toxic population starts out with low camouflage.

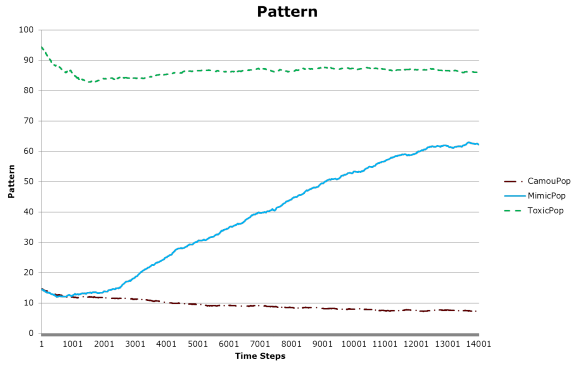

Fig. 4. The average pattern value for each of the three prey populations across 100 runs for the deception hypothesis. The mimic and camouflaged populations start out with the same pattern, which is different from the pattern of the toxic population.

In contrast, Figure 3 shows that the camouflage of the toxic population quickly drops and remains fairly stable. Since the toxic individuals rely on the predators' choice not to eat them, there is an evolutionary pressure to be as distinct as possible from other prey. In this case, the other prey have high camouflage, so the selective pressure encourages the toxic population to lower camouflage.

Figure 4 shows the average pattern of the three prey populations across 100 runs. In this figure, we can see that the pattern of the mimics moves toward that of the toxic population. Both Figure 3 and Figure 4 support the deception hypothesis, since the mimic moves from a position of high camouflage towards a position of no camouflage, thereby mimicking the toxic population. In addition,

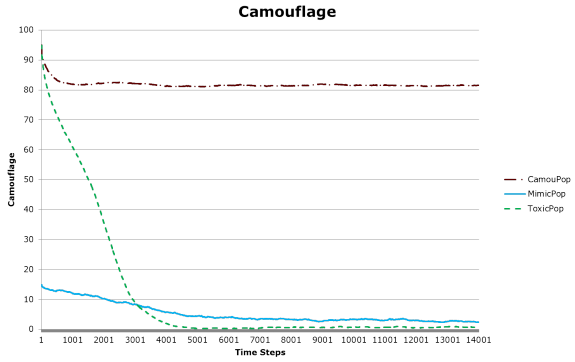

Fig. 5. The average camouflage value for each of the three prey populations across 100 runs for the coincidence hypothesis. The toxic and camouflaged population start out with the same high camouflage, while the mimic populations starts out with low camouflage.

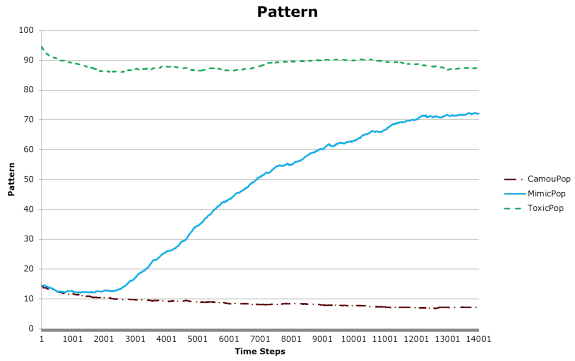

Fig. 6. The average pattern value for each of the three prey populations across 100 runs for the coincidence hypothesis. The mimic and camouflaged populations start out with the same pattern, which is different from the pattern of the toxic population.

the mimics also evolve to have the same pattern as the toxic group. That is, these results suggest that the mimics evolve to trick the predators into not eating them.

3.2 Coincidence

In the coincidence hypothesis, the toxic and camouflaged group start at the same position, with low toxicity and high camouflage. The mimic population, on the other hand, starts out with low toxicity and low camouflage. The coincidence hypothesis predicts that, in order to distinguish itself from the camouflaged population, the toxic population evolves to a position with high toxicity and

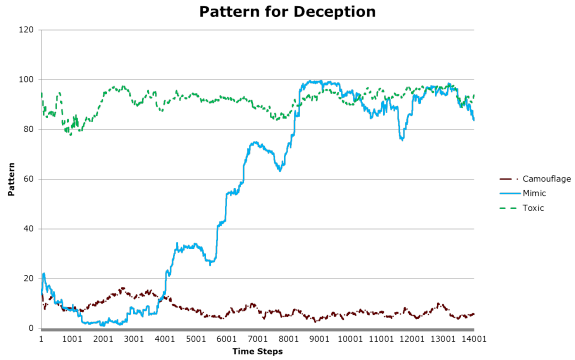

Fig. 7. The pattern of an individual run in the deception hypothesis. The pattern changes with two distinctive bumps, before being similar to the toxic population. After this we see the toxic population moving away from the pattern of the mimics, and the mimics chasing this pattern.

low camouflage, which coincidentally gives the same camouflage as the mimic population (also see Figure 2).

Figure 5 shows the average camouflage of the three prey populations across 100 runs. Note that while the camouflaged population maintains high camouflage, the toxic population evolves to lower its camouflage. In addition, Figure 5 shows that, on average, the mimic population does not increase its camouflage to increase resemblance with the toxic population.

Figure 6 shows the average pattern of the three prey populations across 100 runs. This figure shows that, while the mimic population does not increase its camouflage to resemble the toxic population, the mimic population does evolve to have a pattern that is similar to the toxic population. That is, while Figure 5 supports the coincidence hypothesis, Figure 6 is more suggestive of the deception hypothesis.

3.3 Individual run

While the average results presented in the previous sections give a good impression of the way the prey's phenotype (i.e., camouflage and pattern) evolves over time, closer inspection shows that the average does not fit any individual run particularly well. For this reason, we take a closer look at a representative individual run in this section.

Figure 7 shows the evolution of pattern for all three prey populations of a representative individual run of 14,000 time-steps. Note that while the average results (Figures 4 and 6) suggest that the pattern of the mimic population gradually evolves over 14,000 time steps, Figure 7 suggests a more rapid evolution. Indeed, individual runs typically show a rapid evolution of the pattern of the mimic population. The average results show a more gradual development because the moment at which this rapid evolution starts is different for each run.

Figure 7 shows that the pattern of the mimic population not only evolves in the direction of the pattern of the toxic population, but also continues to converge on the same value. In addition, due to the differences in population size of the mimic and toxic populations, the pattern of the mimic populations exhibits more volatility than that of the toxic population. This corresponds well with the idea of Holmgren and Enquist [7], who say:

> "For mimicry to be established, the movement of the mimic should always be faster than the movement of the model."

After approximately 9000 steps in the simulation, we notice the pattern of the model animals moves away from that of the mimic animals. When the mimics have a higher pattern, the models get a lower pattern and vice versa. This is consistent with the idea of [7]. In the results we can see that the pattern of the mimics changes faster, but the models try to distinguish themselves from the mimics. The reason for this is that model animals that are more simlar to mimic animals are more likely to be eaten by predators, since mimics are harmless for predators. Model animals that look less like the mimics therefore have an evolutionary advantage, a development we can observe in the individual run of the model.

Figure 7 shows that the mimic population changes its pattern in several bumps. These bumps can be explained by the model animals getting less toxic. As a result, the predators start eating more model animals. When we observe the model animals, we can see that they start losing their toxicity when the predators do not eat them, since toxicity is detrimental to individual fitness. However, when the toxicity becomes too low, predators start eating more model animals. In Figure 7, this effect is shown when the pattern of the mimics moves away from the pattern of the model animals.

3.4 Euclidean distance Model and Mimic

Figure 8 shows the distance in phenotype between model and mimic over time. This graph tells us that the distance gets smaller, thus mimicry is created. The euclidean distance is measured as the distance between the mean of the pattern and camouflage between the mimics and the models by the following formula:

$$EUD = \sqrt{(C_{mim} - C_{mod})^2 + (P_{mim} - P_{mod})^2} \tag{3}$$

For the deception hypothesis, the mimic moves towards the model to trick the predators. Over time, we can see that the mimic population becomes increasingly more similar to the model population. This means that a lot of the simulations evolve into mimicry. For the coincidence hypothesis, the toxic group evolves towards the same camouflage as the mimics. After this, the mimics follow the models over the phenotype plane. As a result, the average euclidean distance gets smaller. Since both distances are decreasing, the model supports both hypotheses for mimicry.

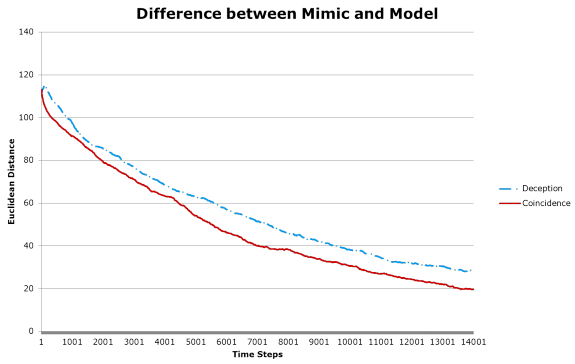

Fig. 8. The Euclidean Distance over time for both the hypotheses. In both hypotheses, the difference between model and mimic decreases over time, arguing for mimicry. The coincidence hypothesis has a little less difference. Keep in mind this is an average over 100 runs, and therefore is not a representation of one run, but a probability of mimic and model being the same.

4 Discussion

In this paper, we constructed an agent-based co-evolutionary model to investigate two possible origins of mimicry. The deception hypothesis predicts that due to selective pressure, mimic animals change their phenotype to resemble the model animals. In contrast, the coincidence hypothesis states that due to selective pressure, model animals change their phenotype to be different from some control population that has high levels of camouflage, and coincidentally get a phenotype that is similar to the mimic animals.

For both these possible origins of mimicry, we can say that they are plausible. The deception has very clear evidence in the deception set-up, where we can see that the mimic animals always change their phenotype to more resemble the phenotype of the model animals. This suggests that mimic animals indeed evolve to deceive predators. For the relatively small population of mimics, it is possible to explore new peaks in the adaptive plane, and successfully deceive predators. For the larger camouflage population, we can see that this population is too big to explore new adaptive peaks.

Our model results also show evidence for the coincidence hypothesis. Given the appropriate starting conditions, the model animals may change their phenotype to more resemble the mimic animals rather than vice versa. However, this can also be explained by the assumed negative fitness contribution of camouflage. This alone may result in both model and mimic animals to experience a selective pressure to reduce camouflage. Indeed, when we consider the pattern alone, the mimics again attempt to deceive the predators by evolving a pattern that resembles that of the model animals.

Note that we considered two different setups to determine the plausibility of the two hypotheses for the origin of mimicry. Both of these setups resulted in

the same ecosystem, with no possibility to determine what was the initial setup. That is, while our results show support for both hypotheses, they do not allow us to draw conclusions about which hypothesis matches best with biological data.

According to Holmgren and Enquist [7], the model animals always attempt to distinguish themselves from mimic animals. By creating distance from the mimics, the predators experience less confusion between model and mimic animals. However, since the mimics follow the phenotype of the models and evolve faster, this is an endless cat and mouse game. In our simulation model, this can be observed in the pattern, where the pattern of the mimic animals closely follows the pattern of the model animals.

Our simulation model can be used to do more research on theories of mimicry. Since the parameters can be easily adjusted, more experiments can be done. Firstly, more experiments can be done with different starting positions of the camouflage, toxicity and pattern values, starting with a control run. In this case, one population without genetic drift would be created to see how preys evolve without other animals. Another example is the toxicity and mimic group starting in the toxic position, and the camouflage on the camouflage position. This would make for another coincidence set-up, which assumes the speciation of the mimicry being a sub-population from the toxic population. Alternatively, the pattern can be altered. The pattern of the camouflage and mimic start the same in this paper, but it can be altered to a situation where the toxic and camouflage population start the same, and see whether the toxic population moves away. Besides this we can see whether the mimics move toward the models, which supports the Coincidence hypothesis.

The dynamics of the model can be altered as well. One possibility is adding more dimensions of recognition. This would mean that instead of 1 pattern, the model would have 50 patterns, which all can be mutated and inherited individually. In these recognition dimensions the scale between 0 and 100 can be removed, so the models and mimics can move through the adaptive space with more freedom. This way, neophobia and the idea of Holmgren and Enquist [7] can be researched in more detail. When the dimensions are implemented we hypothesize that the models will keep evading the phenotype of the mimics and the mimics chasing this phenotype. If the domains are removed, we expect very high and low values in the dimensions, arguing for the very bright colors of the animals.

In our model, we assume that prey consists of three populations of constant size that cannot interbreed. In future work, it would be interesting to see how removing this assumption influences our results. This would create hybrid populations of prey, which may have interesting properties.

To research Müllerian mimicry, more populations can be added which have intermediate values of genetic drift toward toxic. With two toxic populations, a research can be conducted whether the animals imitate each others phenotype or keep their own phenotype. The number dependent theory [11] can be tested in the same way. We hypothesize that when more toxic populations are implemented, there will be one center where all the animals converge to, to make one clear

aposematism. The representation of knowledge can be differentiated. At the moment, the predators have a line in their choice to eat camouflage or not. If a more curved line, or other methods are implemented, the idea of *Novelty and Recognizability* [10] can be researched in more depth. If this is combined with a variation of punishment for toxicity, we expect that neophobia emerge from the simulation.

Lastly, a spatial model can be created, in which agents have a x- and y-coordinate. This way mimicry rings can be researched, which are discussed in great depth by Holmgren and Enquist [10], and found by Bates [1]. When the spatial model is implemented, all the aforementioned can be combined in one simulation, since every place can evolve something else. Especially the borders of different mimicry systems will be interesting to research. Using a bigger adaptation space, better knowledge of the predators and a spatial dimension in the model, we aim to have a better understanding of the origin of mimicry in the future.

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