



# THE ORIGIN OF MIMICRY

Deception or merely coincidence

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**Abstract:** One of the most remarkable phenomena in nature is mimicry, the ability of one species (mimics) to imitate the phenotype of another species (models). Several reasons for the origin of mimicry have been proposed, but no definitive conclusion has been found yet. In this paper, different hypotheses will be tested by an agent based co-evolution model. We consider an adaptive plane with a camouflage and toxic gradient. Animal populations can move over this adaptive plane by evolving. Two options will be compared: (1) Deception, which assumes that mimics evolve to take advantage of the warning color of the models. (2) Coincidence, where models evolve a warning color that coincidentally benefits the mimics. We found that both the hypotheses are a plausible reason for the origin of mimicry, but that deception takes place in the coincidence environment as well. When the model loses its camouflage, the mimics do not move toward the model, but stay in their own place, which supports the idea of coincidence.

## 1 Introduction

One of the most remarkable phenomena in nature is mimicry, the ability of animals (mimics) to mimic the phenotype of other animals (models). Typically, the effect is called mimicry when the mimic imitates a phenotype of a model which can be dangerous for a predator. For the mimic, mimicry has a positive effect on the chance of survival, since it gets confused for the dangerous model. For the models, it might be beneficial (Müllerian, Müller (1879)) or disadvantageous (Batesian, Bates (1862)). Several reasons have been proposed for the origin of mimicry, but no definitive conclusion has been found as yet.

The first one to discover mimicry was Bates (1862). Bates found there are animals that are poisonous with very bright colors, and camouflaged animals which were not poisonous. The effect of having a bright color to scare off predators is known as aposematism. This effect became more remarkable when Bates found animals that had similar colors and shapes as the toxic animals, but were not toxic at all.

This type of mimicry has been called Batesian mimicry, the effect where non-toxic animals imitate the toxic animals. This effect can be found in butterflies (Bates, 1862; Müller, 1879), snakes

(Rabosky, Cox, Rabosky, Title, Holmes, Feldman, and McGuire, 2016) and other animals (Maan and Cummings, 2011). Various research has been done (Mallet and Joron, 1999), but no clear answer has been found for the origin of mimicry.

We distinguish Batesian mimicry and Müllerian mimicry. In Batesian mimicry the mimic is not toxic, which is disadvantageous for the model. The mimic will be eaten or tasted by the predator and found to be harmless, giving a positive feedback stimulus to the predator since the mimic is not poisonous. This results in models being eaten more, since the predators have found positive feedback on eating these colored animals. Hence, the more mimics 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 on the other hand, considers two animals that are both toxic to a certain degree, and therefore enhance each others aposematism. This will be explained by taking animals A and B, which both are toxic and have a similar phenotype. Each animal which starts to look more like the other one will be better remembered, and therefore have a better chance of survival. As a result an evolutionary co-operation mechanism is created.

## 1.1 Camouflage and toxicity

In mimicry we can distinguish between preys and predators, where the predators are hunting the preys. The preys have two defense mechanisms against the predators: (1) camouflage and (2) toxicity.

### 1.1.1 Camouflage

The camouflage of a prey can be beneficial to avoid detection of the prey. To stay undetected the animal needs a good camouflage color, which makes them undetectable for the predators. This causes animals with a higher camouflage to be eaten less (Stevens and Merilaita, 2009). However, when an aposematism is established, a prey that is camouflaged will signal the predators that the prey is safe to eat. Hence, when the camouflaged animals are found, the chance that a predator decides to eat them is very big.

The warning color, or low camouflage, can be beneficial since it scares off predators. However, a warning signal can be hard to establish since the prey has to deviate from its camouflaged color, which makes it more vulnerable to predator attacks. When aposematism is created, the warning colors can be useful. However, aposematism is hard feature to establish. In our research, we represent camouflage as an optical characteristic, but it can be applied to odor, sound, and taste as well.

### 1.1.2 Toxicity

Similarly, synthesizing toxic compounds can be beneficial when they serve their purpose, namely, scaring away predators. However, the first preys to explore this peak in the adaptive plane will still be eaten by the predators since these have not learned which preys are toxic.

The synthesis of toxic has a small negative influence on the fitness. The reason for this decrease in fitness is that these toxic acids or proteins have to be synthesized in the animal, which takes an adaptive cost. However, examples of animals can be found that feed on toxic flora, and use the residues to keep their toxicity intact. An example of this is the *Zygaena* (Holzkamp and Nahrstedt, 1994), which feeds on toxic plants and uses the residues for its own protection. Therefore, the content of toxic in the animals can be a very low cost adaption

in their evolutionary progress. Not all animals get eaten when predators learn of their toxicity. Predators tend to taste their prey first, before consuming them whole. Therefore preys do not have to die when they are tasted, but it is not beneficial for their health either. The evolution of toxicity is also explained by kin selection. When a species is more toxic, the group as a whole will be eaten less and have a benefit. As an individual it is less beneficial to be toxic, but since the group has a benefit when developing toxic, toxicity still evolves.

## 1.2 Hypotheses: Deception & Coincidence

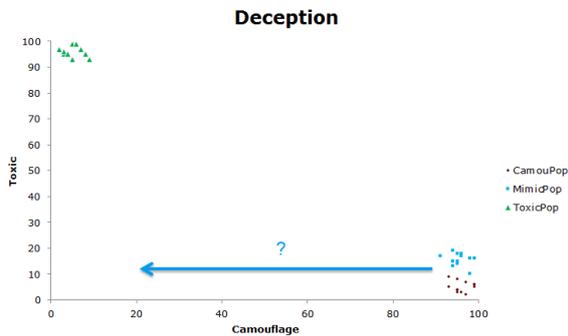
In our research we want to get a closer understanding of the origin of mimicry and the course of evolution in the preys and predators. A common assumption in the literature is that the preys deliberately deceive the predators (Holmgren and Enquist, 1999; Rabosky et al., 2016). However, this assumption can be argued by the idea of coincidence, which argues that mimics had the phenotype all along, and the toxic animals coincidentally got the same phenotype. These two hypotheses will be tested by a co-evolution model. We will elaborate on the hypotheses in the next two sections. This will be done by two definitions: speciation and the adaptive plane

*Speciation* is the term we use for populations which evolve in different sub-populations. For example, an animal population which is all camouflaged can split into one group which stays the same, and another which gets bright colors and becomes a mimic of another population.

*The adaptive plane* is used to explain the evolution of the preys. The plane has a camouflage and toxic dimension over which the populations move. A move is described as a change in features for a population. When the mean toxicity and camouflage changes, we say the population moves over the adaptive plane.

## 1.3 Deception by the preys

In deception, the preys have a dominant role in the evolution of mimicry. As seen from the perspective of the preys, the mimicry group will need to be as similar to the phenotype of the toxic group as possible, to make sure the predators do not mistake



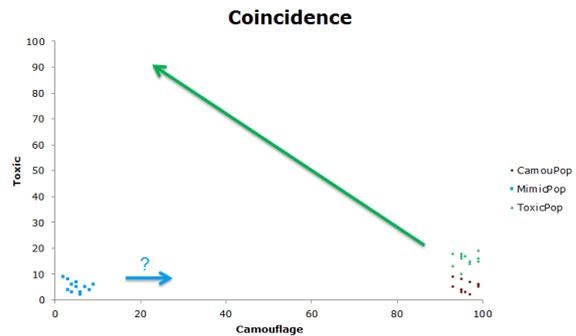
**Figure 1.1:** In deception, the mimic and camouflaged animals will start with the same characteristics. The hypothesis states that the mimicry group will move toward the place without toxic and without camouflage: the mimicry place, since this place is best if the animals do not get eaten.

them for a camouflaged animal. Therefore, when the toxic group is less camouflaged, the mimicry group will have to evolve to have a similar phenotype to maintain the mimicry, as can be seen in Figure 1.1.

Deception can also be explained in terms of speciation. When all the preys are starting as a big population, this is first divided in an camouflage non-toxic population and a non-camouflaged toxic population. This is the starting position for this hypothesis. After this the hypothesis is that the mimicry population is a sub-population of the camouflaged population, by lowering their camouflage. Since the warning color protects them from the predators, we expect that this is beneficial for the mimicry population.

## 1.4 Coincidence

The coincidence hypothesis assumes that the mimicry animals do not change their phenotype, but that the toxic animals evolve a distinct look, which happens to be the same as the mimics, as can be seen in Figure 1.2. The knowledge of the predators is important in this hypothesis. In the case of coincidence, the predators create a discrimination line between the toxic population and the camouflaged population, regardless of the mimic group. This idea is known as: *Pattern Enhancement and Peak Shift*, explained in 5. This results in the toxic



**Figure 1.2:** In coincidence, the toxic and camouflaged animals will start with the same characteristics. The mimicry group is already in place. The toxic population now gets more toxic, and consequently moves toward the lesser camouflaged site. Our hypothesis is that the mimicry group will move towards the toxic group first, after which it moves back with the toxic group.

group moving to the less camouflaged part of the adaptive plane, to create a better distinction between them and camouflaged animals. The mimicry group can stay at the brightly colored place, since the predators take them for toxic animals.

How does mimicry evolve out of this? One explanation is the recognition of poisonous colors in animals. This is explained by the learning of the predators. They learn to recognize the preys that are not camouflaged as toxic and will stop eating them. The mimicry preys can benefit from this by having the same phenotype as the models.

Coincidence can be explained in terms of speciation as well. The big population is first divided in two non-toxic sub-populations, a camouflaged and non-camouflaged (mimic) population. The mimics already have a distinctive color, which can be explained by pre-adaption(Section 5). After this, a sub-population of the camouflaged group becomes toxic, by changing their diet, and starts to lose camouflage to distinguish themselves from the camouflaged population. Now, the mimics and models start to look similar by accident, not because of the mimics, but because of the models.

## 2 Number-dependent theory

A lot of research has been done on mimicry, but still there are no convincing proofs of the origin of mimicry. The number-dependent theory will be elaborated, since it can explain both Müllerian and Batesian mimicry.

Müller (1879) argued for the number-dependent theory, which states that the purifying effect of non-mimicry animals becomes stronger when there are more animals eaten. The purifying effect is the working of the survival of the fittest, and the selection of fit animals compared to less fit animals. With a strong purifying effect, the weak animals have almost no chance of reproducing and only the fittest animals will reproduce. Contrary to this, when there is a weak purifying effect, the weaker animals have a bigger chance of reproducing by accident.

The number-dependent theory can be explained by the following equations. Let  $a_i$  the number of animals in a given sub-prey-population and let  $n$  be the number of animals which are eaten before the predators have learned which animals they should not eat (the learning rate). Lastly, we define  $A$  as the total number of preys with a certain phenotype and  $E_i$  the number of preys eaten per population  $i$ . When all species are totally dissimilar ( $a_i = A$ ), the loss for all species will be  $n$ . However, if populations have the same phenotype, the losses are given by the following equation:

$$E_i = \frac{a_i * n}{A} \quad (2.1)$$

By this formula, Müllerian and Batesian mimicry can be explained. For Müllerian we can derive the following. When there are a lot of animals which look similar,  $A$  gets bigger, and therefore  $E_i$  gets smaller. In other words, fewer preys of each sub-prey population are needed to teach the predators what is toxic. Therefore it has an evolutionary advantage for a species to look a lot like other species.

However, this model only holds when all species are equally toxic, since the predators always get a negative feedback from eating a prey. For Batesian mimicry the formula changes, since the mimics are not toxic and the models are. In this case, the mimics have a negative effect on the learning rate. To establish a mimicry ecosystem, the ratio of mimics to models can not be higher than 1, since the predators

will get more positive reinforcement from eating than punishment. To explain Batesian mimicry we substitute  $n$  for  $n'$  in which  $n'$  is updated according to

$$n' = n + n * \frac{a_{mimic}}{a_{model}}. \quad (2.2)$$

In Equation 2 we can see how  $n'$  is dependent of the ratio number of mimics ( $a_{mimic}$ ) to number of models ( $a_{model}$ ). When there are a lot of mimics, the predators will have more difficulty learning the toxicity of the animals, meaning a larger  $n'$ . As a result, the mimics have a parasitical behavior towards the models. In this situation the equilibrium will be found when there are enough models to keep the predators scared for the color, and the number of mimics can not increase because the predators would eat too much.

Müller argued that the ratio between mimic and model is important in mimicry research. When the mimic is more abundant than the model, the predators will take the mimics for general, and start hunting the model again. On the other hand when there is a rare mimic, the predators will not change their behavior, leaving the mimic model ecosystem in balance. This can be explained by using Equation 2. When the non-toxic mimic is a very small part of  $A$ , the effect on the learning rate of the predators is minimized.

Holmgren and Enquist (1999) argued that that models have to change their phenotype from the mimics because the more they resemble the mimics, the bigger the chance is that they are eaten. This is explained with the formula, where we can see that if the model moves away from the mimic,  $a_{mimic}$  decreases, and therefore the number of animals that need to be eaten decreases. This explains that the most brightly colored preys will have a bigger chance of survival and the population of models will evolve to brighter colors. However, the mimics will receive more benefit when they resemble the models more, and will therefore evolve to have a similar phenotype as the models. Mimicry can only be sustained if the movement of the mimics is faster than the movement of the models.

Mallet and Joron (1999) found that selective pressure is determining in the outcome of mimicry research. When a large part of the preys is attacked by predators (large  $E_i$ ), selection will be stronger and therefore the ecosystem will not change much. On the other hand, when fewer preys are attacked,

the preys will experience a relaxation of selection. Due to this decrease of selection in the population the preys have more opportunity to experiment, causing the population to shift its balance. This way sub-populations are formed to discover other parts of the adaptive landscape.

In this paper, the origin of mimicry will be researched. Two hypotheses will be tested: deception and coincidence. Besides this, the idea of Holmgren and Enquist (1999) will be verified. Lastly the ideas of Mallet and Joron (1999) will be discussed, which are elaborated in Section 5.

The remainder of this paper is set up as follows. The ideas of Mallet will be discussed in section 5. In section 3 we will discuss the model, first general and then in more depth. We present our results in section 4. Section 6 will discuss the results and compare this to the ideas presented in section 1 and section 5.

### 3 Methods

There are 3 different ways to study mimicry (Mallet and Joron, 1999).

- The evolutionary dynamics way, which studies the evolution of the preys but ignores the behavior of the predators. (Gavrilets and Hastings, 1998)
- The receiver psychology way, which focuses on the behavior of the predators, but tends to ignore the evolution of the preys. (Holmgren and Enquist, 1999)
- The traditional natural historical way, which analyzes the behavior of the predator and prey. In these kinds of research, the co-evolution between predators and preys are studied (Bates, 1862).

In this research the natural historical will be used by combining the evolution of the preys and the behavior of the predators. To study this co-evolution of these animals, a model will be created. First we will give a general explanation in Section 3.1, after which the more technical discussion can be found in Section 3.2.

#### 3.1 Model Description

In this paper, a model is presented that simulates the co-evolution of predator and prey to investigate the evolution of mimicry. The predators and preys will co-evolve until mimicry is established, after which the parameters and genetic development of the agents will be analyzed.

The model involves two types of agents: predators and preys. The preys are further divided in three populations: toxic, camouflaged and mimic.

*Predators* have three abilities. Eating preys, learning from their mistakes and reproducing. The predators consist of a neural network to store the memory of the preys. This memory evolves, so there is no adaptive memory in the individual predators. Each time-step, the predator encounters a number of preys. These preys can be found or stay disguised depending on their camouflage. When the prey is found, the predator decides whether or not to eat the preys, depending on the phenotype of the prey. After this, it receives fitness if the animal is not toxic, and loses fitness if it is toxic. When giving birth, the best parent is chosen out of a random sub-set of predators, and copied into the child. The child then has a small chance on mutations, which will be elaborated on in Section 3.2.

*Preys* have one ability, giving birth. Besides this the preys have three characteristics: camouflage, toxicity, and pattern. All of these features range from 0 to 100. To give birth, the fittest two parents are found and the child is created out of the parents. The fitness of the preys is determined by the camouflage, toxicity and times the prey is eaten by a predators. The more camouflage or toxicity a prey has, the less fitness it has. Pattern is added to have an independent value only for the use of signalling the phenotype to predators. This pattern has no influence on fitness, but can be used by predators to recognize the prey.

The preys consist of three populations which can only reproduce within populations. The first population of toxic preys has a small genetic drift toward more toxicity. This genetic drift exists of a small tweak in the mutation. When an animal is mutated, there will be slightly higher probability of increasing toxicity than of decreasing it. The genetic drift of the camouflage works in the same way for the camouflaged population. The mimics have neither drift towards toxic, nor towards camouflage. In the

simulation the toxic population reflects the model, and the mimic population reflects the mimic. The camouflage population is an example for the predators which they can eat. The camouflaged group is also used as a control group, to see if the mimic population has unique behavior compared to other populations. In this paper the term phenotype is used for the combination of camouflage and pattern.

The model makes no use of the spatial properties of animals and is dependent on camouflage for finding preys, and fitness for finding parents. Terms like movement and location of the populations refer to the place on the adaptive plane. There is no location modelled.

## 3.2 Model Analysis

In this section we look at the model in more detail. The eating behavior and knowledge of the predator is discussed in Section 3.2.1, the reproduction of animals in Section 3.2.2, and the setup of different parameters in Section 3.3.

### 3.2.1 Eating of the predators

The eating of the predators is dependent on two steps. This can be seen in Algorithm 3.1. The first is (1) the probability of an animal being found. Camouflage makes the probability to be found lower, so that preys with a high camouflage have a bigger chance to hide from the predators. (2) The choice of the predator. The predator uses a simple feed-forward neural network to propagate the phenotype. The phenotype of the prey lets the predator decide whether to eat a prey or not. This way, toxic animals can distinguish themselves by having a very distinguishable phenotype. The network exists of two input nodes, three hidden nodes and one output node. The nodes are connected by synapses, which all contain a weight. As input, camouflage and pattern of the preys are used, both scaled between -50 and +50. As output a probability of eating is generated, between 0 and 1. The decision threshold is then chosen randomly, so that there is a slight chance predators will still try to eat bright animals occasionally. Each node is connected to all nodes in the next layer. This results in 6 synapses from the input node to the hidden nodes and 3 from the hidden nodes to the output node. The

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### Algorithm 3.1 Prey Eaten

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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 function of predators
if  $random(101) > C_y$  and  $NN(P_y, C_y) >$ 
 $random\_float(1)$  then
    Eat  $Prey_y$ 
end if

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### Algorithm 3.2 Choose Parent

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let  $[P1, P2, P3 \dots Pn]$  random animals
let Parent  $maxFitness([P1,P2,P3\dots Pn])$ 

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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. The learning of the predators is explained in Section 3.2.2.

### 3.2.2 Reproduction

To reproduce, the predators and preys use different principles. For the selection of the parents, both the animals use tournament selection, which works as found in Algorithm 3.2.

*Predators* When the parent is chosen, the child is made as a clone of the parent. The way of reproduction is iteroparous, that is, parents can have several children in their lifetime. For predators the reproduction is different from the preys, in the way that they are asexual. In asexual reproduction only one parent is needed for reproduction, which means that the weights of the predator are copied and given to the child. After this, each weight has a chance of mutation. When a weight is mutated, 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 preys. This way of learning can be seen as a random search.

*Preys* The preys use sexual, iteroparous reproduction with tournament selection. When the two best parents are chosen with the tournament selection, the child is made as a combination of these parents. Different than the predators, the preys have sexual reproduction, meaning that there are

**Table 3.1: The Parameter List shows the different parameters which were used in the model**

Parameter	Predators	Preys
Number of individuals (Tox. = 300, Cam. = 300, Mim. = 30)	10	630
Genetic drift	-	3
Times Prey Found( $Y_{encountered}$ )	3	-
Mutation rate	20	2
Tournament size	3	10
Age	3	5
Chance-being-found	101	-
Camouflage Disadvantage	-	3.0
Toxicity Disadvantage	-	0.2

two parents needed to create a child. There is no distinction made in gender, so that every animal can reproduce with every other. The child is then created by taking the mean of camouflage, toxicity and pattern and giving this to the child. The values of the prey have a value between 0 and 100. The child has a chance of mutation, in which case the features are added by a random number between -10 and 10. If this value exceeds the borders of 0 or 100, it is cut off at that value. In the case of genetic drift, the *GeneticDrift* parameter is added to these numbers, giving more chance for an increasing mutation.

*Generations* The reproduction works in generations, and in each generation the old generation dies. This means that there are no animals older than other animals, but all animals die at the same moment after a predefined number of time-steps. This number of time-steps is not the same for predators and preys, and can be found in Table 3.1 as the age parameter. The preys will survive an attack of a predator, and only die when their generation dies. However, the fitness of a prey deteriorates when it is successfully attacked by a predator, making the chance for reproduction minimal.

### 3.3 Setup of the Parameters

The number of predators is fixed, as well as the number of preys in a population. In every run of the model, the number of predators is lower than the number of preys, corresponding to the real world.

The predators have a lower age than preys, since they learn faster than the preys evolve. The death

of a predator represents a day, after which the predator has learned which animal it should not eat and which it should eat. 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 preys divided by the number of predators. This is optionally 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. The encounters of the predators ( $En_r$ ) can be approximated as follows

$$En_r = (\#Preys/\#Predators) * Y_{encountered}. \quad (3.1)$$

The encounters for each prey ( $En_y$ ) is given by

$$En_y = Y_{encountered}. \quad (3.2)$$

The disadvantages of toxic(T) and camouflage(C) are multiplied by the toxic disadvantage(TD) and camouflage disadvantage (CD) respectively. For example, a prey with *toxicity* = 80 and toxicity disadvantage 0.2, will subtract 16 from the fitness of the prey. The fitness of a prey is updated according to

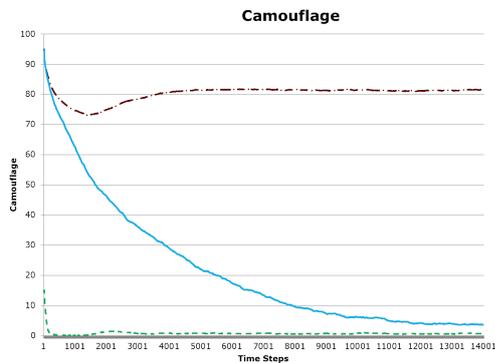
$$F_y = -(T * TD) - (C * CD) - (500 * E_y). \quad (3.3)$$

Here we can see that the fitness of the preys ( $F_y$ ) is calculated by subtracting the toxicity and camouflage, multiplied by their respective disadvantage from 0. After that, 500 is subtracted from the fitness, for each time it gets eaten ( $E_y$ ). The fitness is a value below 0, with 0 being the highest value.

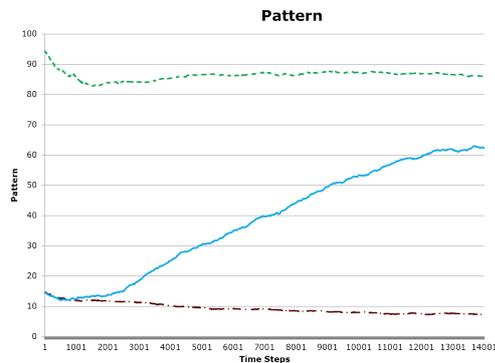
The fitness of the predators is calculated in a different way than the prey fitness. The fitness is determined by the eating of toxic or non-toxic animals, according to the following formula

$$F_r = \sum_{y=1}^{En_y} (E_y(\text{toxic}) - 60). \quad (3.4)$$

Here we can see that the fitness of the predators ( $F_r$ ) depends on the sum of the toxicity of all the eaten preys ( $E_y$ ). All toxicity values are subtracted by 60, so predators get positive feedback when eating animals with lower toxicity than 40, and negative when eating animals which have a higher toxicity value.



**Figure 4.1:** The average camouflage for 100 runs for the deception hypothesis. The starting position of the mimicry and camouflage are the same, and the toxic populations starts low. The mimicry population moves slowly towards the toxic population. This is an average over 100 runs, but one transition from much camouflage to little takes little time. The line can be used as the percentage of runs which already has little camouflage at the given moment.



**Figure 4.2:** The average pattern for 100 runs for the deception hypothesis. We can see that the mimics and the camouflaged animals are initialized at the same position, but the mimics move towards the toxic population. Same as in Figure 4.1 this is an average of 100 runs, so it is not an representative of one run.

## 4 Results

The results are divided in 4 different sections. In Section 4.1 the deception hypothesis will be tested. In Section 4.2 the coincidence hypothesis will be tested. For both of these hypotheses we will show results from 100 runs, in 14000 time steps. After this, in Section 4.3, we will discuss the peculiarities of an individual run, and verify the theorem of Holmgren and Enquist (1999). Lastly, in Section 4.4 the difference between model and mimic will be discussed.

### 4.1 Deception

For the first hypothesis, the mimic and camouflaged population start out with high camouflage. The toxic population starts at it's final position with high toxicity and little camouflage(bright colors). See Figure 1.1 for the graphical view. Proof for deception would be the movement of mimics toward the less camouflaged side, while maintaining their low toxicity level. In Figure 4.1 and 4.2 we can see the average camouflage and pattern over 100 runs. We can see that the mimic population moves towards the lower camouflage level. The camouflaged population moves in the direction of less camou-

flage as well, but due to a big population and the genetic drift is not able to continue. We can see that the camouflage of the toxic population is fairly stable. In the pattern we can see that the pattern of the mimics moves toward the toxic population. These graphs support the idea of deception, since the mimic moves from the camouflaged place in the adaptive plane towards the non-camouflaged place, mimicking the toxic population. Besides this, the mimics use pattern to get an evolutionary advantage by having the same pattern as the toxic group. We can see that the mimics evolve to trick the predators into not eating them.

### 4.2 Coincidence

In the coincidence hypothesis, the toxic and camouflaged group start at the same location, without toxic and camouflage. The toxic group then gets a genetic drift toward more toxicity. The starting situation can be found in Figure 1.2. The question was whether the mimic group would move toward the toxic population (deception) or stay at the same place (coincidence). We can see in Figure 4.3 that, on average, the mimics do not move towards the toxic population. This is an argument for coincidence. When we look at the pattern (Figure 4.4), however, we can see that the mimics try to deceive the predators by using the same phenotype as the

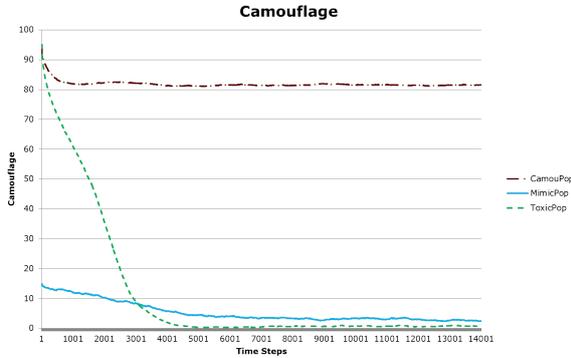


Figure 4.3: The average camouflage for 100 runs for the coincidence hypothesis. Notice that the starting positions of toxic and camouflage are the same, and the mimics are already in place. We can see the toxic population dropping fast, decreasing the camouflage. When we take a look at the mimics, we see that the population does not move towards the toxic population.

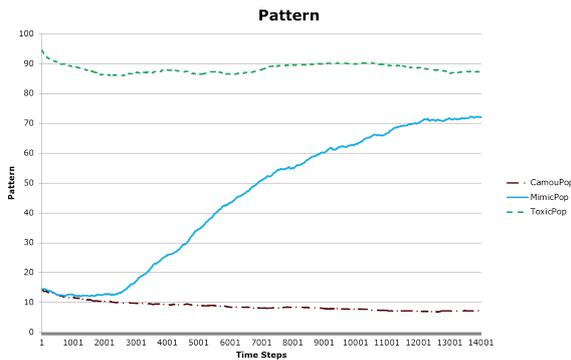


Figure 4.4: The average pattern for 100 runs for the coincidence hypothesis. We can see that, same as in the deception hypothesis, the mimics move towards the toxic population.

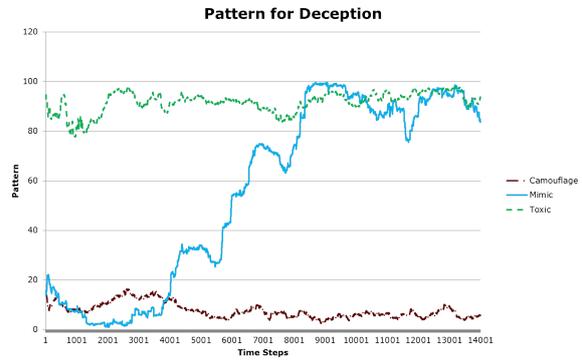


Figure 4.5: The pattern of an individual run in the deception hypothesis. The pattern changes with two distinctive bumps, before being similar to the toxic population. After this we see the toxic population moving away from the pattern of the mimics, and the mimics chasing this pattern.

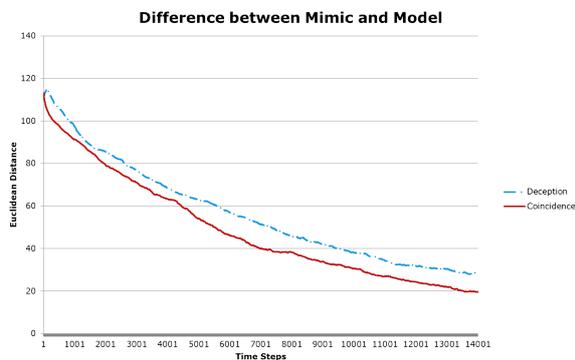
models. The camouflage supports the idea of coincidence, but the pattern supports deception.

### 4.3 Individual Run

Figure 4.5 shows the pattern of an individual run of 14000 time-steps of the simulation. Using this figure we can evaluate the idea of Holmgren and Enquist (1999), which said: "For mimicry to be established, the movement of the mimic should always be faster than the movement of the model". Besides this we will take a look at *Kin Selection*, explained in Section 5.

After approximately 9000 steps in the simulation, we notice the pattern of the models moves away from the mimics. When the mimics have a higher pattern, the models get a lower pattern and vice versa. This is consistent with the idea of Holmgren and Enquist (1999). In the results we can see that the movement of the mimics is faster, but the models try to escape this. This can be explained by the mimic and model population. For models it is disadvantageous to look like the mimic, because the predators will get positive feedback from eating a mimic. Therefore do the models which look less like the mimics have an evolutionary advantage, a development we can observe in the individual run of the model.

We can see that the the mimic changes its cam-



**Figure 4.6: 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.**

ouflage in several bumps. These bumps can be explained by the models getting less toxic and the predators eating them. When we observe the model, we can see that the models start losing their toxicity when the predators do not eat them. This is explained by the individual fitness of the preys. However, when the toxicity drops below 40, which is a positive influence for the predators, the predators will start eating again. This is seen in Figure 4.5 when the mimic moves away from the pattern of the model. Now *Kin Selection* appears for the models. It is as a group better to be toxic, and therefore the whole population starts to be toxic again. When the models are not toxic, it is better for the mimics not to look like the predators, but to have more camouflage. In the choice of the predators, we could see that a discrimination line was made throughout the phenotype plane. As a result, the camouflaged preys were eaten if found, the toxic animals were often skipped. The mimics had to be on the right side of this discrimination line to stay alive. This means there is a form of pattern enhancement, explained in 5.

## 4.4 Euclidean distance Model and Mimic

Figure 4.6 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:

$$EUD = \sqrt{(C_{mim} - C_{mod})^2 + (P_{mim} - P_{mod})^2} \quad (4.1)$$

For the Deception hypothesis, the mimic moves towards the model, to trick the predators. Over time, we can see the more and more mimic populations look similar to models. This means that a lot of the simulations evolve into mimicry. For the Coincidence hypothesis, the toxic group goes towards the mimics first. After this, the mimic follows the model over the phenotype plane. This can be seen as the average Euclidean Distance gets smaller and smaller. Since both distances are decreasing, the model supports both hypotheses for mimicry.

## 5 The Origin of Aposematism and Mimicry

The results of the model can be used to verify certain possible hypotheses, stated by (Mallet and Joron, 1999).

1. *Novelty and Recognizability* can be explained by the predators fearing new colors, which is called neophobia. When predators do not know a color, they fear it, and will refuse eating the prey. The model shows that predators make a discrimination line which animals they do and which they do not want to eat. New patterns or camouflages would always fall into one of these categories. The discrimination line can be seen as a threshold to which the predator decides to eat an animal. With another knowledge representation this origin may be simulated, but not with the simple neural network we used in this research. We hypothesize that when the punishment for eating toxic is very big, predators will not eat newly found animals. However, when this is not the case, predators can

experiment in their eating behavior, making the probability of neophobia smaller.

2. *Preadaptation* Another reason for evolving aposematic features is found before the creation of toxicity. Butterflies for example, need bright colors to attract mates and in territorial male-male interactions. This causes bright colors to evolve in preys, without the toxicity having evolved yet. A change in diet can then cause preys to synthesize toxic, resulting in aposematism. This hypothesis is close to the coincidence hypothesis, since the mimics preadapt to a bright color, after which the toxic animals get bright colors as well. The model shows that, even if the mimics already had bright colors, they would not move toward the camouflage of the toxic animals. Thus, the model supports preadaptation. The idea of preadaptation will be elaborated on in Section 6.1.
3. *Pattern Enhancement and Peak Shift* assumes the memory of the warning colors in the predator is a symbolic representation of the actual colors. This implies that preys which resemble the warning color more will have a bigger opportunity to survive. In the adaptive plane this would mean that it would always be better to have less camouflage because this is more similar to the general representation. This is the case in the model, since the predators take every non-camouflaged animal for a toxic animal. For the mimics, this made it possible to signal a more dangerous signal than the models by having less camouflage. In the pattern we could see that the mimics evolved towards the most toxic location for the predator. Additionally, when the models changed their phenotype, the mimics evolved with them, according to the idea of Holmgren.
4. *Müllerian Mimicry* As discussed earlier, Müllerian mimicry occurs when two animals of similar toxicity start to co-evolve. This means that animals will start to look more alike when combining their distinctive features. When this is combined with another species later on, all these patterns are joined together create a wide variety of aposematic features. However, Müllerian mimicry was not simulated.
5. *Density- and Appearance-dependent Warning Color* This kind of aposematism assumes a phenotype which can change during the lifetime of an animal. When many animals are eaten it would be better to look toxic, whereas when there are not many eaten, it would be better to maintain a camouflage. Since we look at the evolution and genetics of mimicry, this topic is not simulated in the model.
6. *Kin Selection* This way of evolution has an altruistic approach, as it looks at groups as a whole. When a few animals die to scare predators off for the rest, can this be beneficial for the group. Other preys, carrying the same genetics, can evolve to a more aposematic look. This hypothesis is tested by creating populations which evolve together, making the group as a whole better instead of only single animals. For each individual it is better to have less toxic, since this has a negative influence on their fitness. However, for a group as a whole it is better to be toxic, since predators will stop eating the group. It was found that the toxic group emerged in this kind of group behavior. We observed that the toxic group became less toxic as the aposematism was created, until they reached a toxic value of 40 (the toxic threshold for the predators). After passing this, the predators started eating the toxic group again, and the toxic group used kin selection to become toxic again.
7. *Genetic Drift & Shifting Balance* The shifting balance theory consists of three phases, which were found in the model. In phase 1, a subpopulation explores a new adaptive peak due to genetic drift. This was found as the mimics moved away from the camouflaged group to explore another adaptive peak. In phase 2 the population gets fully adapted to this adaptive peak. We found this in the pattern of the preys which evolved to a similar pattern as that of the models. In phase 3 the populations co-evolves with other populations in the same adaptive peak. This happened as the mimics evolved together with the phenotype of the

The simulation has possibilities to research this topic, which will be more elaborated on in Section 6.3

models, even if the models evolved to another phenotype.

## 6 Discussion

During this last section we will discuss the achieved results, and compare them to our hypotheses. In Section 6.1 we will discuss the deception and coincidence hypotheses. We answer the question whether Deception and Coincidence are plausible origins of mimicry. In Section 6.2 the idea of Holmgren and Enquist (1999) is discussed. We show that their idea is found in our model. After this we will discuss the new implementations and usages that can be used by this model in Section 6.3.

### 6.1 Deception and Coincidence

For both the 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 mimics always move toward the models. Similarly, we can see that in the pattern the mimics evolve toward the models to deceive the predators. For a small population of mimics, it is possible to explore new peaks in the adaptive plane, and change their phenotype to get into the mimic place. For the camouflage population, we can see that this population is too big to explore new adaptive peaks.

The coincidence has evidence in the change of camouflage, since we can see that the mimics do not move towards the models in their appearance, but stay in their own place. We can assume that the mimics do not try to mimic the models, when the camouflage has an influence on their fitness. However, when this influence is removed, as can be seen in pattern, the mimics will deceive the predators, and choose the looks of the models.

Both of the hypotheses set-ups concluded in the mimicry ecosystem. However, as soon as the ecosystem is created, it is not possible to see which set-up the system came from. Therefore, we can support both hypotheses, but can not conclude which one matches best with biological data.

### 6.2 The idea of Holmgren and Enquist (1999)

Holmgren argued that the models always move away from the mimics, according to the number dependent theory which was discussed in Section 2. By creating distance from the mimics, the models experience less confusion from the predators. However, since the mimics follow the phenotype of the models and evolve faster, this is an endless cat and mouse game. In the model this can be observed in the pattern, where the mimics circle around the models. However, no quantitative measurement has been done so far, which leaves space for future research.

### 6.3 Future experiments and implementations

The 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 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. 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 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* 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 Mallet and Joron (1999), and found by Bates (1862). 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 adaption 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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