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Report

Predators' Toxin Burdens Influence Their Strategic Decisions to Eat Toxic Prey

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Summary

Toxic prey advertise their unprofitability to predators via conspicuous aposematic coloration [1]. It is widely accepted that avoidance learning by naive predators is fundamental in generating selection for aposematism [2, 3] and mimicry [4, 5] (where species share the same aposematic coloration), and consequently this cognitive process underpins current evolutionary theory [5, 6]. However, this is an oversimplistic view of predator cognition and decision making. We show that predators that have learned to avoid chemically defended prey continue to attack defended individuals at levels determined by their current toxin burden. European starlings learned to discriminate between sequentially presented defended and undefended mealworms with different color signals. Once birds had learned to avoid the defended prey at a stable asymptotic level, we experimentally increased their toxin burdens, which reduced the number of defended prey that they ingested in the subsequent trial. This was due to the birds making strategic decisions to ingest defended prey on the basis of their visual signals. Birds are clearly able to learn about the nutritional benefits and defensive costs of eating defended prey, and they regulate their intake according to their current physiological state. This raises new perspectives on the evolution of aposematism, mimicry, and defense chemistry.

Results and Discussion

Predator cognition plays a key role in the evolution of aposematism and mimicry, where prey advertise their defenses by warning signals, most notably conspicuous color patterns [7]. Although it seems paradoxical that prey should advertise their presence to predators, aposematic prey may benefit from naive predators learning to avoid defended prey more readily when they are conspicuously colored compared to when they are cryptic [2, 3, 8]. The ways in which naive predators learn to associate warningly colored prey with their defenses and remember to avoid them in future encounters now underpins an extensive body of evolutionary theory [5, 9–12], which in turn has inspired a wealth of empirical studies [2–4, 8, 13–16]. However, this learning-focused approach has failed to consider the role of predators once they have acquired the association between the warning coloration and the noxious effects of the toxin. This is a fundamental omission because it is impossible to fully measure selection pressure on aposematic prey without considering how educated predators use their acquired knowledge throughout their lifetimes [17–20].

Perhaps most significantly, there are a number of observations of adult predators attacking and ingesting defended insect prey [21–24]. Although this could be the actions of naive individuals or those with specialist physiological adaptations, an alternative explanation is that educated predators trade off the costs of ingesting toxins with the benefits of valuable nutrients and make strategic decisions to attack and consume defended prey [25]. Consequently, a predator's ability to moderate and process toxins would be a key factor in limiting attack rates on chemically defended prey, and one that could have significant implications for the survival advantage of being aposematic.

In this experiment, we test the idea that educated predators are a significant selective force in the evolution of aposematism and continue to make strategic decisions to consume defended prey based upon their acquired knowledge of the toxic and nutritional content of the prey. By manipulating the toxin burden of generalist avian predators feeding on insect prey, we are able to uncover novel cognitive processes underlying their informed decisions to ingest defended prey, which raise new challenges to current theories of aposematism and mimicry.

In the initial training phase of our experiment, ten adult starlings (*Sturnus vulgaris*) learned to discriminate between mealworm larvae (*Tenebrio molitor*) that were either undefended or defended. On each of 5 consecutive days, subjects were given a single training trial consisting of 16 sequential presentations of 8 chemically defended mealworms (injected with quinine sulfate to make them toxic and coated with Bitrex to make them distasteful; see Experimental Procedures for details) and 8 undefended mealworms (injected and coated with water). Each prey type had a distinct color signal to enable the birds to learn to visually discriminate between the undefended and defended prey, as they would in the wild. Subjects invariably attacked and ate all the undefended prey offered to them during the training phase (all ten birds attacked and ate all eight undefended mealworms in all five training trials). In contrast, birds rapidly learned to attack and eat fewer defended prey than undefended prey even from the very first trial (see Figure 1). However, birds' asymptotic attack rates on defended prey were significantly higher than zero; they continued to readily include them in their diets.

Although the fact that birds continue to eat the defended prey could reflect a limited association between the color signal and the effects of the toxin, we predict that birds had learned the association but were choosing to eat defended prey. In this manner, birds

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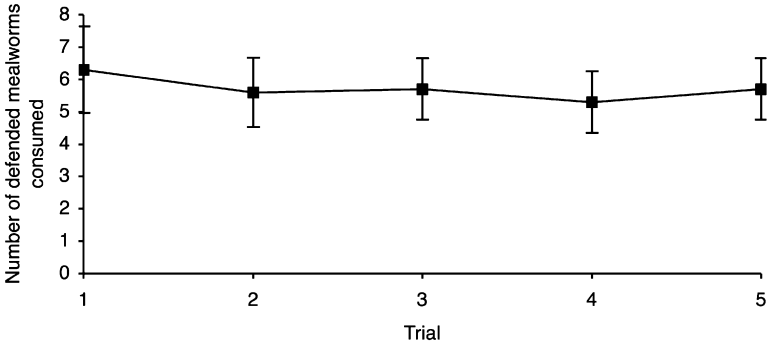

Figure 1. Birds Learned to Avoid Defended Prey

The mean number (± standard deviation) of defended mealworms attacked and eaten in the five training trials. The number of undefended mealworms are not shown because birds invariably ate them in all presentations in all trials.

could regulate their toxin ingestion while exploiting the nutritional value of the defended prey. In the crucial test of this hypothesis, we conducted two further trials in which the toxin burden of each subject was manipulated to test how it affected predators' decisions to ingest defended prey. The toxin manipulation trials followed the same protocol as the training trials, except that before each trial, birds were prefed with three mealworms that were injected with either water or quinine sulfate solution. These mealworms had no associated color or taste cues by which water or quinine could be detected upon attack, and birds showed no hesitation in eating all the mealworms offered prior to the start of each trial. All birds continued to eat all of the undefended prey that were offered in both toxin manipulation trials, whether they had been prefed with water or with quinine. However, birds consistently ate fewer defended mealworms after being prefed with quinine than after being prefed with water (Wilcoxon test, $Z = 2.831$, $p = 0.005$, $n = 10$; see Figure 2). In fact, when prefed with three quinine-injected mealworms, birds reduced their consumption of defended prey by, on average, 2.6 worms, suggesting that they can control the amount of toxin that they are willing to ingest very accurately indeed. We also compared the number of defended mealworms eaten within a trial, by summing the numbers of defended mealworms consumed in the first four presenations with those in the last four. We summed the data across all five training trials and found that birds ate more of the defended worms in the first half of the mealworm presenations compared to the second half (Wilcoxon test, $Z = 2.831$, $p = 0.005$, $n = 10$). Consistent with our hypothesis, birds were therefore reducing the number of defended mealworms ingested as their toxin burdens increased within a trial.

A short series of further trials confirmed that birds had learned to use the color signals and not chemical cues to discriminate against defended prey (see Figure 3). We also ensured that our results were not the result of quinine suppressing birds' appetites; all birds consumed 16 undefended mealworms when prefed with 8 quinine-injected mealworms (see Appetite Test in Experimental Procedures).

Our data clearly demonstrate that avian predators do not simply learn to avoid defended prey, but continue to ingest them at a stable asymptotic rate. Crucially, we have evidence to support the idea that birds have post-ingestive feedback mechanisms to monitor the amount of toxin ingested, and that this information is used to make strategic foraging decisions about when to eat defended prey to regulate their toxin burdens. Although we do not know how the quinine is detected once ingested, one potential explanation is that quinine, like many insect toxins, is an irritant and may make birds feel queasy by irritating the wall of the crop or gut [26]. This would allow birds to detect differences in toxicity between prey items ingested in quick succession (our experiment suggests that a 3 min interval between successive encounters is sufficient) and allow birds to discriminate between toxic and nontoxic prey in the wild.

Regardless of the exact mechanism, the fact that birds make strategic foraging decisions about when to consume toxic prey to regulate their toxin burden and include defended prey in their diets provides new insights into the evolution of aposematism and allows us to make novel predictions about the evolution of insect defenses. First, the initial evolution of aposematism may be more difficult to explain than previously thought. Understanding the initial evolution of aposematism is problematic because it is unclear how a conspicuously colored mutant could survive long enough to pass on its genes to the next generation given that it is easier to locate than its conspecifics [7]. If prey survive predatory attacks, and predators quickly learn to avoid

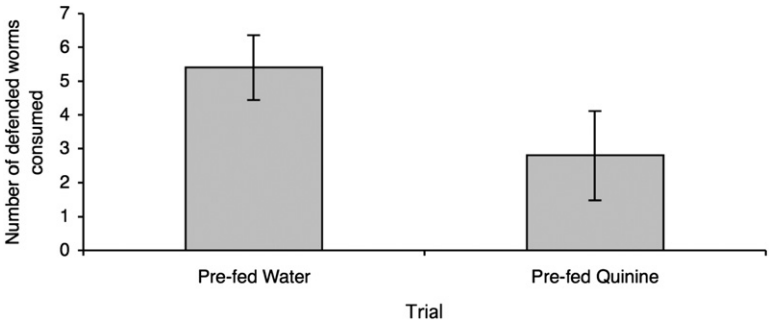

Figure 2. Increasing Birds' Toxin Burdens Decreased Their Consumption of Defended Prey

The mean number (± standard deviation) of defended mealworms attacked and consumed in the toxin manipulation trials. Birds ate fewer defended mealworms when they were prefed with quinine-injected mealworms compared to when they were prefed with water-injected mealworms.

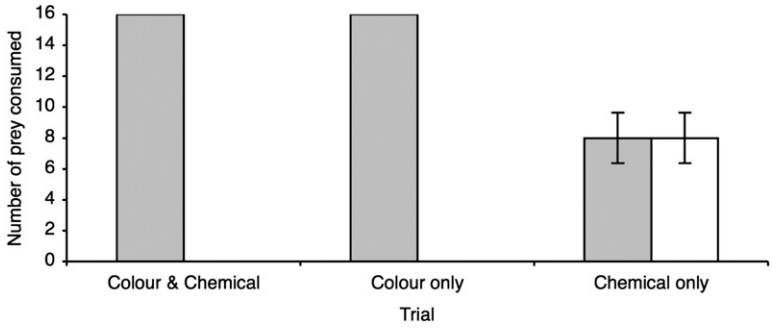

Figure 3. Birds Used Color Signals to Identify Defended Prey

The mean number (± standard deviation) of choices for undefended mealworms (filled bars) and defended mealworms (unfilled bars) in each of the simultaneous choice trials. In the color and chemical trial, all birds invariably chose the mealworms that were undefended. In the color-only trial where only color cues were available, birds also chose only the undefended mealworms. However, in the chemical-only trial where the color signal was not available, birds were unable to distinguish between defended and undefended mealworms and attacked both types equally (Wilcoxon test $Z = 1.0$, $p = 0$). Taken together, these trials demonstrate that birds had learned to discriminate between defended and undefended prey solely on the basis of their color signals.

aposematic prey, this problem is not insurmountable [6, 12]. However, our results suggest that the first conspicuous mutants would have to survive greater levels of predation than previously thought, because even when the learning process is complete, educated predators may still be prepared to eat aposematic prey.

Second, this study highlights the need to consider the selection pressures exerted on aposematic prey across the entire lifespan of a predator. Although research focusing on the learning processes of naive predators has clearly been informative [2, 3, 8, 16], the knowledge acquired by birds about the toxic content of prey clearly plays a central role in their decisions to attack and consume defended prey. It therefore becomes essential that we understand what factors influence birds' decisions to eat defended prey because these factors may well influence the evolution of aposematism and mimicry. This is important because such factors may have been ignored in the past because they were not thought to influence the learning process. For example, the nutrient content of toxic prey, although rarely considered in studies of aversion learning, will in all probability influence birds' decisions to eat toxic prey because birds are likely to trade off the costs of detoxification with the benefits of ingesting nutrients [18, 20, 22]. The ability of avian predators to detoxify defense chemicals will also be important because this will determine how frequently birds can consume defended prey. Interestingly, the physiological process of detoxification may be influenced by ecological factors such as predator state [20] and the availability of the macronutrients required for the detoxification process [27]. As a result, the availability and nutrient content of alternative palatable prey in the environment becomes even more important than initially thought in the evolution of aposematism and mimicry. Consequently, understanding the evolution of aposematism requires us to understand the foraging ecology of predators, not simply the avoidance learning process. The importance of the avoidance learning may therefore have been overstated when modeling the evolution of aposematism and mimicry, and as a result, these evolutionary models must be re-evaluated.

In light of our results, our understanding of mimicry may also need substantial revision. In Müllerian mimicry, two sympatric defended species share very similar visual signals [9]. Assuming that predators eat a fixed number of individuals when learning to associate the prey's shared visual signal with toxicity, then the number of individuals killed is shared across the two species, meaning that fewer individuals of each species are killed [9, 28, 29]. Our results suggest that a similar mutualistic relationship may also occur between sympatric aposematic species that are visually distinct. We would expect to find Müllerian mutualism between species that do not look alike, particularly in cases where different species sequester the same toxin [30]. This is because birds are prepared to ingest only a certain amount of toxin. As a result, if a bird eats an individual of a species that it knows to be toxic, it will benefit every individual the bird knows to be toxic because the amount of toxin the bird can now ingest is reduced.

Our results may also have implications for the agricultural industry. It is common practice for arable farmers to spray seeds with sublethal doses of toxins prior to sowing, in order to prevent birds from eating the seeds before they germinate [31, 32]. However, these deterrents are not particularly effective, and there is evidence that birds continue to eat seeds treated in this manner [31]. Our results suggest that this could be because birds are trading off the benefits of ingesting the nutrients found in the seeds with the costs associated with eating the chemical deterrents. If this is indeed the case, we would predict that the efficacy of avian chemical deterrents could be increased by providing birds with an additional chemically defended food source of greater nutritional value than the chemically defended crop: birds would eat this in preference to the crop because of its higher nutrient content and would increase their toxin load to a level that prevented them eating chemically defended crops.

In addition, our data raise the possibility that the use of chemical deterrents in agriculture could alter the population dynamics of defended insects. If birds eat seeds that have been sprayed with chemical deterrents, they may need to reduce their consumption of chemically defended prey to maintain a safe toxin burden, reducing the benefit of individual insects investing in costly chemical defenses. We know little about how the use of toxic agrichemicals influences predation on defended insects, but this needs to be considered when developing

conservation strategies for commercially important defended insects found in farmland ecosystems.

Our results therefore raise a number of important issues in relation to the evolution of aposematism and mimicry and identify potentially fruitful areas for future research. The study of aposematism has often been distanced from the mainstream study of diet selection, perhaps because of researchers' preoccupation with the avoidance learning process. However, we have demonstrated that educated predators make strategic decisions to eat defended prey, so the evolution of aposematism should now be considered in a broader dietary context (e.g., [33]). Our results also highlight the potential interactions between insect and environmental/agricultural toxins. Clearly, understanding the cognitive mechanisms underlying foraging decisions in birds will be important not only in providing novel theoretical insights but also in potentially developing practical conservation and landscape management strategies.

Experimental Procedures

Subjects and Housing

Ten adult European starlings *Sturnus vulgaris* (six males and four females) were caught in Northumberland and held under English Nature license (No. 20062404). The birds were housed individually in wire cages measuring 45 × 75 × 45 cm, with a drawer at the bottom that could be pulled in and out. They were subject to a 10L:14D cycle via florescent lights, and temperatures were maintained at 16°C–17°C (see [34] for further details). Water was provided ad lib., as were Zoofood Pheasant breeder pellets and fruit except during training and experimenting, when short periods of food deprivation were necessary. All deprivation periods and experimental procedures were in accordance with Home Office regulations and guidelines. At the end of the experiment, birds were returned to a free-flight before being released back into the wild.

Preparation of Artificial Prey

We used mealworms (*Tenebrio molitor*) of similar length (~20 mm) for both undefended and defended prey. Defended mealworms were injected through the mouthparts with 0.02 ml of 2% quinine sulfate solution by means of a hypodermic needle. Quinine at high concentrations is known to be toxic to humans [26] and birds [35], but at low concentrations is used widely as an aversant in learning experiments across a range of taxa (e.g., [4, 36–38]. However, because our data suggest that birds cannot taste quinine when it is administered in this way [34], defended mealworms were made additionally distasteful by coating them with 0.02 ml of Bitrex solution (4 drops of a 2% Bitrex preparation made up to 100 ml with water). Bitrex is a nontoxic bitter-tasting solution used to deter humans from ingesting toxic compounds. Bitrex is also detectable by birds [39, 40], although it is not an aversant for starlings at this and higher concentrations (J.S. and C.R., unpublished data). It was therefore an external chemical cue of internal toxicity to enable rapid avoidance learning. Undefended prey were mealworms that were injected with 0.02 ml of water and coated with 0.02 ml of water.

Training Trials

During the training phase (days 1–7), every subject was given a single training trial on each of 7 consecutive days. Birds were food deprived for 2 hr prior to the start of a trial. Each subject was then placed in its home cage behind a white curtain erected in the same room. The curtain visually isolated subjects from both the experimenter and conspecifics. Birds were monitored with video cameras connected to televisions that were positioned to prevent other birds from seeing the screens. The bottom of the curtain was level with the base of the cage, allowing the drawer to be pulled in and out with minimum disturbance. Each bird had 5 min to acclimatize behind the curtain before the start of a trial.

The procedure was the same for all trials. A trial consisted of 16 prey presentations, in which the drawer of the cage was pulled out, a Petri dish containing a mealworm was placed in the drawer, and the drawer replaced. Petri dishes were used to prevent chemical contamination of the cage floor, and allowed discs of paper to be placed underneath to signal prey type. Subjects were given 1 min to attack the mealworm before the drawer was pulled out and the Petri dish and the paper circle were removed. If the mealworm was attacked and eaten, the Petri dish and the paper circle were removed immediately. Mealworms were presented every 3 min, allowing pairs of birds to be run in parallel, with the presentation of mealworms to one bird corresponding with the interpresentation intervals of the other.

On the first 2 days, birds received single training trials where all 16 mealworms were unmanipulated and palatable and did not have any associated color cues. Subjects readily attacked the prey and ate all 16 mealworms presented in the second trial. On the following 5 days, birds were then given single training trials in which a sequence of 8 defended prey and 8 undefended prey were presented to the birds. Defended and undefended prey were presented in random sequences generated prior to the experiment: unique prey sequences were produced for each trial for each bird. Each prey type was signaled to the birds via purple and green paper discs placed under the dishes, which was balanced across subjects.

We recorded the numbers of undefended and defended mealworms attacked and eaten during each trial. Although the birds could use both color and chemical cues to distinguish between the prey types, they relied upon the visual signals, as shown by the fact that all prey that were attacked (and tasted) were subsequently eaten. Therefore, all our analyses are based upon the numbers of prey eaten.

Toxin Manipulation Trials

At the end of the training phase, subjects received two further trials on consecutive days (days 8 and 9) that followed the same protocol as the training trials. However, prior to each trial, birds received three additional prey presentations of mealworms that contained either 0.02 ml of water or 0.02 ml of 2% quinine sulfate solution. These mealworms did not have any additional cues by which the presence of quinine could be detected, i.e., there were no colored discs or Bitrex used. Half of the birds received the water-injected mealworms on the first day and quinine-injected mealworms on the second day, and for the other five birds, the order was reversed. We recorded the numbers of undefended and defended mealworms eaten during each trial.

Simultaneous Choice Trials

On days 10–12 of the experiment, birds were given three trials where prey were presented simultaneously in the 16 presentations. These trials were designed to test birds' preferences and to assess which cues they used in their decision making. In each presentation, an undefended and a defended mealworm were placed in adjacent Petri dishes (approximately 10 cm apart). In eight presentations, the defended prey was placed on the right hand side of the cage, and for the other eight, it was placed on the left. Birds were allowed to attack only one of the mealworms before the second dish and mealworm were removed. We recorded the number of choices that birds made for each prey type (birds consumed all prey that were chosen). In the first simultaneous choice trial, birds were given the choice between undefended and defended prey with both visual and chemical (Bitrex and quinine) signals present (color & chemical trial). The following day, the same choice was given with only the visual signals present (color-only trial), and finally birds had the choice with only the chemical cues (Bitrex and quinine) present (chemical-only trial).

Appetite Test

One potential criticism of our experiment is that our results may depend on quinine reducing birds' appetites, thereby reducing their attacks on defended prey. Therefore, on day 13, birds were given a final trial of 24 presentations to test whether or not quinine suppressed ingestion of mealworms. The first eight presentations were of a single mealworm injected with 0.02 ml of quinine sulfate solution with no color or taste cues. The next 16 presentations were undefended mealworms (injected with 0.02 ml of water and coated with 0.02 mlof water) with the appropriate color signal that the bird had previously learned. We recorded the prey that were

attacked and eaten, although birds systematically ate all of the prey that were presented. The maximum number of defended mealworms that a bird ate during the experiment was eight, so quinine was not suppressing birds' appetites to the point that they could not eat all the mealworms presented in a trial.

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