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Invited Review

Learning about aposematic prey

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The question, "Why should prey advertise their presence to predators using warning coloration?" has been asked for over 150 years. It is now widely acknowledged that defended prey use conspicuous or distinctive colors to advertise their toxicity to would-be predators: a defensive strategy known as aposematism. One of the main approaches to understanding the ecology and evolution of aposematism and mimicry (where species share the same color pattern) has been to study how naive predators learn to associate prey's visual signals with the noxious effects of their toxins. However, learning to associate a warning signal with a defense is only one aspect of what predators need to do to enable them to make adaptive foraging decisions when faced with aposematic prey and their mimics. The aim of our review is to promote the view that predators do not simply learn to avoid aposematic prey, but rather make adaptive decisions about both when to gather information about defended prey and when to include them in their diets. In doing so, we reveal what surprisingly little we know about what predators learn about aposematic prey and how they use that information when foraging. We highlight how a better understanding of predator cognition could advance theoretical and empirical work in the field.

Key words: **antipredator, defenses, learning, memory, mimicry, prey.**

A QUICK HISTORY LESSON

It was Alfred Wallace, in his correspondence with Charles Darwin, who originally suggested that the conspicuous coloration of Lepidopteran larvae could have evolved to alert predators to the presence of toxins (Wallace 1867). This correlation between warning coloration and toxicity became known as "aposematism" (Poulton 1890), and this term is now used to describe the antipredator strategy of using conspicuous and/or distinctive signals in any sensory modality to warn predators of a defense across a wide range of taxa (Mappes et al. 2005). At around the same time as Wallace, naturalists were also seeing cases of mimicry, where undefended or defended species shared the warning patterns of sympatric aposematic species (Bates 1862; Müller 1879). These phenomena later became known as Batesian and Müllerian mimicry, respectively, and along with aposematism, they have engaged the interest of evolutionary biologists since their first conception (see Figure 1).

It is fair to say that even the early naturalists appreciated the importance of predators' cognitive abilities in the evolution of aposematism and mimicry. Early theorists couched their explanations in terms of "predator avoidance learning," where predators learn to associate aposematic preys' visual signals with the noxious effects of their toxins and avoid them, and their mimics. Using one of the earliest mathematical models in the field of evolutionary biology, Fritz Müller demonstrated that if predators took a fixed number of encounters to learn to identify defended prey, then the cost of educating predators would be shared between 2 mimetic species (Müller 1879). More recently, interest has turned to exploring the broader idea that warning coloration has "special effects" on predator cognition and behavior. This has included investigating not only how predators learn to associate warning signals with toxicity (Gittleman and Harvey 1980; Ham et al. 2006) but also how they remember warning signals (Roper and Redston 1987; Ham et al. 2006), how they generalize their experiences (Gamberale-Stille and Tullberg 1999; Darst and Cummings 2006), and how they initially respond to novel aposematically colored prey (Roper and Cook 1989; Schuler and Roper 1992; Rowe and Guilford 1996; Gamberale and Tullberg 1998). Associative learning has long been, and continues to be, at the very heart of both theoretical models and empirical studies of aposematism and mimicry (Müller 1879; Leimar et al. 1986; Speed 1993; Alatalo and Mappes 1996; Servedio 2000; Lindström et al. 2001b; Speed 2001; Turner and Speed 2001; Skelhorn and Rowe 2006c; Skow and Jakob 2006; Lee et al. 2010), and alongside studies of the genetic architecture of defensive traits (e.g., Mallet and Joron 1999; Beldade and Brakefield 2002; Jiggins et al. 2005) and the physiological costs of producing and/or consuming toxins (e.g., Pasteels et al. 1983; Cohen 1985; Rowell-Rahier and Pasteels 1986; Bowers 1992; Dobler and Rowell-Rahier 1994; Zalucki et al. 2001), it continues to be a key factor underpinning our current understanding of the evolution of prey defenses.

Although viewing predators' decisions to attack and eat defended prey as the result of a classic case of associative learning is certainly appealing, this is likely to be an oversimplification of predator

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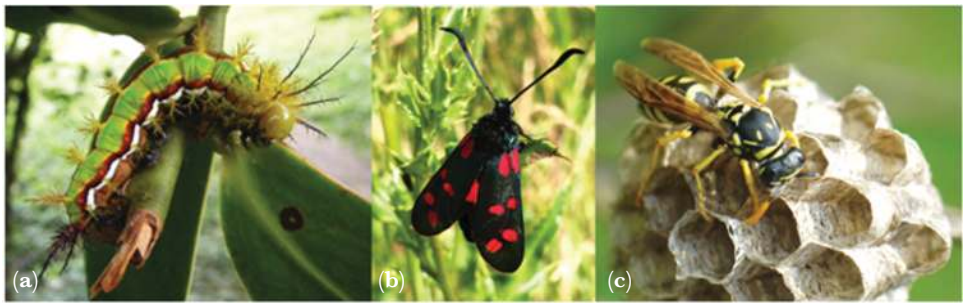

Figure 1
Examples of aposematic prey. (a) Larva of the *Dirphia horcana*. Image by T.J. Hossie. (b) *Zygaena filipendulae*. Image by H.M. Rowland. (c) *Polistes dominula*. Image by T. Reader.

behavior. In particular, we are starting to uncover the complexities of predator decision making and the trade-offs that predators are making when deciding whether or not to attack defended prey. Many authors have suggested that predators can profit from eating aposematic prey when the cost of ingesting toxins is outweighed by the benefits of obtaining the nutrients that these prey contain (Marshall 1908; Speed 1993; Kokko et al. 2003; Sherratt 2003; Sherratt et al. 2004; Rowland, Mappes, et al. 2010), and more recently, attempts have been made to understand how predators should optimize their investment in gathering (and using) information about toxic prey (Sherratt 2011; Halpin et al. 2012; Barnett et al. 2014; Sherratt et al. 2015). The aim of our review is to consider how predators acquire information about aposematic prey and how they use that information in their foraging decisions. We will concentrate our review on toxic aposematic prey, as opposed to those that have physical defenses such as spines or irritating hairs. Toxicity is one of the most common forms of defense utilized by aposematic prey, and it is certainly the most well studied (e.g., Calvert et al. 1979; Marples 1993; Mappes and Alatalo 1997b; Speed 2000; Ruxton et al. 2004; Barnett et al. 2007; Skelhorn and Rowe 2007b; Halpin et al. 2008a; Skelhorn and Rowe 2010; Speed and Ruxton 2014). Similarly, because other reviews have discussed the visual perception of warning signals and their special effects on predator learning (Rowe and Guilford 1999; Mappes et al. 2005; Stevens and Ruxton 2012), we will mention this only briefly and instead concentrate on how predators gather and use information about the toxins and nutrients that aposematic prey contain. In doing so, we reveal what surprisingly little we know about even the most basic (and intensively studied) aspects of predator cognition and highlight how taking a mechanistic approach to the study of prey selection can advance theoretical and empirical work in the field.

THE CLASSIC VIEW OF PREDATOR AVOIDANCE LEARNING OF APOSEMATIC PREY

Predator avoidance learning describes the process by which predators learn to reduce their attacks on aposematic prey over repeated encounters, by associating a prey's warning signals with its toxicity (e.g., Berenbaum and Miliczky 1984; Sillén-Tullberg 1985; Roper and Redston 1987; Gamberale and Tullberg 1996; Lindström et al. 2001b; Ham et al. 2006; Skelhorn and Rowe 2006b; Prudic et al. 2007). Avoidance learning has been widely observed in numerous studies, where predators reduce their attack rates on defended prey over a series of test sessions (see Figure 2). The shape of this

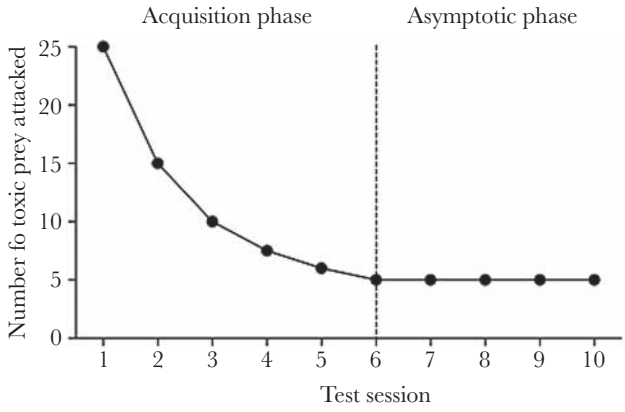

Figure 2
Data from a hypothetical learning experiment showing how predators reduce their attacks on a population of defended prey over repeated test sessions. Individual predators are simultaneously presented with 100 undefended and 100 quinine-coated prey (quinine is both distasteful and toxic to predators; refs.) in each of 10 test sessions. In each session, each predator is allowed to forage freely and attack 50 prey of any type. Over the 10 sessions, the predators reduce their attacks on toxic prey. There are 2 phases to this learning curve. The first is an acquisition phase, which is thought to show the speed with which a predator learns to associate the warning signal with the defense. The second is an asymptotic phase, where predators appear to have learned the association and do not change their attack rate. See the main text for further explanation.

"learning curve" is thought to be largely determined by the classic rules of Pavlovian conditioning, where a conditioned stimulus (CS) becomes associated with an unconditioned stimulus (US) (Pavlov 1927). In the case of aposematism, the warning signal is the CS that becomes associated with the noxious effects of the toxins, which is the US. Over repeated encounters, the CS gains associative strength with the US, and it is the degree of this associative strength, that is, how well the predator associates the warning signal with the toxicity, that determines what the learning curve looks like. For aposematic prey, increasing the salience of their warning signal (e.g., by the signal becoming more conspicuous, distinctive, or novel) or the noxiousness of their toxin should lead to steeper curves and lower asymptotes, and increased survival of prey.

There are numerous studies that are entirely consistent with the idea that the shape of the learning curve is determined by the associative strength between the signal and the toxicity (Roper 1993; Roper and Marples 1997; Gamberale and Tullberg 1998; Hauglund et al. 2006; Skelhorn and Rowe 2006c; Halpin et al. 2008a; Aronsson and

Gamberale-Stille 2013). However, it is also becoming increasingly clear that we need to think more deeply about what cognitive processes underlie avoidance learning curves. First, there is accumulating evidence that the behavior of predators cannot simply be explained by the strength of the association between a toxin and a warning signal, particularly in the asymptotic phase of the learning curve where we now know that animals make informed decisions to include toxic prey in their diets (Barnett et al. 2007; Skelhorn and Rowe 2007b; Halpin et al. 2014). Second, there is an increasing appreciation that predators' decisions to attack defended prey could be influenced by the costs and benefits of acquiring information (Sherratt 2011; Sherratt et al. 2015). Thus, although there is no doubt that associative strength is important in affecting the shape of the overall curve, we think that it is also crucial to consider what other cognitive processes may influence predators' decisions to attack aposematic prey. We will consider how predators behave toward aposematic prey during the asymptotic phase in order to highlight that cognitive processes other than associative learning can influence predators' foraging decisions.

PREDATOR BEHAVIOR IN THE ASYMPTOTIC PHASE

If predator behavior was guided solely by the associative strength between the prey's signal and toxicity, we should not expect the asymptotic attack rate to change unless the signal or toxicity also changed. However, if predators make adaptive decisions based on acquired information, then asymptotic attack rates should be flexible and change in response to any factor that influences the cost or benefit of eating toxic prey. Our recent work using European starlings (*Sturnus vulgaris*) foraging on mealworms (*Tenebrio molitor*) provides strong support for the latter. We examine our own work in detail simply because these are the only experiments that have robustly measured predators' behavior in the asymptotic phase (e.g., Skelhorn and Rowe 2007b, 2009, 2010; Halpin et al. 2012). These experiments have shown that although predators may acquire information through learning about how much toxin different prey contain, their decisions to attack toxic prey at asymptote involve the adaptive use of that information.

Adaptive decision making

If we stop to think for a moment, evolutionary foraging theories predict that predators should strategically include toxic prey in their diets when it benefits them to do so (Stephens and Krebs 1986; Belovsky and Schmitz 1994; Kokko et al. 2003; Sherratt 2003; Oudman et al. 2014). It is sometimes easy to forget that the reason that aposematic prey have evolved defenses at all is because they contain nutrients that predators want. Therefore, continuing to include toxic prey in the diet makes sense if predators benefit from the ingestion of the nutrients prey contain compared with the cost of ingesting the associated toxins. These kinds of "nutrient–toxin trade-offs" have been widely documented in herbivores, which graze on multiple plant species that differ in their nutritional values and toxin contents, and adaptively fine-tune their foraging behavior in order to survive (e.g., Freeland and Janzen 1974; Provenza 1995; Duncan and Young 2002; Dearing et al. 2005; Marsh et al. 2005; Villalba and Provenza 2005). We have now conducted a series of experiments that demonstrate that avian predators also make nutrient–toxin trade-offs when foraging on aposematic prey.

Our experiments have explicitly explored birds' abilities to make adaptive foraging decisions on aposematic prey. In our laboratory experiments, starlings learn to use signals to reduce their intake of toxic prey until they reach a stable asymptotic attack rate on them. We then explore the cognitive processes that the birds are using to make their attack decisions on prey that they know contain toxins. Typically, we manipulate factors that we predict should affect the asymptotic attack rate if birds are making nutrient–toxin trade-offs based on what they have learned about the prey. Unsurprisingly perhaps, we find that the physiological state of the bird affects their decisions. Predators increase their attacks on prey they know to be toxic when they are in a poor energetic state and have low-fat reserves (Barnett et al. 2007, 2012); when ambient temperature is lower and their energetic requirements may be higher (Chatelain et al. 2013); and when the amount of nutrients available from alternative prey is smaller (Halpin et al. 2013). These decisions are not mistakes: Birds can know the toxin content of defended prey but still decide to increase their attack rates on them when in poor energetic state (Barnett et al. 2007). Also, as one would expect, they decrease their consumption of toxic prey when their toxin burdens (the amount of toxin in their bodies that they have yet to metabolize and/or excrete) are increased (Skelhorn and Rowe 2007b). In addition to predator state, intrinsic factors of the prey themselves are also important. Predators increase their consumption of toxic prey when they are nutritionally enriched (Halpin et al. 2014), demonstrating that they learn not only about the toxin content of prey but also about their nutritional value. Conversely, when the toxicity across individual prey is variable, making it potentially difficult to predict toxin intake from a visual signal, predators reduce their attack rates on them perhaps to reduce the risk of eating a highly defended prey (Barnett et al. 2014).

Taken together, these studies show that predators can learn about both the toxin content and nutritional value of aposematic prey and use this information to make adaptive state-dependent decisions about when to include toxic prey in their diets. This highlights the complexity of the cognitive processes underlying predatory decisions and also challenges the notion that we understand how and what predators learn in the acquisition phase; it seems that they learn about much more than just prey toxicity.

PREDATOR BEHAVIOR IN THE ACQUISITION PHASE

Predators are clearly able to learn about various features of aposematic prey. Consequently, we advocate the view that it is much more appropriate to view the acquisition phase as a phase when predators learn *about* defended prey rather than learning to *avoid* them (see also Speed 2000; Kokko et al. 2003; Sherratt 2003; Skelhorn and Rowe 2006a, 2007b; Sherratt 2011; Barnett et al. 2012; Halpin et al. 2014). When viewed in this way, it becomes immediately clear that we know shockingly little about the cognitive processes involved in the acquisition phase and specifically what predators learn, and how they learn, about defended prey.

What do predators learn about toxic prey?

It is well-established that predators can learn about the quantity of toxin that prey contain (e.g., Speed et al. 2000; Lindström et al. 2004; Ihalainen et al. 2007; Webb et al. 2008; Barnett et al. 2012). However, they may also learn more specific quantitative and qualitative information about how toxins vary intraspecifically and interspecifically (Brower et al. 1963; Brower and Calvert 1985; Cohen 1985; Marples et al. 1989; de Jong et al. 1991). For example, they could learn about the variability of a single toxin across individuals within a single population (Barnett et al. 2014; Sherratt et al. 2015)

or about different toxins that have different physiological effects on the predator (Turner and Speed 2001; Sherratt 2003; Skelhorn and Rowe 2005; Maan and Cummings 2012). The benefit of learning about the specific toxin content of a prey population is clear: It allows predators to manage their toxin intake better. This could be by reducing the risk of ingesting a prey where the toxin content is not precisely known (Skelhorn and Rowe 2005, 2007a, 2010) or knowing when it is worth sampling prey to either discriminate among them on the basis of their toxin content or attempt to remove toxins from them (Skelhorn and Rowe 2006b; Holen 2013). Knowing the toxicity or range of toxin contents associated with a particular warning signal could be advantageous for making informed decisions to attack and ingest aposematic prey and their mimics under a range of different situations.

These decisions are even better informed if predators can learn about the nutrient content of their prey, and there is now clear evidence that they do (Halpin et al. 2014). However, we know very little about what nutritional qualities predators learn about, for example, whether they learn about energy content or about specific nutrients (Raubenheimer et al. 2007). We also do not know if predators learn about different nutritional qualities of prey independently from their toxin content or whether they learn something more general about prey "profitability." If we want to understand how predators make decisions, we need to know what predators have actually learned.

How do predators learn about toxic prey?

There may seem like an easy answer to this question; predators simply learn to associate the nutrient and toxin content of the prey with a specific warning pattern, and the combined associative strength of each determines the likelihood of attack. But how do predators learn about nutrients and toxins at the same time? To know exactly how they do this, we cannot simply turn to the experimental psychology literature for possible answers. To our knowledge, there have been no experiments investigating how animals integrate negative and positive reinforcements from the same action, as would be the case when ingesting nutrients and toxins from the same prey. This appears to be pretty unique to the kinds of questions we study; predators receive nutrients and toxins together following an attack, while experimental psychologists would deliver only one of these at a time. Therefore, we do not know whether information about the toxin and nutrient content of prey is encoded independently, and if it is not, whether predators learn about toxicity and nutritional value at similar rates, whether more salient warning signals also enhance the speed at which predators learn about prey nutrient content, and whether some combinations of prey toxicity and nutritional value are easier to learn than others (e.g., low levels of nutrients with high levels of toxicity). We also do not know whether predators learn absolute values of nutrients and toxins or whether they make relative evaluations. Mechanistic studies of predator learning are crucial for our understanding of what predators learn about prey and how they exert selection on prey defenses. But perhaps more importantly, such studies may also provide exciting opportunities to better understand learning and information use per se.

Although it may be perhaps tempting to reinterpret the curves in the acquisition phase as simply the result of how predators learn to associate a color signal with both a food reward and a toxin punishment, and the associative strength that each reinforcer has with the color signal determines the shape of the curves, we would urge against this. Because we know that predators use acquired information to make adaptive foraging decisions, we think it unlikely that changes in foraging behavior in the acquisition phase are simply changes in the strength of CS–US associations. It is likely that, as in the asymptotic phase, predators are making important decisions, but these are based not only on what they already know but also on their motivation to acquire information about the prey (Kokko et al. 2003; Sherratt 2011; Sherratt et al. 2015). Gathering information can be costly in terms of the time and energy invested in sampling prey, and the risk associated with consuming prey with potentially unknown quantities of unidentified toxins. It has been suggested that these decisions are adaptive and that predators make decisions to attack prey based on both their perception that the prey may be toxic and their perceived probability of encountering the prey type in future (Sherratt 2011; Kikuchi and Sherratt 2015; Sherratt et al. 2015). We would go further than this and propose that a wide range of factors will influence predators' decisions to gather information about prey. Such decisions will be influenced not only by informational variables that influence the perception of the potential cost and benefits of sampling prey (novelty, toxicity, abundance, nutritional value, etc.) but also by factors intrinsic to the predator such as its current energetic state, toxic burden, and ability to metabolize toxins. Furthermore, attempts to model the optimal information gathering strategies of predators often assume that prey are either beneficial to the predator (i.e., undefended) or costly (i.e., toxic). However, in natural systems, it is unlikely that there is such a clear dichotomy. There is likely to be a whole spectrum of different prey profitabilities, and even toxic prey may contain important micronutrients that cannot be found in undefended prey (e.g., Brower et al. 1963, 1968; Sargent 1995; Speed 1999; Tullberg et al. 2000). So, although these models may need some refinement, they provide an excellent starting point for understanding how predators make decisions about when to be neophobic if encountering novel prey, or more cautious when sampling variably toxic prey. Importantly, these models not only suggest that shallow learning curves may not represent slow color-toxin associative learning but also reflect that predators are investing more in information gathering during the acquisition phase.

This is an important point, and one we need to be sure that we are clear about. We are not suggesting that associative learning does not affect the shape of the acquisition curve, but rather that it is just one of multiple cognitive processes involved. Associative learning rules may affect the speed of data acquisition. For example, in line with previous studies, we would still expect predators to learn about the toxicity (and maybe even the nutrient content) of prey faster when the signal is conspicuous compared with being cryptic (Gittleman and Harvey 1980; Ham et al. 2006). However, as the predators acquire information about the nutrient and toxin content of prey, they are also deciding how to continue sampling the prey population. These decisions can be considered akin to those used when exploiting versus exploring discreet food patches (e.g., Krebs et al. 1978; Kramer and Weary 1991; Dumont 1996; Provenza et al. 2003; Searle et al. 2005; Sherratt 2011; Kikuchi and Sherratt 2015; Sherratt et al. 2015), with defended toxic prey viewed as suboptimal patches. Viewed in this way, any factor that causes information about toxic prey to be more highly valued will decrease the slope of the curve in the acquisition phase and cause defended prey to be more heavily predated. These predictions need to be tested empirically, but we believe that studying how predators make decisions about how and when to learn will allow us to identify previously unconsidered factors that have a major impact on the evolution of prey defenses.

A BROADER VIEW OF PREDATOR COGNITION

It is clear that in order to understand predators' behavior when faced with aposematic prey we need to take a more holistic view of predator cognition. Obviously, it is important to understand how predators learn to associate the visual appearance of prey with the noxious effects of the toxins they possess, but it is important to realize that learning is not just about predators developing an aversion or a "dislike" for toxic prey types. Predators acquire information about the nutrient and the toxin content of prey, both of which can vary in their degree of predictability across individuals carrying the same warning signal (Pasteels et al. 1983; Brower and Calvert 1985; Cohen 1985; Holloway et al. 1991). Predators also have to trade-off the costs and benefits of acquiring more information about a particular prey type, which will depend on intrinsic (e.g., current physiological state) and extrinsic (e.g., prey frequency) factors. They also have to decide how to use the information they receive to update their knowledge (e.g., Trimmer et al. 2011), for example, should encountering a single highly defended individual in an otherwise moderately defended population significantly affect the evaluation of that prey type? Again, this is likely to be affected by other factors, including how costly ingesting a high dose of toxin is to the predator, and how many moderately defended individuals have been previously encountered. What we want to highlight is that it is crucial to remember that learning is just one of the cognitive processes that are likely to influence predators' foraging decisions. We need to know how predators make adaptive decisions about when to gather information about aposematic prey: from their initial decisions to sample novel prey (with neophobia as the most extreme response), through decisions to "update" their existing knowledge of a particular prey type, to their decisions about cautiously/extensively to handle prey items in order to obtain more information about them. Furthermore, we need to understand how predators use the information they have in order to make adaptive decisions about what prey to include in their diet, and how they trade-off the relative cost and benefits of gathering and using information.

To clearly state the approach, we advocate rather than thinking about predators learning to *avoid* aposematic prey, we should instead think about predators learning *about* them in order for them to make adaptive foraging decisions (e.g., Speed 1993; Skelhorn and Rowe 2007b; Halpin et al. 2014). We acknowledge that adopting this approach will be challenging and will even require us to re-evaluate the way we design and interpret experiments. However, it is important that we develop our understanding of all aspects of predator cognition if we want to understand the evolution of aposematism.

WHY IS ANY OF THIS IMPORTANT?

Incorporating the idea that predators do not simply learn to avoid toxic prey into the study of prey defenses has been a painfully slow process. It has only recently been demonstrated that predators make adaptive decisions about when to eat defended prey (Skelhorn and Rowe 2007b; Barnett et al. 2012), and the cognitive processes underlying these decisions are poorly understood. Our aim has been to highlight that we know a great deal less about predator cognition than is commonly assumed. We have argued that in order to understand the evolution of antipredator defenses it is crucial that we take a more holistic view of predator cognition. We need to understand how predators make strategic decisions about both when to invest in information gathering (and consequently the information they have available to them) and when to eat prey they know to be toxic. Here, we consider some of the reasons why it is important to take this approach.

It allows us to unify the study of different aspects of predator cognition

Consideration of both the costs and the benefits of gathering and using information in predator learning and decision making allows us to study a range of predatory behaviors toward aposematic prey using a more unified approach. Neophobia, foraging biases, taste-rejection behavior, learning, and decision making are often viewed as separate processes, yet they can all be viewed under the umbrella of predators making adaptive decisions to gather more information about a prey type or making use of the current information that they have.

Each foraging decision that a predator makes needs to be seen in a broader context: for example, any wariness toward novel prey that predators express (i.e., neophobia or foraging biases) will be in the context of what they already know about the prey in their environment. It also means that it is possible to evaluate how cognitive processes and foraging decisions at different stages of a predatory encounter are related to each other. For example, the degree to which predators invest in gathering information about the variability of defended prey could influence both predators' decisions about when to include these prey in their diet, and the degree to which they sample prey and try to discriminate between differentially defended individuals postattack (Sherratt et al. 2015). It is therefore vital that we have a single theoretical framework that we can use to study all aspects of cognition and how these interact across the predation process, because the expression of one behavior depends on that of another, and the interaction between cognitive processes can significantly impact on how predators exert selection on prey defenses (Sherratt et al. 2015).

It allows multiple defense strategies to be studied simultaneously

One of the major advantages of the approach we advocate is that it allows research bridges to be made between studies of different types of defensive coloration. Decisions to attack and eat aposematically signaling prey no longer have to be seen as different from those on undefended cryptic prey, where similarly, predators need to evaluate the costs and benefits to attacking palatable prey that vary in their nutritional value (Simpson et al. 2004; Raubenheimer et al. 2007). This means that predators' decisions to attack aposematic prey are made in a sea of many others toward prey with different types of defensive coloration, which are likely to be interdependent. Decisions to attack one particular prey type are determined not only by the costs and benefits of attacking that prey type but also by the costs and benefits of attacking other prey types available (Merilaita and Kaitala 2002; Lindström et al. 2004; Skelhorn and Rowe 2005, 2006a; Halpin et al. 2013).

Taking this step allows us to study the evolution of prey defenses as part of an ecological community, rather than each in isolation. For example, increasing camouflage in a palatable prey type could lead to an increase in predatory attacks on aposematic prey because the costs of investing time in searching for cryptic palatable prey increase the relative benefits to attacking a highly visible aposematic prey (Carle and Rowe 2014). This means that we can determine how the structure of prey communities is likely to influence predators' decisions and consequently the selection pressures acting on prey with different defensive strategies. It will also allow us to

compare the efficacy of different defensive strategies, which may ultimately help us to understand why prey species evolve the defensive strategies they do.

It highlights new factors that affect predation

Accepting that predators do not necessarily avoid toxic prey, but instead make adaptive decisions about when to gather information about them and when to consume them has already allowed us to identify a number of novel factors that influence the selection pressures acting on defended prey. These include qualities of the aposematic prey (e.g., their nutritional value: Halpin et al. 2014; Smith et al. 2014), qualities of alternative prey (their size and how easy it is to find them: Halpin et al. 2013; Carle and Rowe 2014; Smith et al. 2014), the condition of predators (e.g., energetic state and toxin burden: Barnett et al. 2007; Skelhorn and Rowe 2007b; Barnett et al. 2012), and aspects of the external environment (e.g., ambient temperature: Chatelain et al. 2013).

There is also good reason to believe that this is just the tip of the iceberg and that there are many other, as yet unidentified, factors influencing the evolution of antipredator defenses. For example, if predators find themselves more at risk from predatory attack (perhaps due to their physiological condition and/or social dominance [Hegner 1985; Vytenis and Godin 1991; Macleod et al. 2005], increasing predator abundance [Brown 1999], or changes in the physical structure of the habitat [Whittingham and Evans 2004]), they may be time limited or more fearful and more pessimistic when encountering prey that are aposematic. In addition, young predators and those predators hunting in changing environments may have more to gain from investing in information gathering than older predators or those in more predictable environments. In fact, many of the factors identified as important in influencing the relative benefits of exploration and exploitation of palatable prey in an optimal foraging context are also likely to influence predators' decisions about how much information to gather about toxic prey, including current diet quality, the difference in quality between undefended and aposematic prey, the amount of foraging time available, and the age/life expectancy of the predator (Krebs et al. 1978; Kramer and Weary 1991; Eliassen et al. 2007; Mata et al. 2013). Furthermore, determining how these (and other) factors influence predators' decisions to attack toxic prey is crucial because it will help us to understand how and why selection for aposematism and mimicry vary both among and within species.

It will enable theoretical models to generate more accurate predictions

The ability of mathematical models to make valuable theoretical advances in the study of aposematism and mimicry depends on how well they capture the cognitive processes of predators. Indeed, a number of mathematical models appear to suggest that the way in which predators make decisions when faced with toxic prey has an important effect on the evolutionary dynamics of aposematism and mimicry. Indeed, small changes to the algorithms used in associative learning-based models can lead to major changes to the evolutionary predictions they make (Speed 2001; Turner and Speed 2001; Speed and Ruxton 2005). Moreover, theoretical models have taken a number of different approaches to capturing the essence of predator behavior: considering how predators learn associations (Huheey 1988; Speed 1993, 2001; Speed and Ruxton 2007), how state influences their foraging decisions (Kokko et al. 2003; Sherratt 2003; Sherratt et al. 2004; Speed and Ruxton 2014; Sherratt et al. 2015), or how they should optimally invest in information gathering (Sherratt 2011; Kikuchi and Sherratt 2015; Sherratt et al. 2015). Intriguingly, these different approaches often lead to very different (and in some cases conflicting) predictions about the evolution of prey defenses. To give just one example, models that use approaches that fail to consider the cost of learning often predict the evolution of Batesian mimicry when predators are faced with one defended and one undefended species (see Mappes and Alatalo 1997a and refs. therein). On the other hand, models that consider how predators' should optimize their investment in information gathering predict that when the cost of sampling is high, predators should not invest in discriminating between toxic models and undefended mimics, and consequently selection for Batesian mimicry is weak—indeed, selection may even favor the evolution of polymorphisms to further decrease the benefit of learning (predators then need to learn about more prey types) (Sherratt et al. 2015).

Clearly, we need to understand the cognitive processes of predators in order to 1) evaluate which, if any, mathematical models accurately reflect the evolution of prey defenses in real-world situations and 2) develop new models that give novel insights into the evolution of prey defenses. However, we currently do not have enough empirical data to determine whether the decision-making rules generated by, or programed into, these models accurately reflect those used by real-world predators.

It allows us to better design and interpret our experiments

Taking our approach means that we need to be more thoughtful when it comes to designing our experiments and interpreting our data. In particular, we may need to be more careful in how we design our experiments in order that we consider factors that we have not previously considered. For example, if predator state is important in determining how predators learn and make decisions, we will need to ensure not only that predators start learning sessions in a similar state (e.g., that they experience the same food deprivation period or toxin burden), but that we consider changes in state as our sessions progress, particularly where the amount of nutrients and toxins are likely to differ across different experimental groups (Rowland et al. 2007; Halpin et al. 2013).

When it comes to interpreting graphs of avoidance learning, which are commonplace in the literature (e.g., Roper and Wistow 1986; Lindström, Alatalo, Mappes, et al. 2001; Skelhorn and Rowe 2006c, 2006b; Halpin et al. 2008b; Rowland, Hoogesteger, et al. 2010; Skelhorn and Rowe 2010), we also need to be more reflective of what they may represent. If the learning curves can be affected by the nutrient content of the prey, the physiological state of the predator, and how much predators invest in information gathering, we need to be careful that the interpretations of our data are accurate. We can no longer be sure that a reduction in attacks on aposematic prey either within or across learning trials is solely a reflection of how quickly predators learn the association between a prey's signal and its toxicity.

Some experiments do not attempt to determine the cognitive processes underlying predation (i.e., how the predators are learning and making decisions), but instead measure the effect of defenses on prey mortality (Mappes and Alatalo 1997b; Speed et al. 2000; Lindström et al. 2001a; Ihalainen et al. 2008; Rowland, Hoogesteger, et al. 2010). Given our argument, this may seem advantageous because they do not ascribe increased avoidance of aposematic prey to an associative learning process. However, we would urge that the results of such experiments should be treated

with caution. This is because without knowing the cognitive processes underlying the avoidance of aposematic prey, it is difficult to predict the generality of such findings. For example, small changes in environmental conditions have the potential to have quite significant effects on the decisions that predators make, and impact on prey mortality. Only by knowing how the cognitive and physiological processes of predators determine foraging patterns, can we understand the evolution of prey defenses across different situations and potentially predict effects of environmental change.

It allows the cognitive abilities of predators to be compared with other types of forager that encounter toxic foods

Many species, both plants and animals, defend themselves with harmful toxins in order to reduce the chances that they are eaten (Adler 2000; Foley and Moore 2005). This means that many different types of forager also need to make foraging decisions based on the nutrient and toxin content of their foods, including nectivores (e.g., Lerch-Henning and Nicolson 2013; Nicolson et al. 2014; Lerch-Henning and Nicolson 2015), herbivores (e.g., Freeland and Janzen 1974; Dearing et al. 2005; Foley and Moore 2005; Ginane et al. 2005), and frugivores (e.g., Sorensen 1983; Cipollini and Levey 1997; Levey and Rio 2001). However, predators' decisions on aposematic prey are often treated independently from these literatures, and often research on each of these systems tends to be nonoverlapping. We think that there are real benefits from integrating ideas, techniques, and knowledge across different foraging systems in order to develop a more coherent framework for understanding how animals make decisions to eat toxic foods.

This is evident in the success of applying ideas from the herbivory literature to predator–prey interactions and the evolution of prey defenses. Compared with insect prey, the nutritional and toxin content of many forage plants are well known, which have allowed the principles of nutrient–toxin trade-offs to be established and detoxification pathways to be well understood (e.g., Marsh et al. 2005, 2007; Nersesian et al. 2012). As we have already discussed, this has helped to develop the idea that predators regulate their intake of toxic prey according to their physiological state, and identify novel cognitive and evolutionary processes (Kokko et al. 2003; Sherratt 2003; Sherratt et al. 2004; Barnett et al. 2007; Skelhorn and Rowe 2007b; Speed and Ruxton 2014).

However, studies of predators foraging on toxic prey can also help to inform research on other foraging systems, particularly where the mechanisms of learning and decision-making processes are less well studied or assumed to be less important (e.g., Villalba and Provenza 2009). For example, research on the feeding choices of ruminants and other grazing mammals shows that, like predators, they prefer to select foods rich in nutrients and low in toxic plant secondary metabolites (PSMs) (Singer et al. 2002; Baraza et al. 2005; Dearing et al. 2005; Marsh et al. 2007). However, in comparison with what we know about predator cognition, the cognitive processes of mammalian herbivores are even less well understood. Although herbivores can learn cues to help them select plants to help regulate their ingestion of PSMs and balance their nutrient intake (Freeland and Janzen 1974; Singer et al. 2002; Dearing et al. 2005; Ginane et al. 2005; Marsh et al. 2006), how they decide which cues to use and what factors affect how, when, and what they learn are largely unknown.

Comparisons can also be made within a taxon containing different types of forager, for example, avian nectivores and predators. Recent studies on nectivorous birds are starting to uncover how birds physiologically cope with toxins in nectar and how they could learn cues to identify floral rewards containing toxins (Lerch-Henning and Nicolson 2013; Nicolson et al. 2014; Lerch-Henning and Nicolson 2015). One intriguing possibility is that species foraging on different types of toxic foods, such as flowers and insects, may have evolved different cognitive strategies for identifying and regulating their intake of nutrients and toxins. This could occur perhaps because different floral cues are more informative about nutrient content whereas warning signals are more informative about toxin content, or because ingesting toxins from prey are more damaging to predators because they have specifically evolved as a deterrent whereas toxins in nectar may be a by-product of defenses against herbivory (Rhoades and Bergdahl 1981; Adler 2000). These kinds of questions can only be answered if we consider predatory decision making on toxic prey in this broader foraging context.

It allows us to ask more interesting questions about predator cognition

Studies of predator cognition in the context of aposematism and mimicry tend to revolve around understanding how the cognitive processes of predators drive the evolution of these prey defense strategies. However, considering when predators should invest in information gathering or when they should use the information they have, and how they integrate physiological processes into their decision making, allows us to shift focus more toward the evolution of cognitive abilities and behavior of the predators themselves.

One interesting question for future research is to ask if predators are indeed making decisions about when to learn about different prey types, and what information predators use when deciding how much to invest in learning about aposematic prey and their mimics? As we have already discussed, these decisions could be driven by the relative costs (e.g., eating toxins) and benefits (e.g., finding undefended mimics in the population) of learning about aposematic prey, but we currently can only guess at what factors are important; none have been empirically tested. This could provide important insights into why sampling and learning strategies may vary across individuals and species (e.g., Exnerová et al. 2003, 2010).

However, variation in predatory decision making is also likely to be influenced by a predator's physiological state. For example, discriminating among prey that vary in their toxicity may only be beneficial if there is no threat of starvation from missing out on valuable nutrients when toxic prey are rejected (e.g., Halpin et al. 2012), while sampling novel aposematic prey may be more costly to small-bodied predators where the ingestion of a dose of toxin may be have more significant impact compared with a larger-bodied predator (Exnerová et al. 2003). The cognitive strategies of predators foraging on aposematic prey should therefore reflect the costs and benefits of exploiting or exploring toxic (or potentially toxic) prey given their own current knowledge and their physical condition. Understanding how predators integrate physiological information into their decision making is a growing area of research (e.g., Skelhorn and Rowe 2010; Barnett et al. 2014; Speed and Ruxton 2014), which should lead to a better understanding of how predators could be trading off investment in both physiological and cognitive strategies in their attempts to exploit aposematic prey and their mimics as a food source.

CONCLUSION

It has long been recognized that predators do not simply learn to avoid aposematic prey, but instead include these prey in their diet when it

is beneficial to do so: both the nutritional value of toxic prey (Speed 1999; Speed and Ruxton 2014) and the energetic state of predators (Sherratt 2003; Sherratt et al. 2004) may influence predators' decisions to attack defended prey and, consequently, the selection pressures acting on them. However, despite this, the cognitive processes underlying predators' decisions are still poorly understood. We have argued that in order to understand the evolution of prey defenses we need to understand the mechanisms by which predators select prey. In order to do this we need to stop thinking about how predators learn to avoid prey and start concentrating on how they learn about prey. At present, we know very little about how associations between prey coloration and both their toxin and nutrient contents are formed. We also need to understand how predators make adaptive decisions about when to gather and when to use information about defended prey (Sherratt 2011; Sherratt et al. 2015), and to do this, we need to identify the costs and benefits of gathering and using information.

In Box 1, we consider some of the key questions that need to be answered before we can fully understand how predators' cognitive processes influence the evolution of prey defenses. We hope that we have highlighted what little in fact we know about the cognitive processes of predators when faced with defended prey and that it is vital we understand the mechanisms underlying prey selection if we are to understand the evolution of prey defenses. Taking the approach we advocate to study predators' foraging decisions will allow us to understand the selection pressures acting on prey across a range of different environmental conditions, which we argue will allow better extrapolation of laboratory findings to predator–prey systems in the wild.

BOX 1: FUTURE AREAS OF RESEARCH

These questions address what we see as key unknowns about the cognitive processes of predators. Answering them will have important implications for what we understand about the selection pressures acting on aposematic prey and their mimics.

- How do predators associate warning signals with both a reward (the nutrient content) and a punishment (the toxin content)?
- Do warning signals enhance the speed at which predators learn about the nutrient content of toxic prey?
- Is the slope of the curve in the acquisition phase always related to the asymptotic attack rate, that is, can predators learn about defended prey quickly but maintain a high attack rate?
- Do predators learn about the variability in the toxin and nutrient content of individuals within a population?
- Can predators use taste cues to gather information about the nutrient content as well as the toxin content of aposematic prey?
- What factors influence the costs and benefits of gathering information about toxic prey?
- Do predators make absolute or relative evaluations of aposematic prey?
- Are there interspecific differences in the cognitive processes underlying predators' decisions to attack aposematic prey?
- Does investment in postingestive processes to deal with eating toxins affect the cognitive strategies of predators?
- How do predators learn and make decisions about toxic prey in natural environments?

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