Animal Behaviour 98 (2014) 53–67

Contents lists available at ScienceDirect

Animal Behaviour

journal homepage: www.elsevier.com/locate/anbehav

Ecological pharmacodynamics: prey toxin evolution depends on the physiological characteristics of predators

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ARTICLE INFO

Article history:
Received 24 January 2014
Initial acceptance 25 February 2014
Final acceptance 22 July 2014
Available online 23 October 2014
MS. number: 14-00066R

Keywords:
chemical defence
decision making
diet breadth
predator–prey
taste rejection

The use of toxic chemical defences to repel and deter predators is widespread across living organisms, yet there are surprisingly few formal models of toxin evolution. Published models tend to focus on a trade-off between individual benefits and costs of toxicity, and treat predators as simple agents of selection, reducing future attacks when they encounter toxic prey. In this paper we argue, however, that the physiological characteristics of predators may be crucial in determining the nature and outcomes of toxin evolution. To examine this idea we devised and explored a model in which prey defence evolves in the context of predator physiology. We represented this as dose–effect relationships in predators for nutrition and toxins along with variable rates of predator metabolism. Incorporating variables of predator physiology can change views of toxin evolution. A key point is that inclusion of predator physiological variables requires that the nutritional value of prey is explicitly represented in the model, and this directly affects predictions for toxin evolution. In our model costly toxins generally evolve to the point that they are 'minimally unprofitable': just toxic enough to make prey typically unprofitable given their nutritional value to predators. As the nutritional value of prey increases, so the minimally unprofitable toxin level of prey tends to increase in step; hence another general prediction from this model is that toxin levels within prey should often correlate with the nutritional value of the prey. Predator physiology and cognition also contribute to variation in the social nature of defence. We argue that incorporating representations of predator physiology is important in the comprehension of toxin evolution and make suggestions for directions of future work.

Predation is a major determinant of fitness for prey animals through either direct costs resulting from attack or indirect costs incurred to avoid attacks. There are many ways for prey to avoid attacks by predators, including changes in appearance and behaviour (e.g. camouflage, mimicry; Ruxton, Sherratt, & Speed, 2004) and changes in life history patterns (e.g. body size; Higginson & Ruxton, 2010). The use of toxic chemical defences to repel and deter predators is also widespread, found across all animal taxa, yet there are surprisingly few formal models of toxin evolution in animals.

In contrast, understanding the ecology of defensive toxicity via secondary metabolism in plants has a long-established and increasingly tested theoretical basis. For example 'optimal defence theory' (McCall & Fordyce, 2010; McKey, 1974; Stamp, 2003) relates variation in defence investment across plant tissues to the value of each tissue to the plant, and to the intrinsic vulnerability of each tissue to herbivore damage. From the herbivores' perspective there is a growing body of theory too. The 'detoxification limitation hypothesis' (Freeland, 1991; Freeland & Janzen, 1974; Marsh, Wallis, Andrew, & Foley, 2006) assumes, for example, that herbivores can show sufficient nutritional wisdom to regulate their intake of toxins and balance dietary needs (see discussion and developments in Feng, Liu, & DeAngelis, 2008; Forbey et al., 2013; Swihart, DeAngelis, Feng, & Bryant, 2009).

There is, however, in our view a lack of an equivalent, systematic application of theory to the examination of animal chemical defence; this leaves important questions unanswered, such as the following. What determines how much an animal invests in chemical defence? Why are defensive toxins so often variable (Speed, Ruxton, Mappes, & Sherratt, 2012)? What is the role of individual or kin selection in the initial evolution and maintenance of toxicity? What are the ecological consequences of toxin evolution for prey species?

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http://dx.doi.org/10.1016/j.anbehav.2014.09.011
0003-3472/

Formal, published, models of animal toxin evolution focus on the role of costs in determining optimal or evolved toxin levels. Such models tend to treat toxicity as a simple trade-off between the benefits of defence and the fecundity or other costs of toxin investment (Broom, Ruxton, & Speed, 2008; Broom, Speed, & Ruxton, 2005; Broom, Speed, & Ruxton, 2006; Longson & Joss, 2006; Skelhorn & Ruxton, 2008; Speed & Ruxton, 2007; Speed, Ruxton, & Broom, 2006; Svennungsen & Holen, 2007; Svennungsen, Holen, & Leimar, 2011). Some more elaborate models incorporate toxin investment into life history decisions (Higginson & Ruxton, 2010; Higginson, Ruxton, & Skelhorn, 2010); but all of these models have in common a 'black box' approach to predator physiology, making no explicit assumptions about what can be called the 'pharmacodynamics of prey toxicity' (i.e. the relation between dose and effect, rates of detoxification, see Rosenbaum, 2011). Where predator physiology has been explicitly considered in predator–prey interactions, the aim of relevant models is to optimize predator decision making (as with dynamic programming approaches such as in Sherratt, Speed, & Ruxton, 2004) rather than to allow prey to evolve in relation to variable predator physiologies.

Recent empirical work, however, shows that predator assessments of nutrition and toxicity are key determinants of attack decisions: predators appear aware of their physiological toxin levels from previously ingested toxic prey (Barnett, Skelhorn, Bateson, & Rowe, 2012; Skelhorn & Rowe, 2007), rates of metabolic energy use in thermoregulation (Chatelain, Halpin, & Rowe, 2013) and the nutritional value of alternative prey in their environments (Halpin, Skelhorn, & Rowe, 2013). Detoxification rates for prey toxins are becoming increasingly known (Williams, Hanifin, Brodie Jr, & Brodie III, 2012). In the herbivore literature Marsh, Wallis, and Foley (2005) demonstrated that rates of detoxification determine feeding rate in possums, *Trichosurus vulpecula*, and Bergvall et al. demonstrated the sensitivity of foraging deer, *Dama dama*, to tannin levels in food patches (Alm, Birgersson, & Leimar, 2002; Bergvall, Rautio, Kesti, Tuomi, & Leimar, 2006).

Given the importance of predator physiology in determining attack decisions and the conspicuous lack of relevant theory, in this paper we attempt to generate an 'ecological pharmacodynamic' description of predator physiology and apply it to the evolution of prey toxicity. We apply the key idea from the defence limitation hypothesis that 'The rate at which a particular plant metabolite is detoxified is of obvious importance in determining how much a mammal eats per unit time' (Freeland, 1991, p. 63); hence as a first step we introduce a simple theoretical model which represents dose–effect relationships, metabolic rate and detoxification in a predator that causes evolution in a focal prey population.

METHODS

We assume that predators show behavioural flexibility in response to both their environment (i.e. the local prey community structure) and aspects of their own wellbeing (i.e. their state of hunger and levels of previously ingested toxins). The model simulates state-dependent foraging decisions by a predator that attacks and ingests prey if it estimates that by so doing it will make a short-term net gain, given the values of parameters describing the nature of its foraging environment and its physiological and cognitive constraints. In the forager's environment there is (1) a focal species that can evolve its level of toxicity across generations and (2) a set of alternative prey that are always edible. Prey have values for their toxicity level (T, total mass of toxin) and their nutrient level (N, total mass of nutrition). For focal prey that have some toxicity level $T_F > 0, N_F > 0$; for edible, alternative prey, $T_A = 0$, $N_A > 0$.

Predators are characterized by physiological variables measuring the absolute nutrient level (H) and absolute toxin level (D) in their bodies, but these absolute levels are transformed into biologically effective levels (E_H, E_D, respectively) using power functions, so that

$$E_H = (\omega_H H)^{ß_1} \quad (1)$$

and

$$E_D = (\omega_D D)^{ß_2}, \quad (2)$$

where ω_H and ω_D are linear scaling coefficients for nutrition and defence, respectively (set to 1 or 1.5) and $ß_1$, $ß_2$ determine the general shape of the function (accelerated $ß > 1$; decelerated and saturating $ß < 1$; linear $ß = 1$). We examined the effects of variation in predator physiology for a subset of functions using equations 1 and 2. For toxins we have dose–response functions defined by one of two gradient values $\omega_D = 1, 1.5$, which are used with either linear ($ß_1 = 1$) or accelerating ($ß_2 = 1.5$) functions (Fig. 1, red lines show linear functions for two gradients; blue lines show accelerating curves). For nutritional dose–effect curves we use a linear function ($\omega_H = 1$, $ß_1 = 1$, lower red line, Fig. 1) or a decelerated function in which predators become increasingly satiated as they take in more nutrition ($\omega_H = 1$, $ß_1 = 0.5$, black line, Fig. 1). Over a time interval (t to $t + 1$), metabolism reduces the values of H by M_H (and thus reduces E_H) and D by M_D (and E_D), respectively. Hence $H_{t+1} = H_t - M_H$ and $D_{t+1} = D_t - M_D$. Biologically, this represents the energetic costs of routine metabolism and the inherent physiological ability to clear or neutralize circulating previously ingested toxins. We assume for simplicity that the predator can assess its state variables with accuracy, so that it knows its own E_H and E_D levels without error. Predators have a tolerance threshold for both nutrition and toxins (E_{Hmax} and E_{Dmax}, respectively, both set to a value of 2) beyond which they would experience toxicosis. If effective maximum values were exceeded for either or both of these thresholds, the predator would cease to feed until metabolism degrades effective levels to $E_H < E_{Hmax}$ and/or $E_D < E_{Dmax}$. The first of these represents physical satiety (constraints on prey capture driven by the capacity of the digestive system); the second represents the deleterious effects of high levels of circulating toxins on physiological functioning (see Marsh, et al., 2006).

To simulate predator–prey events we assume that each individual prey i has a fixed probability r_i of coming within the attack radius of a predator in a given time unit. How many prey come within this radius per unit time is determined stochastically and increases with the abundance of the focal prey (n_F) and the abundance of alternative prey (n_A). Hence there may be more than one prey present per unit time within this attack radius around the predator; we assume that the predator does not have time to identify and choose between them and hence selects a prey to examine randomly from the possible individuals. A prey chosen in this manner is defined as 'encountered' by the predator. Given that a prey is encountered, the predator must decide whether to attack, and then whether to ingest or release the attacked prey. In general the predator will attack if it anticipates that the effective increase in its nutritional stores exceeds the effective increase in its toxicity levels (i.e. $E_H > E_D$), otherwise it will not attack. In this way the predator will attack if it expects to make a profit. The predator will also not attack if in so doing the estimated effect of consumption on its physiology takes either its effective toxin level or its nutrient store above its maximum tolerance levels, E_{Dmax} or E_{Hmax}.

When an attack does take place, the predator can taste the prey to evaluate the nutrient and toxin qualities of the specific individual