Low Nutritive Quality as Defense Against Herbivores

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Contrary to a widespread assumption in the literature on plant–herbivore interactions, individual plants do not necessarily benefit by possessing traits which lower herbivore fitness. In particular, genes conferring lowered nutritive quality could even increase herbivore damage under certain circumstances. Three special sets of conditions are outlined in which low nutritive quality would lower herbivore-induced damage to an individual plant. These sets are far from exhaustive. It is concluded that the adaptiveness of lowered nutritive quality in herbivore defense is widely possible but in no case demonstrated.

Discussions of plant–herbivore interactions often assume that plant traits are favored by natural selection to the extent that they lower herbivore fitness (e.g. Rhoades, 1979; Feeny, 1976; Rhoades & Cates, 1976). Thus, Feeny (1975, p. 4) claims that:

> ... plants ... may use for protection substances of relatively subtle effect; these may not even be immediately toxic to an invading insect individual but nevertheless can reduce its fitness. This serves the function both of minimizing a population buildup by an attacker and of selecting against those invaders which attempt to colonize the plant. Thus a plant chemical which does not prove immediately toxic ... may nevertheless represent a significant defense against insects.

Feeny suggests that poor nutritional quality of foliage (resulting from low nutrient concentrations or from presence of compounds such as tannins which lower nutrient digestibility) is a widespread anti-herbivore adaptation in forest trees. Clearly, such a plant trait will tend to decrease the fitness of insect herbivores by lowering growth rate—and thus survivorship—of larvae or by lowering adult reproductive output. However, adaptiveness of the trait does not follow automatically from lowered herbivore fitness, at least not when selection is supposed to be effective primarily at and below the level of the individual. Some plant characteristics which lower herbivore fitness could have the effect of lowering fitness of the individual plant as well. Consider the example of low nutritive quality of forest tree foliage, and assume that larvae remain upon the same tree until they reach a certain size, whereupon they pupate, emerge as adults and fly off. A larva on an

247

"adapted" plant may consume many more leaves than would be the case if nutritive quality were high. If the interests of the plant are best served by losing as few leaves as possible and if nutrition-rich leaves are not much different in cost to the plant, then one might expect the immediate selection to be towards foliage of *high* nutritive quality.

There are, however, circumstances where poor nutritive quality of foliage could evolve as an adaptation to insect herbivory. Three possible pathways are presented below. Each requires restrictive assumptions concerning properties of the herbivore and, as will be seen, cannot cover all herbivore–plant interactions.

(1) If herbivores are able to detect differences in the nutritive quality of individual plants, and if they preferentially feed upon more nutritious host individuals, then low nutritive quality of leaves will be advantageous. Selectivity may be exercised by the stage which produces the feeding damage, provided that the feeding stage has the necessary mobility and sensory capacity. Even in the case of leaf-feeding larvae lacking these requirements, the argument will work if females selectively oviposit on plants of higher nutritive quality. Ovipositing female Lepidoptera often appear choosy about the individual plants and the parts of them where they place eggs but beyond attributes confirming host species little seems known of what they are able to be choosy about (M. C. Singer, pers. comm.). On current information this first model seems most likely to be valid for vertebrate herbivores.

(2) If successive herbivore generations tend to feed upon the same host individual then low nutritive quality will act to prevent future buildup of herbivore numbers, thus increasing the plant's fitness. To the extent that neighboring plants are relatives, the requirement for low between-generation mobility in the herbivore is eased. Insects with wingless females and very limited larval dispersal, such as coccids, may be sufficiently sedentary for this model to apply even where neighboring plants are unrelated. It may apply more generally to plants which clone extensively: even with some movement between plants, successive herbivore generations are likely to feed upon the same genetic individual.

This explanation, assuming very non-mobile herbivores, is at an opposite extreme from the first. However, for many important herbivores both models seem too extreme. Many insects both fly freely as adults and do not apparently sample food quality on behalf of their prospective offspring. Even for the relatively immobile coccids, winter moths, psychids and others—albeit these are often important antagonists of plants—it is far from certain that rates of immigration are low enough for this explanation to work.

(3) Consider again the larva which feeds upon an individual hostplant from hatching until it reaches a given pupation size. Assume that the amount of leaf eaten per day is proportional to larval size. If leaves are low in nutritive quality, both more leaves and more time are required to reach pupation. The bulk of the feeding damage occurs during the latter part of the larva's growth, when its greatly increased size enables it to ingest leaves faster. On plants of low nutritional quality, early larval stages are prolonged, increasing the likelihood of mortality—by predation or some other factor—before reaching the most gluttonous stages. As a result, plants of lower nutritive quality might lose fewer leaves overall to herbivory. But late larval stages are prolonged too, so that those which do survive are eating more. Some simple formal model is evidently needed to clarify the conditions where low nutritive quality can evolve through this kind of pathway.

The approach will be to construct a function which describes the expected amount of herbivore damage incurred by a plant from the feeding of a cohort of larvae. We assume that a larva's rate of consumption is proportional to its size so that the following integral should be proportional to the total damage to the plant:

$$D = \int_0^T l_t s_t \, dt,$$

where l_t is survivorship, s_t is size, and T is the time at which the larva reaches pupation size, S. Obviously, T is dependent on the growth function, s_t, for which we will take the simple and fairly realistic assumption of exponential growth throughout the larval period:

$$s_t = e^{gt} \quad g \geq 0.$$

For simplicity of analysis, the unit of size is taken to be size at hatching, and g, growth rate, incorporates the variable we wish to study, the effect of nutritive quality. Note that when g is increased, nutritive quality is higher, and $g = 0$ means nutritive quality is so low that growth is prevented completely.

By definition of pupation size, $S = e^{gT}$, so that $T = (\ln S)/g$.

So now, changing focus to damage as a function of g, we have

$$D(g) = \int_0^{(\ln S)/g} l_t \, e^{gt} \, dt.$$

The form of l_t is the crucial factor determining whether an impediment to nutrition can benefit the plant. We will first take the case of constant mortality and later consider deviations from this pattern.

Let there be a constant mortality μ, so that

$$l_t = e^{-\mu t}.$$

Hence

$$D(g) = \int_0^{(\ln S)/g} e^{(g-\mu)t}\, dt$$

$$= \frac{1}{g-\mu}\left[e^{(g-\mu)(\ln S)/g} - 1\right].$$

Now we want to see how D is affected by changes in g so we find the derivative

$$\frac{dD(g)}{dg} = \frac{1}{(g-\mu)^2}\left[\frac{\mu(\ln S)(g-\mu)}{g^2}e^{(\ln S)[1-(\mu/g)]} - (e^{(\ln S)[1-(\mu/g)]} - 1)\right].$$

Lowered nutritional quality will benefit the plant if $dD(g) > 0$; that is, if

$$\frac{\mu}{g}\left(1 - \frac{\mu}{g}\right)\ln S > 1 - e^{-(\ln S)[1-(\mu/g)]}.$$

Figure 1 shows the values of (μ/g) for which this inequality is true. In Lepidoptera, ratios of final to initial size (our S) usually fall between 500 and 10 000 (examples in Richards & Davies, 1977, p. 364; Campbell, 1962; Rock, 1972). For ratios greater than 500 (ln $S = 6\cdot2$), decreased nutritional quality is certainly favored if $(\mu/g) > 0\cdot2$. To obtain a rough expectation for
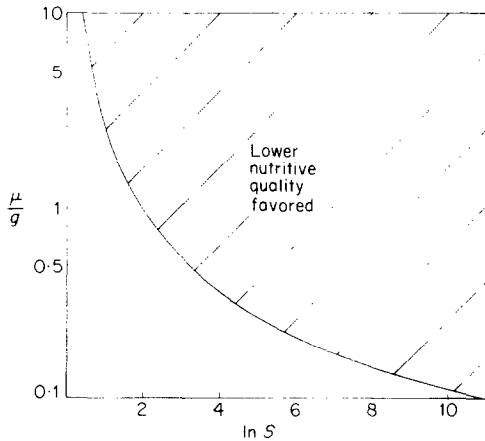

FIG. 1. Values of μ/g and ln S for which lower nutritive quality is favored under the constant mortality sub-model.

(μ/g), note that, for insects feeding primarily as larvae and assumed to be in ecological equilibrium with their host plants,

$$\frac{S}{c}l_T = 1$$

where $1/c$ represents the proportion of biomass loss (for the population) between pupae of one generation and newly hatched larvae of the next. In other words, $1/c$ takes into account factors such as mortality of non-larval stages, the "wastage" of male production (in so far as males do not contribute biomass to the eggs and so do not directly support population growth), and the proportion of adult female biomass not converted into progeny. In our model we can substitute

$$l_T = e^{-\mu T} = e^{-(\ln S)/g}$$

and the above equilibrium equation then gives

$$\frac{\mu}{g} = 1 - \frac{\ln c}{\ln S}.$$

The model thus certainly indicates adaptiveness of no-cost changes towards low nutritive value if

$$1 - \frac{\ln c}{\ln S} > 0\cdot 2, \text{ that is, if } 0\cdot 8 \ln S > \ln c.$$

The lowest S for the above lepidopteran examples is about 500. Even at this ratio the condition holds if c is less than approximately 150; that is, if more than 1/150th of female pupal weight is converted to newly hatched female larvae of the next generation. As this level of efficiency is probably greatly exceeded in most insects, this constant-mortality sub-model tends to confirm the possibility of the evolution of defense through low nutritive quality.

Constant mortality, however, is a weak assumption. What are the effects of disproportionate mortality of either large or small larvae? We consider two extreme cases to see whether benefits to the plant are increased or decreased by a change in either direction.

Suppose all mortality occurs in a certain narrow "size window" which occurs either just after the eggs hatch (I: early instar mortality) or just before the larvae pupate (II: late instar mortality). We suppose that this much higher localized mortality is just enough to bring the population survivorship curve down to l_T; so that, at sizes outside the window, mortality is zero (Fig. 2).
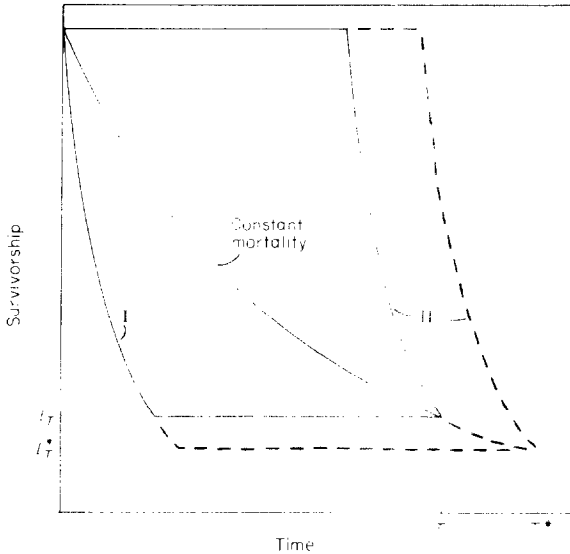
FIG. 2. Effects of low nutritive quality on survivorship in the cases of early instar mortality (I), constant mortality throughout larval stages, and late larval mortality (II). Starred values and dashed lines indicate effects of low nutritive quality.

The effect of lowering nutritive quality is to widen the window in time so that the constant mortality goes on longer and lowers l_T. At the same time, the function s_t is "stretched" over its entire length (in both cases). Thus, on the side of the advantage—the lowering of l_T—case I gains more than case II, because its survivorship function falls much earlier; the same proportion of larvae die in either case, but in case I they die before eating as much. As regards the disadvantage—the increase in T and the need to consume more—both cases are affected the same.

Thus, starting from a constant mortality case which is just neutral for the adaptive advantage of lowering nutritive quality [i.e. a case where $(\mu/g)[1 - (\mu/g)] \ln S = 1 - e^{-(\ln S)[1-(\mu/g)]}$], it is clear that any tendency to form a type I window will make the strategy become adaptive while any tendency to form a type II window will make it maladaptive. In short, if late instars die relatively more, the strategy is less likely to be adaptive.

One further simple sub-model can be used to illustrate a case where late instar mortality is greater. Consider the survivorship pattern $l_t = 1 - pt$. Such linear survivorship is admittedly artificial but approximations occur in nature. If we replace the constant mortality expression with this one and treat p as constant, then following the same reasoning as before our model

yields the result that lower nutritive quality is favored whenever

$$c > S - \frac{1}{2}\frac{S \ln S(S-1)}{S \ln S - (S-1)}$$

For the region of values of c and S already treated as reasonable this is easily fulfilled. However, constant p means that the mortality pattern is wholly dependent on age and not at all on size. Thus benefit to the plant occurs because larvae are being forced to defer their fast-feeding stages until times when fewer are left to feed. In nature, decreased survivorship of late instars, implicit in this survivorship pattern, is likely to result from their larger size rather than from age, so we might expect the mortality rate to ease off on slower growing larvae: this would be the case if, for example, the higher late mortalities occur because birds see large larvae more easily. So another assumption, erring at a different extreme with respect to the likely real situation, would have p dependent on g in such a relationship that l_t maintains its descending linear form but aims, whatever the growth rate, to hit the same survivorship (c/S) at time of pupation. This gives the condition

$$c - 1 + \frac{(S-c)(S-1)}{S \ln S} < 0,$$

which is not true for any feasible c and S. In this case, then, lowered nutritive quality is never favored. It is, no doubt, artificial in that the mutant plant, besides failing to prevent damage, fails even to lower fitness of the herbivore. It is, however, no more unrealistic than the case which has mortality independent of size.

Price (1975, pp. 139–140) divides survivorship curves for herbivorous insects into two categories. One set approximates our original case of constant mortality, with perhaps slightly higher mortality of late stages. The other group shows much greater late stage mortality, approximating the linear case just considered. Our analysis has indicated that more data on the effect of an unusually low nutritive quality on pattern of survivorship in nature is needed before we can say whether insects of this group could be effective selective agents.

We have not considered costs of being unnutritious. Even where one or more the the three pathways is applicable, the evolution of low nutritive quality depends upon costs to plants of producing digestibility-reducing compounds such as tannins or of maintaining low nutrient concentrations in tissues.

In summary, the point of this note is to caution that what hurts herbivores is not necessarily a help to that plant which steps out of line to inflict hurt.

Some herbivore–plant interactions probably do not fit any of our three models, suggesting that sometimes both plant and animal may suffer through mutation to lowered nutritive quality. The common view that low nutritive quality of plant tissue is an anti-herbivore adaptation must be allowed as plausible but it remains uncertain. It will be hard to refute. If life history and mobility parameters of winter moth eliminate it as a force favoring tannin production in oaks (for example), tannin might still be conjectured to be adaptive against some other herbivore. Many other insects besides winter moths ravage oaks and the proposal to seek life history and behavioral data for them all (Southwood, 1961) is daunting.

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