Chapter Ten

LECTINS AND PLANT-HERBIVORE INTERACTIONS

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INTRODUCTION

When an animal takes a bite out of a plant, it is gustatorily and digestively treading on a battlefield implanted and strewn with traits generated by natural selection during millions of years of acts of herbivory. The glycoproteins called lectins are heterogeneous in kind, place and density in this battlefield. How animals respond to this pattern and its parts suggest that lectins may be more than simply one more of nature's many kinds of glue. They are quite unfortunately called "lectins" since it is their sticky nature that is the basis of their biological function; while they are specific in their attachment to certain sugars, the fact that the same sugars can occur on the surface of many different kinds of cells makes them in fact highly variable in the specificity of their stickiness at the cellular level of organization (which surely is the level of concern when one calls them "lectins").

What are the relevant properties of a glue?
- How tightly does it adhere to two or more relevant surfaces?
- How specific is its adherence?
- How quickly and easily can it be dissolved or neutralized by the user?
- How expensive is it?

How can it be used for other purposes?
What is its shelf life?
How much is needed to do the job?
How long does it retain its properties once in place?
What is its setting time?

If we ask these sorts of questions of a child's paste pot, of a welder's rod, of electrician's tape, of a gardener's tanglefoot, of epoxy resins, of a spider's web, of a flower's stigma, to say nothing of what Fort Dix thought up during the Viet Nam war, we quickly note that lectins have a number of properties which should lead, through natural selection, to their becoming on occasion part of a plant's defense repertoire. Just as there are specific places in battle where boiling oil is an important direct defense (though hot oil is generally much more important as a motor lubricant), a moderate-sized protein that adheres tightly to very specific sorts of surfaces can on occasion be an effective defensive weapon.[1] Needless to say, direct defense against other organisms is certainly not the biological function of all lectins any more than providing a microbe binding site on roots is the biological function of the lectins in cecropia moth blood.[2]

Among the array of lectin kinds, concentrations and placements, where is it reasonable to search for a defense function against multicellular herbivores? Before embarking on this query, I must very briefly mention what lectins apparently do to a herbivore that consumes them. They apparently bind to gut surfaces, be they of the animal or of its essential microbial fauna, and alter the function of those surfaces.[3] In short, they fall in the category of digestion inhibitors, as do protease inhibitors,[4,5] polyphenols (tannin, lignin, etc.),[5,6] cellulose,[7] unassimilable starches, etc. Since a lectin molecule is small relative to the herbivore's gut surface area, and since it has only a few active sites,[3] for a lectin to be a significant digestion inhibitor it will have to occur as a relatively large proportion of the diet.

Without dipping further into the philosophical background of why study protein glues as defensive compounds as well as molecules useful to the plant in other ways, I would like to relate the early unfoldings of a study of an

exceptional little mouse, a study that bears strongly on lectins as defenses against seed predators.

LIOMYS SALVINI

Liomys salvini (Figure 1) is a hispid pocket mouse in the same family of professional seed-eaters, Heteromyidae, as is the more familiar desert kangaroo rat, Dipodomys. L. salvini, or 'guarda fiesta' as it is locally known, however is a forest-floor mouse. This tropical mouse is a common inhabitant of the deciduous forests of Guanacaste Province, on the lowland coastal plain of Costa Rica.[8-10] Its diet is almost entirely seeds of herbs, vines, shrubs and trees, which it finds in the litter and in animal dung. It also shells seeds out of certain species of newly fallen fruits. The specific area where I have studied this mouse, Santa Rosa National Park (about 35 km north of Liberia, Guanacaste Province, Costa Rica), has a flora of about 650 species of broad-leafed plants,[11] over half of which are woody perennials with seeds large enough for this mouse to bother with. Of this set of seeds, it is conspicuous that certain species

Figure 1. Adult female Liomys salvini (46 g) from Santa Rosa National Park, Guanacaste Province, Costa Rica (July, 1980).

are flatly rejected (Table 1). These seeds lie about on the forest floor, in or out of their fruits, for months without being eaten. They are never encountered in the pouches of trapped mice. They were rejected when presented to the mice as part of seed mixes on plates at which free-ranging mice nightly foraged in the forest.

It is possible that in some absolute sense some species of seeds are rejected because they are nutritionally a badly imbalanced food; however, this does not explain why they are not eaten as, for example, a carbohydrate source in combinations with other seed species. Some are rejected because they are in a container too hard or unwieldy for L. salvini to gnaw through (Acrocomia vinifera palm nuts are an example), but the great majority of rejected seeds are no harder than those that are eaten and are often bitten into in apparent testing behavior. I find the most reasonable working hypothesis to be that these seeds are rejected for the chemicals that they contain other than those that are normally thought of as dietary nutrients for small mammals. Since this essay is directed at the obnoxious protein portion of a seed's defenses, I will focus on that aspect rather than some of the other known (additional) potential defenses of these seeds (e.g., Mucuna pruriens seeds contain L-dopa,[12]

Table 1. Some species of native forest and forest-edge woody plant seeds and nuts eagerly eaten and thoroughly rejected by free-foraging Liomys salvini in Santa Rosa National Park.

Rejected	Accepted
Acrocomia vinifera	Phaseolus lunatus
Spondias mombin	Enterolobium cyclocarpum
Lonchocarpus acuminatus	Cochlospermum vitifolium
Lonchocarpus costaricensis	Malvaviscus arboreus
Pithecellobium platylobum	Hymenaea courbaril
Canavalia brasiliensis	Forsteronia spicata
Canavalia maritima	Pithecellobium saman
Dioclea megacarpa	Cissus rhombifolia
Mucuna pruriens	Sesbania emerus
Ateleia herbert-smithii	
Cassia grandis	

Dioclea and Canavalia seeds contain canavanine,[13] Ateleia seeds contain 2,4-methanoproline and 2,4-methanoglutamic acid,[14] etc).

With the current understanding of potentially toxic seed proteins, protease inhibitors and lectins are the two protein-aceous candidates to examine with respect to Liomys rejection of seeds as food. Turning briefly to protease inhibitors, in the accepted column of Table 1 there is Enterolobium cyclocarpum, a mimosaceous legume seed that contains a substantial amount of a protease inhibitor (C. A. Ryan, personal communication), but no lectins, at least as measured by agglutination of human, rabbit or hamster blood (I. Liener, personal communication). This seed, in the hard (dormant, ungerminated) or germinated state can serve as the sole diet of L. salvini for a month or more in the laboratory,[10] is avidly sought by L. salvini in the field, and is chosen over many other acceptable seed species in choice tests. This suggests that L. salvini may be sufficiently specialized as a seed predator that it carries gut enzymes or other digestive repertoires generally resistant to protease inhibitors (it is unlikely, however, to be like the bruchid beetle larvae that are resistant to protease inhibitors by having a digestive system that operates largely without proteases[15]). To test this, I fed 4 or 5 L. salvini on diets of pure rat chow, and on rat chow that was 1, 5 or 25% soybean typsin inhibitor (Table 2). There was no reduction in weight (and even a hint of weight increase compared to animals on pure rat chow) or rejection of the adulterated food, nor was there any visible symptom of a toxic or otherwise debilitating effect of the adulterated food. As a working hypothesis, I will assume that L. salvini is highly resistant to protease inhibitors in legume seeds.

There are at least 4 species of seeds in the rejected column of Table 1 that show strong agglutinating activity towards human, rabbit and hamster red blood cells (I. Liener, personal communication): Canavalia brasiliensis, C. maritima, Dioclea megacarpa, and Cassia grandis. The most desirable test would be to extract the lectins from these seeds and feed them to L. salvini. This step is planned. However, black beans (Phaseolus vulgaris) are a rich source of a potent lectin that has been shown to be lethal when incorporated in the diet of the larvae of a bruchid beetle.[1]

Table 2. Weight changes and survival of adult Liomys salvini (hispid pocket mice) fed a variety of diets containing potentially toxic proteins. The animals were wild-caught and maintained in individual cages with ad lib food and water at temperatures approximating those of their natural habitat (Santa Rosa National Park, Guanacaste Province, Costa Rica, May-July, 1980.

Treatment	Mouse number	Initial[1] weight (g)	Weight change (g)	Final Health	Duration of feeding (days)	Weight change per day (g)
Rat chow:						
	37 ♂	32	+1	healthy	9	+0.11
	38 ♀	40	+1	healthy	7	+0.14
	39 ♀	41	-4	healthy	7	-0.57
	40 ♂	59	0	healthy	7	0
	41 ♀	36	+1	eaten by boa	4	+0.25
$\bar{X}$		41.6	-0.2			-0.02
s.d.		10.4	2.2			0.32
Rat chow with 1% soybean trypsin inhibitor:						
	1 ♀	50	0	healthy	6	0
	5 ♀	40	0	healthy	6	0
	7 ♀	41	-1	healthy	6	-0.17
	8 ♀	40	0	healthy	6	0
	10 ♀	47	+1	healthy	6	+0.17
$\bar{X}$		43.6	0.0			0.00
s.d.		4.6	0.7			0.12
Rat chow with 5% soybean trypsin inhibitor:						
	3 ♀	45	+1	healthy	6	+0.17
	9 ♀	43	+2	healthy	6	+0.17
	12 ♀	43	+2	healthy	6	+0.33
	15 ♀	38	+1	healthy	6	+0.17
$\bar{X}$		42.3	+1.3			+0.21
s.d.		3.0	0.5			0.08
Rat chow with 25% soybean trypsin inhibitor:						
	37 ♂	33	0	healthy	5	0
	38 ♀	41	+1	healthy	5	+0.20
	39 ♀	37	+1	healthy	5	+0.20
	40 ♂	59	+1	healthy	5	+0.20
$\bar{X}$		42.5	+0.8			+0.15
s.d.		11.5	0.5			+0.10

Table 2 (continued)

Treatment	Mouse number	Initial[1] weight (g)	Weight change (g)	Final Health	Duration of feeding (days)	Weight change per day (g)
Cooked black beans:						
	51 ♀	39	+1	healthy	6	+0.17
	53 ♀	45	-1	healthy	5	-0.20
	54 ♀	25	+4	healthy	5	+0.80
	58 ♀	31	+1	healthy	5	+0.20
	59 ♂	54	-3	healthy	5	-0.60
$\bar{X}$		38.8	+0.4			+0.07
s.d.		11.4	2.6			0.52
Uncooked black beans:						
	1 ♀	46	-7	dying[2]	3	-2.33
	3 ♀	36	-7	dying[2]	3	-2.33
	5 ♀	41	-6	dying[2]	3	-2.00
	7 ♀	35	-4	dying[2]	3	-1.33
	50 ♀	44	-12	dead	10	-1.20
	52 ♀	38	-6	dead	3	-2.00
	55 ♀	40	-3	dead	3	-1.00
	56 ♀	34	-5	dead	3	-1.67
	57 ♀	35	-4	dead	3	-1.33
$\bar{X}$		38.8	-6.0			-1.69
s.d.		4.3	2.6			0.50
Animals that refused to eat toxic seeds:[3]						
	24 ♀	48	-15	dead	7	-2.14
	21 ♂	36	-9	dead	4	-2.25
	27 ♀	46	-10	dead	6	-1.67
	28 ♂	38	-10	dead	7	-1.43
	29 ♀	38	-9	dead	4	-2.25
	30 ♀	32	-6	dead	2	-3.00
$\bar{X}$		39.7	-9.8			-2.12
s.d.		6.1	2.9			0.55

[1] When a mouse appears more than once in this table, the first time its "initial weight" is the weight at the time of capture. The second time its "initial weight" is the weight in the laboratory following at least 5 days in the laboratory feeding on high quality food.

[2] These 4 mice would not have lived another 12 hours and had the usual traits of starving mice in their last few hours of life (severe shakes, closing eyes, poor coordination, little response to stimulation).

[3] Given the data available, I will assume that mice that voluntarily starve themselves to death die at the same rate as those deprived of all food.

This result is particularly striking because bruchids as a group are seed predators.[16] Black beans are cheap and easily available in Costa Rica. They also suffer no depredation by rats when stored in rat infested habitations.

When L. salvini were offered a diet consisting solely of black beans that had been boiled for one hour, they maintained their body weight and other wise appeared quite healthy (Table 2). However, when given a pure diet of only uncooked black beans, L. salvini lost weight at a rate no different from that of those eating no food at all (Table 2; $t_{13\ d.f.} = 1.54$, n.s). The one animal out of 9 that still appeared healthy after the third day survived for 10 days and probably was in exceptionally good condition at the beginning of the experiment. During the course of the experiment, the mice did eat small but highly variable amounts of uncooked black bean seeds. The mouse that lived so long (number 50 ♀, Table 2) ate roughly half the weight of uncooked black bean seeds per day as it would have were it maintaining its body weight on laboratory rat chow. It lost 27% of its body weight before death, which is nearly twice the loss these mice usually tolerate.

As a working hypothesis, I conclude that it is the lectin in the black beans that is killing the mice. The mode of action is probably the combined effect of direct starvation caused by food rejection (averaged over 34 mouse days, the bean consumption per day per mouse was 0.16 gram, and these mice require 2 to 4 grams per day of laboratory rat chow to maintain their body weight) and reduced nutrient uptake through the intestinal wall, as suggested by there being no conspicuous relationship between rate of weight loss and amount of bean eaten. While there are protease inhibitors in black beans, the impressive ability of L. salvini to live on food rich in soybean trypsin inhibitor and live on protease inhibitor-rich Enterolobium cyclocarpum seeds suggests that protease inhibitors are not the likely cause of black bean seed rejection by L. salvini. There are no known alkaloids or uncommon amino acids in commercial black beans, nor are there any other known potentially toxic molecules besides the proteins. The ultimate test of this working hypothesis depends on the availability of purified black bean and other lectins in 10 to 30 gram amounts. L. salvini is a very abundant seed predator in the forests it occupies. If it can be definitively shown that it cannot eat seeds rich in lectins, it can be stated with certainty that these seed lectins serve as a defense role against rodents irrespective of their other uses to the seedling. Lectins in the diet at naturally occurring concentrations can kill the larvae of a seed predator

bruchid.[1] This demonstrates that seed lectins are functional as are alkaloids, uncommon amino acids, cyanogenic glycosides, etc. in defending seeds against insect seed predators.

If a dietary chemical is lethal to L. salvini, the situation is more than the mere act of hitting a naive gut with just any potentially nasty compound. This seed-eating specialist is unaffected by the very protease inhibitors that are a major reason for humans to boil or otherwise process seeds before eating them. For example, the seeds of E. cyclocarpum are lethal if they are the sole diet of Sigmodon hispidus, another terrestrial rodent in the same habitat (though S. hispidus does quite well on them if boiled).[17] L. salvini can live on a pure diet of HCN-rich Phaseolus lunatus seeds. E. cyclocarpum seeds are also rich in pipecolic acid and albizzine (E. A. Bell, personal communication), two uncommon amino acids with conspicuous insecticidal properties at the concentrations found in seeds.[18,19] Sesbania emerus seeds are rich in canavanine (G. A. Rosenthal, personal communication) yet eaten readily by L. salvini. This animal has a versatile gut yet there appears to be at least one lectin that it cannot handle.

WHAT PROCESSES EVOLUTIONARILY PUT LECTINS IN SEED CHEMICAL REPERTOIRES?

There are five traits of the system that are relevant this question:

1) The forest has many kinds of seeds, and the seeds of each species contain a unique combination of potentially defensive compounds.

2) The forest has many kinds of seed predators, each with the ability to ignore or detoxify some of these compounds, but not all.

3) Lectins are just one of the many protective traits a seed contains.

4) There are two different seed-predator responses to a seed, each likely to generate different traits in the defense array.

a) The rodent-type animals, such as Liomys salvini, try a newly encountered seed, and if is has the appropriate defenses, reject it. Rejection will depend as well on hunger, gut conditioning, body weight of the animal, alternate available foods, health and fat condition of the animal, perceptability of the defense compound, etc. The selective pressure favoring better-defended mutants is essentially constant over the years. This is because each year there are new recruits in the habitat that have to learn about the seeds of that habitat and season, and because there is a rodent-specific rate of forgetting that leads to re-sampling and relearning as each species of plant comes into seed again year after year. Since small rodents are very common, an unprotected mutant will be quickly located and its seed crop probably eliminated by the local set of mice. On the other hand, as selection for resistance traits occurs, the seed will have to be a consistently important part of the diet of the mouse for there to be such a strong selection that the mouse evolutionarily increases or shifts its detoxification abilities to encompass the change. The more usual rodent response to a mutant seed that is better defended should be to eat less of it. This system does not proceed to total inedibility for all seeds because the mice are also dispersal agents, defenses have economic costs to both parent and off-spring, plants and rodent densities fluctuate, rodent detoxification abilities do change, etc.

b) The insect-type animals oviposit on a particular species of host seed (or on its fruit), usually the only species of seed in the habitat that their larvae can eat.[16] Their larvae are specialists on the chemisty of that seed.[20-22] In addition, all mature and immature stages are behaviorally as well as physiologically programmed to deal with the host plant's other traits (e.g., timing of fruiting, fruit chemistry, odor cues for location, susceptibility to parasitoid attack while in that species of seed, etc.). While these animals may be very regular and deadly in killing their host's seeds, they pose no threat in contemporary time to nearly all other plant species present in the habitat, even if the larvae could develop in their seeds. There are undoubtedly many

other seed species in the habitat, which, when only seed chemistry is considered, could serve as hosts for a particular species of insect that does not, in fact, feed on them.

However, probing of other hosts does occur in evolutionary time, but rarely. A probe occurs when an ovipositing female makes an oviposition error or owing to a shortage of its regular hosts, oviposits on almost anything that contains even a fraction of the oviposition stimuli emitted by the usual host. Futhermore, in the latter case (and possibly the former) a successful probe may lead to a shift in the host seed species used, rather than a broadening of the host list. This is because it is likely that the beetle will have a higher fitness as a specialist on either one or the other hosts than as a generalist on both, since thorough bypassing of a plants defenses often requires very fine tuning at the behavioral, morphological, physiological, biochemical, etc. level. Fine tuning may well be impossible owing to differences in timing of seed production, seed chemistry, fruit morphology, etc. If L. salvini or small rodents cannot handle lectins in general, this will select for strong convergence in lectin traits in the seeds these rodents confront; convergence will be on that molecule that works the best at an optimal cost for the genetic lineage of the parent and offspring that bear it.

It is the insect-type seed predator that should be responsible for much of the fine tuning of a lectin's traits, when that lectin is serving primarily as a defense compound in a seed. If each kind of lectin requires a different chemistry of detoxification by a host-specific insect, then a seed's lectin traits will be occasionally evolutionarily modified because the mutant repels a host-specific insect seed predator, just as is the case with alkaloids, uncommon amino acids, cyanogenic glycosides, etc. In contrast to the case where a group of seed predators (e.g., rodents) can bypass a class of compounds (e.g., Liomys versus protease inhibitors) or is repelled by a class of compounds (e.g., rodents versus lectins), each time a lectin's properties are changed it is a novel defense as seen by the host-specific insect. Here, then, natural selection will not result in convergence of traits among lectins, but rather in the continual appearance of

new types. There should even be active selection that results in divergence in lectin types because a mutant, that is a change in a direction already occupied by other lectins, is likely to become susceptible to the host-specific seed predators that can bypass those other lectins. Both diversification and divergence in lectin types should, however, approach an equilibrium level. The level should be determined in part by all those other ecological processes besides physiological seed availability that determine the numbers, kinds and diversity of seed predators in the habitat, and in part by the other non-predator-related selective pressures on lectins.

5) Like other compounds found in the seedling's bag lunch, lectins should be under strong selection to be of multiple use in this weight-, volume-, resource quality-limited container. The ideal combination of compounds in a seed is that which maximizes the fitness of that genetic lineage. Surely this will require a complex balance of

a) partitioning of parental resources among the seeds (seed size, weight, number, etc.),

b) seed photosynthesis and therefore contribution to its own resources, and

c) seed resources for seedling growth and protection against herbivores (probably no molecule is ideal for both functions).

Specifically, lectins will be the focus of selective pressures associated with

a) their use as glue in development, such as in the attachment of symbiotic bacteria to roots or in the binding of different cells within the organism,

b) their degradative destruction as an amino acid and small polypeptide source in seedling metabolism,

c) their use as protective compounds in the cotyledons and in newly produced vegetative tissues (deterrentsto contemporary herbivory by generalists and evolution of herbivory by specialists).

In some plants, past selection will have been such that lectins were never functional as anything more than one kind of glue, a glue of importance only in some very internal biochemical sense. Here, then, other compounds are the defenses and storage compounds in the seeds. But somewhere early in the dim history of legume seeds, there was a combination of herbivore susceptibility and plant lectin synthesis capacity that led to lectin-rich seeds becoming an integral part of the character of a 'successful' species of plant which then radiated in various ways to give us many species of lectin-rich legume seeds. Alternatively, one may hypothesize that this even occurred many times, owing to the general presence of the lectin-synthesis ability of legumes, which in turn pre-disposed them to selection by herbivores that got bad stomach aches from diets rich in protein glues. One cannot chose between these two historical scenarios with the data at hand, but it is obvious what sort of amino acid sequence studies are needed to distinguish among them. They are also not mutally incompatible. Owing to the chemical complexity of proteins, as contrasted with small defensive molecules like alkaloids and uncommon amino acids which can have absolutely convergent end products in their synthesis by different plants, the history of a protein molecule is to some more extreme degreee incorporated in its structure. The question has become not 'Is a lectin for defense?', but rather 'What are the various ways that sticky glycoproteins are functional?' 'What selects for their detailed traits?' and 'What selects for deposition in certain plant parts in sufficient bulk to give a herbivore a gut ache?', bearing in mind that herbivores come in widely differing sizes, food consumption rates and intensity of desire to eat a particular plant part.

WHY ARE LECTINS SO PROMINENT IN SEEDS AND TUBERS?

Assuming that the relatively high concentrations of kinds and amounts of lectins in seeds and tubers is not a sampling artifact, there are several plausible ecological reasons why this severe distributional heterogeneity should occur.

Dilution. Many species of animals that commit severe seed predation on mature seeds are sufficiently specialized on this diet that they eat almost no other food for all or

much of their lives. This means that the contents of the seed are likely to be all or nearly all of their stomach and intestinal contents at a given time. That is to say, whatever is in the seed runs little risk of being diluted out in a larger bulk of different food (as occurs, for example, when a horse digests some of the hard lectin-rich legume seeds it swallows along with leafy feeds). On the other hand, seed contents are very concentrated nutrients, and compared to a foliage-eating animal, a seed-eater consumes a miniscule amount of food. For example, a bruchid beetle larva in a legume seed may only consume twice its last instar body volume in seed contents during its entire development, while a moth caterpillar eating leaves may consume is own volume of food during every 24 hours of active growth. In short, the amount of lectin required to maintain a 5% lectin titer in a seed-eater's gut is easily only 1 to 5% of that which would be required for the same effect in a foliage-eater's gut of the same body weight. That is to say, an expensive digestion inhibitor like a lectin or protease inhibitor may be economically most appropriate for a seed while the much cheaper (per gram) polyphenol digestion inhibitors are most appropriate for foliage. While both may be found in each type of plant part, here I am discussing the forces that keep the disproportionalities in the system.

Bag lunch. Seeds and tubers are storage devices, and in the case of the former, volume- and weight-conscious ones. A lectin (as well as a protease inhibitor) may double as a polypeptide and amino acid storage unit (and of course may even have its evolutionary roots in an innocuous storage protein). However, to the degree that only innocuous storage proteins are found in more vegetative plant parts, we are then again left with the disproportionality question. Futhermore, while it is all very well to beat your sword into a ploughshare, it may be best to put it into the closet and buy a plough when the war is over, because wars have a way of reappearing each generation. Finally, to give you a very high quality sword, your mother may well have used such high quality steel that it makes a lousy ploughshare.

Fitness. Finally, and most definitely not least, there is the simple answer that gram for gram, seeds and tubers have the highest fitness value of any plant part.

From the viewpoint of the juvenile in the seed, the seed is the essence of fitness. For the parent plant, the seeds are one of its throws of the dice to remain in a surviving lineage. Seeds are probably the most thoroughly protected of all plant parts and part of that thoroughness is achieved by containing quite physiologically active compounds of many kinds in high concentrations. Virtually all seeds contain at least 1 potent digestion inhibitor and 2 to 5 small molecules (cyanogenic glycosides, alkaloids, cardiac glycosides, uncommon amino acids, cyanolipids, etc.)[23] unless they are involved in some sort of population-level seed predator satiation (e.g., as in conifers, oaks, bamboos, niloo, chestnuts, dipterocarps, etc.),[24-26] physical protection (hard nuts), or very small size (many herbs). Our own village histories undoubtedly taught us this. Seeds and tubers are where the goodies are, but you cannot eat them unless either you process them (cook them, break them, dig them up, breed out their defenses) or they are so chemically defenseless as to be eaten by the bulk of the herbivorous animals in the habitat. The same ecological story applies to the presence of protease inhibitors and largely indigestible starches and other complex sugars in seeds and tubers, but I shall let that lie as it is not the subject of this symposium. On the other hand, it may be noted that the intensity of protection required is related not only to the value to the owner (mother and seed), but the value to the thief. Seeds and tubers contain the highest concentrations of animal nutrients in the plant world, and often occur at a density in time and space quite high enough to support many species of herbivores for much if not all of the year or generation. Such an array of barbarian hordes cannot be kept at bay with a few ditches and spears.

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