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MINI-REVIEW

Minireviews provides an opportunity to summarize existing knowledge of selected ecological areas, with special emphasis on current topics where rapid and significant advances are occurring. Reviews should be concise and not too wide-ranging. All key references should be cited. A summary is required.

The ecological significance of toxic nectar

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Although plant-herbivore and plant-pollinator interactions have traditionally been studied separately, many traits are simultaneously under selection by both herbivores and pollinators. For example, secondary compounds commonly associated with herbivore defense have been found in the nectar of many plant species, and many plants produce nectar that is toxic or repellent to some floral visitors. Although secondary compounds in nectar and toxic nectar are geographically and phylogenetically widespread, their ecological significance is poorly understood. Several hypotheses have been proposed for the possible functions of toxic nectar, including encouraging specialist pollinators, deterring nectar robbers, preventing microbial degradation of nectar, and altering pollinator behavior. All of these hypotheses rest on the assumption that the benefits of toxic nectar must outweigh possible costs; however, to date no study has demonstrated that toxic nectar provides fitness benefits for any plant. Therefore, in addition to these adaptive hypotheses, we should also consider the hypothesis that toxic nectar provides no benefits or is tolerably detrimental to plants, and occurs due to previous selection pressures or pleiotropic constraints. For example, secondary compounds may be transported into nectar as a consequence of their presence in phloem, rather than due to direct selection for toxic nectar. Experimental approaches are necessary to understand the role of toxic nectar in plant-animal interactions.

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Individuals are often simultaneously under selective pressures exerted by multiple interactions, including both mutualisms and antagonisms. For example, although plant-herbivore and plant-pollinator interactions are typically studied separately, most plants must attempt to attract pollinators while also escaping herbivores. Herbivores and pollinators can therefore both exert selective pressures for plant traits via direct effects on plant fitness (Schemske and Horvitz 1988, Juenger and Bergelson 1997, Strauss and Armbruster 1997). Selection for defense against herbivores may be influenced by selection for attracting pollinators, and vice versa (Strauss 1997). Many traits that affect herbivory in plants are closely related to those affecting pollination. For example, floral resins that once functioned as defenses can be co-opted for pollinator rewards (Armbruster 1997, Armbruster et al. 1997), and pleiotropic effects of an allele determining floral pigmentation may

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influence vegetative resistance to herbivores (Simms and Bucher 1996). In a selection experiment for resistance to beetle herbivores, high-resistance *Brassica rapa* lines were less preferred by pollinators than low-resistance lines, suggesting a tradeoff between herbivore resistance and pollinator preference (Strauss et al. 1999).

Because of these complex interactions, net selection on plant traits, such as production of secondary compounds, is difficult to predict. If plant defensive compounds are present in floral tissues and are deterrent to pollinators, or if the cost of producing the compound results in less attractive floral structures, then pollinators may select against the production of these compounds (Detzel and Wink 1993, Strauss et al. 1999). Alternatively, plant secondary compounds could increase pollinator attraction if decreased herbivory improves floral displays or rewards (Karban 1993, Lohman and Berenbaum 1996, Juenger and Bergelson 1997, Lehtilä and Strauss 1997, Callaway et al. 1999, Krupnick et al. 1999, Strauss et al. 1999, Mothershead and Marquis 2000, Adler et al. in press). Thus, evolution of some plant traits may be constrained by opposing selection from herbivores and pollinators.

Secondary compounds that are associated with resistance to herbivory have been frequently documented in floral nectar (e.g., Baker and Baker 1975, Baker 1977, Guerrant and Fiedler 1981), although nectar is usually studied in the context of pollination rather than herbivory. The general function of nectar, with its array of sugars and amino acids, is to attract pollinators and/or natural enemies of herbivores (Fægri and van der Pijl 1979). Why, then, would secondary compounds, which are generally toxic or repellent, be present in a structure whose function is the attraction of mutualists? Although this phenomenon is widespread (Baker 1977, 1978), it has received relatively little attention from ecologists. Integrating our understanding of multispecies interactions, such as plant-pollinator and plant-herbivore interactions, may shed light on traits, such as secondary compounds in nectar, that previously seemed anomalous.

Nectar secretion and composition

The mechanisms underlying the transport of secondary compounds into nectar are not known. However, the process of nectar secretion, in particular with respect to sugar concentration, has been well studied. I briefly review this literature to provide some insight into the possible mechanisms by which toxic nectar could arise. Nectar is excreted from glands called nectaries located on floral or extrafloral tissues (reviewed in Weberling 1989); here I will discuss only floral nectaries. Nectaries can be found on every type of floral tissue, including calyx, corolla, stamens and carpels. Fine ramifications of the vascular system lead up to nectaries, which may be supplied by both xylem and phloem or phloem alone (Fahn 1988). The sugar concentration in secreted nectar generally decreases as the proportion of xylem in the conducting path increases (Frey-Wyssling and Agthe 1950, Frei 1955).

There are several possible pathways by which nectar components can move from vascular tissue to nectaries and then be transported outward (reviewed in Fahn 1988). Four possible pathways for the flow of "pre-nectar" from phloem endings through the parenchymatous cells of the nectaries and into secretory cells have been suggested: (1) via the apoplast, (2) via exocytosis and endocytosis (Findlay and Mercer 1971), (3) via molecular transport across the plasmalemma and passage through cell walls, or (4) via plasmodesmata. Fahn (1988) reviewed studies of nectary ultrastructure and concluded that the transport of pre-nectar is mainly through the symplast rather than apoplast (but see Genc 1996). The high frequency of plasmodesmata traversing the walls of nectariferous cells suggests that they may play an important role in this process (Fahn 1988, Arumugasamy et al. 1993, Rumpf et al. 1994, Nepi et al. 1996) and provide a low-resistance pathway for the bulk flow of pre-nectar (Gunning and Hughes 1976). Once inside the secretory cells, nectar can be secreted by two main modes of transport: (1) eccrine secretion or active molecular transport across membranes (Lüttge and Schnepf 1976), and (2) granulocrine secretion, or transport via vesicles whose membranes fuse with the plasmalemma (Fahn 1988). Studies of nectary ultrastructure have found evidence for both types of secretion (Zer and Fahn 1992, Arumugasamy et al. 1993, Rumpf et al. 1994, Nepi et al. 1996, O'Brien et al. 1996). When flowers senesce, resorption of nectar constituents occurs in some species (Cruden et al. 1983, Nepi et al. 1996, Torres and Galetto 1998). Transport processes within nectaries, the pathway of secretion, and resorption all have the potential to influence nectar composition (Lüttge and Schnepf 1976).

Nectar is about 90% sugar by dry weight (Lüttge 1977); the other 10% consists of a myriad of compounds, including amino acids, lipids, antioxidants, mineral ions, and secondary compounds (Lüttge and Schnepf 1976, Baker 1977). The chemical composition of nectar varies widely between species, and even between different types of nectaries within the same plant species (Davis et al. 1998). Nectars are characterized by their ratio of sucrose/(glucose + fructose), which is consistent within species but varies widely between species. Amino acids are virtually ubiquitous in nectar, and their composition ranges widely between species but is generally consistent within a species (Baker and Baker 1982). Pollinator taxa have been correlated with both sugar ratios and amino acid composition across species, suggesting that there is selection for a characteristic "taste" that is recognizable to specific pollinators

(Baker and Baker 1982). Some nectars fluoresce under UV illumination while others do not; the color and intensity of fluorescence vary between more than within species, suggesting species-specific differences in compounds responsible for fluorescence (Thorp et al. 1975). Finally, many types of secondary compounds have been found in nectar from different plant species in small but consistent amounts (reviewed below).

The species-specific differences in nectar composition could be explained in two ways that are not mutually exclusive: (1) the secretory process in nectaries controls chemical composition and varies between species or (2) the constituents of nectar reflect the chemical composition of phloem, and phloem composition varies between species. Researchers differ in their emphasis on these possibilities; in his review of secretory tissues, Fahn (1988) states that "nectaries secrete unmodified or only slightly modified substances supplied directly or indirectly by the vascular tissues", while Lüttge and Schnepf (1976) emphasize the role of active transport, rather than passive diffusion, in moving sugars against concentration gradients. The latter opinion is focussed on the transport of sugars rather than other nectar constituents; Lüttge (1977) mentions that compounds other than sugars may move through nectaries by passive diffusion rather than active transport. Many secondary compounds, including alkaloids, iridoid glycosides, glucosinolates, cardenolides, and phenolics, are transported between plant tissues via the phloem (Baker and Baker 1982, Treutter et al. 1985, Mullin 1986, Montllor 1989, Molyneux et al. 1990, Wink 1992, Gowan et al. 1995, Merritt 1996). Therefore differences in non-sugar nectar composition, including secondary chemistry, may be caused by differences in phloem compounds that diffuse into nectar.

Toxic nectar: its nature and occurrence

There are many reports of nectar that is toxic or deterrent to animals, in which responsible compounds are not identified (Table 1). Most of these studies focus on honeybees or on humans poisoned by honey made from nectar of a specific plant. Several reports are anecdotal and describe bee death or narcosis following visits to flowers (Vansell and Watkins 1933, 1934, Pryce-Jones 1942, Eckert 1946, Jaeger 1961, Bell 1971, Crane 1977). Some studies removed nectar from flowers and performed laboratory assays to demonstrate that nectar was the cause of toxicity (Palmer-Jones and Line 1962, Clinch et al. 1972, Berenbaum et al. 1986, Sharma et al. 1986, Paula et al. 1997). Only one study offered both nectar and a sugar solution control in field tests; sugar solution was preferred over nectar by ants in two of four plant species (Feinsinger and Swarm 1978). These studies suggest, albeit largely through description rather than experiments, that some plant species produce nectar that is toxic or deterrent to some floral visitors. More experimental studies comparing nectar and control solutions would strengthen this argument. Also, the emphasis on toxicity to honey bees, which are not the native pollinator for many of these plants, raises the question of whether native pollinators are as affected as introduced species.

In a separate body of literature, numerous studies have demonstrated that secondary compounds occur in nectar without testing the effects of these compounds on floral visitors (Table 2). It is therefore not known whether these compounds occur in sufficient concentrations to have any ecological consequences. Thus these studies do not document nectar that is actually toxic, but identify plant species whose nectar might adversely affect floral visitors. Extensive sampling of hundreds of plant species has demonstrated that alkaloids, phenolics, and nonprotein amino acids are common in nectar (Baker 1977, 1978). Techniques have been available for decades to test for these compounds easily in small quantities (Baker 1977, Guerrant and Fiedler 1981); it is possible that many other compounds that are not so easily screened are also common.

These two distinct bodies of literature demonstrate that there are nectars that contain secondary compounds but whose toxicity is unknown, and also nectars that are toxic to some floral visitors but whose chemistry is unknown. Fortunately, some studies have also established a link between nectar chemistry and toxicity (Table 3). In some cases, secondary compounds were isolated from nectar known to have toxic or repellent qualities, but the compounds were not tested separately (Pryce-Jones 1942, Kozlova 1957, Barragan de Dominguez 1973, Prys-Jones and Willmer 1992). Frankie et al. (1982) correlated decreased pollinator visitation with increased phenolics in nectar, but with a sample size of only three trees. In the most compelling studies, compounds were identified and isolated from nectar that deterred or poisoned floral visitors in the field. These compounds were then offered in sucrose solutions to the same visitors, with or without controls, and produced results (either poisoning or deterrence) similar to those observed in the field (Waller et al. 1972, Majak et al. 1980, Stephenson 1982, Hagler and Buchmann 1993, Carey and Wink 1994). Thus in manipulative experiments, a clear link has been established between nectar secondary compounds and toxicity.

Why does toxic nectar occur?

Although the nature, extent and consequences of toxic nectar are not yet fully understood, several hypotheses regarding the functions of toxic nectar have been proposed by various authors. These are reviewed below,

along with the additional hypothesis that toxic nectar may not provide any benefit to plants. For simplicity, in the remainder of this paper the term 'toxic nectar' will be used to refer to nectar that deters or poisons floral visitors, and it is assumed that secondary compounds are generally, but not always, the cause of this toxicity. Some caveats should be kept in mind: (1) nectar that contains secondary compounds is not always toxic (Guerrant and Fiedler 1981, Haber et al. 1981), (2) nectar that is deterrent or toxic to one floral visitor may not affect others (Stephenson 1981, 1982), and (3) individual secondary compounds may serve multiple roles and interact synergistically (Duffey and Stout 1996). Thus, many of the adaptive hypotheses are not mutually exclusive.

The pollinator fidelity hypothesis

Baker and Baker (1975) were the first to speculate on the functions of "unfavorable substances in floral nectar". They proposed that bees are more resistant to alkaloids than adult Lepidoptera, and that alkaloids in nectar encourage pollination by specialist bees rather than "flower-inconstant" lepidopterans. Thus, toxic nectar could be beneficial by deterring visitors that deliver less intraspecific pollen. This concept was further developed by Rhoades and Bergdahl (1981), who suggested that toxic nectar is analogous to other floral structures that require specialization of pollinators. Just as closed corollas or inaccessible placement of nectar may deter generalist pollinators and/or encourage spe-

Table 1. Reports of nectar that is toxic or deterrent to floral visitors or humans. The compounds responsible for the deterrent or toxic effects of nectar were not identified.

Species	Family	Effects	Reference
Aesculus californica	Hippocastanaceae	toxic to bees	Eckert 1946, Mussen 1979
Astragalus spp.	Fabaceae		
Cuscuta spp.	Convolvulaceae		
Cyrilla racemiflora	Cyrillaceae		
Gelsemium sempervirens	Loganiaceae		
Kalmia latifolia	Ericaceae		
Solanum nigrum	Solanaceae		
Veratrum californicum	Liliaceae		
Zygadenus venesosus	Liliaceae		
Corynocarpus laevigata	Corynocarpaceae	toxic to honeybees	Palmer-Jones and Line 1962
Agauria spp.	Ericaceae	honey toxic to humans	Jaeger 1961
Andromeda spp.	Ericaceae		
Kalmia spp.	Ericaceae		
Rhododendron flavum	Ericaceae		
Rhododendron ponticum	Ericaceae		
Paullinia australis	Sapindaceae		
Angelica triqueta	Apiaceae	toxic to bees	Bell 1971
Astragalus lentiginosus	Fabaceae	toxic to bees	Vansell and Watkins 1934
Azalea pontica	Ericaceae	honey toxic to humans	Kebler 1896
Kalmia latifolia	Ericaceae	honey toxic to humans	
Camellia thea	Theaceae	lethal to honeybee larvae	Sharma et al. 1986
Erythrina fusca	Fabaceae	deterred ants	Feinsinger and Swarm 1978
Hippobroma longiflora	Campanulaceae		
Euphorbia spp.	Euphorbiaceae	honey bitter to humans; induced nausea	Pryce-Jones 1942
Ochroma lagopus	Bombacaceae	toxic to bees and other insects	Paula et al. 1997
Sophora microphylla	Fabaceae	toxic to honeybees	Clinch et al. 1972
Tilia spp.	Tiliaceae	toxic to bees and other insects	Crane 1977
Veratrum californicum	Liliaceae	toxic to bees	Vansell and Watkins 1933

Table 2. Reports of nectar containing secondary compounds.

Species	Family	Secondary compound	Reference
Aesculus hippocastaneum	Hippocastanaceae	saponins	Schulz-Langner 1966
Atropa belladonna	Solanaceae	alkaloids	Detzel and Wink 1993
Brugmansia aurea	Solanaceae		
Nicotiana tabacum	Solanaceae		
Campanula rapunculoides	Campanulaceae	alkaloids	Baker and Baker 1975
Cucurbita pepo	Cucurbitaceae		
Cuscuta salina	Convolvulaceae		
Iris pseudocorus	Iridaceae		
Lotus corniculatus	Fabaceae		
Mimulus moschatus	Scrophulariaceae		
Nymphoides peltatum	Gentianaceae		
Rhododendron ponticum	Ericaceae		
Echium plantagineum	Boraginaceae	pyrrolizidine alkaloids	Culvenor et al. 1981
Lathraea clandestina	Scrophulariaceae	ammonia; high pH	Prys-Jones and Willmer 1992
Liriodendron tulipiferum	Magnoliaceae	nonprotein amino acids	Baker and Baker 1975
36 of 66 species	various	nonprotein amino acids	Baker 1977
50 of 567 species	various	alkaloids	
191 of 528 species	various	phenolics	
86 of 248 floral nectars	various	nonprotein amino acids	Baker et al. 1978

cialists, toxic nectar may be a mechanism to increase pollinator fidelity. This hypothesis assumes both that specialists are more effective pollinators than generalists, and that specialists would be less deterred by toxic nectar than generalists.

The few studies testing this hypothesis do not provide clear support. The generalist butterfly pollinator *Agraulis vanillae* was deterred by the pyrrolizidine alkaloid monocrotaline in artificial nectar (Masters 1991), supporting the idea that unspecialized pollinators may be deterred by toxic nectar. However, a separate study repeated Masters' work and found no effect of monocrotaline on this pollinator (Landolt and Lenczewski 1993). It has been suggested that specialized pollinators are not deterred by pyrrolizidine alkaloids in nectar (Masters 1991), but this has not yet been experimentally demonstrated. A convincing test of this hypothesis would require demonstration not only that toxic nectar deters generalist pollinators more than specialists, but also that generalist pollinators are not as effective as specialists in transferring pollen. Pollinators vary widely in their ability to transfer pollen, and the most common visitors are not always the most effective pollinators (Schemske and Horvitz 1984). No study of the effects of toxic nectar on floral visitors has addressed this point.

The nectar robber hypothesis

Janzen (1977) and Baker (1978) proposed that toxic nectar might deter nectar robbery in the tropics. This idea can be thought of as an extension of the pollinator fidelity hypothesis, in that both hypotheses propose that the function of toxic nectar is to deter undesirable visitors and thus serve as a form of defense. In situations where nectar robbers decrease male and female plant fitness (Roubik 1993, Irwin and Brody 1998), protection against robbers could confer a selective advantage to plants. However, in many cases nectar robbery does not adversely affect plant fitness (Zimmerman and Cook 1985, Arizmendi et al. 1996, Morris 1996); this assumption should be tested before we can confidently ascribe the benefits of toxic nectar to deterring nectar robbers.

Janzen (1977) spurred some of the first broad searches for toxic nectar by suggesting that it deterred nectar robbery by ants. The results of these searches were mixed. In general, once nectar was removed from flowers it was palatable to ants, suggesting that mechanical rather than chemical barriers usually protect nectar (Feinsinger and Swarm 1978, Schubart and Anderson 1978, Guerrant and Fiedler 1981). In some species, nectar was repellent to ants (2 of 4 plant species, Feinsinger and Swarm 1978; 1 of 26 plant species, Guerrant and Fiedler 1981), indicating that toxic nectar may occasionally deter ant robbery. However, Haber et al. (1981) found that most floral nectars, including some that contained alkaloids and phenolics, were readily accepted by ants, indicating that even when nectar contains secondary compounds it may not serve as an effective barrier to nectar robbing. Baker (1978) also pointed out that in their surveys of nectar composition, nonprotein amino acids were more

Table 3. Studies where repellent or toxic properties of nectar are examined and compounds responsible for these properties are identified through correlative or experimental studies.

Species	Family	Cause of toxicity	Effects	Reference
Allium cepa	Liliaceae	high potassium	deterred honeybees	Waller et al. 1972
Aloe littoralis	Liliaceae	phenolics	deterred honeybees	Hagler and Buchmann 1993
Prunus dulcis	Rosaceae	phenolics	dilute honey deterred honeybees	
Tamarix pentrandra	Tamaricaceae	phenolics	deterred honeybees	
Anacardium excelsum	Anacardiaceae	alkaloids, phenolics	did not deter ants	Haber et al. 1981
Byrsonima crassifolia	Malpighiaceae	alkaloids, phenolics	deterred ants	
Crescentia alata	Bignoniaceae	phenolics	did not deter ants	
Hymenaea courbaril	Caesalpineaceae	alkaloids, phenolics	deterred ants	
Tabebuia rosea	Bignoniaceae	phenolics	did not deter ants	
Arbutus unedo	Ericaceae	arbutin (glycoside)	honey bitter to humans	Pryce-Jones 1942
Asclepias spp.	Apocynaceae	galitoxin (?)	toxic to bees	
Astragalus miser v. *serotinus*	Fabaceae	miserotoxin, a nitropropanol glycoside	toxic to honeybees	Majak et al. 1980
Catalpa speciosa	Bignoniaceae	iridoid glycosides	deterred ants and a butterfly	Stephenson 1981, 1982
Calathea lutea	Marantaceae	nonprotein amino acids	did not deter ants	Guerrant and Fiedler 1981
Centrosema plumieri	Fabaceae	nonprotein amino acids	did not deter ants	
Crinum erubescens	Amaryllidaceae	nonprotein amino acids	deterred ants	
Erythrina fusca	Fabaceae	nonprotein amino acids, alkaloids and phenolics	did not deter ants	
Gliricidia sepium	Fabaceae	nonprotein amino acids	did not deter ants	
Hamelia patens	Rubiaceae	nonprotein amino acids and alkaloids	did not deter ants	
Heliconia pognantha	Heliconiaceae	nonprotein amino acids, alkaloids	did not deter ants	
Heliconia wagneriana	Heliconiaceae	nonprotein amino acids	did not deter ants	
Hibiscus tiliaceus	Malvaceae	alkaloids	did not deter ants	
Inga oerstediana	Fabaceae	nonprotein amino acids, alkaloids	did not deter ants	
Jacaratia costaricensis	Caricaceae	nonprotein amino acids	did not deter ants	
Justicia aurea	Acanthaceae	nonprotein amino acids	did not deter ants	
Passiflora vitifolia	Passifloraceae	nonprotein amino acids	did not deter ants	
Posoqueria latifolia	Rubiaceae	trace alkaloids	did not deter ants	
Stachytarpheta jamaicensis	Verbenaceae	trace phenolics	did not deter ants	
Tournefortia hirsutissima	Boraginaceae	phenolics, alklaloids	did not deter ants	
Witheringia riparia	Solanaceae	nonprotein amino acids	did not deter ants	
Ledum palustre	Ericaceae	glycoside	honey toxic to humans	Kozlova 1957
Rhododendron spp. and hybrids	Ericaceae	acetylandromedol	toxic to bees	Carey et al. 1959
Senecio jacobaea	Asteraceae	pyrrolizidine alkaloids	honey bitter to humans	Deinzer et al. 1977
Tabebuia rosea	Bignoniaceae	phenolics	correlated with decreased visits by anthophorid bees	Frankie et al. 1982
Tilia spp.	Tiliaceae	mannose	toxic to honeybees	Crane 1977
Honeys, source unknown		tropane alkaloids	honey toxic to humans	Barragan de Dominguez 1973

common in extrafloral than floral nectaries, and extrafloral nectaries were more commonly visited by ants. Thus toxic nectar is apparently not a broad deterrent of ants.

Some of the most detailed studies supporting the nectar robber hypothesis involved *Catalpa speciosa*, a tree with large, tubular, unobstructed flowers. Despite the sugar-rich accessible nectar, nectar robbers visited these flowers very infrequently (Stephenson 1981), possibly because of iridoid glycosides present in nectar. Ants and skippers were identified as potential nectar robbers and were offered *C. speciosa* nectar, a sucrose solution of the same concentration, or a sucrose solution with added iridoid glycosides. Both species preferred the pure sucrose solution to nectar or sucrose with iridoid glycosides; furthermore, those who drank nectar subsequently showed signs of disorientation or narcosis (Stephenson 1981, 1982). Legitimate bee pollinators were not affected by nectar and did not show a preference for sucrose solution over nectar. From these studies, it was concluded that the iridoid glycosides of *C. speciosa* nectar protect flowers from robbers but do not deter legitimate pollinators.

The drunken pollinator hypothesis

In the orchids *Epipactis purpurata* and *E. helleborine*, toxic nectar is due not to secondary compounds but to the presence of ethanol. Ethanol is not produced by the plant itself, but rather by microorganisms that infect nectar either from the air or by transfer from wasp pollinators (Ehlers and Olesen 1997). Upon drinking the nectar, wasps became "sluggish" and were apparently intoxicated. One effect of this intoxication was that wasps groomed their bodies less frequently for pollinia. The authors hypothesize that this change in behavior may improve pollen transfer between plants, because fewer pollinia are removed by wasps in the course of grooming before being transferred to other plants. Toxic nectar may thus be beneficial to plants with pollinia or large pollen grains, where pollen loads hamper the flight of pollinators.

Because the toxins in *Epipactis* orchids are not produced by the plants, they may be less likely to respond to natural selection for toxic nectar. However, the concept may apply to other plant species, which produce their own toxins in nectar. For example, bumblebees became "drunken" after visiting *Asclepias* flowers, which also produce pollinia (Kevan et al. 1988). There have been some other reports of narcosis and disorientation in bees after drinking toxic nectar (Bell 1971, Clinch et al. 1972), but since these episodes may end in death, they do not always mean that pollen transfer will be improved. "Drunken" pollinators also might not be as effective in locating receptive flowers to deposit pollinia. Thus, altering pollinator behavior might account for the function of toxic nectar in some but not all systems.

The antimicrobial hypothesis

As a rich source of sugars and nutrients, nectar could be susceptible to degradation by microbes. Hagler and Buchmann (1993) suggest that phenolics in nectar could be antimicrobial. Many plants, although not the majority, do contain phenolics in their nectar (Baker 1978, Guerrant and Fiedler 1981). In a survey of nectar composition across a wide geographic range, the percentage of plant species with phenolic constituents in nectar decreased with increasing latitude (Baker 1978); this was also true of alkaloids, which have antimicrobial effects (Verpoorte and Schripsema 1994). Even if microbial diversity or virulence decreases with increasing latitude, a correlation between latitude and nectar toxicity is not sufficient evidence to demonstrate that toxic nectar is beneficial due to antimicrobial properties. Currently, published data to evaluate this hypothesis do not seem to be available.

The antimicrobial hypothesis does not relate toxic nectar directly to specific floral visitors, and deserves particular attention because it provides a more general and therefore plausible explanation for the initial evolution of toxic nectar. Imagine a new mutation that causes a novel secondary compound to be present in nectar. Such a compound would initially be unlikely to deter detrimental floral visitors, such as nectar robbers or generalist pollinators, without also affecting specialized pollinators. A more plausible possibility is that pollinators would eventually specialize on toxic nectar that arose and persisted initially for other reasons. Antimicrobial properties would provide an immediate benefit for secondary compounds in nectar that could outweigh potential costs if toxic nectar deterred legitimate pollinators.

Pleiotropy hypothesis (consequence-of-defense)

All of the previously described hypotheses assume that toxic nectar is in some way adaptive, i.e., that possessing toxic nectar confers a fitness advantage. Currently no studies demonstrate that such a fitness advantage exists. Although studies in one system have shown that toxic nectar deterred potential nectar thieves and did not affect legitimate pollinators (Stephenson 1981, 1982), the connection between this and increased plant fitness assumes that nectar robbing is costly and production of toxic nectar is less costly. Costs of toxic nectar could be production costs, in terms of energy expended or limiting resources used in producing toxins in nectar (Coley et al. 1985, Bazzaz et al. 1987), costs of autotoxicity if toxic compounds are damaging to plant tissues (McKey 1974, Chew and Rodman 1979, Fowden and Lea 1979), or ecological costs if toxic nectar has detrimental effects on mutualists such as potential legitimate pollinators (Simms 1992, Strauss et al. 1999).

Determining whether toxic nectar is adaptive awaits an evaluation of its relative benefits and costs and a demonstration that its possession confers a net fitness advantage.

It is therefore also important to consider the hypothesis that toxic nectar does not provide any fitness advantage, but is rather a consequence of pleiotropic constraints or evolutionary history. In other words, toxic nectar itself may not be a trait that is maintained by selection, but rather a trait that was selected for in prior evolutionary time, or a trait that is a consequence of other traits that are currently under selection. In this case, selection on toxic nectar may be neutral or even negative, but has not been strong enough to eliminate the trait. In particular, toxic nectar may persist in plants as a pleiotropic effect of other traits that are beneficial to the plant. As with the adaptive hypotheses described, little information has been collected to evaluate this hypothesis. Here I describe one possible scenario under which toxic nectar may arise as a pleiotropic consequence of other plant traits that are under selection.

Herbivores as well as pollinators impose selection on plant traits. Although secondary compounds may serve many functions in plants, they have been most consistently associated with resistance to herbivores (Rosenthal and Berenbaum 1991). Toxic compounds in nectar may be the consequence of producing secondary compounds that are transported by phloem and are therefore accessible to nectaries. A related possibility is that toxic nectar is present in plants that contain high levels of secondary compounds as defense in other floral structures such as buds, flowers or ovules. In both cases the underlying hypothesis is that toxic nectar is correlated with, and a consequence of, resistance to herbivory in other plant parts.

The hypothesis that toxic nectar arises as a consequence of defense against herbivores is not mutually exclusive from other hypotheses. Toxic nectar could arise as a consequence of defense against herbivores, and subsequently be selected for if pollinators evolve to specialize on this nectar, or if antimicrobial properties make compounds beneficial regardless of herbivore resistance. Thus, toxic nectar may have arisen as a pleiotropic consequence of herbivore defense but persist due to the evolution of specialist pollinators or other fitness benefits.

There are instances when toxic nectar could not be due to pleiotropic effects of herbivore defense. In some cases the cause of toxic nectar is unrelated to herbivore defense. For example, the nectar of *Lathraea clandestina* is toxic due the presence of ammonia, which is produced in the nectar, possibly by enzymatic degradation of amino acids (Prys-Jones and Willmer 1992). The nectar of onion plants is repellent to honey bees to due to consistently high potassium levels (Waller et al. 1972). The nectar of two *Epipactis* orchids is toxic due to ethanol produced by microorganisms living in nectar (Ehlers and Olesen 1997). In these situations, the cause of toxic nectar appears to be unrelated to production of secondary compounds for herbivore resistance.

Toxic fruit

The existence of toxic ripe fruit poses questions that are analogous to those of toxic nectar: is the presence of secondary compounds in ripe fruit an adaptive trait or a pleiotropic consequence of producing these compounds for defense? Ripe fruit, like nectar, is full of sugars and nutrients whose presumed primary function is the attraction of mutualists such as seed dispersers. Generally, toxins in unripe fruit degrade or are translocated out of the fruit as it ripens, but in some cases ripe fruit still contains high concentrations of secondary compounds (reviewed in Herrera 1982, Ehrlén and Eriksson 1993, Cipollini and Levey 1997). Arguments for both adaptive (Cipollini and Levey 1997, 1998) and nonadaptive (Ehrlén and Eriksson 1993, Eriksson and Ehrlén 1998) explanations for toxic fruit have been put forward, but all authors agree that not enough data are available to evaluate any hypothesis.

Many of the ideas in the debate concerning the adaptive nature of toxic fruit are equally relevant to the issue of toxic nectar. Cipollini and Levey (1997) suggest seven adaptive hypotheses to account for the presence of secondary compounds in ripe fleshy fruit. Although some of these are specific to seed dispersal, several could also be applied to toxic nectar. The 'directed toxicity hypothesis' posits that specific secondary compounds are directed towards seed predators but do not affect beneficial dispersers; this is analogous to the 'pollinator fidelity' and 'nectar robber' hypotheses reviewed here. The 'defense trade-off' hypothesis suggests that secondary compounds are present in toxic fruit to prevent microbial degradation; this idea has also been suggested for toxic nectar (Hagler and Buchmann 1993). The 'attraction/association' hypothesis posits that secondary metabolites provide foraging cues to frugivores. While this concept has not been proposed for toxic nectar, it applies equally well to pollinators visiting flowers. Similarly, the 'attraction/repulsion' hypothesis states that secondary compounds might induce frugivores to leave early during foraging and so disperse seeds further (Sorensen 1983). Because pollinators that remain at the same plant may increase geitonogamy, or the transfer of self-pollen between flowers, encouraging pollinators to leave quickly may be beneficial by increasing outcrossing (De Jong et al. 1993, Klinkhamer and De Jong 1993, Harder and Barrett 1995).

Eriksson and Ehrlén (1998) question whether adaptive hypotheses are necessary to explain toxic ripe fruit.

In a literature review, they examined five adaptive hypotheses concerning toxic fruit and found no strong support for any of them (Ehrlén and Eriksson 1993). Rather, toxic fruit occurred in plant species whose tissues are generally toxic, suggesting that toxic fruit may be a pleiotropic consequence of anti-herbivore mechanisms. These authors suggest that the presence of specific secondary compounds in ripe fruit and not in other tissues would be definitive evidence for an adaptive role of toxic fruit (Eriksson and Ehrlén 1998). Although one study has presented such data (Perera et al. 1984), there are not enough detailed chemical studies of multiple plant tissues to come to any conclusions. Similarly, a detailed chemical analysis of nectar and floral tissue would allow a comparison to determine whether certain compounds occur only in nectar. If this were found, it would provide strong evidence for an adaptive function of secondary compounds in nectar.

Future directions

Much work remains in order to clarify the role of toxic nectar in plant ecology and evolution. Some major avenues of research are suggested below; these are intended only as jumping-off points for future studies.

Phylogenetic patterns and physiological mechanisms

The extent to which nectar composition mirrors phloem composition is currently not known. Studies that measure both nectar and phloem composition of the same species are rare. Differences in nectar vs phloem composition may be due to selective secretion of compounds into nectar, or selective resorption from nectar into nectary tissue (Lüttge 1977, Cruden et al. 1983, Durkee 1983). The composition of alkaloids in nectar and pollen can be different from that of leaves and flowers, suggesting either that certain alkaloids are not transported by the phloem, or that nectaries are secreting certain alkaloids and/or excluding others (Detzel and Wink 1993).

Nectar chemical composition clearly evolves, although the direction of this evolution is disputed. Lüttge (1977) claims that more primitive nectaries, such as those in ferns, secrete nectar with a lower sugar: amino acid ratio, while Baker and Baker (1975) argue that there is a trend towards evolution of higher amino acid concentration in nectar in response to the dietary needs of specialized pollinators. A phylogenetic approach to nectar composition could address the question of whether toxic nectar is a derived or ancestral state, which in turn could shed light on how secondary compounds are transported through nectaries. When a new secondary compound arises as a result of mutation and is transported in the vascular system, the plant may initially lack the ability to sequester the compound effectively, and it may diffuse into nectar. If this were the case, we would expect to see toxic nectar arising simultaneously with the evolution of new secondary compounds, and the lack of toxic nectar would be a derived trait that arises with the ability to sequester the new compound. Alternatively, specific enzymes may be necessary in nectaries to transport compounds into nectar, in which case we would expect to see toxic nectar as a derived trait that arises after the evolution of new secondary compounds. Phylogenetic studies are needed to determine whether toxic nectar occurs concurrently with the evolution of novel secondary compounds or arises subsequently.

Is toxic nectar adaptive?

The most direct way to test whether toxic nectar benefits plants would be to experimentally manipulate this trait by altering the chemical composition of nectar. This could be achieved either by removing nectar from flowers and replacing it with sucrose solutions with or without appropriate secondary compounds, or by adding secondary compounds to existing floral nectar. One could then compare pollination and seed set, preferably in the field with whole plants, and observe whether other ecological effects of toxic nectar, such as deterrence of nectar robbers or microbial degradation, occurred.

Currently, no study has documented within-species variation in nectar toxicity, or that such variation is heritable. This may be difficult, considering the small amounts of nectar available from most species, and that nectar concentration tends to vary widely depending on temperature, humidity, and precipitation. Experiments are needed to determine if nectar toxicity is variable within species and whether this variation affects plant fitness. If this were found, the final step would be to determine how much of this variation is heritable. In order to assert that toxic nectar evolves in response to selection by any agent, it is necessary to demonstrate that this traits exhibits heritable variation. Only after all these points have been investigated can we assert that toxic nectar may have evolved in response to selective pressures exerted by ecological interactions.

Conclusion

Toxic nectar is a widespread but poorly understood phenomenon. Although hypotheses regarding its adaptive function abound, no study of toxic nectar has established that this trait benefits the plant. Clearly, answering this question is of central importance in evaluating the hypotheses reviewed above. If a benefit

of toxic nectar is found, then more specific studies can address whether this is due to decreased nectar robbing, specialist pollinators, antimicrobial properties, altered pollinator behavior, or other reasons. If studies reveal that toxic nectar is not beneficial, then this would be strong evidence that toxic nectar is the result of previous evolutionary forces no longer acting on the plant, or a pleiotropic consequence of other traits such as resistance to herbivores. Integrating our understanding of multispecies interactions, such as plant-pollinator and plant-herbivore interactions, may shed light on traits, such as toxic nectar, that previously seemed anomalous.

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References

Adler, L. S., Karban, R. and Strauss, S. Y. in press. Direct and indirect effects of alkaloids on plant fitness via herbivory and pollination. – Ecology.

Arizmendi, M. C., Dominguez, C. A. and Dirzo, R. 1996. The role of an avian nectar robber and of hummingbird pollinators in the reproduction of two plant species. – Funct. Ecol. 10: 119–127.

Armbruster, W. S. 1997. Exaptations link evolution of plant-herbivore and plant-pollinator interactions: a phylogenetic inquiry. – Ecology 78: 1661–1672.

Armbruster, W. S., Howard, J. J., Clausen, T. P. et al. 1997. Do biochemical exaptations link evolution of plant defense and pollination systems? Historical hypotheses and experimental tests with *Dalechampia* vines. – Am. Nat. 149: 461–484.

Arumugasamy, K., Udalyan, K. and Manlan, S. 1993. Ultrastructure of cyathial nectaries of *Euphorbia heterophylla* and its relation to the mechanism of nectar secretion. – Indian Botanical Contactor 10: 19–27.

Baker, H. G. 1977. Non-sugar chemical constituents of nectar. – Apidologie 8: 349–356.

Baker, H. G. 1978. Chemical aspects of the pollination biology of woody plants in the tropics. – In: Tomlinson, P. B. and Zimmerman, M. H. (eds), Tropical trees as living systems. Cambridge Univ. Press, pp. 57–82.

Baker, H. G. and Baker, I. 1975. Studies of nectar-constitution and pollinator-plant coevolution. – In: Gilbert, L. E. and Raven, P. H. (eds), Coevolution of plants and animals. Univ. of Texas Press, pp. 100–140.

Baker, H. G. and Baker, I. 1982. Chemical constituents of nectar in relation to pollination mechanisms and phylogeny. – In: Nitecki, M. H. (ed.), Biochemical aspects of evolutionary biology. Univ. of Chicago Press, pp. 131–171.

Baker, H. G., Opler, P. A. and Baker, I. 1978. A comparison of the amino acid complements of floral and extrafloral nectars. – Bot. Gaz. 139: 322–332.

Barragan de Dominguez, M. C. 1973. Contribucion al estudio de mieles toxicas colombianas. – Revista Colombiana de Ciencias Quimico-Farmaceuticas 2: 5–31.

Bazzaz, F. A., Chiarello, N. R., Coley, P. D. and Pitelka, L. F. 1987. Allocating resources to reproduction and defense. – BioScience 37: 58–67.

Bell, C. R. 1971. Breeding systems and floral biology of the Umbelliferae, or evidence for specialization in unspecialized flowers. – In: Heywood, V. H. (ed.), The biology and chemistry of the Umbelliferae. Academic Press, pp. 93–107.

Berenbaum, M. R., Zangerl, A. R. and Nitao, J. K. 1986. Constraints on chemical evolution: wild parsnips and the parsnip webworm. – Evolution 40: 1215–1228.

Callaway, R. M., DeLuca, T. H. and Belliveau, W. M. 1999. Biological-control herbivores may increase competitive ability of the noxious weed *Centaurea maculosa*. – Ecology 80: 1196–1201.

Carey, D. B. and Wink, M. 1994. Elevational variation of quinolizidine alkaloid contents in a lupine (*Lupinus argenteus*) of the Rocky Mountains. – J. Chem. Ecol. 20: 849–857.

Carey, F. M., Lewis, J. J., MacGregor, J. L. and Martin-Smith, M. 1959. Pharmacological and chemical observations on some toxic nectars. – J. Pharm. Pharmacol. 11 Suppl: 269T–274T.

Chew, F. S. and Rodman, J. E. 1979. Plant resources for chemical defense. – In: Rosenthal, G. A. and Janzen, D. H. (eds), Herbivores: their interactions with secondary plant metabolites. Academic Press, pp. 271–308.

Cipollini, M. L. and Levey, D. J. 1997. Secondary metabolites of fleshy vertebrate-dispersed fruits: adaptive hypotheses and implications for seed dispersal. – Am. Nat. 150: 346–372.

Cipollini, M. L. and Levey, D. J. 1998. Secondary metabolites as traits of ripe fleshy fruits: a response to Eriksson and Ehrlén. – Am. Nat. 152: 908–911.

Clinch, P. G., Palmer-Jones, T. and Forster, I. W. 1972. Effect on honey bees of nectar from the yellow kowhai (*Sophora microphylla* Ait.). – N.Z. J. Agric. Res. 15: 194–201.

Coley, P. D., Bryant, J. P. and Chapin III, F. S. 1985. Resource availability and plant antiherbivore defense. – Science 230: 895–899.

Crane, E. 1977. Dead bees under lime trees. – Bee World 58: 129–130.

Cruden, R. W., Hermann, S. M. and Peterson, S. 1983. Patterns of nectar production and plant-pollinator coevolution. – In: Bentley, B. and Elias, T. (eds), The biology of nectaries. Columbia Univ. Press, pp. 80–125.

Culvenor, C. C. J., Edgar, J. A. and Smith, L. W. 1981. Pyrrolizidine alkaloids in honey from *Echium plantagineum* L. – J. Agric. Food Chem. 29: 958–960.

Davis, A. R., Pylatuik, J. D., Paradis, J. C. and Low, N. H. 1998. Nectar-carbohydrate production and composition vary in relation to nectary anatomy and location within individual flowers of several species of Brassicaceae. – Planta 205: 305–318.

Deinzer, M. L., Thomson, P. A., Burgett, D. M. and Isaacson, D. L. 1977. Pyrrolizidine alkaloids: their occurrence in honey from tansy ragwort (*Senecio jacobea* L.). – Science 195: 497–499.

De Jong, T. J., Waser, N. M. and Klinkhamer, P. G. L. 1993. Geitonogamy: the neglected side of selfing. – Trends Ecol. Evol. 8: 321–325.

Detzel, A. and Wink, M. 1993. Attraction, deterrence or intoxication of bees (*Apis mellifera*) by plant allelochemicals. – Chemoecology 4: 8–18.

Duffey, S. S. and Stout, M. J. 1996. Antinutritive and toxic components of plant defense against insects. – Arch. Insect Biochem. Physiol. 32: 3–37.

Durkee, L. T. 1983. The ultrastructure of floral and extrafloral nectaries. – In: Bentley, B. and Elias, T. (eds), The biology of nectaries. Columbia Univ. Press, pp. 1–29.

Eckert, J. E. 1946. Injury to bees by poisoning. – In: Grout, R. A. (ed.), The hive and the honey bee. Dadant and Sons, pp. 570–582.

Ehlers, B. K. and Olesen, J. M. 1997. The fruit-wasp route to toxic nectar in *Epipactis* orchids? – Flora 192: 223–229.
Ehrlén, J. and Eriksson, O. 1993. Toxicity in fleshy fruits: a non-adaptive trait? – Oikos 66: 107–113.
Eriksson, O. and Ehrlén, J. 1998. Secondary metabolites in fleshy fruits: are adaptive explanations needed? – Am. Nat. 152: 905–907.
Fægri, K. and van der Pijl, L. 1979. The principles of pollination ecology, 3rd ed. – Pergamon Press.
Fahn, A. 1988. Secretory tissues in vascular plants. – New Phytol. 108: 229–258.
Feinsinger, P. and Swarm, L. A. 1978. How common are ant-repellant nectars? – Biotropica 10: 238–239.
Findlay, N. and Mercer, F. V. 1971. Nectar production in *Abutilon* II. Submicroscopic structure of the nectary. – Aust. J. Biol. Sci. 24: 657–664.
Fowden, L. and Lea, P. J. 1979. Mechanism of plant avoidance of autotoxicity by secondary metabolites, especially by nonprotein amino acids. – In: Rosenthal, G. A. and Janzen, D. H. (eds), Herbivores: their interactions with secondary plant metabolites. Academic Press, pp. 135–160.
Frankie, G. W., Haber, W. A., Baker, I. and Baker, H. G. 1982. A possible chemical explanation for differential flower foraging by anthophorid bees among individuals of *Tabebuia rosea* in a neotropical dry forest. – Brenesia 20: 397–402.
Frei, E. 1955. Die Innervierung der floralen Nektarien dikotyler Pflanzenfamilien. – Ber. Schweiz. Bot. Ges. 65: 60–114.
Frey-Wyssling, A. and Agthe, C. 1950. Nektar ist ausgeschiedener Phloemsaft. – Verh. Schweiz. Naturforsch. Ges. 130: 175–176.
Genc, Z. 1996. Investigation of some *Euphorbia* L. species nectaries by electron microscope. – Turkish J. Bot. 20: 11–19.
Gowan, E., Lewis, B. A. and Turgeon, R. 1995. Phloem transport of antirrhinoside, an iridoid glycoside, in *Asarina scandens* (Scrophulariaceae). – J. Chem. Ecol. 21: 1781–1788.
Guerrant, E. O., Jr. and Fiedler, P. L. 1981. Flower defenses against nectar-pilferage by ants. – Biotropica 13 (Suppl): 25–33.
Gunning, B. E. S. and Hughes, J. E. 1976. Quantitative assessment of symplastic transport of pre-nectar ino the trichomes of *Abutilon* nectaries. – Aust. J. Plant Physiol. 3: 619–637.
Haber, W. A., Frankie, G. W., Baker, H. G. et al. 1981. Ants like flower nectar. – Biotropica 13: 211–214.
Hagler, J. R. and Buchmann, S. L. 1993. Honey bee (Hymenoptera: Apidae) foraging responses to phenolic-rich nectars. – J. Kansas Entomol. Soc. 66: 223–230.
Harder, L. D. and Barrett, S. C. H. 1995. Mating cost of large floral displays in hermaphrodite plants. – Nature 373: 512–515.
Herrera, C. M. 1982. Defense of ripe fruit from pests: its significance in relation to plant-disperser interactions. – Am. Nat. 120: 218–247.
Irwin, R. and Brody, A. K. 1998. Nectar robbing in *Ipomopsis aggregata*: effects on pollinator behavior and plant fitness. – Oecologia 116: 519–527.
Jaeger, P. 1961. The wonderful life of flowers. – E. P. Dutton & Co.
Janzen, D. H. 1977. Why don't ants visit flowers? – Biotropica 9: 1252.
Juenger, T. and Bergelson, J. 1997. Pollen and resource limitation of compensation to herbivory in scarlet gilia, *Ipomopsis aggregata*. – Ecology 78: 1684–1695.
Karban, R. 1993. Costs and benefits of induced resistance and plant density for a native shrub, *Gossypium thurberi*. – Ecology 74: 9–19.
Kebler, L. F. 1896. Poisonous honey. – Am. Pharmaceut. Assoc. Proc. 44: 167–174.
Kevan, P. G., Eisikowitch, D., Fowle, S. and Thomas, K. 1988. Yeast-contaminated nectar and its effects on bee foraging. – J. Apicult. Res. 27: 26–29.
Klinkhamer, P. G. L. and De Jong, T. J. 1993. Attractiveness to pollinators: a plant's dilemma. – Oikos 66: 180–184.
Kozlova, M. V. 1957. Nectar of *Ledum palustre* as a possible source of the toxicity of honey. – Voprosy Pitaniya 16: 1980.
Krupnick, G. A., Weis, A. E. and Campbell, D. R. 1999. The consequences of floral herbivory for pollinator service to *Isomeris arborea*. – Ecology 80: 125–134.
Landolt, P. J. and Lenczewski, B. 1993. Lack of evidence for the toxic nectar hypothesis: a plant alkaloid did not deter nectar feeding by Lepidoptera. – Fla. Entomol. 76: 556–566.
Lehtilä, K. and Strauss, S. Y. 1997. Leaf damage by herbivores affects attractiveness to pollinators in wild radish, *Raphanus raphanistrum*. – Oecologia 111: 396–403.
Lohman, D. J. and Berenbaum, M. R. 1996. Impact of floral herbivory by parsnip webworm (Oecophoridae: *Depressaria pastinacella* Duponchel) on pollination and fitness of wild parsnip (Apiaceae: *Pastinaca sativa* L.). – Am. Mid. Nat. 136: 407–412.
Lüttge, U. 1977. Nectar composition and membrane transport of sugars and amino acids: a review on the present state of nectar research. – Apidologie 8: 305–319.
Lüttge, U. and Schnepf, E. 1976. Organic substances. – In: Lüttge, U. and Pitman, M. G. (eds), Transport in plants II. B: tissues and organs. Springer-Verlag, pp. 244–277.
Majak, W., Neufeld, R. and Corner, J. 1980. Toxicity of *Astragalus miser* v. serotinus to the honeybee. – J. Apicult. Res. 19: 196–199.
Masters, A. R. 1991. Dual role of pyrrolizidine alkaloids in nectar. – J. Chem. Ecol. 17: 195–206.
McKey, D. 1974. Adaptive patterns in alkaloid physiology. – Am. Nat. 108: 305–320.
Merritt, S. Z. 1996. Within-plant variation in concentrations of amino acids, sugar, and sinigrin in phloem sap of black mustard, *Brassica nigra* (L.) Koch (Cruciferae). – J. Chem. Ecol. 22: 1133–1145.
Molyneux, R. J., Campbell, B. C. and Dreyer, D. L. 1990. Honeydew analysis for detecting phloem transport of plant natural products: implications for host-plant resistance to sap-sucking insects. – J. Chem. Ecol. 16: 1899–1910.
Montllor, C. B. 1989. The influence of plant chemistry on aphid feeding behavior. – In: Bernays, E. (ed.), Insect-plant interactions. CRC Press, pp. 125–173.
Morris, W. F. 1996. Mutualism denied? Nectar-robbing bumble bees do not reduce female or male success of bluebells. – Ecology 77: 1451–1462.
Mothershead, K. and Marquis, R. J. 2000. Fitness impacts of herbivory through indirect effects on plant-pollinator interactions in *Oenothera macrocarpa*. – Ecology 81: 30–40.
Mullin, C. A. 1986. Adaptive divergence of chewing and sucking arthropods to plant allelochemicals. – In: Brattsten, L. B. and Ahmad, S. (eds), Molecular aspects of insect-plant associations. Plenum Press, pp. 175–209.
Mussen, E. C. 1979. Buckeye poisoning. – U.C. Apiaries: Univ. of California Cooperative Extension: 1–4.
Nepi, M., Ciampolini, F. and Pacini, E. 1996. Development and ultrastructure of *Cucurbita pepo* nectaries of male flowers. – Ann. Bot. 78: 95–104.
O'Brien, S. P., Loveys, B. R. and Grant, W. J. R. 1996. Ultrastructure and function of floral nectaries of *Chamelaucium uncinatum* (Myrtaceae). – Ann. Bot. 78: 189–196.
Palmer-Jones, T. and Line, L. J. S. 1962. Poisoning of honey bees by nectar from the karaka tree (*Corynocarpus laevigata* J. R. et G. Forst). – N.Z. J. Agric. Res. 5: 433–436.
Paula, V. F., Barbosa, L. C. A., Demuner, A. J. et al. 1997. Entomotoxicity of the nectar from *Ochroma lagopus* Swartz (Bombacaceae). – Ciencia e Cultura 49: 274–277.
Perera, P., Sandberg, F., Van Beek, T. A. and Verpoorte, R. 1984. Tertiary indole alkaloids from fruits of *Tabernaemontana dichotoma*. – Planta Medica 50: 251–253.

Pryce-Jones, J. 1942. Some problems associated with nectar, pollen and honey. – Proc. Linn. Soc. Lond. 155: 129–174.
Prys-Jones, O. E. and Willmer, P. G. 1992. The biology of alkaline nectar in the purple toothwort (*Lathraea clandestina*): ground level defences. – Biol. J. Linn. Soc. 45: 373–388.
Rhoades, D. F. and Bergdahl, J. C. 1981. Adaptive significance of toxic nectar. – Am. Nat. 117: 798–803.
Rosenthal, G. and Berenbaum, M. (eds) 1991. Herbivores: their interactions with secondary plant metabolites, Vol. I: The chemical participants. – Academic Press.
Roubik, D. W. 1993. Direct costs of forest reproduction, bee-cycling and the efficiency of pollination modes. – J. Biosci. (Bangalore) 18: 537–552.
Rumpf, S., Cromey, M. and Webb, C. J. 1994. Ultrastructure and function of the nectaries of New Zealand bracken (*Pteridium esculentum* (Forst. f.) Cockayne). – N.Z. J. Bot. 32: 487–496.
Schemske, D. W. and Horvitz, C. C. 1984. Variation among floral visitors in pollination ability: a precondition for mutualism specialization. – Science 225: 519–521.
Schemske, D. W. and Horvitz, C. C. 1988. Plant-animal interactions and fruit production in a Neotropical herb: a path analysis. – Ecology 69: 1128–1137.
Schubart, H. O. R. and Anderson, A. B. 1978. Why don't ants visit flowers? A reply to D. H. Janzen. – Biotropica 10: 310–311.
Schulz-Langner, E. 1966. Quantitativer Nachweis kleinster Saponinmengen durch beachten der Hämolysedauer. – Planta Medica 14: 49–56.
Sharma, O. P., Raj, D. and Garg, R. 1986. Toxicity of nectar of tea (*Camellia thea*) to honeybees. – J. Apicult. Res. 25: 106–108.
Simms, E. 1992. Costs of plant resistance to herbivory. – In: Fritz, R. and Simms, E. (eds), Plant resistance to herbivores and pathogens. Univ. of Chicago Press, pp. 392–425.
Simms, E. L. and Bucher, M. A. 1996. Pleiotropic effect of flower color intensity on resistance to herbivory in *Ipomoea purpurea*. – Evolution 50: 957–963.
Sorensen, A. E. 1983. Taste aversion and frugivore preference. – Oecologia 56: 117–120.
Stephenson, A. G. 1981. Toxic nectar deters nectar thieves of *Catalpa speciosa*. – Am. Midl. Nat. 105: 381–383.
Stephenson, A. G. 1982. Iridoid glycosides in the nectar of *Catalpa speciosa* are unpalatable to nectar thieves. – J. Chem. Ecol. 8: 1025–1034.
Strauss, S. Y. 1997. Floral characters link herbivores, pollinators, and plant fitness. – Ecology 78: 1640–1645.
Strauss, S. Y. and Armbruster, W. S. 1997. Linking herbivory and pollination-new perspectives on plant and animal ecology and evolution. – Ecology 78: 1617–1618.
Strauss, S. Y., Siemens, D. H., Decher, M. B. and Mitchell-Olds, T. 1999. Ecological costs of plant resistance to herbivores in the currency of pollination. – Evolution 53: 1105–1113.
Thorp, R. W., Briggs, D. L., Estes, J. R. and Erickson, E. H. 1975. Nectar fluorescence under ultraviolet radiation. – Science 189: 476–478.
Torres, C. and Galetto, L. 1998. Patterns and implications of floral nectar secretion, chemical composition, removal effects and standing crop in *Mandevilla pentlandiana* (Apocynaceae). – Bot. J. Linn. Soc. 127: 207–223.
Treutter, D., Galensa, R., Feucht, W. and Schmid, P. P. S. 1985. Flavanone glucosides in callus and phloem of *Prunus avium*: identification and stimulation of their synthesis. – Physiol. Plant. 65: 95–101.
Vansell, G. H. and Watkins, W. G. 1933. A plant poisonous to adult bees. – J. Econ. Entomol. 26: 168–170.
Vansell, G. H. and Watkins, W. G. 1934. Adult bees found dying on spotted loco. – J. Econ. Entomol. 27: 635–637.
Verpoorte, R. and Schripsema, J. 1994. Isolation, identification, and structure elucidation of alkaloids: a general review. - In: Linskens, H. F. and Jackson, J. F. (eds), Modern methods of plant analysis. Vol. 15. Springer-Verlag, pp. 1–24.
Waller, G. D., Carpenter, E. W. and Ziehl, O. A. 1972. Potassium in onion nectar and its probable effect on attractiveness of onion flowers to honey bees. – Am. Soc. Horticult. Sci. 97: 535–539.
Weberling, F. 1989. Morphology of flowers and inflorescences. – Cambridge Univ. Press.
Wink, M. 1992. The role of quinolizidine alkaloids in plant-insect interactions. – In: Bernays, E. (ed.), Insect-plant interactions. CRC Press, pp. 133–169.
Zer, H. and Fahn, A. 1992. Floral nectaries of *Rosmarinus officinalis* L. Structure, ultrastructure and nectar secretion. – Ann. Bot. 70: 391–397.
Zimmerman, M. and Cook, S. 1985. Pollinator foraging, experimental nectar-robbing and plant fitness in *Impatiens capensis*. – Am. Midl. Nat. 113: 84–91.