Form Movement (Reinhold, New York, 1963), p. 153].
10. Mechanical adaptation might be more complex if the properties of the biomaterial, such as *Young's modulus* (*E*), also scale with load. We cannot exclude this possibility, but even if the material properties are scale-dependent, strain or work-to-break similarity is unlikely given the consistently low correlation of length dimensions with load. Since strain energy or the work required to break an element is proportional to $E(ld^2)$, where l is length and d is diameter, ld^2 of the element should be correlated with the tensile load regardless of the scaling in E. This is not true for the elements we considered (for example, $r = .022$ for *Kigelia* stalk ld^2 and fruit mass).
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Detoxification Enzyme Differences Between a Herbivorous and Predatory Mite

Abstract. *The detoxification capabilities of the predatory mite* Amblyseius fallacis *and its herbivorous prey* Tetranychus urticae *are fundamentally different. The activities of mixed-function oxidase and* trans*-epoxide hydrolase are higher in the prey than in the predator; those of* cis*-epoxide hydrolase and glutathione transferase are lower; and esterase activity is similar. Dissimilarities may be related both to differing adaptations to plant allelochemicals and to the higher respiration rate of the predator. Hydrolytic and conjugating reactions appear more important than oxidative pathways in imparting organophosphate resistance to these acarines. These resistances provide insecticide selectivity favorable to the predator and improved integrated pest control.*

Soon after synthetic insecticides became widely used, arthropod pests developed resistance to them. Indeed, some pests were tolerant to these toxicants from the beginning. Herbivorous arthropod pests are consistently less susceptible to conventional insecticides than predators and parasites are (*1*). Insecticide resistance has been described in more than 400 species of pests compared with only 13 of their natural enemies (*2*). Greater insecticide susceptibility of the natural enemies and their reduced capability for resistance limits the effectiveness of pest control, especially that of integrated pest management, which combines pesticidal and biological controls.

Herbivores may be adapted to detoxify pesticides because they must detoxify plant secondary compounds (allelochemicals) in their diets. Much evidence indicates that allelochemicals are produced by plants to deter herbivores (*3*). In response, herbivores can increase detoxification enzyme activities that allow them to feed without suffering from allelochemicals. Mixed-function oxidases (MFO's), a major detoxification system in response to allelochemicals (*4*), also play a role in insecticide detoxification and resistance (*5*). Predators and parasites, which do not feed directly on plants, probably are less exposed to allelochemicals and thus may have lower MFO activities and be less adapted to detoxify or develop resistance to pesticides (*6*). Although predators and parasites may lack well-developed MFO's, they should be as adapted as their prey to the use of nonoxidative detoxification mechanisms, especially hydrolytic pathways (for example, esterases) (*7*) that also function in the basal metabolism of proteins and lipids.

We examined the detoxification enzymes of a pest and its natural enemy and found major differences (Table 1) that support the allelochemical adaptation hypothesis, yet demonstrate that for certain enzymes a carnivore can have an advantage over a herbivore in potential detoxification capability. A strain of the plant-feeding spider mite *Tetranychus urticae* (Koch) that is resistant to organophosphates and a strain that is susceptible to organophosphates were compared with a multiresistant (to organophosphates, pyrethroids, and DDT) and a susceptible strain of the phytoseiid predatory mite *Amblyseius fallacis* (Garman) (*8*). Study of both susceptible and resistant strains of each species allowed us to compare intrinsic detoxification potentials with enzyme modulation due to pesticide exposure.

The *T. urticae–A. fallacis* complex is a useful herbivore-predator model for comparative toxicology. The prey is polyphagous, feeding on more than 200 hosts. The predator is also polyphagous, but its native and principal host is *T. urticae*. Both mites cohabit in natural and agro-ecosystems. They have similar biologies, morphologies, and other characteristics (*6*). Susceptibilities to pesticides and plant toxins and the potential to develop resistance have been studied extensively in both mites (*9*).

Food-deprived mites were collected on a controlled-pore nylon filter that yielded an equivalent distribution of life stages (*8*). Large numbers of animals (10^3 to 10^5 individuals) were used to minimize age-related and other physiological variations that could decrease the reproducibility of results. Whole body homogenates were centrifuged at 12,000*g* to give a supernatant fraction suitable for a detoxification enzyme survey (*10*). Both phase 1 (oxidative and hydrolytic) and phase 2 (conjugative and epoxide-hydrating) reactions, that typify the metabolism of lipophilic toxicants (for example, pesticides and allelochemicals) to more polar and thus excretable products, were measured (*11*).

Aldrin epoxidase, a MFO, was measured by electron-capture detection of the product epoxide dieldrin (*12*). Epoxidation of pesticidal olefins, such as aldrin, by the action of the monooxygenase mediated by cytochrome P-450 is an index of the oxidative capability of the organism for lipophilic toxicants (*13*). In susceptible strains, the herbivorous prey mite had a fivefold higher MFO activity than the carnivore. This finding supports the hypothesis that a herbivore has a higher detoxification potential than its predator as a result of this important phase 1 enzyme group (*6*).

General hydrolytic capability was compared by the α-naphthyl acetate esterase assay, which measures activities of carboxylesterases, lipases, amidases, proteinases, cholinesterases, and thioesterases (*14*). No significant difference between the susceptible predator and susceptible prey mites was found, supporting the hypothesis (*7*) that arthropod herbivores and carnivores should have similar hydrolytic detoxification pathways because of the multiplicity of hydrolytic enzymes involved in endogenous metabolism.

Lastly, three phase 2 enzymes were surveyed in the susceptible strains: (i) glutathione transferase (*15*), (ii) the epoxide hydrolase selective for *trans*-epoxides and typically cytosolic in mammals (*16*), and (iii) the typically membrane-bound epoxide hydrolase that hydrolyzes *cis*-epoxides (*17*). Together, these enzymes can effectively detoxify lipophilic epoxides by generating excretable

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Table 1. Detoxification enzyme levels in a predatory mite *Amblyseius fallacis* and its prey *Tetranychus urticae*. Activity is expressed in picomoles per minute per milligram of protein for combined microsomal and cytosolic fractions. Values are means ± standard error for three to five separate enzyme preparations.

Mite species strain	Activity (pmole/min-mg)				
	Phase 1 enzymes		Phase 2 enzymes		
	Aldrin epoxidase	Esterase ($\times 10^{-3}$)	Glutathione transferase	Epoxide hydrolase	
				trans	*cis*
A. fallacis					
Susceptible	0.27 ± 0.13	318 ± 39	1095 ± 221†	278 ± 24	431 ± 63*
Resistant	0.23 ± 0.08	1788 ± 197¶	1683 ± 144§	600 ± 32¶	834 ± 134‖
T. urticae					
Susceptible	1.44 ± 0.12†	389 ± 15§	102 ± 11	1587 ± 43‡	117 ± 12
Resistant	1.60 ± 0.18	263 ± 45	219 ± 16‖	1613 ± 103	124 ± 18

Interspecific difference between susceptible strains: *$P < .01$; †$P < .005$; and ‡$P < .001$. Intraspecific difference: §$P < .10$; ‖$P < .05$; and ¶$P < 0.005$. Significance by Student's *t*-test is indicated for the strain with the higher enzyme level.

products, the glutathione conjugates and diols (*18*).

Striking interspecific differences were found. *trans*-Epoxide hydrolase activity was sixfold higher in the susceptible prey than in the predator and, like MFO, probably protects the phytophagous arthropod from allelochemicals (*19*). However, the susceptible predator had 11-fold higher glutathione transferase and fourfold higher *cis*-epoxide hydrolase activities than the prey. Both enzymes can protect mammals (and perhaps mites) from the harmful consequences of autoxidation of essential lipids after elevated inhalation of oxidants (*20*). Indeed, carnivorous mites, because of the energy demands of a predatory mode of life, consume more oxygen than do herbivorous mites (*21*) and, in the absence of protective reactions, would be more susceptible to the toxic effects of hyperventilation.

In examining the relation of resistance to detoxification enzymes (Table 1), no intraspecific differences were observed for aldrin epoxidase in either resistant mite. These data suggest that MFO is not a major resistance mechanism in either mite, and they support a parallel study in vivo of the predator in which synergists were used (*22*).

The largest intraspecific difference was noted for esterases, with resistant *A. fallacis* having a sixfold higher activity than the susceptible strain. This result was surprising since many enzymes contribute to the measurement, and presumably only a few enzymes are selected in resistance development. This difference indicates that hydrolytic detoxification is a major mechanism of resistance in *A. fallacis*. Final assignment of the inherent enzyme activities will require further study, since even some glutathione transferases may hydrolyze α-naphthyl acetate (*23*).

In contrast to the finding for *A. fallacis*, general esterase activity appears to be lower in organophosphate-resistant than in susceptible *T. urticae* (*24*). However, a specific carboxylesterase, obscured in an α-naphthyl acetate assay, contributes to development of organophosphate resistance in *T. urticae* (*25*), and for this strain other detoxification enzymes (for example, glutathione transferase), retarded penetration, or target site insensitivity may also be involved.

Glutathione transferase levels are higher in resistant than in susceptible strains in both mite species. Increased glutathione transferase activity is known to contribute to organophosphate resistance in *A. fallacis* (*26*). In addition, in resistant *A. fallacis*, epoxide hydrolase for both substrates was elevated twofold. Although the implications of this finding are unknown, this strain is also pyrethroid and DDT resistant, and these compounds may be detoxified by an epoxide-to-diol pathway (*27*). Thus elevated epoxide hydrolase may partially explain the multiple resistance of this strain, as seems to be the case for a beetle (*28*).

From studies contrasting susceptible and resistant strains, we conclude that hydrolytic and phase 2 reactions (general esterase, glutathione transferase, and epoxide hydrolase) are more important than oxidative pathways (involving MFO) in adapting both mite species to organophosphates. For *A. fallacis*, the same holds for pyrethroids and DDT. This tendency seems more general for development of insecticide resistance in arthropods (*29*) than would be predicted from considerations of the importance of MFO in the metabolism of pesticides (*5*). Furthermore, the specific mechanism for development of resistance, particularly indicated by general esterase, may differ between the acarine herbivore and carnivore.

Our studies of enzymes inherent in susceptible strains of predatory and prey mites support the hypothesis relating detoxification potentials to feeding ecology (*6, 7*) in that MFO was elevated in the herbivore, whereas hydrolytic esterase levels were similar in the two mites. However, the reasons for differing levels of epoxide hydrolase and glutathione transferase, about which much less is known, are unclear. The discovery of elevated *cis*-epoxide hydrolase and glutathione transferase in the predatory mite points out that (i) a workable knowledge of the detoxification capabilities in a predator-prey system requires looking at a wide range of enzyme possibilities and (ii) predators and prey may have been exposed to differing levels or sets of environmental toxins, or the predator responded in a different manner than the prey to the same toxins.

Before generalities can be drawn from these results, other representative herbivore-predator-parasite systems must be examined. More study of the causes of these intrinsic differences is needed, especially in relation to exposure to environmental toxins, including plant secondary compounds. Fundamental differences in detoxification enzymes occur in this herbivore-predator acarine system; these differences are greater than what would be inferred from previous work on insects or between insect and mammal life systems (*5, 13*). Exploration of these differences might lead to selective acaricide development and the biorational design of integrated pest management systems (*30*).

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Naloxone Antagonism of the Thermoregulatory Effects of Phencyclidine

Abstract. *Phencyclidine elicits hyperthermia at low doses and hypothermia at high doses in rats. Naloxone antagonizes both effects. Phencyclidine's effects on thermoregulation are probably mediated by an interaction with a mu opiate receptor.*

An understanding of the pharmacological properties of phencyclidine (PCP) has become important due to the increased incidence of PCP abuse. Recent studies have focused on characteristics that PCP seems to share with the psychotomimetic opioids. Of special interest have been the observations that in rats and squirrel monkeys trained to discriminate PCP from saline the PCP response can be generalized to *N*-allylnormetazocine and that neither effect is antagonized by naloxone (*1*). *N*-Allylnormetazocine is a benzomorphan opioid that is considered the prototypical agonist of the putative sigma opiate receptor (*2*). Thus PCP may also be a sigma opiate agonist, a suggestion supported by evidence of a shared binding site with sigma opiate ligands in rat brain (*3*). There is also evidence, however, that PCP binds to mu opiate receptors sensitive to morphine and naloxone (*4*). We report here that PCP elicits biphasic changes in body temperature that are readily antagonized by naloxone.

Baseline temperatures of female Sprague-Dawley rats (220 to 250 g) were determined rectally with an electronic digital thermometer (IVAC Corp.) accurate to ± 0.1°C (*5*). In groups of three, each rat then received two intraperitoneal injections: one of PCP hydrochloride (0.625 to 40 mg/kg) and one of saline (1 ml/kg), one of PCP and one of naloxone hydrochloride (1.0 mg/kg), one of naloxone and one of saline, or two of saline. Rectal temperatures were again determined 15, 30, 45, and 60 minutes later. Each animal was used for only one treatment on one occasion. Ambient temperature was maintained at 22°C.

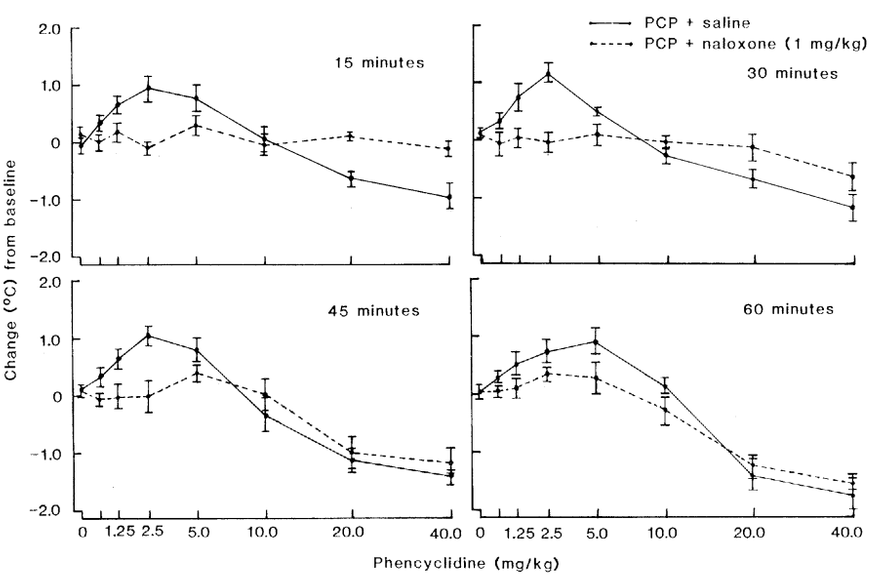

Fig. 1. Time-dependent effects of PCP and PCP plus naloxone on body temperature in rats. Values are means ± standard errors for three experiments.

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