sensitivity is the local concentration of the neurotransmitter (*2, 4*). We have shown recently that a variety of manipulations which modify the sensitivity of the iris sphincter have only one apparent common feature—their effect on the release or destruction of ACh. Furthermore, even in the absence of nerve terminals, ACh is able to depress the sensitivity of the iris sphincter to cholinomimetics (*4*). These and other experiments led to the conclusion that the sensitivity of the target organ is inversely related to the long-term local concentration of the neurotransmitter (*7*). Our results are consistent with this concept, since overstimulation can be expected to lead to a local accumulation of the neurotransmitter, whereas stimulus deprivation can be expected to lower the concentration of ACh at the site of the target organ.

The available evidence suggests that changes in the sensitivity of smooth muscle, as well as other innervated target organs, result from changes in the concentration of cholinergic receptors on the effector cell membrane (*4*). If receptors, like other membrane components, undergo a continuous turnover, changes in concentration result from alterations in either the rate of synthesis or the rate of inactivation of receptor material. Thus, sensitivity of the target organ could be controlled by a negative feedback of ACh (or a product of the interaction of ACh and the receptor) on the rate-limiting step in receptor synthesis. Such a mechanism would be consistent with the observation (*8*) that at least in some systems the synthesis of new protein (or proteins) is required for the achievement of supersensitivity after denervation. The work of Warren and Glick (*9*) indicates, however, that in mammalian cells the rate of synthesis of membrane components is relatively constant, whereas turnover is a variable. Thus, the possibility that changes in cholinergic sensitivity result from changes in the rate of receptor turnover must be considered. A relationship between the frequency of activation of the receptor and the duration of its functional life provides a simple mechanism to account for alterations in receptor turnover rate and concomitant changes in cholinergic sensitivity. Detailed studies on the rates of development of sensitivity changes and on the specificity of these changes should help to distinguish between these possibilities and to elucidate other aspects of maintenance of normal cholinergic sensitivity. The reversible physiological alteration in target organ sensitivity, combined with the in vitro technique of dose-response assays, as used in the present experiments, provides a uniquely suitable system for such studies.

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Butterfly-Plant Coevolution: Has Passiflora adenopoda Won the Selectional Race with Heliconiine Butterflies?

Abstract. *Hooklike trichomes of* Passiflora adenopoda *provide a specific, effectively absolute defense against heliconiine butterfly larvae, a major class of* Passiflora *herbivores. It is suggested that since mechanical defenses are usually more selective in their action against herbivores, they usually are evolved by a plant only after it has accumulated a series of chemical defenses.*

It has long been assumed that many structural features of plant surfaces function to give the plant resistance to the attack of herbivores (*1*). Indeed, the effectiveness of the large thorns of *Opuntia* in discouraging herbivores seems self-evident. However, very little is known about the possible defensive roles of many small plant hairs or trichomes. In fact, a recent review on the biology of plant hairs practically ignores this subject (*2*). Evidence is presented here that at least one such structure, a trichome of *Passiflora adenopoda* D.C. (Passifloraceae), acts as a specific and highly efficient deterrent against heliconiine butterfly larvae, a major class of *Passiflora* herbivores in tropical America (*3*). To my knowledge, this represents the best documented example of the role and mode of action of a cuticular structure in preventing herbivore attack.

All observations on *Passiflora adenopoda* were carried out in an insectary at Stanford. The vine was grown from

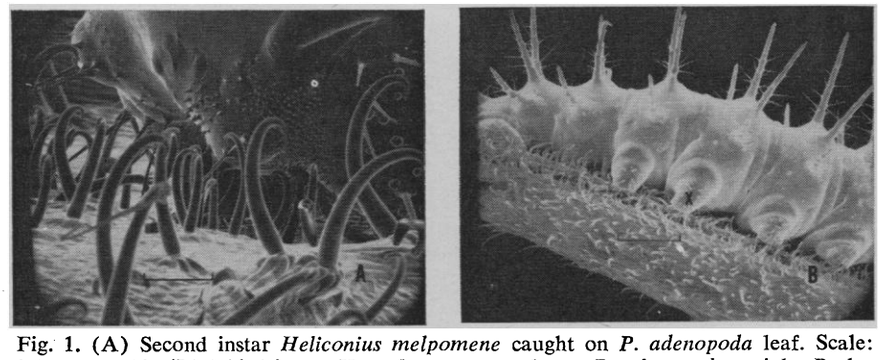

Fig. 1. (A) Second instar *Heliconius melpomene* caught on *P. adenopoda* leaf. Scale: 0.1 mm: ×80. (B) Third instar *H. melpomene* caught on *P. adenopoda* petiole. Proleg marked x is enlarged in Fig. 2. Scale: 1 mm; × 8.5.

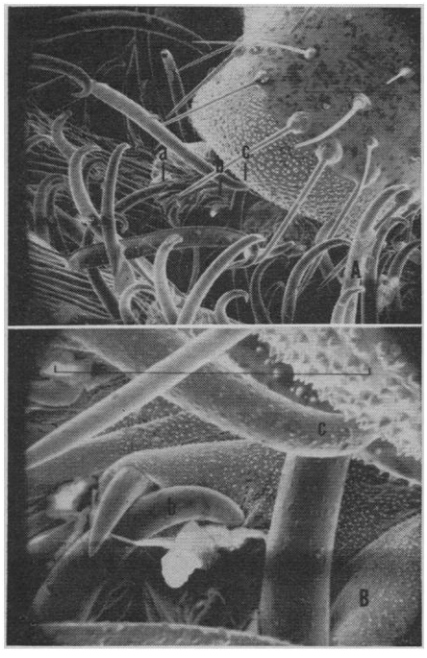

Fig. 2. (A) Detail of trichomes (*a*, *b*, *c*) hooked into *H. melpomene* proleg (X in Fig. 1B). Scale: 0.1 mm; ×85. (B) The same proleg under higher magnification. Scale: 0.1 mm; ×425 for both A and B.

seed collected in January 1968 in Costa Rica (*4*) and has been exposed to heliconiine attack for over 1 year. During this period leaves of *P. adenopoda* were not damaged, whereas other *Passiflora* in the same insectary received heavy damage. To see if *P. adenopoda* was acceptable to *Heliconius erato* and *H. melpomene* larvae, I placed a few second and third instar larvae on the leaves of the plant. The following day none of the larvae had moved more than a few millimeters, and all were dead and desiccated.

Examination of a *P. adenopoda* leaf with a hand lens showed it to be covered with hooklike trichomes. Therefore, I studied several trapped larvae with a scanning electron microscope (*5*) with the result that the defensive function of these trichomes was verified and detailed.

The scanning electron microscope micrographs show that the entire surface of the plant, including the tendrils, is covered with these hooked structures (Fig. 1, A and B) which are of such length and spacing as to securely hook from three to five trichomes into each larval proleg. Figure 2A shows in detail a solidly hooked proleg (labeled X in Fig. 1B). The anterior edge of the proleg has been folded ventrally and posteriorly as a result of being hooked by trichomes *a* and *b*. All three trichomes, which are clearly hooked into the proleg, are bent or broken in the direction in which the proleg was attempting to move.

Higher magnification demonstrates one cause of death. Figure 2B shows a split in the larval skin where trichome *b* enters, and the white blotch just below is hemolymph draining out of the wound (*6*). Death is therefore due to a combination of starvation and loss of hemolymph caused by numerous puncture wounds in the larval integument.

It is difficult to imagine how heliconiines might circumvent the highly effective and specific mechanical defense of *P. adenopoda* without drastic developmental alterations. This raises the question of why more *Passiflora* species have not evolved similar mechanical forms of defense. One possible answer is that hooklike trichomes of *Passiflora* are a recent innovation. Indeed, only three species of some 355 have been reported as having this type of trichome (*7*). Furthermore, chemical modes of defense would be expected in a plant just beginning to evolve defenses against a broad spectrum of herbivores. Chemical defenses are old enough among plants to characterize higher taxonomic categories (*8*).

According to coevolutionary theory (*8*), a succession of chemical defenses against herbivores would be produced in stepwise fashion by a host species. At each step some herbivores become extinct, while others circumvent the chemical challenge, forcing the plant to evolve further deterrent compounds. This process can be looked upon as a kind of herbivore filter which through time tends to reduce the number of higher taxa of herbivores which are able to feed on the plant. Being freed of competition, the remaining herbivores might undergo adaptive radiation on the host plant which itself has diversified during periods of relative protection (*9*). When only one or a few kinds of herbivore are important to the plant (as heliconiines are for *Passiflora*), a highly specific weapon, such as the trichomes, may be more advantageous to the plant in terms of cost as opposed to benefit than would the development of further complex chemicals, even if the absolute cost to the plant (in terms of equivalents of adenosine triphosphate or percentage of carbon fixed) is the same. The plant may also have increasing problems of autotoxicity, the solution of which requires more energy as each additional chemical defense is added.

By evolving hooked trichomes, *Passiflora adenopoda* may have won the coevolutionary race with one major group of *Passiflora* herbivores. However, it undoubtedly must contend with other kinds of herbivores against which these cuticular hooks will have little effect. It does retain extrafloral nectaries which are thought to be part of a mutualistic form of herbivore defense since they attract predaceous ants (*10*). Ants, in a sense, are like simple toxic compounds in the broad spectrum of their defenses against herbivores.

It may be significant that *P. adenopoda* is one of the most widespread species in the genus *Passiflora* (*11*). If this species spreads to the exclusion of other passifloras, a drastic change could occur within the heliconiine community. However, much more ecological information is needed about the species before its impact on the *Passiflora*-heliconiine complex of species can be assessed.

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3. Both the genus *Passiflora* and the tribe Heliconiini of the Nymphalidae are centered in the New World tropics.
4. I thank Woodruff Benson who located the plant.
5. I thank M. J. Mitchell of Kent Cambridge Scientific Inc., Palo Alto, Calif., for providing facilities and help with the scanning electron microscope work. All materials were used fresh and uncoated.
6. The black smeared area in Fig. 2B is caused by "charging up" on the liquid hemolymph.
7. *Passiflora* species with hooked trichomes include *P. adenopoda* D.C. (= *acerifolia* Cham. et Schlecht.), *P. bryonioides* H.B.K. (= *inamoena* A. Gray), and *P. sicyoides* Cham. et Schlecht.; Hans Solereder, *Systematic Anatomy of the Dicotyledons* (Clarendon, Oxford, 1908), vol. 1, p. 386.
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12. I thank members of the Population Biology Group, Stanford University, especially P. R. Ehrlich, R. W. Holm, and M. C. Singer for criticizing the manuscript. I also thank Dr. Dennis Breedlove who identified the plant. This work was supported by O.T.S. Pilot Research Grant, and by NSF grant GB-8038 to P. R. Ehrlich.

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