non of behavioral tolerance" (*5*). As discussed elsewhere (*6*), such an expanded concept of behavioral tolerance is confusing. To avoid such confusion, it would be appropriate to distinguish the "Mitchell effect" from "behavioral tolerance."

To explain the Mitchell effect, I suggested (*7*) that environmental cues present at the time of pharmacological stimulation (CS) become associated with the systemic effect of the drug (UCS). When the drug is administered in the context of environmental cues that have, in the past, been paired with the drug, drug compensatory conditional responses (CR's) attenuate the effect of the drug and are partially responsible for tolerance. The model is based on many experiments concerning the conditioning of drug effects (*8, 9*).

Hayes and Mayer suggest that the model may be inadequate because "Massed trials are more effective than spaced trials in producing behavioral tolerance . . . whereas a Pavlovian interpretation would predict the opposite." This criticism is unwarranted. Massed and spaced trials in Pavlovian conditioning refer to intertrial intervals of seconds and minutes, respectively (*10*). In the drug tolerance work cited by Hayes and Mayer, the interval between injections was varied over a range of weeks [for example, tolerance was more rapid when successive drug administrations occurred 1 or 2 weeks apart than when they occurred 3 weeks apart (*11*)]. There is no empirical or theoretical justification for the assertion that intertrial intervals of 1 to 2 weeks should lead to poorer Pavlovian conditioning than an intertrial interval of 3 weeks. Moreover, it is not established that tolerance is facilitated by such "massed" trials; indeed, some investigators have reported the opposite effect (*12*).

Hayes and Mayer also state that all my data "are the product of designs that involve repeated testing." They are incorrect. This is a further manifestation of their failure to distinguish between testing the effect of a drug and mere exposure to apparatus that will subsequently be used to test the effect of the drug (behavioral tolerance as opposed to the Mitchell effect).

I do not understand the force of Hayes and Mayer's comments about the adequacy of my published demonstrations of a hyperalgesic CR. It is true that additional research is needed. However, morphine-compensatory CR's have been reported in many experiments (*9*).

Hayes and Mayer suggest that their criticisms of the conditioning model are relevant to my other experiments in the area (*13*). However, they do not explain how any alternative model can explain these demonstrations that a variety of nonpharmacological manipulations, known to be effective in generally affecting the strength of CR's (extinction, partial reinforcement, and CS habituation) similarly affect the display of morphine analgesic tolerance; nor do they recognize that the results of these experiments show that mere exposure to the test apparatus may either facilitate or hinder the development of tolerance, in a manner readily predictable by the conditioning model, but not by alternative formulations. Also, work from other laboratories indicates that many procedures which are effective in retarding (*14*) or facilitating (*15*) morphine tolerance similarly affect conditioning.

One design of an experiment that Hayes and Mayer find appropriate for assessing the conditioning model of tolerance has, in fact, been completed (*16*). Furthermore, behavioral tolerance interpretations of this experiment are especially implausible; the role of environmental cues in morphine tolerance, and the existence of a compensatory CR, are demonstrated with a "nonbehavioral" effect of the drug (temperature alteration). Additional recent work has demonstrated that the Mitchell effect does not depend on having the analgesiometric test apparatus as part of the pretest administration environment. An arbitrary audio and visual cue can serve equally well as a CS for the elicitation of a tolerant response (*17*); thus any criticism of the conditioning model which emphasizes the effects of pretest experience with the test apparatus cannot be relevant in interpreting the results of this experiment.

Where appropriate, I have acknowledged the role of pharmacological mechanisms of a nonassociative nature, as well as of associative factors in studies of tolerance (*9, 16*), just as the contribution of learning to tolerance has been acknowledged by others who approached the issue from a pharmacological perspective (*18*).

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Sympatric Speciation: Evidence?

The claims made by Tauber and Tauber (*1*) seem to exceed the information which can be derived from the facts given. From reading the title and abstract, I was led to believe that they had evidence indicating that sympatric speciation must have occurred to account for the existence of *Chrysopa carnea* and *Chrysopa downesi*. However, their data merely indicate that it is possible to interpret this speciation event as having occurred without the necessity of geographic isolation. The evidence does not refute the equally plausible hypothesis that geographic isolation could account for the same speciation event.

The allelic differences at the three loci described explain why these two popu-

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lations are two different species and enable us to understand the selective forces involved in maintaining the distinctions, but they do not enable us to conclude whether these differences arose in sympatry or allopatry. The *G* allele (for dark green color) would be selectively advantageous in avoiding predation for any geographical isolate which coincided with a conifer-rich habitat. The d_1 and d_2 alleles (for restricted early breeding) would also be advantageous under the same conditions in minimizing competition with other conifer-inhabiting arthropod predators.

I agree that it certainly appears that *C. downesi* has been derived from *C. carnea* and that allelic changes at these three loci are probably responsible for this speciation, but there is no evidence that this occurred as a sympatric process rather than as an allopatric one.

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Hendrickson's point of contention is whether our data (*1*) provide evidence for sympatric speciation. Thus the crucial consideration is: what data, beyond direct observation, *do* constitute evidence for sympatric speciation?

As in all empirical sciences, the study of causal processes in evolution involves the formulating and testing of hypotheses. Hypotheses are formed, predictions are made, and data are sought to either corroborate or falsify the hypotheses (*2*).

Until 1966, one of the major objections to the concept of sympatric speciation (that is, speciation without geographic isolation) was that none of the models then proposed provided convincing genetic pathways for this mode of speciation (*3*). In 1966 Maynard Smith presented a theoretical, genetic model for sympatric speciation through disruptive selection in a two-niche situation (*4*). This model contains discrete, testable steps, and it now forms the primary hypothesis for sympatric speciation through disruptive selection. Until our study, the main experimental and observational data supporting this model came from Bush's work with monophagous, frugiverous flies (Tephritidae) which, in the process of speciation, form divergent, sympatric host races (*5*).

We sought to expand the applicability of Maynard Smith's model by testing the consistency of its predictions for nonmonophagous, nonparasitic animals. Our observations of the general insect predators *Chrysopa downesi* and *Chrysopa carnea*, and the results of our experiments with these species, precisely fit the predictions from Maynard Smith's model. First, a single-gene polymorphism adapts homozygous individuals to either of two niches (in our case, two habitats). Second, individuals heterozygous for the polymorphic trait are at a distinct disadvantage in both niches (habitats); this situation subserves the maintenance of a stable polymorphism through disruptive selection. Third, reproductive isolation is the result of very small genetic differences between the forms.

Taken together, these data are most simply explained in the context of sympatric speciation. To assume that the species evolved under geographic isolation, as extreme advocates of allopatric speciation would, would require a more complex series of assumptions—five steps as opposed to our two: (i) geographic isolation of a population of *C. carnea* in a coniferous area; (ii) evolution of the dark-green adult coloration in the isolated population; (iii) elimination of summer breeding by the isolated population; (iv) secondary contact of the two species; and (v) subsequent refinement of the seasonal asynchrony (postponement of breeding by the overwintering coniferous population from midwinter until early spring), as opposed to (i) evolution of a single gene polymorphism involving adult coloration and accompanying habitat association and (ii) evolution of seasonal isolation between the sympatric forms occupying different habitats.

Thus our data (*1*, *6*) which are most simply and most fully explained in terms of sympatric speciation, and which precisely fit an unrefuted model for sympatric speciation, constitute evidence for sympatric speciation.

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Sympatric Speciation: Evidence?

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