against human perturbation and natural disaster; (v) large areas are necessary to minimize the pressures of predation, parasitism, and competition exerted by species abundant in the disturbed areas surrounding the reserves; (vi) failures of small reserves, originally considered to be adequate, have been amply documented; and (vii) the irreversibility of fragmentation demands a conservative preservation strategy.

Simberloff and Abele have performed a useful service by focusing attention on the potential pitfalls of an oversimplified model. We contend, however, that existing theory corroborated by empirical data is sufficient to validate the general conclusion that refuges should contain as large a contiguous area as possible.

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18. Species that never bred were: black-and-white warbler (*Mniotilta varia*), worm-eating warbler (*Helmitheros vermivorus*), hooded warbler (*Wilsonia citrina*). Species that bred sporadically were: Acadian flycatcher (*Empidonax virescens*), American redstart (*Setophaga ruticilla*), blue-gray gnatcatcher (*Polioptila caerulea*), ovenbird (*Seiurus aurocapillus*), scarlet tanager (*Piranga olivacea*), yellow-throated vireo (*Vireo flavifrons*).
19. At the time of the studies of R. R. Graber and J. W. Graber [*Ill. Nat. Hist. Surv. Bull.* **28**, 383 (1963)], all the missing species were recorded as breeding in Illinois.
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We regret being cast as the bêtes noires of conservation, since our report (*1*) was designed to strengthen conservation efforts by eliminating reliance on a species-area equation which alone does not support either of two contrasting refuge strategies, and by tailoring conservation efforts to the idiosyncrasies of the taxa in question. We do not agree with Diamond (*2*) and Terborgh (*3*) that their data are adequate to support the hypothesis of high extinction rates for birds on islands. With one exception, the "evidence" from land-bridge islands rests not on observation of which species were originally present, but rather on inference from the present source fauna and the species-area equation. Even were habitat differences well quantified, which they are not, the wide variance in fitting data to the standard species-area curve (*4*) would make such a deductive leap suspect. For Barro Colorado Island at least, the original birds are documented, but the island has undergone major vegetational change in the last century (*5*) and so can hardly be used as an example of extinction following change in the single variable of area. Perhaps with long-lived animals few appropriate data exist, but this suggests great caution in erecting general theories about extinction.

With respect to the "extreme model," referred to by Whitcomb *et al.* (*6*), we *did* point out that this would be an "oversimplification," and then cited many of the same references which Whitcomb *et al.* use, to exactly the same end: to indicate how the model might be made more realistic. We did not "pass over lightly" (*3*) extinctions; our third paragraph from the end addressed exactly this problem.

Our conclusion still stands: the species-area relationship of island biogeography is neutral on the matter of whether one large or several small refuges would be better. We repeat our earlier statement: "This is not a plea, then, for a specific conservation regime, but rather for more comprehensive autecological consideration."

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Juvenile Hormone and Pest Control

The search for novel chemical pest control agents with improved characteristics, such as increased selectivity for target pests as compared to beneficial arthropods and vertebrates, is a long and difficult process for which the short-term economic incentives are far more elusive than for the development of broad-spectrum pesticides.

The report by McNeil (*1*) appears to

lead to negative conclusions as to the use of juvenile hormone (JH) analogs in integrated insect control practices. However, examination of his data reveals that the generalizations implied in the abstract and in the report cannot be justified on the basis of the evidence presented.

No less serious is the recognition that the methods used by McNeil do not guarantee any relevance to projected effects in the field, and that the statements on higher activity against the parasite as compared to activity against the target pest are based on a biased test procedure. Absolute target pest specificity (if attainable at all) may be the ultimate goal for the integrated control manager, but at present it offers insufficient incentive for commercial development. Appreciable increases of selectivity over currently employed broad-spectrum insecticides may be able to favorably tip the pest-parasite balance in long-term use under field conditions. It is exactly this that juvenile hormone analogs generally have to offer. Their intermediate arthropod selectivity (coupled with a very favorable vertebrate toxicity margin) is extensively documented (*2*) and may satisfy users and producers in this respect.

Although McNeil concedes that "several, if not all stages of the life cycle of the pest's parasitoids are present concurrently," he proceeds to describe his "more realistic test" on only immature parasitoids and fails to mention that the adults are likely to escape damage and should be able to continue to attack any surviving pest hosts.

A second objection is McNeil's presentation of final mortality figures due to IGR (insect growth regulator with JH activity) treatments. These strongly suggest that the IGR's in question have practically no effect on aphids, but kill 100 percent of the parasitoids. However, our experiments (*3*) have shown that third-instar nymphs of several species of aphids would be controlled completely, even at the lowest concentration (0.01 percent) of the IGR in question, provided that one waits for aberrations in the metamorphosis of the aphids to materialize with subsequent mortality or failure to reproduce (or both). Since this cannot be observed on parasitized aphids that mummify, a control group that was unparasitized and treated should have been included in the experiments in order to evaluate the host mortality separately. The results as published, therefore, cannot be interpreted as indicating a differential activity on host and parasite, and any statement to that effect is misleading. It should also be recognized that even the most selective control agent is likely to cause the death of an endoparasitoid when the host is killed prior to the full development of the parasitoid, but this in itself should not be a cause for concern.

Under field conditions, the population dynamics of pest-parasite complexes is further complicated by the presence of hyperparasites, which should also be included in any realistic evaluation.

Even if one would succeed in showing that, in properly designed experiments with candidate control chemicals, a parasite suffers more damage than a pest host, it would still not be justifiable to extrapolate the conclusions, as McNeil did, to other species, to other IGR's, and to the myriad of complex conditions existing in the field.

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At no time did I dispute (*1*) the laboratory findings of Nassar *et al.* (*2*) concerning the effects of certain JH analogs on nonparasitized greenbugs, from which, incidentally, they conclude (by extrapolation?) that the JH analog ZR777 would completely control several aphid species even at a concentration of 0.01 percent. The data I reported [table 1 in (*1*)] represent the mortality of parasitized aphids resulting from the application of JH analogs and not from the effects of a developing parasitoid, and they were necessary to show that the parasitoid mortality recorded (table 2) was the result of JH analog treatments alone. I believe that even if JH analogs had no direct effect on parasitoids, yet killed hosts that contained parasitoids, it would be cause for concern. This is so because the host is already doomed by parasitization and its premature demise due to chemical treatment only results in the loss of a beneficial insect, the parasitoid. I also feel that the marked differences in aphid and parasitoid mortality, as observed within the context of my experiment, are important. This view is supported by the work of Poe (*3*), who studied the effects of ZR619 and ZR777 on the tomato pinworm and an associated endoparasitoid. Emergence of pinworms pupating in sand that had been treated with JH analogs was not suppressed while parasitoid emergence was totally inhibited (*3*).

The statement that my experimental design represented a "more realistic" approach was made with reference to data (*4*) where hosts were treated prior to parasitization, and not at different times after parasitoid attacks, as would be the case under field conditions. The argument that adult parasitoids may escape the effects of JH analog treatments in no way diminishes the importance of the high endoparasitoid mortality within the insect pest.

Contrary to the implications made by Staal and Nassar in their statement that "it would still not be justifiable to extrapolate the conclusions, as McNeill did . . .," I in fact recommended that "Comprehensive studies are essential to evaluate fully the off-target effects of these 'third generation' insecticides prior to their use on a commercial scale in natural ecosystems." The sensitivity of several parasitoids to different JH analogs under laboratory conditions (*1, 3, 5*) convinces me that large-scale use of these products prior to in-depth field studies would be a serious error. If JH analogs, even though effective against the insect pest, seriously disrupt the natural host-parasitoid balance, a situation such as that described by Plapp (*6*) could arise where "An insecticide highly toxic to parasitoids and predators may have the paradoxical effect of actually increasing numbers of the pest it is supposed to control." This is definitely not the objective of a well-designed integrated control program.

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