have the most demonstrable effects in leading to "less desirable" conditions in the estuary. Studies of the effects of thermal discharges have failed to document any substantial damage from present inputs.

Perhaps the most disturbing aspects of Holden's articles are contained not in her "floating statistics," but in the following three sentences: "Although the bay has long been a laboratory for marine scientists, no comprehensive plan has yet been developed to protect its bounty and order its progress. . . . Is the bay dying and if so what can be done to save it? Decisive action will have to await more detailed understanding of the bay." Holden's implication that scientists should devise a master plan for the management and utilization of this great natural resource is perilous, and her statement that there is presently not sufficient scientific information for any decisive action is erroneous. There are certainly countless unanswered scientific questions—there always will be. But the general features of many of the important processes in the bay are known and understood, and scientific predictions can be made. In many respects, scientific information has developed at a faster rate than management's ability to utilize it. Decisions of how to manage the Chesapeake Bay, of how to "order its progress," require not only scientific inputs, but social and economic inputs as well. Management problems rarely have unequivocal answers. They are very frequently value judgments, and natural scientists have no peculiar talents for making such decisions. Science cannot solve all of man's problems, environmental or otherwise. It cannot incontestably determine either what uses of the bay are most important, or even what uses are most desirable. Through science we can learn to understand the bay and even, in part, to control it, but science cannot unequivocally and decisively determine the ways in which we *should* control it. Scientists can, however, help to design and implement the actions necessary to the attainment of the desired ends, once those ends have been selected. Decisive action does not "await a more detailed understanding of the bay." It awaits a crisis. And there is little evidence to suggest that such "decisive action" will be predicated on scientific knowledge.

The Chesapeake Bay has served her many masters well, and she continues to do so. The bay is healthy, but it requires nonsimplistic management.

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3. Contribution 158 of Chesapeake Bay Institute of the Johns Hopkins University. We thank G. Murphy, F. Seiling, J. Carpenter, and S. Fowler for their suggestions. The work was partially supported by the Fish and Wildlife Administration of the State of Maryland and the Fish and Wildlife Service, Bureau of Sport Fisheries and Wildlife, Department of the Interior, through Dingell-Johnson funds, Project F-21-1.

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Chemical Defense Mechanisms and Genetic Polymorphism

Although this comment is not intended to be critical of the admirable article of Whittaker and Feeny (*1*), it does aim to be complementary in that it emphasizes a point crucial to their argument which they overlooked. Evolution of the type they described does not take place at the specific level, but within species. Thus in order to substantiate their thesis, particularly for passive defense mechanisms, it is essential to show that chemical differences between individuals in polymorphic or continuously varying populations convey differential advantages as far as protection is concerned. Admittedly, they did briefly mention *Bufo regularis*, *Strophanthus sarmentosus*, and *Nezara viridula*, but none of these examples fulfills the criteria required.

The impasse can be expressed another way. Where the same form of a character occurs in all members of one species, but in none of a closely related one, it is not possible to avoid confounding the character with the species. This is, of course, the ideal state of affairs for the taxonomist. Yet, when we consider a species monomorphic for a secondary substance, we have no means of telling whether a particular nonparasite could and would attack that species if the putative defensive substance were not present.

The only way around this difficulty is to examine species polymorphic for secondary substances; that is, we must examine species in which some individuals contain secondary substances while others, in the same interbreeding population, do not. If we find that it is only, or predominantly, the form not containing the secondary substance which is attacked by the parasite or eaten by the animal, only then are we justified in concluding that the secondary substance is protective or defensive.

The differential eating of the acyanogenic forms of *Lotus corniculatus* L. (*2, 3*) and *Trifolium repens* L. (*3*) is clearly established, and so, undoubtedly, cyanogenesis in these species can be regarded as a defensive mechanism, although it is likely to be more important to the seedling than to the adult (*4*). Sickle-cell anemia and malaria is now the classic example (*5*); the genetic systems associated with interactions of vascular plants and fungal pathogens are equally elegant (for example, *Linum usitatissinum* (flax) and *Melampsora lini* (flax rust) (*6*), in spite of the fact that in most cases the exact chemical differences between resistant and susceptible host plants are inferred rather than proven. For an example where quantitative rather than qualitative differences are important, it is clear that the concentration of chlorogenic acid in potato tubers determines the differential palatability of three potato varieties to some slug species (*7*). Thus there is good evidence that secondary plant substances do act as defensive mechanisms at the intraspecific level.

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