The results of this experiment, shown in Table 1 and Fig. 1, indicate that infusion according to the rat's individual eating pattern always produced a greater suppression of oral intake than did a continuous 24-hour slow infusion. Examination of the pattern of eating during the infusions suggested a mechanism by which reduction of intake was achieved. During both slow continuous and intermittent meal infusions, the number of meals taken over the 24-hour period was substantially reduced. It seems likely, therefore, that the infused diet provided metabolic signals which postponed the onset of a meal by maintaining the level of some metabolite above a critical threshold. On the other hand, the sizes of meals were reduced only by discrete meal infusion.

The results of this experiment strongly suggest that periodic rapid increases in caloric needs which were not offset by the slow infusion resulted in the regulatory failure in our first experiment. Our third experiment, however, did not rule out the possibility that the suppression of intake when rats were loaded discrete meals simply resulted from filling the animals' stomachs periodically with food. In order to disengage stomach loading per se from stomach filling when the animal was hungry, a fourth experiment was carried out.

In order to control more precisely the time at which an animal would eat a meal, a device was constructed and programed so that every 3 hours a door opened allowing access for 15 minutes to the liquid diet. When animals had adapted to the 3-hour schedules the infusions were begun, while the animals were maintained on the same schedule. Each of six animals was given three intragastric infusions: a continuous slow 24-hour infusion, an infusion 10 minutes before the door opened, and an infusion 90 minutes before the door opened. Animals were loaded meals equal in size to the mean meal size taken on the preinfusion day on which intake was the highest. Table 2 clearly shows that the suppressing effect of loading discrete meals observed in experiment 2 is not the result of periodic stomach filling. In fact, loading meals 90 minutes before animals were due to eat does not suppress oral intake any more effectively than does slow infusion. This experiment therefore strongly suggests that intragastric loading is more effective in suppressing food intake if it occurs when the animal is ready to eat a meal.

Furthermore, it seems that neither continuous slow infusions nor loads which begin once eating is in progress are as effective in suppressing food intake as are loads which anticipate a spontaneous meal. For example, Thomas and Mayer (*6*) infused 50 or 75 percent of rats' total daily intakes either continuously or during spontaneous meals. Calculations based on their data show that each milliliter of intragastric load reduced food intake 0.75 ml under either condition. In our experiments, infusions of 100 percent of the rats' total daily intakes reduced consumption 0.61 ml per milliliter of intragastric load during slow infusions and 0.86 ml per milliliter of intragastric load during simulated meal infusions. Thus, when equated on the basis of a milliliter of suppression of oral consumption per milliliter of infusion, loading which anticipates spontaneous feeding is the most effective means of suppressing food intake by intragastric infusion. Nevertheless, it is possible that complete suppression was not obtained because oropharyngeal stimulation may be necessary for complete satiation. This is suggested by Snowdon's (*3*) demonstration that in rats obtaining all of their food by intragastric self-injection, intragastric intakes were 62 to 76 percent of oral intakes.

The major conclusion to be drawn from this study is that the effectiveness of suppression of oral intake depends not only on the total amount of food infused but also on the temporal distribution of the infusions relative to the time when the animal is about to eat. This finding would indicate that factors other than the total caloric intake may play a more important role in determining satiety than some of the earlier infusion studies had suggested (*4, 5*). Finally, these data may have some clinical application where enteral nutrition is the preferred route of hyperalimentation (*11*).

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Chesapeake Bay: A Second Look

In a recent issue of *Science* (*1*), Holden wrote an article on the Chesapeake Bay, describing some of the uses made of the bay and the different, sometimes conflicting, demands made on the estuary by these activities. Holden compiled several impressive statistics on the areas of oyster bars, clam bars, and beaches that have been closed because of "pollution," as well as statistics on declining oyster production. These figures may leave the reader with the impression that the Chesapeake Bay is "on the ragged edge of becoming badly polluted," as the Chesapeake Bay Foundation has stated. For the most part, Holden's statistics are "accurate," but they are strongly reminiscent of the comic sportscaster's account of partial baseball scores: "In the game

in Detroit between the Tigers and the Baltimore Orioles, it was the Detroit Tigers 5!" From this report, one cannot tell whether the Tigers won or lost. From Holden's account, one cannot begin to objectively assess either the possible harmful effects these activities have had on the bay, or the general state of health of the bay.

Apparently in order to establish the once pristine condition of the bay, Holden begins her story with an often-quoted description by Captain John Smith: "Heaven and earth seemed never to have agreed better to frame a place for man's commodious habitation." This "commodius habitation" is, according to Holden, threatened by pollution whose "ominous signs may be read in seasonal fish kills" and other menacing indicators. It is ironic that in a seldom-quoted account of one of his voyages on the unspoiled, "commodious" Chesapeake Bay, Smith recorded (*2*):

> . . . that abundance of fish, lying so thick with their heads above the water as for want of nets we attempted to catch them with a frying pan . . . neither better fish, nor variety of small fish had any of us ever seen in any place so swimming in the water . . . and some we have found dead upon the shore.

Smith was describing a fish kill in the pristine waters of the upper Potomac (Patawomek) estuary, a fish kill long before the "march of progress" imperiled the health of the bay.

Officials of the Fish and Wildlife Administration of the State of Maryland have no evidence that fish kills have increased either in frequency or in severity since Smith wrote his account. In fact, fish kills are a superficial yardstick for measuring pollution, one whose application may lead to false conclusions.

Holden points out that other "ominous signs may be read in . . . the 28,000 acres of oyster beds and 39,000 acres of clam beds that have been closed due to pollution." The 20,877 acres of oyster bars that were closed in 1970 represent less than 7 percent of the total area of productive oyster bars in Maryland waters, and of these 20,877 acres, 15,007 acres were closed because of pollution (coliform bacteria counts). The remaining 5870 acres were closed because of a Maryland regulation requiring a "buffer zone" around sewage treatment plant discharges. The total area of oyster bars closed each year during the past decade has ranged from more than 27,000 acres in 1963 to less than 10,000 acres in 1965, and has averaged about 20,000 acres. The total area has not increased in the past 4 years. The 39,000 acres of clam beds that were closed represent about 13 percent of the total clam bottom in Maryland waters. The total closed area has decreased over the past 5 years.

Holden made the following statement concerning oyster production:

> The supply of oysters has been severely depleted in recent decades, largely because of overexploitation. In the early 1900's, the annual take from 300,000 acres of oyster bars was between 8 and 10 million bushels. Now the harvest is between 2 and 3 million bushels.

Although statistics on seafood harvests prior to 1929 are not very reliable, there is no doubt that oyster production has declined since the early days of commercial oystering in the bay. At that time, oystermen were harvesting an accumulated resource, a resource virtually untapped except for the small quantities of oysters taken by Indians and settlers. Because bars were literally stripped clean, early harvest figures frequently included seed oysters and empty shells. Modern catch figures include only marketable oysters.

The decline in the bay's oyster production since the turn of the century is attributable to approximately 100 years of overfishing. Maryland's production fell to less than 1.5 million bushels in the 1961–62 season. Many of the bars had been depleted, and the rate of natural recruitment was not sufficient to bring them back. In 1961, Maryland reenacted a seed production program. By the 1965–66 season, production began to climb rapidly, and since 1966, Maryland has led the nation in oyster production by a substantial margin. Over the past 4 years, Maryland's harvest has ranged from more than 3 million bushels for the 1966–67 season to about 2.5 million bushels in 1970–71. Production this past season was limited by the market, not by the crop. Increased oyster production awaits the creation of new markets and new processing techniques.

Since 1963, the value of Maryland's total catch by commercial fishermen has been increasing at the rate of about $1.5 million a year. Over the past decade, Maryland's annual commercial catch of striped bass, averaging more than 3 million pounds, has led the nation. Sport fishermen are estimated to take more than twice this number. Maryland leads the nation in the production of soft-shell clams, and the Chesapeake Bay is the nation's leading producer of blue crabs.

The general trend of the total catch and the total value of the Chesapeake Bay's commercial fisheries has been upward since the 1930's. The success of individual fisheries has fluctuated quite wildly over the same period. Large natural fluctuations of estuarine fisheries are common and may be greater than fluctuations caused by fishing. No other causes of fluctuations have been documented. The Chesapeake Bay remains one of this nation's richest fishing grounds.

When we read statements of the deterioration of the bay, we are reminded of Mark Twain's cable to the Associated Press after he read of his death: "The reports of my death are greatly exaggerated." Several of the bay's tributaries are, in layman's language, "dying." To be more nearly correct, they are much too "alive." Large populations of blue-green algae thrive in the nutrient-rich Potomac and in Baltimore Harbor and Back River. Blue-green algae are also, according to the Environmental Protection Agency in Annapolis, Maryland, beginning to appear in the Gunpowder and the Bush rivers, two small tributaries of the upper bay. The presence of these algae, following the construction of sewage treatment plants on these tributaries, could have been predicted. The adjacent upper bay is still "healthy," but the levels of nutrients, whose primary source is the Susquehanna River, are near the upper limit. The phytoplankton production is high, but the grazing rate is also high, thus preventing a buildup of undesirable algae.

The Chesapeake Bay is certainly threatened with change by man's activities. Some changes have already occurred, but, by most currently accepted standards, the main portion of the bay is healthy. It is because of its healthy state that it bears close watching. This is not to suggest that we are necessarily using the correct yardsticks. Large harvests of shellfish and finfish are poor indicators of a healthy estuary if the shellfish and finfish cannot be consumed because of high concentrations of pesticides or heavy metals. Sewage, pesticides, herbicides, and sediments pose the greatest "threats" to the bay. These by-products of man's activities

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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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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