April 1970 and again from May/June 1970 through November/December 1970. However, in the intervening period, March/April to May/June, which coincides with the premonsoon rise in egg count, *A. duodenale* increased and accounted for about 91 percent of the rise which occurred.

Temporarily arrested development of larval nematodes within their normal definitive hosts is being recognized increasingly as a common alternative to uninterrupted maturation; dormant parasitic larvae are particularly well known among trichostrongylid nematodes of domestic ruminants (*12*). Recent investigations indicate that among these species a potential to enter a diapause-like state within the host is induced by degenerating environmental conditions acting on the free-living larvae (*13*). Thus, seasonal suspension and resumption of parasitic development are attuned to unfavorable and favorable external conditions, respectively, and egg output wasted in seeding an inhospitable environment is reduced to a minimum. Our observations indicate that a similar phenomenon occurs in yet another family of nematodes, the Ancylostomatidae, which parasitizes man and numerous carnivores. This suggests that the seasonal occurrence of dormant parasitic stages may prove to be an adaptation evolved in a broad spectrum of nematodes whose hosts inhabit areas which are seasonally unfavorable for the development of the free-living stages.

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Acanthaster: Effect on Coral Reef Growth in Panama

Abstract. *Analysis of data on coral abundance and growth, and the population size (26 individuals per hectare) and feeding rate of* Acanthaster *indicates that* Pocillopora *reefs on the Pacific coast of Panama are undergoing vigorous growth in the presence of this predator. Prediction of the effects of a population increase in* Acanthaster *to plague proportions (2.5 times that presently observed) suggests that reefs could still maintain a positive growth. However,* Acanthaster *at ten times the present population density would lead to rapid destruction of reefs. It is argued that coral destruction due to* Acanthaster *represents only one of several factors affecting coral reef progression.*

In spite of the various recent efforts directed to the *Acanthaster* problem, especially following the reports of significant coral destruction in Australia (*1*) and Guam (*2*), our present understanding of the amount of coral predation that a reef community can sustain is still largely speculative. This report presents an analysis of a small coral reef in terms of its growth potential and modification by the feeding pressure of the crown-of-thorns sea star *Acanthaster planci* L. (*3*). A first order approximation is offered on the balance between reef growth and *Acanthaster* predation under present conditions, and the projected outcome at increased population densities of this predator.

The coral reef selected for study is a discrete patch reef located on the northwest side of Uva Island, Gulf of Chiriquí, Panama, centered at 7°48′46″N, 81°45′35″W (*4*). The total reef encompasses 13,800 m^2 with the shoal reef crest comprising 52.1 percent and the reef flanks 47.9 percent of the area (*5*). The mean coverage (and .95 confidence limits) of live coral was 0.57 ± 0.10 m^2 on the reef crest and 0.32 ± 0.14 m^2 on the reef flanks (significantly different at $P < .001$), amounting to a total coverage of 6200 m^2 (*6*).

At least 11 scleractinian species and two species of *Millepora* are present on the reef. The reef crest is dominated by *Pocillopora* spp. with *P. damicornis* (Linnaeus) the most abundant species. *Pocillopora* is also the most abundant coral around the reef periphery in deeper water. However, large massive corals, such as *Pavona clivosa* Verrill, *Pavona varians* Verrill, and *Pavona ponderosa* Gardiner, assume an increasing importance along the reef base [5 to 6 m in depth relative to the mean low water springs (MLWS) datum]. Quantitative sampling showed that *Pocillopora* contributes 98.4 percent to the total live coral coverage of the reef.

Work in progress has shown that the Pacific reefs of Panama, contrary to former opinion (*7*), are true structural reefs undergoing vigorous accretionary development (*8*). Preliminary radiocarbon dating of coral carbonate material from the Uva Island study reef indicates a vertical growth of 1 m per 250 years. The horizontal growth of *Pocillopora damicornis*, measured in the nearby Secas Islands, was found to be 1.5 ± 0.2 mm/month at depths of 1.5 and 6 m (data pooled at significance level $P >> .10$) over the period 23 September to 7 November 1972 (*9*). By adding these growth rates to corals which can increase in size peripherally, that is, to colonies bordering unoccupied spaces of the reef surface, horizontal reef growth was estimated to equal 21.4 percent per annum. Since these data were obtained at the height of the wet season, with frequent heavy overcast skies, they probably represent a minimal estimate of the mean annual coral growth (*10*).

Clearly, horizontal reef growth of the magnitude presented here is seldom, if ever, realized for various reasons. For example, the lateral spreading of the Uva Island reef to seaward appears to be restricted by the abrupt deepening of the water. A shoreward extension is possibly limited by the influence of land drainage effects (freshwater dilution, siltation, and so forth). Some biotic factors known to limit coral growth include competition for space with other benthic populations, destruction of the skeleton by borers, and the constant cropping of corals by a variety of animals. For example, the magnitude of attrition due to grazing corallivores (affecting both vertical and horizontal growth) can amount to 6.4 percent of the standing crop of *Pocillopora* in the Gulf of Panama (*8*).

Periodic censusing of *Acanthaster* since March 1970 indicates the presence of a stable population on the Uva Island reef, ranging between 25 and 36 individuals, or 18 and 26 animals per hectare (*11*). Individual size measurements showed modal disk diameters to range between 17 and 19 cm. Size-frequency distributions are unimodal and similar in form to those reported by Dana *et al.* (*12*), suggesting a stable population structure with relatively constant recruitment.

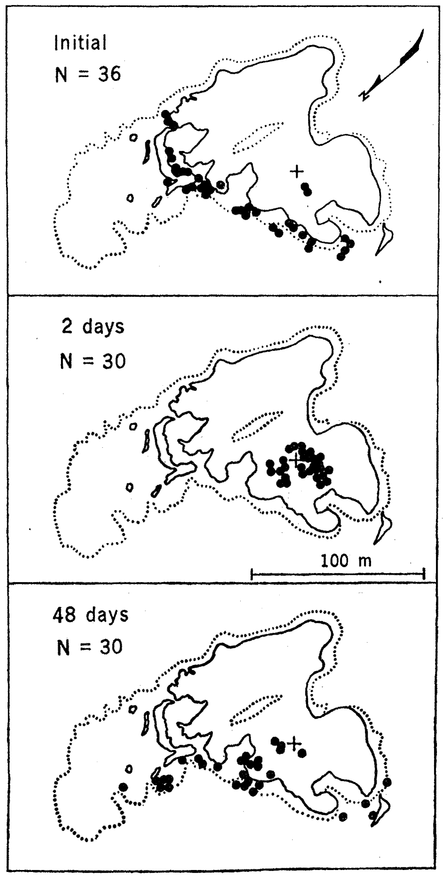

Fig. 1. Distribution of *Acanthaster* on the Uva Island study reef in an undisturbed condition (*Initial*), and in 2 and 48 days after release on the reef crest, at the position marked by a cross (21 September to 8 November 1972). The approximate contour depths are 1 and 4 m (relative to the MLWS datum) for the reef crest and reef flanks, respectively; *N*, number of individuals.

Field mapping showed *Acanthaster* to be distributed along the seaward edge of the reef where coral abundance is relatively low (Fig. 1). The majority of all individuals experimentally transported to the reef crest moved to the deeper reef edge, where they assumed a distribution similar to that found initially, in a maximum period of 48 days. Animals placed on live coral on the reef crest had all positioned themselves in spaces free of live coral in 2 days. This would indicate that *Acanthaster* avoids bottom areas with continuous or very abundant coral cover. It could also be argued that *Acanthaster*'s distribution may be related to water depth (with attendant differences in turbulence and light penetration). The sea star is commonly found, however, at shallow depth where coral growth is sparse. Moreover, Barnes *et al.* (*13*) demonstrated experimentally an instant retraction of the tube feet of *Acanthaster* on contact with *Pocillopora*. This observation was also verified in Panama by short-term field studies of the locomotory behavior. *Acanthaster* that were repositioned to bottom areas with continuous coral cover invariably demonstrated a rapid movement over the live coral; the sea stars came to rest only on contact with a noncoral substratum. All of these observations support the notion of a strong avoidance of live coral. That coral destruction on *Pocillopora* reefs appears to be largely confined to the periphery of the reef core is explained by these findings.

The feeding rate of *Acanthaster* was determined directly from the observation of individuals on known coral surfaces and estimated from the total area of blanched dead coral found in the immediate vicinity of feeding animals. It was assumed that this blanched coral had been destroyed less than 5 days before the observations since it becomes discolored by sediment accumulation and fouling in about 5 days. These data indicate a modal feeding rate of a 0.45-m^2 area of reef surface per individual per month. Feeding rates reported at Guam (*2*) were 1 m^2 per month, over twice the present value for animals with a disk diameter of only 13.8 cm.

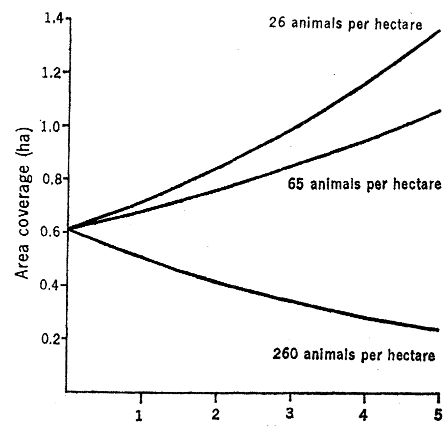

Fig. 2. Hypothetical time course of coral destruction by *Acanthaster* at varying population densities. The *Acanthaster* densities are adjusted in proportion to the coral abundance. Horizontal reef growth was calculated as follows: (area coverage − annual destruction) (horizontal coral growth) + (area coverage − annual destruction), in order to maximize the effects of feeding and minimizing the calculated horizontal reef growth.

Feeding was observed during the day and at night (*14*). From 197 feeding observations, *Pocillopora* comprised (numerically) slightly over 85 percent of the prey. A variety of other scleractinian corals, and the hydrocoral *Millepora*, were also eaten by *Acanthaster*. Several large massive colonies of *Pavona* present along the reef base were preyed on extensively during 1970 and 1971. However, all of 22 colonies examined recently (21 September 1972) showed small areas of live coral (less than 1 percent of the surface area) which had survived. The patches of living coral were usually present in narrow depressions and fissures. Parts of large massive corals (*13*) or whole small colonies in inaccessible positions on the reef floor (*15*) also survived attack on western Pacific reefs. Such surviving colonies are probably important in reef recovery both in terms of their ability to regenerate over grazed surfaces and for eventual restocking of decimated bottom areas.

From the data presented on the abundance of living corals [6100 m^2 of *Pocillopora* (*16*)], horizontal reef growth (21.4 percent per year), and the feeding rate of *Acanthaster* (5.4 m^2 of living coral per year per individual), it is possible to make some reasonably precise predictions on the outcome of continued feeding by this predator. Figure 2 shows the projected growth trend of the Uva Island *Pocillopora* reef at three different population densities of *Acanthaster* over a 5-year period. The present population size of *Acanthaster*, comprising 36 individuals total (26 individuals per hectare), is estimated to result in the destruction of 194 m^2 of coral per year, or 3.2 percent of the reef surface standing crop. If the mortality and recruitment of corals is assumed to be in balance, it is seen that horizontal reef growth more than compensates for the coral destruction due to *Acanthaster*. This analysis also suggests that reef growth would probably not be interrupted by low plague densities of *Acanthaster* (65 individuals per hectare). However, it is predicted that reef destruction would occur rapidly at ten times the present population.

In conclusion, it is apparent that *Acanthaster* represents only one of several factors tending to limit the formation and progression of coral reefs. Further, the number of *Acanthaster* present on the majority of coral reefs investigated (*17*) would appear not to exceed the carrying capacity on the basis of the present analysis. Until more accurate data are available on the growth dynamics of coral reefs, in my opinion it would be imprudent to continue supporting the extermination of *Acanthaster*, except in those areas where it can be demonstrated to constitute a real threat to the continued existence of reefs.

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Nerve Growth Factor: Enhanced Recovery of Feeding after Hypothalamic Damage

Abstract. *A single intraventricular injection of nerve growth factor (NGF), given at the time of brain damage, facilitated the course of recovery from the lateral hypothalamic anorexic syndrome in male rats. In the second and third weeks after the trauma, NGF-treated rats ate more food, regained body weight more rapidly, and fed more vigorously in response to intraventricular administration of norepinephrine than untreated controls. After full recovery, rats that had been treated with NGF were resistant to reinstatement of the hypothalamic syndrome by 6-hydroxydopamine. NGF may facilitate behavioral recovery by promoting the development of supersensitivity to norepinephrine and possibly also by stimulating the growth of regenerating noradrenergic neurons in the brain.*

Nerve growth factor (NGF), a protein with structural similarities to insulin (*1*), is aspecific stimulator of growth and differentiation of peripheral sympathetic and sensory cells (*2*). Although the effects of NGF in the periphery have long been recognized, its action on the central nervous system has only recently been detected. Using histochemical fluorescence methods, Björklund and Stenevi (*3*) demonstrated that NGF stimulates the sprouting and growth of regenerating noradrenergic neurons in the brain. After transection of noradrenergic axons in the caudal hypothalamus by a transplant of iris tissue, a single intraventricular injection of NGF given at the time of damage led 7 days later to a striking increase in the number of newly formed sprouts in and around the transplant. It was suggested that "NGF could be used to accelerate, increase the magnitude of, or improve the final result of, regeneration of central catecholamine neurons" (*3*).

We report here for the first time evidence of the behavioral activity of NGF. This work complements the histochemical studies of Björklund and Stenevi (*3*) and provides, at the same time, some new information about the processes that may underlie the recovery of function after brain damage.

Our experiments were concerned specifically with the recovery of feeding after hypothalamic damage. Feeding may be abolished for several weeks by mechanical or electrolytic destruction of the lateral hypothalamus on both sides (*4*). However, if death is prevented by forced feedings and appro-