the fused cells were not only numerous, but extremely large and bizarre, with nuclei ranging from 40 to 200 per cell (Fig. 1). Since the size and number of multinucleated cells in such cultures are dependent on the amount of virus in the culture (*8*), it can be concluded that significant amounts of leukemia virus were activated in these mice. Spleen weights were also larger in the group of mice receiving both grafts and ALS than in any other group ($P < .01$, *t*-test), although there was no clear correlation in individual animals of this group between spleen weight and recoverable virus.

These results are the first to show activation of leukemia viruses after a homograft transplantation. They also demonstrate that activation of leukemia viruses by immunologic reactions to histocompatibility antigens is not confined to long-term graft-versus-host disease (*4*), but applies to host-versus-graft reactions as well. The observation that both immunosuppression and rejection reactions appeared necessary for maximum virus activation suggests that these two processes may complement each other in the mouse transplant recipient. It would appear that virus becomes activated by the immune reaction and once activated becomes amplified by the recipient's inability to immunologically eliminate it. Whether this sequence will subsequently lead to an increased incidence of malignant lymphomas in these mouse transplant recipients remains to be determined. It is also intriguing to speculate that a similar series of events may account for the frequent appearance of various viral infections and malignant lymphomas in human transplant patients undergoing therapeutic immunosuppression (*1, 9*).

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Arrested Development in Human Hookworm Infections: An Adaptation to a Seasonally Unfavorable External Environment

Abstract. *Contrary to general belief, larvae of* Ancylostoma duodenale *do not always develop directly to adulthood upon invasion of man. In West Bengal, India, arrested development appears to be a seasonal phenomenon which results in (i) reduction of egg output wasted in seeding an inhospitable environment and (ii) a marked increase in eggs entering the environment just before the monsoon begins.*

In man, arrested development of helminths is generally believed to occur among abnormal parasites, which, depending on the species, may survive for extended periods of time. This phenomenon is recognized clinically as larva migrans. As regards hookworms specifically, the larvae of *Ancylostoma braziliense*, a parasite of dogs and cats, wander without maturation in the skin of man, and recognition of their survival and movement is possible since they leave visible, raised, erythematous tracks. Some larvae become temporarily inactive and drug resistant, and Stone and Willis (*1*) suggest that these enter a dormant state, during which their metabolic rate is decreased. They speculate that man's own species of hookworms may become dormant, but that if this happens, it would be difficult to demonstrate, as visible signs (tracks) do not occur. If such dormancy occurs in normal hosts, the epidemiology of human ancylostomiasis is much more complicated than presently believed.

That the parasitic larvae of one of the anthropophilic species of hookworms, *Ancylostoma duodenale*, have the capacity to become latent and subsequently resume development in man is suggested by our recent investigations of ancylostomiasis in West Bengal, India. Larvae acquired during the rainy season of one year appear to remain dormant until just before the monsoon of the following year, when they resume development and mature.

A seasonal investigation of the variation in egg counts in Indian villagers was conducted in Hooghly District, West Bengal, in 1969–1970 (*2*). The monsoon began in June, but the fecal egg counts had already shown a sharp, statistically significant ($P < .005$) increase during the round of stool examination begun in late April and completed in early June (Fig. 1A); in fact, the most marked increase occurred at this time. Were this rise attributable to a proximal increase in exposure, then, considering the 6- to 8-week period required for maturation within the host, the density of infective larvae on the soil surface should have increased sharply in March and April. A parallel investigation of larval abundance and distribution (*3*), based on Beaver's gauze-pad technique (*4*), recovered *no* larvae in March, although larvae were recovered by comparably intensive sampling during the rainy season (Fig. 1B). Meteorological data (Fig. 1C) support Chandler's opinion (*5*) that the premonsoon showers do not provide enough moisture to sustain infective larvae in sufficient abundance to cause a marked increase in the rate of acquisition. The showers of March fell on parched soils which had been losing water since November; furthermore, in March the mean maximum air temperature attained 34°C and the mean weekly daytime temperature at the soil surface ranged from 30° to 39°C. The impact of 30.5 mm of precipitation was inconsequential, and the failure to recover larvae at this time is explicable.

The inference that the premonsoon increase in worm burden reflects transmission which occurred in the previous year is supported by seasonal trends in the rate at which children converted from negative to positive for hookworms (*6*). Some conversions occurred throughout the year (Fig. 2), but a statistically significant increase in the rate was observed only in the round of stool examination conducted in November/December 1970 ($P < .05$). This suggests that transmission was at a maximum at the end of the monsoon season, and had most of the larvae acquired at this time developed directly to adulthood, a rise in egg count concurrent with the increase in conversion rate should have occurred. However, egg counts did not increase at this time (Figs. 1 and 2). Furthermore, the failure of the conversion rate to attain a statistically significant peak concurrently with the premonsoon rise in egg count supports the inference, based on our soil sampling for infective larvae, that this increase cannot be attributed to a marked increase in transmission in March and April.

The course of a self-induced infection in one of us provides additional evidence for the occurrence of arrested development. He (C.G.D.) was exposed percutaneously to infective larvae of *Ancylostoma duodenale* on 24 June 1970. Seven minutes after application, an intense pruritus was experienced over each of two application sites; after

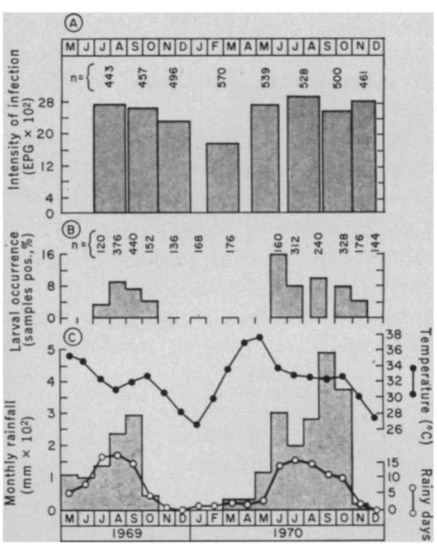

Fig. 1. Seasonal relationship between the intensity of hookworm infection, occurrence of infective larvae, and meteorological conditions. (A) Intensity of infection in terms of eggs per gram of feces (EPG) counted by the Martin-Beaver technique (*14*). The number of people examined per round of stool examination (n) is given over each bar. (B) Occurrence of infective larvae on the soil surface (percent of samples positive). Samples were collected by Beaver's (*4*) damp gauze-pad technique. (C) Total monthly rainfall, number of rainy days, and monthly mean maximum air temperature. A rainy day is defined in accordance with Chandler (*5*) as having at least 0.1 inch (2.54 mm) of rainfall.

30 minutes, numerous macules were observed. Between the 7th and 14th days after infection, a nonproductive cough and pharyngitis were experienced. These events indicate that penetration and normal larval migration had occurred. Beginning in August 1970, when the normal prepatent period should have elapsed, weekly stool examinations were begun, but these remained negative for hookworm eggs through September. The examinations were terminated except for one which proved negative on 28 November 1970, when the subject left the endemic area. Thus, after 22 weeks, or three times the normal prepatent period, ovipositing worms had not yet developed as judged by fecal examinations. Four months later, when fecal examinations were resumed, hookworm eggs were found and weekly egg counts increased significantly from 1640 to 3250 eggs per gram of feces (EPG) during the first 2-month period ($P < .025$). Thereafter, counts continued to increase gradually over the next 3 months to about 5500 EPG, where they leveled off. That some other, later exposure could account for this infection and rise in egg count is unlikely. Considering that the egg counts imply invasion by more than 100 larvae, and that the pruritus experienced at the time of known exposure was severe, it is improbable that a subsequent, accidental exposure could have occurred and escaped notice.

Added support for our conclusions is available in the literature. Maplestone's (*7*) investigations of the dynamics of hookworm infection in eastern India demonstrated a seasonal pattern of variation in egg counts similar to those which we have observed. Furthermore, in a group of convicts imprisoned at the end of the monsoon season, and thereby protected from reexposure, a postmonsoon decline and an enigmatic premonsoon rise in egg count were observed. In five experimentally induced infections with *A. duodenale* in prisoners, Kendrick (*8*) observed that fecal egg counts increased for 15 to 18 months. Throughout this period, and, in fact, through 5 years, more than 140 prisoners who served as controls remained uninfected, demonstrating that extraneous hookworm infections were not acquired in the jail. The rise in egg counts in both prison investigations can be attributed to either a long-term increase in the fecundity of worms (*5*), or, alternatively, to a cumulative increase in the number of females maturing. Other data reported by Kendrick favor the alternative interpretation and, hence, support our hypothesis. In some prisoners, the infections were terminated by chemotherapy before they had run their course. Worm recoveries from these subjects show a statistically significant positive correlation between the number of adult worms recovered and time over a 12-month period ($r = .71$; $P < .025$). Moreover, by anthelmintic treatment and purgation of hospitalized patients, Brumpt and Sang (*9*) recovered *A. duodenale* larvae which had been arrested in their development. They concluded that increases in fecal egg counts of their confined patients must be attributed to the maturation of such larvae.

A recent review (*10*) indicates that where seasonal variation in hookworm egg counts has been demonstrated, both species of human hookworm, *A. duodenale* and *Necator americanus*, occur; in areas where the evidence is to the contrary, only *N. americanus* occurs. This suggests that perhaps only the former becomes arrested in development during the dry season, subsequently resumes development, and causes the premonsoon rise in egg count. Seasonal change in the proportion of *A. duodenale* to *N. americanus* eggs in feces provides evidence for this inference. From September 1969 through December 1970 cultures were made from each positive stool collected from the children under observation, and from these cultures infective larvae were harvested and differentially counted (*11*). These data showed that *A. duodenale* as a percentage of the total larvae produced decreased from September/October 1969 to March/

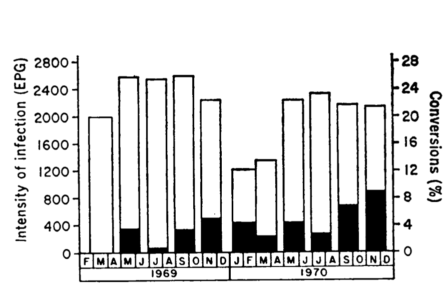

Fig. 2. Seasonal relationship between conversion from negative to positive for hookworm ova in feces and intensity of infection in children. Solid bars show the conversion rate, that is, the percentage of those found negative in one round who were positive in the following round of examination (thus the absence of a solid bar in the first round denotes no data, and should not be construed to mean that there were no conversions). The open bars show the mean egg count (EPG). The premonsoon rise in egg count is statistically significant ($P < .005$).

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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3. A focal method of sampling the soil surface for infective larvae was chosen both to identify the season of transmission and to estimate the frequency and intensity of contact with larvae. As most transmission of hookworms occurs when man becomes stationary while defecating at loci previously polluted with feces, the soil in an annular site 30.54 cm wide around freshly passed stools was sampled for infective larvae; the width of the annulus was based on the length of the average adult villager's foot. Within each annular site, 516 cm^2 (10.6 percent) of the surface area was sampled with gauze pads.
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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