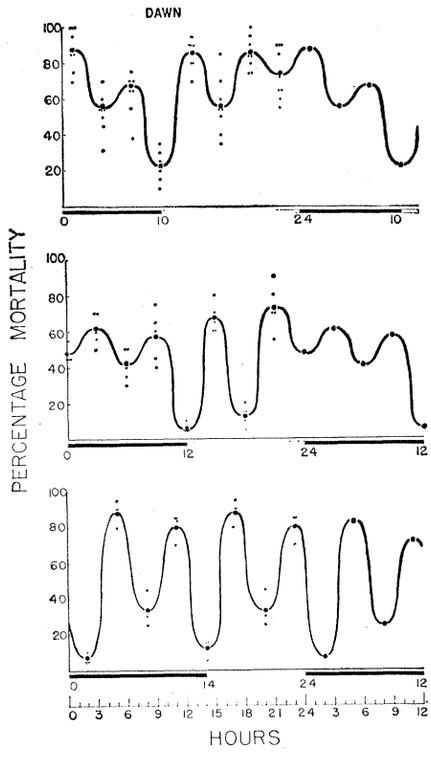

Fig. 1. Susceptibility of the adult boll weevil to methyl parathion as related to time of day of treatment. Curves connecting the large black dots are drawn through the mean percentage mortality produced by each series of treatments. The beginning of the dark period is designated as hour "0"; however, for clarity a second dark period has been plotted. "Clock time" for the dawn of each photoperiod was as follows: 14-hour photophase 6:00 a.m.; 12-hour, 7:00 a.m.; and 10-hour, 9:00 a.m.

groups of weevils were exposed to the same dose of methyl parathion for a 15-minute period. At the end of this period, the weevils were removed from the treated surfaces and returned to the original photoperiod. Treatments made at night were accomplished with the aid of a red photographic light. The insecticide treatments were repeated three to eight times on new groups of weevils over a period of several days. Mortalities were based on records made 24 hours after treatment and were adjusted, by Abbott's formula (*5*), for the small percentages of natural mortality occurring in the untreated control groups.

The weevils showed a definite daily rhythm in their susceptibility to methyl parathion (Fig. 1). Under each photoperiod, the peaks of resistance or susceptibility recurred approximately every 6 hours. Although the basic pattern of the response curve appeared to be the same under each of the photoperiods, the amplitudes of the curves apparently were influenced by the length of the photophase, the daily differences in degree of susceptibility becoming greater as the light periods were shortened. Thus the most dramatic difference occurred under the photoperiod with a 10-hour light cycle. Here the same dose of methyl parathion killed approximately 10 percent of the weevils treated at dawn but almost 90 percent of those treated only 3 hours later.

There also was evidence that the rhythm was entrained to the daily photoperiod. In all photoperiods, the weevils showed a great degree of resistance to methyl parathion at dawn (the beginning of the light period). This has more significance when one realizes that the experiment was conducted so that the clock time of dawn, or lights on, of each photoperiod was as follows: 14-hour photophase, 6:00 a.m.; 12-hour photophase, 7:00 a.m.; and 10-hour photophase, 9:00 a.m. Thus doses of methyl parathion applied at the same clock time, 6:00 a.m., killed slightly more than 20 percent of the weevils entrained to the 14-hour photophase, but nearly 80 percent of those entrained to the 10-hour light cycle. Under these photoperiods, the 6:00 a.m. treatment occurred at dawn of the 14-hour photophase but 3 hours before the dawn of the 10-hour light cycle. This seems to be sufficient proof that it was not the clock time of the day at which the insecticide was applied that was important in determining the susceptibility of the boll weevil to methyl parathion. Rather, it was the phase in the daily rhythm during which the insect was exposed to the toxicant that was important. And this, in turn, apparently was related to the time of occurrence of dawn in each of the photocycles tested.

The cause for this rhythm of susceptibility to the toxicant is not known. Because methyl parathion is a powerful inhibitor of cholinesterase, the results of our study may reflect a cycling of the levels of this enzyme in the boll weevil. However, the daily activity patterns of the insect, or differences in rates of penetration, or absorption, or translocation of the insecticide to the appropriate sites of action may also be important.

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15 April 1964

Photoperiodic Reversibility of Diapause Induction in an Insect

Abstract. *The diapause of the pink bollworm is under photoperiodic control. Diapause is prevented when the dark phases of the daily photoperiod are 8 to 10 hours in duration. If the dark period is extended to 12 hours, diapause is induced. Intercalation of 8- or 10-hour nights may reverse the diapause induction caused by exposure to photocycles having 12-hour dark phases. The 10-hour night was much more effective in reversing induction than the 8-hour night. The intensity of diapause, in part, appeared to be dependent on the previous photoperiodic experiences of the test animals.*

The larval diapause of the pink bollworm, *Pectinophora gossypiella* (Saunders), is under photoperiodic control and occurs when nights are 11 hours or longer (*1*, *2*). Moreover, diapause caused by exposure of the early larval instars to long nights can be reversed for the most part by subsequent exposure of the late instars to short nights. Larvae so treated usually proceed to pupation without any delay in development. This clearly suggests that early inhibition of the endocrine processes controlling development may be released later by the appropriate stimulus. The present study was designed to help elucidate the action of photoperiod on the endocrine system.

Pink bollworms were reared on a cottonseed meal diet fortified with 1 percent Wesson Oil (by weight) (*1*). Newly hatched larvae were placed on the diet and exposed to particular photoperiods at 27°C. Each treatment was replicated three times, and 50 larvae were tested each time. The larvae were held under the test photoperiods for 16 days, long enough for development to proceed well into the last larval instar. At the end of this period, the larvae were transferred to

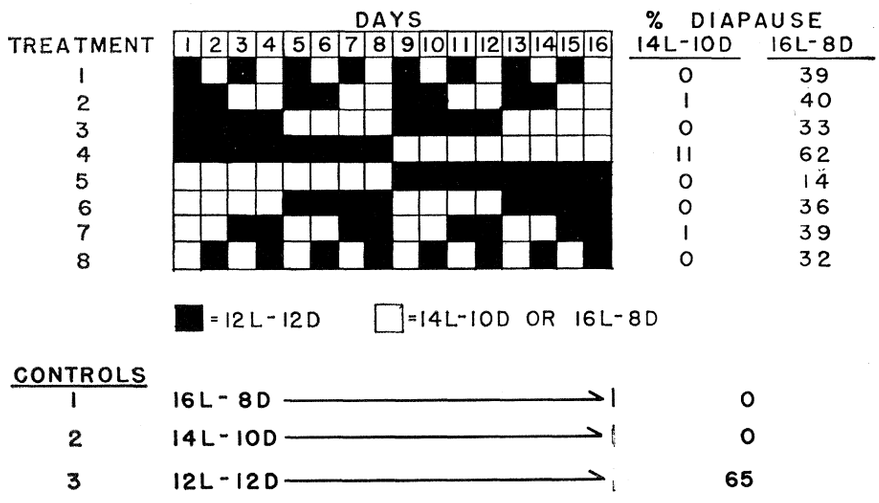

Fig. 1. Effect of intercalation of 14:10 or 16:8 photoperiods with a 12:12 regimen on the induction of diapause. In each treatment, the 14:10 and 16:8 photoperiods were used separately, but in the same type sequence, to oppose the 12:12 photocycle. Each photoperiod was maintained continuously in the controls. *L*, light; *D*, dark.

a photoperiod regimen of 14 hours of light per day and held on that schedule for an additional 24 days. Larvae which had not pupated at the end of the 24-day holding period were considered to be in diapause. In addition, the intensity of diapause in larvae from four treatments to be described was determined by holding the insects until they had either pupated or reached 4 months of age.

Larvae reared under daily photoperiods of 14 hours of light and 10 of dark (14:10) or 16 hours of light and 8 of dark (16:8) did not diapause (Fig. 1). By contrast, exposure to 12 hours of light and 12 hours of dark (12:12) caused 65 percent of the larvae to diapause.

The 14:10 and 16:8 photoperiods also were used in opposition to the 12:12 regimen. Larvae were transferred from one photoperiod to the other according to the scheme in Fig. 1. Sixteen different sequences of photoperiods were employed. In all cases, the larvae received eight photocycles of 14:10 or 16:8 (diapause-preventing) and eight of 12:12 (diapause-inducing).

The induction of diapause caused by 12:12 photoperiods was almost completely reversed, or nullified, by exposure to the 14:10 regimen. In fact, under this arrangement of treatments, there was only one regimen in which more than 1 percent of the larvae were able to diapause—namely, treatment 4. Here, the larvae were exposed to eight consecutive 12:12 photoperiods before being placed in 14:10. Eleven percent of these individuals diapaused. This number of successive early inductive 12:12 photoperiods was sufficient in this percentage of the population to inhibit the endocrine processes controlling development to the extent that they were not activated by later exposure to the 14:10 regimen. The diapause thereby induced was not of a very intense nature. These results show that the endocrine centers do not have to be activated daily for development to proceed without diapause.

The 16:8 photoperiod was much less effective than the 14:10 in reversing induction caused by the 12:12 regimen. These results were unexpected since the data presented for the 14:10 and 16:8 controls indicated that these photoperiods were equally effective in preventing the diapause of the pink bollworm. The difference in the efficiency of the two photoperiods was demonstrated best by the results obtained in treatment 4. Here, the intercalation of the 14:10 photoperiod with one of 12:12 prevented diapause in 89 percent of the population, whereas the intercalation of the 16:8 regimen prevented diapause in only 38 percent of the individuals. The percentage of diapause larvae produced by the 16:8 treatment was approximately the same as that produced by continuous exposure to a 12:12 photoperiod (control 3). In other treatments, however, the 16:8 photoperiod was partially effective in reversing induction caused by the 12:12 cycles. In these, the intercalation of the 16:8 regimen reduced the percentages of diapausing larvae by approximately 40 to 50 percent of that which might have been expected under the 12:12 cycle alone.

The 12:12 photocycles applied during the last 8 days of treatment were relatively inefficient in inhibiting prior activation of the developmental processes. Thus, in treatment 5, early exposure to the 14:10 photoperiod was sufficient to allow all the insects to develop without diapause, and all but 14 percent of those given early ex-

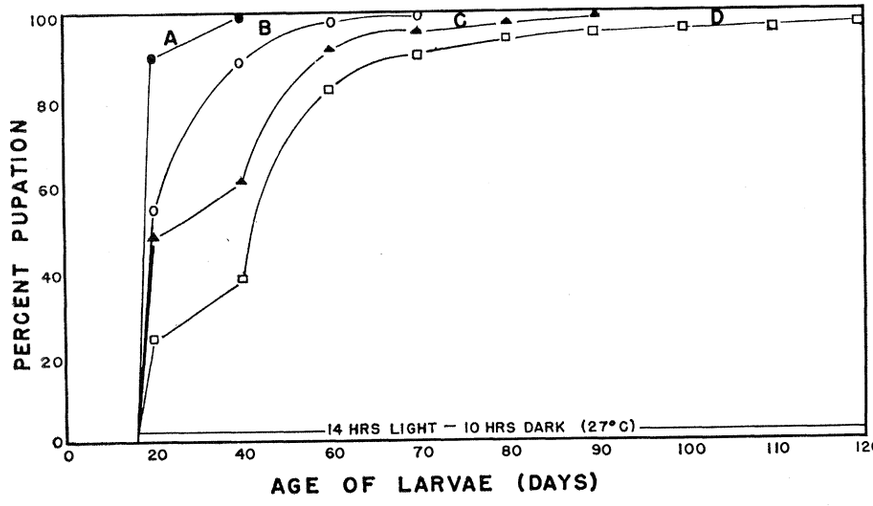

Fig. 2. The cumulative rate of pupation of larvae produced under certain photoperiodic sequences. On day 17, the larvae were placed under the 14:10 photoperiod at 27°C. Nondiapausing larvae in each group had pupated by the end of day 20, thus, records for diapausing larvae begin at day 40. Treatments are identified by reference to Fig. 1 as follows: Curves *A* and *B* were calculated from treatments 1 and 4, respectively, with 14:10–12:12 sequences; curves *C* and *D*, from treatments 1 and 4, respectively, with 16:8–12:12 sequences.

posure to the 16:8 regimen. However, early exposure to the 12:12 photocycle apparently was able to produce a certain degree of inhibition of the endocrine centers; otherwise, the intercalation of the 16:8 photoperiod would have produced sufficient activation of these centers to allow all the larvae to proceed to the pupal stage without diapause.

Larvae produced under treatments 1 and 4 with both the 14:10 and 16:8 photoperiods were observed until they had either pupated or reached 4 months of age. Most of the nondiapausing larvae had pupated by the time they were 20 days old. As shown in Fig. 2, the various photoperiodic regimes apparently had no effect on the time required for these individuals to achieve the pupal stage.

The rate of pupation of the diapausing larvae is shown by records made between the 40th and 120th days. These observations provided a measure of the differences in the intensity of diapause produced by the various photoperiodic exposures. The few diapausing larvae produced in treatments having 14:10 photoperiods proved to have a less intense diapause than those produced under regimes having 16:8 cycles. The treatments which produced the greatest percentages of diapausing larvae also caused the most intense state of diapause. Therefore, it appears that the intensity of diapause is dependent, at least partially, on the quantity and quality of the inductive stimuli previously experienced by the insect. However, because within each treatment group, there was a considerable difference in the rate of pupation of the various individuals, the genetic factors taking part in the diapause phenomena should not be ignored.

The manifestations of larval diapause—cessation of growth, atrophy of the gonads, increase of fat, and lowered respiration—are indicative of an endocrine deficiency (*1*, *3*). Research by Williams (*4*) on pupal diapause has demonstrated that the primary cause of these happenings is a failure of the brain to supply the hormone required to activate the endocrine organs and, more particularly, the prothoracic glands. Also, it has been found in certain species that the endocrine processes are under environmental control (*3*, *4*). Undoubtedly, the diapause of the pink bollworm is provoked when the photoperiod regimen inhibits these endocrine mechanisms.

The full release or activation of the forces controlling growth and development of the pink bollworm apparently is dependent mainly on a night of critical length (*2*). Under this viewpoint, we now see that nights of different length may have different action. This allows one to make certain deductions concerning the dynamics of the system. Although photoperiods having nights 8 or 10 hours long, when given in continuous sequence, appear to be equally effective in preventing diapause, an 8-hour night does not have the force of a 10-hour night in activating previously inhibited endocrine processes. This leaves the impression that the forces causing continued development without diapause gain in strength with an increase in night length from 8 to 10 hours. We also know, however, that as the night increases from 10 to 12 hours, the system undergoes a complete reversal from one that prevents diapause to one that causes it. Thus, it seems, with regard to the length of night, that 8 hours is just barely sufficient for development to continue without diapause, 10 is near optimum and 12 hours is too long.

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14 April 1964

Rectal Glands of Marine and Fresh-Water Sharks: Comparative Histology

Abstract. *The rectal glands of elasmobranchs perform the function of salt-excreting organs. These glands are smaller and show regressive changes in specimens of the bull shark,* Carcharhinus leucas *found in fresh-water environment, compared with specimens of this and other species from a marine habitat.*

The physiological significance of the rectal gland was obscure for a long time, though some morphological investigations were made (*1*). Recently the excretion of sodium chloride was reported as the main function of the gland (*2*). Elasmobranchs are marine fish, but a few adapt to the fresh-water environment (*3*). The bull shark, *Carcharhinus leucas* (Müller and Henle), is common in the Gulf of Mexico and Atlantic Ocean, but it also lives in Lake Nicaragua (*4*) and some fresh-water situations in the United States (*5*). This report deals with morphological differences in the rectal glands of marine and fresh-water *C. leucas*.

The rectal glands investigated were from six marine and ten fresh-water specimens of *C. leucas*. Also, for reference, the rectal glands were investigated in the following additional species of marine sharks: blacktip shark, *C. limbatus* (two specimens), tiger shark,

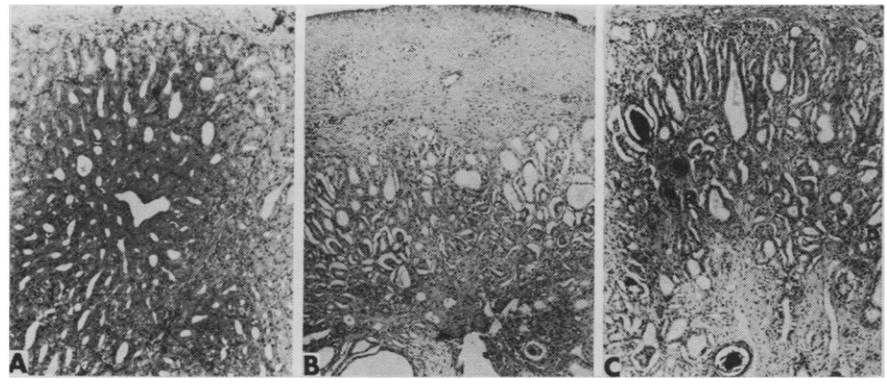

Fig. 1. Rectal glands of bull sharks, *Carcharhinus leucas* (hematoxylin and eosin; × 114). *A*, Marine shark, female (No. 11 in Table 1); *B*, fresh-water shark, male (No. 2 in Table 1); *C*, fresh-water shark, female (No. 3 in Table 1).