SOME INTERACTIONS OF SERRATIA MARCESCENS, NUCLEOPOLYHEDROSIS VIRUS AND BLEPHARIPA PRATENSIS [DIP. : TACHINIDAE] IN LYMANTRIA DISPAR [LEP. : LYMANTRIIDAE]¹ (²)

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The entomopathogens Serratia marcescens Bizio and nucleopolyhedrosis virus were each fed alone and in combination with the parasite Blepharipa pratensis (MEIGEN) to 4th-instar gypsy moth, Lymantria dispar, (L.) larvae. At LD₉₀ for NPV, the presence of the parasite enhanced polyhedrosis about 30 %, but the total number of gypsy moth larvae and pupae killed (85 %) was not significantly different from the number killed by the parasite alone (93 %). When the parasite was combined with S. marcescens, a strain non-pathogenic in L. dispar, total mortality was not significantly different from that in insects exposed only to the parasite (89 and 86 %, respectively), but parasite survival was reduced about 12 %. However, deaths not attributable to the parasite could not be ascribed to the bacterium either.

Many surveys have been made to determine the causes of mortality in gypsy moth populations. Although there have been many estimates of which cause is most important, the primary cause of death is often unknown or indeterminate (CAMPBELL, 1967 ; CAMPBELL & PODGWAITE, 1971). Usually, each agent was examined and evaluated individually as though it operated independently.

But, pathogens do not necessarily operate independently. CAMPBELL (1963) showed that the incidence of Sarcophaga spp. was related directly to injury done to pupae by species of Ichneumonidae. REARDON & PODGWAITE (1976) found positive correlations between the incidence of nucleopolyhedrosis (NPV) and the presence of Apanteles melanoscelus (RATZEBURG), a hymenopteran, and Parasitigena silvestris (R-D), a dipteran. RAIMO and others (1977) found that A. melanoscelus transmits NPV. We found that a maggot of Blepharipa pratensis (MEIGEN), a dipteran, in a gypsy moth pupa precludes parasitization by Brachymeryia intermedia (NESS), a hymenopteran.

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nopteran (GODWIN & ODELL, 1979). A study has shown that the presence of either P. silvestris or B. pratensis significantly increases larval deaths due to indeterminate or unknown causes (GODWIN & ODELL, in preparation).

The developmental pattern of B. pratensis seems to make it an apt adjunct to gypsy moth pathogens, most of which have a relatively low pathogenicity, except NPV (PODGWAITE & CAMPBELL, 1972). B. pratensis females lay their eggs on leaves, and gypsy moth larvae are infested when they ingest an egg. The egg hatches in the gut, and the maggot penetrates the gut and enters the hemocoel. Within 24 h of ingestion, the maggot invades a longitudinal muscle of the abdomen where it remains until the host pupates. At that time, the maggot begins rapid development and kills the pupa in about 4 days.

The penetration of the gut by the maggot may make it possible for bacteria in the gut to then invade the hemocoel. While the maggot resides in the muscle, the muscle undergoes cell proliferation and nuclear and cellular hypertrophy-tumor-like growth (SHIELDS, 1976). Although these tumors have never been shown to incapacitate the host, they do indicate altered physiology. The altered physiology and trauma of gut/hemocoel passage may increase larval susceptibility to disease.

We studied some interactions of Serratia marcescens and NPV, singly and in combination with B. pratensis in the gypsy moth. NPV is a virulent pathogen, which is usually responsible for the collapse of gypsy moth populations. S. marcescens is found in most gypsy moth populations but is not known to cause epizootics (PODGWAITE & CAMPBELL, 1982; PODGWAITE & COSENZA, 1976). In laboratory culture, however, it can be devastating both to the host and to adult fly.

MATERIALS AND METHODS

Gypsy moth egg masses were collected in Hamden, Connecticut, in January and held at an average of 1°C and 80 % RH until June. Then the eggs were pooled, and the larvae were reared according to the method of ODELL & ROLLINSON (1966), except for the diet, which was the BioServ² modification without an antibiotic. Diet was withheld from newly molted 4th-instar larvae until each ingested a parasite egg and/or the bacterium or virus. This period did not exceed 24 h postmolt. The tests were initiated on 2 consecutive days. On the 1st day S. marcescens and B. pratensis were fed singly and in combination. The next day NPV and B. pratensis were fed singly and in combination. One hundred larvae — 5 replicates of 20 larvae each — were used for each treatment and control group on each day.

B. PRATENSIS

Adults were collected in various parts of northeastern United States. Eggs were collected and fed to gypsy moth larvae according to the techniques of ODELL & GODWIN (1979). Each larva was presented 1 egg and watched closely to determine when ingestion took place. Larvae that were fed both a fly egg and a microorganism received the microorganism within 30 min after ingesting the egg.

NPV

NPV suspension was prepared by obtaining polyhedral inclusion bodies (PIB) from NPV-infected gypsy moth larvae and purifying them by K-rotor zonal centrifugation (BREILLATT et al., 1972). Each larva was fed 1 µl of distilled water that contained $1.75 \times 10^2$ PIB. Tests indicated that this dose consistently resulted in a LD₉₀ for 4th-instar larvae from this egg source. The drop of NPV suspension was pipetted