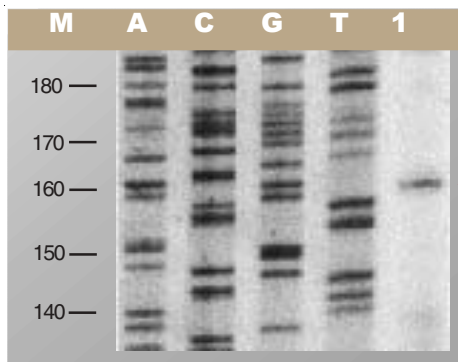


DISRUPTION OF SPRUCE BUDWORM METAMORPHOSIS BY A POLYDNAVIRUS

INTRODUCTION:

Research in this study is aimed at generating novel ideas for the development of innovative spruce budworm (*Choristoneura fumiferana*) control products. More specifically, this study focuses on the endocrine and molecular mechanisms involved in the fatal disruption of budworm metamorphosis by a parasitoid-transmitted virus. The parasitoid in this case is the endoparasitic wasp, *Tranosema rostrale*. Attempts are being made to characterize the disruption mechanisms and identify the regulatory viral genes; the latter may then be used for the genetic manipulation of pest control agents such as CfMNPV, a budworm-specific baculovirus, to improve their efficacy. Parasitization of *C. fumiferana* 5th and 6th instar larvae by *T. rostrale* leads to developmental arrest in the 6th instar host. The ovary of this wasp produces a symbiotic virus called a polydnavirus (PDV), which causes the arrest. Hormonal disruptions induced by the *T. rostrale* PDV have been characterized and a PDV gene whose transcription is very strong in infected 6th instar hosts has been cloned. The recombinant protein has recently been produced for the purpose of conducting bioassays and raising antibodies. Further research will determine if the protein encoded by this gene is responsible for the developmental arrest observed in virus-infected larvae. This protein is suspected of being a translation inhibitor, so physiological effects on earlier instars will be examined as well. A recombinant CfMNPV expressing this new PDV protein will be developed and its effects on larvae, relative to wild-type virus, assessed.



Primer extension analysis performed on total RNA from CxI-injected larvae.

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LOCATION/SITE:

Research has been carried out in the laboratory, and in the field near Quebec City.

RESULTS:

The effect of parasitism by *T. rostrale* or injection of wasp CxI (calyx fluid from ovary; contains PDV) on last-instar *C. fumiferana* 20HE (moulting hormone) titres showed that developmental arrest results from an inability of the host to produce and maintain 20HE at a level sufficient to trigger a molt. Parasitism did not result in abnormally high juvenile hormone levels. A TrPDV cDNA has been cloned and the corresponding transcript was observed to be the most abundant viral transcript in last instar *C. fumiferana* larvae parasitized at the beginning of the stadium. These results suggest that the protein it encodes, termed TrV1, may play an important role in the observed virus-induced pathologies.

CONCLUSIONS:

Endoparasitic wasps have developed various mechanisms to interfere with the metamorphosis of their lepidopteran hosts. Endocrine disruptions associated with the induction of developmental arrest appear to vary as a function of the exact stage at which metamorphosis is blocked. Factors transmitted to the host by the wasp during oviposition appear to be responsible for most of the observed endocrine alterations and the associated developmental arrest. Among these factors, PDVs have received the most attention and hold the most promise for the identification of genes that are instrumental in inducing the observed developmental disturbances. Some genes appear to be good candidates; it remains to be seen whether PDV functions in immune suppression and developmental regulation are associated with the same or separate groups of genes.

MANAGEMENT INTERPRETATIONS:

Since they are endogenous to the budworm ecosystem, the proteins encoded by the viral genes will not likely pose any threat to humans and the environment, and should therefore facilitate registration of recombinant organisms.



SOURCES OF RELEVANT INFORMATION:

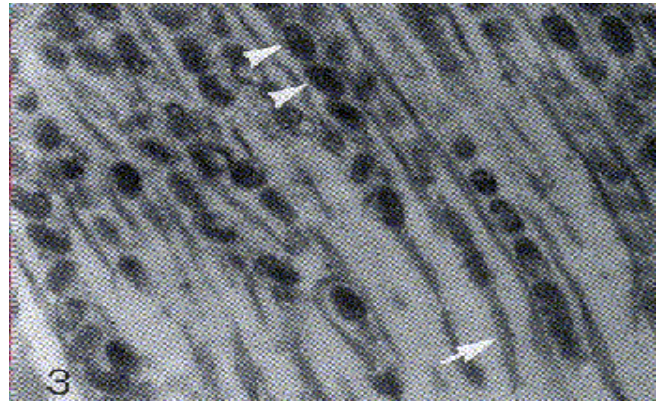
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Electron micrograph showing positioning of *T. rostrale* virions (arrowheads) between chorionic hair-like projections (arrow).

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ISSN 1496-7847

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