学 位 論 文 要 旨

Characterization of a granulovirus isolated from *Spodoptera litura* and its effect on development of the endoparasitoid *Chelonus inanitus*

ハスモンヨトウより分離された顆粒病ウイルスの生物学的特徴と
内部寄生蜂 *Chelonus inanitus* の発育に及ぼす影響

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A new isolate of baculovirus from *Spodoptera litura* (Noctuidae: Lepidoptera) was characterized and its effect on the development of the endoparasitoid *Chelonus inanitus* (Cheloninae: Hymenoptera) was evaluated in this study. The virus was a granulovirus (GV) and closely related to Spodoptera litura granulovirus K1 (SpliGV-K1) strain. This GV showed slow killing phenotype and infected insect gained the body weight more than uninfected host, whereas its phenotype was different from SpliGV-K1 inducing smaller body size of infected host.

C. inanitus is a solitary endoparasitoid, which lays an egg on a host egg, and emerged from host at penultimate instar. Parasitized *S. litura* larvae show precocious initiation of metamorphosis as prepupal behavior, forming prepupal cells, at penultimate (5^{th}) instar before emergence from host as non-parasitized final (6^{th}) instar larvae doing it at prepupal stage. Percentage of emergence, pupation and adult eclosion of the *C. inanitus* was significantly decreased from GV-infected *S. litura* larvae as compared to uninfected host. The lower emergence of the parasitoid larvae from the GV-infected host was due to retarded development of the parasitoid larvae developing in GV-infected host and earlier death of the parasitized and GV-infected host before the emergence of the parasitoid larvae. Body volume of the parasitoid larvae developing in GV-infected host increased but

was significantly lower than that in uninfected host. This may be because the parasitoid larvae could not gain enough nutrition in GV-infected larvae.

All parasitoid larvae emerged from uninfected host showed external feeding on the host cadaver and successfully pupated, whereas significantly less parasitoid larvae emerged from GV-infected host did external feeding on the host cadaver, and it may result in lower pupation rate of parasitoid larvae emerged from GV-infected host. The experiment, in which either infected or uninfected host cadavers were switched on parasitoid larvae emerged from infected or uninfected host, revealed that external feeding of parasitoids was affected both by infection of host cadavers with GV and also by parasitoid developed within GV-infected hosts.

To verify earlier death of GV-infected and parasitized larvae, two hypotheses were examined. One of them was hormonal disorder occurred in parasitized larvae infected with GV, because certain number of the parasitized and GV-infected larvae left the pre-pupal cells and died before emergence of the parasitoid larvae. This abnormal behavior may be due to multiplier effect of parasitism by *C. inanitus* and infection of GV, both of which regulate host endocrinology. Fluctuation of 20-hydroecdyson (20E) and juvenile hormone esterase (JHE) activity were measured to evaluate this hypothesis. In larvae infected with GV-infection alone, no peak of JHE activity was observed, as compared to a broad peak of JHE activity in control (non-parasitized and uninfected) host in the final 6^{th} instar. Titer of 20E was also suppressed in GV-infected host, since a board peak of 20E was observed in control larvae before pupation. Parasitized larvae infected with GV showed higher 20E titers before emergence as that is similar to the only parasitized larvae. Whereas activity of JHE in parasitized and GV-infected larvae was suppressed, that in parasitized larvae showed a small peak. Low activity of JHE may result in relatively higher JH levels, but it is known that JHs were eliminated in hemolymph of uninfected larvae parasitized by *C. inanitus* before emergence.

Second hypothesis was that accelerated GV replication in parasitized host caused the earlier death. Relative amount of viral DNA in total DNA extracted from infected host was significantly higher in parasitized host than in non-parasitized host, and this hypothesis can be accepted. *C. inanitus* has a polydnavirus (PDV) as a symbiont virus encoding genes related to immunosuppression like *viral ankyrin gene* (*vank*). Expression of four *vank* genes from PDV in *C. inanitus* was detected in the later stages of parasitized and GV-infected host. It suggests that the enhancement of the GV replication is due to immunosuppression caused by PDV infection in parasitized host.