A Novel Venom Peptide from an Endoparasitoid Wasp Is Required for Expression of Polydnavirus Genes in Host Hemocytes*

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Maternal factors introduced into host insects by endoparasitoid wasps are usually essential for successful parasitism. This includes polydnaviruses (PDVs) that are produced in the reproductive organ of female hymenopteran endoparasitoids and are injected, together with venom proteins, into the host hemocoel at oviposition. Inside the host, PDVs enter various tissue cells and hemocytes where viral genes are expressed, leading to developmental and physiological alterations in the host, including the suppression of the host immune system. Although several studies have shown that some PDVs are only effective when accompanied by venom proteins, there is no report of an active venom ingredient(s) facilitating PDV infection and/or gene expression. In this study, we describe a novel peptide (Vn1.5) isolated from Cotesia rubecula venom that is required for the expression of C. rubecula bracoviruses (CrBVs) in host hemocytes (Pieris rapae), although it is not essential for CrBV entry into host cells. The peptide consists of 14 amino acids with a molecular mass of 1598 Da. In the absence of Vn1.5 or total venom proteins, CrBV genes are not expressed in host cells and did not cause inactivation of host hemocytes.

Endoparasitoid wasps oviposit their eggs inside the hemocoel of other insects, where their progeny is exposed to host defense reactions, which include humoral and cellular responses. Endoparasitoids have evolved effective mechanisms for successful parasitism that allow them to evade or inhibit the host immune responses (1). Maternal factors are introduced into the host hemocoel, together with eggs, during oviposition. These factors include symbiotic PDVs,1 calyx, and venom proteins (2–4) and may act alone or in conjunction with one or more of the other factors. Inhibition of insect immune responses may involve one or more pathways, such as changes in hemocyte population, down-regulation of phenoloxidase activity, and aberrant hemocyte morphologies (4, 5).

Polydnaviruses associated with hymenopteran parasitoid families Ichneumonidae and Braconidae are referred to as ichnoviruses and bracoviruses (6, 7). The particles are only produced in the female reproductive tract and are released into the oviduct lumen and injected at oviposition into the host hemocoel, where virus genes are expressed in the absence of viral DNA replication (8, 9). Virions are able to enter many host cell types (10, 11). Viral transcripts are detected within a few hours following parasitization, and viral genes are either expressed transiently or persistently (10, 12). The presence of PDVs has been shown to be essential for successful parasitism and development of parasitoids inside their hosts (2, 13, 14). When parasitoid eggs devoid of PDVs are artificially injected into the host hemocoel, the host insect displays a cellular immune response against large exogenous objects that encapsulates the eggs (2).

In endoparasitoids without PDVs, venom proteins, produced by a pair of specialized glands associated with the female reproductive organ, seem to play a major role in host immune suppression and host regulation. For example, in Pimpla hypochondriaca (Braconidae), venom adversely affects the morphology, viability, and immune function of hemocytes of the tomato moth, Lacanobia oleracea (15, 16). Venom proteins also cause developmental alterations in the host. Venom from Aphidius ervi (Braconidae) causes castration of female Acyrthosiphon pisum aphids (17). In Galleria mellonella, venom from Microplitis croceipes reduces larval growth (18). In addition, it has been shown that, in certain cases, venom might have a potent immune-suppressive activity, impairing hemocyte-mediated immune responses before the expression of PDVs or interfering with the activation of the host hemolymph phenoloxidase (18–22).

In some parasitoid systems, PDVs are only effective in conjunction with venom proteins. For example, in Cotesia glomeratus (23) and Apanteles kariyai (24), venom is an essential requirement for successful parasitism. In Cotesia melanoscela, it is reported that venom proteins promote the release of virions into the cytoplasm after uptake by host cells, facilitating the uncoating of PDVs at nuclear pores in host hemocytes (25). In addition, venom proteins enhance virus persistence in the host.

Four genes from Cotesia rubecula bracoviruses (CrBVs) have been found to be expressed in the host Pieris rapae, designated as CrV1–4 (12). They are abundantly expressed in various host tissues, providing active protection by suppressing host cellular responses (26, 27). A 32-kDa protein associated with particles is produced in calyx cells and covers the eggs of the parasitoid, providing passive immune protection while CrBV genes are expressed (28, 29). Several novel proteins have been isolated from C. rubecula venom and found to provide immune protection by inhibiting the activation of host hemolymph prophenoloxidase and melanin formation (20–22). However, there is little known about the functional relationship between CrBVs and venom proteins in this host-parasitoid system.

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‡ The abbreviations used are: PDV, polydnavirus; CrBV, Cotesia rubecula bracovirus; RT, reverse transcription; PBS, phosphate-buffered saline; rpHPLC, reverse phase high pressure liquid chromatography.

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In the present study, we found that venom proteins are essential for the expression of CrBV genes in host cells, although not required for virus entry. A novel venom peptide consisting of 14 amino acids with a molecular mass of 1598 Da (Vn1.5) was found to be sufficient for CrBV gene expression in host cells. In the absence of Vn1.5, CrBV gene transcription was not detected, and hemocyte behavior was not changed, despite the entrance of viruses into the cells.

**EXPERIMENTAL PROCEDURES**

Insect Cultures—The endoparasitoid wasps, *C. rubecula* (Hymenoptera; Braconidae) and its host insect were maintained at 25 °C, on a 14:10 hour (light:dark) photoperiod. *P. rapae* (Lepidoptera: Pieridae) larvae were reared on cabbage plants. Adult wasps were fed with honey-water solution (28).

*Bracovirus Isolation*—CrBVs were purified from newly emerged female wasps as described previously (30). Briefly, isolated ovaries were macerated in PBS (138 mM NaCl, 2.7 mM KCl, 1.47 mM KH2PO4, and 7.3 mM Na2HPO4, pH 7.4) by using micro-scissors. The suspension was centrifuged at 750 × g for 5 min to exclude large cellular debris. The homogenate was then passed through a 0.45-µm syringe filter (MiniStar®) and centrifuged at 15,800 × g in a desktop centrifuge for 20 min. The pelleted virus particles were resuspended in PBS.

**Reverse Phase High Pressure Liquid Chromatography—Venom reservoirs** from 50 female *C. rubecula* wasps were dissected and disrupted in PBS by micro-scissors to release venom proteins into the buffer solution. Reverse phase high pressure liquid chromatography (rpHPLC) of samples was carried out in a Hewlett Packard 1090 liquid chromatograph. The crude venom sample was loaded onto a Vydac reverse phase C18 column and eluted at the flow rate of 0.2 ml/min using a gradient of 5–100% of buffer B (0.04% trifluoroacetic acid in 70% acetonitrile) against buffer A (0.05% trifluoroacetic acid in water) over 82 min. The collected fractions were detected by absorbance at 214 nm, and protein fractions were collected manually. For in vitro hemocyte infection assays (see below), individual fractions were vacuum-dried and dissolved in sterile water.

**Mass Spectrometry**—To determine the molecular mass of venom peptides, electrospray ionization mass spectrometry was carried out by using a PE ScieX API-100. Generally, an amount equivalent to ~7 pmol of purified protein was directly infused, and the resulting raw data were analyzed using the BioMultiView 3.1.1 software.

**Peptide Sequencing**—Approximately 100 pmol of the purified peptides from rpHPLC was vacuum-dried, reconstituted in 8 M urea containing 0.1 M NH4HCO3 and 4 mM dithiothreitol, and finally alkylated by the addition of sodium iodoacetate to the final concentration of 10 mM. The sample was acidified with trifluoroacetic acid to stop the reaction. Peptide sequencing was carried out using a Hewlett Packard G1000A protein sequencer.
Peptide Vn1.5 Synthesis.—Vn1.5 was synthesized byAuspep Pty. Ltd. and analyzed by mass spectral analysis for molecular weight and purity. The lyophilized peptide was reconstituted in sterile water. Various concentrations of the peptide were used in CrBV infection of hemocytes (see below).

CrBV Infection of Hemocytes in Vitro.—Fourth instar P. rapae larvae were surface sterilized in 70% ethanol and bled into cold PBS (saturated with 1-phenyl-2-thiourea to prevent melanization) by removing one of the forelegs. Hemocytes were collected by centrifuging the hemolymph at 750 × g for 5 min and removing the serum. The cells were resuspended in 100 µl of HyQ-SFX insect cell culture medium (HyClone) per insect, bled, and transferred into a 24-well tissue culture plate. 200 µl of the cell suspension containing ~500 cells were allowed to attach for about half an hour at room temperature. Purified CrBVs, equivalent of two wasps, were added to each well. In different treatments, total venom proteins, individual peptides, or mixtures of purified peptides were also added to the cells and mixed by gently pipetting up and down. The cells were incubated for an additional 6 h at 25 °C, after which they were collected by resuspending the cells in the medium and centrifuging at 750 × g for 5 min. Total RNA was then isolated from the cells for reverse transcription-PCR or slot blot assays.

DNA and RNA Isolation.—DNA was isolated from cells by homogenization in a Tris buffer containing 10 mM Tris, 10 mM EDTA, and 1% SDS, pH 8.0. Proteinase K was added to a final concentration of 0.25 µg/µl. The samples were incubated at 37 °C overnight and then treated with RNase A at 37 °C for an additional 30 min. The samples were extracted with phenol/chloroform, and then 2 volumes of ethanol and 0.2 volume of 3 M sodium acetate (pH 5.3) were added to precipitate the DNA. The mixtures were incubated on wet ice for 15 min and centrifuged at 15,800 × g for 20 min. Pellets were washed with 70% ethanol, dried at 37 °C for 5 min, and resuspended in 30 µl of water. Total RNA was isolated from hemocytes as described by Ausubel et al. (31).

Reverse Transcription-PCR (RT-PCR)—Two pairs of primers (CrV1-F and CrV1-R (12) and CrV2-F and CrV2-R (32)) specific to the two CrBV genes, CrV1 and CrV2, were used in reverse transcription reactions using avian myeloblastosis virus reverse transcriptase. A total volume of 10.7 µl, 2 µg of extracted RNA plus 0.1 µg of primer was heated at 95 °C for 5 min to denature RNA before being cooled immediately on ice. After that, 3 µl of 5× avian myeloblastosis virus reverse transcription buffer, 0.5 µl of avian myeloblastosis virus reverse transcriptase (9 units/µl, Promega), 0.5 µl of RNasin (40 units/µl) and 0.5 µl of deoxynucleotide triphosphates (15 mM) were added. The reactions were carried out at 42 °C for 1 h followed by 95 °C for 5 min to inactivate the reverse transcriptase. The resulting DNA fragments were then used in a PCR amplification by adding 5 µl of 10× reaction buffer, 1 µl of forward primer (0.1 µg/µl), 1 µl of reverse primer (0.1 µg/µl), 0.5 µl of deoxynucleotide triphosphates (15 mM), 2.5 units of Taq DNA polymerase (Promega) and 27.5 µl of H2O. After 5 min at 94 °C, 35 amplification cycles were run, including denaturing at 94 °C for 45 s, annealing at 56 °C for 50 s, and extension at 72 °C for 1.5 min. The final extension at 72 °C was carried out for 10 min. All reaction products were electrophoresed on 1% agarose gels and visualized by using ethidium bromide.

DNA and RNA Slot Blot Hybridization—DNA or RNA samples were blotted onto a nylon membrane (Amersham Biosciences) under denaturing conditions according to the manufacturer’s instructions (Bio-Rad). The blots were prehybridized and then hybridized with 32P-labeled cDNA fragment encoding CrV1. A control blot was probed with a P. rapae 18S ribosomal DNA fragment (r18S) to monitor that similar amounts of RNA had been loaded for each sample.
labeled PCR products encoding CrV1 (~900 bp) and CrV2 (~900 bp) at 65 °C overnight. For RNA slot blots, a P. rapae ribosomal 18S DNA fragment probe was used as a control.

Hemocyte Behavior Changes after CrBV Infection with or without Vn1.5—Hemocytes were collected as described above and resuspended in 120 μl of Hy-Q-SFX insect cell culture medium (HyClone) per insect equivalent. 30 μl of a cell suspension containing ~80 cells was transferred into each well of a multiwell plate and incubated for 10 min to allow the cells to attach. Four treatments were set up by adding the following ingredients to the cell monolayers: 1) purified CrBVs and synthesized Vn1.5; 2) CrBVs and total venom proteins; 3) CrBVs alone; and 4) PBS. The cells were incubated for an additional 6 h at 25 °C, fixed with 4% paraformaldehyde, and observed under a phase contrast microscope. Transmission Electron Microscopy—Hemocyte monolayers were incubated with purified CrBVs plus synthesized Vn1.5 or with CrBVs alone as above for 2 h at 25 °C. The cells were then fixed overnight in 4% glutaraldehyde (electron microscope grade) in PBS, with 4% sucrose at pH 7.2. The cells were washed in PBS plus 4% sucrose (2 changes of solution for 10 min each). The samples were post-fixed in 2% osmium tetroxide (OsO4) for 1 h on a rotator and dehydrated in ethanol and propylene oxide. The samples were embedded in resin, and micrographs were taken by using a Jeol 1010 transmission electron microscope.

RESULTS

Venom Is Required for CrBV Gene Expression—To show whether venom components are essential for CrBV gene expression in host cells, in vitro assays were carried out using P. rapae hemocytes. Specific primers from the CrV1 coding region, which is highly expressed in host hemocytes 6 h following parasitization (12), were used as markers in RT-PCR reactions. When hemocytes were infected with purified CrBV particles isolated from female wasps, no CrV1 transcripts were detected in cells after 6 h (Fig. 1A). However, when CrBV particles were added to cells together with venom fluid, CrV1 was expressed strongly (Fig. 1A). The results were confirmed by three independent repeats of the experiment. In addition, slot blot hybridization, using CrV1 cDNA as a probe, showed that the gene is not expressed in inoculated hemocytes in the absence of venom (Fig. 1B). This indicated that venom components are required for CrBV gene expression of host cells, although they are not essential for CrBV entry into host cells (see below).

rhPLC Separation of Venom Proteins and Bioassays—When crude venom preparations from female C. rubecula endoparasitoids were fractionated on a rhPLC column, more than a dozen peptides and proteins were identified (Fig. 2A). To examine which venom component(s) are involved in CrBV function, we collected 12 main fractions and arbitrarily divided the fractions into three groups (Fig. 2A; a, 1–4; b, 5–8; and c, 9–12), each containing four major fractions. The eluted proteins corresponding to each fraction were vacuum-dried and resuspended in sterile water. Proteins or peptides from each group were mixed together (equivalent to two wasps) and applied together with purified CrBV particles to hemocyte monolayers. After 6 h, total RNA was isolated from the inoculated cells and subjected to RT-PCR using CrV1-specific primers. CrV1 transcripts were detected in inoculated cells when a mixture of the 12 fractions was added together with purified CrBVs (Fig. 3A). However, when a mixture of individual groups was added together with purified CrBVs, CrV1 transcripts were only found in group A and not in the other two groups (Fig. 3B).

Because component(s) required for CrBV infection are in group A, individual fractions from this group were used in a subsequent assay. CrV1 transcripts were found in cells infected with CrBVs in the presence of fractions 1, 3, or 4 (Fig. 3C). All subsequent experiments were performed with fraction 1.

Mass Spectrometry and Peptide Sequence Analyses—The size of the peptide in fraction 1 isolated on rhPLC (Fig. 2A) was determined by mass spectrometry to be 1598 Da and designated Vn1.5. The peptide was also N-terminally sequenced, providing 14 amino acids (Fig. 2B) with the predicted molecular weight matching the mass of the peptide determined by mass spectrometry. The estimated isoelectric point of Vn1.5 is 10.01. The sequence of Vn1.5 was compared with other proteins and peptides in protein databases, but no significant similarity was found.

Attempts to N-terminally sequence fractions 2–4 only produced amino acid sequences in fraction 2. The other two fractions might contain proteins/peptides that are N-terminally blocked. Sequence analysis of fraction 2 produced 14 amino acids (Fig. 2C) with the predicted molecular mass of 1640 Da and a theoretical pI of 6.18. The mass of the peptide was also analyzed by mass spectrometry and determined as 1640 Da, indicating that the sequence contained the full-length sequence. We designated this peptide Vn1.6.

Effect of Synthetic Peptide Vn1.5 on the CrBV Gene Expression—To further confirm the function of Vn1.5 in CrBV gene expression, the peptide was synthesized and used for in vitro
bioassays. Hemocytes were inoculated with purified CrBV particles in the presence or absence of synthetic Vn1.5. To examine possible concentration dependence, three different magnitudes of peptide concentrations—5 μg/ml, 0.5 mg/ml, and 50 mg/ml—were applied. CrV1 transcripts were detected in treated cells at all three concentrations (Fig. 4A). Although the intensity of signals increased slightly with the increase in concentration (Fig. 4), the difference in gene expression did not reflect three magnitudes in peptide concentrations. These results further confirmed that Vn1.5 alone was sufficient to promote the expression of CrBV genes at low concentrations.

To examine whether other CrBV genes were also dependent on the venom peptide for their expression, we used CrV2 coding DNA as a probe. When CrV2-specific primers were used for RT-PCR, CrV2 transcripts were also detected in the infected cells in the presence of 1.5 μg/ml Vn1.5 or total venom proteins equivalent to one wasp (Fig. 5). Conversely, no transcript was detected in cells inoculated with CrBV particles alone.

**DISCUSSION**

In contrast to insect ectoparasitoid-host interactions, where venom has a paralyzing function, venom has a different function in endoparasitoids, where it is mainly involved in alterations of host physiology. In addition, depending on whether or not endoparasitoids produce virus-like particles, such as PDVs, venom may complement or replace PDV functions in host regulation. For example, venom from *P. hypochondriaca* (Brachidae), which seems to lack any virus-like particles, adversely affects the morphology, viability, and the immune function of hemocytes of the tomato moth, *Lacanobia oleracea* (16). In endoparasitoids with PDVs, a combination of maternal factors suppresses host immune responses, and venom proteins play a synergistic role with PDVs (23–25,33). In *C. melanoscela*, total venom was found to promote the release of virions into the cytoplasm of infected host cells and enhanced persistence of the virus in the host (25).

In *C. rubecula*, maternal factors introduced into the body of the host at oviposition are essential for successful completion of parasitism. An immunosuppressive gene (*CrV1*) from *C. rubecula* bracoviruses is involved in the destabilization of cytoskel-
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etin in host hemocytes (12, 26). In addition, a calyx protein (Crp32) on the surface of CrBV particles and eggs provides short term protection against host encapsulation reactions before CrBV genes are expressed (28, 29). When crude venom preparations isolated from female C. rubecula endoparasitoid wasps were separated by rpHPLC, over a dozen proteins were found, some of which are involved in the inhibition of melanization by blocking the activation of prophenoloxidase (20–22).

In the present study, we explored the interaction between venom proteins and CrBV genes with regard to virus entry and gene expression. When P. rapae hemocytes were incubated with CrBV particles in vitro, viral transcripts were only detected in infected cells in conjunction with venom proteins. It is clear that venom is required for CrBV expression in host cells, although electron microscopy observations revealed that venom components were not essential for CrBV entry into host cells. Using various mixtures of venom proteins separated on rpHPLC, we found that a 1586-Da peptide (Vn1.5) was sufficient for the expression of CrBV genes when used together with purified particles in in vitro assays. However, another peptide from C. rubecula venom consisting of 14 amino acids and a molecular mass of 1640 Da (Vn1.6) did not support expression of CrBV genes. Considering that the peptide is similar in size to Vn1.5, this indicates that only specific peptides are able to support the expression of CrBV genes. Vn1.5 is basic and hydrophobic, whereas Vn1.6 is acidic. These or other biochemical properties of Vn1.5 might contribute to its ability to facilitate CrBV expression. In addition to Vn1.5, two other fractions were also found to equally promote expression of viral genes. Whether these fractions contain proteins/peptides that have biochemical properties similar to Vn1.5 that facilitate the expression of CrBV genes remains to be investigated.

Synthetic Vn1.5 peptides in conjunction with purified virus were shown to facilitate the expression of CrBV genes, whereas synthetic peptides or CrBV particles alone were not able to cause behavioral changes in hemocytes. This suggests that calyx-derived virus particles and venom-derived Vn1.5 peptides are both required to express CrBV genes in host hemocytes, similar to natural parasitism (12, 26).

Transmission electron microscopy observations showed that venom proteins, including Vn1.5, are not required for the virus entry, release into the cytoplasm from endosomes, or attachment to the nuclear membrane of CrBVs. This suggests that the peptide might facilitate virus chromatin restructuring, uncoating of genomic DNA at the nuclear pore, or expression of CrBV genes at transcriptional level. Further investigations are required to elucidate the exact involvement of Vn1.5 and perhaps other venom proteins in facilitating the expression of polydnaviral genes. However, this is the first report of the characterization of a venom peptide from an endoparasitoid wasp involved in promoting the expression of polydnavirus genes at the molecular and functional level.

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