Densovirus Oral Infection Targets and Disrupts the Peritrophic Matrix of the Lepidopteran Pest Spodoptera frugiperda

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A crucial step in the pathogenesis of orally infectious viruses is to overcome the gut barrier of their host. In Insects, the gut barrier is composed by a single-layered epithelium lined by a protective chitinous matrix, the peritrophic matrix (PM) that displays functions similar to the mucous secretions of the vertebrate digestive tract. The PM thus constitutes a first-line defense that viruses must cross to reach the epithelium and initiate infection. This process is poorly known for most insect viruses, whether they are pathogenic for or transmitted by insects. Here we address this issue by investigating the lectin-like activity of an insect parvovirus on the digestive tract of a caterpillar host. The Junonia coenia densovirus (JcDV) is orally infectious and pathogenic for caterpillars of the pest Spodoptera frugiperda. Upon ingestion, the naked capsids of JcDV rapidly concentrate on the PM suggesting their strong affinity for glycans. Our results showed that JcDV interaction with the PM is mediated through its affinity for carbohydrates including GlcNAc, GalNAc, mannose and fucose. Moreover, we discovered that, in addition to a lectin-like activity, capsids likely carry a yet to be determined activity which triggers a localized disorganization of the PM ultrastructure. Such disruption of the PM, may aid viral particles to go through and reach the epithelium. Finally, our results showed that JcDV early invasion results in an arrest chitin synthesis by midgut cells and changes in their transciptomic profile. In conclusion, this study has revealed that JcDV earliest stage of pathogenesis triggers the dysfunction of the gut barrier, which may help building biocontrol strategies using densoviruses.