TRANSMISSION OF LETTUCE INFECTIOUS YELLOWS VIRUS IS DETERMINED BY A VIRUS CAPSID PROTEIN MEDIATED VIRION RETENTION MECHANISM IN THE FOREGUT OF WHITEFLY VECTORS

James Ng¹; Angel Chen¹; Greg Walker².

¹Department of Plant Pathology and Microbiology, University of California, Riverside, CA 92521, USA; jamesng@ucr.edu ²Department of Entomology, University of California, Riverside, CA 92521, USA.

Members of the emerging and economically important genus Crinivirus are transmitted in a non-circulative manner by specific whitefly vectors via illunderstood mechanisms. Non-circulative transmission refers to the fact that upon acquisition, virions do not circulate through, replicate, or invade the salivary glands of the insect vector, before they can be inoculated into a plant. Plant infection by criniviruses occurs exclusively in the phloem and virions can only be acquired by the vector during prolonged feeding. Once, acquired, virions can persist in the vector from hours to days, but lose their ability to be transmitted when the vector molts. Among criniviruses, transmission of Lettuce infectious yellows (LIYV) by the whitefly Bemisia tabaci biotype A is best studied. However, little is known about the fate of the virus once it enters the vector. We have been investigating virus-vector interactions associated with the whitefly transmission of LIYV using an immunofluorescent localization approach in which virions or recombinant virus capsid components and reacting antibodies are acquired by live whiteflies. Our studies revealed that fluorescent signals, indicating the retention of virions, were specifically localized in the cibarium or foreguts of whitefly vectors but not within those of whitefly nonvectors. Moreover, the specific retention of virions strongly corresponded with the whitefly transmission of the virus. When four recombinant (r) LIYV capsid components were individually acquired by whitefly vectors, only the recombinant minor coat protein, rCPm, was retained in a significantly higher number of individuals. Notably, a CPm (transmission defective) mutant was defective in specific virion retention, whereas the CPm restored virus showed wild-type levels of specific virion retention and transmission. Taken together, these data suggest that the transmission of LIYV is determined by a CPmmediated virion retention mechanism in the cibarium or foreguts of whitefly vectors.