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Interacciones Moleculares Planta/Virus/Vector

Las enfermedades virales en cultivos vegetales afectan seriamente la producción y calidad de los alimentos. El conocimiento a nivel molecular de las interacciones entre factores de la planta, virus y vector transmisor que determinan la severidad de las epidemias virales es crucial para el diseño de estrategias biotecnológicas que atenúen sus efectos. Nuestro grupo las estudia mediante aproximaciones genómicas, proteómicas y funcionales.

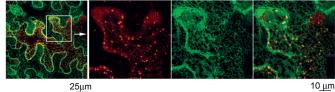
uestro grupo persigue identificar y estudiar interacciones entre factores del virus y de la planta, sus bases moleculares y su implicación funcional, en la hipótesis de que las alteraciones que los virus provocan en la homeostasis celular, a través de sus proteínas y ácidos nucleicos, son responsables en gran medida de la patogenicidad viral. En particular se estudian interacciones entre factores del huésped y determinantes de patogenicidad virales que interfieren en procesos de la planta, entre ellos resistencias específicas a virus, respuestas genéricas a estreses, bióticos y abióticos, así como efectos de alteraciones medioabientales en interacciones compatibles entre virus de RNA y plantas. Para ello se estudian las propiedades a nivel molecular y celular, de interactomas y actividades biológicas mediante ensayos funcionales, de determinantes de patogenicidad como el factor HCPro de potyvirus, 2b de cucumovirus o P25 de potexvirus. Por otro lado, se persigue el conocimiento de las rutas celulares responsables de la manifestación de necrosis sistémica, una sintomatología extrema inducible por determinantes virales (como P25 y HCPro) en el curso de infecciones compatibles. Resultados recientes sugieren que la base genética de la necrosis sistémica se asemeja en algunos aspectos a la de la muerte celular programada inducida por patógenos incompatibles en respuestas de defensa mediadas por genes de resistencia. Para identificar los genes y los circuitos celulares involucrados en la expresión de síntomas, usamos herramientas de transformación genética de plantas y aproximaciones de genética reversa, basadas en silenciamiento génico inducido por virus. Otra línea de investigación persigue el silenciamiento eficiente de genes de insectos vectores de virus mediante RNAs bicatenarios expresados en las plantas de las que se alimentan.

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Live potyviral HCPro dimers appear

as red dots associated to green fluorecing cortical endoplasmic reticulum



that in response to osmotic stress



redistribute as red filaments that co-localize with green fluorecing microtubules

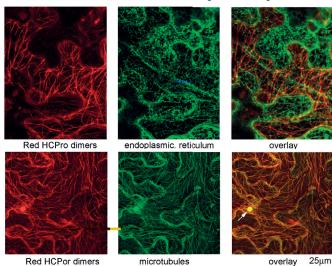


Figura 1 | Figure 1

Visualización en vivo del factor potyviral no estructural HCPro en células epidérmicas de *Nicotiana benthamiana*, en inclusiones y en pequeños puntos asociados al retículo endoplásmico, que en respuesta a estrés osmótico relocalizan hacia el citoesqueleto de microtúbulos. HCPro suprime las defensas del silencimiento antiviral y media la transmisión horizontal del virus por áfidos.

Live visualization of dimers of the potyviral non-structural protein HCPro in epidermal cell of *Nicotiana benthamiana*. HCPro localizes as inclusions and as small dots associated to the endoplamic reticulum, which in response to osmotic stress relocates towards the microtubule cytoskeleton. HCPro suppresses antiviral silencing defenses and mediates viral horizontal transmission by aphids.



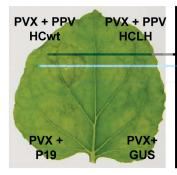
Molecular Plant/Virus/Vector Interactions

Plant virus diseases in commercial crops seriously affect the yield and quality of the food on which we depend. The understanding at the molecular level of interactions between factors from the plant, virus and transmission vector that determine the severity of disease outbreaks is crucial to the design of biotechnology strategies that attenuate their effects. Our group studies them by combined genomic, proteomic and functional approaches.

ur group pursues the identification and study of interactions between factors from the virus and from the plant, their molecular basis and functional significance, under the hypothesis that the alterations that viruses cause to plant homeostasis through its proteins and nucleic acids are to a great extent responsible for viral pathogenicity. In particular we study interactions between host factors and viral pathogenicity determinants that interfere processes in the plant, among them specific antiviral resistances, generic responses to both biotic and abiotic stresses, as well as effects of environmental alterations on compatible interactions between plants and RNA viruses. In this way we study the molecular and cellular properties, interactomes and biological activities in functional assays of determinants such as the potyviral HCPro, the cucumoviral

2b, or the potexviral P25 protein. On the other hand, knowledge is also pursued on the cellular routes responsible for the elicitation of systemic necrosis, an extreme symptom that can be induced by viral determinants (such as P25 and HCPro) during the course of compatible infections. Recent findings suggest that the genetic basis for the systemic necrosis resembles in some aspects that of programmed cell death induced by incompatible pathogens during defense responses mediated by resistance genes. To identify the genes and circuits involved in symptom expression we are using plant genetic transformation and reverse genetic approaches based on virus-induced gene silencing. Another research line pursues the efficient silencing of insect vector genes by the use of double-stranded RNAs expressed from the plants they feed on.

A





В

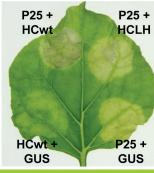




Figura 2 | Figure 2

La expresión de PVX junto con proteínas virales induce necrosis en *Nicotiana benthamiana*. Se infiltraron hojas con combinaciones de construcciones que expresan PVX mas PPV HCwt, PPV HCLH, TBSV P19 o GUS (A), y PVX P25 más GUS, HCwt, o HCLH (B). Las hojas se fotografiaron (paneles izquierdos) y se tiñeron con DAB (paneles derechos) indicativo de producción de H2O2.

Expression of PVX together with viral proteins elicits necrosis in *Nicotiana benthamiana*. Leaves were infiltrated with combinations of constructs expressing PVX plus either PPV HCuH, TPSV P19 or GUS (A), and PVX P25 plus either GUS, HCwt, or HCLH (B), as indicated. Leaves were photographed (left panels) and then stained with DAB (right panels) indicative of H₂O₂ production.

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