J Glycobiol 2017, 6:2(Suppl)
DOI: 10.4172/2168-958X-C1-009

3rd Glycobiology World Congress

June 26-28, 2017 London, UK

N- and O-glycosylation are essential for *Ustilago maydis* virulence

Jose Ignacio Ibeas
Universidad Pablo de Olavide, Spain

Cell wall and secreted proteins are the vehicles for the interaction between fungi and their host. Most of these proteins are glycosylated and this posttranslational modification is essential for their localization and function. Although the role for glycoproteins in these interactions has been studied in fungal animal pathogens for many years, almost nothing is known for phytopathogens fungi. Ustilago maydis has raised as an excellent model for the study of plant-pathogen interactions and its relation with the maize plant is one of the systems in which studies can be tackled from both plant and pathogen perspective. In the last years our group is focused on the study of the role for N- and O-glycosylation during the maize infection for this fungus. We have identified many of the proteins involved in both process, demonstrate that N- and O-glycosylation are both essential for virulence and identified mutants in different stages across the infection process. So we have shown that mutants for Pmt4 (O-mannosyltransferase) are not able to form appressorium, a structure required for plant penetration, mutants for Gls1 (glucosidase I) cannot progress into the plant once the plant has been penetrated and Gas2 (glucosidase II b-subunit) mutants are unable to induce plant tumor formation after a defective progression into the plant. By applying in silico analysis we have identified Pmt4 targets which are essential to complete plant infection. Now we are using 2D gel analysis to identify cytoplasmic, secreted and cell wall glycoproteins involved in the infection process.

joibecor@upo.es

Components from inter alpha trypsin inhibitor protein-glycosaminoglycan-protein complexes mediating the binding to the domain III from envelope protein of dengue virus

Dayron Martin Prieto
Center for Genetic Engineering and Biotechnology, Cuba

Dengue disease can be caused by any of the four virus serotypes of dengue virus complex i.e., DV1-4. Symptomatic dengue disease can vary from very mild symptoms to a life threatening syndrome with hemorrhagic manifestations. The interplay between viral and host factors that determine virus pathogenesis is not well understood. In previous work, a proteomic approach was used to study DV interactome in human plasma. Results pointed to inter alpha trypsin inhibitor complexes (IαIc) as putative interaction partners of DV. IαIc consist of protein-glycosaminoglycan-protein structures, containing serum derived Hyaluronan Associated Proteins (SHAP), covalently linked by a chondroitin sulfate chain (CS) with a serine protease inhibitor named Bikunin. SHAP bind covalently to hyaluronic acid in extracellular matrix, enhancing leukocytes adhesion during acute inflammatory response, a process typically altered in patients with severe dengue. Plasma levels of IαIc are lower in children infected with DV and the magnitude of the decrease correlate with disease severity. In this work, IαIc were highly purified from human plasma and identified by mass spectrometry. IαIc components were separated by chemical hydrolysis of CS and used to further validate and characterize the direct interaction with recombinant proteins comprising the domain III of the envelope protein of DV1-4: DIIIE1-4. Two types of independent interactions were identified; one of low affinity with the light chain Bikunin, presumably via the CS chain of this protein and a stronger interaction was found with SHAP. Our results suggest the direct participation of IαIc in the pathogenesis of DV1-4 infection in humans.

dayron.martin@cigb.edu.cu