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The cellular SUMOylation system as a novel target for anti-influenza therapies

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The recent emergence of the so-called swine origin H1N1 2009 influenza virus, which exhibits high genetic compatibility with H5N1 avian influenza viruses, has provided more favorable conditions for the potential appearance of a new highly pathogenic H5N1 human pandemic virus. Considering the known speed at which mutations conferring resistance to current antivirals get introduced into viral populations, and the likelihood of those mutations being present in a new pandemic strains, it is imperative to develop new broad-spectrum anti-influenza therapies that could be rapidly implemented in the event of such new pandemic. To this end, the targeting of cellular components required for viral replication has gained attention as one of the most promising approaches. Here, we characterize the cellular SUMOylation system as a valuable target for the development of novel anti-influenza therapies. Briefly, through a combination of *in vitro* and tissue culture studies using different cell lines and viral strains, our studies have demonstrated that i) several influenza viral proteins are SUMOylated during infection, ii) modulating the activity of the cellular SUMOylation system before infection affects the normal progression of the influenza life cycle, and iii) influenza infection triggers a global increase in cellular SUMOylation. Altogether, these findings indicate that SUMOylation may regulate the activity of various viral proteins and play an important role for viral fitness and pathogenesis. This is supported by our recent data indicating that SUMOylation affects the activity of both, the viral RNA-dependent RNA-Polymerase and the non-structural protein NS1. In consequence, compounds capable of either, deregulating the SUMOylation of specific viral proteins, or blocking the global increase in cellular SUMOylation observed during viral infection, may prove effective in blocking influenza viral multiplication.

Biography

Dr. Rosas-Acosta obtained a BSc in Microbiology at the Universidad de Los Andes, in Bogota, Colombia, in 1989. After working as a research assistant for 4 years, he performed doctoral studies in Biomedical Sciences at New York University and postdoctoral work in Virology at Texas A&M University and the Texas A&M Health Science Center – College of Medicine. Dr. Rosas-Acosta joined the University of Texas at El Paso as an Assistant Professor in Biological Sciences and member of the Border Biomedical Research Center in 2007. Dr. Rosas-Acosta's lab was the first to report on the relevance of the SUMOylation system for influenza virus infection.