Instituto Universitario de Investigación
Biocomputación y Física de Sistemas Complejos
Universidad Zaragoza

# BIFI-Talks 2021


## Prof. Nuria Verdaguer

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## From structure to mechanisms of Zika virus induced neurodevelopmental disease


Zika virus (ZIKV) infection was declared a global threat for public health due to its association with congenital neurodevelopmental birth defects, such as microcephaly, a rare medical condition characterized by a significant decrease in brain size. ZIKV infection delays cell cycle progression and induces cell death of neural progenitor cells (NPCs), leading to reduced production of neurons. Despite the enormous basic and clinical research efforts, no specific therapy has yet been approved for the treatment or prevention of ZIKV infection, thus a better understanding of its pathogenesis is an urgent need. Our recent studies of the effect of ZIKV proteins on NPC proliferation show that the protein NS5 that is essential for viral genome replication, is also sufficient to alter the mode of division of NPCs. NS5 interacts and depletes primary cilia and centrosome components, causing an atypical non-genetic ciliopathy and premature neuron delamination. While the Methyltransferase (MTase) and RNAdependent RNA polymerase (RdRP) activities of ZIKV-NS5 appear to be dispensable, the Y25, K28 and K29 residues that are involved in NS5 oligomerization, are essential for the localization and interactions with proteins of the cilia base, promoting ciliopathy and premature neurogenesis. In this talk I will summarize our recent findings, exploring these unexpected interactions between ZIKV-NS5 and different cilia/centrosome proteins. These data will shed new light into the mechanisms by which NS5 interferes with the molecular machinery of primary cilia formation, highlighting new potential targets for therapeutic intervention.


***DIA Y HORA:* 7 DE MAYO A LAS 12:30**

***ONLINE:* ZOOM Seminar**

**Link:** https://us02web.zoom.us/j/83011713089