Zika virus inhibits eIF2α-dependent stress granule assembly

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Zika virus is the most recent emerging arbovirus with pandemic potential. It is primarily transmitted by mosquito bites, but may also be sexually transmitted. While adults who contract Zika will rarely have complications of neurological order, the virus imposes a significant risk to pregnant women because of its association with microcephaly and other fetal malformations. The Mouland lab’s efforts are to identify strategies by which the virus counteracts the stress responses elicited by infected host cells and therefore potentially identify new targets for antiviral therapy.

In this publication, the lab observed that cells do not assemble stress granules in response to Zika virus infection and identified a previously unknown strategy by which Zika inhibits stress granule assembly by promoting the dephosphorylation of eIF2α. Because eIF2α signalling is a hub for many cellular stress responses, these findings potentially have implications for diverse virus-host cell interactions. The results in this manuscript demonstrate that Zika virus counteracts host anti-viral stress responses to promote a cellular environment propitious for viral replication.

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