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IDENTIFYING THE MECHANISMS IN WHICH ZIKA VIRUS CROSSES THE PLACENTA AND INDUCES FETAL INFECTION

Meghal Sheth

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Zika virus (ZIKV) is a mosquito borne flavivirus that was first discovered in Uganda in 1947. Between 2007 and 2016, ZIKV has spread to the Americas and ZIKV has been discovered to have a devastatingly profound neurological impact on adults, causing Guillain Barré Syndrome. ZIKV in pregnant women can also induce congenital complications like microcephaly in fetuses. 29% of fetuses in ZIKV infected mothers have shown developmental abnormalities, and no vaccines or therapies have been developed to combat ZIKV infection.

My research sought to understand how ZIKV specifically crosses the placenta and induces infection. The inhibition of furin (FI), a proprotein convertase that has been found to activate viruses like Chikungunya (CHIKV), was assessed due to its ability to decrease the spread of ZIKV. An antimalarial drug, Hydroxychloroquine (HCQ), was also assessed to see if there are therapeutic effects on infected placental trophoblast cells. JEG-3 choriocarcinoma trophoblast cultures were infected with ZIKV and were then co-treated with FI and HCQ. The viral burden was then measured via qRT-PCR. The trophoblast cultures co-treated with the furin inhibitor had no statistically significant decrease in viral burden compared to the ZIKV only treatment, however the HCQ co-treatment did have a significant decrease in viral burden. My findings show that ZIKV infection does not appear to use the furin convertase to induce infection in placental cells. However, HCQ treatment, which blocks autophagy processing required by ZIKV, is effective in limiting infection.

The next step is to better understand the mechanism by which HCQ modulates ZIKV infection and move closer to employing it as a therapeutic avenue to prevent the damaging effects of ZIKV on fetuses.