Investigate How the Zika Virus Infection Induces Microcephaly in Foetuses?

Ayaan Mustafa Siddiqui
Medical Student, University of Exeter, United Kingdom

Abstract:

The association between Zika virus (ZIKV) infection in pregnancy and fetal microcephaly remains incompletely understood despite intense research interest. This review aimed to evaluate proposed mechanisms by which ZIKV might impair neurodevelopment and to assess how convincingly current evidence supports a causal relationship. Studies published since 2005 were identified through structured database searches using predefined inclusion and exclusion criteria, focusing on human, in vitro and animal models that examined ZIKV neurotropism, placental transmission and fetal brain outcomes. Evidence consistently shows that ZIKV exhibits marked neurotropism, preferentially infecting neural progenitor cells, disrupting proliferation and inducing cell death in developing brain tissue. Murine and organoid models further demonstrate cortical thinning and reduced brain volume following congenital exposure, broadly mirroring clinical microcephaly phenotypes. However, heterogeneity in experimental design, viral strains, timing and dose of infection, and outcome measures limits direct comparison across studies and weakens causal inference. Overall, current data strongly suggest that ZIKV can contribute to microcephaly, but they do not yet delineate a single, definitive pathogenic pathway. More integrative, longitudinal research across epidemiological, clinical and mechanistic domains is needed to clarify risk, refine causal models and inform the development of any future preventive or therapeutic strategies.

Keywords:

Zika virus; congenital infection, microcephaly, fetal neurodevelopment, teratogenic mechanisms.