

"WHY IS OUR BABY'S HEAD SMALL?" THE PATHOGENESIS OF MICROCEPHALY RESULTING FROM ZIKA VIRUS AND OTHER CONGENITAL INFECTIONS

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The main pathogens associated with congenital infection and affliction, Zika virus, Cytomegalovirus, Rubella virus, Herpes simplex virus, Herpes varicella virus, and Toxoplasma gondii, all of which are often manifest with microcephaly as well as other stigmata, are briefly reviewed. The trimester of maternal infection and the role of non-primary infection with regard to fetal outcome are noted. The epidemiology and manifestations of infections with these organisms in non-pregnant hosts, the pregnant woman, and in the fetus are reviewed. Pathogen specific characteristics are highlighted, including the differences between Zika virus and the other teratogenic pathogens noted above. The pathology of microcephaly is reviewed in detail with a discussion of the similar neuropathogenesis of the infections caused by these agents including some of the other flaviviruses. The immunopathologic findings for these congenital infections are outlined. Hypotheses which might explain why: some infants are not infected in the presence of primary maternal infection, while others acquire subclinical infection, but still others are severely afflicted are discussed. These hypotheses include: pathogen strain differences, tropism to and susceptibility of various pathogens to developing fetal tissues, the role of various subsets of maternal immunity, and aspects of fetal immune responses.