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Epithelial cells of the amniotic membrane sustain persistent cytomegalovirus infection in the human placenta

Lenore Pereira, Takako Tabata, Matthew Pettit, June Fang-Hoover and Martin Zydek
University of California San Francisco, USA

Human cytomegalovirus (HCMV) is the leading viral cause of birth defects, including neurological deficits, impaired hearing and vision loss. We previously reported that epithelial cells in amniotic membranes of placentas from newborns with intrauterine growth restriction and underlying congenital infection contain HCMV proteins in cytoplasmic vesicles. Here we immunostained amniotic membranes from diagnosed symptomatic congenital infection and preterm deliveries and detected viral proteins and aberrant epithelial cell morphology in cases of virus transmission. Primary amniotic epithelial cells (AmEpC) infected with pathogenic viral strains dysregulated stem cell proteins, and viral replication was gestational age dependent. In contrast to highly differentiated retinal pigment epithelial cells, infected AmEpC failed to make intact viral assembly compartments and instead formed diffusely localized multivesicular bodies, proliferated and survived for months releasing progeny without plaque formation. Explants of amniochorionic membranes infected *ex vivo* upregulated IFN- β in surrounding epithelial cells. Infected AmEpCs produced IL-6 and upregulated the anti-apoptotic proteins survivin and Bcl-xL by mechanisms dependent and independent of the activated signal transducer and activator of transcription 3. Both survivin and Bcl-xL were expressed by control and infected amniotic membranes in utero, suggesting an opportune environment to sustain persistent infection. Interventions that target signaling pathways contributing to HCMV persistence in AmEpC could reduce the viral load and inflammation in the fetal compartment and improve outcome.

Biography

Lenore Pereira is a Molecular Virologist for over 30 years and Professor in the Department of Cell and Tissue Biology at the University of California San Francisco. She has focused the last 16 years on the biology of human *Cytomegalovirus* replication and pathogenesis at the uterine-placental interface. She has published over 120 papers and invited reviews. Recently, her group in collaborative studies with Dr. Eva Harris at the University of California Berkeley identified ZIKV target cells and immune mechanisms that protect the placenta.

Lenore.Pereira@ucsf.edu

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