PhD Project Proposal Presentation

Title: Trophoblast HLA Expression and Maternal NK Cell Regulation during *Toxoplasma gondii* Infection

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Venue: Dr. Shanta Rao Auditorium

Abstract

During pregnancy, the maternal–fetal interface presents a unique immunological environment in which tolerance and defense must be tightly balanced. This immune tolerance is maintained through the specialized expression of Human Leukocyte Antigen (HLA) Class I molecules on extravillous trophoblasts (EVTs), including the classical HLA-C and the non-classical HLA-E, HLA-F, and HLA-G. These molecules interact with maternal immune cells, predominantly decidual natural killer (dNK) cells, to regulate immune responses, prevent fetal rejection, and support placental development. Disruption of this finely tuned system has been implicated in adverse pregnancy outcomes.

Infections during pregnancy can perturb this immune balance and compromise maternal–fetal tolerance. Among these, *Toxoplasma gondii*, a globally prevalent intracellular parasite, is capable of infecting placental tissues and is associated with adverse outcomes such as congenital defects and miscarriage. In addition to direct infection, *T. gondii* is known to modulate host immune signaling pathways. However, its specific impact on trophoblast HLA expression and the downstream consequences for maternal NK cell function at the maternal–fetal interface remains poorly understood. Furthermore, parasite strains differ in their effector repertoires and their capacity to modulate host immune pathways, suggesting that strain-dependent immune regulation may influence pregnancy outcomes.

To address this gap, the present study aims to investigate whether *T. gondii* infection alters trophoblast HLA expression, elucidate the underlying regulatory pathways, and evaluate the functional consequences for maternal NK cell activation. By integrating molecular, mechanistic, and functional approaches, this work seeks to advance our understanding of infection-associated immune dysregulation at the maternal–fetal interface and its role in adverse pregnancy outcomes.