

Ph.D Proposal Presentation

Batch 2025

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Guide: Dr. Kumari Nishi

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Department: Neuroendocrinology

Date: 14.10.2025

Time: 11:45 am – 1:00 pm

Venue: Dr. Shanta Rao Auditorium

Title: Deciphering the molecular switches of spermiation: Insights from the NO-sGC-cGMP pathway

Abstract

The Nitric Oxide (NO)-Soluble Guanylyl Cyclase (sGC)-cyclic Guanosine Monophosphate (cGMP) pathway is pivotal in male reproductive physiology, influencing erectile function, spermatogenesis, and sperm quality. NO, synthesized by nitric oxide synthase (NOS) isoforms (eNOS, nNOS, iNOS) from L-arginine, activates sGC to produce cGMP, leading to vasodilation, inhibition of platelet aggregation, and regulation of blood pressure. In Leydig cells, NO promotes steroidogenesis, crucial for testosterone synthesis. In our previous study (Gaonkar et al., 2024), sub-chronic administration of L-NAME, a NOS inhibitor, induced persistent hypertension and impaired spermatogenesis in male Wistar rats, even after two months of withdrawal. Observations included reduced sperm count, abnormal morphology, elevated intra-testicular testosterone, and spermiation failure, marked by retained step 19 spermatids in seminiferous tubules.

Molsidomine, an NO donor, and Sildenafil, a PDE5 inhibitor that elevates cGMP levels, have shown therapeutic effects on vascular and reproductive functions. However, the mechanistic link between NO-sGC-cGMP pathway dysregulation and spermiation remains unclear. This study aims to elucidate the role of this pathway in Leydig cell steroidogenesis and Sertoli-germ cell interactions, focusing on germ cell migration and differentiation during spermiation. Understanding these molecular mechanisms will clarify how NO deficiency disrupts spermiation and spermatogenesis, providing insights into male infertility associated with NO pathway dysfunction. The proposal will be discussed in detail during presentation.