OPHTHALMOLOGY
COLLEGE OF MEDICINE

SUMMER RESEARCH OPPORTUNITIES FOR UNDERGRADUATE students

FOR APPLICATION YEAR: 2026

PROJECT TITLE: Environmental Timing Cues and Circadian Control of Brain, Behavior, and Disease

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Project Description

Our laboratory investigates how environmental timing cues, particularly light and feeding schedules, influence physiology, behavior, and disease susceptibility. Modern human schedules often diverge from natural light–dark cycles—for example, shift work or indoor occupations expose individuals primarily to artificial lighting. Such misalignment disrupts circadian rhythms, leading to altered sleep–wake cycles, metabolic dysregulation, and changes in systemic physiology. Using mice as experimental models, we examine how environmental manipulations interact with circadian circuits to shape behavior, neural activity, and cellular function. The main research area of the lab includes -

Aim 1: We aim to define how light schedules, feeding paradigms, and behavioral conditioning influence circadian regulation and downstream physiology. Our studies focus on central circadian networks, including the suprachiasmatic nucleus (SCN), which coordinates daily rhythms of activity, sleep, and metabolism. By systematically varying light exposure and feeding schedules, we assess how environmental timing cues affect behavior, neural circuit function, and microbiological composition. We further leverage genetic models to dissect mechanisms of light entrainment. For instance, deletion of the Opn4 gene disrupts intrinsically photosensitive retinal ganglion cell (ipRGC) function, impairing photic entrainment and reducing the ability of mice to maintain stable circadian rhythms. These experiments establish how environmental and genetic factors jointly shape circadian stability, behavioral flexibility, and systemic physiology.

Aim 2. We investigate how local circadian disruption contributes to vulnerability in neurodegenerative disease, focusing on Alzheimer’s disease (AD). Circadian and sleep deficits are among the earliest changes observed in

AD, preceding cognitive decline and exacerbating neuronal vulnerability, amyloid pathology, and synaptic dysfunction. Postmortem studies reveal dysregulation of core clock genes in hippocampal and cortical circuits, even when central pacemaker function is preserved. To model these regional deficits, we developed an inducible mouse line enabling temporal and circuit-specific disruption of Per2 rhythmicity in limbic excitatory neurons. Using this system, we test whether early-life hippocampal circadian disruption exacerbates amyloid stress and triggers transcriptional, glial, and synaptic changes. Parallel analyses examine how limbic neurons respond differently to circadian disruption compared with canonical circadian centers.

Collectively, these studies provide mechanistic insight into how environmental and molecular circadian perturbations influence neural function, systemic physiology, and disease vulnerability. By integrating environmental manipulations with genetic and circuit-level approaches, our laboratory seeks to uncover fundamental principles of circadian regulation and identify strategies for rhythm-aligned therapeutic interventions