

**YOU ARE INVITED TO ATTEND THE
DEFENSE OF THE DOCTORAL
DISSERTATION**

**“CARBONIC ANHYDRASE 1 PREVENTS FERROPTOSIS AND
OPERATES AS A METABOLIC CHECKPOINT THAT REGULATES
MAST CELL DEVELOPMENT”**

By

Krupa Chavan
PhD Biomedical Sciences
Infection, Immunity, and Inflammation Program

Bachelor of Dental Surgery, 2014, Sinhgad Dental School and Hospital, Pune, India

Master of Science in Dentistry, 2020, Rutgers School of Dental Medicine, Newark,
NJ

Thesis Advisor: Dr. Mark Siracusa, PhD
Associate Professor
Department of Medicine

Monday, October 26th, 2026
Cancer Center, G1196
1:00 P.M

Join Zoom presentation
<https://rutgers.zoom.us/j/96124457435?pwd=GY0hkIBQeS9dNBhzizIxxAjE72lqdu.1>
Meeting ID: 961 2445 7435
Password: 019845

ABSTRACT

Mast cells (MCs) are rare tissue-resident innate immune cells that are predominantly located in barrier tissues including the skin, lungs, and gastrointestinal tract. MCs are derived from hematopoietic progenitor cells (HPCs) which migrate from the bone marrow to the periphery where they mature and reside. Mature mast cells are further classified as connective tissue mast cells (CTMC) and mucosal mast cells (MMC) based on their location, granule content and function. They are the first line of defense against pathogens, parasites, toxins, and environmental threats and are key effectors of a complex range of type 2 immune responses via their release of diverse effector molecules. However, when they are dysregulated mature mast cells also drive the development of inflammatory diseases like allergic inflammation, mastocytosis, autoimmune diseases and other mast-cell related disorders. Despite the therapeutic need, a gap in understanding the factors that regulate mast cell development has limited our ability to design novel therapeutics to effectively target mast cells. Our previous studies have identified a population of HPCs that possess mast cell potential and are defined by their expression of the metabolic enzyme carbonic anhydrase (Car)1. Here we show that genetically and pharmacologically targeting Car1 is sufficient to prevent the development of both CTMC and MMC in models of *Trichinella spiralis* parasitic infection, mastocytosis, atopic dermatitis, and food allergy. Mechanistically, Car1 is required to prevent ferroptosis in MCs by regulating cellular metabolism and oxidative stress. Collectively, these findings establish Car1 as a metabolic regulator of mast cell development, highlight its therapeutic potential for targeting pathologic CTMC as well as MMC, and further advance our understanding of mast cell biology and the mechanisms governing lineage fate determination.