RUTGERS HEALTH
School of Graduate Studies
Health Sciences Campus - Newark

Office of Student Affairs and Admissions
SGS at New Jersey Medical School
Rutgers, The State University of New Jersey
185 South Orange Avenue, MSB C-696
Newark, NJ 07103

http://njms.rutgers.edu/gsbs/
p. 973-972-4511
f. 973-972-7148

YOU ARE INVITED TO ATTEND THE DEFENSE OF THE DOCTORAL DISSERTATION

"Metabolic Regulation of Liver ILC1 Immune Responses"

By

Pooja N. Kayala
Infection, Immunity, and Inflammation Program
B.S. University of Mumbai, 2017
M.S. Vellore Institute of Technology, 2019

Thesis Advisor:
Aimee M. Beaulieu, Ph.D.
Assistant Professor and Chancellor Scholar
Center for Immunity and Inflammation
Department of Microbiology, Biochemistry and Molecular Genetics

Wednesday, December 3rd, 2025
Cancer Center, G-1196
10:00 AM

Join Zoom presentation

https://rutgers.zoom.us/j/5540286906?pwd=Ba3L45tO4h3f37bAlf3vuqETJnd1Vl.1&omn=91059082502

Meeting ID: 554 028 6906 Password: 99999

ABSTRACT

The liver plays important roles in host metabolism and immunity, and is a major site of metastatic cancer. The liver is populated by diverse immune cell types, including "helper-lineage" Group 1 innate lymphoid cells (ILC1s) and classic NK cells (cNKs). ILC1s and cNKs are now known to play redundant and non-redundant roles in liver immunity, including defense against pathogens, control of metastatic seeding and tumor growth, and tissue repair after liver injury. Although the liver is a highly metabolic organ, little is known about the metabolic processes that support the distinct functions of liver ILC1s and cNKs.

Here, we show that liver ILC1s and cNKs exhibit distinct metabolic profiles and requirements. Specifically, we find that liver ILC1s preferentially rely on glycolysis rather than oxidative phosphorylation (OXPHOS) to meet basal energetic demands at rest and after activation. In contrast, liver cNKs are highly dependent on OXPHOS, similar to splenic cNKs, consistent with their higher mitochondrial content and activity, as compared to liver ILC1s. The glycolytic preference of liver ILC1s was tissue-specific, as ILC1s and NK cells in other tissues, including the salivary glands, uterus, and lamina propria, all exhibited a greater dependence on OXPHOS than liver ILC1s. Functionally, liver ILC1s required glycolysis but not OXPHOS for IFNγ production, although OXPHOS was important for responses associated with cytotoxicity, including granzyme B expression and degranulation. In line with their glycolytic profile, liver ILC1s imported more glucose than liver cNK cells, both at baseline and after activation. Moreover, inhibition of glucose uptake with the pan-GLUT inhibitor, Glutor, impaired IFNγ production by ILC1s, indicating that active glucose import via GLUT transporters is required for ILC1 effector responses. Transcriptional profiling studies indicate that liver ILC1s and liver cNKs both express GLUT1 and GLUT3, whereas liver ILC1s also express GLUT8. Nevertheless, studies involving transgenic mice indicate that GLUT1 is not strictly required for glucose uptake or IFNγ production by liver ILC1s, possibly due to compensation by other GLUT transporters. Taken together, our data indicate that, unlike liver cNKs, liver ILC1s preferentially rely on glycolysis rather than OXPHOS for basal energy demands and effector functions.