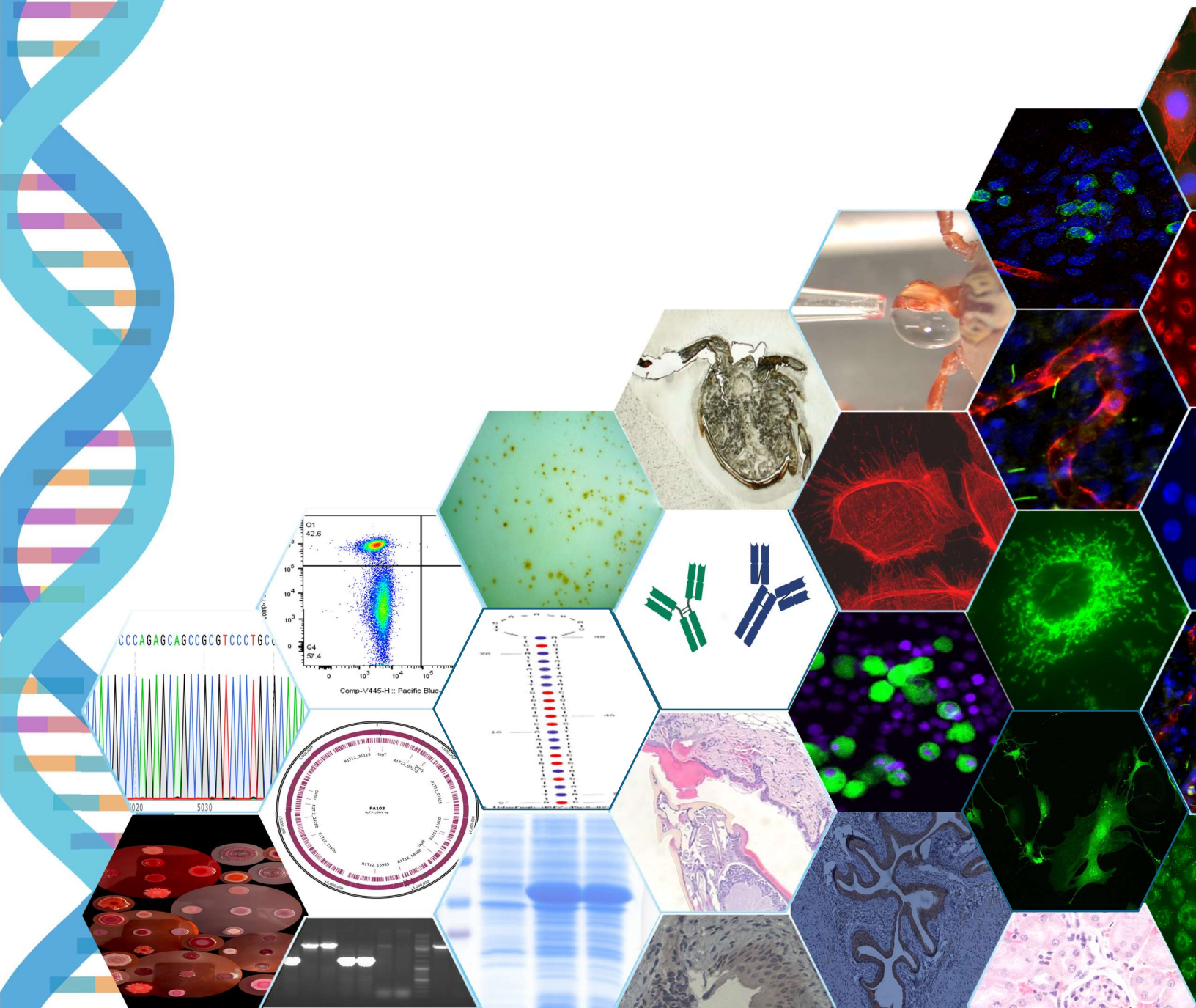
# MICROBIOLOGY AND IMMUNOLOGY RESEARCH SYMPOSIUM 2026

Frederick P. Whiddon College of Medicine
University of South Alabama

# Abstract Book



# 2026 Microbiology and Immunology Research Symposium

*September 25, 2026*

*Terrace Room, Student Center*


12:25 PM Dr. Timothy Casselli, Assistant Professor, Whiddon COM Department of Microbiology and Immunology and M&I Symposium Committee Chairman, gives opening remarks and introduces the symposium keynote speaker.

12:30 PM Keynote Presentation "**More than Meets the Eye: What Fat Cells Taught Me About Disease and Discovery**" by Dr. Jacqueline M. Stephens, Professor and Claude B. Pennington, Jr. Endowed Chair in Biomedical Research, Louisiana State University (LSU) Pennington Biomedical Research Center; Director, Adipocyte Biology Laboratory; and Principal Investigator, Metabolic Basis of Disease Center.

1:30 PM Intermission.

1:45 PM Student Research Presentations begin.

*First Presentation*— Oluwagbenro Adesunloro
*Second Presentation*— Shovon Lal Sarkar
*Third Presentation*— Allen Okine
*Fourth Presentation*— Sarah Macon-Foley
*Fifth Presentation*— Elisa Pizzocaro

3:00 PM Intermission.

3:10 PM Student Research Presentations resume.

*Sixth Presentation*— Manley Hicks
*Seventh Presentation*— Sicily Hardy
*Eighth Presentation*— Brianna Mitchell
*Ninth Presentation*— Hoa Tran
*Tenth Presentation*— Qudus Ojomo
*Eleventh Presentation*— Ahmed Garba

4:30 PM The 2026 M&I Symposium concludes, and Dr. Casselli gives closing remarks. Transition to the USA Faculty Club for reception and awards ceremony.

4:45 PM Reception and Awards Ceremony begins.

6:45 PM Reception and Awards Ceremony concludes.

# Amyloid-β beyond the brain: evidence for local and systemic induction during urinary tract infection

Presenter: Oluwagbenro Adesunloro

Mentor: Allyson Shea, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Amyloid-β (Aβ), generated from amyloid precursor protein (APP), is known for its role in Alzheimer's disease but may also function in the innate immune response. Elevated circulating Aβ has been reported in patients with sepsis; however, whether less severe infections, such as urinary tract infections (UTIs), similarly induce systemic and local Aβ production remains unclear. We hypothesized that UTI increases Aβ levels both locally and in circulation during UTI. To test this, plasma samples were obtained from UTI patients (n = 76) and healthy controls (n = 20) within the USA Health system. Aβ levels were quantified and correlated with clinical parameters. Patients with UTI had significantly higher plasma Aβ40 levels than healthy controls ($P < 0.001$). To assess the associated inflammatory response, cytokines were measured using a multi-analyte flow assay. Caspase-1, IL-6, IL-8, and IL-18 were significantly elevated in UTI patients compared with healthy controls ($P < 0.001$), supporting activation of known innate immune pathways. In parallel, we used an *in vitro* bladder epithelial cell model to assess local Aβ production during infection with the uropathogenic *Escherichia coli* strain CFT073. At 3 hours post-infection, APP levels were significantly reduced compared with uninfected ($P = 0.0059$) and LPS-treated cells ($P = 0.0071$), while Aβ40 levels were significantly increased ($P = 0.0127$ and $P = 0.0055$, respectively), consistent with enhanced APP processing to Aβ during infection. Collectively, these findings identify Aβ40 as a potential innate immune effector produced systemically and locally during UTI. This extends Aβ biological relevance beyond neurodegeneration and suggests a role in infection-associated host defense.

# Differential production of tick saliva microRNAs associated with *Rickettsia* infection

Presenter: Shovon Lal Sarkar

Mentor: Kevin R. Macaluso, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

The Gulf Coast tick, *Amblyomma maculatum*, is a key vector for *Rickettsia parkeri*, which causes spotted fever group rickettsiosis. Ticks transmit rickettsial agents via saliva containing bioactive molecules, including miRNAs, a type of small RNA that regulates biological and molecular processes and varies with the presence of pathogens. Previous studies reported that manipulating miRNAs may influence tick development, feeding, and pathogen transmission. While miRNAs in ticks in the presence of pathogen have been studied, little is known about saliva miRNAs and their potential role. We hypothesize that rickettsial infection induces distinct saliva miRNA profiles in tick. To test this, saliva from 72 ticks infected with *R. parkeri* or *Candidatus* Rickettsia andeanae was analyzed and compared to saliva from 36 uninfected ticks. miRNAs were isolated, quantified, and sequenced, revealing robust profiles in all samples. Downstream analysis identified over 110 confident miRNAs, with 6 upregulated and 5 downregulated in *Rickettsia parkeri*-infected ticks; we will confirm these by RT-qPCR. Notably, we selected Ama-miR-275 and Ama-let-7 for functional analysis using *in silico* methods. Gene Ontology and network analyses predicted roles of miRNAs in metabolism, protein binding, development, and cellular biogenesis in the targeted host. Future studies will employ *in vivo* RNAi assays with anti-miRNAs to elucidate saliva miRNAs' roles in tick feeding and pathogen transmission to the host. Ultimately, this research aims to characterize differentially expressed miRNAs to develop intervention strategies against tick-borne rickettsial diseases.

# Comparison of *Rickettsia* quantification and estimation of bacterial load per plaque

Presenter: Allen Okine

Mentor: Kevin R. Macaluso, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Accurate quantification of *Rickettsia* burden is an essential component to examine infectivity dynamics. The widely used methods for bacterial quantification includes direct microscopic counts with Peroff-Hausser counting chamber, quantitative PCR (qPCR), and plaque assay. The relationship between bacterial burden within individual plaques, plaque size, and its corresponding infectious titer remains poorly defined. The aim of this study is to determine how *Rickettsia* burden within individual *Rickettsia* plaques relates these infectious titers and how this relationship changes with plaques size as the duration of infectivity increases. Individual plaques of varying sizes and incubation period were isolated and evaluated to determine the relationship between bacterial abundance and plaque-forming capacity. The number of infectious bacteria measured by plaque assay was about 1,000-fold lower than the bacterial quantity determined by qPCR and direct microscopic counting of the stock. Also, plaque size increased over time, and larger plaques contained higher bacterial loads. Approximately $10^3$ to $10^6$ *Rickettsia* genomic equivalent per plaque were identified by qPCR, demonstrating that bacterial load increased with plaque size. Overall, these findings suggest that *Rickettsia* burden increases as plaques develop. Comparing qPCR, direct microscopic counting, and the plaque assay, at the single-plaque level provides a framework for distinguishing total detectable bacterial burden from infectious units and may improve the interpretation and standardization of *Rickettsia* infectivity assays.

# Defining the cutaneous immune microenvironment during tick co-feeding on hosts with prior virus exposure

Presenter: Sarah Macon-Foley

Mentor: Meghan Hermance, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Tick co-feeding transmission occurs when infected and uninfected ticks feed simultaneously on the same vertebrate host, enabling virus transfer between ticks without requiring host viremia. This transmission mechanism is thought to be critical for maintaining many tick-borne viruses (TBVs) in nature. Although antibodies against several TBVs have been detected in wildlife species, indicating a prior exposure to the virus, how this previous exposure impacts tick co-feeding transmission of TBVs remains poorly understood. To investigate this, we used an *in vivo* tick transmission model consisting of Langat virus (LGTV)-infected *Ixodes scapularis* nymphs ("donors") and pathogen-free *I. scapularis* larvae ("recipients") co-feeding on either virus-naïve mice or previously LGTV-exposed mice. Recent studies in our lab demonstrated a significant decrease in co-feeding transmission efficiency on previously LGTV-exposed mice compared to virus-and-tick-naïve mice (Fisher's exact, $P < 0.0001$). However, the previously LGTV-exposed mice did not develop a detectable viremia, indicating localized viral transmission at the tick co-feeding site. We therefore employed single-cell RNA sequencing (scRNA-seq) of the tick co-feeding site to characterize the cutaneous immune microenvironment and identify transcriptional changes that occur during co-feeding in the context of virus-naïve or previously LGTV-exposed mice. Validation of our scRNA-seq findings via flow cytometry of the co-feeding site is underway. Ultimately, these studies will define how prior virus exposure of the vertebrate host reshapes the cutaneous immune microenvironment during tick co-feeding and identify host responses associated with reduced virus transmission between co-feeding ticks.

# Evaluation of amyloid-β processing in the central nervous system during experimental Lyme neuroborreliosis

Presenter: Elisa Pizzocaro

Mentor: Timothy Casselli, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Lyme disease (LD) is a multisystem disease caused by the inflammatory response to infection with the tick-transmitted bacterium *Borrelia burgdorferi* (*Bb*). Approximately 15% of LD cases develop Lyme neuroborreliosis (LNB), characterized by cerebrospinal fluid (CSF) infection, inflammation, and neurological manifestations.

Amyloid precursor protein (APP) is a widely expressed transmembrane protein that can be proteolytically processed to generate amyloid-β (Aβ), a peptide associated with neurodegenerative diseases. Previous studies have shown that infection can alter APP processing and promote Aβ accumulation in the central nervous system (CNS); however, APP processing during LNB remains poorly studied. We hypothesized that *Bb* infection alters APP processing products in CNS-associated tissues and fluids.

Aβ species including Aβ38, Aβ40, and Aβ42 were measured by immunoassay in CSF and brain homogenates from *Bb*-infected and control C3H mice, a model that recapitulates key CSF features of LNB. *Bb*-infected samples were collected at days 7 and 28 post-inoculation, corresponding to peak CNS and peripheral inflammation, respectively.

Only Aβ40 and Aβ42 were reliably detected in CSF and brain homogenates from all mice. Variability in Aβ38 levels were observed within groups, consistent with previous reports in patient samples. Aβ species were detected at higher levels in CSF than in brain homogenates or circulation. No significant differences in Aβ levels were observed between *Bb*-infected and control mice. These findings suggest that, in this murine model, *Bb* infection did not induce detectable changes in APP processing. Future experiments will quantify soluble APP in CNS-associated tissues and fluids to further evaluate APP processing during LNB.

# Uncovering the molecular mechanisms of macrophage exhaustion in monoclonal antibody therapies

Presenter: Manley Hicks

Mentor: Michael R. Elliott, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Immunotherapies such as monoclonal antibodies (mAbs) have revolutionized cancer treatment. However, the efficacy of some mAbs is constrained by the finite capacity of the patient immune system. For example, the anti-CD20 mAb rituximab robustly depletes B cells during initial treatment of leukemia but loses potency upon repeated administrations. This acquired resistance is associated with a reduced ability of macrophages to perform antibody-dependent cellular phagocytosis (ADCP), the primary cytotoxic mechanism of rituximab. Mechanistically, sustained ADCP drives macrophages into hypophagia, an exhausted state characterized by significantly attenuated ADCP capacity and loss of Fc gamma receptor 1 (CD64), a key mediator of ADCP. Notably, we have shown that this exhaustion is not a global defect of phagocytosis, as hypophagic macrophages retain their capacity to clear apoptotic cells. This distinction suggests that ADCP-specific components, such as CD64, may represent key regulatory nodes of hypophagia. Based on these observations, we hypothesize that loss of surface CD64 is the principal driver of macrophage hypophagia. Supporting this, our published data demonstrate that IFN-gamma-induced upregulation of CD64 restores ADCP in hypophagic macrophages. But because IFN-gamma exerts broad pleiotropic effects, a more targeted strategy is required to isolate the role of CD64. To address this, we are generating transient CD64 expression systems in primary mouse macrophages to enable precise temporal control of receptor expression, allowing us to directly test its contribution to hypophagia. Collectively, this work aims to enhance the efficacy of mAb therapies by establishing a foundation for targeted adjuvant strategies to bypass macrophage hypophagia.

## Urinary pyridone ribosides impact UPEC growth and virulence

Presenter: Sicily Hardy

Mentor: Allyson Shea, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Urinary tract infections (UTIs) are among the most common bacterial infections worldwide and are major drivers of antimicrobial resistance and antibiotic use. Uropathogenic *Escherichia coli* (UPEC), the leading cause of UTIs, utilizes metabolic flexibility as a virulence strategy to colonize the nutrient-limited urinary tract. While canonical bacterial virulence has been extensively characterized, how host urinary metabolites contribute to pathogenesis remains underexplored in comparison. Pyridone ribosides (2-, 4-, and 6PYR) are $NAD^+$ catabolites that increase with age and inflammation. Mass spectrometry (LC-MS) revealed higher urinary PYR levels in postmenopausal (PM) women without UTI (median 86.19 μM, n = 25) than in PM women with an active UTI (median 21.53 μM, n = 11), suggesting that bacteria may consume these metabolites. Consistent with this hypothesis, UPEC grows in nitrogen-free MOPS minimal medium supplemented with 4PYR as the sole nitrogen source, demonstrating that PYR can potentially serve as an additional nitrogen source in the urinary tract. Detection of 4PYR in bacterial pellets grown under the same culture conditions by LC-MS further supported bacterial uptake. Additionally, UPEC grown in nitrogen-free MOPS with 4PYR showed a 3.8-fold increase in *hlyA* toxin expression relative to the standard MOPS control, suggesting a potential signaling role for 4PYR in regulating virulence-associated gene expression. These findings demonstrate that 4PYR may serve as a nitrogen source for UPEC in the urinary tract while also influencing virulence, linking host urinary metabolites to bacterial metabolism and gene regulation.

# Antibody-dependent cellular phagocytosis of monoclonal antibody-opsonized B cells triggers human macrophage cytokine release

Presenter: Brianna Mitchell

Mentor: Michael R. Elliott, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Monoclonal antibodies (mAbs) treat cancer, autoimmunity, and infectious diseases. However, initial mAb administration in treatment-naïve patients triggers a rapid spike in circulating cytokines, termed cytokine release syndrome (CRS). Some develop first-dose infusion reactions (FDIRs) with fever, hypotension, rash, chills, and dyspnea. In chronic lymphocytic leukemia (CLL), initial mAb administration causes CRS in 64% of patients, 77% with FDIRs. At low doses, rituximab, an αCD20 mAb for CLL, rapidly elevates inflammatory mediators (e.g. IL-6, IL-8, CXCL10). Despite this, mechanisms of cytokine release during initial mAb therapy remain unknown. Within minutes after αCD20 infusion, liver and spleen macrophages rapidly engulf opsonized circulating B cells via antibody-dependent cellular phagocytosis (ADCP), the key cytotoxic mechanism of αCD20 mAbs. Rapid ADCP following initial αCD20 infusion correlates with CRS, highlighting tissue-resident macrophage ADCP as a potential driver of αCD20-induced cytokine release. We hypothesize mAb-mediated ADCP causes macrophages to release inflammatory cytokines contributing to CRS. To test this, human monocyte-derived macrophages (hMDMs) were cultured with αCD20- or αCD52-opsonized Ramos B cells. Over four hours, live-cell imaging confirmed rapid hMDM ADCP of mAb-opsonized Ramos cells; ELISA showed αCD20 and αCD52 increased supernatant IL-6 and IL-8 compared to controls. Multiplex cytokine profiling showed mAb-mediated ADCP selectively increased MCP-1/CCL2 and IL-8/CXCL8, while other CRS-associated cytokines remained near baseline, identifying MCP-1 and IL-8 as ADCP-responsive cytokines. Ongoing studies are defining how complement- and Fcγ receptor-dependent ADCP and its individual stages contribute to cytokine release. Together, these studies will provide mechanistic insight for safer, more accessible mAb therapies while preserving therapeutic efficacy.

# Caspase-1 deficiency alters actin cytoskeletal organization in pulmonary microvascular endothelial cells

Presenter: Hoa Thi Tran

Mentor: Jonathon Audia, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

In cells of the innate immune system, caspase-1 is a proinflammatory cysteine protease best known for its role in inflammasome-mediated activation of IL-1β, IL-18, and the executioner of pyroptosis, gasdermin D. Our prior work elucidated a novel role for caspase-1 in the response of pulmonary microvascular endothelial cells (PMVECs) to infection-induced stress using *Pseudomonas aeruginosa* as a model pathogen. Our unpublished data also showed that caspase-1-deficient PMVECs exhibited delayed ExoT-induced cell rounding, suggesting that caspase-1 may influence actin cytoskeletal responses to infection. However, the mechanisms underlying caspase-1-directed actin organization in PMVECs remain poorly defined. Here we demonstrate that *Casp1*$^{-/-}$ PMVECs exhibit a dysregulation of actin organization. Our study determined that *Casp1*$^{-/-}$ PMVECs exhibited increased phosphorylation of cofilin, an actin-binding protein that regulates actin filament (F-actin) turnover when active. Consistent with reduced cofilin activity, *Casp1*$^{-/-}$ PMVECs showed markedly increased F-actin accumulation compared with wild-type cells. Functionally, *Casp1*$^{-/-}$ PMVECs displayed enhanced wound closure within 24 hours compared with wild-type PMVECs, suggesting increased migratory activity. Interestingly, *Casp1*$^{-/-}$ PMVECs also displayed increased expression of α-smooth muscle actin (α-SMA), a contractile actin isoform predominantly expressed in vascular smooth muscle cells but minimally expressed in endothelial cells. Increased α-SMA expression is commonly associated with endothelial-to-mesenchymal transition and is frequently used as a marker of a mesenchymal-like phenotype. Together, our study identifies caspase-1 as a potential contributor to actin cytoskeletal dynamics in lung endothelial cells and suggests that loss of caspase-1 alters cofilin-dependent actin remodeling and promotes cytoskeletal reorganization toward a mesenchymal-like cytoskeletal phenotype.

# Dominant-negative *Stat3*$^{V463\Delta}$ mutation drives macrophage dysfunction in a model of STAT3 Hyper IgE Syndrome

Presenter: Qudus Ojomo

Mentor: Robert Barrington, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Background: STAT3 Hyper-IgE Syndrome (STAT3-HIES) is a rare primary immunodeficiency caused by dominant-negative *STAT3* mutations. It is characterized by eczema, elevated IgE, and recurrent infections. Recurrent pulmonary infections are major source of morbidity and mortality in STAT3-HIES. Studies utilizing conditional LysM-Cre-mediated *STAT3* deletion demonstrated that *STAT3*-deficient macrophages exhibit hyperactivation, increased MHC-II and CD86, and excessive proinflammatory cytokine production. We investigated whether this phenotype reflects macrophage function in STAT3-HIES using *Stat3*$^{V463\Delta}$ mice, which harbor an autosomal dominant *STAT3* mutation identified in patients.

Methods: *Stat3*$^{V463\Delta}$ and wild-type (WT) mice were infected intratracheally with *Pseudomonas aeruginosa* PA103 ($10^5$ CFU), and bronchoalveolar lavage was collected for single-cell RNA sequencing (scRNA-seq). Bone marrow-derived macrophages (BMDMs) were assessed for phagocytosis using pHrodo-labeled *E. coli* bioparticles and intracellular bacterial killing in a gentamicin protection assay with *E. coli* K-12. LPS-induced CD86, MHC-II, and reactive oxygen species (ROS) production were all assessed by flow cytometry.

Results: scRNA-seq analysis suggests a downregulation of TLR4-NF-κB signaling and enhanced expression of MerTK, a key efferocytosis receptor. Phagocytosis was comparable between *Stat3*$^{V463\Delta}$ and WT BMDMs (n=4), whereas *Stat3*$^{V463\Delta}$ BMDMs exhibited delayed intracellular killing of *E. coli* K-12 over 6h (n=2). LPS-induced CD86 expression and ROS generation were reduced, while MHC-II expression remained comparable.

Conclusion: These preliminary findings identify impaired antimicrobial function and reduced activation in *Stat3*$^{V463\Delta}$ macrophages, contrasting with conditional *STAT3*-deficient macrophages. Defective bacterial killing may therefore contribute to the susceptibility of STAT3-HIES patients to pulmonary infection. Ongoing work is testing whether efferocytosis programs may also be functionally altered in *Stat3*$^{V463\Delta}$ macrophages.

# Detection, *in vitro* propagation, and quantification of *Rickettsia buchneri*

Presenter: Ahmed Garba

Mentor: Meghan Hermance, Ph.D.

Department of Microbiology and Immunology, University of South Alabama
Frederick P. Whiddon College of Medicine, Mobile, AL, United States

*Ixodes scapularis* is a vector of several medically important pathogens and also harbors bacterial endosymbionts. One such endosymbiont is *Rickettsia buchneri*, a maternally-inherited bacterium found across all *I. scapularis* life stages with a strong tropism for the ovaries. Current evidence shows that *R. buchneri* can inhibit growth of other tick-borne bacteria, including *Rickettsia parkeri,* in tick cell culture; however, whether *R. buchneri* interacts with pathogens naturally transmitted by *I. scapularis* remains unknown. Investigating these microbial interactions within the tick requires a reliable system for detecting, propagating, and quantifying *R. buchneri*. Field-collected and laboratory-reared *I. scapularis* were screened for *Rickettsia* by conventional PCR targeting the citrate synthase (*gltA*) gene. Overall, 8/30 (26.7%) adult ticks were *Rickettsia*-positive, of which 5/8 (62.5%) were confirmed as *R. buchneri* by Sanger sequencing and BLAST analysis. To establish *R. buchneri in vitro*, cells from a *R. buchneri*-infected ISE6 tick cell line were combined with uninfected ISE6 cells and transferred through three sequential propagations. Each propagation was monitored weekly by determining the proportion of *Rickettsia*-infected ISE6 cells in Giemsa-stained cytocentrifuge preparations. Infection increased from 15% at week one to 100% at week four across all propagations. At ≥80% infection, cells were mechanically disrupted to release intracellular *Rickettsia*. The cell-free *Rickettsia* were then semi-purified and quantified by direct microscopic counting of viable bacteria, yielding a concentration of $1.02 \times 10^{10}$ *Rickettsia*/mL. Ultimately, these data lay the foundation for subsequent investigation of *R. buchneri* growth and interactions with *I. scapularis*-borne pathogens.