Infectious pathogens still remain a major cause of human diseases, whereas defective or excessive immunity causes an array of many other disorders including cancer, autoimmune disease, and allergic disease. The Department of Medical Microbiology and Immunology at the University of Toledo is dedicated to the fight against these common disorders.

Microbiology explores the relationships between microbial pathogens and their human hosts, and immunology studies the nature of host defense system against environmental insults. The Department contributes to the research mission of the University by advancing basic and translational research with the ultimate goal of developing innovative vaccines and therapeutics for infectious diseases, cancer, and inflammatory disorders caused by immune dysregulation. Cutting-edge technologies in biomedical science are provided through the Flow Cytometry Core Lab, Bioinformatics, Genomics/Proteomics Core Lab, and BSL-3 Lab housed in the Department. The Department also contributes to the teaching mission of the University by educating medical and graduate students in basic principles of immunology, bacteriology, virology, and host-pathogen interaction. Moreover, the Department contributes to the service mission of the University by providing advanced expertise in microbiology and immunology and by promoting regional, state, national and international research programs concerning infectious diseases, inflammatory disorders, and graft rejection.

Our research program is directed at understanding the immune function of platelets in both protection from pathogenic infection and platelets as a causative or compounding factor in autoimmune diseases and clotting disorders. These studies have been published in the American Journal of Pathology, Journal of Thrombosis and Haemostasis, and Journal of Immunology.
Dr. Huntley’s laboratory primarily studies Francisella tularensis, the causative agent of the zoonotic disease tularemia. F. tularensis is well-recognized as one of the most dangerous bacterial pathogens known because of its low infectious dose, ease of aerosolization, multiple routes of infection, and ability to induce severe disease and death. Dr. Huntley’s laboratory uses a multi-disciplinary approach to: (1) study changes in F. tularensis membrane proteins during mammalian and tick infections; (2) generate F. tularensis membrane protein mutants and examine their virulence in vitro and in vivo; (3) characterize the function of individual membrane proteins; (4) develop and test new vaccines to prevent tularemia; and (5) define immune responses that protect against F. tularensis infection.

More recently, Dr. Huntley’s laboratory has received funding to study cytomicrobial blooms, cyanobacterial hepatotoxins (e.g. MC-LR), and is developing new methods to remove MC-LR from municipal drinking water. To accomplish these goals, current projects are: (1) selecting for and isolating MC-LR degrading bacteria from Lake Erie; and (2) using these MC-LR-degrading bacteria as biocatalysts to remove MC-LR from drinking water.

We research the mechanisms by which bacteria sense and respond to their extracellular environment. *Vibrio cholerae* is the bacterium that causes epidemic cholera, a disease that continues to spread in areas of the world where people lack access to clean drinking water. Due to increasing antibiotic resistance among *V. cholerae* strains, there is a need to develop additional therapeutic agents for cholera treatment. Current projects in the Matson lab include identification and characterization of small molecule inhibitors of a *V. cholerae* stress response pathway that may be developed into cholera therapeutics. Additional studies aim to characterize transcriptional responses of *V. cholerae* to various stresses to determine pathways associated with bacterial fitness and pathogenesis. Identification of new antiviral strategies will lead to the development of successful therapeutic applications.

Our laboratory is interested in studying the host responses against virus infections in cells and mice. Innate immune responses, activated very early during virus infection, are critical for inhibition of virus replication. The interferon system is recognized as a major antiviral innate immune response mechanism against a broad range of viruses. We are investigating how a key transcription factor IRF3 and the induced genes (ISGs) mount antiviral protection. Using both RNA (e.g. Paramyxoviruses) and DNA (e.g. Herpesviruses) viruses, which are the important human pathogens, we are interested to uncover novel host response mechanisms to protect against them. Identification of new antiviral strategies will lead to the development of successful therapeutic applications.

Our laboratory has four areas of interest. One focuses on how gene regulation of *F. tularensis* infection is controlled. Our work indicates that the production of a number of *F. tularensis* virulence factors, including lipopolysaccharides, LPS, and outer membrane proteins, is controlled by the transcriptional regulator, the RcsB protein. RcsB is a global regulator that controls many aspects of bacterial physiology, including metabolism, virulence, and survival. Our studies have shown that the expression of RcsB is regulated by a conserved DNA sequence, the RcsB box, which is present in the promoters of many *F. tularensis* virulence genes. We are currently investigating the role of RcsB in the regulation of virulence factor expression and in the host-virus interaction.Huntley's laboratory is particularly interested in studying the mechanisms by which bacteria sense and respond to their extracellular environment. *Vibrio cholerae* is the bacterium that causes epidemic cholera, a disease that continues to spread in areas of the world where people lack access to clean drinking water. Due to increasing antibiotic resistance among *V. cholerae* strains, there is a need to develop additional therapeutic agents for cholera treatment. Current projects in the Matson lab include identification and characterization of small molecule inhibitors of a *V. cholerae* stress response pathway that may be developed into cholera therapeutics. Additional studies aim to characterize transcriptional responses of *V. cholerae* to various stresses to determine pathways associated with bacterial fitness and pathogenesis. Identification of new antiviral strategies will lead to the development of successful therapeutic applications.

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Molecular Mechanisms of Host-pathogen Interactions

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**Faculty Highlights & Achievements**

- Well-funded department with over $2.4 million supporting strong research projects in both basic and translational research in a collegial atmosphere, with approximately 20 publications each year
- Strong record of successful faculty recruitment, retention, and timely promotions with tenure
- Medical student performance consistently above the national average on Step 1 USMLE Exam in sections of microbiology, immunology, and immune systems
- Highly successful graduate program with students earning major awards and fellowships, timely graduation, and establishing careers in science and industry
- Outstanding service record with the university, Toledo and surrounding communities, and in the scientific community (nationally and internationally)
- Recent Dean’s Awards selected by UT peers:
  - 2013 Outstanding Research Award (Stepkowski)
  - 2014 Research Excellence—Sustained Category (Stepkowski)
  - 2014 Research Excellence-New Investigator (Ferreira)
  - 2014 Mentoring Excellence (Pan)
  - 2013 Outstanding Research Award (Stepkowski)
  - 2013 Teaching Excellence-Basic Science (Ferreira)
  - 2012 Distinguished University Professor (Blumenthal)

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