# Welcome to the Department of Microbiology, Immunology & Parasitology

## LSU Health Sciences Center
## School of Medicine at New Orleans
## http://www.medschool.lsuhsc.edu/microbiology

It's an exciting time to be involved in studies of microorganisms and their interactions with human and animal hosts. Recent advances in microbiology, immunology, molecular genomics, and bioinformatics have provided tantalizing glimpses into the biology of these "bugs", how they cause disease and the influence of our own microbiota on the disease process, how the immune system has evolved to deal with these threats, and how, in turn, microorganisms have evolved complex survival strategies in the face of host defenses. Devastation caused by the COVID-19 pandemic is a stark reminder of these issues. SARS-CoV-2, along with recent outbreaks of mpox in Africa and beyond, the ongoing problems of influenza virus, methicillin-resistant 'superbugs' (MRSA), HIV/AIDS, drug-resistant TB, and life-threatening fungal infections, among others, are constant reminders of the dynamic nature of our field.

Faculty in the Department of Microbiology, Immunology and Parasitology (MIP) direct translational research programs in immunology, virology, bacteriology, mycology and parasitology, linking basic and clinical sciences within the LSU Health Sciences Center and with researchers at other campuses. The MIP Department provides a strongly interactive environment that has underpinned the development of several new interdisciplinary research and development programs with funding from the National Institutes of Health, Private Foundations and Industry. Our recent State-funded initiatives include the Louisiana Vaccine Center, established in collaboration with colleagues from Tulane and Xavier University, while MIP faculty have also forged strong linkages with related programs here at the Health Sciences Center and the LSU School of Veterinary Medicine in Baton Rouge.

Each of these initiatives underpins a dynamic and integrated education program in MIP, that includes mentoring and career development of postgraduate students and postdoctoral trainees from Louisiana, across the United States, and around the globe. Students pursuing PhD and MS degrees in our program are mentored by an accomplished and dedicated faculty and supported by state-of-the art research facilities.

Please contact us for further information concerning any aspect of our research and education programs.

**Alistair Ramsay, Ph.D.**
**G. John Buddingh Professor and Department Head**

**MIP Graduate Coordinators:**
**Doug Johnston, PhD**
**Jennifer Cameron, PhD**

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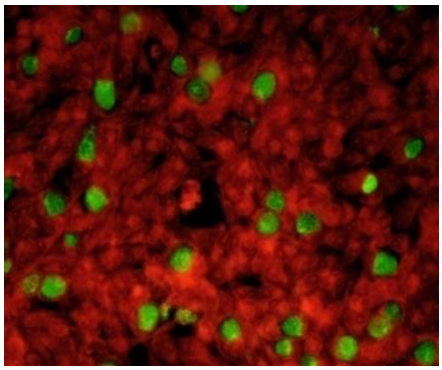
# DEPARTMENT OF MICROBIOLOGY, IMMUNOLOGY, & PARASITOLOGY FACULTY RESEARCH CAPSULES

## IMMUNOLOGY AND INFECTIOUS DISEASES / CANCER

**Pam Kozlowski, Ph.D.** **[pkozlo@lsuhsc.edu; 568-6956; CSRB 606]**

Research in Dr. Kozlowski's laboratory is focused on the identification of mucosal and systemic IgA antibody responses that could protect against HIV-1 or SARS CoV-2 infection. IgA antibodies transported into mucosal fluids are ideally suited for host protection, and vaccines that induce HIV or SARS CoV-2-specific IgA antibodies in secretions could prevent transmission of these viruses at mucosal surfaces. Studies in the lab involve measuring the specificity and function of the antiviral antibodies produced in infected individuals or in nonhuman primates immunized with different types of vaccines.

**Alison Quayle, Ph.D.** **[aquayl@lsuhsc.edu; 568-4070; CSRB 543]**



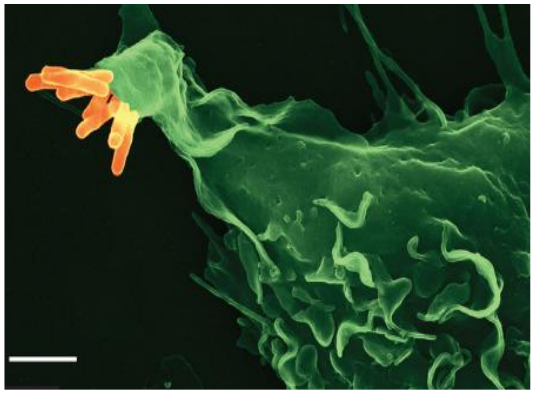
The central theme of our research is immune defense in the human female genital tract, with a focus on the unique obligate intracellular bacteria *Chlamydia trachomatis*, and HIV. Broadly, our research interests encompass the study of: (1) *Chlamydia trachomatis*-specific adaptive responses in the human endocervix, (2) immuno-evasive strategies used by *C. trachomatis* to adapt to, and survive in, the human genital milieu and (3) *C. trachomatis* and HIV co-infection and (4) the impact of a dysbiotic vaginal microbiome (bacterial vaginosis) on genital immunity. We collaborate with the LSU/Crescent Care Sexual Health Clinic for our clinical studies and use *in vitro* modeling to elucidate basic mechanisms.

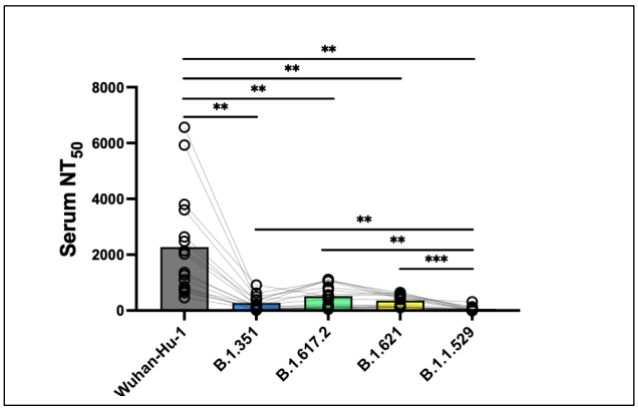
***An endocervical isolate of Chlamydia trachomatis grown in HeLa cells**. Chlamydial lipopolysaccharide is stained green, visualizing the chlamydial inclusion, and structural proteins of the cell are stained red.*

**Alistair Ramsay, Ph.D.** **[aramsa@lsuhsc.edu; 568-8324; CSRB 339]**

Current research interests of the Ramsay lab are centered on immune biology of pulmonary infections by intracellular pathogens with a view to the development of new or improved vaccine strategies. Our ultimate goal is to generate information that will inform the development of improved vaccines, particularly those that stimulate protective immunity at mucosal and systemic sites of infection.

A primary focus is investigating host:pathogen interactions in *Mycobacterium tuberculosis* infection, using immune assays, genomics and bioinformatics. Related to this, we are interested in finding improved TB immunization strategies, based largely on the development and evaluation of recombinant BCG and viral vectors expressing immunogenic vaccine targets in *M. tuberculosis* and engineered for enhanced immunogenicity. In COVID-19 research we have developed a pseudovirus-based assay for neutralizing antibody responses against SARS-CoV-2 and its variants as a tool to facilitate our characterization of functional immunity in both SARS-CoV-2 convalescents and vaccinees over time (see Fig. below).






***Mycobacterium tuberculosis*** *associated with a macrophage cell (left), and evidence of resistance of* ***SARS-CoV-2*** *variant strains to neutralizing antibodies generated following vaccination with RNA vaccine encoding Spike glycoprotein of Wuhan-Hu-1 pandemic strain.*

**Guoshun Wang, D.V.M., Ph.D.** **[gwang@lsuhsc.edu; 568-7908; CSRB 607]**

The Wang lab has two major research directions: 1) understanding cystic fibrosis (CF) disease pathogenesis, and developing CF gene and stem cell therapy, and 2) elucidating alcohol-induced anti-inflammation and immunosuppression.

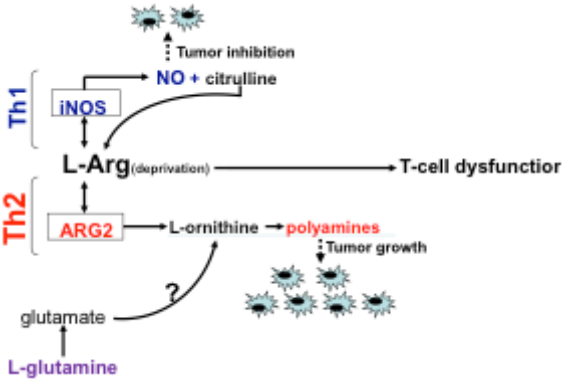
CF, the most common life-threatening genetic disorder in the Caucasian population, is caused by mutations in CF transmembrane conductance regulator (CFTR) gene, encoding a cAMP-activated chloride channel. Clinically, CF intestinal and lung diseases claim the most morbidity and mortality with the cardinal pathology characterized by infection, inflammation and obstruction. It is not fully established how the chloride channel defect leads to these clinical complications. The Wang lab made the initial discovery that the CFTR defect undermines chloride transport to neutrophil phagosomes, which impairs the production of hypochlorous acid (HOCl), a potent microbicide for effective bacterial killing. This defect compromises host defense against infection and resolution of inflammation. The lab is further elucidating the contribution of this innate immune defect in CF lung and intestinal disease pathogenesis, and is also pursuing genomic editing to correct the root-cause defect of CF.

Alcohol is known to have anti-inflammatory and immunosuppressive effects. However, molecular mechanisms underlying this long-observed phenomenon are not well defined. Previous research from this lab has shown that ethanol, in the absence of glucocorticoids, upregulates Glucocorticoid-Induced Leucine Zipper (GILZ), a critical steroid-responsive gene, through non-canonical activation of Glucocorticoid Receptor (GR). We are now characterizing how alcohol exploits the GR signaling pathway to modulate the body's immune system.

**Arnold H. Zea, Ph.D.** **[azea@lsuhsc.edu; 599-0906; CSRB 531]**

The Zea lab is focused in the immune biology of cancer and tuberculosis. In cancer, Dr. Zea is studying the mechanisms by which L-arginine and L-glutamine metabolism regulates tumor growth-inhibition and immune responses (see Figure below). This work will help to better understand mechanisms of resistance and tumor evasion and to develop new therapeutic strategies to control and possibly eradicate tumors. The knowledge and experience gained in cancer-related research has allowed Dr. Zea to explore whether similar mechanisms can occur in infections by *Mycobacterium tuberculosis* (*Mtb*) and Non-Tuberculous Mycobacteria (NTM). He is studying *in vitro* and *in vivo* mechanisms by which *Mtb*-cyclic-AMP and NTM-cyclic-AMP (cAMP) regulates arginase induction, nitric oxide and cytokine production used by *Mycobacteria* to survive and persist inside macrophages.

The main goal of these projects is to identify pathways involved in L-arginine metabolism that can be targeted to inhibit tumor and/or *Mycobacterial* growth/persistence. These findings could facilitate the development of new, unconventional therapies that could eliminate tumors and tuberculosis, based on their dependence on L-arginine or L-glutamine. It also has the potential to advance treatments for multi-drug and extensively-drug resistance tuberculosis, where current first line drug therapies are ineffective.

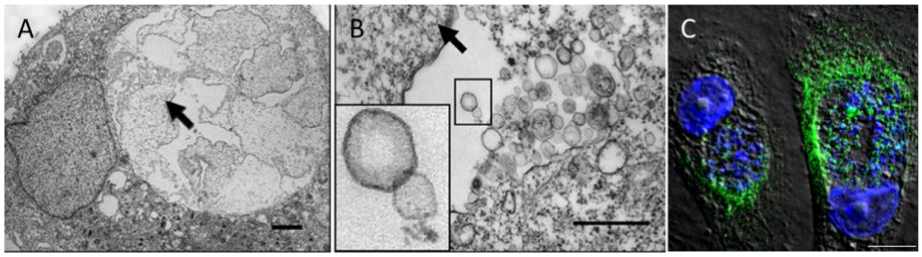



***Impairment of antitumor immunity in patients with cancer (and TB) could be due to the shift from a Th1 to Th2 cytokine response****. This shift prompts the tumor microenvironment to produce high ARG2 and polyamine levels to sustain tumor growth, instead of high levels of INOS and NO to inhibit its proliferation. The depletion of L-Arginine (L-Arg) induces T-cell dysfunction as another advantage for tumor growth. The effect of L-glutamine on modulating L-arginine metabolism is being investigated.*

## BACTERIAL PATHOGENESIS AND BIOINFORMATICS

**Li Shen, M.D., Ph.D.** **[lshen@lsuhsc.edu; 568-4076; CSRB 359]**

Research in Dr. Shen's lab is focused on pathogenesis of human intracellular pathogens, such as *Chlamydia trachomatis* that cause the most prevalent sexually transmitted bacterial infections with serious reproductive complications in both men and women. Molecular, genetic, biochemical, cell biology, and RNA-sequencing approaches are utilized in combination. (i) *Control of the virulence associated type III secretion system (T3SS) in C. trachomatis*. *Chlamydia* uses the T3SS to deliver virulence proteins termed 'effectors' to counteract host innate immunity. Evidence emerges that there is an intimate link between gene expression and the T3SS in *C. trachomatis*. We are interested in defining the mechanisms that are utilized by *Chlamydia* to carefully regulate the T3SS activity at multiple steps, including gene expression, substrate recognition, and spatiotemporal effector secretion. (ii) *Mechanisms and consequences of C. trachomatis responses to antimicrobial insults.* Despite aggressive diagnosis and antibiotic treatment program, rates of *C. trachomatis* infections continue to rise. We seek to develop novel tools that allow us to quantitatively probe diverse developmental forms of *C. trachomatis* during infection. We will characterize key signaling pathways, in *C. trachomatis* and its host cells, that contribute to *Chlamydia* adaptation and survival during exposure to external and internal antimicrobial insults. It is expected that insights obtained will pave the way for the future development of novel therapies targeting the pathways against *Chlamydia* infections.

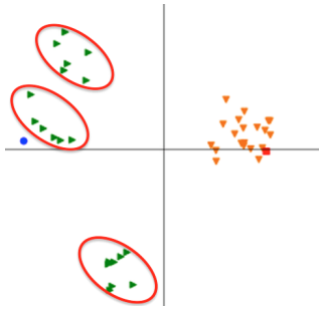



*Chlamydial persistent forms (A) and the member vesicles induced by ampicillin in HeLa cells (B). Translocation of virulence factor CPAF (green) into the cytosol of human primary endocervcal epithelial cells (C).*

**Christopher Taylor, Ph.D.** **[ctay15@lsuhsc.edu; 568-4065; CSRB 601A]**

The human body is host to diverse communities of microbial organisms collectively referred to as the human microbiome. These communities include bacteria, fungi and archaea, some of which perform important

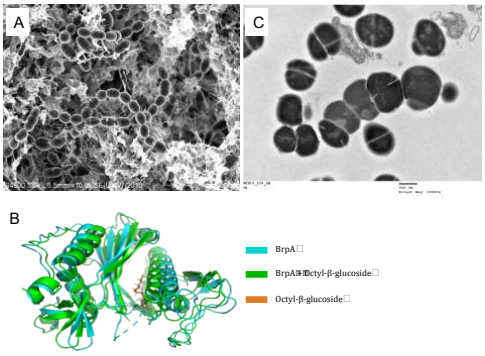
metabolic functions. Interventions such as antibiotic treatment and environmental interaction can disrupt these communities and changes in community structure have been shown to play a major role in several diseases. We use 16S ribosomal RNA collected from these communities in tandem with high-throughput sequencing to study and analyze these microbial communities and their relationship with human health and disease. The primary focus of the lab is computational analysis and visualization of microbial community structure performed in collaboration with clinicians and basic scientists. In addition to the human microbiome, we also investigate microbial communities in model organisms such as mouse, rat, and non-human primate studies with a focus on translational research (*see Fig. overleaf*). In addition to 16S rRNA analysis of microbial community structures, we also use shotgun metagenomic sequencing to assess microbial communities. The primary source of funding for our lab is NIAID with several grants to study the reproductive tract and interactions of microbiota with sexually transmitted infections (primarily Chlamydia) and bacterial vaginosis.



***Principle Coordinates Analysis Plot*** *of mice given a gut microbiota transplant (green triangles) from mice on a high-fat diet (blue circle) and mice given a gut microbiota transplant (orange triangles) from mice on a standard chow diet (red square). The gut microbial communities of mice receiving the transplant are most similar to each respective transplant donor community. Red ovals surround samples from mice that were housed in the same cage illustrating that gut microbial community drift is impacted by cohabitation.*

**Z. Tom Wen, Ph.D.** **[zwen@lsuhsc.edu; 941-8465; Dental School 6305]**

The focus of Dr. Wen's research at the Dental School is on molecular characterization of oral biofilms and identification of novel strategies against human dental caries. With the support of the NIH, major efforts are being directed to (i) identification of genes required for biofilm formation by *S. mutans,* the key etiological agent of dental caries; (ii) uncovering how different species of bacteria communicate affecting the development ultimately, oral health; and (iii) identification of novel strategies including natural compounds effective against cariogenic biofilms and dental caries.

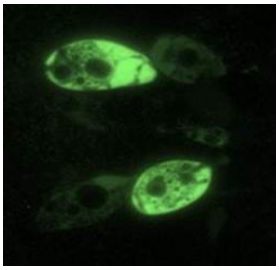



A. SEM image of *S. mutans* biofilms at x10.0k; B. BrpA structure with and without Octyl-β-glucoside; C. TEM image of a *mecA* mutant featuring major defects in cell division at x10.0k.

## PATHOGENIC EUKARYOTES

**Ben Kelly, Ph.D.** **[bkell2@lsuhsc.edu; 568-6115; CSRB 346]**

Dr. Kelly's laboratory studies the biology of the protozoan pathogens, *Leishmania* and *Trypanosoma cruzi*. These parasites are transmitted via the bite of their insect vectors and are the etiologic agents of leishmaniasis and Chagas disease, respectively. Currently, there are no really effective treatments to combat these debilitating and often fatal diseases that have infected approximately 20 million people worldwide. Research in my laboratory focuses primarily on understanding molecular functions of specific parasite proteins required for viability and virulence. We are especially interested in identifying

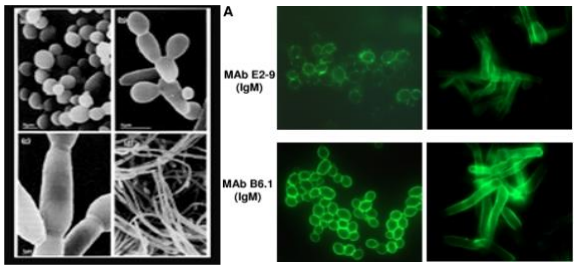


*Leishmania parasites expressing a LACK–Green Fluorescent Protein chimeric transgene.*

important molecular functions unique to these parasites, as these may represent potential drug targets for better, low toxicity therapies against these diseases.

Current research in the lab includes use of conventional and CRISPR-based gene-knockout and gene-tagging approaches to understand how a molecular scaffolding protein, termed "LACK", promotes expression of parasite genes important for virulence and survival in the mammalian host.

**Hong Xin, M.D., Ph.D.** **[hxin@lsuhsc.edu; 568-8121, CSRB 541]**

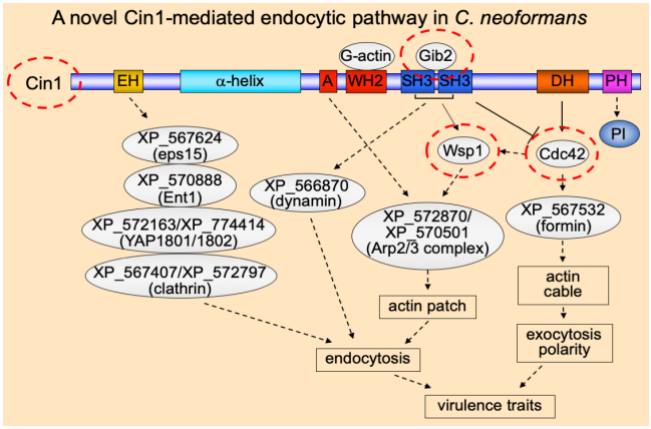



*Candida albicans* Antibodies bind to *Candida*

Hematogenously disseminated candidiasis in humans has become the third leading cause of hospital-acquired blood stream infections and despite antifungal therapy at least 40% of affected individuals will die of this disease. As there is no approved antifungal vaccine for use in humans and significant therapeutic challenges remain, our approach is disease prevention through active vaccination and/or passive immunization with protective antibodies. We first reported that a tetanus toxoid conjugated glycopeptide vaccine induced dual (double) antibody-dependent protective immunity without the need for adjuvant, which is feasible for human use. We have demonstrated a novel double chimeric peptide vaccine that functions synergistically to improve the level of protection against disseminated candidiasis. Up to now, we have developed a panel of protective monoclonal antibodies (mAbs), which are specific for fungal cell-surface glycan and peptides with high homologous among all medically relevant *Candida* species, including *C. auris.* Furthermore, we showed that the combination of two mAbs is a much more effective immunoprotective approach as compared to single mAb treatment. Strikingly, these "universal" mAbs, as an adjunct to the existing therapy, greatly enhanced efficacy of antifungal treatment against the diseases in immunocompromised mouse models, indicating the great clinical relevance. Our research goal is to develop the multi-epitope vaccine and mAb-based therapy that protects against medically significant *Candida* species in humans, as well as addressing fundamental questions concerning vaccine-induced immunity in high-risk, immune compromised populations.

**Ping Wang, Ph.D.** **[pwang@lsuhsc.edu; 568-2446; CSRB 358]**

Research in my laboratory is focused on understanding the molecular basis of pathogenesis using two human fungal pathogens, *Cryptococcus neoformans* and *Rhizopus delemar,* as model organisms. For *C. neoformans,* we examine how G protein-binding proteins and their regulators mediate signal transduction pathways that coordinate cellular growth and differentiation required for virulence. We also dissect a novel intersectin (Cin1)-mediated intracellular trafficking in the secretion of virulence factors and determine whether Cin1 alternative splicing has a role in the neurotropic property of the fungus, a unique mechanism of cryptococcal pathogenesis.

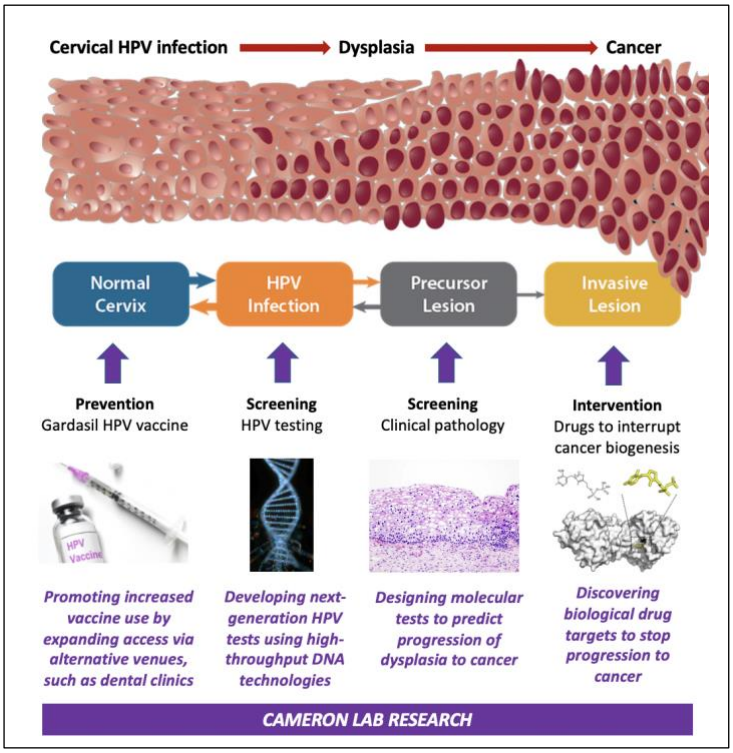



For *Rhizopus delemar,* our focus is to develop tools to advance genetic studies of mucormycosis virulence mechanisms. We have started with adopting CRISPRCas9 technology for gene editing, whose optimization will facilitate the construction of mutant libraries. Such technical development would promote the discovery of novel targets for antifungal therapy.

# MOLECULAR VIROLOGY, VIRAL PATHOGENESIS & VIRAL CANCERS

**Jennifer E. Cameron, Ph.D** **[jcame2@lsuhsc.edu; 568-2196; CSRB 539]**

It is estimated that at least 10% of cancers worldwide are caused by infections with 'tumor viruses'. One of the most common tumor viruses is human papillomavirus (HPV), which causes cervical cancer in women as well as anal cancers and some oral and throat cancers in both men and women. We seek to understand the underlying molecular biology of HPV cancer biogenesis so that we can apply that knowledge to improve cancer prevention strategies. For example, our work has recently focused on the expression patterns of tiny regulatory molecules known as microRNAs that can differentiate aggressive from benign dysplasia, the precursor to HPV cancer. Additionally, our work has revealed compelling clinical evidence that when two tumor viruses - HPV and Epstein-Barr virus (EBV) - share a biological niche, they may coordinately promote dysplasia development.



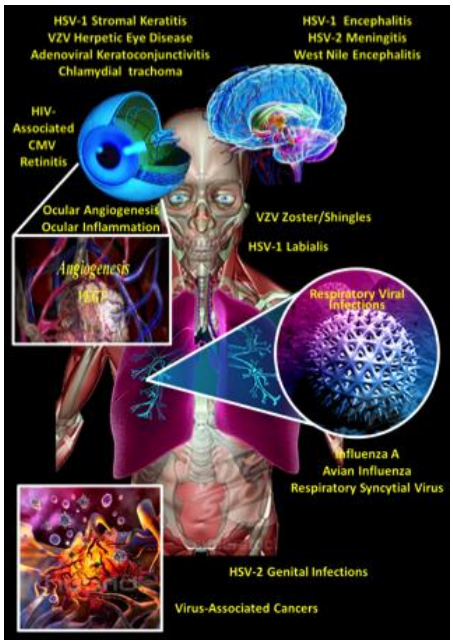

We are, therefore, interested in unraveling the biological consequences of HPV-EBV co- infection to reveal key aspects of cancer biogenesis that can be exploited for effective medical intervention. The work employs cutting-edge genomic technologies to investigate these and other clinically important questions in specimens donated by patients with HPV infection.

In response to the emergence of SARS coronavirus type 2, Dr. Cameron's lab has also participated in research to characterize the innate and adaptive immune responses important for protection against COVID-19 morbidity and mortality. The lab is also evaluating protease inhibitor compounds that interfere with SARS-CoV-2 replication by blocking the activity of the two viral proteases. Promising compounds will enter the developmental pipeline as potential antiviral therapies for COVID-19.

**Timothy Foster, Ph.D.** **[tfoste@lsuhsc.edu; 568-4075; CSRB 522]**

The Foster lab investigates cellular and molecular virus-host interactions that can be utilized to simultaneously inhibit pathogen replication and suppress deleterious host-mediated inflammatory responses, especially within the eye. To ensure visual clarity and acuity, the eye is normally maintained as an avascular immunologically privileged organ. However, infection of the ocular surface by common viral or bacterial pathogens can result in vision-threatening vascularization and host-mediated inflammatory responses that cannot be resolved by current anti-pathogen treatment regimens. Through our studies, we have developed targeted therapeutics that modulate host metabolic pathways that are required for both pathogen replication and induction of inflammation-associated disease sequelae (US patent applications 61/664,464, 13/828,669, & 14/039909 and international patent application PCT/US13/31623 by Foster *et al*.).

These approaches are unlike current drugs in that: 1) Disruption of these pathways prevents replication of a broad range of intracellular pathogens, including most viruses and some bacteria such as *Chlamydia*; 2) Targeting of host pathways, rather than pathogen-specific mechanisms, constrains development of drug resistance; 3) They can be used for treatment of current drug resistant pathogens; 4) They block formation of vision-threatening host-mediated sequelae by modulating inflammatory responses; 5) They inhibit pathological vascularization, a current area of intense research for ophthalmic, as well as for anti-cancer therapeutics; 6) They promote healing of traumatic wounds induced either surgically or through pathogen replication. Therefore, our lab's efforts have broad reaching implications that go far beyond exploring a single pathogen or disease presentation. Consequently, we employ a broad range of cellular and molecular techniques, as well as animal models in order to discern therapeutic efficacy of a drug and its potential mechanisms of action.




*Virus and Inflammation-Associated Disease Processes Targeted by Metabolic Therapeutics in Development.*

**Michael Hagensee, M.D., Ph.D.** **[mhagen@lsuhsc.edu; 210-3324; LCRC 708]**

*HPV viral particle*

The Hagensee laboratory studies the role of human papillomavirus (HPV) in human malignancies. Studies are focused on the increase in HPV-related cervical cancer in HIV+ women and the increase in oral cancer in HIV+ men and women.

A new area of focus is the role of HPV in anal cancer from basic biology to clinical trials and similar increases in anal cancer rates in HIV+ individuals. This increase may be due, in part, to an interaction with another DNA virus, Epstein Barr Virus (EBV) which also causes human cancers. Current projects include detection of HPV and EBV in clinical specimens, determination of the systemic and local immune response against each virus, in-vitro modeling techniques and development of xenograft mouse models. Results from these studies will aid in improved diagnostics and preventive measures for these cancers. Additional projects include development of self-testing methods to detect HPV DNA to improve cervical cancer screening and studies into improvement of HPV vaccine implementation. More recently, we have started to explore the serological response to SARS-COV-2 namely detection of serum antibodies against COVID-19, how long do they last after natural infection or vaccination and responses to the new variants of COVID-19.

# DEPARTMENT OF MICROBIOLOGY, IMMUNOLOGY, & PARASITOLOGY GRADUATE STUDENTS (*alphabetically by mentor*)

**CAMERON LAB:**

**Ashley Winters**
Ashley's PhD project focused on miRNAs as biomarkers for identifying women who will advance from low grade cervical intraepithelial neoplasia (LG-CIN) to high grade cervical intraepithelial neoplasia (LG-CIN).
This work is part of an overall lab focus aimed at the development of a clinical test, which could obviate the need for medical observation and indicate treatment intervention for women at risk of progression to HG-CIN and cancer. Ashley graduated with her PhD in December 2024.

**FOSTER LAB:**

**Thomas Galbato**
Thomas is working on therapy of host-directed inflammatory disease for his PhD project. The role of the serotonin 2A receptor in Inflammatory and age-related disease, including macular degeneration in the eye is a particular focus of his studies.

**JOHNSTON LAB:**

**Lyndsey Nash Gisclair**
Lyndsey's PhD project focused on evaluating endothelial cell responses to infection with the parasite *Trypanosoma cruzi*, the causative agent of Chagas' Disease. About 30% of infected individuals develop life-threatening cardiomyopathy due to excessive inflammation and fibrosis. The project included development of a novel 3D multicellular cardiac co-culture model to characterize endothelial responses to *T. cruzi* infection and endothelial dysfunction to inform potential therapies or preventives for Chagas' Disease. Lyndsey graduated with her PhD in May 2025.

**KELLY LAB:**

**Isabel Stephany-Brassesco**
Isabel's PhD project involves identification of RACK1-proximal proteins in parasitic trypanosomatids and the importance of rsp17 in ribosome LACK interactions and LACK function. This work is a key component of the lab emphasis on molecular understanding of the function and regulation of trypanosomatid protein pathways towards improved drug therapies.

**KOZLOWSKI LAB:**

**Justin Smith**
Justin's PhD project is focused on evaluating potential effector functions of IgA from rhesus macaques immunized with newly developed trimeric HIV envelope proteins. IgA antibodies in this species are not well characterized and it remains unclear if they can efficiently neutralize virus or mediate Fc-dependent antiviral effector functions. These studies will inform development of new methods for measuring functions of IgA antibodies generated by HIV vaccine candidates and their correlation with protection.

**QUAYLE LAB:**

**Caleb Ardizzone**
Caleb's PhD project was focused on eludicating protective immunity to *Chlamydia trachomatis* in the female genital tract, and the factors that compromise this. Caleb graduated with his PhD in May 2024.

**Clayton Jacobs**
Clay is an MD/PhD student who joined the Quayle and Taylor labs in August 2024.

**RAMSAY LAB:**

**Jared Sheehan**
Jared's PhD project focused on characterizing development and decay of binding isotypes and functional (virus-neutralizing) antibody responses against SARS-CoV-2 and its variants over time, both in sera from COVID-19 mRNA vaccinees and in convalescent sera from patients infected with SARS-COV-2. Jared graduated with his PhD in May 2024.

**RIVERA LAB (Jessica Rivera MD/PhD, Dept of Orthopedics, secondary appointment in MIP):**

**John Carleton**
John is an MD/PhD student who joined the Rivera lab in August 2024.

**TAYLOR LAB:**

**Jacob Elnaggar**
Jacob's PhD project focused on assessment of the vaginal microbiome through the use of 16S rRNA sequencing, shotgun metagenomic sequencing, and qPCR assays. He developed informatics methods to analyze and visualize the microbial composition of vaginal samples over time using both sequencing methodologies and qPCR assays to obtain better assessments of the abundances of microbial organisms. Jacob graduated with his PhD in May 2024 and returned to medical school to complete his MD degree.

**John Lammons**
John is an MD/PhD student working on gut-derived mechanisms promoting HIV- and alcohol-induced gastrointestinal dysfunction using integrated meta-/multi-omics approaches.

**WANG, G LAB:**

**Dianne Wellems**
Dianne's PhD project concerned elucidation of the role of cystic fibrosis transmembrane conductance regulator (CFTR) in myeloid cells in cystic fibrosis (CF). Dianne graduated with her PhD in December 2023.

**Callie Scull**
Callie's PhD project investigated how the function of the CFTR gene in innate immune cells affects the intestinal microbiota, a potential mechanism underlying cystic fibrosis (CF) intestinal disease that clinically manifests as intestinal infection, inflammation, and obstruction. Callie graduated with her PhD in December 2023.

**XIN LAB:**

**Jonothan Colon**
Jonothan's PhD project involved development of immunotherapeutic strategies to protect the host from multidrug-resistant *Candida auris* infection. He used mouse models of invasive *C. auris* infection to evaluate multi-peptide vaccines as novel immunotherapeutics, and a panel of peptide-related monoclonal antibodies as a passive vaccination strategy. Jonothan graduated with his PhD in May 2024 and is currently in medical school.