UF College of Dentistry
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Shannon M Wallet,

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About Shannon M Wallet

Shannon Wallet, Ph.D., is Chair and Louis and Marge Atkins Endowed Professor of the Department of Oral Biology.

Wallet holds a bachelor's in medical technology from North Carolina State University and a degree in clinical laboratory science from Duke University. She earned her doctorate in oral biology from the University of North Carolina at Chapel Hill in 2005 and joined UF in 2006 as a tenure-accruing assistant professor in the department of periodontology. She was promoted to associate professor and was awarded tenure in July 2013. After 12 years in various roles with UFCD, Wallet was the associate dean for faculty affairs when she left in 2018 for a position in the Department of Foundational Sciences at the East Carolina University School of Dental Medicine. She became the director of basic science and served as interim chair of foundational sciences at ECU, before leaving in 2019 to serve as the associate dean for research for the Adams School of Dentistry Interim chair for the Division of Oral and Craniofacial Health Sciences.

In addition to her administrative duties as a department chair, Wallet teaches dental students and residents in basic sciences. Wallet also has a robust research program which is focused on mechanisms associated with altered innate immune functions which lead to dysregulated adaptive immunity. Her current research program focuses on the contribution of epithelial cell biology and signaling to innate and adaptive immune homeostasis and dysfunction. Using novel and unique tools, her team is able to evaluate their findings in the human conditions which makes translation of their findings more feasible.

Teaching Profile

Courses Taught

- 2011-2018,2022-2024
  DEN6128 Host Defense
- 2018
  GMS7980 Research for Doctoral Dissertation
- 2009-2018
  DEN8290 Special Topics
- 2018
  BME7980 Research for Doctoral Dissertation
- 2017-2018
  DEN5127 Infectious Diseases
- 2009,2012-2016,2016-2023,2023
  GMS5905 Special Topics in Biomedical Sciences

- 2013-2014,2016
  DEN6681 Craniofacial Pathobiology
- 2014-2016,2024
  GMS6140 Principles of Immunology
- 2012-2015
  DEN6680 Principles and Craniofacial Biology and Emerging Therapies
- 2011,2014
  DEN5013 Found Professionalism
- 2014
  DEN6910 Supervised Research
- 2011-2014
  DEN6971 Research for Master's Thesis
- 2011-2012
  DEN6421C Perio Tx Plan
- 2011-2012
  GMS6161 Introduction to Oral Biology II
- 2010-2011
  IDH4905 Individual Work
- 2011
  GMS6160 Introduction to Oral Biology I

Teaching Philosophy

While at the University of North Carolina at Chapel Hill (UNC-CH), East Carolina University (ECU), and University of Florida (UF), I had the privilege to teach several types of students including predoctoral (DMD) and postdoctoral (MS) dental students as well as PhD students. I contribute to the teaching missions primarily through teaching didactic courses in the DMD, advanced education and PhD curricula. The format of these courses ranged from small-group learning to passive learning (i.e. lecture) to flipped classroom learning. In my experience, each teaching methodology has a place within the education system to impart the knowledge and skill sets needed for learners to progress. For instance, in Host Defense, there is a level of information that needs to be provided prior to asking students to utilize this information in critical thinking exercises, thus passive learning is the most appropriate way to convey this information. Conversely, in more upper level courses it is understood that this fundamental knowledge has been gained and thus we ask students to utilize this information during critical thinking exercises using small groups. Finally, in our Grant Writing course a flipped classroom works best whereby students utilize both their foundation of knowledge and critical thinking skills to evaluate their own writing and that of others. One final aspect of my teaching philosophy is that which addresses the heterogeneity of learning styles identified in today's learners. Simply, there is not one teaching method that will satisfy all learning styles or all learners, but it is the responsibility of educators to recognize and respect that there are multiple types of learners. I also have had the opportunity to advise and mentor future scientists at all levels through their research experiences. Here again each type of student requires a different mentoring style and/or philosophy. This is in part because there are several levels of commitment to research which range from simple curiosity to incorporation of research into their career goals. Therefore, I work to match students' needs and capabilities to ensure success in the research endeavor. Here my philosophy is that students should demonstrate an ability to read the literature, know the deficiencies in their area of research, be able to design a research plan to answer the question(s) left by these deficiencies, execute the plan, troubleshoot any problems which arise and analyze and interpret the data. In my career to date, I have successfully advised and mentored 40+ undergraduates, graduated four pre-doctoral fellows and two MS students, while also having a history of serving on over 50

graduate student committees. Finally, I have also held several administrative positions to assess dental educational experiences, including curriculum committees, CODA self-study teams and student performance committees. These experiences have left me with novel skill sets, tools, and approaches to education that I am eager to share with other educators.

Research Profile

The Wallet Laboratory's research interests are focused on mechanisms associated with altered innate immune functions, which lead to dysregulated adaptive immunity. Most recently, our research program has grown due to productive collaborations in pulmonary mucosal immunology. As such, our program has four major arms integrated through with a central philosophy. Specifically, our laboratory focuses on the contribution of epithelial cell biology and signaling to innate and adaptive immune homeostasis and dysfunction. We study the contribution of what I term 'epithelial cell innate immune (dys)function' to five major disease conditions: SARS-CoV-2 infection, burn and inhalation injury, periodontal disease (autoinflammation), type 1 diabetes (autoimmunity), and pancreatic cancer. While appearing to be a diverse research program, we have found that many of the mechanisms and systems in play are surprisingly (or maybe not so surprisingly) similar allowing for rapid translation of our findings. Importantly, previous investigations into the role of epithelial cells in immunobiology have been hindered by a lack of robust primary cell culture techniques, which our laboratory has been able to overcome using both animal and human tissues. Thus, using our novel and unique tools we are able to evaluate our findings in the human conditions, again making translation of our findings that much more feasible. Below are brief yet more detailed descriptions of each arm of my research program:

1. SARS-CoV2 & COVID COVD-19 pathogenesis can take a mild course with few or no symptoms, resembling other common upper respiratory diseases, whereby individuals do not require hospitalization and typically recover within two weeks. In some cases, disease progresses to a more severe state requiring hospitalization and often results in acute respiratory distress syndrome (ARDS) and death. Although the severity of COVID-19 can vary, it is clear the severity is due to the type and magnitude of the immunological response to SARS-CoV2 infection. There is very little information about the independent and cooperative effects of all phases of the immune response: innate and adaptive, namely humoral (B cell/serologic), and cellular (T cell) and their clinical sequelae. We are focusing our investigations on defining unique and overlapping mechanisms of the mucosal immune-mediated pathogenesis of COVID-19 which lead to mild and severe disease. The nature and magnitude of an innate immune response to a pathogen directly affects the nature and magnitude of the specific humoral response systemically and in the pathogen-tropic tissue (i.e. Oronasopharynx/lung). Resulting antibodies also shape many subsequent innate immune functions of canonical immune cells such as neutrophils and macrophages, as well as barrier functions of mucosal cells such as epithelial cells. The additive effect of these interactions directly impacts the short- and long-lived Ab repertoire. We hypothesize that the magnitude and nature of the bi-directional interactions between innate immunity and the antibody repertoire regulate the severity of COVID-19. We predict that in mild disease, innate immune response in sites of early infection (e.g. oronasopharynx), the subsequent mucosal Ab repertoire and its interactions with innate immunity cause effective viral clearance and appropriate immune resolution. However, in severe disease, innate immune response, mucosal Ab repertoire and the subsequent interactions are inappropriately amplified leading to tissue damage, barrier failure and further viral spread. To address this hypothesis, we are

using our expertise in developing peripheral and mucosal immune signatures and indices used as clinical biomarkers to: 1) Unveil the innate immunological landscape in the systemic and mucosal compartments across the span of natural disease in SARS-CoV2+ individuals with mild and severe disease. 2) Identify innate immune signatures which promote protective vs. pathogenic Ab repertoires and develop serologic innate immunological signatures (SISS) which are representative of mild and severe disease. We have extensive experience in defining mechanisms of innate immune dysfunction at mucosal We are also using this expertise to: 1) Delineate how polyclonal (pAb) and monoclonal Ab (mAb) repertoires from mild and severe disease modulate neutrophil, macrophage, and mucosal epithelial cell innate immune functions and to 2) Evaluate the effect of these Ab repertoires on the SARS-CoV2 induced systemic and mucosal innate immunological landscapes using murine models of COVID-19.

2. In periodontal disease (PD), subgingival bacteria initiate and sustain a non-resolving inflammation that is ineffective at controlling the infection. In addition, PD-associated inflammation is multifactorial in nature, whereby immune plasticity in both the innate and adaptive immune compartments have been observed. Immune plasticity during infection may impart deleterious outcomes during PD, as a consequence of chronic inflammation; however, the cues that drive this plasticity, and what the precise impact this plasticity has on proper immune cell activation and function during PD is at best poorly understood. Our preliminary findings suggest that oral epithelial cells (OECs) serve as a source of inflammatory cues capable of regulating the not only oral bone loss in response to poly-microbial infection, but plasticity of both innate and adaptive immune cells. In addition, we recently reported that the macrophage immune response to PD-associated bacteria challenge is directly influenced by its prior activation by inflammatory stimuli (i.e. polarization). Further, poorly orchestrated T cell activation is appreciated as a key facet of PD. With this information, we propose that activation of the oral epithelium drives an inflammatory milieu, which directly and indirectly shapes innate and adaptive immune plasticity in PD. Thus, our laboratory uses integrated approaches to elucidate the mechanistic features of OCE-elicited immune signaling on macrophage plasticity and functional handling of PD-associated pathogens. In addition, the outcomes of direct and indirect mechanisms of OEC tuning of the T cell compartment are evaluated. Lastly, macrophage-T cell crosstalk under PD pathogen-elicited inflammatory and immunoregulatory environments are investigated. We implement a combinatorial approach employing in vitro and in vivo experimentation to detail the cellular interactions at key checkpoints in host response to infection. The results from these studies will set the foundation for the contribution of immune plasticity and its role in shaping the immunologic response guiding PD, providing critical insight for future studies targeting therapeutic intervention approaches for PD based on immunologic tuning of the host response to PD-associated pathogens. Therapeutics/treatments: Current treatment of chronic periodontitis involves mechanical removal of supra- and sub-gingival bacterial plaque and calculus. This process is known as scaling and root planing (SRP) and is often accompanied by the use of locally administered adjunctive therapies. However, locally applied antibiotics and/or the use of chlorhexidine rinses only result in modest outcomes. Antibiotics are often avoided as most compounds are unable to penetrate biofilms16 and lead to bacterial resistance. Likewise, chlorhexidine stains teeth and causes desquamation, altered taste, and increased calcified deposits. Effective treatment modalities for periodontal disease are needed to preserve oral and systemic health. Indeed, there is a profound need to develop an adjunctive therapeutic that: a) possesses broad-spectrum activity against periodontopathogens with minimal toxicity to mammalian cells; b) penetrates tissue and biofilms to act on subgingival plaque; c) provides anti-inflammatory activity; and, d) promotes tissue healing. Here our group is taking a two-pronged approach to address this void in the field and are collaborating with biomedical engineers at UF and chemists at UNC-CH. With our collaborators at UF, an enzyme fusion protein using indoleamine 2,3-dioxygenase 1 (IDO) and galectin-3 (G3) has been developed,

with the goal of creating a tissue-anchored and thus localized immunomodulatory nanomedicine. G3 is a carbohydrate-binding protein, as a means to restrict enzyme diffusion via binding to tissue glycans. G3 binds N-acetyllactosamine and other β-galactoside glycans, as well as glycosaminoglycans, abundant components in mammalian tissues, whereby IDO and the resultant production of kynurenine metabolites is a general regulator of inflammation in response to sterile and pathogenic inflammatory stimuli. Similarly with our collaborators at UNC-CH, To facilitate pharmacological applications of NO-releasing polymers, the development of macromolecular NO donors that store and spontaneously release therapeutically relevant levels of NO at physiological pH has incorporated hyaluronic acid (HA) to allow for sustained delivery. We hypothesize that this dual-action local acting therapeutic that improves clearance of periodontal pathogens (nitric oxide; NO) and promotes wound healing (hyaluronic acid; HA) represents a novel treatment paradigm for periodontal diseases that will reduce the frequency and/or augment SRP interventions, promoting improved resolution of disease especially in compromised patient populations (e.g., those afflicted with diabetes).

2. Burn and Inhalation Injury. The American Burn Association estimates that ~450,000 people in the United States will suffer cutaneous burn injury each year, resulting in ~3,500 deaths. There are multiple influences on morbidity and mortality in burn patients, with inhalation injury and sex among the most significant. Combined burn and inhalation (B+I) injury occurs in 5-30% of all burn patients and leads to increased acute lung injury (ALI) and subsequent bacterial infections with the associated increased morbidity and mortality. Burn injury induces an influx of highly activated innate immune cells (primarily neutrophils and macrophages) to the pulmonary vasculature where they are poised to protect the airway from infection. This recruitment is a double-edged sword in that these highly activated immune cells, upon further stimulation (such as inhalation injury) have the potential to contribute to local tissue damage. Indeed, B+I injury has an acute two-fold effect in the lungs: local tissue hypoxia and local tissue damage. Two key hallmarks of hypoxia are 1) induction of cell death and 2) formation of further tissue-damaging and immune-activating radical oxygen species (ROS) by resident immune cells which leads to further inflammation and recruitment. This cycle of tissue damage and immune cell recruitment/activation is difficult to control leading to the morbidity and mortality associated with burn injury.

Our collaborators have previously demonstrated in human samples and a mouse model that inhalation injury generates the release of numerous Damage-Associated Molecular Patterns (DAMPs). Pulmonary innate immune cells, as well as non-canonical immune responder cells (e.g. epithelial cells), bind DAMPs through Toll-Like Receptors (TLRs). These interactions induce further ROS, inflammatory cytokines and chemokines which results in additional damage and immune recruitment. The overall objective of this line of investigation is to delineate mechanisms needed to break the cycle of uncontrolled inflammation in order to define appropriate treatments to restore tissue homeostasis and prevent the associated morbidity and mortality. Again, our group takes multiple approaches to address this void in the field. Most currently, we are evaluating the role of Nuclear Factor-Erythroid-2-Related Factor (NRF2) in this immune regulation. ‘Cellular stress’ including hypoxia and immune activation induces the dissociation of the repressive Keap1 (Kelch-like ECH-Associated Protein 1)-NRF2 complex, allowing NRF2 to translocate to the nucleus and promote transcription of anti-oxidant, superoxide de-toxifying, and anti-inflammatory immune gene. Our preliminary data demonstrate that Nrf2-/- knockout mice have profound mortality after B+I injury with significant increases in pulmonary epithelial cell shedding, edema and elevated local inflammatory cytokine. However, our preliminary data also demonstrate that pulmonary immune cell NRF2 protein transcription is rapidly increased after B+I, although protein is not translocated to the nucleus resulting in decreased transcription of downstream antioxidant genes. Our collaborators have published that exogenous pharmacological activation

of NRF2 in immune cells from burn patients significantly reduced expression of inflammatory mediators responsible for immune cell recruitment and associated with poor outcome of injury. Nuclear receptors, such as estrogen (E2) receptors (ERs), have been described as a significant inhibitor of NRF2 translocation, although their contribution to restoring homeostasis following burn injury has not been evaluated. This may be of importance in females, as they have significantly higher rates of inpatient mortality after B+I injury, although the current understanding of the interplay between sex hormones, immune function, and burn injury is limited. We propose that the NRF2-mediated homeostasis following burn and inhalation injury is insufficient, but that pharmacological activation of the NRF2 pathway can reduce the acute oxidative damage and hyper-inflammation providing a novel therapeutic approach to improve long term outcomes following B+I injury. To address our hypotheses, we are using our animal model of burn and inhalation injury, which accurately recapitulates hypoxia, immune dysfunction, and susceptibility to bacterial infection and 2) clinical samples collected from burn and B+I inpatients at the North Carolina Jaycee Burn Center to evaluate the translatability of defined mechanisms and intervention as well as evaluate indices to predict poor patient outcomes.

3. Type 1 Diabetes (T1D) is an autoimmune disease with both genetic and environmental contributions, yet a single environmental factor has yet to be identified. We propose that rather than the specific identity of the environmental factor, the way in which an environmental signal is received is most important to disease. Among individuals at risk of developing T1D, seroconversion to islet autoantibody (IA) production is the best and earliest biological predictor of disease progression. Thus, critical events leading to the destructive β cell autoimmunity associated with T1D are linked to the development of IAs. In subjects with high risk HLA alleles, 9 gene regions marked by single nucleotide polymorphisms (SNPs) are associated with IA seroconversion. Our collaborator's analysis of these IA risk loci reveals enrichment for genes/proteins that regulate only three cell signaling pathways (RAS/MAPK, PI3K/AKT and JAK/STAT), whereby four IA seroconversion risk variants (PTPN22, SH2B3, ERBB3 and UBASH3A) each impact all three. Thus, we propose that regulation of environmental sensing is at least in part controlled by the one or more of these risk variants associated with the development of IAs. Appropriate communication of the gastrointestinal tract (GI) with the environment is required for local and systemic immunologic homeostasis, which is in part regulated by the intestinal epithelial cell (IEC) through their physical and biochemical interactions with innate and adaptive immune populations. To this end, contribution of immunity within the GI tract to the expression of autoimmunity at distal sites has been previously demonstrated. Thus, we propose that the seroconversion risk variants affect immune signaling pathways within the IEC contributing to the maintenance of autoimmunity in T1D, through the secondary expansion and/or polarization of autoreactive effector T-cells. To study human disease, a major need is cell-specific human isogenic systems that can be used to dissect the role of individual T1D risk gene variant alleles. Our preliminary studies support use of a disease-in-a-dish strategy that pairs human induced pluripotent stem cells (iPSCs) with CRISPR/Cas9 gene modification to fulfill these needs. Thus, our laboratory couples iPSCs and CRISPR/Cas9 technology with our method of reproducible primary human IECs culture to achieve haploinsufficient, mono-allelic homozygosity at risk or protective alleles to evaluate alterations in IEC-immune mechanisms which contribute to the maintenance of β cell autoimmunity.

4. Pancreatic cancer (PC) will be the second leading cause of cancer-associated deaths by 2020. PC is resistant to conventional chemoradiation regimens and clinical trials evaluating new agents continue to demonstrate unacceptably high rates of failure. As promising alternatives, therapies designed to stimulate antitumor immune responses have achieved unprecedented success with other solid tumors. Vaccination, one immunotherapeutic modality applied to PC, has unfortunately achieved a limited degree of success. Similarly, immune checkpoint inhibition has also achieved promising results in solid tumors, but again clinical

outcomes following checkpoint inhibition alone in PC have been disappointing. Thus, it has been proposed that immune modulation may not be a viable option in PC, where it has been hypothesized that in cancers with a limited array of somatic mutations, such as PC, development of fewer neoantigens prevent the development of a vast enough repertoire of tumor-specific T cells to be effective in mounting antitumor responses. Yet others have demonstrated that patients with gastrointestinal tumors, including PC, do actually harbor neoantigen-reactive T cells. Most importantly, expansion of these neoantigen-reactive T cells ex vivo and subsequent adoptive cell therapy (ACT) resulted in stable regression of a metastatic cholangiocarcinoma, previously resistant to multiple chemotherapy regimens. Comparable clinical responses in PC would revolutionize the current treatment paradigm, significantly expanding the population of patients eligible for potentially curative therapy. We are keenly aware, that expansion of PC-reactive T cells ex vivo and subsequent ACT will not be sufficient to treat PC due to a tumor micro-environment of tolerance. Indeed, we recently published, that while cancer cells exert mastery over the local tumor-associated stroma (TAS) to configure escape from immunity, the TAS also contributes to the production of an immunosuppressive tumor microenvironment. As outlined in our recent review, we propose that these combined effects at least in part contribute to the observed failure of vaccine mediated in vivo expansion of tumor-specific T cells, as well as mono-modal checkpoint inhibitor therapies. We hypothesize that ACT will need to be coupled with appropriate checkpoint inhibition and/or vaccination to be effective in the treatment of PC, by providing both a robust population of highly functional and tumor-specific T cells as well as inhibition/reversion of T cell tolerance/ exhaustion in the tumor microenvironment. Thus, our program is currently evaluating the frequency, phenotype and function of ex vivo expanded PC-reactive T cells, using platforms that can be used in a heterogeneous patient population as well as in a more personalized manner. In addition, we will utilize and expand upon the existing knowledge on the PC-tumor microenvironment of tolerance to inform the selection of appropriate checkpoint inhibitors to be evaluated as adjunctive mono- and multi-modal therapies to ACT. Most importantly, we will translate ACT and the appropriate adjunctive multi-modal checkpoint inhibitor therapies into clinical trial. We have assembled an interdisciplinary team each with unique expertise to reach the overarching goal of our research program – to develop effective and translational immunotherapy for PC. We are making strides towards this goal utilizing models and tools currently in our laboratories to do so in the human condition.

In addition to my primary research objectives, my collaborative research programs, have allowed me to be involved, at some level, in investigating the basic biology of health, multiple autoimmune conditions, autoinflammation, sepsis, and exercise induced inflammation that have resulted in 95+ total publications to date. I have been blessed with the opportunities to couple my passions and expertise with that of others to bring together multiple research communities with the goal of advancing human health and hope to be able to continue to do so for years to come.

Open Researcher and Contributor ID (ORCID)

0000-0003-4650-651X

Publications

2024

Exosomal microRNA following severe trauma: Role in bone marrow dysfunction.

The journal of trauma and acute care surgery. 96(4):548-556 [DOI] 10.1097/TA.0000000000004225. [PMID] 38151766.

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2024

Gut mycobiome dysbiosis after sepsis and trauma.

Critical care (London, England). 28(1) [DOI] 10.1186/s13054-023-04780-4. [PMID] 38212826.

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2023

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Shock (Augusta, Ga.). 59(2):180-189 [DOI] 10.1097/SHK.0000000000002023. [PMID] 36516458.

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2023

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2023

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2018

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Grants

Sep 2023 ACTIVE

BA220315: Extracellular vesicles as Biomarkers, Mediators Of Immune Dysfunction and Universal Countermeasures against Radiation Syndrome and Associated Polytrauma

Role: Co-Investigator
Funding: US ARMY MED RES AND DEVELOPMENT COMMAND

May 2023 ACTIVE

Nitric oxide-releasing hyaluronic acid therapeutics for treating periodontal disease

Role: Principal Investigator
Funding: UNIV OF NORTH CAROLINA CHAPEL HILL via NATL INST OF HLTH NIDCR

Jan 2023 ACTIVE

Nitric oxide-releasing Glycosaminoglycans for treating complex wounds

Role: Principal Investigator
Funding: UNIV OF NORTH CAROLINA CHAPEL HILL via NATL INST OF HLTH NIDDK

Sep 2022 ACTIVE

Multi-modal rescue of pulmonary NRF2-insufficiency after burn and burn + inhalation injury to regulate innate immune dysfunction

Role: Co-Investigator
Funding: NATL INST OF HLTH NIGMS

Aug 2017 – Jul 2021

Tissue-Targeted Enzyme for Localized Tryptophan Catabolism to Direct Subcutaneous and Oral Mucosal Inflammatory Responses

Role: Project Manager
Funding: NATL INST OF HLTH NIDCR

Mar 2017 – Dec 2020

Initiating Mechanisms of Cancer Cachexia

Role: Project Manager
Funding: FL DEPT OF HLTH BANKHEAD-COLEY CANCER RE

Jul 2016 – Jun 2021

Functional role of skeletal muscle in the innate immune response to sepsis

Role: Project Manager
Funding: NATL INST OF HLTH NIGMS

Apr 2016 – Feb 2021

The CD226 amd TIGIT costimulatory Axis in Type 1

Role: Project Manager
Funding: NATL INST OF HLTH NIDDK

Sep 2015 – Aug 2022

Regulatory Mechanisms Controlling Expression of P. gingivalis Surface Structures

Role: Project Manager
Funding: NATL INST OF HLTH NIDCR

Apr 2015 – Jun 2021

L-Arg Availability Affects the Physiological State of Porphyromonas gingivalis

Role: Project Manager
Funding: NATL INST OF HLTH NIDCR

Feb 2015 – Jan 2020

Role of Intestinal Bacteria in Human Norovirus Infection

Role: Project Manager

Funding: NATL INST OF HLTH NIAID

- **Jun 2014 – Nov 2021**

  Mechanisms and Treatment Response of Aggressive Periodontitis in Children

  Role: Project Manager
  Funding: NATL INST OF HLTH NIDCR

- **Jun 2014 – May 2016**

  Intestinal Epithelial Cell Contributions to Permissiveness of Norovirus Infection of B cells

  Role: Principal Investigator
  Funding: UF DSR OPPORTUNITY FUND

- **Jul 2013 – Jun 2018**

  Epithelial Cell Function in the Progression of Periodontal Disease

  Role: Principal Investigator
  Funding: NATL INST OF HLTH NIDCR

- **Jan 2012 – Dec 2016**

  Institutional Research Grant

  Role: Project Manager
  Funding: AMERICAN CANCER SOCIETY

- **Sep 2011 – Nov 2017**

  The effects of aging on experimental models of pain inhibition and facilitation

  Role: Project Manager
  Funding: NATL INST OF HLTH NIA

- **Jul 2011 – Jun 2016**

  Osteclast Function in T1D

  Role: Principal Investigator
  Funding: AMER DIABETES ASSOCIATION

- **Sep 2010 – Jun 2017**

  Substance abuse and immunity in HIV+ adolescents by systems biology

  Role: Project Manager
  Funding: NATL INST OF HLTH NIDA

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