Member: Sari Acra, MD, MPH
Rank: Professor, Director of the Division of Pediatric Gastroenterology
Department: Pediatrics
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

My major research focus is nutrition, and recent clinical nutrition research in our group has focused on measuring energy expenditure in chronic disease states. We are also defining the molecular mechanisms underpinning chronic diarrhea in children. A representative publication is: Gaibee Z, Warner N, Bugda Gwilt K, Li W, Guan R, Yourshaw M, Whittaker Hawkins R, Zorbas C, St-Germain J, Tabatabaie M, Mao S, Pinsk V, Yerushalmi B, Wang LK, Nelson SF, Wozniak L, Shouval DS, Matar M, Assa A, Frost N, Jimenez L, **Acra S**, Walters T, Mouat S, Li M, Lafontaine DLJ, Tyska M, Raught B, Avitzur Y, Lencer WI, Goldenring JR, Martín MG, Thiagarajah JR, Muise AM. The genetic architecture of congenital diarrhea and enteropathy. *N Engl J Med*. 2025;392(13):1297-1309. PMCID: PMC11968080

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
RC2DK118640	Congenital diarrhea and enteropathy (PediCODE) consortium and biorepository	09/15/2019-08/31/2029	Not PI or MPI

Summary of Digestive Disease Research

My research measures energy metabolism in children using whole-room indirect calorimeters (metabolic chambers). This system couples movement sensors to provide a near-free-living environment in which a study subject or patient's energy expenditure, substrate oxidation, physical activity, and body composition are measured accurately on a minute-by-minute basis. Through this approach, we investigate the physiological regulation of energy metabolism in children with inflammatory bowel disease and other chronic disease states, while improving technologies for measuring body composition and physical activity. I have also collaborated with other VDDRC members to study the etiology of chronic diarrhea in children.

VDDRC-Related Training and Activities

Former Pilot & Feasibility Award Recipient
Retreat Speaker

VDDRC Collaborations

Supported by my preliminary VDDRC Pilot-funded work in conjunction with VDDRC member Dr. Goldenring, we hypothesized that identification of specific cellular abnormalities in children with Undiagnosed Neonatal Diarrhea (UDND) syndrome, whole-genome sequencing of affected neonates and their parents, and studying the disease by recapitulating it in defined biologic systems would lead to insights into the causes of UDND cases, and potentially lead to individualized treatments. This work is now funded as a U2C NIDDK grant with Dr. Goldenring serving as Co-Principal Investigator. We have also collaborated with Drs. Choksi, Goettel, Hiremath, Kaji, Peek, Tyska, Washington, Williams, K. Wilson, Yan, and many other VDDRC investigators. A collaborative VDDRC publication is Kaur H, Ali SA, Short SP, **Williams CS**, **Goettel JA**, **Washington MK**, **Peek RM**, **Acra SA**, **Yan F**. Identification of a functional peptide of a probiotic bacterium-derived protein for the sustained effect on preventing colitis. *Gut Microbes*. 2023;15(2):2264456. PMCID: PMC10566403.

Future VDDRC Core Use

We will utilize the services of the Cell Imaging and Flow Cytometry Cores for detailed examination of samples from children with UDND syndrome. We will also continue to use the Biostatistical Component for database development, statistical analysis, interpretation of whole-exome findings, and collaboration on presenting results.

Member: Samuel Bailin, MD, MSCI
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

I am a new Associate Member of the VDDRC. I completed my Internal Medicine residency and Infectious Diseases fellowship at VUMC and joined as faculty in 2021. My work specializes in caring for persons living with HIV, and my translational research program focuses on understanding the pathophysiology of metabolic diseases in persons living with HIV. A representative publication is: **Bailin SS**, Kropski JA, Gangula RD, Hannah L, Simmons JD, Mashayekhi M, Ye F, Fan R, Mallal S, Warren CM, Kalams SA, Gabriel CL, Wanjalla CN, Koethe JR. Changes in subcutaneous white adipose tissue cellular composition and molecular programs underlie glucose intolerance in persons with HIV. *Front Immunol*. 2023:14:1152003. PMCID: PMC10499182.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
K23DK135414	Adipose tissue T cell polarization and metabolic health in persons living with HIV	04/01/2023-01/31/2027	$171,700

Summary of Digestive Disease Research

Adipose tissue is a critical regulator of systemic metabolic processes, and changes in adipose tissue immune cell populations that modulate adipose tissue function may, in part, contribute to the risk of metabolic disease in persons with HIV (PWH). Defining the mechanisms through which adipose tissue innate and adaptive immune cells contribute to metabolic diseases is critical for identifying preventative and therapeutic strategies that reduce morbidity and mortality in PWH. I used single-cell RNA sequencing (scRNA-seq) to define adipose tissue compositional and transcriptional changes in PWH using cross-sectional data and compared these changes with diabetic HIV-negative adipose tissue. I found that higher proportions of CD4+ and CD8+ T effector memory (TEM) cells and lipid-associated macrophages (LAMs) were associated with glucose intolerance in PWH, and that higher CD4+ and CD8+ TEM proportions were associated with more fibroblastic cells in PWH only. Our overall hypothesis is that proinflammatory TEM cells expressing interferon-γ and tumor necrosis factor α are increased in PWH on ART, infiltrate adipose tissue and promote macrophage polarization, are associated with the adipose tissue viral reservoir, and contribute to the development of metabolic disease.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Pilot & Feasibility Project Reviewer
Retreat Speaker

VDDRC Collaborations

Although I am a new VDDRC member, I have already developed productive collaborations with other VDDRC members, including Drs. Below, Gabriel, Koethe, Mashayekhi, Meenderink, Niswender, and Wanjalla. A representative VDDRC collaborative publication is: **Bailin SS**, **Gabriel CL**, Gangula RD, Hannah L, Nair S, Carr JJ, Terry JG, Silver HJ, Simmons JD, **Mashayekhi M**, Kalams SA, Mallal S, Kropski JA, **Wanjalla CN**, **Koethe JR**. Single-cell analysis of subcutaneous fat reveals profibrotic cells that correlate with visceral adiposity in HIV. *J Clin Endocrinol Metab*. 2024;110(1):238-253. PMCID: PMC11651702.

Future VDDRC Core Use

I will continue to utilize the Biostatistical Component and plan to use the Cell Imaging and Flow Cytometry Cores.

Member: Curtis Gabriel, MD, PhD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

I am a new VDDRC member and currently an Assistant Professor of Medicine in the Division of Gastroenterology, Hepatology and Nutrition. During graduate school, I worked with Amy S. Major to investigate the pathogenesis of fatty liver disease in a mouse model of systemic lupus erythematosus. After medical school, I completed my internal medicine residency and gastroenterology fellowship in the PSTP program at Vanderbilt. My current research focuses on the mechanisms that give rise to steatotic liver disease in people with HIV. A representative publication is: Bailin SS, Ma S, Perry AS, Terry JG, Carr JJ, Nair S, Silver HJ, Shi M, Mashayekhi M, Kropski JA, Ferguson JF, Wanjalla CN, Das SR, Shah R, Koethe JR, **Gabriel CL**. The primacy of adipose tissue gene expression and plasma lipidome in cardiometabolic disease in persons with HIV. *J Infect Dis*. 2025;231(2):e407-e418. PMCID: PMC11841643.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
K23DK135420	The gut-liver axis in HIV-related non-alcoholic fatty liver disease	09/01/2023-05/31/2027	$169,813
VDDRC P&F Award P30DK058404	Fecal lipidomics in HIV-related MASLD	06/01/2026-05/31/2027	Pilot funding not listed

Summary of Digestive Disease Research

Liver disease is a leading cause of mortality among persons with HIV (PWH), and PWH bear a disproportionate burden of metabolic dysfunction-associated steatotic liver disease (MASLD). Although the underlying mechanisms are not well understood, intestinal dysbiosis and intestinal barrier dysfunction may contribute to MASLD pathogenesis in PWH. HIV infection alters the intestinal microbiome, modifies the plasma metabolome, and impairs intestinal barrier function; however, data on the microbiome and metabolome in PWH with MASLD remain limited. My research has shown that hepatic steatosis is associated with differences in key phospholipid levels, including phosphatidylcholine, in PWH with MASLD. My ongoing research focuses on elucidating the links between the intestinal microbiome and the plasma lipidome in PWH with MASLD.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Retreat Speaker
Seminar Speaker

VDDRC Collaborations

Although I am a new VDDRC member, I have already developed fruitful collaborations with other VDDRC members, including Drs. Bailin, Below, Brown, Koethe, Mashayekhi, Meenderink, Niswender, and Wanjalla. A representative VDDRC collaborative publication is: **Wanjalla CN**, **Gabriel CL**, Fuseini H, **Bailin SS**, **Mashayekhi M**, Simmons J, Warren CM, Glass DR, Oakes J, Gangula R, Wilfong E, Priest S, Temu T, Newell EW, Pakala S, Kalams SA, Gianella S, Smith D, Harrison DG, Mallal SA, **Koethe JR**. CD4+ T cells expressing CX3CR1, GPR56, with variable CD57 are associated with cardiometabolic diseases in persons with HIV. *Front Immunol*. 2023:14:1099356. PMCID: PMC9971959.

Future VDDRC Core Use

I will continue to utilize the Biostatistical Component and the Translational Analysis Core. Future research projects will utilize the Cell Imaging and MS/Omics Cores to define the pathogenesis of MASLD in PWH at a molecular and cellular level.

Member: Clint M. Hasenour, PhD
Rank: Research Assistant Professor
Department: Molecular Physiology and Biophysics
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

The liver is the major organ where glucose biosynthesis is coupled to mitochondrial metabolism. Previous studies demonstrate that overnutrition accelerates whole-body gluconeogenesis (GNG) and citric acid cycle (CAC) activity *in vivo*. A limitation of prior research is that the unique contributions of the liver to whole-body GNG and CAC fluxes have been difficult to disentangle *in vivo*. The inability to discern hepatic from renal metabolic fluxes represents a significant knowledge gap, as obesity may not only cause an ectopic accumulation of lipid but also an "ectopic redistribution" of gluconeogenic function. A representative publication is: Bednarski TK, Rahim M, **Hasenour CM**, Banerjee DR, Trenary IA, Wasserman DH, Young JD. Pharmacological SERCA activation limits diet-induced steatohepatitis and restores liver metabolic function in mice. *J Lipid Res*. 2024 Jun;65(6):100558. PMCID: PMC11179628.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
K01DK135924	*In vivo* hepato-renal metabolic flux dysregulation in obesity	04/01/2023-02/28/2027	$111,658
R03DK147764	Factors controlling metabolic plasticity in the kidney during obesity and injury	05/14/2026-03/31/2028	$75,000

Summary of Digestive Disease Research

Our research group investigates the etiology and treatment of metabolic disease through the lens of multi-organ fluxomics while focusing on an understudied aspect of gluco(dys)regulation. This work matters because it will identify organ-specific metabolic nodes that may be better targets for improving glycemic control and reducing liver damage.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member

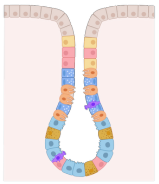
VDDRC Collaborations

I am a new member of the VDDRC, and our group has begun to establish collaborations with VDDRC members, including Drs. Bailin, Gabriel, and Winn.

Future VDDRC Core Use

I will continue using the Biostatistical Component and expand my studies to include the Cell Imaging and MS/Omics Cores.

Member: Mirazul Islam, PhD
Rank: Postdoctoral Fellow
Department: Medicine and Cell and Developmental Biology
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

I am a postdoctoral fellow in Dr. Robert Coffey's laboratory. Our group has contributed to the understanding of development and disease along the entire GI tract. One VDDRC publication is: **Islam M**, Yang Y, Simmons AJ, Shah VM, Musale KP, Xu Y, Tasneem N, Chen Z, Trinh LT, Molina P, Ramirez-Solano MA, Sadien ID, Dou J, Rolong A, Chen K, Magnuson MA, Rathmell JC, Macara IG, Winton DJ, Liu Q, Zafar H, Kalhor R, Church GM, Shrubsole MJ, Coffey RJ, Lau KS. Temporal recording of mammalian development and precancer. *Nature*. 2024;634(8036):1187-1195. PMCID: PMC11525190.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
VDDRC P&F Award P30DK058404	Role of BTG2 in maintaining quiescence of human LGR5^{+} colonic stem cells	07/30/2026-06/30/2027	Pilot funding not listed
GI SPORE Pilot Project Award P50CA236733	Role of BTG2 and LGR5^{+} stem cells in colorectal cancer	01/14/2026-07/31/2027	Pilot funding not listed
GI SPORE Career Enhancement Award P50CA236733	Development of a TOB2 reporter platform for investigating dormant cell states in colorectal cancer	07/01/2026-06/30/2027	Pilot funding not listed

Summary of Digestive Disease Research

Our group is focused on the study of the role of the EGF receptor (EGFR) and its ligands in GI diseases. We have a particular interest in investigating the trafficking of EGFR ligands in polarizing colonic epithelial cells as well as stem cells within the context of the development of colorectal diseases.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle

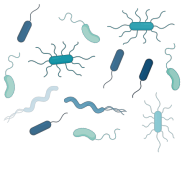
VDDRC Collaborations

I have collaborated extensively with multiple VDDRC members. I have worked closely with Drs. Coffey, Lau, Magnuson, Markham, and Shrubsole. A representative collaborative publication is **Islam M**, Bechard ME, Yang Y, Simmons AJ, Xu Y, Higginbotham JN, Zhao P, Cao Z, Tasneem N, Glass SE, **Markham NO**, Revetta F, Ramirez-Solano MA, Liu Q, Franklin JL, **Lau KS**, **Coffey RJ**. Unbiased recording of clonal potency reveals species-specific regulation of mammalian intestine. *bioRxiv*. 2025:2025.06.10.658707. PMCID: PMC12259079.

Future VDDRC Core Use

I will continue to use the Biostatistical Component, and we will continue to rely heavily on the Cell Imaging, Flow Cytometry, MS/Omics, and Translational Analysis Cores for our studies in novel *ex vivo* human models (organoids) and mouse models of disease.

Member: Shannon Kordus, PhD
Rank: Research Instructor
Department: Pathology, Microbiology and Immunology, and Biochemistry
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

I am a Research Instructor in Borden Lacy's laboratory. Our laboratory focuses on understanding the molecular mechanisms by which bacterial protein toxins contribute to human disease. Specific interests include defining the virulence properties of the *C. difficile* toxins and the development of novel strategies for *C. difficile* infection prevention and therapy. A recent VDDRC-supported publication is: **Kordus SL**, Kroh HK, Rodríguez RC, Shrem RA, Peritore-Galve FC, Shupe JA, Wadzinski BE, Lacy DB, Spiller BW. Nanobodies against *C. difficile* TcdA and TcdB reveal unexpected neutralizing epitopes and provide a toolkit for toxin quantitation *in vivo*. *PLoS Pathog.* 2023;19(10):e1011496. PMCID: PMC10621975.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
VDDRC P&F Award P30DK058404	Determing host signals that trigger toxin release in *Clostridiodes difficile*	07/30/2026-06/30/2027	Pilot funding not listed

Summary of Digestive Disease Research

The primary focus of our laboratory has been to understand the structural biology of the three toxins produced by *C. difficile* and the roles these toxins play in pathogenesis. Most of this work has been focused on TcdA and TcdB, two homologous glucosyltransferase toxins that are considered the major virulence factors in *C. difficile* infection. More recently, we have studied the *C. difficile* transferase (CDT) toxin, a third toxin that can affect disease severity.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Named Supported Investigator

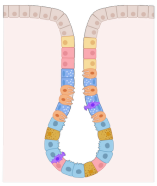
VDDRC Collaborations

I collaborate with Drs. Lacy, Washington, and Markham on projects related to human tissue, cell, and animal models of *C. difficile* toxin function. A recently published collaborative paper is: Nabukhotna K, **Kordus SL**, Shupe JA, Cano Rodríguez R, Smith A, Bohannon JK, **Washington MK**, **Lacy DB**. Purified CDT toxins and a clean deletion within the CDT locus provide novel insights into the contribution of binary toxin in cellular inflammation and *Clostridioides difficile* infection. *PLoS Pathog*. 2024;20(9):e1012568. PMCID: PMC11444381.

Future VDDRC Core Use

I will continue to use the Biostatistical Component, and our lab will continue to use the Cell Imaging, Flow Cytometry, MS/Omics, and Translational Analysis Cores to support our independent and collaborative projects on elucidating and defining the structural basis for gastrointestinal microbial toxins and to evaluate our models of infection.

Member: Mark Magnuson, MD
Rank: Professor
Department: Molecular Physiology and Biophysics
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

My laboratory uses genetically altered mice, next-generation sequencing, and bioinformatics to study the regulation and function of genes that play critical roles in development and cell function. A representative publication is: Trinh LT, Osipovich AB, Liu B, Shrestha S, Cartailler JP, Wright CVE, **Magnuson MA**. Single-cell RNA sequencing of Sox17-expressing lineages reveals distinct gene regulatory networks and dynamic developmental trajectories. *Stem Cells*. 2023;41(6):643-657. PMCID: PMC10465087.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
VDDRC P&F Award P30DK058404	Molecular basis for Sox17 control of biliary system development	06/01/2025-05/31/2027	Pilot funding not listed

Summary of Digestive Disease Research

The transcription factor SOX17 contributes to hepatopancreatic-biliary system formation and vascular-hematopoietic emergence. To better understand Sox17 function in the developing endoderm and endothelium, we developed a dual-color temporal lineage-tracing strategy in mice combined with single-cell RNA sequencing to analyze 6934 cells from Sox17-expressing lineages at embryonic days 9.0-9.5. Our analyses showed 19 distinct cellular clusters combined from all 3 germ layers. Differential gene expression, trajectory, and RNA-velocity analyses of endothelial cells revealed a heterogeneous population of uncommitted and specialized endothelial subtypes, including 2 hemogenic populations that arise from different origins. Similarly, analyses of posterior foregut endoderm revealed subsets of hepatic, pancreatic, and biliary progenitors with overlapping developmental potency. Calculated gene-regulatory networks predict gene regulons that are dominated by cell-type-specific transcription factors unique to each lineage. Vastly different Sox17 regulons in endoderm versus endothelial cells support differential interactions between SOX17 and other regulatory factors, enabling lineage-specific regulatory actions.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Director, Complementary Awards Program-Supported Genome Editing Core
Pilot & Feasibility Award Recipient in the current funding cycle

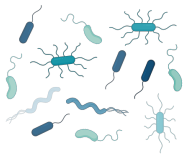
VDDRC Collaborations

My laboratory has developed collaborations with other VDDRC members, including Drs. Coffey, Islam, Lau, and Shrubsole. A representative collaborative publication is: **Islam M**, Yang Y, Simmons AJ, Shah VM, Musale KP, Xu Y, Tasneem N, Chen Z, Trinh LT, Molina P, Ramirez-Solano MA, Sadien ID, Dou J, Rolong A, Chen K, **Magnuson MA**, Rathmell JC, Macara IG, Winton DJ, Liu Q, Zafar H, Kalhor R, Church GM, **Shrubsole MJ**, **Coffey RJ**, **Lau KS**. Temporal recording of mammalian development and precancer. *Nature*. 2024;634(8036):1187-1195. PMCID: PMC11525190.

Future VDDRC Core Use

I will continue to use the Biostatistical Component and Flow Cytometry Core and plan to use the MS/Omics Core in the next funding cycle.

Member: Nicholas O. Markham, MD, PhD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

I previously received a Pilot and Feasibility Award and was a former member of the Academy of Investigators within the VDDRC. My interests include understanding how gastrointestinal epithelial cells interact with microbial toxins and how changes in the gut microbiome affect human diseases, such as colon carcinogenesis. One representative VDDRC publication is: Lunnemann HM, Shealy NG, Reyzer ML, Shupe JA, Green EH, Siddiqi U, Lacy DB, Byndloss MX, **Markham NO**. Cecum axis (CecAx) preservation reveals physiological and pathological gradients in mouse gastrointestinal epithelium. *Gut Microbes*. 2023;15(1):2185029. PMCID: PMC10012889.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
Veterans Administration IK2BX005699	Gastrointestinal cell type-specific signaling and *C. difficile* toxin pathogenesis	10/01/2021-09/30/2026	$315,450

Summary of Digestive Disease Research

Data from my VDDRC Pilot indicate that the *C. difficile* toxin TcdB transactivates EGFR, which is essential for stem cell function. I have now optimized a murine intrarectal toxin-instillation model, allowing our group to study TcdB's effects on EGFR and stem cell function. In a separate but related project, we have used spatial transcriptomics and multiplex immunofluorescence to show that early-stage colorectal cancers contain rich bacterial niches filled with species, like *C. difficile*, that are relatively low-abundance but disproportionately strong effectors of tumor transcriptional programming.

VDDRC-Related Training and Activities

Academy of Investigators Member
Pilot & Feasibility Award Recipient in the last 10 years
Pilot & Feasibility Project Reviewer
Retreat Poster Judge
Seminar Speaker

VDDRC Collaborations

I have ongoing collaborations with numerous VDDRC members, including Drs. Algood, Byndloss, Choksi, Coburn, Coffey, Goettel, Goldenring, Kaji, Lacy, Lau, Nicholson, Piazuelo, Shrubsole, Skaar, Singh, Washington, Williams, K. Wilson, and Zhu. A recent VDDRC collaborative publication is: Green EH, Kotrannavar SR, Rutherford ME, Lunnemann HM, Kaur H, Heiser CN, Ding H, Simmons AJ, Liu X, **Lacy DB**, **Washington MK**, **Shrubsole MJ**, Liu Q, **Lau KS**, Sears CL, **Coffey RJ**, Drewes JL, **Markham NO**. Multiomic spatial atlas shows deleted in malignant brain tumors 1 (DMBT1) glycoprotein is lost in colonic dysplasia. *J Pathol*. 2025;266(1):51-65. PMCID: PMC11985286.

Future VDDRC Core Use

I will continue using the Biostatistical Component, Flow Cytometry, MS/Omics, and Translational Analysis Cores to analyze host responses to *C. difficile* toxin. I plan to use the Cell Imaging Core in the next funding cycle.

Member: Mona Mashayekhi, MD, PhD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

My laboratory research focuses on the role of chronic inflammation in obesity-related cardiometabolic diseases. A representative publication is **Mashayekhi M**, Beckman JA, Nian H, Garner EM, Mayfield D, Devin JK, Koethe JR, Brown JD, Cahill KN, Yu C, Silver H, Niswender K, Luther JM, Brown NJ. Comparative effects of weight loss and incretin-based therapies on vascular endothelial function, fibrinolysis and inflammation in individuals with obesity and prediabetes: A randomized controlled trial. *Diabetes Obes Metab*. 2023;25(2):570-580. PMCID: PMC10306232.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
K23HL159351	The effect of SGLT2 inhibition on adipose tissue inflammation and endothelial function	07/01/2023-06/30/2028	$171,700

Summary of Digestive Disease Research

Obesity contributes to heightened chronic inflammation, which promotes the development of cardiometabolic diseases. No current therapies specifically target inflammatory pathways activated in obesity to improve these outcomes. Glucagon-like peptide-1 receptor agonists (GLP1-RAs) are a newer w class of weight-loss medications that reduce cardiometabolic disease beyond what is expected from their glucose- and weight-reduction benefits alone. GLP-1RAs reduce inflammation in animals, but this has not been defined in humans. Our research program aims to characterize whether and how GLP1-RAs reduce obesity-associated inflammation in humans by altering the immune metabolic profile.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Named Supported Investigator
Retreat Speaker
Seminar Speaker

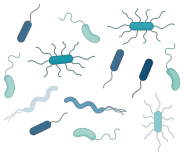
VDDRC Collaborations

I have established productive collaborations with several VDDRC members, including Drs. Ayala, Bailin, Brown, Gabriel, Koethe, Meenderink, Niswender, Samuels, Wanjalla, and Winn. A representative VDDRC collaborative publication is **Mashayekhi M**, Safa BI, Nian H, Devin JK, Gamboa JL, Yu C, Chen R, Beckman JA, **Koethe JR**, Silver HJ, **Niswender K**, Luther JM, Brown NJ. RISING STARS: Effects of a GLP-1 receptor polymorphism on responses to liraglutide. *J Endocrinol*. 2025;267(1):e250174. PMCID: PMC12556785.

Future VDDRC Core Use

I will continue to utilize the Biostatistical Component and Flow Cytometry Cores in my research. I plan to utilize the Cell Imaging Core in the next funding cycle.

Member: Leslie Meenderink, MD, PhD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

I am a new VDDRC member. I conducted undergraduate research at Yale University in Dr. Paul Forscher's laboratory, investigating cytoskeleton dynamics in neuronal growth cones. During my MD/PhD at Vanderbilt University, my PhD research in Dr. Steve Hanks' lab focused on the role of p130Cas signaling in focal adhesion dynamics and cellular motility. During my internal medicine residency, infectious disease fellowship, and postdoctoral research, I used live-cell imaging in Dr. Matthew Tyska's laboratory to understand the mechanisms underlying assembly of the intestinal brush border. Since starting my own laboratory at VUMC, I have focused on how the intestinal epithelium establishes and maintains a barrier against intestinal microbes. Using human tissue samples, mouse models, and cell culture, along with advanced light and electron microscopy, our research seeks to understand how the intestinal epithelium balances commensal bacteria and defends against pathogenic microbes. A representative publication is: Cowell RP, Bickart AC, Shashi SR, Vasquez A Jr, Brackman LC, Abdel-Dayem MA, Chen W, Bowman DM, Krystofiak ES, Essalki Y, Gross I, Harrison DG, Wanjalla CN, Algood HMS, Tyska MJ, **Meenderink LM**. Brush border intermicrovillar adhesion limits bacteria attachment to the small intestine brush border. *bioRxiv*. 2025:2025.11.25.690363. PMCID: PMC12772999.

Summary of Digestive Disease Research

My current research aims to understand how the gut's microscopic surface architecture is disrupted in, and contributes to, IBD pathogenesis and bacterial infection. Microvilli (MV) are the initial site of contact between intestinal epithelial cells and luminal bacteria. Perturbations in MV structure and density result in nutrient malabsorption and osmotic diarrhea as seen in IBD and infection with pathogenic bacteria. A recent study showed that even in uninflamed tissue, patients with Crohn's disease have lower levels of MV structural proteins. These findings imply MV defects are not only present in IBD during active inflammation but also persist despite therapy and may contribute to relapse. Interestingly, our data demonstrate that mice lacking the MV structural protein CDHR2 recapitulate several of the epithelial defects seen in Crohn's patients, providing a unique *in vivo* model system. Our laboratory is now addressing the hypothesis that defects in normal enterocyte apical surface structure and function persist in the absence of active inflammation and provide a path for microbial entry, driving inflammation, infection, and disease relapse. Through this work, we will identify defects in enterocyte structure in Crohn's patients and characterize how these defects contribute to persistent changes in the microbial content. These epithelial structural defects may provide a new target for therapeutic intervention.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Retreat Speaker

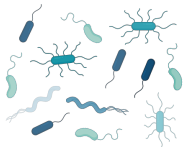
VDDRC Collaborations

I have established productive collaborations with several VDDRC members, including Drs. Algood, Bailin, Choksi, Gabriel, Goettel, Goldenring, Koethe, Lau, Mashayekhi, Piazuelo, Tyska, Wanjalla, Washington, and Williams. A representative VDDRC collaborative publication is: Short SP, Brown RE, Chen Z, Pilat JM, McElligott BA, **Meenderink LM**, Bickart AC, Blunt KM, Jacobse J, Wang J, Simmons AJ, Xu Y, Yang Y, Parang B, **Choksi YA**, **Goettel JA**, **Lau KS**, Hiebert SW, **Williams CS**. MTGR1 is required to maintain small intestinal stem cell populations. *Cell Death Differ*. 2024;31(9):1170-1183. PMCID: PMC11369156.

Future VDDRC Core Use

I will continue to use the Biostatistical Component and the Flow Cytometry Core. Future studies will utilize the Cell Imaging, MS/Omics, and Translational Analysis Cores.

Member: Maribeth R. Nicholson, MD, MPH
Rank: Associate Professor
Department: Pediatrics
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

My research program focuses on the clinical and translational evaluation of *C. difficile* infections (CDI) in children and adults, with a focus on the role of the intestinal microbiome, diagnostic challenges, and vaccine development. A representative publication is **Nicholson MR**, Donskey CJ. Multistep testing algorithms for *Clostridioides difficile* infection. *JAMA*. 2023;330(10):966-967. PMCID: PMC10528531.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
Vedanta Biosciences	A randomized, double-blind, placebo-controlled phase 3 study of VE303 for prevention of recurrent *Clostridioides difficile* infection: The restorative303 study	05/29/2024-05/28/2029	$161,496

Summary of Digestive Disease Research

I have established a translational research program centered on *C. difficile* infections in children and adults. Our studies have prospectively enrolled pediatric and adult patients, collecting serum and fecal samples to examine the intestinal microbiome, anti-toxin antibody responses, and the prevalence/immune reactions to *C. difficile* colonization in patients with additional comorbidities. We have then used these human observations to inform *C. difficile* vaccine development through collaborations within the VDDRC.

VDDRC-Related Training and Activities

Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Retreat Poster Judge
Seminar Speaker

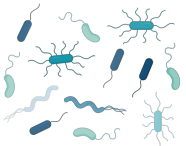
VDDRC Collaborations

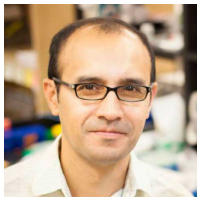
I have ongoing collaborations with VDDRC members Drs. Acra, Byndloss, Lacy, Markham, Markle, Olivares-Villagomez, Skaar, and Washington. An example collaborative VDDRC publication is: Thomas AK, Peritore-Galve FC, Ehni AG, Lança BBC, Coggin J, Brady EJ, Yoder SM, Shrem R, Rodríguez RC, Kroh HK, Gibson-Corley KN, **Washington MK**, **Olivares-Villagómez D**, Creech CB, **Nicholson MR**, Spiller BW, **Lacy DB**. Mucosal vaccination clears *Clostridioides difficile* colonization. *Nature*. 2026. Online ahead of print. PMID: 41708852.

Future VDDRC Core Use

I will continue to use the Biostatistical Component and the MS/Omics Core for our clinical and translational work and have applied for a competitive extension of my VDDRC Pilot & Feasibility Award.

Member: Danyvid Olivares-Villagómez, PhD
Rank: Senior Research Scientist
Department: Medicine
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

My research focuses on osteopontin, a ubiquitous protein found in many tissues and organs. This protein is also present in breast milk. Our group is studying the importance of breast milk-derived osteopontin in intestinal homeostasis. We also study the importance of a commensal, *Turicibacter sanguinis*, during intestinal inflammation. One example of VDDRC-sponsored publications: 1) Hoek KL, McClanahan KG, Latour YL, Shealy N, Piazuelo MB, Vallance BA, Byndloss MX, Wilson KT, **Olivares-Villagómez D**. Turicibacterales protect mice from severe *Citrobacter rodentium* infection. *Infect Immun*. 2023;91(11):e0032223. PMCID: PMC10652940.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
R21AI187749	*Turicibacter* modifies intestinal infection severity by modulating intestinal metabolites	08/01/2025-07/31/2027	Not PI or MPI

Summary of Digestive Disease Research

My research focuses on immune responses mediated by cells associated with the intestinal epithelium, particularly intraepithelial lymphocytes (IELs). I am particularly interested in the interactions between different IEL populations and the protein osteopontin. I am also interested in how these cells respond during intestinal bacterial infections.

VDDRC-Related Training and Activities

Academy of Investigators, Former Member
Pilot & Feasibility Award Recipient in the current funding cycle
Retreat Poster Judge

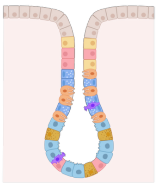
VDDRC Collaborations

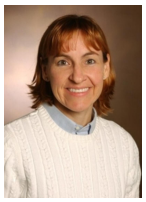
We collaborate with many VDDRC members, including Drs. Algood, Behringer, Byndloss, Cover, Lacy, Nicholson, Piazuelo, Washington, Weitkamp, K. Wilson, and Zhu, for various aspects of our research focused on IEL biology. An example of these VDDRC collaborations is represented in the following publication: Thomas AK, Peritore-Galve FC, Ehni AG, Lança BBC, Coggin J, Brady EJ, Yoder SM, Shrem R, Rodríguez RC, Kroh HK, Gibson-Corley KN, **Washington MK**, **Olivares-Villagómez D**, Creech CB, **Nicholson MR**, Spiller BW, **Lacy DB**. Mucosal vaccination clears *Clostridioides difficile* colonization. *Nature.* 2026;652(8112):1289-1297. PMCID: PMC13128438.

Future VDDRC Core Use

We will continue to rely significantly on the Flow Cytometry Core for enriching subsets of immune cell populations by flow sorting, as well as the Translational Analysis Core. We will also continue utilizing the Biostatistical Component for all our studies.

Member: Maria Blanca Piazuelo, MD
Rank: Research Associate Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

I serve as Associate Director of the VDDRC Translational Analysis Core and will provide expertise in GI pathology. My primary research interest is pre-malignant lesion development in the stomach and colon. A representative VDDRC publication is Gobert AP, Hawkins CV, Williams KJ, Snyder LA, Barry DP, Asim M, Allaman MM, McNamara KM, Delgado AG, Wang Y, Zhao S, Rose KL, **Piazuelo MB**, Wilson KT. Hypusination in intestinal epithelial cells protects mice from infectious colitis. *Gut Microbes*. 2024;16(1):2438828. PMCID: PMC11649231.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
P01CA116087	*H. pylori*-induced inflammation and gastric cancer	01/01/2009-02/28/2029	Listed under Contact PI

Summary of Digestive Disease Research

My research interests focus on morphological alterations in inflammatory and precancerous processes in the gastrointestinal tract in human tissues and animal models. Our group has conducted studies in Colombia comparing the population in a high gastric cancer risk area (in the Andes Mountains) with an area of low gastric cancer risk on the Pacific coast. Both populations have a very high prevalence of *H. pylori* infection. With a multidisciplinary team, we found that the phylogeographic origin of *H. pylori* strains infecting participants was associated with the severity of gastric histopathological lesions and oxidative damage to the gastric mucosa. Additional studies concluded that *H. pylori* strains from the high-risk area have a greater ability to induce oxidative stress and DNA damage than strains from the low-risk area.

VDDRC-Related Training and Activities

Director, Histopathology Service in the Translational Analysis Core
Former Pilot & Feasibility Award Recipient
Pilot & Feasibility Project Reviewer

VDDRC Collaborations

I collaborate extensively with multiple VDDRC investigators, serving as a pathologist on numerous studies evaluating gastrointestinal inflammatory response and precancerous lesions in human subjects and in rodent models. Specifically, I have ongoing collaborations with Drs. Algood, Byndloss, Cassat, Choksi, Coburn, Cover, Goettel, Markham, Meenderink, Olivares-Villagomez, Peek, Schneider, Washington, Williams, and K. Wilson, among many others. One VDDRC collaborative publication is: Brackman LC, Jung MS, Green EH, Joshi N, Revetta FL, McClain MS, **Markham NO**, **Piazuelo MB**, **Algood HM**. IL-17 signaling protects against *Helicobacter pylori*-induced gastric cancer. *Gut Microbes*. 2024;16(1):2430421. PMCID: PMC11639209.

Future VDDRC Core Use

I will continue to use the Biostatistical Component and Translational Analysis Core to evaluate GI inflammation and injury in rodent models and human samples.

Member: Jason Samuels, MD
Rank: Assistant Professor
Department: Surgery
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

I lead clinical trials investigating multimodal treatment strategies for obesity and have extensive experience caring for over 500 bariatric surgery patients. Given the wide-ranging impact of obesity on health outcomes, a critical need exists to explore the efficacy of adjuvant weight loss therapies after gastric bypass surgery. Tirzepatide (TRZ), a glucagon-like peptide-1 receptor agonist, shows remarkable effectiveness in medical obesity, with 25% weight loss after sustained therapy. However, nearly two-thirds of patients taking medications like TRZ have mild to moderate gastrointestinal (GI) symptoms, including nausea, vomiting, and abdominal pain. These medication side effects could be a consequence of gastroparesis via vagal stimulation of the stomach and may represent a major driver of weight loss. Limited data exist on the use of newer agents, such as TRZ, in patients who have undergone gastric bypass, which disrupts the vagal nerves that manage food transit and gastric emptying. My research focuses on this major, timely gap: whether gastric bypass surgery might mitigate these GI symptoms while enabling enhanced weight loss with adjuvant TRZ use in the postoperative period. A representative publication is **Samuels JM**, Ye F, Irlmeier R, Silver H, Srivastava G, Spann M. Real-world titration, persistence & weight loss of semaglutide and tirzepatide in an academic obesity clinic. *Diabetes Obes Metab* 2025;27(11):6200-6209. PMCID: PMC12515774.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
K23DK143312	Use of adjuvant anti-obesity medications to enhance surgical weight loss	09/09/2025-07/31/2030	$163,800
Intuitive Foundation 2024-4335	Comparative analysis of skeletal muscle adaptations and gastrointestinal adverse effects following bariatric surgery versus GLP-1 receptor agonist therapy	07/01/2025-06/30/2026	$60,000
American College of Surgeons	Use of adjuvant anti-obesity medications to enhance surgical weight loss	07/01/2025-06/30/2027	$40,000

Summary of Digestive Disease Research

My clinical and translational research focuses on using the anti-obesity medication to enhance surgical weight loss. The aims of this research are to 1) determine the impact of adjuvant TRZ administration on weight, total fat mass, and lean body mass in patients with a history of gastric bypass, 2) investigate the frequency and severity of GI discomfort associated with TRZ, and 3) investigate the impact of TRZ on GI motility in patients with prior Gastric Bypass.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Retreat Speaker

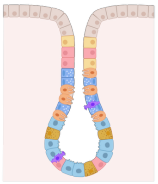
VDDRC Collaborations

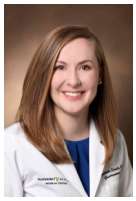
Although I am a new VDDRC member, I have established collaborations with numerous VDDRC members, including Drs. Ayala, Flynn, Mashayekhi, Niswender, and Yu. A representative VDDRC collaborative publication is **Samuels JM**, **Niswender KD**, Roumie CL, Spann MD, **Flynn CR**, Ye F, Blankush J, Irlmeier R, Funk LM, Patel MB. Adverse event comparison between glucagon-like peptide-1 receptor agonists and other anti-obesity medications following bariatric surgery. *Diabetes Obes Metab* 2024;26(9):3906-3913. PMCID: PMC12223922.

Future VDDRC Core Use

I will continue to utilize the Biostatistical Component for data analysis.

Member: Elizabeth A. Scoville, MD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

I am currently an Assistant Professor of Medicine in the Division of Gastroenterology at Vanderbilt University Medical Center. My research interests focus on the mechanisms underlying non-response to treatment in inflammatory bowel disease (IBD), including genetics and inflammatory pathways. I am a former recipient of the VDDRC Pilot Award. A representative publication is: Raffa GA, Tyree RN, Carson K, Allaman MM, Beaulieu DB, Dalal RL, Pabla BS, **Scoville EA**, Horst SN, Schwartz DA, Washington MK, Wilson KT, Coburn LA. Non-invasive determination of disease activity in Crohn's disease by serum luminex profiling. *Sci Rep*. 2026;16(1):8867. PMCID: PMC12988101.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
Crohn's & Colitis Foundation of America	IBD Plexus: Partnering to Accelerate Science SPARC IBD	09/17/2018-01/17/2027	$3,093

Summary of Digestive Disease Research

My research has focused on defining inflammatory mediators in IBD, primarily related to adipose tissue signaling pathways and metabolic changes, and was funded by a VDDRC Pilot and Feasibility Award. Our work has shown that non-targeted serum metabolomic profiling can differentiate between Crohn’s disease, ulcerative colitis, and control patients.

VDDRC-Related Training and Activities

Academy of Investigator, Former Member
Pilot & Feasibility Award Recipient in the last 10 years
Pilot & Feasibility Project Reviewer

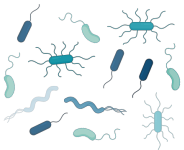
VDDRC Collaborations

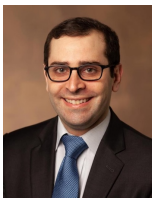
I have several ongoing collaborations with VDDRC members, such as Drs. Choksi, Coburn, Coffey, Goettel, Lau, Piazuelo, Washington, Williams, and K. Wilson. One publication resulting from these VDDRC collaborations is: Li J, Simmons AJ, Hawkins CV, Chiron S, Ramirez-Solano MA, Tasneem N, Kaur H, Xu Y, Revetta F, Vega PN, Bao S, Cui C, Tyree RN, Raber LW, Conner AN, Pilat JM, Jacobse J, McNamara KM, Allaman MM, Raffa GA, Gobert AP, Asim M, **Goettel JA**, **Choksi YA**, Beaulieu DB, Dalal RL, Horst SN, Pabla BS, Huo Y, Landman BA, Roland JT, **Scoville EA**, Schwartz DA, **Washington MK**, Shyr Y, **Wilson KT**, **Coburn LA**, **Lau KS**, Liu Q. Identification and multimodal characterization of a specialized epithelial cell type associated with Crohn's disease. *Nat Commun*. 2024;15(1):7204. PMCID: PMC11339313.

Future VDDRC Core Use

I will continue to use the Biostatistics Component of the VDDRC for statistical analysis of clinical data.

Member: Jeffrey Singer, MD, PhD
Rank: Postdoctoral Fellow
Department: Medicine
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

I am a Postdoctoral Fellow in Eric Skaar's laboratory. My research focuses on mucosal immunology and host/microbe biology in the intestine and liver. One recent VDDRC-supported publication is: Yarosz EL, Kumar A, **Singer JD**, Chang CH. Cullin 3-mediated regulation of intracellular iron homeostasis promotes thymic invariant NKT cell maturation. *Immunohorizons*. 2023;7(3):235-242. PMCID: PMC10122431

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
VDDRC P&F Award P30DK058404	Role of nutritional immunity in chronic liver disease	07/30/2026-06/30/2027	Pilot funding not listed

Summary of Digestive Disease Research

The primary focus of my research has been to understand why patients with decompensated liver disease are highly susceptible to infections, with particular interest in metal acquisition by *Klebsiella pneumoniae*.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle

VDDRC Collaborations

I collaborate with Drs. Peek, Skaar, and Washington on projects related to metal acquisition and human gastrointestinal infections.

Future VDDRC Core Use

We will continue to utilize the Biostatistical Component as well as the Cell Imaging and the Translational Analysis Cores to monitor and quantify inflammation in our animal models of infection, the MS/Omics Core for Imaging Mass Spectrometry, and the Flow Cytometry Core.

Member: Nadia “Markie” Sneed, PhD, APRN, FPN-BC
Rank: Director, Whole Foods Research Lab; Assistant Professor
Department: Nursing
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

I have extensive clinical, academic, and research training in nutrition science, nursing, public health, and patient-centered outcomes research. My research focuses on identifying non-pharmacological nutritional interventions that utilize food as medicine to treat metabolic diseases such as obesity in at-risk families. I am also interested in studying the mechanisms by which ultra-processed foods (UPFs) drive metabolic conditions like obesity by exploring their effects on the gut microbiome using omics-based approaches (e.g., metagenomics, metabolomics). A representative publication is: **Sneed NM**, Ukwuani S, Sommer EC, Samuels LR, Truesdale KP, Matheson D, Noerper TE, Barkin SL, Heerman WJ. Reliability and validity of assigning ultraprocessed food categories to 24-h dietary recall data. *Am J Clin Nutr*. 2023;117(1):182-190. PMCID: PMC10196599.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
DRTC Pilot Award P30DK020593	Whole foods for teens: A pilot dietary intervention to reduce body adiposity in adolescents with obesity.	04/01/2025-03/30/2027	Pilot funding not listed

Summary of Digestive Disease Research:

My research evaluates whole-foods-based dietary strategies to improve metabolic health across the lifespan without caloric restriction. Our lab conducts randomized controlled trials in adults and adolescents to examine how diet composition influences metabolic outcomes–specifically obesity. Through the *Whole Foods for Families* and *Whole Foods for Teens* programs, we integrate clinical, behavioral, and biologic measures - including body composition and gut microbiome profiling - to identify mechanisms linking diet to metabolic health. This work generates critical data to support the development of scalable, family-centered dietary interventions for obesity prevention.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle

VDDRC Collaborations

I am a new VDDRC member and am currently working to establish collaborations with other VDDRC members, including Drs. Mashayekhi, Niswender, and Samuels.

Future VDDRC Core Use

I will continue to utilize the Biostatistical Component for my research studies.

Member: Ashley Spann, MD, MSACI
Rank: Assistant Professor
Department: Medicine and Biomedical Informatics
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

I am currently a board-certified internal medicine physician pursuing combined fellowship training in Clinical Informatics and Gastroenterology, Hepatology and Nutrition at Vanderbilt. My goals are to become dually equipped to apply technology for patients affected by GI illness and liver disease and to impact patient care and clinical research in a novel way that has not yet been done. Particular informatics interests include the use of predictive modeling for improving outcomes and management of patients with end-stage liver disease, and the integration of these models for use in clinical care through clinical decision support tools. A representative publication is: **Spann A**, Bishop KM, Weitkamp AO, Stenner SP, Nelson SD, Izzy M. Clinical decision support automates care gap detection among primary care patients with nonalcoholic fatty liver disease. *Hepatol Commun*. 2023;7(3):e0035. PMCID: PMC9915945.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
American Association for the Study of Liver Diseases	Harold Amos Medical Faculty Development Program Award	07/01/2024-06/30/2028	$105,000
PCORI Award	Comparing low-touch and high-touch multimodal interventions for chronic pain in patients with cirrhosis	04/01/2026-03/31/2032	$75,000

Summary of Digestive Disease Research

I am an academic hepatologist with particular interests in transplant hepatology within an active, practical research program focused on using machine learning methods and health system tools to not only advance care for the treatment of patients with end-stage liver disease, but also to be able to identify patients at risk of disease progression to trigger preventative measures for these patients. Other informatics interests include large-scale data assessment for modeling that could potentially impact liver transplantation and organ allocation.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle

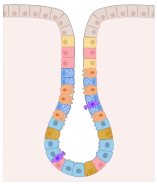
VDDRC Collaborations

Although I am a new VDDRC member, I actively collaborate with several VDDRC members, including Drs. Brittian and Izzy. A representative collaborative publication is: **Spann A**, Bishop K, Marbach S, Ji X, Slaughter J, Weitkamp A, Stenner S, Nelson S, Lopez C, Theobald C, **Izzy M**. Electronic decision aids enhance management of primary care patients with steatotic liver disease: Proof of concept pilot study. *Hepatol Commun*. 2025;9(9):e0794. PMCID: PMC12412744.

Future VDDRC Core Use

I will continue to utilize the Biostatistical Component for my research studies.

PI Name: Marcus C.B. Tan, MBBS, FACS
Rank: Associate Professor
Department: Surgery
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

My long-term interest is centered on the mechanistic understanding and treatment of pancreatic lesions. Specifically, my research focuses on the biology of pre-malignant pancreatic cysts, which I conducted as a VDDRC Pilot and Feasibility Project. A representative VDDRC publication is: Quoc-Huy Trinh V, Ankenbauer KE, Torbit SM, Taranto CP, Liu J, Batardiere M, Kumar B, Maurer HC, Revetta F, Chen Z, Kruse ARS, Judd AM, Copeland C, Wong J, Ben-Levy O, Jarvis B, Brown M, Brown JW, Das K, Makino Y, Spraggins JM, Lau KS, Azadi P, Maitra A, **Tan MCB**, DelGiorno KE. Mutant GNAS drives a pyloric metaplasia with tumor suppressive glycans in intraductal papillary mucinous neoplasia. *Cell Rep*. 2025;44(12):116684. PMCID: PMC12860476.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
UG1CA189828	Comparing the clinical impact of pancreatic cyst surveillance programs	08/31/2020-12/31/2028	Not PI or MPI
R01DK145538	Tuft cells resolve pancreatic injury through a Type 2 Immune circuit	09/01/2026-05/31/2030	Not PI or MPI

Summary of Digestive Disease Research

Our group believes that better understanding the mechanisms underlying pancreatic cyst differentiation and growth will improve identification of lesions at higher risk of malignant transformation, thereby improving patient selection for curative surgery. Towards this end, we are currently defining tumor-nerve interactions that drive the development of pancreatic cystic lesions, work that was funded by the VDDRC Pilot and Feasibility Component.

VDDRC-Related Training and Activities

Academy of Investigators, Former Member
Pilot & Feasibility Award Recipient in the last 10 years

VDDRC Collaborations

Although I am a relatively new member of the VDDRC, my previous participation in the Academy of Investigators sparked several new collaborations with Drs. Arrojo e Drigo, Choi, DelGiorno, Goettel, and Lau. An example of a collaborative VDDRC publication is: Won Y, Jang B, Lee SH, Reyzer ML, Presentation KS, Kim H, Caldwell B, Zhang C, Lee HS, Lee C, Trinh VQ, **Tan MCB**, Kim K, Caprioli RM, **Choi E**. Oncogenic fatty acid metabolism rewires energy supply chain in gastric carcinogenesis. *Gastroenterology*. 2024;166(5):772-786. PMCID: PMC11040571.

Future VDDRC Core Use

We will use the MS/Omics and Translational Analysis Core for our VDDRC Pilot-initiated work and the Cell Imaging Core for future studies.

PI Name: Kasey C. Vickers, PhD
Rank: Associate Professor
Department: Medicine, Molecular Physiology and Biophysics, and Pathology, Microbiology and Immunology
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

My research program focuses on the role(s) of cell-free RNA in intercellular communication and gene regulation in inflammation and lipid metabolism. We have made major contributions to fundamental understanding of extracellular RNA carriers, including lipoproteins, ribonucleoproteins, and non-vesicle extracellular particles. One recent VDDRC-related publication is: Michell DL, Massick C, Koya KS, Cavnar AB, Semler EM, Raby C, Gibson BHY, Moore-Lotridge SN, Zhu W, Ramirez-Solano M, Sheng Q, Allen RM, Linton MF, Schoenecker JG, **Vickers KC**. Plasminogen as a ribonucleoprotein: regulatory small RNA cargo modulates fibrinolysis and inflammation. *Blood*. 2025;146(15):1850-1861. PMCID: PMC12978156.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
P01CA229123	exRNA in colorectal carcinoma: Biogenesis and function	01/20/2020-05/31/2031	Not PI or MPI
R35CA197570	Early to late events in colorectal neoplasia: Basic biology to clinical implementation	04/01/2017-07/31/2031	Not PI or MPI

Summary of Digestive Disease Research

We aim to gain a fundamental understanding of cell-free small RNAs (sRNAs) and their regulators within cell-to-cell communication networks to predict, prevent, and treat metabolic disease. Our laboratory studies the mechanisms and consequences of cell-free sRNA secretion, transport, and biological functions in intestinal inflammation and colorectal carcinogenesis. We have made major contributions to understanding the impact of sRNAs on cholesterol and lipid metabolism. As an independent investigator, my team has demonstrated that lipoproteins and other protein carriers deliver functional host and microbial small RNAs (sRNAs) to recipient cells, where they regulate target gene expression and inflammation. In the last decade, we also defined how this novel endocrine-like pathway is altered in colorectal cancer (CRC), inflammatory bowel disease, and other diseases. Recently, we discovered that bacterial sRNA cargo on circulating LDL particles accumulates at the intestinal mucosa and induces inflammatory macrophages and cytokine secretion within atherosclerotic lesions. Currently, our group is interested in defining the functional role of post-transcriptional RNA modifications on cell-free sRNAs in disease, specifically related to immunological memory of past insults (innate immune training) and intestinal inflammation. Our laboratory is well-suited for both wet and dry work, as we routinely perform and analyze high-end omics studies, including demethylation-based high-throughput sRNA sequencing (sRNA-seq), bulk RNA sequencing, single-cell RNA sequencing, whole-genome DNA sequencing, direct nanopore sequencing, and mass spectrometry-based proteomics, lipidomics, and epitranscriptomics. Our research program has opened new fields of study for cell-free sRNA research and, in the process, developed new tools and concepts that can be extended to other inflammatory diseases, including intestinal inflammation and carcinogenesis.

VDDRC-Related Training and Activities

Academy of Investigators, Former Member; Pilot & Feasibility Award Recipient in the last 10 years; Pilot & Feasibility Project Reviewer

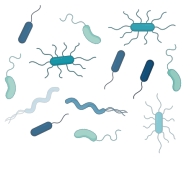
VDDRC Collaborations

I have active collaborations with numerous VDDRC members, including Drs. Algood, Brown, Coffey, Philip, and J. Wilson. A recent representative publication is: Tutanov OS, Massick C, Ramirez M, Higginbotham JN, Jimenez L, Castleberry M, Cao Z, John E, Hamilton MS, Zhang Q, Jeppesen DK, Michell DL, Glass SE, Patel P, Rose KL, Krystofiak E, Chen HC, Sheng Q, Liu Q, Patton JG, Weaver AM, Franklin JL, **Vickers KC**, **Coffey RJ**. A comprehensive analysis of supermere, exomere, and extracellular vesicle isolation and cargo in colorectal cancer. *Cell Rep*. 2025;44(10):116287. PMCID: PMC12632813.

Future VDDRC Core Use

We will continue to use the Cell Imaging, Flow Cytometry, and MS/Omics Cores for comprehensive analysis of extracellular vesicles, lipoproteins, ribonucleoproteins, and NVEPs within digestive disease research projects.

Member: Celestine Wanjalla, MD, PhD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: GII





Rationale for VDDRC Membership

Persistent exposure to HIV antigens and co-infection with chronic herpes viruses such as CMV or hepatitis B/C contribute to chronic inflammation in persons living with HIV (PLWH) and appear to be important in metabolic dysfunction. My laboratory has adopted a multidisciplinary approach to 1) define virus-specific innate and adaptive immune cells in the peripheral blood of persons with metabolic dysfunction using existing multiparameter flow cytometry and single-cell techniques, 2) optimize techniques to better understand the antigenic drivers of the adaptive immune response at the tissue level, and 3) apply/implement changes in metabolic disease risk assessment and therapeutic approaches to improve outcomes in patients. A representative VDDRC publication is: Obare LM, Simmons J, Oakes J, Zhang X, Nochowicz C, Priest S, Bailin SS, Warren CM, Mashayekhi M, Beasley HK, Shao J, Meenderink LM, Sheng Q, Stolze J, Gangula R, Absi T, Ru Su Y, Neikirk K, Chopra A, Gabriel CL, Temu T, Pakala S, Wilfong EM, Gianella S, Phillips EJ, Harrison DG, Hinton A, Kalams SA, Kirabo A, Mallal SA, Koethe JR, **Wanjalla CN**. CD3+ T-cell: CD14+ monocyte complexes are dynamic and increased with HIV and glucose intolerance. *J Immunol*. 2025;214(3):516-531. PMCID: PMC11952877.

Summary of Digestive Disease Research

The HIV reservoir and other co-infections, such as cytomegalovirus (CMV), promote chronic inflammation, which further contributes to accelerated aging. Our overarching hypothesis is that chronic inflammation accelerates aging-related diseases such as metabolic dysfunction in people living with HIV, even in the absence of detectable viral load. Overall, our research aims to identify mechanisms driving aging in people living with HIV, ultimately paving the way for targeted therapies that could slow down aging and reduce comorbidities in this population. Our findings will inform the development of targeted anti-inflammatory therapies, potentially transforming the management of aging-related comorbidities.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Retreat Speaker

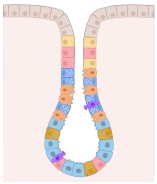
VDDRC Collaborations

I have established collaborations with several VDDRC members, including Drs. Algood, Bailin, Gabriel, Koethe, Lau, Mashayekhi, Meenderink, Niswender, Tyska, and Winn. A representative collaborative publication is: Obare LM, Priest S, Ismail A, Mashayekhi M, Zhang X, Stolze LK, Sheng Q, Nthenge K, Vue Z, Neikirk K, Beasley HK, **Gabriel C**, Temu T, Gianella S, Mallal SA, **Koethe JR**, Hinton A Jr, **Bailin SS**, **Wanjalla CN**. Cytokine and chemokine receptor profiles in adipose tissue vasculature unravel endothelial cell responses in HIV. *J Cell Physiol*. 2024;239(11):e31415. PMCID: PMC12694733.

Future VDDRC Core Use

I will utilize the Flow Cytometry Core and the Biostatistical Component for data analysis.

PI Name: Mary Kay Washington, MD, PhD
Rank: Professor
Department: Pathology, Microbiology and Immunology
VDDRC Status: Associate Member
Research Group: HRP





Rationale for VDDRC Membership

In the VDDRC, I serve as Director of the Translational Analysis Core. My research is focused on the pathology of inflammatory and pre-malignant diseases in the GI tract and the histopathologic assessment of mouse models of GI disease. One of my VDDRC-related publications is: Spiga L, Fansler RT, Wu Y, Grote A, Langford-Butler M, Miller AK, Neal M, Hale OF, Singla D, Calcutt MW, Rose AE, Bresson MM, Schrimpe-Rutledge AC, Berdy B, Codreanu SG, Washington MK, Bratton BP, Sherrod SD, McLean JA, Zengler K, Sears CL, Behringer MG, Gnirke A, Tao L, Livny J, Olivares-Villagómez D, Earl AM, Zhu W. An anaerobic pathogen rewires host metabolism to fuel oxidative growth in the inflamed gut. *Cell*. 2026 Apr 30:S0092-8674(26)00401-0. Online ahead of print. PMCID: PMC13185528.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
P01CA116087	*H. pylori*-induced inflammation and gastric cancer	01/01/2009-02/28/2029	Not PI or MPI

Summary of Digestive Disease Research

I am an experienced diagnostic gastrointestinal pathologist with extensive expertise in collaborative projects, tissue resource administration, and interpreting histopathology in animal models of human disease. I have led several consortia to define the nomenclature, features, and characteristics of premalignant lesions that develop in the GI tract of rodent models.

VDDRC-Related Training and Activities

Director, VDDRC Translational Analysis Core
Retreat Poster Judge
Retreat Speaker

VDDRC Collaborations

I have long-standing collaborative projects with numerous VDDRC members (>40), including Drs. Acra, Algood, Behringer, Cassat, Choksi, Coburn, Coffey, Cover, Flynn, Goettel, Goldenring, Guo, Hiremath, Izzy, Jiang, Kaji, Kordus, Lacy, Lau, E. Lee, Y. Lee, Markham, Markle, Meenderink, Olivares-Villagomez, Peek, Piazuelo, Scoville, Shrubsole, Singh, Williams, K. Wilson, Yan, Zheng, and Zhu. One collaborative VDDRC publication is Heiser CN, Simmons AJ, Revetta F, McKinley ET, Ramirez-Solano MA, Wang J, Kaur H, Shao J, Ayers GD, Wang Y, Glass SE, Tasneem N, Chen Z, Qin Y, Kim W, Rolong A, Chen B, Vega PN, Drewes JL, **Markham NO**, Saleh N, Nikolos F, Vandekar S, Jones AL, **Washington MK**, Roland JT, Chan KS, Schürpf T, Sears CL, Liu Q, **Shrubsole MJ**, **Coffey RJ**, **Lau KS**. Molecular cartography uncovers evolutionary and microenvironmental dynamics in sporadic colorectal tumors. *Cell*. 2023;186(25):5620-5637. PMCID: PMC10756562.

Future VDDRC Core Use

I will continue to use the Biostatistical Component, Translational Analysis, and Cell Imaging Cores for our collaborative studies using rodent models of pre-neoplasia, as well as Imaging Mass Spectrometry within the MS/Omics Core.

Member: Nathan Winn, PhD
Rank: Assistant Professor
Department: Medicine
VDDRC Status: Associate Member
Research Group: OMN



Rationale for VDDRC Membership

My research focuses on the physiological and molecular links between immunometabolism in adipose tissue and metabolic disease. We leverage a combination of transgenic mouse models and surgical manipulations to obtain vascular access for determination of glucose and lipid fluxes. We have developed novel mouse models of yo-yo dieting or 'weight cycling' to study adaptive mechanisms of weight regain on metabolism. My current research aims to define the underlying mechanisms that diminish the capacity to adapt to weight regain. A representative VDDRC publication is: Cottam MA, Caslin HL, **Winn NC**, Hasty AH. Multiomics reveals persistence of obesity-associated immune cell phenotypes in adipose tissue during weight loss and weight regain in mice. *Nat Commun*. 2022 May 26;13(1):2950. PMCID: PMC9135744.

Digestive Diseases-Related Grants

Grant Number	Title	Project Period	Annual Direct Costs
K01DK136926	Uncovering mechanisms of pancreatic adaptability to weight cycling	04/28/2025-07/31/2028	$135,518

Summary of Digestive Disease Research

Most individuals who lose weight regain it within 1-5 years. This 'weight cycling' further increases the risk of metabolic disease compared to weight maintenance. Our group developed a mouse model of weight cycling to uncover mechanisms by which weight regain poses additional risk of metabolic disease. We demonstrated that weight-cycled diet-induced obese (WC-DIO) animals have worse metabolism than equally obese mice that have not been weight-cycled. The study of weight cycling is practical and clinically relevant. Despite the success of contemporary weight loss medicines, adherence to these drugs is <50% after a year of treatment, resulting in rapid weight regain. These clinical and real-world findings of weight rebound underscore the need to unravel the mechanisms by which weight cycling increases metabolic disease risk.

VDDRC-Related Training and Activities

New VDDRC Associate Member
Academy of Investigators Member
Pilot & Feasibility Award Recipient in the current funding cycle
Named Supported Investigator
Retreat Speaker

VDDRC Collaborations

I have established collaborations with other VDDRC members, including Drs. Arrojo e Drigo, Ayala, Flynn, Hasenour, Mashayekhi, Skaar, and Wanjalla. A representative collaborative publication is: Murdoch CC, Weiss A, Enriquez KT, Traina KA, Drury SL, **Winn NC**, Lantier LL, **Skaar EP**. Severe dietary zinc deficiency does not significantly alter energy balance in adult mice. *J Nutr Metab*. 2025:2025:6911386. PMCID: PMC12401606.

Future VDDRC Core Use

We will continue to use the Biostatistical Component and the Cell Imaging Core, and future studies will use the Translational Analysis and MS/Omics Core, which will increase due to the integrative nature of our studies.