Pediatric Gastroenterology Hepatology & Nutrition

M.D. and Ph.D. Fellowship Training Program

Benjamin L. Shneider, M.D.

Section Head

Faith Ihekweazu, M.D. M.S.

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Director of Research Training

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Associate Fellowship Director

Texas Children's Hospital

Baylor College of Medicine

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Message from the Program Director

Baylor College of Medicine/Texas Children's Hospital is home to one of the nation's **premier fellowship programs** in Pediatric Gastroenterology, Hepatology, and Nutrition. As one of the largest programs nationwide, we proudly support 12 fellowship positions. Our fellows gain invaluable experience working directly with **leading national and international experts** across all sub-specialties, including Advanced Hepatology, Transplant Hepatology, Eosinophilic Gastrointestinal disorders, Neurogastroenterology and Motility, Intestinal Rehabilitation, Hepatobiliary and Pancreaticobiliary diseases, Aerodigestive diseases, Celiac disease, Inflammatory Bowel disease, and Advanced Therapeutic Endoscopy. We offer **customizable fellowship tracks** in basic science research, clinical research, and clinical sub-specialties, intentionally designed to align with each fellow's career aspirations. This ensures our graduates are exceptionally well-prepared to become **leaders in the field**. Fellows also benefit from extensive research opportunities, leveraging our **T32 NIH grant**, the Texas Medical Center **Digestive Disease Center**, and the unparalleled resources of the **Texas Medical Center**, the world's largest medical center.

Faith Ihekweazu, M.D., M.S.
Program Director
Pediatric Gastroenterology, Hepatology and Nutrition

Section of Pediatric Gastroenterology, Hepatology, and Nutrition
Baylor College of Medicine, Texas Children's Hospital
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Texas Children's Hospital

U.S.News BEST HOSPITALS RANKINGS

Section of Pediatric Gastroenterology, Hepatology & Nutrition

Baylor College of Medicine

Message from the Head of Section

I am delighted that you are interested in the training programs here at Texas Children's Hospital and the Baylor College of Medicine. Our fellowship offers a tremendous opportunity for both breadth and depth of investigation in clinical, translational and basic investigation. Teaching and investigation are key components of our mission statement, "to strive for outstanding and compassionate patient care through evidence-based and expert practice achieved through research and education." There has been exciting growth in the programs at Texas Children's Hospital and in our Section of Pediatric Gastroenterology, Hepatology and Nutrition. Our faculty of over 40 talented clinicians provide care to children in the Houston metropolitan area (pop. 7,000,000) and we receive referrals from all of Texas, nationally and worldwide.

Our basic research laboratories have been completely renovated and re-equipped with state-of-the-art molecular instrumentation. Clinical research has been reinvigorated in the section with the development of a clinical research coordinator team and we now conduct over 200 IRB approved clinical research protocols. Our teams are actively involved in a number of NIH-funded clinical consortia. We will continue to push the boundaries of our knowledge and our research and clinical practice – we hope you will consider joining us.

Benjamin L. Shneider, M.D.
George Peterkin Endowed Chair
Professor of Pediatrics and Head of Section,
Pediatric Gastroenterology, Hepatology and Nutrition

Message from the Department Chair

Greetings from Houston! I'm Lara Shekerdemian, Chair of Pediatrics at Baylor College of Medicine and Pediatrician In-Chief at Texas Children's Hospital, where I have the privilege of leading the nation's largest department of Pediatrics. Having more than 25 years' experience in clinical and academic medicine and on three continents - 15 of those fantastic years have been at Baylor/Texas Children's Hospital in Houston Texas. Prior to becoming Chair and In-Chief, I was Division Chief of Critical Care Medicine.

Thank you for considering Baylor College of Medicine/ Texas Children's Hospital for your training in Pediatric Gastroenterology, Hepatology & Nutrition. This division includes more than 40 talented faculty members who all contribute to a world-class service, and are eager to be part of your educational journey. Our Department of Pediatrics has more than 1,200 faculty members, giving you the opportunity to work alongside a broad range of subspecialists while caring for patients. Houston, as the energy capital of Texas, attracts people from across the state, the nation, and the world. This rich mix of patients brings a wide spectrum of medical conditions, offering you an exceptional and well-rounded clinical experience. We look forward to sharing more with you about the Department of Pediatrics at Baylor College of Medicine and Texas Children's Hospital.

Lara S. Shekerdemian, MD, MHA
Ralph D. Feigin Endowed Chair and Professor of Pediatrics
Department of Pediatrics, Baylor College of Medicine
Pediatrician-in-Chief, Texas Children's Hospital

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Texas Children's Hospital

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Baylor College of Medicine

BACKGROUND

The Pediatric GI Fellowship Program, established in 1973 by Dr. Buford L. Nichols, offers an unparalleled opportunity to train at the forefront of the field, preparing fellows to become leaders in pediatric digestive health. At Baylor College of Medicine, fellows gain exposure to a vast and diverse patient population through the partnership with Texas Children's Hospital, one of the nation's premier children's hospitals. Our faculty handle over 26,000 outpatient and 3,800 in-patient consultations annually, providing a comprehensive spectrum of pediatric gastrointestinal and liver conditions**.**

Clinical training is enhanced through several Texas Children's Hospital Centers and Clinics:

Gastrointestinal Endoscopy and Therapeutic Endoscopy..... Douglas S. Fishman, M.D., Director
Inflammatory Bowel Disease Program..... Richard Kellermayer, M.D., Director
Hepatology and Liver Transplant Program.....Daniel Leung, M.D., Medical Director
Neurogastroenterology and Motility Program..... Eric Chiou, M.D., Director
Research Training is enhanced by:
NIH Institutional Training Grant (T32 DK07664) .....Douglas Burrin, Ph.D., Director
.....Benjamin Shneider, M.D., Associate Director
Pancreaticobiliary Program.....Douglas S. Fishman, M.D., Director

Subspecialty Clinics:
Aerodigestive Disorders Clinic..... Eric Chiou M.D., Co-Director
Very Early Onset IBD (VEO-IBD) Clinic.....Lina Karam, M.D., Director
Eosinophilic Disorders Clinic..... Anthony Olive, M.D., Co-Director
Multidisciplinary Abdominal Pain Program (MAPP clinic).....Andrew Chu, M.D., Director
Celiac Disease Clinic.....Douglas S. Fishman, M.D., Director
Intestinal Rehabilitation Clinic..... Lynette Van Buren, M.D., Director
Viral Hepatitis Clinic..... Daniel H. Leung, M.D., Director

Research training leverages the expertise of faculty across the Texas Medical Center and is enriched by the extensive research resources available throughout the Medical Center—particularly those of Baylor College of Medicine. Trainees have access to a wide spectrum of research opportunities, from clinical studies—including outcomes and translational research—to cutting-edge basic science laboratories at the forefront of molecular medicine.

OVERVIEW OF TRAINING

Mission Statement - To strive for outstanding and compassionate patient care through evidence-based and expert practice achieved through research and education

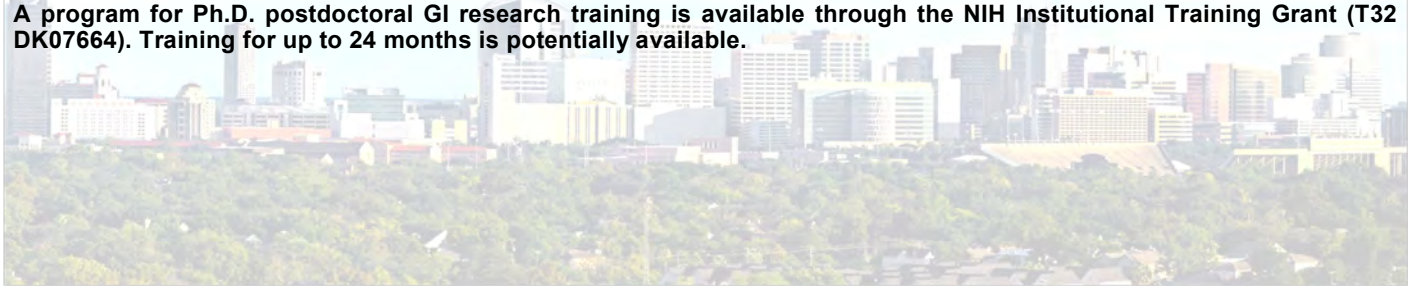
The goal of our Fellowship Program is to educate pediatricians to become outstanding clinicians who also can compete at the cutting edge of research in academia.

The program is designed around the guidelines developed by the North American Society for Pediatric Gastroenterology and Nutrition, and is intended to allow trainees to meet the requirements for certification by the Sub-Board for Pediatric Gastroenterology of the American Board of Pediatrics.

All entrants must have completed the equivalent of three years of ACGME accredited residency training in pediatrics.

The Fellowship Program includes clinical training and research training and is completed by all Fellows. The basic training consists of 12 months of clinical training and 24 months of research training. This 36-month program includes clinical service, teaching, and research. It provides the trainee with the state-of-the-art knowledge and skills required for an academic career.

A program for Ph.D. postdoctoral GI research training is available through the NIH Institutional Training Grant (T32 DK07664). Training for up to 24 months is potentially available.



CLINICAL AND RESEARCH FACULTY

Clinical and Research Faculty
Pediatric Gastroenterology, Hepatology and Nutrition

Chief of Section:
Benjamin L. Shneider, M.D.
Kjersti Andersen, P.A.
Ricardo Arbizu, M.D., M.S.
Ricardo Arbizu Alvarez, M.D.
Kesha Balakrishnan, M.B.B.S.
Anna Banc-Husu, M.D., MSCI
Keisha Barton, M.D.
Donovan Berens, M.D.
Holly Breeden, M.D.
Catherine Brigman, M.D.
Savini Britto, M.D.
Douglas Burrin, Ph.D.
Partha Chakraborty, M.D.
Ojasvini Choudhry Chandon, M.D.
Donna A. Cheung, M.B.B.S.
Misti-Rae Dominguez, P.A.
Donna Garner, R.N., M.S.N.
Sanjiv Harpavat, M.D., Ph.D.
Paula M. Hertel, M.D.
John Hollier, M.D., MS
Faith D. Ihekweazu, M.D., MS
Amy Issa, M.D.
Amir Jazayeri, M.D.
Craig Jensen, M.D.
David Jones, M.D.
Lina B. Karam, M.D.
Isha Kaul, M.D.
Richard E. Kellermayer, M.D., Ph.D.
Kristi D. King, M.P.H., R.D., L.D.
Seiji Kitagawa, M.D.
Daniel H. Leung, M.D.
Krupa Mysore, M.D., M.S.
Geoffrey A. Preidis, M.D., Ph.D.
Anthony P. Olivé, M.D.
Yen Pham, M.D.'
Anitta Philip, M.D.
Priya Raj, MS, M.D.
Naseem Ravanbakhsh, M.D.
Tanisha Richards, MSN, APRN, FNP-C
Henry Shiau, M.D.
Lainey Song-Grant, P.A.
Krishnakant Soni, Ph.D.
Mary Elizabeth Tessier, M.D.
Nicole D. Triggs, MSN, APRN, CPNP
Kristin Whitfield Van Buren, M.D.
Bryan S. Vartabedian, M.D.
Seema Mehta Walsh, M.D.
Allyson N. Wyatt, MD

Associated NIH Training Grant Research Faculty

David Allison, Ph.D……Pediatrics, Children's Nutrition Research Center
Robert A. Britton, Ph.D…… Molecular Virology and Microbiology
Shelly Buffington, Ph.D……..Center for Precision Environmental Health
Hashem B. El-Serag, M.D, M.P.H…… Medicine, Gastroenterology and Health Services
Mary K. Estes, Ph.D..…….. Medicine, Molecular Virology & Microbiology
Gregory Guthrie, Ph.D……...Pediatrics, Children's Nutrition Research Center
Amy Hair, M.D……Pediatrics, Neonatology
Joseph Hyser, Ph.D..…….. Medicine, Molecular Virology & Microbiology
Hamed Jafar-Nejad, M.D…… Molecular and Human Genetics
Fong Lam, M.D……...Pediatrics, Critical Care Medicine
Brendan Lee, M.D., Ph.D…… Pediatrics, Molecular and Human Genetics
Fasiha Kanwal, M.D.,M.S.H.S……..Medicine, Gastroenterology and Health Services
Brendan Lee, M.D., Ph.D……..Human Molecular Genetics
Andrea McAlester, Ph.D……Pathology and Immunology
Jason Mills, M.D., Ph.D……Medicine, Gastroenterology and Health Services
Sasirekha Ramani, Ph.D……...Medicine, Molecular Virology & Microbiology
J. Marc Rhoads, M.D…… Pediatric Gastroenterology– *University of Texas Medical School*
Tor Savidge, Ph.D…… Pathology and Immunology
Lanlan Shen, Ph.D…….... Pediatrics, Children's Nutrition Research Center
Allison Speer, M.D……..Pediatrics, Surgery—*University of Texas Medical School*
Caitlin Vonderohe, D.V.M., Ph.D…….Pediatrics, Children's Nutrition Research Center
Jill Weatherhead, M.D., Ph.D……...Tropical Medicine, Infectious Disease
Ruth Wooten-Kee, Ph.D……Pediatrics, Children's Nutrition Research Center

M.D. RESEARCH TRAINING / COURSEWORK

SCHEDULING OF TRAINING

Clinical and research months may be allocated in various ways. The first year of training always will have 12 weeks of research distributed in 2 week blocks among the clinical months to identify their research mentor and project area.

	Clinical Training	Research Training
Year 1	**Inpatient Clinical Training = 20 weeks Consults** **Liver Service = 13 weeks & Outpatient Clinic = 6 weeks**	**12 weeks (6 X 2 week rotations)** **(Includes conference and vacation) ***
Year 2	**Inpatient Consult Service = 6 weeks** **Fellows Continuity Clinic = Half day per week**	**46 weeks**
Year 3	**Inpatient Consult Service = 6 weeks** **Fellows Continuity Clinic = Half day per week**	**46 weeks**

COURSEWORK

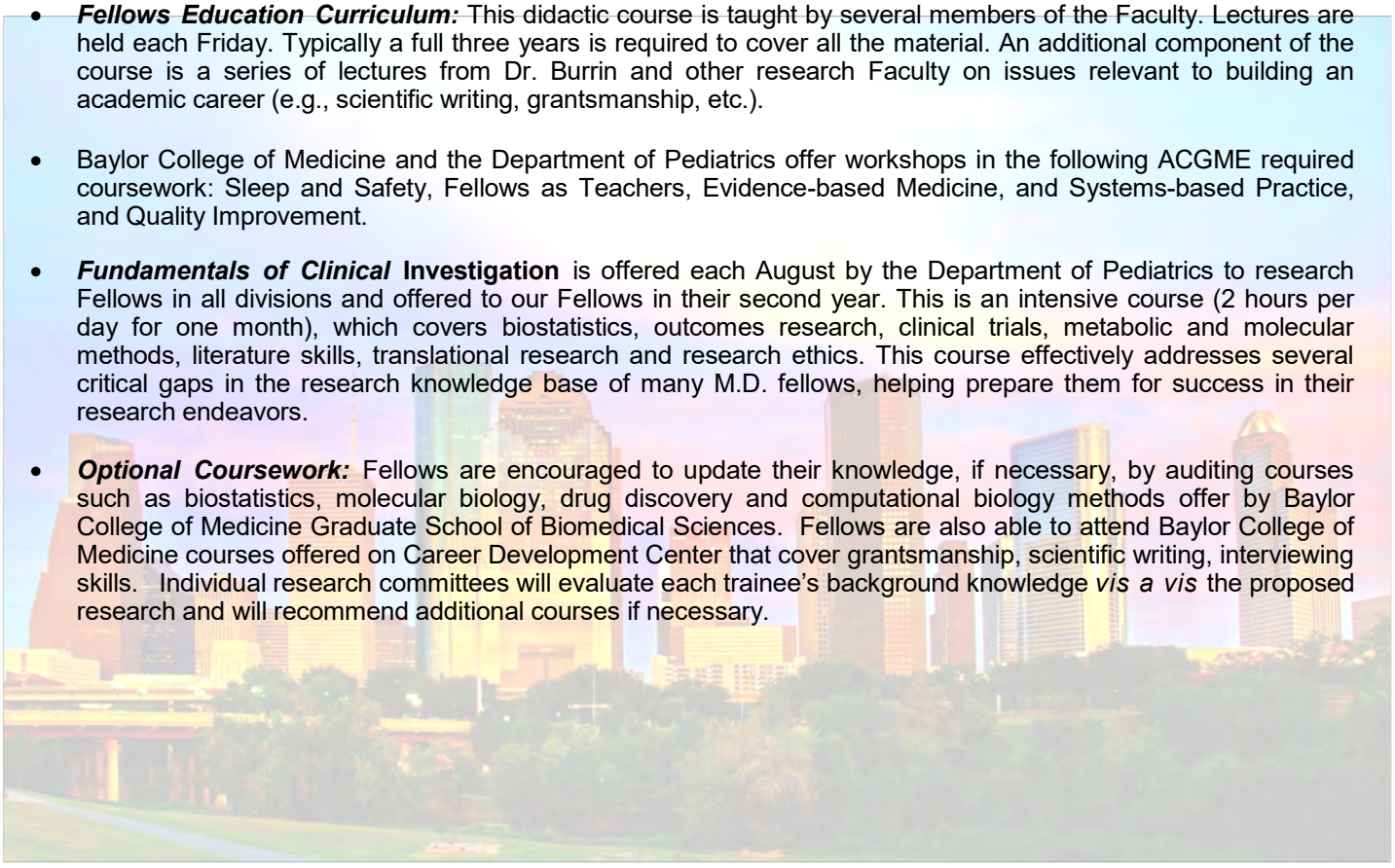
There are several courses available of all trainees:

- ***Fellows Education Curriculum:*** This didactic course is taught by several members of the Faculty. Lectures are held each Friday. Typically a full three years is required to cover all the material. An additional component of the course is a series of lectures from Dr. Burrin and other research Faculty on issues relevant to building an academic career (e.g., scientific writing, grantsmanship, etc.).

- Baylor College of Medicine and the Department of Pediatrics offer workshops in the following ACGME required coursework: Sleep and Safety, Fellows as Teachers, Evidence-based Medicine, and Systems-based Practice, and Quality Improvement.

- ***Fundamentals of Clinical* Investigation** is offered each August by the Department of Pediatrics to research Fellows in all divisions and offered to our Fellows in their second year. This is an intensive course (2 hours per day for one month), which covers biostatistics, outcomes research, clinical trials, metabolic and molecular methods, literature skills, translational research and research ethics. This course effectively addresses several critical gaps in the research knowledge base of many M.D. fellows, helping prepare them for success in their research endeavors.

- ***Optional Coursework:*** Fellows are encouraged to update their knowledge, if necessary, by auditing courses such as biostatistics, molecular biology, drug discovery and computational biology methods offer by Baylor College of Medicine Graduate School of Biomedical Sciences. Fellows are also able to attend Baylor College of Medicine courses offered on Career Development Center that cover grantsmanship, scientific writing, interviewing skills. Individual research committees will evaluate each trainee's background knowledge *vis a vis* the proposed research and will recommend additional courses if necessary.



M.D. CLINICAL TRAINING

Dr. Faith Ihekweazu is the Fellowship Director responsible for clinical training and program administration. The other members of the clinical Faculty are listed on page 5.

For more information contact program administrator: Lisa Kuchik (lkuchik@bcm.edu)

Inpatient: All Fellows receive 12 months of inpatient training at Texas Children's Hospital. We have 3 inpatient services that are each staffed by our fellows: two GI consultation services and one Hepatology service. Each service consists of a Gastroenterology Fellow, an attending physician, and an Advanced Practice Provider (APP). The three teams split call days during the week, with the on-call team being responsible for consults overnight.

- All patient care is performed by the APP and/or Fellow under the supervision of the attending. For example, consults are seen by the Fellow or APP and then are presented to the attending physician for discussion and teaching.
- All procedures are performed by the Fellow under supervision of the attending physician.
- Monday through Thursday the Fellows are "on call" once to twice a week on alternating days. Weekend call usually occurs every ten weeks for each Fellow. Fellows take night call and weekend call from home. There is a second fellow on call Saturday 5pm – Sunday 7am to allow the primary weekend fellow a rest period from consults, inpatient issues, and parent phone calls.
- Because attending physicians rotate weekly, the Fellow has the opportunity to work with a number of different Faculty and, as a result, is exposed to a variety of clinical perspectives.

Outpatient: Through all years of the Fellowship, the trainee attends Gastroenterology, Hepatology and Nutrition outpatient clinics.

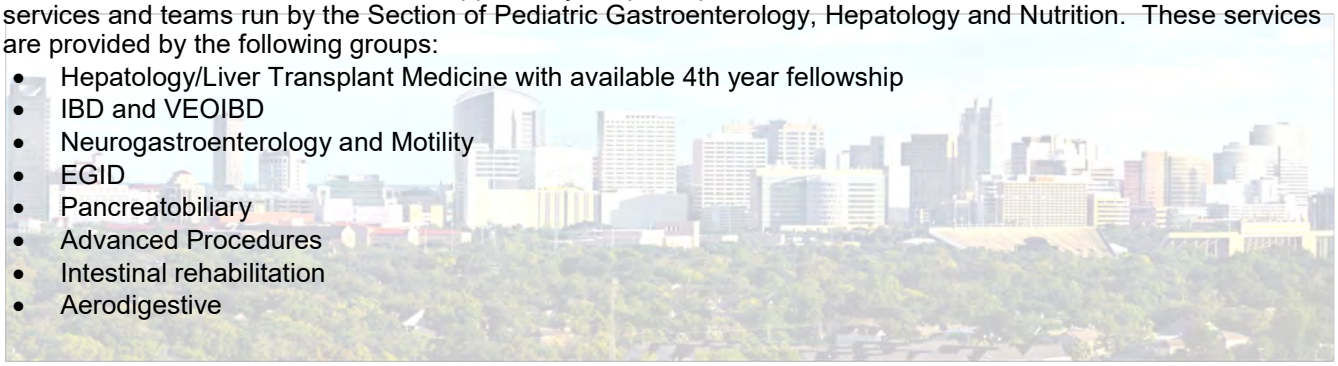
- During their first year, fellows will spend 6 weeks rotating through outpatient clinics in the medical center and the community hostpials, including nutrition-related clincs.
- Second and third year fellows have responsibility for continuity patients through a weekly 1/2 day Fellow's Clinic. Fellows' clinic is staffed by selected teaching faculty. During Fellow's Clinic there is exposure to initial evaluation and diagnostics, clinical management , and patient follow up for the duration of fellowship training. Responsibility for all patient/family communication belongs to the continuity fellow, with coverage arranged among fellows during times the fellow is unavailable.
- One week each month, second and third year fellows will spend time in subspecialty clinics in place of their continuity clinic. These include the General Gastroenterology, Hepatology, Inflammatory Bowel Disease, Intestinal Rehabilitation, Pancreatobiliary, and Neurogastroenterology and Motility clinics. Additionally, they can join our multi-disciplinary clinics including Very Early Onset IBD clinic, Prader-Willi Clinic, Eosinophilic Gastrointestinal Disorder Clinic, Aerodigestive clinic, Multidisciplinary Abdominal Pain Program, and Anorectal malformations clinic.

Skills: Expertise that the trainee will acquire includes:

- Diagnostic and management skills in a wide array of gastrointestinal, hepatic, pancreatic and nutritional disorders.
- State-of-the-art training in gastrointestinal procedures, including diagnostic upper and lower endoscopy, therapeutic endoscopy (e.g., stricture dilation, variceal band ligation), intestinal motility, and capsule endoscopy.

Other Activities: Fellows have the opportunity to participate in the activities of a number of other clinical services and teams run by the Section of Pediatric Gastroenterology, Hepatology and Nutrition. These services are provided by the following groups:

- Hepatology/Liver Transplant Medicine with available 4th year fellowship
- IBD and VEOIBD
- Neurogastroenterology and Motility
- EGID
- Pancreatobiliary
- Advanced Procedures
- Intestinal rehabilitation
- Aerodigestive



M.D. AND PH.D. RESEARCH TRAINING OVERVIEW

RESEARCH TRAINING

Dr. Douglas Burrin oversees the research training of the Fellows. The Section has maintained a NIH T32 Institutional Training Grant since 1991, directed by Dr. Burrin, Principal Investigator. This grant provides funds for selected M.D. and Ph.D. who seek a career with an emphasis on research. The research training program is designed to accommodate a wide range of research experience and the individual interests of each trainee. We also have developed distinct research training tracks (see details p. 8) to help clinical fellows find the most appropriate training experience to meet their career objectives

For M.D. Fellows: during year one, six (2 week) blocks of training are dedicated to research rotations. This gives the Fellow firsthand experience and provides the basis of choosing a mentor and an area of research interest.

As noted previously, the training program for M.D. Fellows includes a minimum of 24 months of research training and is designed to fulfill the research requirements that must be met by a Fellow before he/she is eligible to take the sub-board examination in pediatric gastroenterology. These requirements emphasize the importance of research training in the education of an academic pediatric gastroenterologist.

For Ph.D. Fellows: the expectation is that 24 months of research support will be provided. During this time, work also is geared toward obtaining future research support for the Fellow (e.g., NIH F32 grants) and preparing them for an academic career and the transition to a full-time Faculty position.

THE RESEARCH PROJECT

Each M.D. and Ph.D. trainee pursues a research project under the direction of a mentor selected from the list of potential mentors (p. 20). Mentors are chosen on the basis of solid research, an excellent record in training new investigators, and a strong desire to see our Fellows have a successful research career. To facilitate the choice of mentor, M.D. Fellows spend their first three research months rotating between candidate mentors. Ph.D. Fellows directly select a research mentor from among Training Grant mentors (p. 20). After selecting a research mentor and identifying a research project, each trainee forms a Scholarship Oversight Committee (SOC) including the mentor and three (or more) additional members. Dr. Burrin serves as chairman of the research committee.

Using the SOC members as consultants, the Fellow writes a research proposal, which will be the foundation for studies to be completed during the research years. This proposal is reviewed by the research committee and orally defended before this committee. M.D. and Ph.D. Trainees present an oral progress report to their SOC at least twice per year. They also are expected to give a presentation at the Pediatric GI Research Workshop once per year. Additional meetings of the research committee are called as needed.

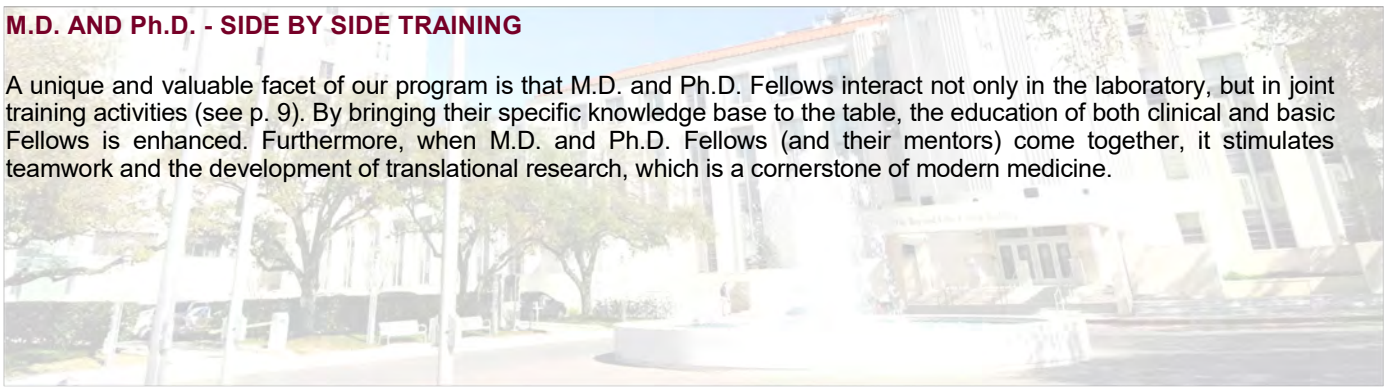
NIH T32 TRAINING GRANT

Only selected M.D. Fellows receive support from this prestigious training award and it is viewed as an important commitment toward a research career as a physician scientist. M.D. Fellows apply for Training Grant support in the Spring of their first year of Fellowship. The Training Grant provides research support for 24 months for M.D. Fellows.

All Ph.D. Fellows are supported by the NIH T32 Training Grant. Ph.D. candidates interested in the program can submit an application any time throughout the year provided that a slot is available. Applicants selected into the program are usually supported for 24 months.

M.D. AND Ph.D. - SIDE BY SIDE TRAINING

A unique and valuable facet of our program is that M.D. and Ph.D. Fellows interact not only in the laboratory, but in joint training activities (see p. 9). By bringing their specific knowledge base to the table, the education of both clinical and basic Fellows is enhanced. Furthermore, when M.D. and Ph.D. Fellows (and their mentors) come together, it stimulates teamwork and the development of translational research, which is a cornerstone of modern medicine.



M.D. RESEARCH TRAINING TRACKS

RESEARCH TRAINING TRACKS

As the field of Pediatric Gastroenterology, Hepatology and Nutrition has evolved, the breadth and depth of areas of clinical expertise and research endeavor have increased dramatically. Given the commitment of our program to the training of academic physicians, it has become clear that a single track of training is not adequate to meet the needs of the field and our trainees. Three distinct tracks of training have been developed, with a focus on research activities in the second and third year of fellowship. The tracks are designed to help identify, at an early stage/first year in fellowship, the most appropriate training experience to meet the individual's career objectives. It should be noted at the outset that there is no intrinsic enhanced value of one track over another. They are designed to meet the distinct needs of trainees. All are rigorous in training and in expectation of academic pursuit and product.

INVESTIGATOR TRACK

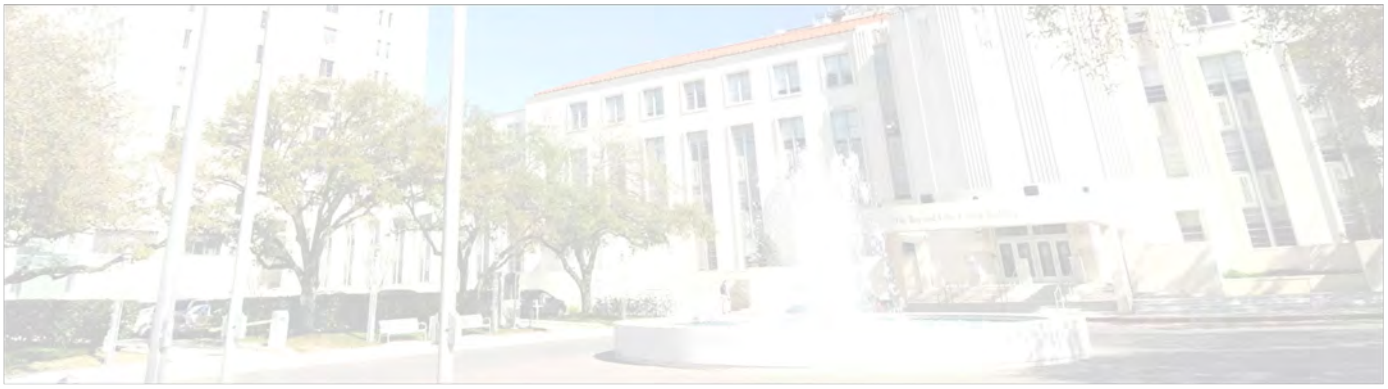
This track is designed to permit training in a wide-range of endeavors that will permit the trainee to experience the full range of the process of scientific investigation (hypothesis development, planning and executing investigations, interpreting and presenting the newly developed data in the context of the existing literature). A key goal will be to develop the skills necessary for independent assessment of scientific evidence. Mentors and the scientific oversight committees for this track will work with the trainee to develop lines of investigation that are important, feasible and conceptualized to permit meaningful data acquisition in the first 12 to 15 months of the research training. This will allow the trainee adequate time to delve into the interpretation of the new data and yield ample opportunity for presentation/publication of the data in the context of existing information. This process will give the trainee the requisite skills for critical interpretation of new data, which could be laboratory or clinically based.

CLINICAL NICHE TRACK

This track permits simultaneous training in investigation along with a more in-depth clinical experience in a particular niche in Pediatric Gastroenterology, Hepatology and Nutrition. This track is designed for those individuals who desire a patient care focused career in a specific sub-specialty area such as inflammatory bowel disease, neurogastroenterology and motility, hepatology, advanced procedures, or aerodigestive disorders, as examples. In this track, advanced clinical training is carefully integrated with a rigorous research endeavor to lay the groundwork to develop clinical specialists who have the ability to carry out patient oriented research. The scope of investigation and/or research product is tailored to accommodate the need for additional clinical training without diminishing the academic rigor of the line of investigation and/or the product that is developed as a result of the investigation. To pursue this track, a specific clinical niche needs to be identified by the trainee within 9 months of entering fellowship. View the Clinical Niche Track details.

PHYSICIAN-SCIENTIST TRACK

This track is designed to provide clinically relevant research training with the specific goal of attaining an independently funded career as a physician-scientist in Pediatric Gastroenterology, Hepatology and Nutrition. The near term goal of the program is for the individual to obtain NIH K-award (or equivalent thereof) funding within three years of completion of the fellowship. Individuals interested in this track will follow a distinct application and interview process when applying for a fellowship position. Criteria for acceptance into this track are very rigorous; the successful applicant typically will have extensive research experience prior to starting fellowship. The scientific endeavor in this track can include basic, translational, clinical or educational research.



PEDIATRIC GI WORKSHOP

This biweekly meeting is held over the noon hour and hosts presentations by Faculty and Fellows. The meeting is designed for Fellows and Faculty to share their research findings and foster collaboration among researchers with an interest in pediatric gastroenterology, hepatology and nutrition within Baylor College of Medicine and other institutions. All Fellows are required to present their research progress in this workshop each year. Twice per year, the workshop also hosts a "Visiting Young Investigator" - an up and coming person in pediatric gastroenterology who presents their research and meets informally with our M.D. and Ph.D. Fellows and Junior Faculty to discuss how to launch an academic career.

PEDIATRIC GI JOURNAL CLUB

This is a monthly gathering of Fellows or Faculty involved in our research training program. The meeting is designed to have Fellows select a clinical and basic science journal article that highlights a new research concept or advance with relevance to pediatric gastroenterology, hepatology and nutrition. The journal club is organized by the 2nd year Fellows and all trainees are required to present one clinical and one basic science article per year. Each Fellow leads an informal discussion of the design, outcomes and significance of the paper. The meetings are held in the evenings, hosted in the homes of our Faculty mentors as well as at the hospital, with food provided. These meetings are a vital social gathering for interaction among Fellows and Faculty to discuss their research and get acquainted personally. New collaborations and ideas often emerge from these meetings.

PEDIATRIC GI SCIENTIFIC SOCIAL

This is a quarterly meeting among Faculty and Fellows involved in the NIH Training Grant. The meeting is held in the evening and is designed to be a more intimate and informal gathering that includes dinner at a site in the Texas Medical Center. The program invites two mentors and one trainee from the Training Grant to give a brief, informal overview of their research intended to foster interaction, questions, and feedback among the attendees. The goal of this program is to give Fellows an opportunity to "think on their feet" and respond to feedback from Faculty mentors on their research. It also is another effective forum that often leads to research collaborations among Faculty mentors.

DIGESTIVE DISEASE CENTER GI FORUM

This is a weekly seminar series hosted by the Texas Medical Center Digestive Disease Center (DDC) (see p. 14) where local Faculty and invited guests from around the world present their research in the areas of gastroenterology and hepatology. The DDC is an integral component of the GI and liver research community in the Texas Medical Center and brings together Faculty from Baylor College of Medicine, University of Texas Health Science Center, M.D. Andersen Cancer Center, Rice University, University of Houston, and Texas A&M Institute of Biosciences and Technology. The DDC also hosts the Annual Frontiers in Digestive Disease on the Texas Medical Center campus, which is a one-day thematic symposium that features oral presentations from invited speakers, local Faculty, and trainees along with a poster sessions. It is yet another forum for our trainees to interact, network and socialize with Faculty and other members of the GI research community in Houston.

CAREER DEVELOPMENT WORKSHOPS

These are informal lectures given by training grant Faculty on vital skills and knowledge needed for successful Fellowship and Junior Faculty research career. Topics include how to write a manuscript, how to get published in peer-reviewed journals, how to apply for grant funding, and how to prepare oral presentations and posters for scientific meetings. Similar presentations on these and other career development topics also are given during the Baylor College of Medicine Department of Pediatrics Fellows Day held annually in the Spring.

CONFERENCES / VISTING FACULTY / BENEFITS

CONFERENCE SCHEDULE

Conferences are an important part of any Fellowship training program. The Section of Gastroenterology, Hepatology and Nutrition holds conferences on a regular basis in addition to those held by the Department of Pediatrics. The following table shows the current calendar.

Day	Time	Conference
Monday	12:00pm -1:00pm	GI Research Workshop (first and third Monday)
	1:00pm - 2:00pm	Hepatology Team Conference
Tuesday	12:00pm - 12:45pm	Division Meeting (monthly, second Tuesday)
	12:45pm - 1:45pm	Pedi GI Grand Rounds (monthly, second Tuesday)
	1:00pm - 2:00pm	Evidence-Based Case Conference (weekly/except second Tuesday of month)
	6:00pm - 7:30pm	Journal Club (first Tuesday of month, except July)
Wednesday	1:00pm - 2:00pm	Liver Transplant MRB Conference (weekly)
	1:00pm –2:00pm	Liver Pathology Conference (first Wednesday)
Thursday	8:30am - 9:30am	Children's Nutrition Research Center Conference (weekly)
	12:00pm - 1:00pm	IBD Conference (Monthly, first week)
	12:00pm - 1:00pm	Clinical Research Conference (Monthly, third week)
	4:00pm - 5:00pm	Digestive Disease Center GI Forum (weekly)
***IBD Conference, Team E Conference, and Clinical Research Conference are rotating conferences**		
Friday	8:30am - 9:30am	Pediatric Grand Rounds (weekly)
	12:00pm - 1:00pm	Pediatric Gastroenterology, Hepatology and Nutrition Fellow Core Curriculum

VISITING FACULTY PROGRAMS

Because of its size of and the amount of activity in the Texas Medical Center, we are afforded numerous opportunities to interact with visiting clinicians and researchers. In addition, the Section of Gastroenterology, Hepatology and Nutrition has its own invited speaker program, known as the Visiting Young Investigator series which is funded by our NIH T32 Training Grant. In this series, young pediatric gastroenterologists from around the country with established research credentials are brought in as role models for Fellows, Ph.D. trainees, and Junior Faculty. Each investigator gives a formal research seminar at the GI Research Workshop (see above) and has informal discussions (including career advice) with the Fellows and Junior Faculty.

VACATION AND BENEFITS

All Residents and Fellows are provided 44 paid days off per academic year (July 1 – June 30). This time off is non-vested (meaning the house staff physician is not paid for it if he or she leaves before having utilized), does not accrue, and does not roll over from one academic year to the next. These 44 days include:

*__21__ vacation days
*__14__ sick days (to be used only for personal illness)
*__9__ paid time off (PTO) days
This includes personal days, holiday, and educational leave (standard leave).

Jury Duty: Paid leave will be provided for jury duty as required by law.

SCIENTIFIC MEETINGS/ JOB PLACEMENT

NATIONAL SCIENTIFIC MEETING ATTENDANCE OPPORTUNITIES

Each M.D. and Ph.D. Fellow is provided with travel funds to attend one national conference per year. Additional conference travel funds are available to T32 Training Grant trainees and additional travel funds may be available if the Fellow is presenting a paper or a poster, but permission must be sought individually from the Chief of the Section. Examples of typical annual conferences attended by Fellows and Faculty are:.

- North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (held in November)
- Digestive Disease Week - A combined meeting of American Gastroenterological Association, American Association for the Study of Liver Diseases, American Society for Gastrointestinal Endoscopy, Society for Surgery of the Alimentary Tract (held in May)
- The Liver Meeting - American Association for the Study of Liver Diseases (held November)
- Pediatric Academic Societies' Annual Meeting - Sponsored by American Pediatric Society, Society for Pediatric Research, Academic Pediatric Association, and American Academy of Pediatrics (held in May)
- Experimental Biology - Federation of American Societies for Experimental Biology is a combined meeting of the following societies: The American Physiological Society, American Society for Biochemistry and Molecular Biology, American Society for Pharmacology and Experimental Therapeutics, American Society for Investigative Pathology and American Association of Immunologists (held in April)

In addition to these national meetings, M.D. Fellows have the opportunity to participate in special Fellow conferences, which are organized by the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN):

- First Year Fellows Conference
- Second Year Fellows Conference
- Third Year Fellows Conference

The sponsoring companies cover all expenses, travel, food and accommodations for these conferences. During their research time, Fellows may have the opportunity to attend small conferences in their area of research. However, total conference attendance is limited to three per year in order to protect time in the training program.

JOB PLACEMENT

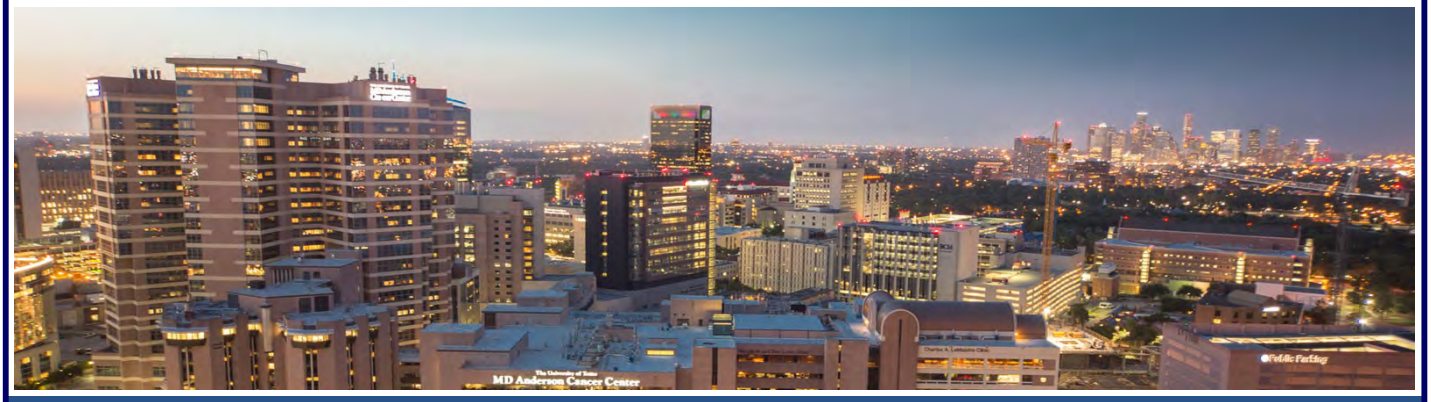
Assistance in obtaining positions after Fellowship training is taken very seriously by the Faculty. In addition to opportunities that are advertised, Faculty members often are aware of unpublished openings and are dedicated to finding the best match for each Fellow. The positions of the most recent previous Fellows are listed on the following page.

CURRENT POSITIONS OF FORMER MD TRAINEES

Name	Period Training	Current Position
Gulati, Ajay	2004-2007	Professor of Pediatrics, University of North Carolina, Chapel Hill, NC
Kellermayer, Richard	2006-2008	Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Himes, Ryan	2006-2009	Section Head, Pediatric GI, Ochsner Medical Center in New Orleans, LA
Jain, Ajay Kumar	2006-2009	Section Head, Pediatric GI, Saint Louis University, St. Louis, MO
Mehta Walsh, Seema	2007-2010	Associate Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Van Buren, Lynette	2007-2010	Associate Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Schaible (Kratzer), Tiffany	2008-2011	Associate Professor of Pediatrics, Wake Forest University, Winston-Salem, NC
Harpavat, Sanjiv	2011-2012	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Mir, Sabina	2010-2013	Associate Professor of Pediatrics, University of North Carolina, Chapel Hill, NC
Ng, Kenneth	2010-2013	Associate Professor of Pediatrics, Johns Hopkins School of Medicine, Baltimore, MD
Hollier, John	2012-2015	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Hiremath, Girish	2013-2016	Associate Professor of Pediatrics, Vanderbilt University, Nashville, TN
Mysore, Krupa	2013-2016	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Ihekweazu, Faith	2013-2016	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Preidis, Geoff	2014-2018	Associate Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Osgood, Peter	2015-2018	Assistant Professor of Pediatrics, Northwestern University Feinberg School of Medicine, Chicago, IL
Barton, Keisha	2016-2019	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Febo-Rodriquez, Liz	2017-2020	Assistant Professor of Pediatrics, University of Texas Medical Branch, Houston, TX
Britto, Savini	2017-2020	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Shiau, Henry	2017-2020	Assistant Professor Pediatrics, Baylor College of Medicine, Houston, TX
Dike, Peace	2017-2021	Assistant Professor Pediatrics, Stanford University, Palo Alto, CA
Mercedes, Rebecca	2020-2023	Assistant Professor of Pediatrics, Vanderbilt University, Nashville, TN
Sakhuja, Shruti	2020-2023	Assistant Professor of Pediatrics, University of Chicago, Chicago, IL
Watson, Ashleigh	2021-2024	Assistant Professor of Pediatrics, University of Utah, Salt Lake City, UT
Zimmerman, Sloane	2021-2024	Assistant Professor of Pediatrics, George Washington University, Washington D.C.
Simon, David	2021-2024	Assistant Professor of Pediatrics, St. Louis University, St. Louis, MO
Haribai, Minoti	2021-2024	Assistant Professor of Pediatrics, NYU, New York
Berens, Donovan	2022-2025	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX
Halaby, Carine	2022-2025	Assistant Professor of Pediatrics, George Washington University, Wash DC
Philip, Anitta	2022-2025	Assistant Professor of Pediatrics, Baylor College of Medicine, Houston, TX

CURRENT POSITIONS OF FORMER Ph.D. TRAINEES

Name	Period of Training	Current Position
Blutt, Sarah	1999-2001	Associate Professor, Molecular Virology Microbiology, Baylor College of Medicine, Houston, TX
Zhou, Yong	2007-2009	Associate Professor, University of Texas Health Science Center, Houston, TX
Hyser, Joseph	2008-2010	Associate Professor, Molecular Virology Microbiology, Baylor College of Medicine, Houston, TX
Wooten-Kee, Ruth	2008-2010	Assistant Professor Pediatrics, Children's Nutrition Research Center, Baylor College of Medicine, Houston, TX
Pflughoeft, Kathryn	2011-2012	Research Assistant Professor, University of Nevada School Medicine
Cana, Ross	2013-2015	Assistant Professor, BioSciences, Rice University, Houston, TX
Engevik, Mindy	2014-2016	Assistant Professor, Departments of Regenerative Medicine and Microbiology, Medical University of South Carolina, Charleston, SC
Guthrie, Greg	2015-2017	Assistant Professor Pediatrics, Children's Nutrition Research Center, Baylor College of Medicine, Houston, TX
Galley, Jeff	2016-2018	Research Scientist, Nationwide Children's Hospital, Ohio State University, Columbus, OH
Tan, Kai Li	2019-2020	Research Scientist, Emergent Biosolutions, Gaithersburg, MD
Midani, Firas	2019-2022	Assistant Professor, Molecular Virology Microbiology, Baylor College of Medicine, Houston, TX
Engevik, Kristen	2020-2023	Assistant Professor, Departments of Regenerative Medicine and Microbiology, Medical University of South Carolina, Charleston, SC
Vonderohe, Catilin	2020-2023	Assistant Professor Pediatrics, Children's Nutrition Research Center, Baylor College of Medicine, Houston, TX



EDUCATIONAL ENVIRONMENT

BAYLOR COLLEGE OF MEDICINE

Baylor College of Medicine is located in the Texas Medical Center in Houston, Texas which is the world's largest medical center. Baylor College of Medicine is dedicated to promoting health for all people through education, research and public service. The College pursues this mission by sustaining excellence in educating medical and graduate students, biomedical scientists and allied health professions, as well as advancing basic and clinical biomedical research. The College is ranked among the nation's top medical schools for research and 5th for primary care and 10th for pediatrics in the United States. It has more than 5700 full-time, part-time, emeritus, and voluntary Faculty and conducts independent research amounting to more than $687 million annually. The College enrolls 736 medical students in a four-year program, approximately 600 Ph.D. graduate students, more than 1500 residents and Fellows in postgraduate medicine and surgery. Baylor College of Medicine has more than 90 research and patient care centers. This thriving environment has fostered international recognition, especially in the Departments of Pediatrics, Molecular and Cellular Biology, Molecular Virology & Microbiology, Molecular and Human Genetics, the Howard Hughes Research Institute, and the Human Genome Center. www.bcm.edu

THE DEPARTMENT OF PEDIATRICS

Under the leadership of Dr. Gordon Schutze, Pediatrics is one of the pre-eminent departments in the U.S. The Department has trained more than 60% of the pediatricians in Texas and nearly 5% of all pediatricians in the United States. The Department of Pediatrics receives more than $104 million per year in extramural grant support, the majority of which comes from NIH. In any given year, the members of the department publish more than 800 papers in the peer-reviewed medical literature. Today, the Department of Pediatrics has more than 1200 Faculty. Some of these Faculty members provide staffing for the Pediatric Services at Ben Taub General Hospital (a public hospital providing care to citizens of Harris County). In addition, there are also 8 research centers in specific disciplines, the most relevant to this program being the Center for Cell and Gene Therapy, the Children's Nutrition Research Center, the Obesity Center, and the Liver Center. www.bcm.edu/departments/pediatrics

BAYLOR CLINICAL SCIENTIST TRAINING PROGRAM

Baylor College of Medicine has a college-wide multidisciplinary didactic 1-5 year training program known as the Clinical Scientist Training Program. This program is committed to promoting the education and training of highly motivated Junior Faculty and clinical fellows to become successful, independent clinical investigators and future leaders in academic medicine and biomedical research. The program offers M.S. and Ph.D. degrees in Clinical Investigation through the Graduate School of Biomedical Sciences. Both programs are designed for individuals with a significant commitment to clinical research.

M.D. trainees interested in clinical research pursue either the M.S. or Ph.D. degree from the Clinical Scientist Training Program. The core courses for the program are entitled Fundamentals of Clinical Investigation, Clinical Investigation for the Career Scientist, Responsible Conduct of Research for Clinical Investigators, and electives (Intermediate Biostatistics at the UT School of Public Health). Fundamentals of Clinical Investigation is offered in August every year. Fulfilling the course requirements, students will write a K-23 research proposal that will be developed into a thesis and the students also will write a R01 proposal. www.bcm.edu/education/programs/clinical-scientist-training/

TEXAS MEDICAL CENTER DIGESTIVE DISEASE CENTER

Mentors and trainees associated with this training program derive significant benefit from the Texas Medical Center Digestive Disease Center (DDC). The DDC is a NIH-funded center that promotes coordinated digestive disease activities, and the only one in the Southwest or Gulf States area. The mission of the DDC is to facilitate on-going research in digestive diseases, promote translational research between basic and clinical areas, develop new projects, nurture new investigators, and provide GI educational activities. The DDC supports three Basic Science Cores (Cellular and Molecular Morphology, Functional Genomics and Proteomics, Integrative Biology), and one Clinical Core (Study Design and Clinical Research). The Center draws together a multidisciplinary group of investigators, including basic scientists with proven tract records of success, and well coordinated clinical programs dealing with pediatric and adult GI patients.

In addition, Fellows supported on our NIH Training Grant are eligible, in their third year, to apply for pilot/feasibility funding (up to $50,000) from the DDC in order to develop research projects suitable for external funding. www.bcm.edu/gastro/DDC

Baylor College of Medicine
GIVING LIFE TO POSSIBLE

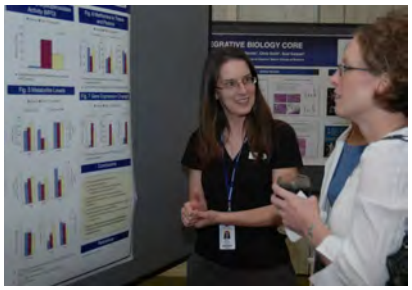
THE UNIVERSITY OF TEXAS
MD ANDERSON
CANCER CENTER
Making Cancer History

UTHealth
The University of Texas
Health Science Center at Houston

EDUCATIONAL ENVIRONMENT

PEDIATRIC RESEARCH AND FELLOWS SYMPOSIUM

The Department of Pediatrics supports research training through their sponsorship of an annual research symposium where trainees have the opportunity to present their research in either oral or poster format. The symposium also includes faculty presentations on topics relevant to the development of academic physicians. www.bcm.edu/pediatrics



CHILDREN'S NUTRITION RESEARCH CENTER

The Children's Nutrition Research Center (CNRC) is the first federally funded human nutrition research center to investigate the nutritional needs of children room conception though adolescence. The 11-story Center has 50 full-time Faculty members dedicated to defining the nutrient needs of children from conception through adolescence, pregnant women, and nursing mothers. Since 1978, CNRC research has helped form the foundation of national nutrition policies and clinical nutrition practices that have improved the health of mothers and children of all ages. It is operated by Baylor College of Medicine in cooperation with Texas Children's Hospital and the Agricultural Research Service of the U.S. Department of Agriculture (USDA/ARS). The CNRC houses an Energy Metabolism and Exercise Laboratory with indirect room calorimetry, an advanced Body Composition Laboratory, a Behavioral Studies Unit and Children's Eating Laboratory. These facilities enable the Center scientists to conduct some of the world's most advanced nutritional studies, which have earned the CNRC an international reputation for research excellence.

GASTROINTESTINAL (GI) PROCEDURES SUITE

Texas Children's Hospital houses the 7,300-sq. ft. Gastrointestinal Procedures Suite. The GI Procedures Suite is at the cutting edge of patient care and research. More than 3300 procedures (e.g., upper endoscopy, colonoscopy and liver biopsy) are performed each year in the Suite as well as a wide array of diagnostic tests (e.g., 24-hour esophageal pH monitoring, breath testing). The Suite is nationally known for its leadership role in sedation techniques in children and is the headquarters of the PEDS-CORI project (Pediatric Endoscopy Database System—Clinical Outcomes Research Initiative). The suite is composed of three endoscopic rooms, two non-endoscopic procedure rooms, a prep room, holding area, recovery room and endoscope disinfection room. www.bcm.edu/pediatrics

TEXAS CHILDREN'S HOSPITAL

Texas Children's Hospital (TCH) is an internationally recognized full-care pediatric hospital. It is the largest children's hospital in the United States. TCH is consistently ranked as a national leader among pediatric hospitals by U.S. News & World Report. Texas Children's ranked 3rd among all 183 pediatric institutions with services over nearly 4.9 million patient encounters and nearly 7,000 births take place annually. Texas Children's Hospital founded in 1924 is the largest children's hospital in the U.S. and is nationally ranked in 10 pediatric subspecialties categories (5nd in GI). The hospital has over 2000 faculty in 40 subspecialty sections, and 17 centers. The hospital's Feigin Center is a 20-story facility dedicated entirely to pediatric research. Texas Children's Hospital is Houston's only freestanding children's hospital and is the primary teaching hospital of Baylor College of Medicine. The hospital's award-winning medical staff consists of board-certified, primary -care physicians, pediatric subspecialists, pediatric surgical subspecialists, and dentists. In addition, TCH offers a dedicated and highly skilled nursing staff, health care professionals and support staff of more than 6,000.

Also located adjacent to the Clinical Care Center is the TCH Pavilion for Women which provides a full continuum of family-centered care to women, mothers and their babies, beginning before conception and continuing through all the years of a woman's life. In 2018, TCH opened a new 19-floor Legacy Tower with new surgical operating rooms, intensive care unit, heart center and Helipad roof.

TCH West Campus houses a 48-bed acute care hospital located 25 miles from the Texas Medical Center. It houses a pediatric emergency center, surgical suites, and an advanced diagnostic imaging center.

In 2017, TCH opened the Woodlands Campus with a 560,000-square-foot, state-of-the-art facility with 25 emergency center rooms, 72 outpatient rooms, 12 radiology rooms, four operating rooms, 28 critical care beds and 32 acute care beds. In 2024, TCH North Austin Campus on opened and features 48 beds, 52 specialty care clinics, and a maternal-fetal medicine clinic. www.texaschildrens.org

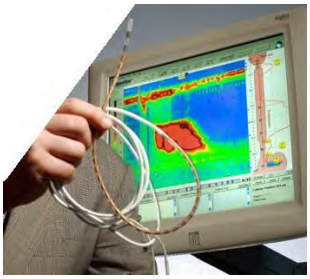
EDUCATIONAL ENVIRONMENT

INFLAMMATORY BOWEL DISEASE PROGRAM

The first of its kind in Houston and the Southwest, Texas Children's Inflammatory Bowel Disease (IBD) Center offers a comprehensive, multidisciplinary approach to the diagnosis and treatment of Crohn's disease and ulcerative colitis in children. A team of specialists provides individualized care, education and cutting-edge research to help patients and families manage the long-term aspects of the disease. We also treat other disorders with chronic diarrhea in pediatric patients, such as recurrent Clostridium difficile infection. Our program offers state-of-the-art diagnostic and therapeutic procedures such as whole exome sequencing, a wide array of endoscopic investigations including enteroscopy and capsule endoscopy, and MRI enterography. We are a pediatric center for fecal microbiota transplantation (FMT) in the treatment of recurrent Clostridium difficile infection, which is supported by the state-of-the-art Texas Children's Microbiome Center. https://www.texaschildrens.org/departments/inflammatory-bowel-disease-program

THE NEUROGASTROENTEROLOGY AND MOTILITY PROGRAM

The Neurogastroenterology and Motility Center at Texas Children's Hospital's goal is to provide world-class evaluation, therapy, and research in childhood functional and motility gastrointestinal disorders. Since 2008, a full motility laboratory including state-of-the art high resolution manometry, esophageal impedance, and breath testing has been available for diagnostic evaluations. All GI Fellows will have exposure to patients seen within the Center, and are welcome to attend Center outpatient clinics, observe/perform manometry procedures, or choose a research project relating to functional or motility disorders.



TEXAS CHILDREN'S HOSPITAL MICROBIOME CENTER

The Texas Children's Microbiome Center is a cutting-edge laboratory housed within Texas Children's Hospital. Staffed by leading scientists in the field of metagenomic science, the Texas Children's Microbiome Center is strategically placed to partner with pediatric gastroenterology and other specialties. Clinical research coordinators are available for recording valuable information regarding the current condition of the child as well as historical medical information of importance. The Texas Children's Microbiome Center Sequencing Core is skilled in the extraction of nucleic acid from a variety of specimen types, and the TCMC provides metagenomic analysis of the gut microbiome by next-generation sequencing partnered with bioinformatics support. www.texaschildrens.org

CAMP SIA (Survived It All)

Camp SIA is a free camp for our patients who have life-long conditions including liver and bowel disease. The camp is held at the Camp For All facility in Burton, Texas. Camp SIA allows our GI patients time to meet and do activities with other kids who have similar conditions. Camp is great fun, not only for our campers, but for our medical staff as well. Medical staff is on hand 24-hours a day including a doctor, nurses, medical assistants, and a dietitian.
https://www.texaschildrens.org/departments/inflammatory-bowel-disease-program

EDUCATIONAL ENVIRONMENT

HEPATOLOGY AND LIVER TRANSPLANT PROGRAM

Texas Children's Liver Center is the largest pediatric liver disease program in the South, and is among the largest in the United States. The Center's highly skilled transplant surgeons and liver specialists provide first-level clinical care to children with all forms of pediatric liver disease. Working in a state-of-the-art facility with a focus on family-centered care, our experienced team performs 35-45 liver transplants a year in children, ranging from young infants (younger than a month) to young adults, making us one of the most experienced liver transplant programs in the United States and the world. The Liver Transplant Program has performed more than 825 pediatric liver transplants with 35-45 per year. The liver transplant team at Texas Children's Hospital is much more than just a surgical team. Our transplant coordinators work with surgeons, cardiologists, social workers, child life specialists, dietitians, pharmacists and physical and occupational therapists.

Within the Pediatric Hepatology Program, the Biliary Atresia Clinic provides comprehensive medical, surgical and transplant care for infants and children with biliary atresia. The Viral Hepatitis Program at Texas Children's Hospital provides comprehensive screening, education, and treatment for children and their families affected by viral hepatitis B and C. The Intestinal Rehabilitation Clinic provides multidisciplinary services that evaluate and treat patients with short bowel syndrome (SBS) and associated medical and social issues.

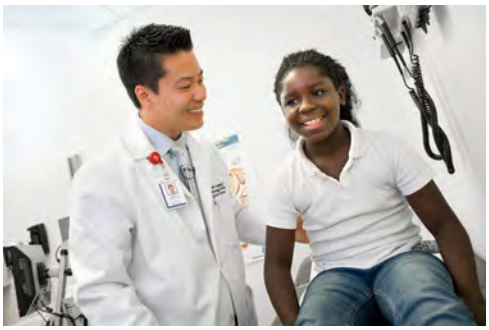
INTESTINAL REHABILITATION CLINIC

Provides multidisciplinary services that evaluate and treat patients with short bowel syndrome (SBS). The most frequent manifestations include growth delay, nutritional deficiencies, malabsorptive diarrhea, liver disease, bacterial overgrowth in the intestines, and oral feeding aversions. The multidisciplinary services offered by this clinic include gastroenterology, home parenteral nutrition prescriptions and coordination with home healthcare, nutrition, social services, and vascular access team. Over 150 patients with SBS are seen annually in the Intestinal Rehabilitation Clinic with over 50 home parenteral nutrition prescriptions managed by our multidisciplinary team. Our team is well-known and established in the field of SBS and provides state of the art treatment.

GASTROENTEROLOGY NURSING STAFF

The GI nurses are an integral part of the management of follow up care of GI patients. Due to the complex nature of our patients, we are fortunate to have a number of specialized nurses who provide continuity of care in the clinic via their phone triage system. In this way the nursing staff aids the Fellows in day to day management of the outpatient GI patient.

https://www.texaschildrens.org/departments/digestive-disorders/our-team

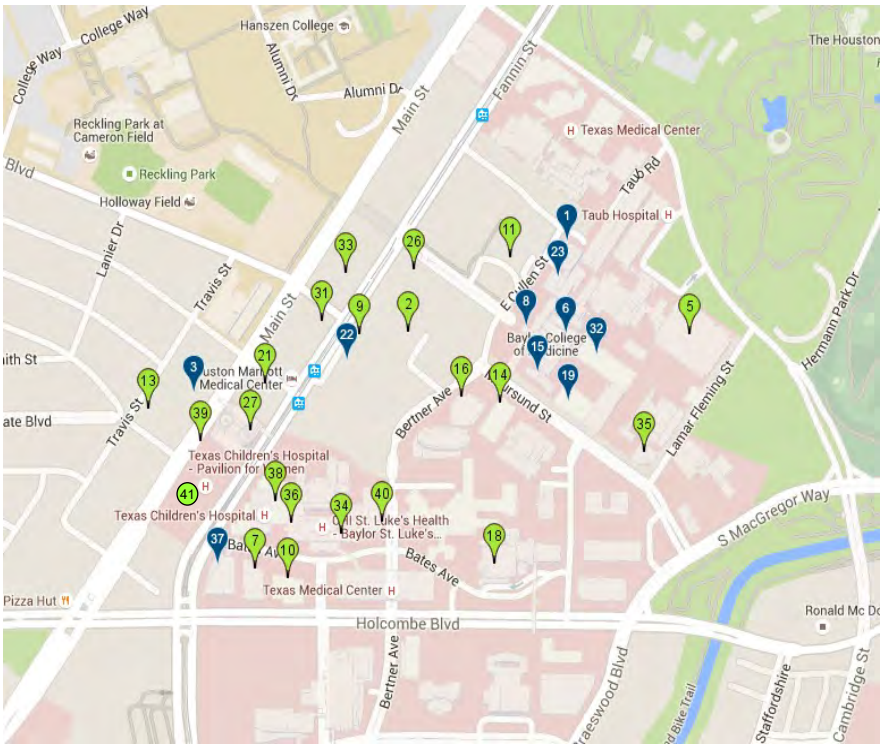


THE TEXAS MEDICAL CENTER

THE TEXAS MEDICAL CENTER

TMC is the world's largest medical complex located on 2.1 square miles it's the 8th largest business district in the U.S. Its 49 member institutions are engaged in not-for-profit patient care, education, and research. Among its components are 2 medical schools (BCM and the University of Texas-Houston), 2 graduate schools of biomedical sciences, a school of public health, 4 schools of nursing, a dental school, a college of pharmacy, 2 major comprehensive cancer research centers (M.D. Anderson Cancer Center and BCM). These institutions include 21 renowned hospitals, 14 support organizations, 10 academic institutions, eight academic and research institutions, seven nursing programs, three public health organizations, three medical schools, two pharmacy schools, and a dental school. Encompassing over 50 million developed square feet on approximately 1,000 acres in the heart of Houston, TMC has over 120,000 employees who serve over 10 million patients each year. The TMC institutions received $595M across 1,182 awards in 2024. The Medical Center is situated in a largely residential area of Houston. Thus, abundant housing is within walking distance. Rice University's tree-lined campus is adjacent to the Medical Center, as are the recreational facilities of Hermann Park.

1 Alkek Bldg (BCM)
2 Alkek Tower
3 Baylor Clinic
5 Ben Taub Hospital
6 Ben Taub Research Center
7 Children's Nutrition Research Center
8 Cullen Building (BCM)
9 Dunn Tower
10 Texas Children's Hospital Feigin Research Center
11 HAM-TMC Library (Jesse Jones Bldg)
13 **Texas Children's Hospital Pavilion Tower II**
14 Jan and Dan Duncan Neurological Research Institute at TCH
15 Jewish Institute for Medical Research
16 John P. McGovern Commons
18 MD Anderson Cancer Center
19 Margaret Alkek Biomedical Research Building
21 Medical Tower (Baylor Faculty)
22 Houston Methodist Hospital
23 Michael E. DeBakey Center (BCM)
26 Neurosensory Center
27 O'Quinn Medical Tower
31 Scurlock Tower
32 Smith Research Wing
33 Smith Tower
34 Baylor St. Luke's Medical Center
35 TIRR (The Institute for Rehabilitation and Research)
36 Texas Children's Hospital
37 Texas Children's Hospital Mark Wallace Tower
38 Texas Children's Hospital West Tower
39 Texas Children's Pavilion for Women Tower I
40 Texas Heart Institute—Denton A. Cooley Building
41 **Texas Children's Hospital Legacy Tower**



THE CITY OF HOUSTON

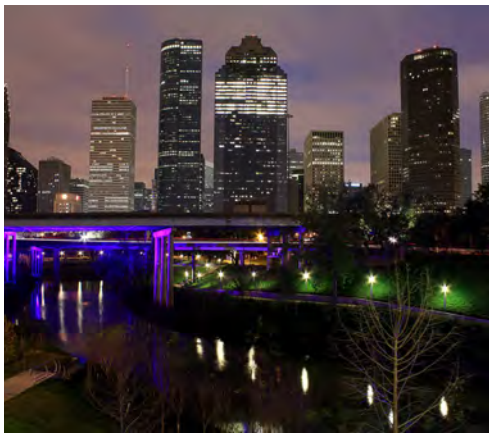
FUN IN THE CITY

As the fourth most populous city in the United States, with over 2.3 million residents representing a rich blend of cultures, Houston is a city full of surprises. While long recognized as a global leader in the energy sector, Houston is also a major center for the arts, education, aerospace, health care, and international trade. Houston consistently ranks among the top U.S. metropolitan areas for economic opportunity and diversity. It is one of only a few cities in the country with permanent, professional companies in all four major performing arts disciplines: opera, symphony, ballet, and theater. The Museum District in Midtown is home to 19 museums, including the Museum of Fine Arts, Houston Museum of Natural Science, and Contemporary Arts Museum Houston, drawing millions of visitors annually. Nearby, the city's dynamic nightlife, eclectic dining, and vibrant arts scene continue to grow.

Downtown Houston is the epicenter of entertainment, featuring renowned venues such as the Wortham Theater Center, Jones Hall, Alley Theatre, and Bayou Music Center. Discovery Green, a 12-acre urban park in the heart of downtown, hosts year-round concerts, public art installations, family events, and fitness activities.
Houston is also a passionate sports city. Its warm climate supports outdoor activities year-round, and the city is home to major professional teams:

- The **Houston Texans** (NFL), playing at NRG Stadium
- The **Houston Rockets** (NBA), at Toyota Center
- The **Houston Astros** (MLB), 2017 and 2022 World Series champions, at Daikin Park
- The **Houston Dynamo FC** (MLS) and **Houston Dash** (NWSL), both playing at Shell Energy Stadium (formerly BBVA Compass Stadium)

Just an hour south, the beaches of Galveston offer a coastal escape, including the historic Galveston Island Pleasure Pier amusement park. For easy access to sporting and cultural events, the METRO light rail system continues to expand, connecting key areas across the city.
One of Houston's most iconic annual events is the **Houston Livestock Show and Rodeo**, held at NRG Stadium. Drawing millions of visitors each year, this three-week celebration features rodeo competitions, a star-studded concert lineup, carnival attractions, a parade, trail rides, BBQ contests, and more—solidifying its status as Houston's signature event.
With its blend of cultural richness, economic vitality, and community spirit, Houston offers something for everyone.



FACULTY RESEARCH INTERESTS

Allison, David	Nutrition, Energetics, Obesity Epidemiology
Britton, Robert A.	Basic and Translational Therapeutic Microbiology
Buffington, Shelly	Nutrition and Gut-Brain Axis Function
Burrin, Douglas G.	Nutritional Regulation of Neonatal GI and Liver Health and Disease
Chiou, Eric	Disorders of Esophageal Motility
El-Serag, Hashem	Clinical Epidemiology and Outcomes of GI and Liver Disorders
Estes, Mary K.	Gastrointestinal Virus-Cell Interactions, Pathogenesis and Immunity
Fishman, Douglas	Advances in Therapeutic Endoscopy and Pancreaticobiliary Disease
Gao, Xia	Targeted Nutritional Intervention and Therapeutics for Metabolic Diseases
Hair, Amy	Nutritional Impact on Gut-Liver Microbiome in Preterm Infants
Guthrie, Gregory	Pathogenesis and Treatment of Neonatal Cholestatic Diseases
Harpavat, Sanjiv	Developmental Etiology and Clinical Studies of Biliary Atresia
Hertel, Paula M.	Infantile Disorders of Biliary Function: Closing Current Knowledge Gaps
Hollier, John M.	Treatment of Functional Abdominal Pain Disorders in Children
Hyser, Joseph	Cellular Signaling Pathways in GI Health and Infectious Diarrhea
Ihekweazu, Faith	Shaping the Gut Microbiome to Modulate Intestinal Inflammation
Jafar-Nejad, Hamed	Role of O-glucose Glycans in Notch Signaling and Liver Development
Kanwal, Fasiha	Clinical Epidemiology of Liver Diseases
Kellermayer, Richard	Epigenomic and Microbiome Aspects of Inflammatory Bowel Disease
Lee, Brendan	Biochemical Genetics of the Urea Cycle and Nitric Oxide Synthesis
Leung, Daniel	Viral Hepatitis and Cystic Fibrosis Liver Disease
Mills, Jason	Regeneration, Metaplasia, and Tumorigenesis in the GI tract
Mysore, Krupa	Immune Regulation of Pediatric Liver Diseases
Preidis, Geoffrey	Malnutrition and its Effects on the Liver, Gastrointestinal Tract, and Gut Microbiome
Ramani, Sasirekha	Prevention and Pathogenesis of Viral Gastroenteritis in Infants
Rhoads, J. Marc	Probiotic Mechanism of Action in GI Diseases
Savidge, Tor	Enteric Neuroimmune Microbe Interactions and Infectious Disease
Shen, Lanlan	Epigenetic Regulation of GI Development and Disease
Shneider, Benjamin L.	Basic and Translational Research in Bile Acid Homeostasis and Disease
Speer, Allison	Tissue-engineered Intestine for Pediatric Intestinal Failure
Tessier, Mary Elizabeth	Microbiome Regulation of Enterohepatic Bile Acid Signaling
Thevananther, Sundararajah	Molecular Mechanisms of Liver Regeneration, NASH, and Hepatocellular Carcinoma
Wooten-Kee, Ruth	Targeting Nuclear Receptors in Normal and Liver Disease Conditions

Obesity, Aging, Statistical Methods, and Research Trustworthiness

Endowed Professor, Department of Pediatrics
Chief of Nutrition
Director, USDA-ARS Children's Nutrition Research Center
Baylor College of Medicine

DAVID ALLISON

Recent Publications

Cruz-Cano R, Allison DB. Report uncertainty information to improve trust in science. Nat Hum Behav. 2025;9(1):9-12. https://doi.org/10.1038/s41562-024-02084-3

Trueblood JS, Allison DB, … [15 authors in total]. The misalignment of incentives in academic publishing and implications for journal reform. Proc Natl Acad Sci USA. 2025;122(5):e2401231121. https://doi.org/10.1073/pnas.2401231121

Vorland CJ, O'Connor LE, Henschel B, Huo C, Shikany JM, Serrano CA, … [12 authors in total] Allison DB, Brown AW. "Shaking the ladder" reveals how analytic choices can influence associations in nutrition epidemiology: beef intake and coronary heart disease as a case study. Crit Rev Food Sci Nutr. 2025:1-16. https://doi.org/10.1080/10408398.2025.2525459

Dhurandhar EJ, Maki KC, Dhurandhar NV, … [12 authors in total], Allison DB. Food noise: definition, measurement, and future research directions. Nutr Diabetes. 2025;15:30. https://doi.org/10.1038/s41387-025-00382-x

French SJ, Kanter M, Maki KC, Rust BM, Allison DB. The harms of high protein intake: conjectured, postulated, claimed, and presumed, but shown? Am J Clin Nutr. 2025;122(1):9-16. https://doi.org/10.1016/j.ajcnut.2025.05.002

My research team has two broad and intersecting major foci. The first may be termed energetics and involves the study of energy intake, expenditure, and metabolism; obesity; weight loss; food intake; and the causes, consequences, and effects of these processes and conditions, especially but not only on longevity. Our second line of research is more methodological and involves promoting greater rigor in science—both in our own work and that of others—especially but not only regarding the use of statistics.

Our research on energetics uses animal models, epidemiology, and clinical trials, among other methods, and my collaborators and I consistently seek to challenge investigators in the field to consider new ideas and new approaches that may serve as catalysts for future studies. Recent achievements include a 2023 paper in Science that laid out key questions remaining to be answered regarding the prevention and treatment of obesity and the development and publication (pending modest revisions) of the most thoroughly psychometrically validated measure of the newly identified construct of "food noise" and which is being adopted into major pharmaceutical trials.

My group also develops new statistical methods where they are needed. I teach courses on statistics and currently have two courses funded as NIH R25 grants that aim to promote better use of statistical methods. I regularly identify statistical errors in the literature and work with authors, editors, and others to attempt to correct and learn from those errors. I also write tutorial papers and am particularly interested in causal inference, design and analysis of randomized controlled trials, analysis of longevity data, and analysis of clustered and nested data. Additionally, I direct the Comparative Data Analytics Core of the NIH Nathan Shock Center for the Basic Biology of Aging housed at the University of Alabama at Birmingham.

Alongside my research activities, I greatly enjoy and prioritize mentoring. I have mentored over 70 pre- and postdoctoral trainees, most of whom hold current positions in academic research. I have won a number of mentoring awards, including the nation's highest award for mentoring, the Presidential Award for Excellence in Science, Mathematics and Engineering Mentoring (PAESMEM). I take mentoring seriously and am proactive in practicing it, making the effort to study effective methods and freely sharing my knowledge with others.

I was recruited to the CNRC as of June 30 and will be a new member of the T32 Executive Committee. I am a member of the National Academy of Medicine and have been continuously funded by the NIH for over 30 years.

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Therapeutic Microbiology

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ROBERT BRITTON

Recent Publications

Zhu D, Galley J, Pizzini J, Musteata E, Douglas MV, Chazin WJ, Skaar EP, Tabor JJ, Britton RA. Microbial Biosensor for sensing and treatment of intestinal inflammation. Adv Sci (Weinh). 2025 Jul;12 (27):e2504364.

Di Rienzi SC, Danhof HA, Forshee MD, Roberts A, Britton RA. Limosilactobacillus reuteri promotes the expression and secretion of enteroendocrine- and enterocyte-derived hormones. FASEB J. 2025 Mar 31;39(6):e70408.

Danhof HA, Lee J, Thapa A, Britton RA, Di Rienzi SC. Microbial stimulation of oxytocin release from the intestinal epithelium via secretin signaling. Gut Microbes. 2023 Dec;15(2):2256043.

Wang Y, Zhu D, Ortiz-Velez LC, Perry JL, Pennington MW, Hyser JM, Britton RA, Beeton C. A bioengineered probiotic for the oral delivery of a peptide Kv1.3 channel blocker to treat rheumatoid arthritis. Proc Natl Acad Sci U S A. 2023 Jan 10;120(2):e2211977120.

Auchtung JM, Preisner EC, Collins J, Lerma AI, Britton RA. Identification of Simplified Microbial Communities That Inhibit Clostridioides difficile Infection through Dilution/ Extinction. mSphere. 2020 Jul 29;5(4):e00387-20.

Collins J, Robinson C, Danhof H, Knetsch CW, van Leeuwen HC, Lawley TD, Auchtung JM, Britton RA. Dietary trehalose enhances virulence of epidemic Clostridium difficile. Nature. 2018. 18;553 (7688):291-294.

Therapeutic Microbiology Laboratory. The Britton laboratory is interested in the role of microbes in health and disease, with a focus on identifying microbes with therapeutic properties for a variety of ailments. We use bacterial genetics, genomics, microbial ecology, and physiology to investigate individual microbes and microbial community structure and function.

The role of intestinal bacteria in health and disease. Recent work into the role of intestinal bacteria in a variety of disease states including inflammatory bowel disease, obesity, and diabetes has established a clear link between these bacteria and our health. The Britton laboratory is focused on two areas of research in this area: the role of probiotic bacteria in treating disease and the role of the intestinal microbiota in preventing pathogen invasion.

Probiotic Lactobacillus reuteri. Much of our work focuses on characterizing how different strains of Lactobacillus reuteri impact various aspects of the host response including inflammation, bone health, pathogen invasion and intestinal function. We use a variety of in vitro and animal models to explore how L. reuteri impacts health. Our overall goals are to identify novel probiotic strains that can be used to prevent or ameliorate disease and to develop a platform for the delivery of biotherapeutics.

Microbiota and prevention of pathogen invasion. We are interested in understanding how the intestinal microbiota provides a barrier to incoming pathogens and how perturbations of the microbiota result in an established infection. We have focused most of our attention on the pathogen Clostridium difficile, which is the most common cause of antibiotic associated diarrhea and is quickly becoming the most common cause of nosocomial infections. We have developed mini-bioreactors and mice colonized with a human intestinal microbiota to address which members of the community are responsible for inhibiting C. difficile invasion. Our ultimate goal is to develop a probiotic cocktail derived from the human intestinal microbiota that will suppress C. difficile invasion.

Regulation of gut hormone production by the microbiota. Obesity and type 2 diabetes are emerging problems in the United States population, including young children. Our laboratory is screening next-generation probiotics for their ability to modulate gut hormone production in an effort to find microbes that can impact weight gain, behavior, and inflammation.

Engineering microbes to sense and respond to disease. Using synthetic biology technology, we are creating microbes that can sense intestinal inflammation and respond with the secretion of therapeutics to resolve disease. We are also optimizing L. reuteri as a platform to deliver a wide variety of gut delivered therapies and have demonstrated that oral delivery can result in systemic distribution of protein-based therapies.

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DOUGLAS G. BURRIN

Nutritional Regulation of Neonatal Gut and Liver Health and Disease

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Department of Pediatrics
USDA-ARS Children's Nutrition Research Center
Director, Fellowship Research Training
Gastroenterology, Hepatology and Nutrition
Baylor College of Medicine

Recent Publications

Melendez Hebib V, Taft DH, Stoll B, Liu J, Call L, Guthrie G, Jensen N, Hair AB, Mills DA, Burrin DG. Probiotics and Human Milk Differentially Influence the Gut Microbiome and NEC Incidence in Preterm Pigs. Nutrients. 2023 May 31;15(11):2585.

Vonderohe C, Guthrie G, Burrin DG. Fibroblast growth factor 19 secretion and function in perinatal development. Am J Physiol Gastrointest Liver Physiol. 2023 Mar 1;324(3):G190-G195.

Call L, Molina T, Stoll B, Guthrie G, Chacko S, Plat J, Robinson J, Lin S, Vonderohe C, Mohammad M, Kunichoff D, Cruz S, Lau P, Premkumar M, Nielsen J, Fang Z, Olutoye O, Thymann T, Britton R, Sangild P, Burrin D. Parenteral lipids shape gut bile acid pools and microbiota profiles in the prevention of cholestasis in preterm pigs. J Lipid Res. 2020 Jul;61(7):1038-1051.

Burrin, D, P Sangild, B Stoll, T Thymann, R Buddington, J Marini, O Olutoye, and RJ Shulman. Translational Advances in Pediatric Nutrition and Gastroenterology: New Insights from Pig Models. Annu Rev Anim Biosci. 8:321-354, 2020

Metabolomic signatures distinguish the impact of formula carbohydrates on disease outcome in a preterm piglet model of NEC. Call L, Stoll B, Oosterloo B, Ajami N, Sheikh F, Wittke A, Waworuntu R, Berg B, Petrosino J, Olutoye O, Burrin D. Microbiome. 2018.19;6(1):111.

Our laboratory works on basic and translational projects designed to establish how nutritional support, enteral versus parenteral, effects gut and liver function and susceptibility to disease in early development. We have used the neonatal piglet to established unique models of parenteral nutrition-associated liver disease (PNALD), necrotizing enterocolitis (NEC) and short-bowel syndrome (SBS) to address clinically -relevant problems in pediatric gastroenterology.

Current projects in the laboratory seek to identify the cellular and molecular mechanism that lead to PNALD and metabolic dysfunction associated with prematurity and neonatal parenteral nutrition (PN) support. Our recent studies in preterm pigs show that new generation lipid emulsions prevent PNALD and uniquely shape the gut microbiome. We are currently exploring how specific nutrients in commercial lipid emulsions, such as phytosterols, alter the susceptibility to PNALD and shape the gut microbiome and metabolome. We are exploring how parenteral lipid emulsions affect bile acid homeostasis and activity of the farnesoid X receptor–fibroblast growth factor 19 (FGF19) signaling pathway in the liver and gut.

Studies also are aimed at establishing how dietary carbohydrate composition shapes the gut microbiome and the incidence of NEC. Our recent studies suggest that lactose vs. corn syrup solid-based formula protects against NEC in preterm pigs and that this correlates with changes in the gut microbiome and metabolomic profiles. New studies are focusing on how human donor milk and probiotics shape the gut perinatal microbiome and may reduce the risk of NEC. We are also focused on how developmental changes in host citrulline and arginine metabolism measured using in vivo stable isotopic kinetic studies may contribute to intestinal perfusion and ischemia that has been linked to NEC. We are pursuing the molecular basis of this finding using metabolic studies in cultured enteroids.

We take an integrative experimental approach dictated by the research question to address relevant functions at the whole animal, tissue, cellular or molecular level. We use sophisticated metabolic, cell biological and molecular approaches, such as stable isotope metabolomics,16S rDNA sequencing, RNA-seq transcriptomics, and confocal microscopic imaging to identify the cellular localization of specific signals involved in the metabolism, proliferation and survival of relevant cell types, including gut enteroids and hepatocyte spheroids.

I have established collaborations with BCM colleagues investigating the role of nutrition and bile acid receptor agonist. I am actively involved the digestive research community in the Texas Medical Center as a mentor to postdoctoral fellows and students through our the NIH funded (T32) training grant. I serve on the Internal Advisory Committee, Texas Medical Center Digestive Disease Center (DDC) and Director of the Pilot Feasibility Grant program.

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Host-Microbe Interactions in Neurodevelopment and Behavior

Assistant Professor, Tenure-Track
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Alkek Research Tower
McNair Scholar
Baylor College of Medicine

SHELLY BUFFINGTON

Recent Publications

Di Gesù CM, Buffington SA. The early life exposome and autism risk: a role for the maternal microbiome? Gut Microbes. 2024. 16(1):2385117.

Di Gesù* CM, Matz* LM, Bolding IJ, Fultz RS, Hoffman KL, Gammazza AM, Petrosino JF, Buffington SA. 2021. Maternal gut microbiota mediate intergenerational effects of high-fat diet on descendant social behavior. Cell Rep. 2023;42(5):112498. .

Buffington SA, Viana Di Prisco G, Auchtung TA, Ajami NJ, Petrosino JF, Costa-Mattioli M. 2016. Microbial reconstitution reverses maternal diet-induced synaptic and social deficits in offspring. Cell. 165(7):1762-1775.

Sgritta M, Dooling SD, Buffington SA, Momin EN, Francis MB, Britton RA, Costa-Mattioli M. 2019. Mechanisms underlying microbial-mediated changes in social behavior in mouse models of autism spectrum disorder. Neuron. 101 (2):246-259.

Buffington SA, Dooling SD, Sgritta M, Noecker C, Murillo OD, Felice DF, Turnbaugh PJ, Costa-Mattioli M. 2021. Dissecting the contribution of host genetics and the microbiome in complex behaviors. Cell. 184(7):1740-1756.

The goal of our research program is to elucidate the mechanisms by which maternal environmental exposures leading up to and throughout the reproductive lifespan impact neurodevelopmental health outcomes and risk for neurodevelopmental disorders in descendant generations. We are particularly interested in the duality of the maternal gut microbiome as both a potential contributor to and a therapeutic target for reducing risk for neurodevelopmental disorders in children. Through studying host-microbe interactions contributing to neurodevelopment, brain function, and behavior, we are identifying fundamental mechanisms by which the brain processes information and drives complex behavior. Simultaneously, we are discovering new and innovative therapeutic approaches to reduce risk for neurodevelopmental disorders linked to maternal environmental exposures.

Why are we as neuroscientists interested in studying the maternal gut microbiome and how environmental factors perturb this critical ecosystem? Maternal gut microbiota affect early-life offspring development via four primary routes: 1) pre- and 2) postnatal exposure to maternal microbially-derived metabolites, 3) modulation of immune signaling at the maternal-fetal interface, and 4) postnatal mother-to-infant vertical transmission of gut microbiota. During pregnancy, maternal gut microbiota provide metabolites essential for fetal growth, immune cell maturation, and neural circuit formation. At parturition, maternal vaginal and skin microbes pioneer colonizing the infant gut, but only transiently, and are then replaced by maternal gut strains that persist throughout development. Consequently, ‘dysbiosis’ of the gut microbiome is increasingly implicated in the growing burden of non-communicable chronic diseases, including neurodevelopmental disorders. The underlying mechanisms, however, remain unresolved and the potential for therapeutic targeting of the maternal gut microbiome to reduce disease risk in children unrealized.

To close these gaps, we take a highly interdisciplinary approach at the intersection of neuroscience, anaerobic microbiology, and immunology combined with functional integrated multi-OMICs to study host-microbe interactions mediating intergenerational effects of maternal environmental exposures on descendant neurodevelopment and behavior, with the ultimate goal of delivering transformative antenatal care to prevent or mitigate the adverse effects of maternal environmental exposures on neurodevelopment in descendants.

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Disorders of Esophageal Motility

Assistant Professor
Departments of Pediatrics, Gastroenterology
Baylor College of Medicine

ERIC CHIOU

Recent Publications

Virani FR, Chiou EH, Lambert EM. Pediatric Laryngopharyngeal Reflux: Epidemiology, Clinical Presentation, Diagnosis, and Therapeutic Outcomes. Otolaryngol Clin North Am. 2025;58(3):507-517.

Baez-Bravo GA, Chiou EH, Olive A, Davis CM. Pooled Phase 2 and 3 Efficacy and Safety Data on Budesonide Oral Suspension in Adolescents With Eosinophilic Esophagitis. Pediatrics. 2024 Dec 1;154(Suppl 4):S33-S34.

Chiou EH, Olive AP, Davis CM. Gut Microbiota Dysbiosis in Suspected Food Protein Induced Proctocolitis - A Prospective Comparative Cohort Trial. Pediatrics. 2024 Dec 1;154 (Suppl 4):S35-S36.

Kaul I, Chiou EH. The Role of Pediatric Gastroenterologists in the Evaluation of Complex Aerodigestive Disorders. Curr Gastroenterol Rep. 2022 Dec;24(12):211-221.

Ramachandran V, Shah KP, Fishman DS, Chiou EH. Post-fundoplication high-resolution esophageal manometry in a patient with Ehlers-Danlos syndrome. Ann Gastroenterol. 2018 Sep-Oct;31 (5):633.

E Chiou, R Rosen, H Jiang, S Nurko. Diagnosis of supra-esopahgeal gastric reflux: Correlation of oropharyngeal pH with esophageal impedance monitoring for gastroesophageal reflux. Neurogastroenterology and Motility; 23: 717-e326, 2011.

The focus of my research is on disorders of esophageal motility in children. Gastroesophageal reflux disease (GERD) is considered one of the most common gastrointestinal motility disorders, affecting an estimated 1.8% to 8.2% of all children and adolescents worldwide. We now understand that the majority of acid reflux occurs as a result of transient relaxations of the lower esophageal sphincter. Despite the prevalence of GERD, there are still many unanswered questions regarding its diagnosis and management.

I am particularly interested in studying extra-intestinal manifestations of GERD in the airways and lungs. Although varied conditions such as asthma, otitis media, sleep apnea and chronic laryngitis have all been linked to GERD, there is still poor understanding regarding the pathogenesis of these complications. A major reason for this is the lack of an accurate and reliable tool for the diagnosis of reflux in the anatomic areas above the esophagus. Investigations have looked at the accuracy of measuring changes in pH in the proximal esophagus and posterior oropharynx, as well as the use of esophageal impedance monitoring to measure both acid and non-acid reflux. Current projects are looking at the role of gastroesophageal reflux in affecting changes to the human oral microbiome and its relationship to dental lesions. In addition, we are studying the use of transmission electron microscopy to look for ultrastructural changes in the esophageal epithelium known as dilated intercellular spaces (DIS) which have been associated with GERD and alterations in epithelial permeability.

My second area of investigation includes evaluating the use of high-resolution manometry techniques for the diagnosis of esophageal motility disorders in general. We have compared the use of this new modality to conventional techniques and are interested in understanding how these new measures of motility can be used to improve diagnosis, management and clinical outcomes.

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Clinical Epidemiology and Outcomes of GI and Liver Disorders

Professor
Margaret M and Albert B Alkek Professor
and Chair, Department of Medicine
Director, Texas Medical Center Digestive Disease
Baylor College of Medicine

HASHEM B. EL-SERAG

Recent Publications

El-Serag HB, Duong H, Luster M, Kanwal F, Hill DD, Burroughs M, Hernandez C, Haber BA, Larsen LM, Marcinak JF, Wegrzyn LR, Kramer JR. Risk of Hepatocellular Cancer in U.S. Patients With Compensated Cirrhosis Treated With Direct-Acting Antivirals Versus Interferon. Aliment Pharmacol Ther. 2025 Apr;61(7):1226-1237.

El-Serag HB, Thrift AP, Duong H, Ning J, Khaderi S, Singal AG, Asrani SK, Marrero JA, Powell H, Rizwan K, Najjar O, Amos CI, Luster M, Al-Sarraj A, Salem E, Scheurer ME, Chhatwal J, Kaochar S, Kanwal F. Serum levels of total bile acids are associated with an increased risk of HCC in patients with cirrhosis. Hepatol Commun. 2024 Oct 10;8 (11):e0545.

El-Serag H, Kanwal F, Ning J, Powell H, Khaderi S, Singal AG, Asrani S, Marrero JA, Amos CI, Thrift AP, Luster M, Alsarraj A, Olivares L, Skapura D, Deng J, Salem E, Najjar O, Yu X, Duong H, Scheurer ME, Ballantyne CM, Kaochar S. Serum biomarker signature is predictive of the risk of hepatocellular cancer in patients with cirrhosis. Gut. 2024 Feb 16:gutjnl-2024-332034.

Kim HS, Xiao X, Byun J, Jun G, DeSantis SM, Chen H, Thrift AP, El-Serag HB, Kanwal F, Amos CI. Synergistic Associations of PNPLA3 I148M Variant, Alcohol Intake, and Obesity With Risk of Cirrhosis, Hepatocellular Carcinoma, and Mortality. JAMA Netw Open. 2022 Oct 3;5(10):e2234221.

Hashem B. El-Serag, M.D., M.P.H., is a gastroenterologist/hepatologist physician investigator with research expertise in epidemiology and health services research. Her received formal research training and obtained a master's degree in public health. He is a recipient of two VA Career Development Awards which aided in developing his expertise in research methods applied to the epidemiology (including genetic epidemiology) and outcomes of digestive diseases. Previously, Dr. El-Serag served as leader of the Cancer Prevention and Population Sciences Program in the Dan L. Duncan Comprehensive Cancer Center and the program lead for the Clinical Epidemiology and Outcomes Program at the Houston Center for Innovations for Quality, Effectiveness and Safety. Since 2022, he was selected to serve as Baylor College of Medicine's inaugural Vice President of the Learning Health System.

Dr. El-Serag has been funded by >75 research grants including multiple from the NIH, VA, Cancer Prevention Research Institute of Texas (CPRIT), professional societies and industry, and have more than 660 publications (H-index 161). The methodology employed in my studies include primary data collection and secondary analyses of extant disease registers and databases, most notably the national VA databases, Medicare, SEER, and Optum, and recently the largest prospective cohort of patients with cirrhosis, the Texas HCC Consortium. I have been Director and PI for a P30-funded Digestive Diseases Center since 2015. The THCCC cohort has served as the backbone of our ongoing P01 funded program to examine the prevention of hepatocellular carcinoma among patients with metabolic dysfunction associated fatty liver disease (MASLD).

Over the years, he has successfully mentored students, residents, fellows, and junior faculty, providing them with the tools and knowledge necessary to excel in research and clinical practice. His mentorship approach is rooted in a commitment to individualized guidance, emphasizing critical thinking, scientific rigor, and professional development. Dr. El-Serag's extensive experience in clinical and translational research allows him to offer invaluable insights into complex research methodologies and the practical application of scientific discoveries. Under his mentorship, numerous mentees have secured independent funding, published in high-impact journals, and advanced their careers, further attesting to his effectiveness as a mentor in fostering the next generation of researchers.

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Gastrointestinal Virus-Cell Interactions, Pathogenesis and Immunity

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Departments of Molecular Virology and Microbiology, and Medicine-GI
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MARY K. ESTES

Recent Publications

Li NF, Crawford SE, Mittiga SR, Coarfa C, Nguyen-Phuc H, Utama B, Blutt SE, Ramani S, Estes MK. Macrophage phagocytosis of human norovirus-infected cells in an ex vivo human enteroid-macrophage coculture model. mBio. 2025 Aug 13;16(8):e0118025.

Patil K, Ayyar BV, Hayes NM, Neill FH, Bode L, Estes MK, Atmar RL, Ramani S. 2'-Fucosyllactose inhibits human norovirus replication in human intestinal enteroids. J Virol. 2025 25;99 (2):e0093824.

Ettayebi K, Kaur G, Patil K, Dave J, Ayyar BV, Tenge VR, Neill FH, Zeng X-L, Speer AL, Di Rienzi SC, Britton RA, Blutt SE, Crawford SE, Ramani S, Atmar RL, Estes MK. Insights into human norovirus cultivation in human intestinal enteroids. mSphere. 2024;9 (11):e0044824.

Murakami K, Tenge VR, Karandikar UC, Lin SC, Ramani S, Ettayebi K, Crawford SE, Zeng XL, Neill FH, Ayyar BV, Katayama K, Graham DY, Bieberich E, Atmar RL, Estes MK. Bile acids and ceramide overcome the entry restriction for GII.3 human norovirus replication in human intestinal enteroids. Proc Natl Acad Sci U S A. 2020 Jan 21;117(3):1700-1710.

Blutt SE, Klein OD, Donowitz M, Shroyer N, Guha C, Estes MK. Use of organoids to study regenerative responses to intestinal damage. Am J Physiol Gastrointest Liver Physiol. 2019 Dec 1;317 (6):G845-G852.

Ettayebi K, Crawford SE, Murakami K, Broughman JR, Karandikar U, Tenge VR, Neill FH, Blutt SE, Zeng XL, Qu L, Kou B, Opekun AR, Burrin D, Graham DY, Ramani S, Atmar RL, Estes MK. Replication of human noroviruses in stem cell-derived human enteroids. Science. 2016. 23;353(6306):1387-1393.

I am a Distinguished Service Professor with co-appointments in the departments of Molecular Virology and Microbiology and Medicine at Baylor College of Medicine (BCM). I direct a multidisciplinary research program focused on understanding gastrointestinal (GI) disease, including host–microbe interactions, and developing methods to prevent and treat global disease. My research includes molecular studies that have resulted in significant basic science discoveries related to GI virus replication and viral pathogenesis, as well as the translation of these discoveries into candidate vaccines. Our norovirus vaccine is being developed by Hillevax vaccines and is in field trials, and our rotavirus virus-like particle (VLP) vaccine is now used by Immucell to produce a colostrum product to prevent calf scours. Until November 1, 2014, I was the founding director of the Texas Medical Center Digestive Diseases Center (DDC), with a theme of GI Infections and Injury. I now serve on its Internal Advisory Committee and Co-direct an Enteroid/Organoid core. The DDC actively fosters an environment that facilitates and enriches research by encouraging interactions between basic scientists and clinical investigators, in addition to providing scientific core services, training, and access to state-of-the-art equipment and technological expertise that are difficult to obtain on an individual basis. Various honors recognize my work, such as service on NIH, WHO, and AGA review groups and committees; prior service as president of the American Society for Virology; election to the NAM, NAS, and NAI; an AGA Lifetime Achievement Award; an AGA William Beaumont Prize in Gastroenterology; an AGA Distinguished Mentor Award; a BCM Mentoring Award; and an ASM Lifetime Achievement Award.

We are using viruses that infect distinct types of cells (enterocytes, crypt cells, M cells) in the GI tract as probes to learn about the biology, host response and gene expression of these cells. Our work on the molecular biology of GI viruses uses two viruses, rotaviruses, the major cause of diarrhea in children and animals worldwide, and noroviruses, the cause of almost all (>96%) outbreaks of epidemic gastroenteritis in all age groups. Most studies are multidisciplinary and involve in vitro and in vivo models of infection including studies in volunteers, animal models and novel ex vivo human intestinal mini-gut enteroid cultures.

Our research program is divided into several areas including molecular analysis of GI viral-host interactions that affect pathogenesis and also include studies to develop strategies for mucosal immunization with non-replicating virus-like particles (VLPs), identification of intestinal glycan receptors for GI viruses, and understanding the stem cell response to infection. Cloning and sequencing the first human calicivirus genome from Norwalk virus (NV) led to development and use of new diagnostic assays that have resulted in changing our understanding of the natural history and epidemiology of infections with these viruses. Notably, NV and related viruses are now recognized as important causes of disease in children and in immunocompromised individuals, including transplant patients. Recombinant NV VLPs are a promising candidate vaccine. Recent identification of a correlate of protection and initial efficacy studies with promising results are stimulating norovirus vaccine development and field trials are being planned.

Finally, the molecular basis of the restricted replication of Norwalk viruses to the GI tract of humans is being dissected by using the first infectious cDNAs of Norwalk virus in mammalian cells. Histo-blood group antigens are host susceptibility factors and the role of the innate immune response in blocking replication is being pursued.

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Amino acid metabolism and nutritional intervention in obesity-associated fatty liver and cancer

Assistant Professor
Department of Pediatrics
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Amino acid metabolism, metabolomics, nutrition, obesity and cancer
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XIA GAO

Recent Publications

Felix JB, Saha PK, de Groot EL, Tan L, Sharp R, Anaya ES, Li Y, Quang H, Saidi N, Abushamat L, Ballantyne CM, Amos CI, Lorenzi PL, Klein S, Gao X, Hartig SM. N-acetylaspartate from fat cells regulates postprandial body temperature. Nat Metab. 2025 Jul 23;.

Wang W, Aguilar M, Datta S, Alley A, Tadesse M, Wang X, Gao X#, Zhang R#. Dual inhibitor of MDM2 and NFAT1 for experimental therapy of breast cancer: in vitro and in vivo anticancer activities and newly discovered effects on cancer metabolic pathways. Front Pharmacol. 2025;16:1531667.

Gao X, Sanderson SM, Dai Z, Reid MA, Cooper DE, Lu M, Richie JP Jr, Ciccarella A, Calcagnotto A, Mikhael PG, Mentch SJ, Liu J, Ables G, Kirsch DG, Hsu DS, Nichenametla SN, Locasale JW. Dietary methionine influences therapy in mouse cancer models and alters human metabolism. Nature. 2019 Aug;572(7769):397-401.

Gao X, Locasale JW, Reid MA. Serine and Methionine Metabolism: Vulnerabilities in Lethal Prostate Cancer. Cancer Cell. 2019 Mar 18;35(3):339-341

Gao X, Lee K, Reid MA, Sanderson SM, Qiu C, Li S, Liu J, Locasale JW. Serine Availability Influences Mitochondrial Dynamics and Function through Lipid Metabolism. Cell Rep.2018 Mar 27;22(13):3507-3520.

Gao X, van der Veen JN, Zhu L, Chaba T, Ordoñez M, Lingrell S, Koonen DP, Dyck JR, Gomez-Muñoz A, Vance DE, Jacobs RL. Vagus nerve contributes to the development of steatohepatitis and obesity in phosphatidylethanolamine N-methyltransferase deficient mice. J Hepatol. 2015 Apr;62 (4):913-20.

I am a former DDC Pilot Feasibility Awardee. My NIH K99-funded projects aim to understand amino acid metabolism in cell proliferation and cell fate transitions, and to develop targeted nutritional interventions and therapeutics for metabolic diseases such as obesity, fatty liver disease, and cancer. I was recruited to the CNRC in 2021 and received a prestigious Research Scholar Award funded by the Cancer Prevention and Research Institute of Texas (CPRIT). My work has been published in Nature, Cell Metabolism, and the Journal of Hepatology.The Gao Lab employs metabolomics alongside in vitro and in vivo mouse models to investigate the interactions between nutrition and metabolism in the development, progression, and treatment of metabolic diseases. Current efforts focus on understanding the dynamics of amino acid metabolism in relation to the development and progression of metabolic dysfunction-associated steatotic liver disease (MASLD). T32 collaborations include Dr. Shen and Dr. Burrin.
Theme: Molecular Hepatology.

Ongoing projects in our lab focus on two major areas: cancer and obesity-associated fatty liver diseases.

Cancer Projects: Funded by R00 and CPRIT, these projects aim to evaluate the immune relevance of dietary methionine restriction in tumor inhibition and explore its potential as an adjuvant cancer therapy. We utilize a wide range of syngeneic mouse models and patient-derived xenograft models to assess immune system involvement and identify immunological factors contributing to the antitumor effects of methionine restriction. Additionally, we employ high-throughput in vitro drug screening, combined with in vivo validation, to determine the feasibility of methionine restriction as a therapeutic strategy. Our lab's metabolomics and lipidomics platforms enable in-depth investigation of the mechanisms underlying the observed phenotypes.

Obesity and Fatty Liver Disease Projects: Supported by USDA and a DDC Pilot Award, these projects aim to evaluate metabolic dynamics in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), with a particular focus on amino acid metabolism. Using a metabolomics approach, we have mapped metabolic changes during MASLD development and progression in a diet-induced mouse model (GAN DIO-NASH). We are currently conducting lipidomics and proteomics analyses on the same samples to identify novel nutritional and metabolic factors involved in MASLD pathogenesis. Additionally, we are investigating the role of the microbiome in host metabolism, disease progression, and treatment.

Our lab is actively seeking talented students and postdoctoral fellows to join our team.

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Neonatal Liver Disease, Cholestasis, Fibrosis, and Therapeutic Targets

Assistant Professor
Department of Pediatrics-Nutrition
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GREGORY GUTHRIE

Recent Publications

Guthrie G, Vonderohe C, Meléndez Hebib V, Stoll B, Burrin D. Multicomponent parenteral lipid emulsions do not prevent liver injury in neonatal pigs with obstructive cholestasis. JCI Insight. 2025. 17;10(10): e189196.

Guthrie G, Stoll B, Chacko S, Mohammad M, Style C, Verla M, Olutoye O, Schady D, Lauridsen C, Tataryn N, Burrin D. Depletion and enrichment of phytosterols in soybean oil lipid emulsions directly associate with serum markers of cholestasis in preterm parenteral nutrition-fed pigs. JPEN J Parenter Enteral Nutr. 2022 Jan;46(1):160-171.

Guthrie G, Vonderohe C, Burrin D. Fibroblast growth factor 15/19 expression, regulation, and function: An overview. Mol Cell Endocrinol. 2022 May 15;548:111617.

Guthrie G, Burrin D. Impact of Parenteral Lipid Emulsion Components on Cholestatic Liver Disease in Neonates. Nutrients. 2021 Feb 4;13 (2):508.

Guthrie G, Stoll B, Chacko S, Lauridsen C, Plat J, Burrin D. Rifampicin, not vitamin E, suppresses parenteral nutrition-associated liver disease development through the pregnane X receptor pathway in piglets. Am J Physiol Gastrointest Liver Physiol. 2020 Jan 1;318(1):G41-G52.

I am an Assistant Professor in the Department of Pediatrics at Baylor College of Medicine and a former postdoctoral trainee on this T32 program. My research program, supported by foundation grants, pilot funding, and an NIDDK Career Development Award (K01 DK129408), focuses on understanding and treating liver disease, with a particular emphasis on neonatal cholestasis, fibrosis, and therapeutic target identification. My long-term goal is to delineate the molecular mechanisms driving liver injury—including fibrosis, inflammation, and oxidative stress—and to evaluate potential pharmaceutical interventions to prevent or treat liver diseases such as biliary atresia and parenteral nutrition-associated liver disease.

My laboratory employs a clinically relevant neonatal piglet model for in vivo studies and extensive in vitro approaches. I have expertise in isolating and culturing all primary hepatic cell types and utilize both 2D and advanced 3D culture systems, including hepatic organoids and multi-cellular spheroids, to investigate cell–cell interactions in the context of liver fibrosis. My research incorporates molecular techniques such as adeno-associated virus (AAV)-mediated gene modulation to identify and characterize therapeutic targets, complemented by downstream analyses including RNA-seq and metabolomics. I have studied bile acid homeostasis in neonatal obstructive cholestasis, particularly in the context of parenteral lipid emulsions, and examined the molecular and cellular mechanisms contributing to pediatric biliary atresia using bile duct ligation models. I have also developed organoid liver models and hepatocyte/cholangiocyte spheroid systems to determine cell-specific responses to high bile acid concentrations, inflammation, and oxidative stress, with the goal of identifying strategies to reduce the need for liver transplants.

My K01 research on obstructive cholestasis and parenteral lipid emulsions was recently highlighted in JCI Insight (PMID 42044694). I am active in the Texas Medical Center DDC Enrichment Program Committee and lead the GI Focus Group, a monthly forum for early career faculty, postdocs, and senior graduate students to present research. I have mentored T32 fellows, both current (Elefson) and graduated (Vonderohe), and maintain active collaborations with Burrin, Harpavat, Jafar-Nejad, Mysore, and Vonderohe. My research theme is Molecular Hepatology.

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Neonatal Nutrition and Microbiome Research to Improve Preterm Infant Health

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I am an Associate Professor of Pediatrics (with tenure) in the Division of Neonatology at Baylor College of Medicine and a Neonatologist at Texas Children's Hospital. I serve as Program Director of both the Neonatal Nutrition Program and the Neonatal Nutrition Research Program, where I have designed and led multiple clinical and translational studies focused on optimizing and innovating nutrition for extremely premature infants in the Neonatal Intensive Care Unit (NICU).

With the largest NICU in the United States, I have conducted seven large-scale nutritional clinical studies enrolling more than 1,000 very low birth weight infants, including studies evaluating how exclusive human milk–based diets improve outcomes for preterm infants. My NIH-funded R01 research focuses on how nutrition impacts the gut–liver–microbiome axis in preterm infants, integrating basic science and clinical research to generate translational projects aimed at improving neonatal care. Additional ongoing projects include:

As a physician–scientist with over 10 years of mentoring experience, I have developed a structured mentoring program for trainees in neonatal nutrition research. I currently mentor medical students, graduate students, residents, postdoctoral fellows, and junior faculty, providing support in study design, data analysis, and translational research execution. Many projects are designed to generate clinically actionable findings that directly improve outcomes for premature infants.

I have built a robust research infrastructure that includes research coordinators, research dietitians, and a dedicated statistician, and I maintain collaborations with the USDA/ARS Children's Nutrition Research Center and other leading institutions nationwide. My T32 collaborations include Burrin, Preidis, and Sasirekha under the Clinical and Translational Research theme.

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Recent Publications

Hair AB, Sullivan KM, Ahmad I, Zaniletti I, Acker SN, Premkumar MH, Reber K, Huff KA, Nayak SP, DiGeronimo R, Kim J, Roberts J, Markel TA, Brozanski B, Sharma J, Piazza AJ, Yanowitz TD; Children's Hospitals Neonatal Consortium Necrotizing Enterocolitis Focus Group. Initial surgery for spontaneous intestinal perforation in extremely low birth weight infants is not associated with mortality or in-hospital morbidities. .J Perinatol. 2024 Dec;44(12):1746-1754.

Adeniyi-Ipadeola GO, Hoffman KL, Yang H, Javornik Cregeen SJ, Preidis GA, Ramani S, Hair AB. Human milk cream alters intestinal microbiome of preterm infants: a prospective cohort study. Pediatr Res. 2024 May;95(6):1564-1571.

Holzapfel LF, Unger JP, Gordon P, Yang H, Cluette-Brown JE, Gollins LA, Hair AB, Martin CR. Fatty acid concentrations in preterm infants fed the exclusive human milk diet: a prospective cohort study. J Perinatol. 2024 May;44(5):680-686.

Hair AB, Patel AL, Kiechl-Kohlendorfer U, Kim JH, Schanler RJ, Hawthorne KM, Itriago E, Abrams SA, Blanco CL. Neurodevelopmental outcomes of extremely preterm infants fed an exclusive human milk-based diet versus a mixed human milk + bovine milk-based diet: a multi-center study. J Perinatol. 2022 Nov;42(11):1485-1488.

Bergner EM, Taylor SN, Gollins LA, Hair AB. Human Milk Fortification: A Practical Analysis of Current Evidence. .Clin Perinatol. 2022 Jun;49(2):447-460.

SANJIV HARPAVAT

Developmental Etiology and Clinical Studies of Biliary Atresia

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Baylor College of Medicine

Recent Publications

Harpavat S, Borovsky KA, Scheurer ME, Cavallo L, Erhiawarie FE, Vasudevan S, Vogel AM, Cerminara D, Tessier EM, Patel KR, Devaraj S, Shneider BL. A phase 2 trial of short-term intravenous N-acetylcysteine in biliary atresia after Kasai portoenterostomy. Hepatol Commun. 2025, 9(7):e0729.

Upton AM, Rabbani TA, Hernandez JA, Shneider BL, Harpavat S. The "maximum echogenicity" at the right portal vein: Biliary atresia versus Alagille syndrome. J Pediatr Gastroenterol Nutr. 2025, 81(2):212-216.

Upton AM, Hernandez JA, Shneider BL, Rabbani TA, Devaraj S, Patel KR, Vasudevan SA, Vogel AM, Harpavat S. An ultrasound approach to visualize the "duct at the hilum" in infants undergoing evaluation for biliary atresia. J Pediatr Gastroenterol Nutr. 2025, 81(2):204-211.

Harpavat S, Garcia-Prats JA, Anaya C, Brandt ML, Lupo PJ, Finegold MJ, Obuobi A, ElHennawy AA, Jarriel WS, Shneider BL. Diagnostic Yield of Newborn Screening for Biliary Atresia Using Direct or Conjugated Bilirubin Measurements. JAMA. 2020, 323(12):1141-1150.

Harpavat S, Garcia-Prats JA, Shneider BL. Newborn Bilirubin Screening for Biliary Atresia. N Engl J Med. 2016, 375(6):605-6.

Harpavat S, Finegold MJ, Karpen SJ. Biliary atresia patients have elevated direct/conjugated bilirubin levels shortly after birth. Pediatrics 2011, 128:e1428-33.

Our research program focuses on biliary atresia, the #1 reason for pediatric liver transplant. There are multiple gaps in our understanding of biliary atresia's etiology, diagnosis, and treatment. Our initial work focused on the commonly held notion that biliary atresia is acquired in the few weeks of life. Two findings supported this explanation: (a) infants often appear healthy at birth, and (b) biliary atresia does not follow a genetic pattern (discordant in twins, does not run in families). However, through clinical studies, we found that infants with biliary atresia already have elevated direct or conjugated levels at birth, suggesting that the disease starts in utero [PMID: 22106076]. We are building on this initial observation through a series of studies:

Exploring possible etiologies of biliary atresia. This work focuses on sequencing biliary tissue, to look further for genetic changes which may be associated with disease. It is based on our review of existing data suggesting that biliary atresia may already be present as early as 12-15 weeks gestation [PMID: 31335837]

Developing a newborn screening test for biliary atresia, which will allow for earlier diagnosis/better outcomes. Infants with biliary atresia have better outcomes when identified and treated earlier. Unfortunately, in the United States, the biliary atresia diagnosis is often delayed. To address this issue, we performed a prospective, multi-center screening study which established that newborn direct/conjugated bilirubin measurements can effectively identify infants with biliary atresia [PMID: 27509119, 32207797]. Now we are assaying different ways to measure conjugated bilirubin concentrations from the state newborn screening blood spot cards, with the goal of making screening easily available to every baby born in the United States.

Implementing a new ultrasound approach that bypasses current complex, invasive diagnostic testing for biliary atresia. We have developed a new diagnostic approach using abdominal ultrasound, that has the potential to bypass standard diagnostic testing which is time-consuming, invasive, and expensive. This ultrasound approach can be performed quickly in feeding (non-fasting) infants [PMID: 40452357, 40452374]. Current work includes conducting a global implementation study as well as working with colleagues in the Department of Radiology to create AI algorithms capable of performing the approach.

Testing new therapies that have the potential to reduce need for liver transplant. This work builds on our single-center trial of N-acetylcysteine in biliary atresia [PMID: 40489761]. Currently we are collaborating with DDC member Gregory Guthrie, Ph.D., to trial therapies in piglet models, as well as translating these findings to conduct future clinical trials.

Identifying genetic factors which explain differences in patient outcomes. This work is an extension of our previous analysis of whole exome sequencing of patients with biliary atresia [PMID: 36942736], exploring genetic differences among infants.

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Infantile Disorders of Biliary Function: Closing Current Knowledge Gaps

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Recent Publications

Newton KP, Jayasekera D, Blackford AL, Behling C, Wilson LA, Fishbein MH, Molleston JP, Xanthakos SA, Vos MB, Schwimmer JB; NASH CRN. Longitudinal response to standard of care in pediatric metabolic dysfunction-associated steatotic liver disease: Rates of improvement and worsening, and factors associated with outcomes. .Hepatology. 2025 Jan 3:10.1097/EP. 0000000 000001216.

Wang A, Blackford AL, Behling C, Wilson LA, Newton KP, Xanthakos SA, Mouzaki M, Molleston JP, Jain AK, Hertel P, Harlow Adams K, Schwimmer JB; NASH CRN. Development of Fibro-PeN, a clinical prediction model for moderate-to-severe fibrosis in children with nonalcoholic fatty liver disease. .Hepatology. 2024 Jun 1;79(6):1381-1392.

Jain AK, Buchannan P, Yates KP, Belt P, Schwimmer JB, Rosenthal P, Murray KF, Molleston JP, Scheimann A, Xanthakos SA, Behling CA, Hertel P, Nilson J, Neuschwander-Tetri BA, Tonascia J, Vos MB; Nonalcoholic Steatohepatitis Clinical Research Network (NASH CRN). Nutrition assessment and MASH severity in children using the Healthy Eating Index. Hepatol Commun. 2023 Dec 7;7(12):e0320.

Hassan S, Hertel P. Overview of Progressive Familial Intrahepatic Cholestasis. Clin Liver Dis. 2022 Aug;26(3):371-390.

Hertel PM, Hawthorne K, Kim S, Finegold MJ, Shneider BL, Squires JE, Gupta NA, Bull LN, Murray KF, Kerkar N, Ng VL, Molleston JP, Bezerra JA, Loomes KM, Taylor SA, Schwarz KB, Turmelle YP, Rosenthal P, Magee JC, Sokol RJ; Childhood Liver Disease Research Network Presentation and Outcomes of Infants With Idiopathic Cholestasis: A Multicenter Prospective Study. (ChiLDReN).J Pediatr Gastroenterol Nutr. 2021;73(4):478-484.

Disorders of bile flow (cholestasic liver diseases) that present during infancy and childhood are diverse and, in many respects, still poorly understood. Collectively, these diseases are not uncommon, but individually, they are rare. Biliary atresia, the most common indication for liver transplantation in children, occurs in only approximately 1:10,000 live births. Many other infantile cholestatic liver diagnoses are even less common.

Often, the etiology of cholestasis during infancy is multifactorial. Bile flow may be impaired by numerous factors such as infection, hemolysis, and parenteral nutrition, and bile flow in the immature liver is particularly vulnerable to becoming impaired following one or more of these insults. Genetic factors may also contribute to vulnerability to cholestasis; variants in genes such as those implicated in alpha-1 -antitrypsin deficiency and progressive familial intrahepatic cholestasis are known to play roles in cholestasis in early life. The cause(s) of cholestasis may remain undetermined in many young patients, however.

Texas Children's Hospital and Baylor College of Medicine Division of Pediatrics Gastroenterology and Nutrition participates in an NIH-funded, multi-center consortium known as "ChiLDReN" (Childhood Liver Disease Research Network – childrennetwork.org). The Network consists of fourteen clinical sites in the United States and Canada, each of which enrolls children with cholestatic liver diseases in protocols that include four long-term observational studies as well as therapeutic drug trials. The strength of the Network lies in its prospective, longitudinal (up to 10 years) study of children with liver diseases, as well as its ability to capture substantial numbers of study subjects diagnosed with rare diseases by virtue of involvement of multiple top pediatric tertiary care hospitals.

Dr. Hertel is responsible for the conduct of the ChiLDReN Network observational protocols at the Texas Children's Hospital site, is Chair of the Idiopathic Neonatal Hepatitis working group, and is an active participant in the PFIC (progressive familial intrahepatic cholestasis) and Biliary Atresia Genomics and Pathophysiology workgroups. A wonderful opportunity exists to access the Network's data and tissue repository by submission (and approval) of ancillary proposals – please contact Dr. Hertel or one of our other ChiLDReN site Co-Investigators for more information. Dr. Hertel would be pleased to direct those interested to a contact with compatible interests.

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JOHN M. HOLLIER

New Paradigms for Treating Functional Abdominal Pain Disorders in Children

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Recent Publications

Hollier JM, Czyzewski DI, Self MM, Liu Y, Weidler EM, van Tilburg MAL, Varni JW, Shulman RJ.Associations of Abdominal Pain and Psychosocial Distress Measures With Health-Related Quality-of-Life in Pediatric Healthy Controls and Irritable Bowel Syndrome. J Clin Gastroenterol. 2020 Jun 16.

Hollier JM, van Tilburg MAL, Liu Y, Czyzewski DI, Self MM, Weidler EM, Heitkemper M, Shulman RJ. Multiple psychological factors predict abdominal pain severity in children with irritable bowel syndrome. Neurogastroenterol Motil. 2019 Feb;31(2):e13509.

Hollier JM, Vaughan AO, Liu Y, van Tilburg MA, Shulman RJ, Thompson DI. Maternal and Child Acceptability of a Proposed Guided Imagery Therapy Mobile App Designed to Treat Functional Abdominal Pain Disorders in Children: Mixed-Methods Predevelopment Formative Research. JMIR Pediatr Parent. 2018 Jun 29;1(1):e6. doi: 10.2196/pediatrics.8535.

Hollier JM, Czyzewski DI, Self MM, Weidler EM, Smith EO, Shulman RJ. Pediatric Irritable Bowel Syndrome Patient and Parental Characteristics Differ by Care Management Type. J Pediatr Gastroenterol Nutr. 2017 Mar;64(3):391-395.

Hollier JM, Hinojosa-Lindsey M, Sansgiry S, El-Serag HB, Naik AD. Clinical and psychosocial variables associated with behavioral intentions to undergo surveillance endoscopy. BMC Gastroenterol. 2014 Jun 10;14:107.

I am a former postdoctoral trainee on this T32 and Texas Children's Hospital Pediatric Pilot Award Recipient. I was mentored by Drs. Rob Shulman, former TG Program Director, and Hashem El-Serag in the field of disorders of gut-brain interaction. I have been supported by an NIDDK Career Development Award (K23) and an (R03). The long-term goals of Dr. Hollier's research are to (1) investigate the association between innate and environmental factors and disorders of gut-brain interaction in children, (2) exploit novel technologies to increase access to evidence-based interventions for affected pediatric patients, and (3) disrupt traditional healthcare systems to deliver evidence-based, technological interventions to promote better health outcomes for affected pediatric patients and their families. I utilize a variety of clinical and community databases to characterize better affected patient populations. I have also developed a patient-centered guided imagery therapy mobile application with input from young pediatric patients with disorders of -brain interaction and their caregivers. The app is being tested in a randomized clinical pilot trial to assess whether this approach reduces pain and psychological distress. T32 collaborations include Burrin, El-Serag, Shneider. Theme: Clinical and Translational.

Unfortunately many novel discoveries in the clinical research arena do not move beyond journal publications and often sit idle or die in the medical literature. This poor transition from bench to bedside is a major inefficacy of our biomedical research roadmap and needs to be addressed to enhance overall population health.

The overarching theme of my research strives to improve the delivery of optimal care to pediatric patients and demonstrate subsequent improvements in patient outcomes. My research utilizes clinical research, health services research, and implementation science to accomplish these goals. Functional abdominal pain disorders, like irritable bowel syndrome, are highly prevalent in children and serve as a template for my current research investigations.

I am particularly interested in quantifying the effectiveness of psychological therapies like guided imagery therapy on affected patients' abdominal pain symptoms and quality-of-life when delivered via electronic mobile health application. My research team currently is developing a mobile health application with the guidance of a multidisciplinary team and will conduct a clinical trial using the mobile health application to assess its efficacy. My group is also exploring the role of sleep disturbances on the various factors known to be associated with these disorders using actigraphy. Future work will explore implementation of an electronic mobile health application as a treatment for functional abdominal pain disorders within pediatric clinics. This process will help ensure that evidence-based practices will reach the bedside and avoid the abyss of novel yet ignored clinical remedies.

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Cellular Signaling Pathways and Circuits in Gastrointestinal Health and Infectious Diarrhea

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JOSEPH HYSER

Recent Publications

Engevik MA, Banks LD, Engevik KA, Chang-Graham AL, Perry JL, Hutchinson DS, Ajami NJ, Petrosino JF, Hyser JM. Rotavirus infection induces glycan availability to promote ileum-specific changes in the microbiome aiding rotavirus virulence. Gut Microbes. 2020 Sep 2;11 (5):1324-1347.

Chang-Graham AL, Perry JL, Strtak AC, Ramachandran NK, Criglar JM, Philip AA, Patton JT, Estes MK, Hyser JM. Rotavirus Calcium Dysregulation Manifests as Dynamic Calcium Signaling in the Cytoplasm and Endoplasmic Reticulum. Sci Rep. 2019 Jul 25;9(1):10822.

Chang-Graham AL, Danhof HA, Engevik MA, Tomaro-Duchesneau C, Karandikar UC, Estes MK, Versalovic J, Britton RA, Hyser JM. Human Intestinal Enteroids With Inducible Neurogenin-3 Expression as a Novel Model of Gut Hormone Secretion. Cell Mol Gastroenterol Hepatol. 2019 Apr 25;8(2):209-229.

Chang-Graham AL, Perry JL, Strtak AC, Ramachandran NK, Criglar JM, Philip AA, Patton JT, Estes MK, Hyser JM. Rotavirus Calcium Dysregulation Manifests as Dynamic Calcium Signaling in the Cytoplasm and Endoplasmic Reticulum. Sci Rep. 2019 Jul 25;9(1):10822.

Hyser JM, Estes MK. Pathophysiological Consequences of Calcium-Conducting Viroporins. Annu Rev Virol. 2015. Nov;2(1):473-96.

Perry JL, Ramachandran NK, Utama B, Hyser JM. Use of genetically-encoded calcium indicators for live cell calcium imaging and localization in virus-infected cells. Methods. 2015. Nov 15;90:28-38.

I am a former post-doctoral trainee on this T32 and DDC Pilot Feasibility Awardee. My early research used the skills and data generated as a TG Fellow to successfully apply for a NIDDK Career Development Award (K01) and I have gone on to establish independent R01 (DK115507) and R21 (AI137710) funded research projects. The long-term goals of the Hyser Laboratory are to characterize signaling pathways in the gut and liver that are critical for maintaining normal function, and determine how pathogenic and commensal microbes exploit these signaling pathways to their benefit. A major focus of studies in our lab involves live microscopy imaging of cellular signaling dynamics using cells, enteroid mini-guts, and animal models expressing fluorescent biosensors for important secondary messengers, including Ca2+, cAMP, nitric oxide. My recent work published in Science 2020 and Science Advances 2025 using cell lines and enteroids stably expressing the GCaMP6 Ca2+ sensor, discovered that individual rotavirus-infected cells elicit a potent paracrine purinergic signal that requires viroporin activity and activates Ca2+ signals in neighboring uninfected cells. This paracrine signal is mediated by the repeated release of purines, which diffuse to, and activate purinergic receptors on the neighboring cells. I am actively involved in mentoring of >10 trainees and is Co-Director of the BCM Immunology and Microbiology Graduate Program. I am active in the Texas Medical Center DDC Enrichment Program Committee.T32 collaborations include Britton, Estes, Speer, Sasirekha, Thevananther. Theme: Immunology and Microbiology. (2 TG trainees in past yr).

Cells communicate using a myrid of extracellular molecules that are sensed by receptors that are coupled to intracellular signaling pathways. The integration of these pathways helps define cell specialization and regulates virtually all cellular functions. The long-term goal of the Hyser laboratory is to characterize signaling pathways in the gut and liver that are critical for maintaining normal function, and determine how pathogenic and commensal microbes exploit these signaling pathways to their benefit.

Our research program is divided into three broad (and overlapping) themes:
1) Host signaling pathways underlying rotavirus diarrhea.
2) Characterization of signaling pathways exploited by enteric diarrheal pathogens.
3) Engineering enteroids to study gastrointestinal functions.

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Shaping the Gut Microbiome to Modulate Intestinal Inflammation

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FAITH IHEKWEAZU

Recent Publications

Ihekweazu FD, Fofanova TY, Queliza K, Nagy-Szakal D, Stewart CJ, Engevik MA, Hulten KG, Tatevian N, Graham DY, Versalovic J, Petrosino JF, Kellermayer R. Bacteroides ovatus ATCC 8483 monotherapy is superior to traditional fecal transplant and multi-strain bacteriotherapy in a murine colitis model. Gut Microbes. 2019;10(4):504-520.

Ihekweazu FD, Versalovic J. Development of the Pediatric Gut Microbiome: Impact on Health and Disease. Am J Med Sci. 2018 Nov;356 (5):413-423.

Ihekweazu FD, Kellermayer R. (2016) Fecal Microbiota Transplantation: is it time for children? In M. Manfredi & G.L. de'Angelis (Eds.), Probiotics in Children (pp 309-326). New York, NY: Nova Science Publishers, Inc.

Ihekweazu FD, Fofanova T, Nagy-Szakal D, Hulten K, Queliza K, Opekun A, Petrosino JF, Graham DY, Kellermayer R. Complex and Defined Bacteriotherapy Can Inhibit Acute Colitis in Mice. Oral Presentation, World Congress of Pediatric Gastroenterology, Hepatology and Nutrition, Montreal, Canada, 2016: No. 818.

Ihekweazu FD, Ajjarapu A, Kellermayer R. Diagnostic Yield of Routine Enteropathogenic Stool Tests in Pediatric Ulcerative Colitis. Ann Clin Lab Sci. 2015 Fall;45(6):639-42

Due to the limited therapies, 20% of pediatric patients with Inflammatory Bowel Disease (IBD) become refractory to traditional medications, requiring surgery within 5 years of diagnosis. Conventional IBD therapy includes immunosuppressive agents, which carry significant side effects, including infection and malignancy. In order to address this critical need for new therapeutic options, I have concentrated on the role of the gut microbiota in disease pathogenesis.

I have focused my research on bacteriotherapy as a therapeutic modality for IBD, studying both human and murine models of disease. While my initial interest was in using fecal microbiota transplantation (FMT) for the treatment of IBD, I learned that the fecal microbiome is complex, dynamic, and highly variable, complicating the dissection of the key therapeutic attributes. Therefore, I sought to identify stable bacteria that carry the therapeutic effects of FMT. I determined that Bacteroides ovatus monotherapy led to improved outcomes compared to FMT (decreased weight loss and inflammation, enhanced epithelial recovery, increased survival) in a murine model of colitis. Taking this further, I now aim to determine the mechanism behind the anti-inflammatory effect of B. ovatus therapy during murine colitis, focusing on its ability to produce short chain fatty acids and thereby enhance epithelial barrier function and modulate the immune system. My long-term goal is to advance the field by identifying next generation probiotics, which will be effective and safe for the treatment of IBD.

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HAMED JAFAR-NEJAD

Roles of glycosylation and genetic modifiers in cholestatic diseases and intestinal immune response

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Molecular & Human Genetics
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Development, Disease Models and Therapeutics Graduate Program
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Recent Publications

Fox D, Xie J, Burwinkel JL, Adams JM, Chetal K, Keivandarian M, Faingelernt Y, Subramanian S, Lopez MF, Salomonis N, Zarrin-Khameh N, Gao G, Huppert SS and Jafar-Nejad H (2025). AAV-mediated silencing of Sox4 leads to long-term amelioration of liver phe-notypes in mouse models of Alagille syndrome. ***Gastroenterology*** Online ahead of print.

Niknejad N, Fox D, Burwinkel JL, Zarrin-Khameh N, Cho S, Soriano A, Cast AE, Lopez MF, Huppert KA, Rigo F, Huppert SS, Jafar-Nejad P and Jafar-Nejad H (2023). Antisense oligonucleotide silencing of a glycosyltransferase, *Poglut1*, improves the liver phenotypes in mouse models of Alagille syndrome. ***Hepatology*** 78 (5):1337-1351.

Pandey A[#], Galeone A, Han SY, Story BA, Consonni G, Mueller WF, Steinmetz LM, Vaccari T and Jafar-Nejad H[#] (2023). Gut barrier defects, intestinal immune hyperactivation and enhanced lipid catabolism drive lethality in NGLY1-deficient *Drosophila*. ***Nature Communications*** 14 (1):5667. ([#] corresponding authors)

Adams JM, Huppert KA, Castro EC, Lopez MF, Niknejad N, Subramani- an S, Zarrin-Khameh N, Finegold MJ, Huppert SS, Jafar-Nejad H. Sox9 Is a Modifier of the Liver Dis- ease Severity in a Mouse Model of Alagille Syndrome. ***Hepatology***. 2020 Apr;71(4):1331-1349.

Adams JM, Jafar-Nejad H. Determining Bile Duct Density in the Mouse Liver. ***J Vis Exp***. 2019 Apr 30;(146):10.3791/59587.

Adams JM and Jafar-Nejad H (2019). The roles of Notch signaling in liver development and disease. ***Biomolecules***. 9: 608

The long-term goals of our research are (1) to understand how glycosylation and deglycosylation regulate signaling pathways, animal development, and the pathophysiology of human diseases, and (2) to identify dosage-sensitive genetic modifiers of select human rare diseases in animal models, with the ultimate goal of developing mechanism-based therapeutic strategies.

A major focus of our group over the last decade has been the development of the biliary system in the context of Alagille syndrome (ALGS), a multisystem disorder characterized by bile duct paucity and primarily caused by mutations in the Notch pathway ligand *JAG1*. Alarmingly, only 24-40% of ALGS patients with early cholestasis reach their 18th birthday without a liver transplant. Coexisting cardiovascular and renal anomalies often preclude liver transplantation in ALGS, and even among transplant recipients, complications are common. Currently, no FDA-approved therapies exist to promote biliary development in ALGS or other diseases with bile duct paucity.

To address this unmet need, we have developed mouse models that span the spectrum of ALGS liver disease severity. Through genetic studies, we identified two dosage-sensitive suppressors of the liver phenotypes in these models: the glycosyltransferase *Poglut1* and the transcription factor *Sox4*. We have reported that postnatal knockdown of these genes—via antisense oligonucleotides for *Poglut1* and adeno-associated virus for *Sox4*—dramatically improves liver phenotypes in ALGS models. Ongoing work includes mechanistic and preclinical studies aimed at advancing these candidates toward clinical trials, as well as exploring their relevance to other cholestatic diseases.

We are also investigating the role of N-linked glycosylation and deglycosylation in intestinal immunity and metabolism. Using *Drosophila* as a model, we found that loss of the deglycosylating enzyme NGLY1 disrupts gut barrier integrity, triggers innate immune activation, and induces severe lipid catabolism. Our data suggest that an altered gut microbiome contributes to the developmental delay in *Ngly1*-mutant *Drosophila* larvae. Current efforts focus on determining the molecular basis and the physiological consequences of altered gut microbiome in *Ngly1*-deficienct *Drosophila* and in mutants for select *N*-glycosylation pathway components. These studies aim to uncover shared and distinct roles of glycosylation-related genes in shaping the gut microbiome and modulating the host's response to it. Ultimately, this work may provide novel insight into the pathophysiology of congenital disorders of glycosylation and deglycosylation.

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Clinical Epidemiology of Liver Diseases

Professor and Section Chief-Gastroenterology
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FASIHA KANWAL

Recent Publications

Kanwal F, Tapper EB, Ho C, Asrani SK, Ovchinsky N, Poterucha J, Flores A, Ankoma-Sey V, Luxon B, Volk M. Development of Quality Measures in Cirrhosis by the Practice Metrics Committee of the American Association for the Study of Liver Diseases. Hepatology. 2019 Apr;69(4):1787-1797.

Kanwal F, Kramer JR, Mapakshi S, Natarajan Y, Chayanupatkul M, Richardson PA, Li L, Desiderio R, Thrift AP, Asch SM, Chu J, El-Serag HB. Risk of Hepatocellular Cancer in Patients With Non-Alcoholic Fatty Liver Disease. Gastroenterology. 2018 Dec;155(6):1828-1837.

Kanwal F, Kramer J, Asch SM, Chayanupatkul M, Cao Y, El-Serag HB. Risk of Hepatocellular Cancer in HCV Patients Treated With Direct-Acting Antiviral Agents. Gastroenterology. 2017 Oct;153(4):996-1005.

Kruse RL, Kramer JR, Tyson GL, Duan Z, Chen L, El-Serag HB, Kanwal F. Clinical outcomes of hepatitis B virus coinfection in a United States cohort of hepatitis C virus-infected patients. Hepatology. 2014 Dec;60(6):1871-8.

I am a gastroenterologist, health services researcher, and a Professor of Medicine at Baylor College of Medicine, where I serve as the Chief for the Section of Gastroenterology and Hepatology. I am the Editor-in-Chief of American Gastroenterological Association clinical journal, Clinical Gastroenterology and Hepatology. I have the scientific background, expertise and experience to serve as a mentor for the proposed T32 training program.

My research has focused on health care delivery for patients with liver diseases. Using prospective and retrospective data, my studies provided critical insight into the magnitude of quality problems in liver disease healthcare delivery. I led a randomized controlled trial that implemented a collaborative care model for depression management in liver clinics at 4 different hospitals. This work served as a platform for the American Association for the Study of Liver Disease (AASLD) position statement on Quality Measures for Cirrhosis, for which I am the lead author. One of the quality measures is now part of the measures included in the Centers for Medicare and Medicaid pay for performance programs. This research has been pivotal in setting the stage for the first large-scale quality improvement collaborative in cirrhosis, recently funded by AASLD, that will test and implement strategies to translate evidence into effective clinical practice.

Another focus of my research is outcomes of patients with liver disease, including risk of hepatocellular cancer (HCC) in several emerging populations of patients with liver cirrhosis. I recently completed a grant that examined risk of HCC in a broad spectrum of patients with nonalcoholic fatty liver disease (NAFLD). My current grants from the NIH, Cancer Prevention Research Institute of Texas (CPRIT) and the Veterans Administration (VA) are supporting development of risk-prediction models that combine clinical, electronic medical record, and survey data to improve clinical risk stratification of patients with liver disease, including risk of progression to HCC. For these grants, I have developed disease specific databases (NAFLD, cirrhosis, hepatitis C, and hepatitis B) using the VA nationwide data sources that combine comprehensive and longitudinal clinical, administrative, laboratory, pharmacological data. All databases are linked to Vital Status and cancer registry data. Collectively, these data will serve as valuable and cost-effective resources for mentored research on access, disparities, quality, and cost of healthcare in digestive diseases.

I have mentored MD and PhD students as well as residents, fellows, and junior faculty, both within and outside of Baylor. One of my mentees currently holds an American Cancer Society grant and another holds NIH career development award. Three other mentees successfully competed for national society and internal BCM seed grants. In addition to these mentees, four of my mentees have transitioned from fellowship to faculty positions at academic institutions.

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Role of Epigenome and Microbiome in Inflammatory Bowel Disease

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Department of Pediatrics
Gastroenterology, Hepatology and Nutrition
Children's Nutrition Research Center

RICHARD KELLERMAYER

Recent Publications

Nicholson MR, Mitchell PD, Alexander E, Ballal S, Bartlett M, Becker P, Davidovics Z, Docktor M, Dole M, Felix G, Gisser J, Hourigan SK, Jensen MK, Kaplan JL, Kelsen J, Kennedy M, Khanna S, Knackstedt E, Leier M, Lewis J, Lodarek A, Michail S, Oliva-Hemker M, Patton T, Queliza K, Russell GH, Singh N, Solomon A, Suskind DL, Werlin S, Kellermayer R, Kahn SA.Efficacy of Fecal Microbiota Transplantation for Clostridium difficile Infection in Children. Clin Gastroenterol Hepatol. 2020 Mar;18(3):612-619

Kugathasan S, Denson LA, Walters TD, ...Kellermayer R, Kappelman MD, Steiner S, Markowitz JF, Cho J, Xavier RJ, Huttenhower C, Aronow BJ, Gibson G, Hyams JS, Dubinsky MC. Prediction of complicated disease course for children newly diagnosed with Crohn's disease: a multicentre inception cohort study. Lancet. 2017. 29;389(10080):1710-1718.

Shiau H, Ihekweazu FD, Amin M, Fofanova T, Miloh T, Kellermayer R. Unique Inflammatory Bowel Disease Phenotype of Pediatric Primary Sclerosing Cholangitis: A Single-Center Study. J Pediatr Gastroenterol Nutr. 2017 Oct;65(4):404-409.

Fofanova TY, Petrosino JF, Kellermayer R. Microbiome-Epigenome Interactions and the Environmental Origins of Inflammatory Bowel Diseases. J Pediatr Gastroenterol Nutr. 2016;62:208-19.

Nagy-Szakal D., Mir SAV., Harris RA., Dowd SE., Yamada T., Lacorazza D., Tatevian N., Smith CW., de Zoeten EF., Klein J., Kellermayer R. Loss of omega-6 fatty acid induced pediatric obesity protects against acute murine colitis. FASEB J. 2015 Aug;29(8):3151-9.

I am a former DDC Pilot Awardee, currently serving as the Director of the Texas Children's Inflammatory Bowel Disease Program and as a physician-scientist with over 180 peer-reviewed publications (H-index 43). My clinical and research focuses on understanding the genetic, epigenetic, and developmental origins of inflammatory bowel diseases (IBD), with particular emphasis on the gut microbiome and advancing microbial therapeutics. My research integrates expertise in molecular biology, microbiology, genetics, and pediatrics to investigate pediatric GI diseases, with a particular focus on IBD and primary sclerosing cholangitis. Our lab's research efforts have encompassed clinical studies, murine models of IBD, human tissue collection and biobanking, genome-wide DNA methylation and gene expression profiling, pyrosequencing, real-time RT-PCR, and comprehensive genetic, epigenetic, and metagenomic analyses. We have developed extensive clinical expertise in the management of pediatric IBD and PSC-IBD, bridging translational research with clinical practice. I was recognized for my mentorship (Dept Pediatrics Mentorship Award) and have mentored more than 32 MD clinical postdoctoral fellows in pediatric GI most with productive publication records and many currently hold academic clinical faculty positions at major medical schools. T32 collaborations include Britton, Burrin, Savidge. Theme: Clinical and Translational.

Our laboratory approaches inflammatory bowel diseases from the perspective of the developmental origins hypothesis. This hypothesis postulates that at various stages of development critical changes in organismic structures can occur as a result of environmental influences. Such critical changes are then maintained throughout life and influence our susceptibility to common disorders, such as inflammatory bowel diseases (IBD), for example. IBD develops on the basis of an exaggerated immune response against the enteric microflora that is transmitted by the intestinal epithelium. One molecular process that has been recognized to potentially play a major role in the developmental origins of human diseases is DNA methylation. DNA methylation is the most stable epigenetic process and can respond dynamically to microbiota changes in the intestinal mucosa. Therefore, epigenetic and microbiome alterations may be intimately related in the mammalian gut. We are studying how nutritional and genetic changes can modify the intercalating network of the mucosal epigenome and microbiome in association with mammalian colitis. Our recent clinical and basic research focus is complex (fecal microbiota transplantation) and select bacteriotherapy to treat murine models of colitis and IBD. These studies should provide the basis for novel diagnostic, preventative and therapeutic measures for IBD.

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Microvascular Inflammation and Thrombosis in Acute and Chronic Models of Liver and Lung Injury

FONG WILSON LAM

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Department of Pediatrics, Pediatric Critical Care Medicine
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Recent Publications

Stayer K, Pathan S, Biswas A, Li H, Zhu Y, Lam FW, Marini J, Thevananther S. Exogenous arginine differentially regulates inflammatory cytokine and inducible nitric oxide synthase expression in macrophages. Immunohorizons. 2025 Jul 14;9(8). doi: 10.1093/immhor/vlaf028. PubMed PMID: 40694828; PubMed Central PMCID: PMC12282984.

Iqbal M, McLennan AL, Mukhamedshin A, Dinh MTP, Liu Q, Junco JJ, Mohan A, Srivaths PR, Rabin KR, Fogarty TP 3rd, Gifford SC, Shevkoplyas SS, Lam FW. Ultra-low extracorporeal volume microfluidic leukapheresis is safe and effective in a rat model. Nat Commun. 2025 Feb 24;16 (1):1930. doi: 10.1038/s41467-025-57003-5. PubMed PMID: 39994179; PubMed Central PMCID: PMC11850925.

Riley AF, Rose R, Denfield S, Thomas JA, Vogel AM, Coleman R, Lam FW. Assessment of echocardiographic interpretation of dual-lumen cannula during venovenous extracorporeal membrane oxygenation use for pediatric respiratory failure. Echocardiography. 2024 Jul;41(7):e15878. doi: 10.1111/echo.15878. PubMed PMID: 38979777.

Lam FW, Brown CA, Ronca SE. Recombinant Rod Domain of Vimentin Reduces SARS-CoV-2 Viral Replication by Blocking Spike Protein-ACE2 Interactions. Int J Mol Sci. 2024 Feb 20;25(5). doi: 10.3390/ijms25052477. PubMed PMID: 38473724; PubMed Central PMCID: PMC10931652.

Courson JA, Langlois KW, Lam FW. Intravital Microscopy to Study Platelet-Leukocyte-Endothelial Interactions in the Mouse Liver. J Vis Exp. 2022 Oct 6; (188). doi: 10.3791/64239. PubMed PMID: 36282718; PubMed Central PMCID: PMC9915146.

Shan Z, Li L, Atkins CL, Wang M, Wen Y, Jeong J, Moreno NF, Feng D, Gui X, Zhang N, Lee CG, Elias JA, Lee WM, Gao B, Lam FW, An Z, Ju C. Chitinase 3-like-1 contributes to acetaminophen-induced liver injury by promoting hepatic platelet recruitment. Elife. 2021 Jun 10;10. doi: 10.7554/eLife.68571. PubMed PMID: 34110284; PubMed Central PMCID: PMC8233036.

Lam FW, Brown CA, Valladolid C, Emebo DC, Palzkill TG, Cruz MA. The vimentin rod domain blocks P-selectin-P-selectin glycoprotein ligand 1 interactions to attenuate leukocyte adhesion to inflamed endothelium. PLoS One. 2020;15 (10):e0240164. doi: 10.1371/journal.pone.0240164. eCollection 2020. PubMed PMID: 33048962; PubMed Central PMCID: PMC7553327.

As a Pediatric Intensivist-Scientist, I care for children with critical illness as well as make biomedical discoveries that underly the pathophysiology of their disease, particularly inflammation and thrombosis, and its treatment. My overall career goal is to reduce the morbidity and mortality in critically ill patients by using and enhancing my clinical, educational, and research expertise.

In our laboratory, we investigate how cellular interactions within the microvasculature—particularly among neutrophils, platelets, and endothelial cells—drive inflammation and thrombosis in critical illness. Our work integrates molecular biology, biophysics, and bioengineering to uncover mechanisms of disease and develop novel therapeutic strategies.

Currently, our research spans three major areas:

Microfluidics-Based Apheresis Devices: In conjunction with collaborators from the University of Houston, we are designing and testing innovative microfluidic platforms for selective blood purification in vitro, in rodents, and in preclinical pig models. These devices aim to selectively separate blood components from circulation, offering new approaches to treat systemic inflammation and thrombosis, particularly in children.

Modulators of Inflammation and Thrombosis: We are developing biologics and small molecules that target key pathways in thromboinflammation and viral infection. Our work has identified extracellular vimentin and fibrin modulation as promising strategies to reduce leukocyte-endothelial interactions and microvascular thrombosis.

Pathways of Liver Injury and Fibrosis: We are exploring novel cellular and molecular mechanisms that contribute to liver injury and fibrotic remodeling, with a focus on how leukocytes and platelets affect liver outcomes.

Our team combines expertise in pediatric critical care, leukocyte and platelet biology, vascular biology, and bioengineering. We value curiosity, collaboration, and translational impact. Our laboratory has a history of collaboration throughout the Texas Medical Center and University of Houston. Post-doctoral fellows in our lab will have opportunities to lead interdisciplinary projects, publish high-impact research, and contribute to innovations that improve outcomes for children with life-threatening conditions.

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Biochemical Genetics of the Urea Cycle and Nitric Oxide Synthesis

Professor
Robert and Janice McNair Endowed Chair and Professor
Chairman, Department of Molecular and Human Genetics
Baylor College of Medicine

BRENDAN LEE

Recent Publications

Kho J, Polak U, Jiang MM, Odom JD, Hunter JV, Ali SM, Burrage LC, Nagamani SC, Pautler RG, Thompson HP, Urayama A, Jin Z, Lee B. Argininosuccinate lyase deficiency causes blood-brain barrier disruption via nitric oxide-mediated dysregulation of claudin expression. JCI Insight. 2023 Sep 8;8(17):e168475.

Stroup BM, Marom R, Li X, Hsu CW, Chang CY, Truong LD, Dawson B, Grafe I, Chen Y, Jiang MM, Lanza D, Green JR, Sun Q, Barrish JP, Ani S, Christiansen AE, Seavitt JR, Dickinson ME, Kheradmand F, Heaney JD, Lee B, Burrage LC. A global Slc7a7 knockout mouse model demonstrates characteristic phenotypes of human lysinuric protein intolerance. Hum Mol Genet. 2020 Aug 3;29(13):2171-2184.

Kho J, Tian X, Wong WT, Bertin T, Jiang MM, Chen S, Jin Z, Shchelochkov OA, Burrage LC, Reddy AK, Jiang H, Abo-Zahrah R, Ma S, Zhang P, Bissig KD, Kim JJ, Devaraj S, Rodney GG, Erez A, Bryan NS, Nagamani SCS, Lee BH. Argininosuccinate Lyase Deficiency Causes an Endothelial-Dependent Form of Hypertension. Am J Hum Genet. 2018. 2;103(2):276-287.

Burrage LC, Sun Q, Elsea SH, Jiang MM, Nagamani SC, Frankel AE, Stone E, Alters SE, Johnson DE, Rowlinson SW, Georgiou G; Members of Urea Cycle Disorders Consortium, Lee BH. Human recombinant arginase enzyme reduces plasma arginine in mouse models of arginase deficiency. Hum Mol Genet. 2015.15;24(22):6417-27.

The overall mission of my research program is to translate the study of structural birth defects and inborn errors of metabolism into a deeper understanding of development, disease mechanisms, and potential therapeutic strategies. My work spans from basic developmental biology to interventional clinical trials, with a long-standing focus on biochemical genetic disorders—particularly urea cycle disorders (UCDs)—as models for complex diseases involving nitric oxide dysregulation.

Our research has included the development of mouse models for UCDs, stable isotopic metabolic studies in patients, longitudinal observational studies, and both investigator-initiated and industry-sponsored clinical trials. Alongside these efforts, we have pursued novel treatments such as cell, protein, and viral gene therapies for UCDs, hyperbilirubinemia, and hemophilia, as well as investigations into immune responses to these therapies.

Clinically, our studies began with stable isotopic measurements to improve diagnosis of urea cycle disorders and to evaluate nitrogen source bioavailability (enteral vs. parenteral) in the urea cycle. These efforts expanded to exploring nitric oxide flux and how urea cycle enzymes, such as argininosuccinate lyase and arginase, regulate nitric oxide synthesis in conditions including liver fibrosis and necrotizing enterocolitis.

Over the past decade, I have directed multiple NIH-funded initiatives, including the BCM/TCH site for the UCD Rare Diseases Clinical Research Consortium, and currently serve as PI for the NIH Brittle Bone Disorders RDCRC, the NIH Undiagnosed Diseases Network Clinical Site at BCM, and the REJOIN consortium within the NIH HEAL initiative. My team has led over 20 clinical research protocols, supporting more than 1,000 inpatient and outpatient research visits, within the Lawrence Family Bone Disease Program of Texas—a collaboration among UT MD Anderson Cancer Center, UT Health, and BCM. Leveraging this infrastructure, I founded and direct the BCM Center for Skeletal Medicine and Biology, supported by advanced resources such as the MicroCT Imaging and Bone Histomorphometry Cores.

Training the next generation of scientists has been central to my mission. I co-founded the BCM Medical Research Pathway program, enabling medical students to spend a dedicated year on basic, translational, or clinical research. Over 25 years, I have mentored more than 46 postdoctoral fellows, 25 PhD students, 10 MD trainees, and 20 undergraduates, as well as numerous NIH career development awardees. I remain committed to diversifying the scientific workforce through initiatives such as HHMI EXROP, the NHGRI Diversity Action Plan, and the All of Us Evenings with Genetics Scholars program. Additionally, I serve as PI for the BCM Medical Genetics Training Grant, now in its 46th–50th years of continuous NIH funding, supporting ABMGG-track MD and MD/PhD training in medical genetics research.

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Viral Hepatitis and Cystic Fibrosis Liver Disease

Associate Professor
Department of Pediatrics
Gastroenterology, Hepatology and Nutrition
Director of Viral Hepatitis Clinic
Texas Children's Hospital

DANIEL H. LEUNG

Recent Publications

Gonzalez-Peralta RP, Wen JW, Hardikar W, Karnsakul WW, Whitworth S, Lin CH, Indolfi G, Rosenthal P, Balistreri W, Schwarz KB, Honegger JR, Zhang X, Svarovskaia EC, Suri V, Kersey K, Leung DH. Long-term efficacy and safety of sofosbuvir-based direct-acting antiviral regimens in paediatric patients with hepatitis C virus infection: an international registry study. Lancet Child Adolesc Health. 2025 ;9(4):248-254.

Ali S, Nisar A, Zhang A, Nagamani S, Aceves-Ewing NM, Rawls B, Quan T, Enns G, Goss J, Leung DH, Shneider BL, Jain S, Hazard FK, Schady D, Burrage LC. Prevalence of fibrosis in hepatic explants and biopsies from individuals with urea cycle disorders. Mol Genet Metab. 2025 Aug;145 (4):109175.

Ruan W, Cerminara DN, Galvan NTN, Fishman DS, Harpavat S, Hertel PM, Mysore KR, Tessier ME, Fuller K, Faraone M, Cotton RT, O'Mahony CA, Rana A, Goss JA, Leung DH. Sirolimus utilization in pediatric liver transplantation: A large high-volume quaternary center experience. J Pediatr Gastroenterol Nutr. 2025 Aug;81(2):167-176.

Freeman AJ, Sellers ZM, Mazariegos G, Kelly A, Saiman L, Mallory G, Ling SC, Narkewicz MR, Leung DH. A Multidisciplinary Approach to Pretransplant and Posttransplant Management of Cystic Fibrosis-Associated Liver Disease. Liver Transpl. 2019 Apr;25 (4):640-657.

Calvopina DA, Chatfield MD, Weis A, Coleman MA, Fernandez-Rojo MA, Noble C, Ramm LE, Leung DH, Lewindon PJ, Ramm GA. miRNA-Seq identifies a serum miRNA panel, which combined with APRI can detect and monitor liver disease in pediatric Cystic Fibrosis. Hepatology. 2018. Jul 16. doi: 10.1002/hep.30156.

Dr. Leung oversees the Viral Hepatitis Clinic at Texas Children's Hospital which features a robust clinical program, managing and counseling nearly 200 established patients with hepatitis B and C. 7 clinical trials in the treatment of hepatitis are conducted from the clinic with new protocols studying the safety and efficacy of direct acting antivirals for children infected with HCV. Other research protocols within the hepatitis clinic include the study of sheerwave elastography, serum biomarkers, and simple biomarkers.

Dr. Leung's research spans disciplines within hepatology and gastroenterology such as cystic fibrosis (CF), the intestinal microbiome, and nutritional outcomes. Dr. Leung currently has funding from the NIH, CF Foundation, CF Therapeutics Development Network, and Texas Children's Hospital Pediatric Pilot Award. His efforts as site PI of the NIH funded CF Liver Disease Network has led to BCM being recognized as the largest enrolling site in the country. For the last 5 years, Dr. Leung has served as Lead Co-Principal Investigator of the Cystic Fibrosis Foundation (CFF) funded Multi-center Infant Nutrition Observational Study in Children identified by Newborn screen (BONUS) study in tandem with Dr. Drucy Borowitz, a matriarch in the field of pediatric cystic fibrosis. His unique skill set in gastrointestinal and hepatology care within cystic fibrosis was recognized by the CFF and he was awarded a Developing Innovative GastroEnterology Specialty Training (DIGEST) Program grant through 2017 to be further mentored by pioneers within the CF GI and Liver field. He is currently a co-investigator for a NIH R01 funded project in the CF Infant microbiome and sits on the Cystic Fibrosis Foundation and Therapeutic Development Network Clinical Research Award and Grants Committee, which has awarded more than $2 million in annual grant funding.

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YUYING LIU

Probiotics and Gut Microbiota in Immune Regulation and Autoimmune Disorders

Associate Professor
Pediatric Gastroenterology
McGovern Medical School
The University of Texas Health Science Center at Houston

Recent Publications

Okeugo B, Armbrister SA, Daniel RC, Saleh ZM, Wang J, Giorgberidze S, Rhoads JM, Liu Y Reduced autoimmunity associated with deletion of host CD73. Immunohorizons. 2025 Jan 23;9(1):vlae004.

Nessim Kostandy E, Suh JH, Tian X, Okeugo B, Rubin E, Shirai S, Luo M, Taylor CM, Kim KH, Rhoads JM, Liu Y. Probiotic Limosilactobacillus reuteri DSM 17938 Changes Foxp3 Deficiency-Induced Dyslipidemia and Chronic Hepatitis in Mice. Nutrients. 2024 Feb 12;16(4):511.

Liu Y, Armbrister SA, Okeugo B, Mills TW, Daniel RC, Oh JH, van Pijkeren JP, Park ES, Saleh ZM, Lahiri S, Roos S, Rhoads J. Probiotic-Derived Ecto-5'-Nucleotidase Produces Anti-Inflammatory Adenosine Metabolites in Treg-Deficient Scurfy Mice. Probiotics Antimicrob Proteins. 2023 Aug;15 (4):1001-1013.

Liu Y, Freeborn J, Armbrister SA, Tran DQ, Rhoads JM. Treg-associated monogenic autoimmune disorders and gut microbial dysbiosis. Pediatr Res. 2022 Jan;91(1):35-43.

Lactobacillus reuteri effects on maternal separation stress in newborn mice. Park ES, Freeborn J, Venna VR, Roos S, Rhoads JM, Liu Y.Pediatr Res. 2021 Nov;90(5):980-988.

I am an Associate Professor of Pediatric Gastroenterology at UTHealth with a broad background in molecular and cell biology, protein engineering, and immunology, with specific expertise in mucosal immunology. I established and currently direct the Pediatric GI Research Lab, where my goal is to identify the role of probiotics and other gut microbial factors in regulating immune-related disorders, including necrotizing enterocolitis (NEC) and autoimmune disorders involving regulatory T (Treg), TH1, and TH17 cells, such as primary immune deficiency (IPEX syndrome, IPEX-like syndrome), inflammatory bowel disease (IBD), and multiple sclerosis (MS).

My research specifically explores the immune-modulatory mechanisms of probiotics, probiotic-modulated gut microbiota, and microbiota-associated metabolites. I discovered the central role of tolerogenic dendritic cells in educating Th cell differentiation into Tregs via toll-like receptor 2 in the NEC model, as well as a novel mechanism in which probiotics alter the adenosine metabolite inosine to inhibit T cell differentiation through the adenosine receptor 2A (A2A).

My current NIH-funded research focuses on four main themes: (1) the effects of probiotics on gut-brain signals in the neonatal stress model, (2) human breast milk factors and their impact on probiotic function in healthy newborn mice and autoimmune disorders, (3) probiotic-educated T cell function, and (4) maternal-infant transfer of microbially modified immunity. I am also involved in clinical research projects, including a completed infantile colic probiotic study and an ongoing probiotic autism study. My lab processes patient samples, analyzes circulating immune cells, plasma and fecal inflammatory biomarkers, gut permeability markers, and stool DNA for microbiota analysis, and monitors probiotic product quality by measuring CFUs and using multiple omics approaches to evaluate whether dysbiosis and abnormal circulating metabolites can be improved by probiotics.

In addition to leading these research efforts, I actively mentor and support clinical fellows in their research training, assisting with IRB protocol preparation, study design, sample processing, assay development, data analysis, and meeting presentations. I have also collaborated extensively through T32 programs with colleagues including Britton, Burrin, and Rhoads in the areas of Immunology and Microbiology.

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ANDREA A. HILL MCALESTER

Dietary Regulation of Immune Tissue Repair Functions in Intestinal Health and Disease

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Department of Pathology and Immunology
TMC DDC Enrichment Program Committee, GCC Immunology Group
Immunometabolism, Mucosal Immunology, Microbiology
Baylor College of Medicine

Recent Publications

Gil AM, Zegarra-Ruiz DF, Wu WJ, Norwood K, Assie A, Samuel BS, Major AM, Diehl GE, McAlester AAH. Dietary lipids induce PPARd and BCL6 to repress macrophage IL-23 induction after intestinal injury and LPS exposure. Sci Rep. 2025 Jul 27;15 (1):27344. doi: 10.1038/s41598-025-12448-y. PMID: 40717126;

Andrea A. Hill, Myunghoo Kim, Daniel F. Zegarra-Ruiz, Lin-Chun Chang, Kendra Norwood, Adrien Assie, Wan-Jung Wu, Michael C. Renfroe, Hyo Won Song, Angela M. Major, Buck S. Samuel, Joseph M. Hyser, Randy S. Longman, and Gretchen E. Diehl. Acute high-fat diet impairs macrophage-supported intestinal damage resolution. Authorship note: Andrea A. Hill (McAlester) and Myunghoo Kim are co-first authors. Andrea A. Hill (McAlester) and Gretchen E. Diehl are co-senior authors. JCI Insight. 2023 Feb 8;8(3). doi:10.1172/jci.insight.164489.

Myunghoo Kim*, Carolina Galan*, Andrea A. Hill*, Wan-Jung Wu, Hannah Fehlner-Peach, Deborah Schady, Matthew L. Bettini, Kenneth W. Simpson, Randy S. Longman, Dan R. Littman, Gretchen E. Diehl. Critical role for the microbiota in CX3CR1+ intestinal mononuclear phagocyte regulation of intestinal T cell responses. Immunity. *Authors contributed equally to this work. 2018 Jul 17;49 (1):151-163.e5.

Andrea A. Hill and Emily Anderson-Baucum, Corey D.Webb, Arion Kennedy and Alyssa Hasty. Activation of NF-κB drives the enhanced survival of adipose tissue macrophages in an obesogenic environment. Molecular Metabolism. 2015. 2015 Oct;4(10):665-77.

Our research is supported by an NIDDK Career Development Award (K01-DK 121934), DDC Pilot Award, CDMRP DOD Research Discovery Award. The long-term goals of our research are to elucidate the direct influence of dietary lipids on reparative intestinal immune and intestinal epithelial cell responses to injury and to develop novel therapeutics targeting these mechanisms to aid in treating diet-associated inflammatory diseases. The overall themes of the research program are: (1) Defining the direct influence of dietary composition on intestinal immune and epithelial cell reparative responses in intestinal disease. (2) Identifying the reparative factors and molecular pathways affected by diet. (3) Developing dietary interventions and novel therapeutics to support and sustain the healing of the intestinal mucosal barrier. (4) Create novel inflammation-modulating biopolymer therapeutics and identify new therapeutic targets that facilitate intestinal healing.

We are currently investigating the direct impact of diet on intestinal epithelial cell (IEC) repair responses and how it affects communication between intestinal epithelial cells and immune cells, which is crucial for proper intestinal healing. Our recent research has shown how specific lipid components directly affect intestinal immune reparative responses, including cytokine responses to microbes in the injured intestine. The lipid transport and signaling functions of the lipid receptor CD36 mediate these effects. The transport of lipids inside immune cells, especially macrophages, by CD36 suppresses antimicrobial reparative cytokines, IL-23 and IL-22, which are needed to promote IEC repair through activation of a transcriptional repressor complex composed of PPARd and BCL6. An ongoing research project in the lab aims to understand further how CD36, PPARd, and BCL6 regulate macrophage tissue repair functions using transgenic animal models, pharmacological methods, and in vitro exposure of macrophages to lipids and microbial stimuli. These studies will help identify factors and molecular pathways affected by dietary lipids that control macrophage antimicrobial reparative responses to intestinal damage.

We are also interested in understanding how specific lipids and complex lipid compositions influence the dynamic communication between the immune system and tissue cells, which is essential for proper wound healing. In collaboration with our BCM GEMS core, we have developed wound healing assays using a human-macrophage and enteroid-derived epithelial monolayer co-culture system, providing us with a valuable human-relevant tool to study the dietary effects on intestinal damage repair. This high-throughput system enables us to directly assess the impact of individual lipids and their combinations on extracellular and intracellular factors and pathways that regulate macrophage reparative functions and signaling, which directly and indirectly drive IEC wound healing responses. Using this system, we aim to define and create a network of molecular and metabolic pathways modulated by dietary lipids that intersect with macrophage reparative and lipid metabolism functions. This will be achieved through bulk sequencing and metabolomics of in vitro human- and mouse-derived macrophages exposed to multi-dietary lipids and damage signals (e.g., LPS or apoptotic cells). Insights from this project will lead to innovative nutritional interventions that support immune tissue repair responses, helping to prevent or reverse many chronic inflammatory diseases.

Studies also aim to develop inflammation-modulating biopolymer therapeutics (IMT) to constrain tissue-damaging cytokine signaling and promote reparative immune responses and intestinal damage repair. By modulating the physicochemical properties of the polymers, specifically their composition and surface charge, we can alter the cytokine milieu in the damaged intestine in a manner that supports proper tissue healing. Ultimately, this research has the potential to significantly improve the quality of life for patients with gastrointestinal and other inflammatory diseases where modulation of immune function could be beneficial.

Dr. McAlester is an active member of the Texas Medical Center DDC Enrichment Program Committee and the Gulf Coast Consortia Immunology Group.

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Regeneration, Metaplasia, and Tumorigenesis in the GI Tract

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Jones Building
Section Chief of Research, Vice Chief
Section of Gastroenterology and Hepatology
Baylor College of Medicine

JASON C. MILLS

Recent Publications

Brown JW, Lin X, Nicolazzi GA, Liu X, Nguyen T, Radyk MD, Burclaff J, Mills JC. Cathartocytosis: Jettisoning of cellular material during reprogramming of differentiated cells. Cell Rep. 2025 Jul 29;44(8):116070.

Jin RU, Xu Y, Lih TS, Huang YZ, Nittolo TM, Sells BE, Dres OM, Wang JS, Li QK, Zhang H, Mills JC. SOX2 regulates foregut squamous epithelial homeostasis and is lost during Barrett's esophagus development. J Clin Invest. 2025 Jun 19:e190374.

Cho CJ, Nguyen T, Rougeau AK, Huang YZ, To S, Lin X, Thalalla Gamage S, Meier JL, Mills JC. Inhibition of Ribosome Biogenesis In Vivo Causes p53-Dependent Death and p53-Independent Dysfunction. Cell Mol Gastroenterol Hepatol. 2025 Mar 11;19(7):101496.

Fashemi BE, Rougeau AK, Salazar AM, Bark SJ, Chappidi R, Brown JW, Cho CJ, Mills JC*, Mysorekar IU*. IFRD1 is required for maintenance of bladder epithelial homeostasis. iScience. 2024 Oct 28;27(12):111282.

Adkins-Threats M, Arimura S, Huang YZ, Divenko M, To S, Mao H, Zeng Y, Hwang JY, Burclaff JR, Jain S, Mills JC. Metabolic regulator ERRγ governs gastric stem cell differentiation into acid-secreting parietal cells. Cell Stem Cell. 2024 Jun 6;31(6):886-903.e8.

Miao ZF*, Sun JX, Huang XZ, Bai S, Pang MJ, Li JY, Chen HY, Tong QY, Ye SY, Wang XY, Hu XH, Li JY, Zou JW, Xu W, Yang JH, Lu X, Mills JC*, Wang ZN*. Metaplastic regeneration in the mouse stomach requires a reactive oxygen species pathway. Dev Cell. 2024 May 6;59(9):1175-1191.e7.

Deans-Fielder K, Wu T, Nguyen T, To S, Huang YZ, Bark SJ, Mills JC*, Shroyer NF*. Mechanisms Driving Fasting-Induced Protection from Genotoxic Injury in the Small Intestine. Am J Physiol Gastrointest Liver Physiol. 2024 May 1;326(5):G504-G524.

As a human pathologist and cell and developmental biologist, I lead my laboratory's investigations into how cells in tissue adapt to various stressors, particularly those that cause cells to change their functional identity in the GI tract. We are interested in how gastric epithelial cells in adults form from the stem cell and how this process goes askew during metaplasia. We are also interested in how mature, differentiated cells can be recruited back into the cell cycle during tissue regeneration and in tumorigenesis. We have proposed that there is a specific cellular-molecular program with three distinct stages that mature cells can use to return to a regenerative state in response to injury, which we call *paligenosis*. Like apoptosis or mitosis, paligenosis is conserved across cell types and organisms. Although paligenosis drives tissue repair, it can also increase risk for mutation accumulation by allowing old cells to proliferate, furthering chronic, precancerous conditions in stomach, pancreas, and esophagus.

Approaches in our lab range from structural studies at the protein level to clinical trials and translational efforts that apply paligenosis findings toward better prevention or treatment of tumors in the GI tract. Our lab also has vast experience in bioinformatics and multi-omics approaches, including a full-time bioinformatician on staff, and we have published our own in-house expression analysis software/algorithms. Our work has been most focused on stomach, but we also study intestines, pancreas and esophagus, and we have established mouse, organoid, and human tissue models for the study of paligenosis.

Currently in our lab, we have multiple NIH-funded projects ongoing. One project is investigating the hypothesis that the molecular mechanisms that stem cells use to differentiate into acid-pumping parietal cells in the gastric epithelium are driven by the nuclear hormone transcription factor ERRγ. This investigation relies on immunostaining approaches, electron microscopy and confocal microscopy imaging, ChIP-seq, and mass spectrometry techniques. Another project is investigating the hypothesis that early events of paligenosis are driven by the PERK kinase wing of the integrated stress response and the dynamic regulation and autophagy of rough endoplasmic reticulum. This investigation uses both established and customized mouse models in combination with specific inhibitors and an RNA-sequencing multi-omics approach. And another project in collaboration with the Mysorekar Lab is investigating the hypothesis that aging phenotypes driven by IFRD1 disrupt ribosome dynamics during protein translation, modification, and trafficking between the endoplasmic reticulum and Golgi, leading to bladder dysfunctions such as urinary tract infections. This study also investigates the effects of various pharmacological interventions, and uses techniques such as qRT-PCR, western blotting, and immunostaining in combination with specialized mouse and organoid models.

I currently serve as Vice Chief and Chief of Research in the adult GI division, and also act as co-director of the Texas Medical Center Digestive Diseases Center, and director of its Tissue Analysis and Molecular Imaging sub-core. This center is one of 17 Digestive Diseases Centers supported by the NIH-NIDDK P30 mechanism (P30DK056338) and fosters multidisciplinary basic and translational science by bringing together 103 center members. I am also serving as co-Associate Director for Education at the Daniel L. Duncan Comprehensive Cancer Center. In recognition for my contributions to medical research, I was inducted into the American Association of Physicians in 2022.

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Immune Regulation and Infections in Chronic Liver Diseases and Transplantation

Assistant Professor
Department of Pediatrics
Gastroenterology, Hepatology and Nutrition
Baylor College of Medicine

KRUPA MYSORE, M.D., M.S.

Recent Publications

Mysore KR, Cheng K, Suri LA, Fawaz R, Mavis AM, Kogan-Liberman D, Mohammad S, Taylor SA. Recent advances in the management of pediatric cholestatic liver diseases. J Pediatr Gastroenterol Nutr. 2025 Apr;80(4):549-558.

Dike PN, Schady D, Himes R, Goss JA, Guffey D, Cerminara D, Mysore KR. Incidence and risk factors for chronic rejection in pediatric liver transplantation. Liver Transpl. 2024 Sep 24. doi: 10.1097/LVT.0000000000000488. .

Mysore KR, Kannanganat S, Schraw JM, Lupo PJ, Goss JA, Setchell KDR, Li XC, Kheradmand F, Shneider BL. Innate immune cell dysfunction and systemic inflammation in children with chronic liver diseases undergoing transplantation. Am J Transplant. 2023 Jan;23(1):26-36. doi: 10.1016/j.ajt.2022.09.004.

Mysore KR, Phan TL, Himes RW, Schady D, Eldin KW, Prusty BP, Munoz FM. Human Herpesvirus 6 Infection in Pediatric Liver Transplantation: Single-Center Study of Incidence, Outcomes, and Management. J Pediatric Infect Dis Soc. 2021 Jan 25;piaa166. doi: 10.1093/jpids/piaa166.

Mysore KR, Ghobrial RM, Kannanganat S, Minze LJ, Graviss EA, Nguyen DT, Perez KK, Li XC. Longitudinal Assessment of T Cell Inhibitory Receptors in Liver Transplant Recipients and their association with Post-transplant Infections. Am J Transplant. 2018 Feb;18(2):351-363. doi: 10.1111/ajt.14546.

My clinical and research interests are in pediatric liver diseases and transplantation. I actively collaborate with divisions of Immunology and Infectious diseases at Texas Children's Hospital. There are a large number of infectious complications in patients with chronic liver diseases (CLD) and transplantation. We lack the understanding of the immunological deficits predisposing to these infections. My work focuses on basic and translational projects designed to establish how immune dysregulation in CLD and transplantation alters hosts susceptibility to infection. We have used the human blood samples and tissues in the laboratory, using powerful techniques such as flow cytometry, ELISPOT assays, PCR etc. to analyze immune cells. The laboratory data is correlated with clinical patient data which address clinically-relevant problems in pediatric hepatology and transplantation. My Master's in Clinical Investigation with CSTP has helped me expand translational research studies in the laboratory and facilitated building a biorepository of specimens from children and adolescents who have received liver transplants; a vital resource to investigate biomarkers in pediatric transplantation.

Current projects in the laboratory seek to identify the cellular and molecular mechanism that lead to T cell and dendritic cell dysfunction associated with pediatric CLD in the immediate pre-transplant phase. Our recent studies in adult CLD show that patients with increased expression of T cell inhibitory markers in circulation in the pre-transplant period have increased susceptibility to infections in the immediate post-transplant phase. We are currently identifying underlying mechanisms in liver diseases which alter the T cell co-signaling pathways while simultaneously establishing role of these inhibitory pathways in pediatric liver diseases. Alterations in T cell -dendritic cell interactions and antigen presentation in CLD are being studied as well.

Studies also are aimed at establishing role of immune cells in vaccine response in transplant patients. We specifically are aiming to identify differences in vaccine response across transplant patients and the underlying immune mechanisms for these variabilities. Other active areas of her clinical research include work on implementing ABO-incompatible liver transplant management protocol and evaluating the incidence of Human Herpes Virus (HHV-6) virus in pediatric liver transplantation at Texas Children's Hospital. Representative publications from recent work are listed below.

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Malnutrition and Effects on the Liver, Gastrointestinal Tract, and Gut Microbiome

Associate Professor
Department of Pediatrics
Gastroenterology, Hepatology and Nutrition
Baylor College of Medicine

GEOFFREY PREIDIS

Recent Publications

Lynch LE, Lahowetz R, Maresso C, Terwilliger A, Pizzini J, Melendez Hebib V, Britton RA, Maresso AW, Preidis GA. Present and future of microbiome-targeting therapeutics. J Clin Invest. 2025 Jun 2;135 (11):e184323.

Wan X, Soni KG, Choi JM, Jung SY, Conner ME, Preidis GA. Inhibition of SREBP-1c rescues hepatic CYP7B1 expression and bile acid synthesis in malnourished mice. Am J Physiol Gastrointest Liver Physiol. 2025 Jul 1;329(1):G232-G243.

Morgan RL, Preidis GA, Kashyap PC, Weizman AV, Sadeghirad B; Probiotics Reduce Mortality and Morbidity in Preterm, Low-Birth-Weight Infants: A Systematic Review and Network Meta-analysis of Randomized Trials. McMaster Probiotic, Prebiotic, and Synbiotic Work Group. Gastroenterology. 2020 Jun 24:S0016-5085(20)34849-6.

Preidis GA, Weizman AV, Kashyap PC, Sadeghirad B, Morgan RL. AGA Technical Review on the Role of Probiotics in the Management of Gastrointestinal Disorders. Gastroenterology. 2020 Jun 9:S0016-5085 (20)34732-6.

Shin AS, Preidis GA, Shulman RJ, Kashyap PC. Gut microbiome in adult and pediatric functional gastrointestinal diseases. Clin Gastroenterol Hepatol 2019. Jan;17(2):256-274.

Preidis GA, Kim KH, Moore DD. Nutrient sensing nuclear receptors PPAR-alpha and FXR control liver energy balance. J Clin Invest 2017;127:1193-1201.

I am an Associate Professor in the Department of Pediatrics at Baylor College of Medicine and a former postdoctoral trainee on this T32. The skills and data I generated as a T32 Fellow supported my successful application for an NIDDK Career Development Award (K08) and a DDC Pilot Feasibility Award, which led to the development of an independently funded research program supported by multiple NIH grants, including R01 (DK133301, DK142021) and U01 (DK112194) awards. I also serve as a new member of the T32 Executive Committee.

The long-term goals of my laboratory are to define mechanisms through which early-life undernutrition alters metabolism and to determine how undernutrition influences intestinal and liver function. Our work examines how malnutrition affects hepatic secretion, transcriptional programming, gastrointestinal motility, and host–microbiome interactions to impact growth. This research is directly relevant to children suffering from nutritional deficiencies due to medical and socioeconomic factors, including protein–energy undernutrition in the developing world and low birthweight newborns in neonatal intensive care units.

Mentorship is a major focus of my career. I have served as the primary research mentor for four T32 trainees (MD clinical fellows), all of whom have published extensively and now hold academic faculty positions at institutions including the University of Tennessee Health Science Center, Stanford Medical School, George Washington University School of Medicine, and Baylor College of Medicine. I also serve as the primary research mentor for six postdoctoral fellows and two PhD students and am actively involved in training as Director of the Enrichment Program of our Digestive Diseases Center and Associate Director of the BCM Medical Scientist Training Program.

My T32 collaborations include Burrin, El-Serag, Estes, Hair, Mills, Ramani, Shen, Shneider, Speer, Tessier, and Wooten-Kee, with a research focus on Mucosal Biology. Over the past ten years, four T32 trainees have worked in my laboratory.

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Probiotic Mechanism of Action in GI Diseases

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Division Head of Pediatric Gastroenterology
Fellowship Program Director
University of Texas Health Sciences Center at Houston

J. MARC RHOADS

Recent Publications

Liu Y, Hoang TK, Park ES, Freeborn J, Okeugo B, Tran DQ, Rhoads. Probiotic-educated Tregs are more potent than naïve Tregs for immune tolerance in stressed new-born mice. JM.Benef Microbes. 2023 Mar 14;14(1):73-84.

Nessim Kostandy E, Suh JH, Tian X, Okeugo B, Rubin E, Shirai S, Luo M, Taylor CM, Kim KH, Rhoads JM, Liu Y. Probiotic Limosilactobacillus reuteri DSM 17938 Changes Foxp3 Deficiency-Induced Dyslipidemia and Chronic Hepatitis in Mice. Nutrients. 2024 Feb 12;16(4):511.

Hoang TK, Freeborn J, Wang T, Mai T, He B, Park S, Tran DQ, Roos S, Rhoads JM, Liu Y. Human Breast Milk Promotes the Immunomodulatory Function of Probiotic Lactobacillus reuteri DSM 17938 in the Neonatal Rat Intestine. J Probiotics Health. 2019;7 (1):210. doi: 10.35248/2329-8901.19.7.210.

Fatheree NY, Liu Y, Taylor CM, Hoang TK, Cai C, Rahbar MH, Hessabi M, Ferris M, McMurtry V, Wong C, Vu T, Dancsak T, Wang T, Gleason W, Bandla V, Navarro F, Tran DQ, Rhoads JM. Lactobacillus reuteri for Infants with Colic: A Double-Blind, Placebo-Controlled, Randomized Clinical Trial. J Pediatr. 2017. 191:170-178.

I serve as Assistant Director, of our Texas Medical Center DDC. One major research focus in our lab is on mucosal immune responses, aiming to determine the mechanisms by which probiotics reduce GI inflammation in rodent models and in cultured T cells and dendritic cells. I published several mechanistic investigations demonstrating beneficial effects of Limosilactobacillus reuteri in mice exposed to necrotizing enterocolitis. Since then, we have focused on a type of autoimmunity seen in children with very early onset inflammatory bowel disease called IPEX syndrome. I found L. reuteri in vitro has a major inhibitory effect on the maturation of all 3 classes of proinflammatory T-cells (Th1, Th2, and Th17). More recently, he our lab group headed by Yuying Liu have identified major shifts in several metabolites that are mechanistically linked to the anti-inflammatory effects of probiotic(s), specifically the adenosine-A2A receptor pathway. Currently, my group is completing analysis of an R01-funded study (HD095158) of a combination probiotic (BB-12 and LGG) in 100 children with autism spectrum disorder with GI symptoms, using validated behavioral questionnaires to determine if improving GI symptoms can aid behavior management. We are using multiple omics to understand if dysbiosis and abnormal circulating metabolites can be ameliorated by this probiotic. Other projects include how probiotic feeding of pregnant dams results in T-cell changes in the breast milk which may prevent inflammation. T32 collaborations include Britton, Burrin, Luna, Horvath, Estes, Mills, and Preidis. Theme: Immunology and Microbiology

Since 2007, our basic research has been focusing on mechanisms of neonatal necrotizing enterocolitis (NEC) which is the leading cause of gastrointestinal morbidity in premature infants. We have found the critical roles of Toll-like receptor (TLR) and TLF-signaling regulators and mediators, as well as an imbalance of Foxp3+ regulatory T (Treg) and effort memory T (Tem) cell, which promote the development of NEC. In addition, we have been studying the effects of probiotic *Lactobacillus reuteri* (LR) strains on the development of NEC. Our studies showed the LR reduced the incidence and severity of NEC in animal models via modulation of TLR4 and NFKB signaling in the intestine. LR also causes aredistribution to the Tcell subsets in the intestinal mucosa.

Recently, we observed the LR markedly prolong the survival of Foxp3-deficient scurfy (sf) mice, which may provide evidence for using probiolic LR to treat patients with IPEX syndrome (immune dysregulation, polyendocrinopathy, enteropthy, X-linked) or patients with other autoimmune diseases. We are investigating changes in the microbiota produced by this disease and severe changes in the fecal and plasma metabolic profile. The changes seen are markedly modified by oral L. reuteri treatment.

Drs. Liu & Rhoads also are actively investigating the mechanisms by which LR regulates intestinal inflammation invitro and animal models. The project is designed to investigate how mucosa dendritic cells and Tcells (TH1, TH17, and Tregs) respond to probiotics, and which cells control gastrointestinal inflammation of the newborn. Moreover, we will show if LR treatment increases fecal microbial richness and diversity and alters their metabolic products, in order to understand local systemic beneficial effects. Ultimately, we hope to provide novel insight into the mechanisms of how probiotics regulate neonatal intestinal inflammation.

In our clinical research on colic, which we showed to be an inflammatory condition in babies who cry excessively, we were first to demonstrate elevated fecal calprotectin in this population, indicating that there is an intestinal inflammation in these babies. We are analyzing the microbiota and fecal calprotectin in babies treated with *L. reuteri versus* placebo in a “road to discovery” trial.

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Multidisciplinary Studies on Enteric Infections and Vaccines

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RAMANI SASIREKHA

Recent Publications

Patil K, Ayyar BV, Hayes NM, Neill FH, Bode L, Estes MK, Atmar RL, Ramani S. 2'-Fucosyllactose inhibits human norovirus replication in human intestinal enteroids. J Virol. 2025.

Adeniyi-Ipadeola GO, Hankins JD, Kambal A, Zeng X-L, Patil K, Poplaski V, Bomidi C, Nguyen-Phuc H, Grimm SL, Coarfa C, Stossi F, Crawford SE, Blutt SE, Speer AL, Estes MK, Ramani S. Infant and adult human intestinal enteroids are morphologically and functionally distinct. mBio. 2024.

Atmar RL, Neill FH, Hayes NM, Opekun AR, Graham DY, Estes MK, Ramani S. Lack of Detection of Norwalk Virus in Saliva Samples From a Controlled Human Infection Model. Open Forum Infect Dis. 2024.

Ngo VL, Wang Y, Wang Y, Shi Z, Britton R, Zou J, Ramani S, Jiang B, Gewirtz AT. Select Gut Microbiota Impede Rotavirus Vaccine Efficacy. Cell Mol Gastroenterol Hepatol. 2024.

Our research program involves multidisciplinary studies on enteric infections and vaccines with a focus on rotavirus and norovirus, the two leading causes of viral gastroenteritis worldwide. Our primary goals are to understand host factors that contribute to disease, and to identify mechanisms to improve responses to enteric infectious agents and vaccines so that we can address fundamentally important questions of population relevance on host-virus/vaccine interactions. Research in the Ramani Lab can be categorized into 3 major areas outlines below. The studies involve a combination of laboratory assays using organoid models and population studies and thus take a complete bench-to-bedside approach to infectious diseases.

Rotavirus Vaccine Research Program: The primary area of research focuses on the underperformance of live, attenuated rotavirus vaccines in low- and middle-income countries. Building on a long-term interest in identifying correlates of protection from infection and vaccination and what mediates vaccine failure, our overall goal is to develop interventions to improve infant health in these populations. Two ongling R01 grants are aimed at mechanistically understanding how the enteric virome and the intestinal microbiome impact rotavirus vaccine replication. On the translational perspective, new grants from the Gates Foundation are enabling efforts to develop novel interventions to improve current oral rotavirus vaccines and support the development of the next generation rotavirus vaccines.

Norovirus Research Program: Studies on human norovirus range from efforts to understand the mechanisms of norovirus-induced gastroenteritis to using genomic tools to understand virus evolution and host responses. These collaborative studies with Dr. Atmar's lab are supported by a NIH program project grant and a Genomic Centers for Infectious Diseases U19 grant. Additionally, the Atmar and Ramani Labs together provide laboratory support for public health surveillance for rotavirus and norovirus in the US Pediatric Population as part of a CDC-funded U01 grant awarded to investigators at Texas Children's Hospital.

Advancing Infant Organoid Models for Gastrointestinal Diseases and Vaccines: Key to our research program is the use of state-of-the-art human intestinal organoid cultures. In collaboration with the DDC Gastrointestinal Experimental Model Systems Core, our lab played a lead role in the establishment and characterization of infant intestinal organoids being used extensively by investigators within and outside the Texas Medical Center to study GI infectious and non-infectious diseases. With support from the Gates Foundation, our lab is also working on advancing organoid models from children with environmental enteric dysfunction.

Dr. Ramani is a former DDC Pilot Feasibility Awardee. She is active in the Texas Medical Center DDC Enrichment Program Committee. T32 collaborations include Britton, Burrin, Estes, Hair, Hyser, Speer, Vonderohe. Theme: Immunology and Microbiology

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Emerging Concepts in Gut Microbiome - Enteric Nervous System Function and Disease

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Texas Children's Microbiome Center
Department Pathology and Immunology
Baylor College of Medicine

TOR SAVIDGE

Recent Publications

Wu Q, Badu S, So SY, Treangen TJ, Savidge TC. The pan-microbiome profiling system Taxa4Meta identifies clinical dysbiotic features and classifies diarrheal disease. J Clin Invest. 2023 Nov 14:e170859. doi: 10.1172/JCI170859.

Wu Q, Boonma P, Badu S, Yalcinkaya N, So SY, Garey KW, Williams K, Arnold LE, Shulman RJ, Kellermayer R, Savidge TC. Donor-recipient specificity and age-dependency in fecal microbiota therapy and probiotic resolution of gastrointestinal symptoms. NPJ Biofilms Microbiomes. 2023 Aug 3;9 (1):54. doi: 10.1038/s41522-023-00421-4.

Aguirre AM, Yalcinkaya N, Wu Q, Swennes A, Tessier ME, Roberts P, Miyajima F, Savidge T, Sorg JA. Bile acid-independent protection against Clostridioides difficile infection. PLoS Pathog. 2021 Oct 19;17(10):e1010015. doi: 10.1371/journal.ppat.1010015.

Savidge TC, Urvil P, Oezguen N, Ali K, Choudhury A, Acharya V, Pinchuk I, Torres AG, English RD, Wiktorowicz JE, Loeffelholz M, Kumar R, Shi L, Nie W, Braun W, Herman B, Hausladen A, Feng H, Stamler JS, Pothoulakis C. Host S-nitrosylation inhibits clostridial small molecule-activated glucosylating toxins. Nat Med. 2011 17:1136-41 (2011).

I currently serve as Professor in the Department Pathology and Immunology at Baylor College of Medicine. I am also a Director of Texas Children's Microbiome Center and I am Principal Investigator of an independently NIH funded research laboratory focusing on neuro-immune-microbe interactions. I am also a Full Member of the NIDDK-funded Texas Medical Center Digestive Diseases Center and regularly review faculty evaluations and serve on several educational committees for the College. I am an international authority on C. difficile infections and microbial-mucosal interactions in enteric disease, with authored publications in Cell, Science, Nature Medicine, PNAS, Journal of Clinical Investigation and Gastroenterology. I served as Editor-In-Chief for a volume in Methods in Microbiology and currently serve on the editorial boards for the American Journal of Physiology (GI and Hepatology) and Gut Microbes. I served as an inaugural Chair for the Microbiome and Microbial Gastrointestinal Diseases Section for the Institute of the American Gastroenterology Association and played an active role in organizing and reviewing scientific materials presented at Digestive Diseases Week. I currently serve on the scientific advisory board for Texas Children Hospital and have provided scientific recommendations for the NASA Human Research Roadmap for the 2035 Mission to Mars. I regularly serve on related NIH study sections and as Principal Investigator currently manage several grants funded by NIH. I have supervised numerous postdoctoral workers and graduate students, several of whom have now successfully taken up faculty and distinguished industry positions.

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Epigenetic Regulation of GI Development and Disease

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LANLAN SHEN

Recent Publications

Chen F, Zhang Y, Li W, Shen L, Creighton CJ. Global DNA methylation differences involving germline structural variation impact the transcriptome of pediatric brain tumors. Nature Communications. 2025 May 21;16(1):4713.

Li J, Riggins K, Yang L, Chen C, Castro P, Alfarkh W, Zarrin-Khameh N, Scheurer M, Creighton CJ, Musher B, Li W, Shen L. DNA methylation profiling at base-pair resolution reveals unique epigenetic features of early-onset colorectal cancer in underrepresented populations. Clinical epigenetics. 2025 Jan 23, 17:11.

Yang L, Peery RC, Farmer LM, Gao X, Zhang Y, Creighton CJ, Zhang L, Shen L. Dietary folate and cofactors accelerate age-dependent p16 epimutation to promote intestinal tumorigenesis. Cancer Res Commun. 2024 Jan 10, 4(1):164.

Yang L, Chen X, Lee C, Shi J, Lawrence EB, Zhang L, Li Y, Gao N, Jung SY, Creighton CJ, Li JJ, Cui Y, Arimura S, Lei Y, Li W, Shen L. Functional characterization of age-dependent p16 epimutation reveals biological drivers and therapeutic targets for colorectal cancer. J Exp Clin Cancer Res. 2023 May 4, 42 (1):113.

Shi J, Xu J, Chen Y, Li J, Shen L, Li JJ, Li W. The concurrence of DNA methylation and demethylation is associated with transcription regulation. Nature Communications. 2021 Sep 6, 12 (1):5285

My laboratory is interested in the epigenetic mechanisms in health and disease with a focus on transcriptional regulation by DNA methylation across development and during tumorigenesis. We use a combination of genomics, epigenomics, and next- generation gene editing approaches and a variety of model systems including cell culture, ex vivo organoids, germ-free and gnotobiotic mice.

Research Projects:

p16 Epimutation: Function in Tumorigenesis and As A Target for Cancer Therapy. p16 (or p16INK4a) is a tumor suppressor gene that regulates cell cycle. p16 epimutation - inactivation of p16 by promoter DNA methylation - is one of the most common epigenetic events in human colorectal cancer. We published the first mouse model of targeted p16 promoter methylation (J Clin Invest. 2014; 124:3708). Using these mice, we demonstrate that engineered p16 epimutation drives spontaneous tumor development. Recently, our studies have revealed that p16 epimutation operates synergistically with mutant K-ras and Apc(Min) to promote lung and colon tumor progression. Our overall goals are to understand epigenetic causality of human cancers and to identify epigenetic vulnerabilities that can be targeted for therapeutic intervention.

Gut Microbiota and Developmental Epigenetic Regulation of Intestinal Stem Cells. We are interested in understanding how DNA methylation processes interplay with environmental cues (nutrition and microbiota) to guide the emergence and behavior of intestinal stem cells (ISCs). Using mouse models, we demonstrate that the suckling period is critical for epigenetic changes in the development of ISCs, potentially affecting intestinal health for life (Genome Biol. 2015; 16:211). We show that postnatal intestinal epigenetic regulation is significantly altered by germ-free conditions. Interestingly, the successful restoration of DNA methylation depends on the timing of microbial colonization (i.e. pre-weaning vs. after-weaning), supporting a critical window phenomenon. Currently, we are applying state-of-the-art sequencing- based techniques to achieve the ultimate genome-wide, unbiased assessment of microbial effects on ISC epigenome. We are using cutting-edge genome editing to dissect the epigenetic mechanisms that regulate ISC function in response to the gut microbiota. Our ultimate goal is to develop epigenetically targeted probiotic therapies to provide lifelong protection against intestinal disease.

Dr. Shen's USDA and NIH-funded research is focused on epigenetics and the transcriptional regulation by DNA methylation in intestinal stem cells across development and tumorigenesis. Her research team is using a combination of genomics, epigenomics, and next-generation gene editing approaches and a variety of model systems including intestinal epithelial organoids, and germ-free and gnotobiotic mice. They have demonstrated that the suckling period is critical for epigenetic changes in the development of intestinal stem cells, potentially affecting intestinal health for life. In addition, they have shown that epigenetic regulation of intestinal epithelial cells is significantly altered by germ-free conditions. Furthermore, the successful restoration of intestinal DNA methylation patterns depends on the timing of microbial colonization (i.e. pre-weaning vs. after-weaning), supporting a critical window phenomenon. Currently, they are applying state-of-the-art sequencing-based techniques to achieve the ultimate genome-wide, unbiased assessment of the microbiome effects on the intestinal stem cell epigenome; and using cutting-edge organoid and CRISPR epigenome editing tools to dissect the microbiota-mediated epigenetic mechanisms that regulate intestinal stem cell function. T32 collaborations include Burrin, Preidis, Kellermayer. Theme: Mucosal Biology. (1 TG trainee in the past 10 yrs).

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BENJAMIN SHNEIDER

Translational and Clinical Research in Cholestasis, Fibrosis and Portal Hypertension

Professor
Chief, Section Gastroenterology, Hepatology and Nutrition
George Peterkin Endowed Chair
Department of Pediatrics
Baylor College of Medicine

Recent Publications

Grammatikopoulos T, Jaramillo C, Molleston J, Pimenta J, Ackermann O, Superina R, De Franchis R, Tutan S, Ling S, Ramamurthy U, Shneider BL.J Considerations in the development of the International Multicenter Pediatric Portal Hypertension Registry. Pediatr Gastroenterol Nutr. 2025 Jan;80(1):197-202.

Molleston JP, Goodrich NP, Ye W, Leung DH, Sokol RJ, Shneider BL. Prospective analysis of liver stiffness measurement by vibration controlled elastography as a predictor of outcomes in biliary atresia. Gastroenterology 168:393-395, 2025.

Bass LM, Ye W, Hawthorne K, Leung DH, Murray KF, … Shneider BL, on behalf of ChiLDReN. The Risk of Variceal Hemorrhage and Pre-Transplant Mortality in Children with Biliary Atresia. Hepatology. 76:712-726, 2022.

Shneider BL, Spino CA, Kamath BM, Magee JC, Ignacio RV, et. al. for ChiLDReN and UK IMAGO/IMAGINE Investigators. Impact of Long-term Administration of Maralixibat on Children with Cholestasis Secondary to Alagille Syndrome. Hepatol Commun. 6:1922-1933,2022. ostomy Predicts Short-Term Outcomes in Biliary Atresia. J Pediatr. 2016 Mar;170:211-217

Benjamin Shneider is the Head of the Division of Pediatric Gastroenterology, Hepatology and Nutrition, Professor of Pediatrics and the George Peterkin Endowed Chair. Dr. Shneider's leadership goal is to leverage the wide-ranging clinical and research expertise of an exceptionally talented division to provide compassionate, state-of-the-art, discipline-leading and evidence-based care for children with all types of gastrointestinal, hepatic, pancreatic and nutritional disorders. Rapidly-paced advances in biomedical knowledge provide an unprecedented opportunity to simultaneously advance both clinical care and the science of medicine. Dr. Shneider is a pediatric hepatologist and physician-scientist with varied areas of expertise including a clinical focus in pediatric hepatology primarily involving cholestatic liver diseases, basic and translational investigations of bile acid homeostasis, pediatric liver transplantation and pediatric portal hypertension. He is Associate Program Director and an Executive Committee member of the Pediatric GI T32 program.

Dr. Shneider has led multi-center clinical trials in and clinical investigations of cholestatic liver diseases in children. These have included investigator-initiated interventional clinical trials. Dr. Shneider is an internationally recognized thought-leader in pediatric portal hypertension, where he conducts numerous innovative studies including multi-center studies of the natural history of biliary atresia and other progressive cholestatic liver diseases. He leads an international consortium, The International Multicenter Pediatric Portal Hypertension Registry (IMPPHR), focused on understanding the natural history of and therapeutic approaches to variceal hemorrhage in children. His recent translational studies have examined the plasma proteome in pediatric cholestasis with reference to liver stiffness. Dr. Shneider has been continuously funded by the NIDDK for over 30 years for basic, translational and clinical investigations and is the PI for the BCM U01 site participating in the Childhood Liver Disease Research Network (ChiLDReN); he has been a PI in this network since inception in 2002. He has an extensive record in mentoring (>35) students, resident, fellows, post-doctoral fellows and faculty over his career; 14 remain in academic clinical faculty positions and three have received NIH funding.

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Tissue-engineered intestine and cell-based therapies for intestinal failure

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ALLISON SPEER

Recent Publications

Bordelon RC, Herath M, Speer AL. Innervation of Human Intestinal Organoids. J Vis Exp. 2025 Jan 17:(215).

Speer AL, Lally KP, Pedroza C, Zhang Y, Poindexter BB, Chwals WJ, Hintz SR, Besner GE, Stevenson DK, Ohls RK, Truog WE, Stoll BJ, Rysavy MA, Das A, Tyson JE, Blakely ML. Surgical Necrotizing Enterocolitis and Spontaneous Intestinal Perforation Lead to Severe Growth Failure in Infants – A Preplanned Secondary Analysis of the Necrotizing Enterocolitis Surgery Trial. Annals of Surgery. 2024 Sept 1;280(3):432-443.

Beanland BT, McNeill EP, Sequeira DJ, Xue H, Shroyer NF, Speer AL. Investigation of murine host sex as a biological variable in epithelial barrier function and muscle contractility in human intestinal organoids. FASEB J. 2022 Nov;36(11):e22613.

McNeill EP, Gupta VS, Sequeira DJ, Shroyer NF, Speer AL. Evaluation of murine host sex as a biological variable in transplanted human intestinal organoid development. Dig Dis Sci. 2022 Dec;67(12):5511-5521.

I am a pediatric surgeon and basic-translational scientist with clinical and research interests in pediatric intestinal failure and rehabilitation. I serve as the Surgical Director of the Short Bowel Syndrome Therapy and Rehabilitation (STAR) team at UTHealth and Children's Memorial Hermann Hospital. My long-term goal is to establish myself as an independent investigator focused on improving current treatments and developing novel regenerative medicine strategies for intestinal failure, including tissue-engineered intestine and cell-based therapies. My short-term goals are directed toward career development and investigating enteric nervous system (ENS) development and function within human intestinal organoid (HIO)-derived tissue-engineered intestine, the role of biomechanical forces during ENS development in HIOs, and optimization of human pluripotent stem cell-based therapy for Hirschsprung disease and other enteric neuropathies using the ganglionic HIO model. I have transplanted HIOs into over 300 immunodeficient hosts with greater than 80% engraftment and less than 2% mortality and have generated enteric neural crest cells for incorporation into HIOs, which is crucial for transplant studies. My work has shown that in vivo HIO transplantation enhances select tight junction gene expression and results in larger HIOs with fewer lumens in male versus female hosts. Current projects focus on testing whether serotonin receptors promote neurogenesis and intestinal barrier function.

I have been involved in clinical surgical research since 2005 and in intestinal tissue engineering, regenerative medicine, stem cell biology, and related developmental biology since 2009. My work has been recognized with the Ethicon-Society of University Surgeons Surgical Research Fellowship Award (2011), a pilot/feasibility award from the NIH-funded Texas Medical Center Digestive Diseases Center (2017), the NIH Long Repayment Program award (2018), an American Pediatric Surgical Association Foundation award (2020), a Men of Distinction Foundation award (2022), a NIH/NIDDK Career Development K08 award (2022), and an American Neurogastroenterology & Motility Society Transition award (2023).

I have found it essential to build my own mentoring team as an early-stage investigator. My scientific advisory committee guides my research, career development, and grant planning, teaching new skills and techniques while reviewing manuscripts and experimental plans. My mentoring team includes Noah F. Shroyer, PhD (primary mentor for my K08), Robert O. Heuckeroth, MD/PhD, and Michael A. Helmrath, MD, all of whom are NIH-funded. I have trained more than eight medical students and four postdoctoral fellows. My T32 collaborations include Burrin, Estes, and Mills, under the Mucosal Biology theme.

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Microbiome Regulation of Enterohepatic Bile Acid Signaling

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Recent Publications

Tessier MEM, Schraw JM, Beer S, Harpavat S, Kyle Jensen M, Magee JC, Ng V, Scheurer ME, Taylor SA, Shneider BL. The association of human milk intake and outcomes in biliary atresia. J Pediatr Gastroenterol Nutr. 2025 Jan;80 (1):163-173. doi: 10.1002/ jpn3.12403. Epub 2024 Nov 11. PMID: 39526563; PMCID: PMC11863988.

Tessier MEM, Cavallo L, Yeh J, Harpavat S, Hoffman KL, Petrosino JF, Shneider BL. The Fecal Microbiome in Infants With Biliary Atresia Associates With Bile Flow After Kasai Portoenterostomy. J Pediatr Gastroenterol Nutr. 2020 Jun;70(6):789-795. doi: 10.1097/ MPG.0000000000002686. PMID: 32443032.

Tessier MEM, Shneider BL, Petrosino JF, Preidis GA. Bile acid and microbiome interactions in the developing child. J Pediatr Gastroenterol Nutr. 2025 May;80 (5):832-839. doi: 10.1002/ jpn3.70014. Epub 2025 Feb 17. PMID: 39959949; PMCID: PMC12068970.

Tessier MEM, Shneider BL, Brandt ML, Cerminara DN, Harpavat S. A phase 2 trial of N-Acetylcysteine in Biliary atresia after Kasai portoenterostomy. Contemp Clin Trials Commun. 2019 May 2;15:100370. doi: 10.1016/ j.conctc.2019.100370. PMID: 31193715; PMCID: PMC6542754.

I am a former pediatric resident and pediatric gastroenterology fellow at BCM and Texas Children's Hospital. My research has been supported by an NIDDK Career Development Award (K23) and DDC Pilot Award. My research during fellowship focused on the dysregulation of enterohepatic circulation and bile flow in Clostridiales difficile infection. Through these basic and translational studies, she evaluated the impact of the microbiome on bile flow and vice-versa. These investigations laid the foundation to expand her studies to pediatric cholestasis and biliary atresia, a fibro-obliterative disorder of the extrahepatic biliary tree and the leading indication for liver transplant in children. Little is known about its pathogenesis, treatments options are limited, and the clinical course is variable and often characterized by progressive deterioration. Bile flow is crucial to the success of the one nontransplant treatment options for biliary atresia, the Kasai portoenterostomy. My K23 has established a multicenter study to assess the impact of the microbiome on bile flow and outcomes in infants with cholestasis, particularly biliary atresia. This study lays the foundation for clinical and translational studies in biliary atresia, including clinical trials of microbiome manipulation. I am also also involved in local and multicenter studies evaluating the microbiome in pediatric primary sclerosing cholangitis as well as is site PI for the Prospective Observational Study of Primary Sclerosing Cholangitis through the Childhood Liver Research disease network. T32 collaborations include Burrin, Harpavat, Shneider, Thevananther. Theme: Clinical and Translational, Immunology and Microbiology.

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Targeting P2Y2 purinergic signaling to mitigate sepsis-associated liver injury and improve outcomes in sepsis

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SUNDARARAJAH THEVANANTHER, Ph.D.

Recent Publications

Stayer, K, Pathan S, Biswas A, Li H, Zhu Y, Lam FW, Marini J, Thevananther S. Exogenous arginine differentially regulates inflammatory cytokine and inducible nitric oxide synthase expression in macrophages. *ImmunoHorizons*. 2025 Jul 14; 9(8): vlaf028.

Arunachalam AR, Samuel SS, Mani A, Maynard JP, Stayer KM, Dybbro E, Narayanan S, Biswas A, Pathan S, Soni K, Kamal AHM, Ambati CSR, Putluri N, Desai M, Thevananther S. P2Y2 purinergic receptor gene deletion protects mice from bacterial endotoxin and sepsis-associated liver injury and mortality. *Am. J. Physiol. Gastrointestinal. Liver Physiol*. 325(5):G471-G491, 2023

Maynard JP, Lee, J-S, Sohn BH, Yu X, Lopez-Terrada D, Finegold MJ, Goss JA, Thevananther S. P2X3 purinergic receptor overexpression is associated with poor recurrence-free survival in hepatocellular carcinoma patients. *Oncotarget*, 6 (38):41162-79, 2015

Tackett BC, Sun H, Mei Y, Maynard JP, Cheruvu S, Mani A, Hernandez-Garcia A, Vigneswaran N, Karpen SJ, Thevananther S. P2Y2 purinergic receptor activation is essential for efficient hepatocyte proliferation in response to partial hepatectomy. *Am. J Physiol Gastrointest Liver Physiol*. 307(11):G1073-87, 2014.

A major goal of my laboratory is to gain mechanistic insights into sepsis-associated liver injury and improve outcomes in sepsis.

The liver plays a significant role in regulating a wide range of metabolic, homeostatic, and host-defense functions. Hepatic dysfunction contributes to the severity of sepsis and is an independent risk factor for increased morbidity and mortality in patients. There is an urgent unmet need to develop innovative approaches to mitigate liver injury and restore metabolic and inflammatory homeostasis in septic patients.

Innate immune activation orchestrated by toll-like receptor (TLR) signaling acts as a 'double-edged sword' in the pathogenesis of sepsis-associated liver injury. While inflammation is necessary to fight infection, TLR4-mediated activation of hyperinflammation (cytokine storm) and liver injury can deplete systemic arginine levels, leading to multi-organ injury and mortality in sepsis patients.

TLR4-mediated macrophage activation leads to nucleotide release, contributing to elevated nucleotide levels in the extracellular milieu. While extracellular nucleotides have the potential to activate P2Y2 purinergic receptors expressed in macrophages and hepatocytes, the pathophysiologic relevance of P2Y2 purinergic signaling in sepsis-associated liver injury remains unknown.

Our ongoing studies are designed to test the *hypothesis that TLR4-mediated activation of P2Y2 purinergic signaling, via macrophage activation, as well as dysregulation of hepatocyte metabolic function, contributes to inflammatory liver injury and adverse outcomes in sepsis.*

Aim 1. To elucidate the role of TLR4-mediated activation of P2Y2 signaling as a key mediator of hepatic macrophage activation, leukocyte infiltration, and inflammatory liver injury.

Aim 2. To define the role of P2Y2 signaling in the dysregulation of hepatocyte fatty acid metabolism, lipotoxicity, and liver injury.

Aim 3. To determine if P2Y2 purinergic receptor antagonist treatment can attenuate sepsis-associated multi-organ injury and improve survival in mice.

Successful completion of this study will enhance our understanding of P2Y2 purinergic receptor-mediated signaling in hepatocytes and macrophages and establish the rationale for targeting P2Y2 purinergic signaling to mitigate sepsis-associated liver injury and improve outcomes in sepsis.

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Perinatal Physiology, NEC Pathogenesis, and Translational Interventions

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CAITLIN VONDEROHE

Recent Publications

Burrin D., Vonderohe CE, and Guthrie G. Functional importance of bile acid-FXR signaling in neonatal immunity and disease. Cellular & Molecular Immunology 2025.

Vonderohe C, Stoll B, Guthrie G, Melendez Hebib V, Dawson G, Burrin DG. 2023. Birth Modality Affects the Enterohepatic FXR-FGF19 Axis in Neonatal Piglets. Endocrinology. 164:1. https://doi.org/10.1210/endocr/bqac188

Vonderohe C, Guthrie G, Burrin DG. Fibroblast Growth Factor 19 Secretion and Function in Perinatal Development. . Am J Physiol Gastrointest Liver Physiol. 2023. doi: 10.1152/ajpgi.00208.2022

Vonderohe C, Guthrie G, Stoll B, Chacko S, Dawson G, Burrin DG. Tissue specific mechanisms of bile acid homeostasis and activation of FXR-FGF19 signaling in preterm and term neonatal pigs. Am J Physiol Gastrointest Liver Physiol. 2021. G117-G133.

I am a licensed veterinarian with doctoral training in swine nutrition, which uniquely qualifies me to manage and manipulate preterm piglet models and translate findings to clinical stakeholders. I am a former postdoctoral trainee on this T32 and a DDC Pilot Awardee. My research is supported by an NIDDK Career Development Award (K08), a Pediatrics Pilot Award, and the USDA. My long-term goal is to establish and maintain an independent, highly translational research program investigating the effects of preterm birth on perinatal hepatic, pulmonary, and gastrointestinal pathophysiology. I focus on characterizing the comorbidities of preterm birth and leveraging nutrition and targeted pharmaceutical therapies to improve clinical outcomes in this fragile patient population.

A major focus of my lab is necrotizing enterocolitis (NEC), a devastating intestinal disease in preterm infants with high mortality and long-term comorbidities such as short bowel syndrome and poor growth. Using preterm and term piglet models, I aim to characterize fundamental physiological differences between term and preterm infants and establish their predisposition to pathologic conditions like NEC. My current projects include: 1) investigating the impact of perinatal glucocorticoid exposure on the development of the gut and liver in preterm neonates (DK135845) and 2) evaluating the efficacy of manipulating arginine metabolism to prevent and treat NEC. The central hypothesis of my K08 project is that glucocorticoids upregulate developmental responses to bile acid homeostasis and FGF19 signaling during the perinatal period. I characterize developmental differences in molecular and cellular signaling pathways in preterm and term hepatocytes and intestinal enteroids and examine their association with cortisol, bile acid homeostasis, and FGF19 signaling. I also test the effects of birth modality (vaginal versus cesarean section) and glucocorticoid treatment (maternal and neonatal) on gut-liver bile acid homeostasis and FXR-FGF19 signaling in neonatal pigs.

Additionally, my lab studies the potential of modulating systemic arginine levels to prevent and treat inflammatory diseases like NEC. Using a drug that converts intravascular arginine into citrulline, we observed reduced inflammatory infiltrates in a juvenile porcine model of pediatric sepsis, and I am further exploring the mechanism and clinical potential of this approach for NEC. My T32 collaborations include Burrin, Guthrie, Hair, Lam, Mills, Preidis, and Ramani.

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Mechanisms of Parasitic Infection and Immune-Mediated Organ Damage

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JILL WEATHERHEAD

Recent Publications

Wu, Y, G Adniyi-Ipadeola, M Adkins-Threats, M Seascock, C Suarez-Reyees, R Fujiwara, ME Bottazzi, L Song, JC Mills, JE Weatherhead. Host gastric corpus microenivronment facilitates Ascaris suum larval hatching and infection in a murine model. PLoS Neglected Tropical Diseases. 2024. 18(2): e0011930. doi: 10.1371/journal.pntd.0011930.

Wu, Y, C Suarez-Reyes, A Kneubehl, JE Weatherhead. Repeat Ascaris challenge reduces worm intensity through gastric cellular reprogramming. bioRxiv. 2024. doi: 10.1101/2024.08.29.610358.

Wu, Y, E Li, M Knight, G Adeniyi-Ipadeola, L Song, A Burns, AC Gazzinelli-Guimaraes, R Fujiwara, ME Bottazzi, JE Weatherhead. Transient Ascaris suum larval migration induces intractable chronic pulmonary disease and anemia in mice. PLOS Neglected Tropical Diseases. 2021. 15 (12): e0010050. doi: 10.1371/journal.pntd.0010050.

Weatherhead, JE, P Gazzinelli-Guimaraes, JM Knight, R Fujiwara, P Hotez, ME Bottazzi, D Corry. Host immunity and inflammation to pulmonary helminth infections. Front Immunol. 2020. 11:594520.

I am a tenured Associate Professor of Pediatrics and Medicine in the Sections of Pediatric Tropical Medicine, Pediatric Infectious Diseases, and Adult Infectious Diseases at Baylor College of Medicine (BCM), board certified in internal medicine, pediatrics, pediatric infectious diseases, and adult infectious disease, with a sub-specialty certificate in tropical medicine and traveler's health (CTropMed) from the American Society of Tropical Medicine and Hygiene (ASTMH). As a physician-scientist, my clinical and research work is driven by a commitment to improve the lives of children living in parasite-endemic, poverty-stricken communities worldwide. I apply my clinical and scientific expertise to answer fundamental questions about parasite pathogenesis and host–pathogen interactions, using in vitro and in vivo model systems to understand parasite-induced immunopathology and identify novel preventative and therapeutic interventions.

I have developed an active translational research program investigating ascariasis, the most common helminth infection globally, affecting nearly 500 million people. Ascariasis causes significant life-long morbidity, particularly in children with high worm burden, yet no interventions currently prevent infection due to limited understanding of larval infection mechanisms. My laboratory was the first to demonstrate that ingested Ascaris eggs exploit the host gastric microenvironment to enhance larval hatching and initiate infection. We further discovered that repeat Ascaris infection induces gastric mucosal injury, triggering cellular reprogramming known as pyloric metaplasia, which inhibits future infection and likely represents an evolutionary anti-Ascaris mechanism. Building on this work, I am now evaluating how Ascaris-induced pyloric metaplasia affects anti-helminth immunity, Helicobacter pylori colonization, and gastric pathology. This research will identify gastric mucosal receptors that permit larval translocation and initiation of infection and may establish Ascaris infection as a risk factor for H. pylori–related gastric disease.

My research is supported by an NIAID Career Development Award (K08-A143968), NIAID R03 grants (AI173826 and AI180714), and a DDC Pilot Feasibility Award.

As an early-stage investigator, my goal is to define host immune responses to parasites to develop vaccines and therapeutics that prevent parasite-induced morbidity. This work has the potential to advance understanding of parasite pathogenicity and inform novel interventions that reduce morbidity for millions of children globally. My T32 collaborations include Estes and Mills, under the Immunology and Microbiology theme.

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Nuclear receptor signaling in liver disease

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CLAVIA RUTH WOOTON-KEE

Recent Publications

Wooton-Kee CR, Yalamanchili HK, Mohamed I, Hassan M, Setchell KDR, Narvaez Rivas M, Coskun AK, Putluri V, Putluri N, Jalal P, Schilsky ML, Moore DD. Changes in the FXR -cistrome and alterations in bile acid physiology in Wilson disease. Hepatol Commun. 2025. Vol. 23;9 (6):e0707;

Suh JH, Cheon I, Jung HJ, Lee SH, Heo MJ, DeBerge M, **Wooton-Kee CR**, Kim KH. Bile acid regulation of xenobiotic nuclear receptors on the expressions of orosomucoids in the liver. Am J Physiol Endocrinol Metab. 2025 ;328(6):E940-E951.

Wooton-Kee CR. Therapeutic implications of impaired nuclear receptor function and dysregulated metabolism in Wilson's disease. Pharmacol Ther. 2023; 251:108529.

Wooton-Kee CR, Robertson M, Zhou Y, Dong B, Sun Z, Kim KH, Liu H, Xu Y, Putluri N, Saha P, Coarfa C, Moore DD, Nuotio-Antar AM. Metabolic dysregulation in the Atp7b(-/-) Wilson's disease mouse model. Proc Natl Acad Sci U S A. 2020 Jan 28;117(4):2076-2083.

Wooton-Kee CR, Jain AK, Wagner M, Grusak MA, Finegold MJ, Lutsenko S, Moore DD. Elevated copper impairs hepatic nuclear receptor function in Wilson's disease. J Clin Invest. 2015. Sep;125 (9):3449-60.

Nuclear hormone receptors are highly conserved transcription factors that regulate a variety of biological processes, such as steroid hormone biosynthesis and metabolism. My laboratory's research program centers on how nuclear receptor dysfunction contributes to the pathogenesis of metabolic liver diseases.

Previous studies with the estrogen receptor demonstrated a deleterious effect of copper on estrogen receptor structural confirmation and activity. Wilson disease is an autosomal recessive disorder that results in toxic copper accumulation primarily in the liver and brain, therefore a robust model of copper toxicosis. Our long-term studies focus on the impact of copper on the activity of hepatic nuclear receptors in Wilson disease. We found that chronic and toxic levels of copper reduced the activity of hepatic nuclear receptors, resulting in dysregulation of hepatic metabolism and pathways involved in cellular proliferation and regeneration. Our current studies are focused on determining the mechanisms underlying copper-mediated nuclear receptor dysfunction and the utility of nuclear receptor ligand therapy in Wilson disease mouse models.

We recently began studies to explore how maternal metabolism and hepatic gene expression adapt during lactation. Lactation is a physiological state that requires a profound increase in energy demand to meet the nutritional requirements of the mother and the offspring. Despite the well-known benefits of lactation for both the mother and offspring, there is a paucity of metabolomic data in lactating dams. Our USDA-funded studies focus on maternal liver-gut metabolic adaptations during lactation and whether lactation confers protection against fatty liver and MASH development. This work is advancing our understanding of physiological hepatic plasticity and informing broader questions about liver function in the context of reproductive and metabolic transitions.

T32 collaborations include Drs. Burrin, Guthrie, Harpavat, Mysore, and Preidis. Theme: Molecular Hepatology.

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