BIOGRAPHICAL SKETCH

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NAME: Huimin Yu

eRA COMMONS USER NAME (credential, e.g., agency login): HUIMINYU

POSITION TITLE: Assistant Professor of Medicine

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Shanghai Jiao Tong University, China	M.D.	08/2004	Medicine
Johns Hopkins University, MD	Ph.D.	12/2010	Biology and Genetics
University of Virginia, VA	Resident	08/2015	Internal Medicine
Johns Hopkins University, MD	Fellow	12/2018	Gastroenterology

A. Personal Statement

I am committed to a career as a physician-scientist with a focus on inflammatory bowel disease (IBD) and host-pathogen interactions. My goal is to translate my research into better patient care, especially for those who suffer from IBD. I received my M.D degree from Shanghai Jiao Tong University School of Medicine. I then pursued Ph.D. and postdoctoral training with Dr. Jeremy Nathans, an HHMI investigator, at Johns Hopkins University, studying the role of three Frizzled receptors (Frizzled 1, 2 and 7) in development. I published two first-author papers in ***Development*** and co-author paper in ***Neuron***. During my residency training, I did some clinical study about the Crohn disease. After residency training in internal medicine at University of Virginia, I started fellowship training in the physician-scientist track in Gastroenterology at Johns Hopkins University, where I continued to develop expertise in the field of IBD. During my fellowship, I studied the pathogenesis of Enterotoxigenic *E. coli* (ETEC) diarrhea in human mini-gut organoids with Dr. Mark Donowitz. I joined the faculty at Johns Hopkins University with 75% effort dedicated to research. My current focus is (1) to study the role and mechanism of creeping fat formation; and (2) to identify the pathways that causally link adipogenesis, intestinal inflammation, and intestinal fibrosis in IBD.

B. Positions and Honors

Positions

2010 – 2012	Postdoctoral Fellow	Johns Hopkins University School of Medicine
2012 – 2015	Resident in Medicine	University of Virginia School of Medicine
2015 – 2018	Clinical and Research Fellow in Medicine	Johns Hopkins University School of Medicine
2019 – present	Assistant Professor in Medicine	Johns Hopkins University School of Medicine

Honors

1997:	Distinguished Student Award, Shanghai Municipality (provincial level honor)
1998:	Distinguished Student Award in Public Health Research, Shanghai Municipality
1999:	6th National “Challenge Cup” Academic Science & Technology Competition Award
1999:	BaoGang National Excellent Student Scholarship, BaoGang Educational Foundation
2000:	Excellent Student Leader Award, Shanghai Jiao Tong University
1996 – 2003:	Excellent Academic Scholarship (1st Prize), Shanghai Jiao Tong University
2004:	NSF Pre-Doctoral Fellowship Program Competition, Honorable Mention

2004 – 2010: H.A. and Mary K. Chapman Young Investigator Fellowship, Chapman Foundation

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C. Contributions to Science

1. Developmental biology. During my Ph.D. study and postdoctoral training with Dr. Jeremy Nathans, I studied the role of three Frizzled receptors (Frizzled 1, 2 and 7) in development by using single, double, and triple knockout mouse models. The goal of this work is to understand the molecular and cellular basis of human disease during development**.** I found that mutations in these genes cause diverse phenotypes, including cleft palate, congenital heart defects, and neural tube defects. My work linked the mechanisms responsible for the palate, ventricular septum, and neural tube closure, each of which involves directional tissue movements followed by tissue fusion. My studies suggested that Frizzled signaling may be involved in some of the most common congenital anomalies in humans. My studies also showed an extended web of genetic interactions between these three Frizzleds. In addition, I also studied X chromosome inactivation (XCI) by using a dual-color genetic system for cell-type selective visualization of XCI mosaicism. Our group defined the topography of XCI mosaicism at single-cell resolution.

a. Wu H, Luo J, **Yu H**, Rattner A, Mo A, Wang Y, Smallwood PM, Erlanger B, Wheelan SJ, Nathans J. (2014) Cellular resolution maps of X chromosome inactivation: implications for neural development, function, and disease. *Neuron* 81:103-119.

b. **Yu H**, Ye X, Guo N, Nathans J. (2012) Frizzled2 and Frizzled7 function redundantly in convergent extension and closure of the ventricular septum and palate: evidence for a network of interacting genes. *Development* 139:4383-4394.

c. **Yu H**, Smallwood P, Wang Y, Vidaltamayo R, Reed R, Nathans J. (2010) Frizzled1 and Frizzled2 genes function in palate, ventricular septum and neural tube closure: general implications for tissue fusion processes. *Development* 137: 3707-3717.

2. Inflammatory bowel disease (IBD). During my residency at the University of Virginia, I did a retrospective cohort study of narcotic misuse in 931 patients with Crohn's disease (CD) under the guidance of Dr. Brian Behm, an IBD specialist. We found that 20% of patients with CD were using chronic narcotics with higher rates (37%) in those with a concurrent FGID. We recommended screening for narcotic misuse in patients with CD with a concomitant FGID and using prescription monitoring programs to identify others at risk for misuse. After I became faculty at Hopkins since 01/2019, I have been focusing on the role of inflammation in the pathogenesis of IBD.

a. **Yu H**, Crocker J, Foley S, Tuskey A, Behm B. (2014) Polypharmacy in Crohn’s Disease. Poster presented at *Digestive Diseases Week 2014.*

b. Crocker J, **Yu H**, Conaway M, Tuskey A, Behm B. (2014) Narcotic use and misuse in Crohn’s disease. *Inflammatory Bowel Diseases* 20: 2234-2238.

3. Host-pathogen interactions. During my GI fellowship, I used human enteroids (mini-intestines) as a model to study the pathogenesis of Enterotoxigenic *E. coli* (ETEC) diarrhea under the guidance of Dr. Mark Donowitz. I developed a model of ETEC diarrhea by exposing ETEC to human enteroids with a demonstration of elevated intracellular and extracellular cGMP. I also characterized how ETEC exposure alters the epithelial tight junction and explored the role of innate immunity by using enteroid-macrophage co-culture system.

a. Foulke-Abel J, **Yu H**, Sunuwar L, Lin R, Fleckenstein J, Kaper J, Donowitz M. Phosphodiesterase 5 (PDE5) restricts intracellular cGMP accumulation during enterotoxigenic Escherichia coli infection. Gut Microbes. 2020; 1-12.

b. **Yu H**, Sunuwar L, Fleckenstein J, Donowitz M, Foulke-Abel J. (2017) Enterotoxigenic *E. coli* (ETEC) pathogenesis modeled in human enteroid monolayers demonstrates MRP-related cyclic nucleotide secretion. Oral presentation at *Digestive Diseases Week 2017*.

c. **Yu H**, Hasan N, In Julie, Estes M, Kovbasnjuk O, Zachos N, Donowitz M. (2017). The contributions of human mini-intestines to the study of intestinal physiology and pathophysiology. *Annual review of physiology* 73: 291-312.

My Bibliography:
https://www.ncbi.nlm.nih.gov/sites/myncbi/1JkirDLvQKV/bibliography/55063844/public/?sort=date&direction=ascending

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D. Additional Information: Research Support and/or Scholastic Performance

Current Research Support

Discovery Fund Challenge Awards Yu (PI) 01/01/2020 - 12/31/2021
Johns Hopkins School of Medicine
Title: **The role of creeping fat in intestinal fibrosis and stricturing in Crohn's disease**
Role: Principal Investigator

Completed Research Support

Hopkins GI core center pilot grant Yu (PI) 09/01/2019 - 08/31/2020
Hopkins Conte Digestive Diseases Basic and Translational Research Core Center
Title: **Inflammation in intestinal epithelial organoids**
Role: Principal Investigator