Name: Joseph T Blumer

Email: jblumer@wisc.edu

Major Professor: Dawn Belt Davis, MD, PhD

Degree Objective: PhD, Endocrinology and Reproductive Physiology

Background: BS Biology and Spanish, University of Wisconsin-Madison

Current Research Project:

My research focuses on studying the interactions of the novel diabetes gene, transcription factor 19 (Tcf19), in the mouse pancreatic β-cells and high fat high sucrose diet feeding. Particularly, I am interested in how early life experiences impact future metabolic disease. Transcription factor 19 (*Tcf19*) is a novel transcriptional regulator that has been identified as a potential causal gene in genome-wide association studies for both TID and TIID. *Tcf19* is expressed in both humans and rodents. Although ubiquitously expressed, *Tcf19* is most highly expressed in the pancreatic islet and is upregulated in mouse models of non-diabetic obesity. Knockdown experiments in the rat β-cell line, INS1, show that TCF19 is necessary for promoting β-cell proliferation and survival. We have generated a mouse model with whole-body knockout of *Tcf19* (wbTcf19KO) and after 10 weeks of high fat diet (HFD) feeding wbTcf19KO mice become significantly glucose intolerant, a hallmark of gestational diabetes. The next phase of research is to determine the impact on developing fetuses when pregnant dams are at increased genetic and environmental risk for gestational diabetes.

Positions and Employment

2021- Spring Grading TA – Public Health Genomics, UW-Madison
2018-Present Graduate Research Assistant – Davis Lab, UW-Madison
2017-2018 Research Intern – Davis Lab, UW-Madison
2011-2016 CNA – Surgery and Procedure Center, Monroe Clinic

Funding

2020 Metabolism and Nutrition Training Program NIH T32DK007665

Awards

2020 CaMPS Robert Gunn Student Award Finalist

2018 CSCTR-MWAFMR 2018 Trainee Travel Award
2017 Department of Medicine Research Day Poster Award
2016 Dean's List
2014 Dean's List

Professional Memberships

2019-Present American Physiological Society
2018-2019 Korean-American Scientists and Engineers Association

Committees

2020-Present Endocrinology and Reproductive Physiology Student Committee, UW-Madison

Mentoring

2020-Present	**Keelin Ricciardi**	**Undergraduate**
2019-2020	**Anya Beric**	**Undergraduate (Biology 152 Independent Project)**
2017-2018	**Bilal Malas, BS**	**Undergraduate**
2017-2018	**Lucille Anzia, BS**	**Undergraduate**

2017	Katherine Bekasova	**Summer High School Intern**
2016	**Priya Mathur**	**Summer High School Intern**

C. Contributions to Science

Undergraduate

I began my research career during my time as an undergrad student at UW-Madison in the lab of Dr. Davis. I started by working on my Biology 152 Independent Project under the direct mentorship of Dr. Amelia Linnemann. I learned a variety of lab techniques with emphasis on Western blotting, which was used to show that interleukin 6 (IL-6) promotes the phosphorylation of STAT3, a cytoplasmic inhibitor of autophagy, allowing STAT3 to dimerize and enter the nucleus. This work culminated in a peer-reviewed publication in *The FASEB Journal*, on which I was 2nd author, describing the mechanisms by which IL-6 protects pancreatic beta-cells from stress induced apoptosis through stimulation of autophagy.

Publications:

Linnemann AK, **Blumer J**, Marasco MR, Battiola TJ, Umhoefer HM, Han JY, Lamming DW, Davis DB. "Interleukin 6 protects pancreatic β cells from apoptosis by stimulation of autophagy." *The FASEB Journal,* vol. 31, no. 9, Sept. 2017, pp. 4140-4152., doi:10.1096/fj.201700061rr.

Research Intern

As a research intern in the Davis Lab I transitioned from studying IL-6 to Transcription factor 19 (Tcf19). I worked with an undergraduate student to characterize a whole body Tcf19 knockout (wbTcf19KO) mouse model, and designed and executed experiments to image and quantify beta-cell mass, proliferation, and apoptosis from these mice. The results of these experiments are included in the manuscript I am currently in the process of writing, on which I will be a co-first-author. Additionally, I assisted other graduate students and post-doctoral fellows in the lab with their experiments studying the role of cholecystokinin in protecting beta-cells from apoptosis, recently published as a preprint in BioRxiv and submitted for review to *Translational Research* journal.

Local Poster Presentation:

UW Department of Medicine Research Day (2017)

Tcf19 Regulates Key Stress Response Pathways and DNA Damage Repair Genes

Danielle A. Fontaine, Jee Young Han, Sukanya Lodh, **Joseph Blumer**, Avtar Roopra, Dawn Belt Davis.

Published Abstracts from National Meetings

Combined Annual Meeting of CSCTR and MWAFMR (2018)

JT Blumer, GH Yang, DA Fontaine, JY Han, A Roopra, DB Davis "TCF19, A NOVEL DIABETES GENE, REGULATES STRESS RESPONSE PATHWAYS IN THE PANCRATIC beta-CELL." *JOURNAL OF INVESTIGATIVE MEDICINE* 66 (4), 823-824, 2018

Submitted Publications:

Hung Tae Kim, Arnaldo H. de Souza, Heidi Umhoefer, Jee Young Han, Lucille Anzia, Steven J. Sacotte, Rashaun A. Williams, **Joseph T. Blumer**, Jacob T. Bartosiak, Danielle A. Fontaine, Mieke Baan, Carly R. Kibbe, Dawn Belt Davis. "Cholecystokinin Suppresses Beta-Cell Apoptosis, Including in Human Islets in a Transplant Model" *bioRxiv* [Preprint] March 3, 2021; doi: https://doi.org/10.1101/2021.03.02.433645

Graduate

Since joining the Davis Lab as a graduate student, I have focused on the role of Tcf19 specifically in the pancreatic beta-cells. I expect to publish at least two first-author peer reviewed manuscripts based on the results generated from the aims of this proposal. I have recently contributed as co-author on a preprint examining the impact of TCF19 expression on DNA damage pathways in the β-cell.

Local Oral and Poster Presentations:

VA Hospital Research Day (2019 – Poster)

Tcf19 knockout mice have impaired insulin secretion and altered β–cell gene expression
Jee Young Han, **Joseph T Blumer**, Sukanya Lodh, Grace H Yang, Danielle A Fontaine, Dawn B. Davis

Virtual Madison Scholars Symposium (2020 – Oral)

Whole-Body Tcf19 Knockout Mouse Islets Have Increased Stress-Related Gene Expression And Reduced Proliferative Capacity

Poster Presentations at National Meeting/Published Abstracts:

Midwest Islet Club (2019)
Tcf19 knockout mouse islets have increased stress-related gene expression and reduced proliferative capacity. **Joseph T Blumer**, Jee Young Han, Grace H Yang, Sukanya Lodh, Danielle A Fontaine, Hannah Foster, Dawn B Davis

366-OR: TCF19 Promotes Functional Beta-Cell Mass and Proliferative Capacity by Regulating DNA Damage Repair Pathways. GH YANG, JEEY HAN, S LODH, **JT BLUMER**, D FONTAINE, DB DAVIS
Diabetes 68 (Supplement 1), 2019

Tcf19 Knockout Mouse Islets Have Increased Stress-related Gene Expression and Reduced Proliferative Capacity. **JT BLUMER**, JY HAN, GH YANG, S LODH, DA FONTAINE, DB DAVIS
The FASEB Journal 34 (S1), 1-1, 2020

Submitted Publications:

Yang GH, Fontaine DA, Lodh S, **Blumer JT**, Roopra AS, Davis DB. "Elucidating the crosstalk between inflammation and DNA damage repair pathways in diabetes through the diabetes susceptibility gene, Tcf19", *BioRxiv* 438736 [Preprint] April 6, 2021, Available at https://doi.org/10.1101/2021.04.06.438736