BIOGRAPHICAL SKETCH
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NAME: Tomblin, Justin

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

POSITION TITLE: Postdoctoral Researcher

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
Marshall University, Huntington, WV	B.Sc.	05/2011	Biotechnology
Marshall University, Huntington, WV	Ph.D.	05/2016	Biomedical Sciences

A. Personal Statement

I have the necessary background and preparation to be considered as a future junior investigator for this COBRE, which is focused on cellular transport physiology in obesity related disorders. My postdoctoral training has given me an in depth understanding on micro-RNAs and their function, and how this relates to intestinal nutrient absorption, in the context of obesity and inflammation. My specific focus will be to decipher the network of micro-RNAs responsible for the changes in the level of nutrient transporters noted in the small intestine during the obese state. The molecular mechanisms will be elucidated via micro-RNA sequencing studies performed in two separate accepted *in vivo* models of obesity. Once we uncover micro-RNAs of interest, we can then target them and study whether their silencing or overexpression enhances or reduces the absorption of important transporters in the gut, such as the glutamine transporter, B0AT1. Because of my training under the supervision of the principal investigator Dr. Uma Sundaram, I can confidently say that my molecular background will play an ample part in carrying out the aims of the current research proposal being submitted.

B. Positions and Honors

Positions and Employment

2016- Postdoctoral Fellow, Department of Clinical and Translational Sciences, Joan C. Edwards School of Medicine, Marshall University, 1600 Hal Greer Blvd. Huntington, WV

Other Experience and Professional Memberships

2013- Society of Toxicology (SOT)
2015- The American Society for Pharmacology and Experimental Therapeutics (ASPET)

Honors

2014 Best Research Performance Award for the academic year 2013-2014
Biomedical Sciences Graduate Program
2015 Graduate Research Fellowship Award
NASA West Virginia Space Grant Consortium
2016 Best Oral Presentation, Basic Science Category
MUSOM Research Day 2016

C. Contribution to Science

1. My recent publication on regulation of the amino acid transporter SLC7A5/LAT1 by the aryl hydrocarbon receptor (AHR) in breast tumor cells was a novel report. We found via RNA sequencing studies that the known AHR agonist, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), stimulated the expression of SLC7A5 in MCF-7 cells. We then showed direct binding of AHR to the SLC7A5 gene promoter to regulate its expression. We also showed that using a known AHR antagonist blocked TCDD induction of SLC7A5. Finally, silencing of AHR with targeting short interfering RNA specific for AHR also reduced SLC7A5 RNA and protein levels in multiple breast tumor types, including triple-negative breast tumor cells. The outcome of these findings provides a better understanding of how AHR plays multiple roles in tumor cell biology, including facilitating the uptake of important amino acids like leucine and tryptophan via upregulation of SLC7A5. These findings present new evidence for why targeting AHR in tumor cells could be an important therapeutic option.

    a. Tomblin JK, Arthur S, Primerano DA, Chaudhry AR, Fan J, Denvir J, Salisbury TB. 2016 Aryl hydrocarbon receptor (AHR) regulation of L-Type Amino Acid Transporter 1 (LAT-1) expression in MCF-7 and MDA-MB-231 breast cancer cells. Biochem Pharmacol. 2016 Mar 1. pii: S0006-2952(16)00134-9. doi: 10.1016/j.bcp.2016.02.020.

2. In another study, we have shown that insulin like growth factor 2 (IGF-2) can stimulate the RNA and protein expression of AHR, and subsequent recruitment of AHR to the cyclin D1 (CCND1) gene promoter in breast cancer cells to increase CCND1 expression. AHR-null cells showed reduced CCND1 induction upon IGF-2 stimulus. This other report revealed that AHR could be important for breast cancer in the context of obesity, where circulating levels of IGF-2 are higher.

    a. Tomblin JK, Salisbury TB 2014 Insulin like growth factor 2 regulation of aryl hydrocarbon receptor in MCF-7 breast cancer cells. Biochem Biophys Res Commun. 2014 Jan 17;443(3):1092-6.

Complete List of Published Work in MyBibliography:
https://www.ncbi.nlm.nih.gov/myncbi/browse/collection/51763993/?sort=date&direction=ascending

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