BIOGRAPHICAL SKETCH
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NAME: Salisbury, Travis

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

POSITION TITLE: Associate Professor of Biomedical Sciences

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
Kent State University, Kent, OH	B.S.	05/1997	Zoology
Kent State University, Kent, OH	Ph.D.	05/2003	Physiology
Case Western Reserve, School of Medicine, Cleveland, OH	Postdoctoral	10/2003	Molecular Endocrinology
Washington State University , Pullman , WA	Postdoctoral	09/2009	Molecular Endocrinology

A. Personal Statement

I have 13 years of experience in studies on regulation of gene expression in response to signaling. My work on regulated gene expression started during my postdoctoral training. My research identified that gonadotropin releasing hormone (GnRH), which signals through a G protein coupled receptor (GPCR), regulates gene expression by activating the transcriptional coactivator β-catenin. Finding that GPCR signaling regulated β-catenin activity was novel, because β-catenin had historically been associated with the developmental Wnt signaling pathway. Currently, I supervise an active laboratory, teach medical and graduate students and I was promoted to associate professor with tenure in 2015. My work remains focused on signal-regulated gene expression. Our recent studies have identified that the aryl hydrocarbon receptor (AHR), which is a ligand-activated transcription factor, responds to and mediates cancer signaling pathways that are important for breast cancer proliferation, survival and invasion. This includes two recent RNA-seq papers that we published in Biochemical Pharmacology. Our most recent report (2016) involving RNA-seq, coupled with chromatin immunoprecipitation (ChiP) and gene knockdown experiments, identified that L-Type Amino Acid Transporter 1 (LAT1) is a direct AHR gene target. Having identified that LAT1 is a direct AHR gene target is central to our hypothesis that obesity upregulates LAT1 and associated leucine signaling in breast tumors through AHR dependent mechanisms. In addition to increasing our understanding of obesity and breast cancer, the mentoring I will receive as a COBRE mentee will foster my career development and expertise in nutrient transport and breast cancer research.

1. 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. Tomblin JK, Arthur S, Primerano DA, Chaudhry AR, Fan J, Denvir J, Salisbury TB. Biochem Pharmacol. 2016 Apr 15;106:94-103. PubMed PMID: 26944194 PubMed Central PMCID: PMC4813787

2. Salisbury TB, Tomblin JK, Primerano DA, Boskovic G, Fan J, Mehmi I, Fletcher J, Santanam N, Hurn E, Morris GZ, Denvir J. Endogenous aryl hydrocarbon receptor promotes basal and inducible expression of tumor necrosis factor target genes in MCF-7 cancer cells. Biochem Pharmacol. 2014 Oct 1;91(3):390-9. PubMed PMID: 24971714; PubMed Central PMCID: PMC4157967.

3. Tomblin JK, Salisbury TB. 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. PubMed PMID: 24380854; PubMed Central PMCID: PMC3932621.

4. Salisbury TB, Binder AK, Grammer JC, Nilson JH. GnRH-regulated expression of Jun and JUN target genes in gonadotropes requires a functional interaction between TCF/LEF family members and beta-catenin. Mol Endocrinol. 2009 Mar;23(3):402-11. PubMed PMID: 19131506; PubMed Central PMCID: PMC2654513.
5. Salisbury TB, Binder AK, Nilson JH. Welcoming beta-catenin to the gonadotropin-releasing hormone transcriptional network in gonadotropes. Mol Endocrinol. 2008 Jun;22(6):1295-303. PubMed PMID: 18218726.

B. Positions and Honors

Positions and Employment

2003 – 2004	Postdoctoral Fellow, Case Western School of Medicine
2004 - 2009	Postdoctoral Fellow, Washington State University
2009-2015	Assistant Professor, Marshall University, School of Medicine
2015-	Associate Professor with tenure, Marshall University, School of Medicine

C. Contribution to Science

1. My postdoctoral publications directly investigated the mechanism by which the transcriptional coactivator β-catenin was required for maximal expression of the LHβ gene, which encodes the β subunit of luteinizing hormone (LH) in response to GnRH signaling. This publication identified that β-catenin increases transcription of the LHβ gene by binding to the transcription factor steroidogenic factor 1 (SF1). We then published that GnRH signaling induces a functional interaction between β-catenin and the TCF/LEF family of transcription factors, and that this was required for the induction of JUN expression and JUN target genes. Collectively, these publications demonstrated that GnRH can signal through β-catenin to regulate the expression of genes that define a functional gonadotrope and are essential for reproduction. Publications by other laboratories have now demonstrated that the regulation of SF-1 or TCF/LEF gene targets by hormones that signaling through G protein coupled receptors (GPCRs) occurs at many gene promoters and in different tissues such as the ovary or testis.

    a. Salisbury TB, Binder AK, Grammer JC, Nilson JH. GnRH-regulated expression of Jun and JUN target genes in gonadotropes requires a functional interaction between TCF/LEF family members and beta-catenin. Mol Endocrinol. 2009 Mar;23(3):402-11. PubMed PMID: 19131506; PubMed Central PMCID: PMC2654513.
    b. Salisbury TB, Binder AK, Nilson JH. Welcoming beta-catenin to the gonadotropin-releasing hormone transcriptional network in gonadotropes. Mol Endocrinol. 2008 Jun;22(6):1295-303. PubMed PMID: 18218726.
    c. Salisbury TB, Binder AK, Grammer JC, Nilson JH. Maximal activity of the luteinizing hormone beta-subunit gene requires beta-catenin. Mol Endocrinol. 2007 Apr;21(4):963-71. PubMed PMID: 17244763.

2. My current studies are investigating signal-induced regulation of gene expression in human breast cancer cells. We have focused on adipokines (which refers to paracrine and endocrine factors released by adipocytes), aryl hydrocarbon receptor (AHR) ligands, tumor necrosis factor (TNF) and insulin like growth 2 (IGF2). We have discovered that the regulation of gene expression and cancer processes (such as proliferation or viability) by each of these diverse signals (adipokines, TCDD, IGF2 and TNF) in breast cancer cells is mediated by AHR. Our continued work on RNA-seq projects has led to a manuscript that was published last year (2016) in Biochemical Pharmacology that demonstrated that signal-induced regulation of L-Type Amino Acid Transporter 1 (LAT1) by AHR occurs through a distal enhancer in intron 2 of the LAT1 gene. These publications and our proposed experiments are providing evidence to target AHR in breast cancer in obesity

    a. 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. Tomblin JK, Arthur S, Primerano DA, Chaudhry AR, Fan J, Denvir J, Salisbury TB. Biochem Pharmacol. 2016 Apr 15;106:94-103. PubMed PMID: 26944194 PubMed Central PMCID: PMC4813787

b. Salisbury TB, Tomblin JK. Insulin/Insulin-like growth factors in cancer: new roles for the aryl hydrocarbon receptor, tumor resistance mechanisms, and new blocking strategies. Front Endocrinol (Lausanne). 2015;6:12. PubMed PMID: 25699021; PubMed Central PMCID: PMC4313785.

c. Salisbury TB, Tomblin JK, Primerano DA, Boskovic G, Fan J, Mehmi I, Fletcher J, Santanam N, Hurn E, Morris GZ, Denvir J. Endogenous aryl hydrocarbon receptor promotes basal and inducible expression of tumor necrosis factor target genes in MCF-7 cancer cells. Biochem Pharmacol. 2014 Oct 1;91(3):390-9. PubMed PMID: 24971714; PubMed Central PMCID: PMC4157967.

d. Tomblin JK, Salisbury TB. 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. PubMed PMID: 24380854; PubMed Central PMCID: PMC3932621.

e. Salisbury TB, Morris GZ, Tomblin JK, Chaudhry AR, Cook CR, Santanam N. Aryl hydrocarbon receptor ligands inhibit igf-2 and adipokine stimulated breast cancer cell proliferation. ISRN Endocrinol. 2013;2013:104850. PubMed PMID: 24171117; PubMed Central PMCID: PMC3793317.

Complete List of Published Work in My Bibliography:
https://www.ncbi.nlm.nih.gov/pubmed/?term=salisbury+tb

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

Ongoing Research Support

Edwards Cancer Foundation, Edwards Comprehensive Cancer Center (ECCC), Marshall University, School of Medicine 2017. The goal of this clinical grant is to investigate the regulation and function of nutrient transporters and mTOR1 in breast cancer tissue from obese compared with lean women undergoing treatment for their breast cancer at the ECCC.
Salisbury (PI)

WV-INBRE Next Generation Sequencing pilot grant, Marshall University 2016-2017
The goal of this projected is to define adipocyte regulated genes in human breast cancer cells that are also aryl hydrocarbon receptor gene targets.
Salisbury (PI)

Completed Research Support

Marshall University School of Medicine Pilot Award, Marshall University 2014-2015
The goal of this pilot award was to investigate whether adipocyte-secreted factors promote breast cancer by increasing the regulation and function of amino acid transporters
Salisbury (PI)

Research Starter Grant from the Pharmaceutical Manufacturers Association of America 2012-2013
The goal of this project was to investigate the role of the aryl hydrocarbon receptor in breast cancer cells stimulated by the adipocyte-derived secretome.
Salisbury (PI)

WV-INBRE next generation sequencing challenge grant, Marshall University 2012-2013
The goal of this project was to establish signal-induced aryl hydrocarbon receptor gene targets in human breast cancer cells.
Salisbury (PI)

WV-INBRE next generation sequencing challenge grant, Marshall University 2011
The objectives of this proposal was to identify genome-wide AHR binding sites in breast cancer cells
Salisbury (PI)

Cell Differentiation and Development Center (CDDC) grant, Marshall University 2011-2012

The objectives of this proposal was to investigate gene expression effects of endocrine disrupting chemicals in ovarian cells.
Salisbury (PI)