BIOGRAPHICAL SKETCH

NAME: Leesa LaFever Sampson, Ph.D.

eRA COMMONS USER NAME: lafevelm

POSITION TITLE: Senior Staff Scientist

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Western Kentucky University	B.S.	06/2003	Biology
Vanderbilt University	Research Assistant	07/2005	Cell Signaling and Invertebrate Stem Cells
Vanderbilt University	Ph.D.	12/2010	Cell and Developmental Biology
Cincinnati Children's Hospital Medical Center		3/2017	Cell Signaling and Mammalian Stem Cells
Vanderbilt University		Current	Stem Cell Biology/Genome Engineering

A. Personal Statement

I have a solid scientific education in Cell and Developmental Biology with an emphasis on Cell Signaling with thirteen years of overall experience in invertebrate and mammalian stem cell *in vivo* regulatory studies. I have accumulated multiple honors throughout my scientific career including multiple NIH training grants, a NIDDK F32 NRSA award, and an internal Digestive Health grant through the Cincinnati Children's Digestive Health Center. I have attended and presented my research in poster and oral formats at national and international conferences. I have an active interest in mentoring and have taught undergraduate students in the classroom and mentored high-school, undergraduate, and graduate students in the laboratory. My current position in the Center for Stem Cell Biology at Vanderbilt University will focus on generating new genetic tools using state of the art genome modification technologies for local researchers.

B. Research and/or Professional Experience

Employment

1. **Research Assistant:** *June 2003-July 2005*: Vanderbilt University, Department of Cell and Developmental Biology, Daniela Drummond-Barbosa, PhD. Full time.
2. **PhD Student:** *July 2005-July 2009*: Vanderbilt University, Department of Cell and Developmental Biology, Daniela Drummond-Barbosa, PhD. Full time.
3. **PhD Student:** *July 2009-December 2010*: Johns Hopkins School of Public Health, Department of Biochemistry and Molecular Biology, Daniela Drummond-Barbosa, PhD. Full time.

4. **Postdoctoral Fellow:** *January 2011-April 2011*: University of Cincinnati, Department of Cancer and Cell Biology, George Thomas, PhD. Full time. *(G.Thomas moved his main lab to Spain shortly after I joined the group.)*
5. **Postdoctoral Fellow:** *April 2011-December 2014*: Cincinnati Children's Hospital Medical Center, Department of Experimental Hematology and Cancer Biology, Yi Zheng, PhD. Full time.
6. **Research Associate:** *January 2015-March 2017*: Cincinnati Children's Hospital Medical Center, Department of Experimental Hematology and Cancer Biology, Yi Zheng, PhD. Full time.
7. **Senior Staff Scientist**: *March 2017-current*: Vanderbilt University, Center for Stem Cell Biology, Mark Magnuson, PhD. Full time.

Honors

1. **Award of Excellence:** 1999- 2003, Western Kentucky University, full tuition and board
2. **Basil C. Cole Biology Scholarship:** 2002-2003, Western Kentucky University
3. ***Magna Cum Laude* B.S. in Biology:** 2003, Western Kentucky University
4. **Program in Developmental Biology Retreat Poster Presentation Award:** 2004, Vanderbilt University
5. **Developmental Biology NIH T32 Training Grant:** 2006-2008, Vanderbilt University
6. **Reproductive Biology NIH T32 Training Grant:** 2008-2009, Vanderbilt University
7. **Cancer Therapeutics NIH T32 Training Grant:** 2011-2012, University of Cincinnati
8. **Travel Grant Award for James W. Freston Conference in Gastrointestinal Stem Cell Biology and Pathobiology:** 2012
9. **NIH National Institute of Diabetes, Digestive, and Kidney Disease Ruth L. Kirschstein NRSA F32. "mTOR signaling in murine intestinal stem cell and progenitor homeostasis":** 2012-2014
10. **Cincinnati Children's Hospital/University of Cincinnati Digestive Health Center Annual Symposium 2nd Place Poster Presentation:** 2013
11. **Cincinnati Children's Hospital Digestive Health Center Pilot and Feasibility Grant** with co-PI's, Yi Zheng and Noah Shroyer: 2013-2014

Professional Societies and Public Advisory Committees

1. **American Association for the Advancement of Science:** 2006-2007
2. **International Society for Stem Cell Research:** 2013-2014
3. **American Gastrological Association:** 2012-2015

C. Contributions to Science

1. Identifying the first direct role for insulin in regulating ovarian tissue.

In the early 2000s, it was understood that diseases like diabetes and obesity lead to fertility problems such as polycystic ovarian syndrome, but mainstream thought was that insulin indirectly regulates ovarian function. As a research assistant in the lab of Dr. Daniela Drummond-Barbosa at Vanderbilt University we used *Drosophila* genetic techniques to remove the single insulin receptor gene from the fly ovary, which is sustained by germline stem cells, and demonstrated that insulin signaling directly stimulates germline stem cell growth and proliferation. Later studies showed similar direct roles for insulin on mammalian ovarian follicles indicating conservation of function. As a research assistant in 2005 and young graduate student in 2008, my contribution to these studies was largely technical in nature. I helped design experiments, acquired and analyzed data, and generated figures.

2008 Hsu, H.J.*, **LaFever, L.***, and Drummond-Barbosa, D. 2008. Diet controls normal and tumorous germline stem cells via insulin-dependent and -independent mechanisms in *Drosophila*. *Developmental Biology* **313**(2): 700-712.

*These authors contributed equally

2005 **LaFever, L.** and Drummond-Barbosa, D. 2005. Direct control of germline stem cell division and cyst growth by neural insulin in *Drosophila*. *Science* **309**(5737): 1071-1073.

2. Uncovering specific roles for the highly-conserved mTOR kinase in different *Drosophila* ovary stem cell types.

In the mid-2000s, little was known about the role of vital genes such as Target of Rapamycin (TOR) kinase in individual cell types because its disruption is embryonically lethal. From studies in cell culture, TOR appeared vital for cell survival and was identified as an obvious target for cancer therapies. As a PhD candidate in the lab of Daniela Drummond-Barbosa at Vanderbilt University, I used the *Drosophila* ovary model to conditionally remove dTOR (*Drosophila* TOR) kinase from either the germline stem cells (germline cell type) or follicle stem cells (epithelial cell type). We found that while dTOR is critical for germline stem cells to generate eggs, follicle stem cells could still generate daughter cells, indicating dTOR was less critical in this lineage. Later conditional studies in other tissues in both invertebrates and mammals showed that TOR was indeed playing tissue-specific roles and underlined the importance of examining protein function in different contexts. My role in this study involved study design, performing most experiments, analyzing data, preparing figures, and assisting with writing the manuscript.

2010 **LaFever, L**., Feoktistov, A., Hsu, H.J., and Drummond-Barbosa, D. 2010. Specific roles of Target of rapamycin in the control of stem cells and their progeny in the *Drosophila* ovary. *Development* **137**(13): 2117-2126.

3. Discovering stress-specific roles for mTOR kinase in stem and progenitor cells of the mammalian small intestine.

When I began my postdoctoral project in 2011, it was unclear if mammalian intestinal stem cells, recently characterized by new molecular markers like *Lgr5*, were nutrient and stress-responsive. In the laboratory of Yi Zheng at Cincinnati Children's Hospital, I aimed to address this deficiency, and potentially explain why mTOR (mechanistic Target of Rapamycin) inhibitor therapies were not proving useful for gastrointestinal cancers, by using an intestinal epithelial-specific disruption mouse model for mTOR kinase, the key kinase of the core intracellular nutrient and stress-sensing pathway. Our studies unveiled that mTOR is critical for stem cell-based regeneration under situations of injury, but not during homeostasis. Our studies suggest that mTOR disruption would require additional injury stress input to impair intestinal stem cell population efficiently. My contribution to this study was at the level of an independent investigator with critical guidance and input from my mentor and other gastrointestinal researchers at Cincinnati Children's Hospital. I designed the study, performed most experiments or organized technical assistance, analyzed data, prepared figures, wrote the manuscript, submitted for publication, and handled the rebuttal/revision process of peer review with assistance from my PI.

2015 **Sampson, L.L.,** Davis, A.K., Grogg, M.W., Zheng Y. mTOR disruption causes intestinal epithelial cell defects and intestinal atrophy post-injury in mice. *FASEB J*, doi: 10.1096/fj.15-278606 (2015).

http://www.ncbi.nlm.nih.gov/sites/myncbi/leesa.sampson.1/bibliograpahy/49246105/public/?sort=date&direction=ascending

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