## BIOGRAPHICAL SKETCH

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NAME: Quentin Felty

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

POSITION TITLE: Associate Professor

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 |
|---|---|---|---|
| University of Washington, Seattle | BS | 06/94 | Biology |
| Univ. of Alabama at Birmingham | MSPH | 06/98 | Environmental Toxicology |
| Univ. of Alabama at Birmingham | MA | 12/00 | Education |
| Univ. of Alabama at Birmingham | PhD | 06/04 | Environmental Health Sciences |

### A. Personal Statement

As a Prinicipal Investigator, I have been successful in obtaining grants and conducting extramurally funded research projects. I have served as PI on over $1M in external funding and over $1.5M as a co-PI. These accomplishments are testimony to a track record of excellent collaborative and team building ability. Our research program has a record of successful and productive research projects in the area of molecular toxicology studies of the adverse effects of exposure to estrogenic chemicals; identification of redox signaling molecules; and role of reactive oxygen species in breast cancer and vascular cell proliferative disorders that impact cancer invasiveness. The primary focus of our research is to elucidate how estrogenic and/or environmental estrogenic chemicals activate ID3 and influence the susceptibility of vascular lesion formation – estrogen is a major risk factor for vascular lesions in pulmonary arterial hypertension, and to determine whether an inhibitor of ROS and/or ID3 could prevent the development of plexiform vascular lesions. Our laboratory has found that reactive oxygen species (ROS) are important signaling molecules in the development of PCB-induced non-malignant vascular lesions. We have the necessary expertise to assist in the proposed research with regard to redox signaling and induced pluripotent stem cells, which is key to the performance of the project. In summary, our laboratory has demonstrated a record of successful and productive research projects in the area of redox cell signaling, estrogenic induced growth of cancer and vascular lesions.


1. Das J.K., Voelkel, N.F., and **Felty Q**. ID3 contributes to the acquisition of molecular stem cell-like signature in microvascular endothelial cells: Its implications for understanding microvascular diseases. *Microvascular Research,* 98:126-138 (2015). doi:10.1016/j.mvr.2015.01.006
2. Al-Husseini A., Kraskauskas D., Mezzaroma E., Nordio A., Farkas D., Drake J.I., Abbate A., **Felty Q.**, Voelkel N.F. Vascular endothelial growth factor receptor 3 signaling contributes to angioobliterative pulmonary hypertension. *Pulmonary Circulation*, 5(1):101-116 (2015). doi:10.1086/679704.
3. Das J.K. and **Felty Q**. Microvascular Lesions by Estrogen-Induced ID3: Its Implications in Cerebral and Cardiorenal Vascular Disease. *J Mol Neurosci*., 55(3):618-31 (2015). doi: 10.1007/s12031-014-0401-9
   Das J.K. and **Felty Q**. PCB153-Induced Overexpression of ID3 Contributes to the Development of Microvascular Lesions. *PLoS One*, 9(8):e104159. (2014). doi: 10.1371/journal.pone.0104159
4. **Felty, Q.**, Sakao, S., and Voelkel N.F. Pulmonary Arterial Hypertension: A Stem Cell Hypothesis, Chapter 16, In *Lung Stem Cells in the Epithelium and Vasculature*, Editors: Amy Firth, Jason X.-J. Yuan. Springer International Publishing, Switzerland. ISBN: 978-3-319-16231-7, p. 289-306. (2015) doi:10.1007/978-3-319-16232-4

**B. Positions and Honors**

**Positions and employment**

8/11-present Associate Professor, Env & Occupational Hlth, Florida International University (FIU), Miami, FL
8/05 - 7/11 Assistant Professor, Env & Occupational Hlth, Florida International University (FIU), Miami, FL
7/04 - 8/05 Postdoctoral Fellow, FIU School of Public Health, Miami, FL.
6/01 - 6/04 Research Assistant, UAB Dept. of Environmental Health Sciences, Birmingham, AL.
2000-2004 NCI Cancer Prevention and Control Training Program Fellow

**Other experience and Professional Memberships**

**Member of Review Panels**

2015 Member, the Basic Cell, Cell Structure & Survival 2 Peer Review Committee, American Heart Association (AHA) Spring and Fall.

2009 Member, Endocrinology peer review panel of the Breast Cancer Research Program for the Department of Defense (DoD) Congressionally Directed Medical Research Programs (CDMRP).

2008-2010 Member, Molecular Signaling, Region II Peer Review Committee Meeting, American Heart Association

2007 Member, Molecular & Cellular Biology & Genetics Review Panel, Susan G. Komen Breast Cancer Foundation

2007 Member, Epi, Exposure Assessment & Lab Methods Review Panel, National Institute for Occupational Safety and Health (NIOSH)

2006 Member, Intramural Program Grant Review Panel, National Institute for Occupational Safety and Health (NIOSH)

**Professional Memberships**

American Association for Cancer Research (AACR)-Full member
Society of Toxicology (SOT)-Full member

**Honors**

2014 University Presidential Recognition for Faculty Research, Dr. Felty was recognized with a selected group of 12 outstanding faculty researchers by FIU President Mark Rosenberg.

2013 American Association for Cancer Research MSI Faculty Scholar in Cancer Research Award

2010 FIU Top Scholars Award (FIU Presidential Recognition for Excellence in Faculty Scholarship 2010)

2011 Plenary session organizer and co-chair; Modeling of Gene-Environment Interactions to Analyze the Susceptibility and Progression of Chronic Human Diseases for the International Congress on Modeling and Simulation (MODSIM 2011).

2011 Editorial Board Member for the journal PLoS ONE

2011 Editorial Board Member for the Journal of Environmental & Analytical Toxicology

2004 Graduate Travel Award from the Society of Toxicology

2000-04 NCI Cancer Prevention and Control Training Fellowship

**C. Contribution to Science**

The primary focus of my research program is characterizing underlying genetic, epigenetic and environmental factors that contribute to variability in human responses to environmental estrogenic chemicals and development of complex human diseases. Pulmonary hypertension is roughly twice as common in women compared to men. Severe pulmonary hypertension is characterized by clustered proliferation of endothelial cells in the lumina of small size pulmonary arteries resulting in the formation of complex vascular structures known as plexiform lesions. Whether the formation of these vascular lesions is caused by estrogen or environmental pollutants that mimic estrogens is not known. Animal studies in models of pulmonary hypertension and scant clinical data suggest that estrogens are protective. Yet, the long-term use of estrogen therapy has been shown to increase the risk of cardiovascular disease in women. The dose of estrogens reportedly used in animal models and clinically may offer a potential explanation for the estrogen paradox. Clinically, estrogen is given at a "low dose" to minimize thrombotic risk and hormone-dependent malignancies. However, few *in vitro* and *in vivo* studies have studied the adverse effects of low dose estrogen exposure. Since estrogen is a known mitogen of endothelial cells that promotes vessel formation, exogenous estrogen exposure in the form of hormone therapy for contraception or management of menopause may support the angioproliferative endothelial phenotype. Therefore, our overall research goal is to investigate the molecular mechanism by which estrogenic chemicals may promote vascular lesion formation. By elucidating the relationship between exposure to the estrogens and dysregulation of signaling that leads to vascular lesions, and by providing methods to assess early events in the hazards associated with estrogen and environmental estrogenic compounds, the research will provide public health and environmental toxicology an important understanding of the mechanisms and biomarkers necessary for the assessment of estrogen exposure-associated vascular hazards in humans.

My early publications directly addressed estrogen-induced mitochondrial ROS signaling in cancer.

1. **Felty, Q.**, Singh, K. P., and Roy, D. Estrogen-induced G1/S transition of G0-arrested estrogen-dependent breast cancer cells is regulated by mitochondrial oxidant signaling, *Oncogene* 24, 4883-4893. (2005) doi:10.1038/sj.onc.1208667
2. **Felty, Q.**, Xiong, W. C., Sun, D., Sarkar, S., Singh, K. P., Parkash, J., and Roy, D. Estrogen-induced mitochondrial reactive oxygen species as signal-transducing messengers, *Biochemistry* 44, 6900-6909. (2005) doi:10.1021/bi047629p

I have extended my ROS signaling research to the area of vascular toxicity of endocrine disrupting chemicals.

1. **Felty, Q.** Gene Environment Interactions and Vascular Lesions, Chapter 6*,* In *Environmental Factors, Genes, and the Development of Human Cancers,* Springer Press, New York, USA. ISBN 978-1-4419-6751-0. p. 139-152 (2010).
2. **Felty, Q.** Proteomic 2-D DIGE profiling of human vascular endothelial cells exposed to environmentally relevant concentration of endocrine disruptor PCB153 and physiological concentration of 17β-estradiol. *Cell Biol Toxicol,* 27(1):49-68, (2011). doi:10.1007/s10565-010-9170-6

**Complete List of Published Work in My Bibliography**

http://www.ncbi.nlm.nih.gov/sites/myncbi/quentin.felty.1/bibliograpahy/46514045/public/?sort=date&direction=descending

**D. Research Support**

**Active Research Support**

**Felty, Q.** (Principal Investigator) 05/18/16-05/18/17
FIU BSI Collaborative Sandbox Team Seed Funding Program **$10,000**
Title: "PCBs in concert with ID3 epigenetically drives the formation of non-malignant vascular lesions in lung"

**Completed Research Support (Last 3 Years)**

**Felty, Q.** (Principal Investigator) 01/01/15-12/31/15
NIH, SC3 Award (1SC3GM084827-01A1) **No cost extension**
Title: "Estrogen-induced Pyk2 signaling in the abnormal growth of vascular cells"

**Felty, Q.** (Principal Investigator) 01/01/10-12/31/14
NIH, SC3 Award (1SC3GM084827-01A1) **$760,500**

Title: “Estrogen-induced Pyk2 signaling in the abnormal growth of vascular cells”

**Felty, Q.** (Principal Investigator) 07/01/09-06/30/13
Florida Department of Health, Bankhead-Coley Award (09BN-06) **$375,000**
Title: “Metastases and Promotion of Aggressive Angiogenic Phenotype in Breast Cancer”