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## BIOGRAPHICAL SKETCH

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NAME Arendt, Lisa Marie	POSITION TITLE Assistant Professor		
eRA COMMONS USER NAME (credential, e.g., agency login) LMARENDT			
<i>EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)</i>			
INSTITUTION AND LOCATION	DEGREE <i>(if applicable)</i>	MM/YY	FIELD OF STUDY
University of Wisconsin, Madison	B.S.	1996	Genetics
University of Wisconsin, Madison	D.V.M.	2002	Veterinary Medicine
University of Wisconsin, Madison	Ph.D.	2007	Cell and Molec. Biology
Tufts University, Boston		2014	Postdoctoral Training

### A. Personal Statement

My research goal is to investigate how stromal changes under conditions of obesity alter epithelial cell populations in the normal breast and during tumorigenesis. Utilizing adipose stromal cells isolated from reduction mammoplasty tissues from lean and obese women and well as from mammary glands of obese and lean mice, I will examine changes in primary breast epithelial cell populations in during normal development as well as during tumor development in vivo, with complementary in vitro models. With my expertise in veterinary medicine, I have a strong background in cancer biology, pathology and animal models to utilize in my research. During both my thesis work and my postdoctoral training, I studied breast cancer and mammary gland development using animal models. In particular during my postdoctoral training, I worked frequently with the human in mouse (HIM) modeling technique, which provides a unique model to observe preneoplastic progression to disorganized tumors. My previous studies in this area have examined how obesity alters macrophage activation and angiogenesis using a high fat diet model in mice as well as a novel HIM model. I have successfully published in the area of obesity-associated breast cancer, as well as multiple other peer-reviewed articles in the field of breast cancer research.

### B. Positions and Honors

#### Positions and Employment

2002-2008	Research Assistant, UW-Madison, School of Veterinary Medicine
2004	Laboratory Instructor, Veterinary Histology, UW-Madison, School of Veterinary Medicine
2008-2014	Senior Research Associate, Department of Anatomy, Tufts University
2014-2015	Research Assistant Professor, DMCB Department, Tufts University
2015-	Assistant Professor, Department of Comparative Biosciences, UW-Madison

#### Other Experience and Professional Memberships

2002-	Member, American Veterinary Medical Association
2002-	Member, Wisconsin Veterinary Medical Association
2004-	Member, Endocrine Society
2004-	Member, American Association for Cancer Research
2014	Chair, Gordon Research Symposium for Mammary Gland Biology, Barga, Italy

#### Honors

1994-1998	Wisconsin All-State Scholar
1994-1998	Proctor and Gamble Scholarship
1996	Phi Kappa Phi
1997	Golden Key National Honors Society
1999	Meril Summer Student Research in Biomedical Sciences Fellowship
2000	Excellence in Student Research Award
2013	Tufts Medical Cancer Center Poster Award Recipient
2014	Cover art competition winner, Journal of Endocrinology

### C. Peer-reviewed Publications

1. Rose-Hellekant TA, Arendt LM, Schroeder MD, Gilchrist K, Sandgren EP, Schuler LA. Prolactin induces ER alpha positive and ER alpha negative mammary cancer in transgenic mice. *Oncogene*, 2003; 22: 4664-4574.
2. Arendt LM, Rose-Hellekant TA, Sandgren EP, Schuler LA. Prolactin potentiates transforming growth factor alpha induction of mammary neoplasias in transgenic mice. *Am J Path*, 2006; 168:1365-1374.
3. Arendt LM, Schuler LA. Prolactin drives ERa-dependent ductal expansion and synergizes with TGFa to induce mammary tumors in males. *Am J Path*, 2008; 172:194-202.
4. Arendt LM, Schuler LA. Transgenic models to study actions of prolactin in mammary neoplasia. *J Mammary Gland Biol Neoplasia*, 2008; 13:29-40.
5. Arendt LM, Grafwallner-Huseth TL, Schuler LA. Prolactin-growth factor crosstalk reduces mammary estrogen responsiveness despite elevated ERa expression. *Am J Path*, 2009; 174: 1065-1074.
6. Arendt LM, Evans LC, Rugowski DE, Garcia-Barchino MJ, Rui H, Schuler LA. Ovarian hormones are not required for PRL-induced mammary tumorigenesis, but estrogen enhances neoplastic processes. *J Endocrinology*, 2009; 203: 99-110.
7. Carver KC, Arendt LM, Schuler LA. Complex prolactin crosstalk in breast cancer : new therapeutic implications. *Mol Cell Endocrinol*, 2009; 307: 1-7.
8. Arendt LM, Rudnick JA, Keller PJ, Kuperwasser C. Stroma in breast development and disease. *Semin Cell Dev Biol*, 2010; 21: 11-18.
9. McCreedy J, Arendt LM, Rudnick JA, Kuperwasser C. The contribution of dynamic stromal remodeling during mammary development to breast carcinogenesis. *Breast Cancer Res*, 2010; 12: 205.
10. Jeselsohn R, Brown NE, Arendt LM, Klebba I, Hu MG, Kuperwasser C, Hinds PW. Lobular progenitor cells are the cellular targets of MMTV-ErbB2 tumorigenesis and require cyclin D1 for their maintenance. *Cancer Cell*, 2010; 17: 65-76.
11. Keller PJ, Lin AF, Arendt LM, Klebba I, Jones AD, Rudnick JA, Dimeo TA, Gilmore H, Jefferson DM, Graham RA, Naber SP, Schnitt S, Kuperwasser C. Mapping the cellular and molecular heterogeneity of normal and malignant breast tissues and cultured cell lines. *Breast Cancer Res*, 2010; 12:R87.
12. Arendt LM, Rugowski DE, Grafwallner-Huseth TA, Garcia-Barchino MJ, Rui H, Schuler LA. Prolactin-induced mouse mammary carcinomas model estrogen resistant luminal breast cancer. *Breast Cancer Res*, 2011; 13: R11.
13. Chaffer CL, Brueckmann I, Scheel C, Kaestli AJ, Wiggins PA, Rodrigues LO, Brooks M, Reinhardt F, Su Y, Polyak K, Arendt LM et al. Normal and neoplastic nonstem cells can spontaneously convert to a stem-like state. *PNAS*, 2011; 108: 7950-7955.
14. Keller PJ, Arendt LM, Kuperwasser C. Stem cell maintenance of the mammary gland: it takes two. *Cell Stem Cell*, 2011; 9:496-497.
15. Rudnick JA, Arendt LM, Klebba I, Hinds JW Iyer V, Gupta PB, Naber SP, Kuperwasser C. Functional heterogeneity of human breast fibroblasts can be defined by a prostaglandin secretory phenotype that promotes expansion of cancer-stem like cells and tumor growth. *PlosOne*, 2011; 6: e24605.
16. Asher JM, O'Leary KA, Rugowski DE, Arendt LM, Schuler LA. Prolactin promotes mammary pathogenesis independently from cyclin D1. *Am J Path*, 2012; 181:294-302.
17. Keller PJ, Arendt LM, Skibinski A, Logvinenko T, Klebba I, Dong S, Gilmore H, Schnitt S, Naber S, Garlick J, Kuperwasser C. Defining the cellular precursors to human breast cancer. *PNAS*, 2012; 109: 2772-2777.

18. Iyer V, Klebba I, McCready J, Arendt LM, Betacur-Boissel M, Wu MF, Zhang X, Lewis MT, Kuperwasser C. Estrogen promotes ER-negative tumor growth and angiogenesis through mobilization of bone-marrow derived monocytes. *Cancer Res*, 2012; 72: 2705-2713.
19. Arendt LM, McCready J, Keller PJ, Naber SP, Baker DB, Seewaldt V, Kuperwasser C. Obesity promotes breast cancer by CCL2-mediated macrophage recruitment and angiogenesis. *Cancer Res*, 2013; 73: 1-14.
20. McCready J, Arendt LM, Glover E, Iyer V, Lyle S, Naber SP, Jay DG, Kuperwasser C. Adipose stromal cells present during lactation promote angiogenesis and breast tumor growth. *Breast Cancer Res*, 2014; 16: R2.
21. Arendt LM, Keller PJ, Skibinski A, Goncalves K, Naber SP, Kuperwasser C. Immature lobules of the human breast are enriched for progenitor cells. *Breast Cancer Res*, 2014; 16:453.
22. Arendt LM, St. Laurent J, Keller PJ, Callebero S, Lyle S, Naber SP, et al. Hormonal and non-hormonal regulation of distinct lineage-committed progenitor cells in the human breast requires WNT and TBX3. *PlosOne*, 2014; 9: e111442.
23. Mazumdar S\*, Arendt LM\*, Philips S, Sedic M, Kuperwasser C, Gill G. CoREST1 promotes tumor formation and tumor stroma interactions. *PlosOne*, 2015; 10:e0121281. \*These authors contributed equally to this work.
24. Schubert SM, Arendt LM, Zhou W, Baig S, Walter SR, Buchsbaum RJ, et al. Ultra-sensitive protein detection via Single Molecule Arrays towards early stage cancer monitoring. *Science Reports*, 2015; 5:11034.
25. Wronski A, Arendt LM, Kuperwasser C. Humanization of the mouse mammary gland. *Methods Mol Biol*, 2015; 1293:173-186.
26. Arendt LM and Kuperwasser C. Form and function: how estrogen and progesterone regulate the mammary epithelial hierarchy. *J of Mammary Gland Biol and Neoplasia*, 2015; in press.

## D. Research Support

### Current Research Support

Komen Foundation      Arendt (PI)      7/1/15-6/30/18 (in progress)

Obesity-Activated Macrophages Promote Tumorigenesis

Career Catalyst Award: The goal of this study is to investigate how macrophages, activated in the obese microenvironment, secrete cytokines that alter epithelial cell populations in the normal breast and promote the formation cancer stem-like cells during tumorigenesis.

### Completed Research Support

Expedition Inspiration      Arendt (PI)      10/1/13-1/31/15

Obesity-Activated Macrophages Promote Tumorigenesis

Brenda Williams Young Investigator Award: The goal of this study was to determine how macrophages polarized by obese mammary tissue contribute to the tumor microenvironment and elucidate how obesity-associated macrophages promote aggressive breast tumors.

Breast Cancer Alliance      Arendt (Co-PI)      1/1/12-12/31/12

Mechanisms of Breast Cancer Progression Associated with Obesity

Exceptional Project Grant: The goal of this study was to determine the role of macrophages in angiogenesis in obesity-associated breast cancer and assess small molecule inhibitors to prevent early tumor development.

5K01RR021858-05      Arendt (PI)      4/1/06-3/31/11

Interactions of PRL, Estrogen, and TGF $\alpha$  in Mammary Cancer

NCRR-SERCA: The goal of this study was to use transgenic mouse models to understand the interactions of prolactin (PRL), estrogen, and transforming growth factor alpha (TGF $\alpha$ ) in the formation of preneoplastic lesions and progression to malignancy in breast cancer.