OMB No. 0925-0001 and 0925-0002 (Rev. 09/17 Approved Through 03/31/2020)

BIOGRAPHICAL SKETCH

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NAME: Jaekwang Jeong

eRA COMMONS USER NAME (credential, e.g., agency login): jjeong1230

POSITION TITLE: Associate Research Scientist

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 |
| --- | --- | --- | --- |
| Pukyung National University, Pusan, South Korea | B.S. | 02/2002 | Microbiology |
| Pukyung National University, Pusan, South Korea | M.S. | 02/2004 | Microbiology |
| University of Maryland, College Park | Ph.D. | 05/2011 | Cell Biology |
| Yale University School of Medicine | Postdoctoral | 03/2017 | Cancer Cell Biology |
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**A. Personal Statement**

I have been trained in broad areas from fundamental cell and molecular biology to advanced cancer biology. During my Ph.D. period, I found a unique mechanism in secretory epithelial cells in the lactating mammary gland. The formation of the milk-lipid globule membrane is driven by interactions between the membrane protein, butyrophilin (BTN), and the redox enzyme, xanthine oxido-reductase (XOR) at the apical surface. Therefore, these training provided me a strong background in the cell biology. My goal is to establish an independent research lab focused on how the dysregulation of normal breast biology contributes to breast cancer. Therefore, I joined Dr. Wysolmerski’s laboratory as his research has focused on how breast cells handle calcium signaling both during lactation and in cancers. I advanced my research interest into cancer cell biology. Initially, my research has been focusing on the PMCA2, a calcium pump protein, and NHERF1, a PDZ domain containing a scaffolding protein, and I found a novel HER2 driven protein-protein interaction which includes HER2, PMCA2, NHERF1, Ezrin, and actin cytoskeleton. This protein complex is important for HER2 signaling and membrane retention which causes malignant transformation in transgenic mice. The discovery of this new complex and pathway will stimulate the development of novel drugs that would be effective treatments for HER2-positive tumors either alone or in combination with established therapies.

**B. Positions and Honors  
Professional experience:**

2017-Present Associate Research Scientist, Department of Internal Medicine, Yale School of Medicine

2013-2015 NIH Postdoctoral Training Fellowship, Yale School of Medicine

2012-2013 Postdoctoral Fellow, Yale School of Medicine

2011-2012 NIH Postdoctoral Training Fellowship, Yale School of Medicine

**Honors**

2012-2013 James Hudson-Alexander Brown Coxe Fellowship award, Yale School of Medicine

2010 Best Poster presentation Bioscience Day, University of Maryland, College Park

2008 Best Poster presentation MOCB annual symposium, University of Maryland, College Park

**C. Contributions to Science**

**1. Defined the role of PMCA2, NHERF1, and Ezrin in breast cancer.** PMCA2 is highly expressed calcium pump in the HER2 positive breast cancer. PMCA2 is over-expressed in breast cancer cells and we showed that high levels of PMCA2 expression are an independent predictor of death in a large cohort of patients with breast cancer. Our recent studies expanded that PMCA2 interacts with the scaffolding proteins, NHERF1 and Ezrin, and this interaction regulates ErbB2 localization and signaling in breast cancers.

1. **Jeong J**, Choi J, Kim W, Takyar FM, Dann P, Wysolmerski JJ. (2018) Inhibition of Ezrin Causes PKCa -mediated Internalization and Subsequent Degradation of ErbB2/HER2 in Breast Cancer Cells. ***J. Biol. Chem*.** 294(3), 887-901
2. **Jeong J**, Kim W, Kim LK, Vanhouten J, Wysolmerski JJ. (2017) HER2 signaling regulates HER2 localization and membrane retention. ***PloS One***, 3;12(4):e0174849
3. **Jeong J**, Vanhouten JN, Kim W, Dann P, Sullivan C, Choi J, Sneddon WB, Friedman PA, Wysolmerski JJ. (2017) The Sca\_olding protein NHERF1 Regulates the Stability and Activity of the Tyrosine Kinase HER2**. *J. Biol. Chem*.** Apr 21;292(16):6555-6568
4. **Jeong J**, VanHouten JN, Dann P, Kim W, Sullivan C, Yu H, Liotta L, Espina V, Stern DF, Friedman PA, Wysolmerski JJ. (2016) PMCA2 regulates HER2 protein kinase localization and signaling and promotes HER2-mediated breast cancer. ***Proc Natl Acad Sci U S A*.** 113(3):E282-90

**2. Defined the role of MAL2, a lipid raft resident protein, in breast cancer.** MAL2-mediated lipid raft formation is required for the retention of an intact HER2 signaling complex at the plasma membrane as well as enhanced HER2 signaling in breast cancer cells.

1. **Jeong J**, Shin JH, Li W, Hong JY, LimJ, Hwang JY, Chung JJ, Yan Q, Liu Y, Choi J, WysolmerskiJJ. MAL2 mediates the formation of stable HER2 signaling complexes within lipid raft-rich membrane protrusions in breast cancer cells. (2020) Manuscript in Preparation. **Corresponding Author.**

**3. Defined the role of PMCA2, NHERF1, and Ezrin in normal mammary gland.** PMCA2 and NHERF1 expressed highly in the lactating mammary gland. We identified that PMCA2 and NHERF1 levels decrease during mammary involution and that loss of PMCA2 acts as an important trigger for mammary gland involution after weaning. We identified that the interaction between PMCA2 and NHERF1 is critical for controlling the intracellular calcium levels during lactation.

1. Grinman D, Athonvarungkul D, Wysolmerski JJ, **Jeong J** (2020) Calcium Metabolism and Breast Cancer; Echoes of Lactation? ***Current Opinion in Endocrinology and Metabolic Research***, Under Review
2. **Jeong J**, Kim W, Hens J, Dann P, Schedin P, Friedman PA, Wysolmerski JJ. (2019) NHERF1 is required for proper localization of PMCA2 and suppression of early involution in the lactating mammary gland. ***Endocrinology*,** en.2019-00230

**4. Demonstrated the mechanism of lipid droplet secretion during lactation of mammary gland.** The interactions between butyrophilin 1a1 (BTN1a1) and xanthine oxido-reductase (XOR) at the apical surface is critical for secretion of lipid droplets during lactation. We found that loss of BTN1A1 causes defect of lipid droplet secretion during lactation.

1. **Jeong J**, Schaack J, Jenkins LM, Dinan JC, Weigert R, Mather IH. Btn1a1 and XDH function in milk-lipid secretion. (2020) Manuscript in Preparation.
2. **Jeong J**, Kadegowda AKG, Meyer TJ, Jenkins L, Dinan JC, Weigert R, Mather IH. (2020) The butyrophilin 1a1 (Btn1a1) knock out mouse revisited; Ablation of Btn1a1 leads to concurrent cell death and renewal in the mammary epithelium during lactation. Manuscript in Preparation.
3. **Jeong J**, Lisinski I, Kadegowda AK, Shin H, Wooding FB, Daniels BR, Schaack J, Mather IH. (2013) A test of current models for the mechanism of milk-lipid droplet secretion. ***Traffic***. 14(9):974-86
4. **Jeong J**, Rao, A.U., Xu, J., Ogg, S.L., Hathout, Y., Fenselau, C., and Mather I.H. (2009) The PRY/SPRY/B30.2 domain of butyrophilin 1A1 (BTN1A1) binds to xanthine oxidoreductase: Implication for the function of BTN1A1 in the mammary gland and other tissues. ***J. Biol. Chem*.** 284: 22444-22456

**3. Other co-author papers**

**a)** Kim W, Takyar FM, Swan K, **Jeong J**, VanHouten J, Sullivan C, Dann P, Yu H, FiaschiTaesch N, Chang W, Wysolmerski J. (2016) Calcium-Sensing Receptor Promotes Breast Cancer by Stimulating Intracrine Actions of Parathyroid Hormone-Related Protein. ***Cancer Res*.** Sep 15;76(18):5348-60.