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Major Professor: Dr. Jing Zheng

Degree Objective: Ph.D. Endocrinology and Reproductive Physiology

Background:

2006 to 2011, Bachelor of Medicine, Bachelor of Surgery, Harbin Medical University, China

2011 to present, Ph.D. candidate, University of Wisconsin-Madison, United Sates

Current Research Project: During pregnancy, fetal and placental vasculatures undergo dramatic growth and remodeling to accommodate the remarkable increases in fetal and placental blood flows required for supporting the developing fetus. During these processes, vascular endothelial cells reside under low oxygen environments (~2-13% O2 or pO2 ~17-100 mmHg). This physiological chronic hypoxia (pCH) is critical for cell growth and development. Vascular Endothelial Growth Factor-A (VEGF) and Fibroblast Growth Factor-2 (FGF2) actively regulate many endothelial functions (e.g., proliferation, migration, and vasodilator production). Actions of VEGF and FGF2 are mediated via a serial of protein kinases (e.g., ERK1/2, Akt1, and p38 MAPK) and also via heterotrimeric GTP-binding proteins (G proteins). G protein subunit α-11 & 14 (GNA11 & 14) as transducer participates in various transmembrane and intracellular signaling systems, including phospholipase C (PLC), ERK1/2, Akt1, and Ca++, all of which are key signaling molecules for endothelial functions. NO is a potent vasodilator synthesized by eNOS in endothelial cells and also critically mediates VEGF- and FGF2-stimulated angiogenesis. It has been established that eNOS activation can be regulated by phosphorylation and [Ca^{2+}] at different levels. GNA11, as an essential mediator for activation of VEGF receptor 2 (KDR), has been shown to be required for VEGF-stimulated human umbilical vein endothelial (HUVE) cell proliferation and migration via PLC-dependent activation of RhoA (a small GTPase), as well as VEGF-induced ERK1/2 activation. However, roles of GNA11 & 14 in mediating VEGFA- & FGF2-induced endothelial functions is still remain unknown.

Honors:

Grants Received:

2014 SSR Larry Ewing Memorial Trainee Travel Grant

Publications:

- Zhao YJ, Zou QY, Li Y, Li HH, Wu YM, Li XF, Wang K, Zheng J. Expression of G-protein subunit α-14 is increased in human placentas from preeclamptic pregnancies. J Histochem Cytochem. 2014 May;62(5):347-54. doi: 10.1369/0022155414521213. Epub 2014 Jan 14. PubMed PMID: 24423937; PubMed Central PMCID: PMC4005364.

- Li Y, Zhao YJ, Zou QY, Zhang K, Wu YM, Zhou C, Wang K, Zheng J. Preeclampsia Does Not Alter Vascular Growth and Expression of CD31 and Vascular Endothelial Cadherin in Human Placentas. J Histochem Cytochem. 2015 Jan;63(1):22-31. doi: 10.1369/0022155414558063. Epub 2014 Oct 31. PubMed PMID: 25362142.

- Li Y, Wang K, Zou QY, Zhou C, Magness RR, Zheng J. A possible role of aryl hydrocarbon receptor in spontaneous preterm birth. Med Hypotheses. 2015 May;84(5):494-7.

- Li Y, Wang K, Zou QY, Magness RR, Zheng J. 2,3,7,8-Tetrachlorodibenzo-p-dioxin differentially suppresses angiogenic responses in human placental vein and artery endothelial cells. Toxicology. 2015 Oct 2;336:70-8.

National Presentations:

- Zou QY, Zhao YJ, Li Y, Chen DB, Zheng J. 2013. IL6 and IL8 Differentially Regulate Human Endothelial Functions. Reproductive Sciences. Poster presentation.

Other Presentations:

ERP Service: