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Major Professor: Ian Bird

Degree Objective: Ph.D. Endocrinology and Reproductive Physiology

Background: BS Molecular Biology, Univ Wi Madison.

Current Research Project:

Pregnancy-related increases in endothelial nitric oxide (NO) production are necessary for enhanced vasodilation and so increased blood perfusion of the uterus. Failure of this adaptation can result in preeclampsia and associated fetal growth restriction. Increases in sustained phase Ca²⁺ signaling, which are a hallmark of pregnancy adaptation, are critically linked to increased endothelial nitric oxide synthase (eNOS) activity in response to agonists such as ATP, which stimulates repeated Ca²⁺ bursts in UA endothelial cells (UAEC) in a manner dependent on Cx43 gap junction function. Vascular endothelial growth factor (VEGF), which is elevated in preeclamptic pregnancies, is a known inhibitor of Cx43 function and we have recently reported VEGF inhibits Ca²⁺ burst and NO production on subsequent ATP challenge in intact UA endothelium. My project focuses on elucidating the signal transduction pathways responsible for the VEGF inhibition of the ATP stimulated Ca²⁺ response by using VEGF receptor subtype specific agonists and pharmacological inhibitors of known signaling pathways. VEGF is also capable of producing its own Ca²⁺ response and associated eNOS activity in UAEC, which may be under control of a separate pathway than that responsible for the inhibition of the ATP stimulated Ca²⁺ response. I am currently characterizing the VEGF stimulated Ca²⁺ response, to better understand how it may compare to the ATP stimulated Ca²⁺ response and if it truly is separate from the inhibitory pathway(s).

Honors:

2009 FASEB Summer Research Conference (Ion Channel Regulation) Travel Award

UW-Madison School of Medicine and Public Health-Herman I. Shapiro Distinguished Graduate Fellowship 2010/2011

Grants Received:

NIH T32 Trainee; NIH Ruth L. Kirschstein National Research Service Award NIH T32-HD041921 2008-2010.

UW-Madison School of Medicine and Public Health-Herman I. Shapiro Distinguished Graduate Fellowship 2010/2011

Publications:



Yi, F. X., Boeldt, D. S., Gifford, S. M., Sullivan, J. A., Grummer, M. A., Magness, R. R., Bird, I. M. (2010). Pregnancy enhances sustained Ca²⁺ bursts and endothelial nitric oxide synthase activation in ovine uterine artery endothelial cells through increased connexin 43 function. *Biology of Reproduction*, 82(1), 66-75.

Yi FX, Boeldt DS and Bird IM. Pregnancy Induced Reprogramming of Endothelial Function in Response to ATP: Evidence for Post Receptor Ca²⁺ Signaling Plasticity. In: *Extracellular ATP and Adenosine as the Regulators of Endothelial Cell Function.*, edited by Gerasimovskaya E and Kaczmarek E Springer Publications, 2010, p. 197-213.

National Presentations:

Poster Presentation: Boeldt DS, Grummer MA, Yi FX, Magness RR, Bird IM. (2009) Pregnancy-Specific Changes in VEGF Ca²⁺ Signaling in Uterine Artery Endothelial Cells. 2009 FASEB Summer Research Conference: Ion Channel Regulation. Snowmass, CO. Abstract 17.

Poster Presentation: Boeldt DS, Grummer MA, Magness RR, Yi FX, Bird IM. (2010) Differential Regulation of Cx43 Phosphorylation and Inhibition of Cell Coupling in Uterine Artery Endothelial Cells. 57th Annual Meeting for the Society for Gynecologic Investigation. Orlando, FL. Abstract 917.

Poster Presentation: Boeldt DS, Grummer MA, Magness RR, Yi FX, Bird IM. (2010) VEGF Inhibition of ATP Stimulated Ca²⁺ Signaling in Uterine Artery Endothelial Cells: A Potential Role for Cx43 Gap Junction Phosphorylation. 2010 FASEB Summer Research Conference: Calcium and Cell Function. Steamboat Springs, CO. Abstract 53.

Other Presentations:

Oral Presentation: Boeldt DS, Grummer MA, Yi FX, Bird IM (2008) Using an Adenoviral Approach to Investigate Pregnancy Specific Changes in Ca²⁺ Signaling in UAEC. *Endocrinology Reproductive Physiology Seminar* Jan 24, 2008.

Selected for Oral Presentation: Boeldt DS, Grummer MA, Yi FX, Bird IM. (2008) Using an Adenoviral Approach to Investigate Pregnancy Specific Changes in Ca²⁺ Signaling in UAEC. *Endocrinology-Reproductive Physiology Training Program 2008 Annual Scientific Symposium*, Abstract p.7.

Oral Presentation: Boeldt DS, Grummer MA, Yi FX, Bird IM (2008) Comparing ATP and VEGF Ca²⁺ Signaling in Uterine Artery Endothelial Cells. *Endocrinology Reproductive Physiology Seminar* Sept 11, 2008.

Oral Presentation: Boeldt DS, Grummer MA, Yi FX, Bird IM. (2009) VEGF Ca²⁺ Signaling in Uterine Artery Endothelial Cells. *Perinatal Research Labs Tuesday Talks* Jan 13, 2009.

Selected for Oral Presentation: Boeldt DS, Grummer MA, Yi Fx, Bird IM. (2009) Pregnancy-Specific Changes in VEGF Ca²⁺ Signaling in Uterine Artery Endothelial Cells. *Endocrinology-Reproductive Physiology Training Program 2009 Annual Scientific Symposium*, Abstract p.9.



Oral Presentation: Boeldt DS, Grummer MA, Yi FX, Bird IM (2010) VEGF as a Mediator of Preeclampsia: VEGF Induced Endothelial Dysfunction in Uterine Artery Endothelial Cells. Endocrinology Reproductive Physiology Seminar Oct 7, 2010.

Oral Presentation: Boeldt DS, Grummer MA, Yi FX, Bird IM (2010) VEGF as a Mediator of Preeclampsia: VEGF Induced Endothelial Dysfunction in Uterine Artery Endothelial Cells. Perinatal Research Labs Tuesday Talks Nov 9, 2010.

Poster Presentation: Boeldt DS, Grummer MA, Magness RR, Yi FX, Bird IM. (2010) Differential Regulation of Cx43 Phosphorylation and Inhibition of Cell Coupling in Uterine Artery Endothelial Cells. Endocrinology-Reproductive Physiology Training Program 2010 Annual Scientific Symposium, Abstract p.9.

ERP Service:

ERP Seminar Committee 2007 - 2009

ERP Curriculum Committee 2008 – 2010

ERP Recruitment Committee 2008 - Current