BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Charles Evans Wood	Professor and Chair
eRA COMMONS USER NAME (credential, e.g., agency login) cewood	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
University of California at Berkeley	A.B.	06/74	Biochemistry
University of California at San Francisco	Ph.D.	06/80	Endocrinology
University of California at San Francisco	postdoctoral	05/83	Fetal Physiology

Please refer to the application instructions in order to complete sections A, B, C, and D of the Biographical Sketch.

A. Personal Statement

My laboratory is focused on the mechanisms controlling the fetal responses to stress and the mechanism of prematurity. Our experimental model is the chronically catheterized fetal sheep model, and our work combines a wide range of techniques including in vivo experimentation, molecular biology endpoints, and systems biology analysis of transcriptomics.

B. Positions and Honors

Positions and Employment

1983-1988: Assistant Professor of Physiology, University of Florida College of Medicine.
1988: Tenure granted by the University of Florida.
1988-1993: Associate Professor of Physiology, University of Florida College of Medicine.
1993-present: Professor of Physiology, University of Florida College of Medicine.
2000-2003: Interim Chair, Department of Physiology and Functional Genomics, Univ. of Florida College of Medicine
2003-present: Chair, Department of Physiology and Functional Genomics, Univ. of Florida College of Medicine

Other Experience and Professional Memberships

1986-2001: Editorial Board, *Am. J. Physiol. (Reg., Int., Comp. Physiology)*
1987-1990: Editorial Board, *Domestic Animal Endocrinology*
1992-1996: Peer Review Research Committee, Florida Affiliate American Heart Association
1999-2003: NIH Human Embryology and Development study section, regular member
1996-1999: Research Committee, Florida/Puerto Rico Affiliate American Heart Association
1997-2000: Chair, Peer Review Committee #1, Florida/Puerto Rico Affiliate American Heart Association
2004-2006: Chair, Research Committee, Florida/Puerto Rico Affiliate American Heart Association
2011-present: Editorial Board, *Am. J. Physiol. Reg., Int. Comp Physiol.*
2013-present: Editorial Board, *Physiological Reports*
2012-2015: Pregnacy and Neonatology study section, member (Chair, 2013-2015)
Various: *Ad hoc* member of several NIH study sections and special emphasis panels.
Member: Endocrine Society, American Physiological Society, Society for Gynecologic Investigation, Society for Neuroscience, Fetal and Neonatal Physiological Society.

Honors

1979-1980: Chancellor's Research Fellowship (UCSF)
1980: Who's Who in American Colleges and Universities
1980-1982: Postdoctoral Traineeship, UCSF (Training Grant #HL07192)
1982-1983: Postdoctoral Fellowship, UCSF (NIH NRSA #AM06407)
1983-1986: NIH New Investigator Award (#HD17705)
1988-1993: Established Investigator of the American Heart Association

C. Selected Peer-Reviewed Publications (Selected from 154 peer-reviewed publications)

Wood, C.E., M.B. Rabaglino, E. Richards, N. Denslow, M. Zarate, E.I. Chang, and M. Keller-Wood. Transcriptomics of the Fetal Hypothalamic Response to Brachiocephalic Occusion and Estradiol Treatment. Physiological Genomics, 46(14):523-32, 2014. PMCID: In process

Keller-Wood, M., X. Feng, C.E. Wood, E. Richards, R. Anthony, G.E. Dahl, and S. Tao. Elevated maternal cortisol leads to relative hyperglycemia and increased stillbirth in ovine pregnancy. Am. J. Physiol. Reg. Int. Comp. Physiol. 307(4):R405-13, 2014. PMCID: In process

Wood, C.E., M.B. Rabaglino, E.I. Chang, N. Denslow, M. Keller-Wood, and E. Richards. The Genomics of the Fetal Hypothalamic Cellular Response to Transient Hypoxia: Endocrine, Immune, and Metabolic Responses. *Physiological Genomics* 45(13):521-27, 2013. PMC3727022 [Available on 2014/7/1]

Rabaglino, M.B., E. Richards, N. Denslow, M. Keller-Wood, and **C.E. Wood**. Genomics of Estradiol-3-Sulfate Action in the Ovine Fetal Hypothalamus. *Physiological Genomics* 44(13):669-77, 2012. PMCID: PMC3426428.

Wood, C.E.. Fetal Hypothalamus-Pituitary-Adrenal Responses to Estradiol Sulfate. *Endocrinology* 152(12)4966-4973, 2011. PMCID: PMC3230050.

James M.O., W. Li, D.P. Summerlot, L. Rowland-Faux, and C.E. Wood. Triclosan is a Potent Inhibitor of Estradiol and Estrone Sulfonation in Sheep Placenta. Environ Int. 36(8):942-949, 2010.

Gersting, J.A., C.E. Schaub, and C.E. Wood. Ontogeny of Prostaglandin Endoperoxide Synthase Enzyme Expression in the Ovine Fetal Central Nervous System and Pituitary. Mechanisms of Development: Gene Expression Patterns 9:603-611, 2009. PMID: 19706338

Schaub, C. and C.E. Wood. Blockade of Estrogen Action Decreases Estrogen Receptor Alpha Expression in the Fetal Brain. Neonatology 96(2):115-119, 2009. PMC2793321

Wood C.E., M. Powers Fraites, and M. Keller-Wood. Blockade of PGHS-2 Inhibits the Hypothalamus-Pituitary-Adrenal Axis Response to Cerebral Hypoperfusion in the Sheep Fetus. Am J Physiol Regul Integr Comp Physiol. 296(6):R1813-9, 2009.

Wood C.E., M. Powers Fraites, and M. Keller-Wood. Blockade of PGHS-2 Inhibits the Hypothalamus-Pituitary-Adrenal Axis Response to Cerebral Hypoperfusion in the Sheep Fetus. Am J Physiol Regul Integr Comp Physiol. 296(6):R1813-9, 2009. PMCID: PMC2692792

Gersting, J.A., C. Schaub, M. Keller-Wood, and C.E. Wood. Inhibition of Brain PGHS-2 Prevents the Preparturient Increase in Fetal ACTH Secretion in the Sheep Fetus. Endocrinology, 149(8):4128-36, 2008. PMCID: PMC2488234

Schaub, C., J. Gersting, M. Keller-Wood, and C.E. Wood. Development of ER-α and ER-β Expression in the Developing Ovine Brain and Pituitary. Gene Expression Patterns 8(6):457-63, 2008

Powers, M. and C.E. Wood Ketamine Inhibits Fetal ACTH Response to Cerebral Hypoperfusion. Am. J. Physiol. Reg., Int., Comp. Physiol. 292(4):R1542-1549, 2007. PMCID: PMC2793322

Wood, C.E. Estrogen-HPA Interactions in the Fetus: The Interplay Between Placenta and Fetal Brain. J. Soc. Gyn. Invest. 12(2):67-76, 2005.

Wood, C.E., and D. Giroux. Central Nervous System Prostaglandin Endoperoxide Synthase-1 and -2 Responses to Oestradiol and Cerebral Hypoperfusion in Late-Gestation Fetal Sheep. J. Physiol. (Lond.) 549.2:573-581, 2003. PMCID: PMC2342963

Wood, C.E., K.E. Gridley, and M. Keller-Wood. Biological activity of 17β-estradiol-3-sulfate in ovine fetal plasma and uptake in fetal brain. Endocrinology. 144(2):599-604, 2003. PMID: 12538622

Purinton, S.C. and C.E. Wood. Oestrogen augments the fetal ovine hypothalamus-pituitary-adrenal axis in response to hypotension. J. Physiol. (Lond.) 544(Pt 3): 919-29, 2002. PMCID: PMC2290634

Purinton, S.C., H. Newman, M.I. Castro, and C.E. Wood. Ontogeny of estrogen sulfatase activity in ovine fetal hypothalamus, hippocampus, and brainstem. Am. J. Physiol. (Reg., Int., Comp. Physiol.) 276:R1647-R1652, 1999. PMID: 10362743

Wood, C. E. Insensitivity of term fetal sheep to cortisol: Possible relation to the control of parturition. Endocrinology, 122:1565-1572, 1988.

C. Research Support. Ongoing research projects pertinent to the proposed research.

WOOD, C.E.

ONGOING

5 R01 HD33053-15 (Wood, PI) 12/1/12-11/30/17
NIH/NICHD
Fetal Reflex and Endocrine Responses
The major goals of this project are to test the hypothesis that ketamine, a clinically-useful NMDA receptor blocker, inhibits the inflammatory response to fetal stress. The experiments in this project include investigation using whole animal, cellular, and transcriptomics (systems biology) approaches.

1 R01 HD050414 (Wood, PI) 1/1/09-12/31/13
NIH/NICHD
Renal Transporters and Fetal Neuroendocrinology
The major goal of this project is to investigate the role of sulfoconjugated estrogens as controllers of fetal neuroendocrine responses to stress, and to investigate the role of organic anion transporters as mediators of the entry of sulfoconjugated estrogens into the fetal brain.

1R01 HD057871-01 (Keller-Wood, PI; Wood Co-Investigator) 12/1/08-11/2014 (NCE 2013-14)
NIH/NICHD
Effects of maternal cortisol on fetal and neonatal growth and metabolism
These studies will test the mechanisms by which either increased or decreased maternal cortisol can alter fetal growth and metabolism.

1 R01 HD056288 (Keller-Wood, PI; Wood Co-Investigator) 6/01/08- 5/31/14 (NCE 2013-14)
NIH/NICHD
The baroreflex in pregnancy: effects of adrenal and placental steroids.
The proposed studies are designed to address the hypothesis that increases in cortisol contribute to the normal decrease in baroreflex sensitivity in pregnancy by effects within the CNS, as well as by indirect effects on blood volume. We further hypothesize that these effects are modulated by progesterone and estradiol, and that the vulnerability of the late gestation pregnant ewe and the parturient woman to decreases in plasma cortisol is caused by changes in blood volume and/or progesterone occurring at term or after delivery.

1R21 ES020545-01 (Wood, James, multiple PI grant) 7/1/11-6/30/14 (NCE 2013-14)
NIH/NIEHS
Fetal Endocrine Disruption by Triclosan
The environmental endocrine disruptor, Triclosan, has
been shown to potently inhibit a key enzyme in the pathway to the formation of fetal estrogen in the placenta, estrogen sulfotransferase (SULT1E1). This application proposes studies in a fetal sheep model of in utero fetal development to evaluate the effects of Triclosan on levels of circulating fetal estrogen availability and downstream genomic consequences in fetal target tissues.

COMPLETED

R01 HD42135 (Wood, PI) 4/1/02-3/31/07
NIH/NICHD
Estrogen Influences on Fetal Adrenocorticotropin
The major goal is to investigate the interaction between increases in fetal estrogen production and increases in activity of the ovine fetal hypothalmo-pituitary-adrenal axis.

1 R01 DK62080 (Keller-Wood, PI; Wood Co-Investigator) 3/1/03-2/28/08
NIH/NIDDK
Cortisol at MR mediate fetal physiologic/genomic effects
The goal of this study is to identify the roles of mineralocorticoid and glucocorticoid receptors in the control of electrolyte and fluid balance by the fetal lung and kidney, and on control of the fetal pituitary-adrenal axis

using both physiologic endpoints (lung liquid composition, urine production and composition and plasma ACTH), and molecular markers of steroid action in these cells (sgk, ENaC and 5HT1A protein and mRNA).

2 R01 DK38114-11 (Keller-Wood, PI; Wood Co-Investigator) 7/1/99 – 6/30/08
NIH/NIDDK
Control of Corticotropin during Pregnancy
The major goals of this project are to determine the role changes in corticosteroid receptors in the brain during pregnancy, and to test the hypothesis that progesterone alters mineralocorticoid function and ACTH secretion in pregnancy.