BIOGRAPHICAL SKETCH

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NAME: Joey P. Granger, Ph.D.

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

POSITION TITLE: Professor of Physiology and Medicine

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
Univ. of Louisiana, Lafayette, LA	B.S.	1979	Biology/Chemistry
Univ. of Mississippi Medical Ctr., Jackson, MS	Ph.D.	1983	Physiology/Biophysics

A. Personal Statement

Joey Granger is the Billy S. Guyton Distinguished Professor and Professor of Physiology and Medicine. He also serves as Director of the Cardiovascular Renal Research Center. He is internationally recognized for his research in hypertension and renal physiology. He has a 25 year history of successful funding from NIH and is currently funded by several grants from the NHLBI. Dr. Granger has mentored many outstanding investigators at UMMC and other academic institutions (>35 trainees). Many of his trainees have obtained positions in the academic and biotechnology industry. He was recently awarded the 2008 American Physiological Society Bodil M. Schmidt-Nielsen Distinguished Mentor and Scientist Award. Dr. Granger also has extensive administrative and leadership experience, currently serving as the Dean of the Graduate School and on the leadership committees of the American Physiology Society, American Heart Association-Council for High Blood Pressure Research, and the Inter-American Society of Hypertension. He also recently served as Chairman of the Hypertension Microcirculation Study section, CSR, NIH. He recently served as President of The American Physiological Society. He is currently Chair-Elect of the Council for High Blood Pressure Research for the American Heart Association.

B. Positions and Honors

Professional Experience:

1985-86 Asst. Professor, Dept. of Physiology and Biophysics, Mayo Med. School, Rochester, MN
1985-86 Associate Consultant, Mayo Clinic and Foundation, Rochester, MN
1986-88 Assistant Professor, Dept. of Physiology, Eastern Virginia Medical School, Norfolk, VA
1988-90 Assoc. Professor, Dept. of Physiology, Eastern Virginia Med. School, Norfolk, VA
1990-Pr. Professor, Dept. of Physiology and Biophysics, Univ. Mississippi Medical Center, Jackson, MS
2008-Pr. Dir., Center for Excellence in Cardiovascular Renal Research, Univ. of MS Med.Center
2004-Pr. Billy S. Guyton Distinguished Professor, Univ. of Mississippi Medical Center

Honors and Awards (Selected):

Granger is currently an Associate Editor for *Hypertension* and serves as Co-Editor with his brother, Neil Granger, on the eBook series entitled *Integrative Systems Physiology*. He has also served as the Editor of the *Council for High Blood Pressure Newsletter* and an Associate Editor for *News in Physiological Sciences* and *American Journal of Physiology: Regulatory and Integrative Physiology.* He is serving or has served as a member of Editorial Boards of *American Journal of Hypertension, American Journal of Physiology: Renal*

Physiology, American Journal of Physiology: Regulatory and Integrative Physiology, Journal of CardioMetabolic Syndrome and *the Journal of the American Society of Hypertension*.

Granger served as President of the American Physiology Society in 2012 and was recently elected as Chair of the Council on Hypertension of the American Heart Association. He also currently serves on Leadership committees of the Inter-American Society of Hypertension. He also served on numerous scientific committees of the Council for High Blood Pressure Research, Inter-American Society of Hypertension, American Society of Hypertension, and the American Physiological Society. Within the American Physiological Society he has served as a Councilor, Chair of Committee on Committees, Long-Range Planning Committee, Career Opportunities Committee, Program Committee, President of the Gulf Coast Physiological Society, Chair of the Water and Electrolyte Homeostasis Section, Section Advisory Committee, Nominating Committee, Publication Committee, Finance Committee, Strategic Planning Committee, and as a mentor for the Frontiers in Physiology program and APS/NIDDK minority fellowship program and the APS Summer Undergraduate Research program. Granger has also served on scientific study sections for the American Heart Association, National Institutes of Health, NASA, and the Veterans Administration. He recently served as chair of the Hypertension and Microcirculation NIH study section. He also served on the National Board Medical Exam Physiology Test Development Committee.

Granger has received numerous research awards including the 2010 American Heart Association Distinguished Scientist Award, American Physiological Society 2008 E.H. Starling Distinguished Lecture Award, American Physiological Society 2008 Bodil M. Schmidt-Nielsen Distinguished Mentor and Scientist Award, Dahl Memorial Lecture of the American Heart Association, American Society of Hypertension Young Scholar Award, the International Society of Hypertension Demuth Research Award, Inter-American Society of Hypertension Young Investigator Award, the Regulatory and Integrative Physiology Young Investigator Award of the American Physiological Society Water and Electrolyte Section, the Harold Lamport Award of the Cardiovascular Section of the American Physiological Society, the Henry Pickering Bowditch Lecture of the American Physiological Society, and the Established Investigator Award of the American Heart Association.

C. Contribution to Science

Granger's overall research program over the last 30 years has examined molecular mechanisms in the pathogenesis of hypertension. His early research demonstrated the importance of renal interstitial hydrostatic pressure in mediating renal pressure natriuresis and how this mechanism is deranged in various models of hypertension. He was also one of the first to demonstrate the importance of atrial natriuretic peptide (ANP) in long-term control of sodium balance and arterial pressure. He also demonstrated the intrarenal actions of synthetic ANP He also demonstrated that ANP had potent actions on the renin-angiotensin system and that chronic physiological elevations in plasma ANP produced long-term improvement in renal pressure natriuresis and reductions in arterial pressure.

1. Granger, J. P., T. J. Opgenorth, J. Salazar, J. C. Romero, and J. C. Burnett, Jr. Long-term hypotensive and renal effects of chronic infusions of atrial natriuretic peptide in conscious dogs. Hypertension 8:II-112-II-116, 1986.

2. Granger, J. P., J. Haas, and F. G. Knox. Effects of direct increase in renal interstitial hydrostatic pressure on sodium excretion. Am. J. Physiol. 254:F527-F532, 1988.

His later work investigated the role of the renal endothelin and nitric oxide systems in various models of salt-sensitive hypertension. His laboratory demonstrated that nitric oxide plays a critical role in protecting the preglomerular vessels from ANGII and norepinephrine vasoconstriction and loss of nitric oxide leads to renal failure and hypertension. They also demonstrated that nitric oxide regulated renin release via a macula densa mediated mechanism. Finally, in a series of studies the Granger laboratory demonstrated an important effect of endothelin on renal function and blood pressure regulation in a number of models of hypertension.

3. Alberola, A., F. J. Salazar, T. Nakamura, and J. P. Granger. Renal hemodynamic effects of angiotensin II (AII): Interactions with endothelium derived nitric oxide. Am. J. Physiol. 267:R1472-R1478, 1994.

4. Wilkins, F. C., Jr., A. Alberola, H. L. Mizelle, T. J. Opgenorth, and J. P. Granger. Systemic hemodynamics and renal function during long-term pathophysiological increases in circulating endothelin. Am. J. Physiol.

5. Schnackenberg, C., B. Tabor, M. Strong, and J. P. Granger. Intrarenal NO blockade enhances renin secretion rate by a macula densa mechanism. Am. J. Physiol. 272:R879-R886, 1997.

6. Schnackenberg, C., C. Wilkins, and J. P. Granger. Role of nitric oxide in modulating the vasoconstrictor actions of angiotensin II in preglomerular and postglomerular vessels in dogs. Hypertension 26(2): 1024-1029, 1995.

7. Kato, T., S. Kassab, F. C. Wilkins, K. Kirchner, J. Keiser, and J. P. Granger. Endothelin antagonist improve renal function in spontaneously hypertensive rats. Hypertension 25(2):883-887, 1995.

8. Granger, J. P., C. G. Schnackenberg, J. Novak, B. Tucker, T. Miller, S. Morgan, and S. E. Kassab. Role of nitric oxide in modulating the long-term renal and hypertensive action of norepinephrine. Hypertension 29(2):205-209, 1997.

9. Kassab, S., J. Novak, T. Miller, K. A. Kirchner, and J. Granger. Cardiovascular and renal actions of endothelin receptor antagonism in Dahl salt-sensitive hypertension. Hypertension 30(3):682-686 1997

His current research focuses on the role of endothelial and neurohormonal factors in mediating hypertension in animal models of pregnancy-induced hypertension or preeclampsia. His laboratory over the last 5 years has published over 50 peer-reviewed manuscripts on the topic of pathophysiology of preeclampsia. Utilizing the RUPP (Reduced Uterine Perfusion Pressure) model of placental ischemia, which was developed by the Granger laboratory, they demonstrated that placental ischemia in the pregnant rat has many of the features of preeclampsia in women. They are currently using this model for the investigation of the mechanisms linking placental ischemia and cardiovascular dysfunction in preeclampsia and for identifying potential drug targets for the treatment of preeclampsia. They are also investigating molecular mechanisms whereby obesity increases the risk for developing preeclampsia. Dr Granger was recently appointed by American College of Obstetricians and Gynecologists on Hypertension in Pregnancy Working Group, where he was responsible for outlining future directions in preeclampsia research.

10. Alexander, B. T., S. E. Kassab, M. T. Miller, S. R. Abram, J. F. Reckelhoff, W. A. Bennett, and J. P. Granger. Reduced uterine perfusion pressure during pregnancy in the rat is associated with increases in arterial pressure and changes in renal nitric oxide. Hypertension. 37: 1191-1195, 2001.

11. Alexander, B. T., A. N. Rinewalt, K. L. Cockrell, W. A. Bennett, and J. P. Granger. Endothelin-A receptor blockade attenuates the hypertension in response to chronic reductions in uterine perfusion pressure. Hypertension. 37:485-489, 2001

12. LaMarca B.B., G. Gadonski, K. Cockrell, E. Sullivan and J.P. Granger. Endothelin type A receptor blockade attenuates TNF alpha-induced hypertension in pregnant rats. Hypertension 46:1-5, 2005

13. Murphy SR, BB LaMarca, K Cockrell, JP Granger. Soluble fms-Like Tyrosine-1 Induced Hypertension: Role of Endothelin. Hypertension 2010 Feb;55(2):394-8. NIHMS172853

14. Gilbert JS, Verzwyvelt JD, D Colson, M Arany, M J Ryan, and J P Granger. Vascular Endothelial Growth Factor Improves Renal and Endothelial Function, and Normalizes Blood Pressure in Hypertensive Pregnant Rats. Hypertension 2010 Feb;55(2):380-5. NIHMS172854

15. Spradley FT, Sasser JM, Musall JB, Sullivan JC, Granger JP Nitric oxide synthase-mediated blood pressure regulation in obese melanocortin-4 receptor-deficient pregnant rats. Am J Physiol Regul Integr Comp Physiol. 2016] PMID:27534879

16. Spradley FT, Palei AC, Granger JP.Differential body weight, blood pressure and placental inflammatory responses to normal versus high-fat diet in melanocortin-4 receptor-deficient pregnant rats.J Hypertens. 2016 Oct;34(10):1998-2007. PMID:27467764

17. Spradley FT, Tan AY, Joo WS, Daniels G, Kussie P, Karumanchi SA, Granger JP.Placental Growth Factor Administration Abolishes Placental Ischemia-Induced Hypertension. Hypertension. 2016 Apr;67(4):740-7. PMID:26831193

18. Sildenafil Treatment Ameliorates the Maternal Syndrome of Preeclampsia and Rescues Fetal Growth in the Dahl Salt-Sensitive Rat.Gillis EE, Mooney JN, Garrett MR, Granger JP, Sasser JM .Hypertension. 2016 Mar;67(3):647-53 2016 PMID:26729752

My NCBI - Bibliography: http://www.ncbi.nlm.nih.gov/pubmed/?term=granger+jp

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D. Research Support

Ongoing:

U54 GM115428 NIH/NIGMS	8/18/16 – 7/31/21	Mississippi Center for Clinical and Translational Research Wilson, JG, PI Core leader-: J P Granger
NIH/NRSA 7	1/14 – 6/30/17	Effects of obesity on the development of hypertension during pregnancy" Trainees : Frank Spradley Mentor: Joey P. Granger
NIH 1T32HL105324	2010-2022	Hypertension and Cardiorenal Diseases Research Training Program PI: J.P. Granger

The major goal of this project is to train graduate students and postdocs in cardiovascular-renal research.

NIH 2 P01 HL051971	2014-2019	Renal Control of Body Fluids and Circulatory Dynamics Project II of PPG PI: J.P. Granger (30%)

Project II of the proposal seeks to determine interactions between metabolic factors and angiogenic and endothelial factors in the pathophysiology of pregnancy-induced hypertension.

NIH 1P20 GM 104357-01	2013-2018	Cardiorenal and Metabolic Diseases Research Center Co-Investigator: J.P. Granger

The major goal of this COBRE grant is to develop a Cardiorenal and Metabolic Resources Research Center.

1R01HL121527-01	2014-2019	A Novel Protein Delivery System for Therapy of Preeclampsia, Co-Investigator: J P Granger

This grant will investigate the use of novel therapeutic polypeptides in the treatment of preeclampsia-associated hypertension and investigate the beneficial effects on offspring cardiovascular health