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## BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2.  
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NAME Raizada, Mohan K.		POSITION TITLE Distinguished Professor	
eRA COMMONS USER NAME (credential, e.g., agency login) mraizada			
EDUCATION/TRAINING ( <i>Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.</i> )			
INSTITUTION AND LOCATION	DEGREE (if applicable)	MM/YY	FIELD OF STUDY
University of Lucknow, India	B.Sc.	1966	Biol., Chem., & Botany
University of Lucknow, India	M.Sc.	1968	Biochemistry
Central Drug Research Institute/ University of Kanpur, India	Ph.D.	1972	Biomedical Sciences

### **A. Personal Statement:**

My interest in the role of the renin-angiotensin system in cardiovascular disease and hypertension spans three decades. Our research group was among the first to propose that the balance between the vasoprotective axis and vasodeleterious axis, rather than a single component of the renin-angiotensin system, is critical in the maintenance of normal cardiovascular homeostasis. We were also among the first to provide conceptual evidence that neuroinflammation in the autonomic brain regions particularly in the paraventricular nucleus, is the central mechanism that may be responsible for drug-resistant neurogenic hypertension. Thus our group is investigating the hypothesis that activation of microglia in the PVN initiates a cascade of events starting with the activation of sympathetic nerve activity to the bone marrow that impairs the balance between the inflammatory and angiogenic progenitor cells regulation impacting cardiovascular pathophysiology associated with hypertension. This innovative concept, if proven, would offer us new and novel targets for drug resistant hypertension therapeutics

### **B. Positions and Honors:**

2007– Present - Distinguished Professor, Department of Physiology & Functional Genomics, UF  
1987-2006 – Professor, Department of Physiology & Functional Genomics, University of Florida  
1993-1998 – Associate Dean, Graduate Education, College of Medicine, UF  
1981-1986 – Associate Professor, Department of Physiology, University of Florida  
1979-1981 – Assistant Professor, Department of Physiology and Biophysics, University of Iowa  
1977-1978 – Associate, Department of Physiology and Biophysics, University of Iowa  
1974-1976 – Postdoctoral Associate, Lady Davis Institute for Medical Research, Montreal  
1973-1974 – Postdoctoral Fellow, Medical College of Wisconsin, Milwaukee

### **Honors:**

2014- Carl Ludwig Distinguished Lectureship, NCAR Section of the American Physiological Society  
2013- Arthur C Corcoran Memorial Lecture, HBPRC of the American Heart Association  
2014- Ranbaxy Award for Outstanding Achievements in Biomedical Research  
2007- Distinguished Professor, University of Florida  
2003-2013- MERIT Award, NIH  
1985-1990- Established Investigator, American Heart Association

### **Other Experience and Professional Memberships:**

2009-2010 NIH-VCMB study section, Regular Member  
2006- 2008 AHA Peer Review Committee, Regular Member

2002-2006	NIH-HM study section, Regular Member
2005- Present	British Physiological Society
2004- Present	American Heart Association
1996-2000	NIH-ECS study section, Regular Member
1983- Present	Member, American Physiological Society
1980- Present	The Endocrine Society

### **C. Selected and Relevant Peer-reviewed Publications (From a total of 305):**

- 1) Zubcevic J, Santisteban MM, Pitts T, Baekey DM, Perez PD, Bolser DC, Febo M, **Raizada MK**. *Hypertension* 2014; 63:129-39. Epub 2014 Mar 31. No abstract available.
- 2) Zubcevic J, Jun JY, Kim S, Perez PD, Afzal A, Shan Z, Li W, Santisteban MM, Yuan W, Febo M, Mocco J, Feng Y, Scott E, Baekey DM, **Raizada MK**. Altered inflammatory response is associated with an impaired autonomic input to the bone marrow in the spontaneously hypertensive rat. *Hypertension* 2014; 63:542-50.
- 3) Hu P, Thinschmidt JS, Yan Y, Hazra S, Bhatwadekar A, Caballero S, Salazar T, Miyan JA, Li W, Derbenev A, Zsombok A, Tikhonenko M, Dominguez JM 2nd, McGorray, SP, Saban DR, Boulton ME, Busik JV, **Raizada MK**, Chan-Ling T, Grant MB. CNS inflammation and bone marrow neuropathy in type 1 diabetes. *Am J Pathol* 2013; 183:1608-20.
- 4) Qi Y, Zhang J, Cole-Jeffrey CT, Shenoy V, Espejo A, Hanna M, Song C, Pepine CJ, Katovich MJ, **Raizada MK**. Diminazene aceturate enhances angiotensin-converting enzyme 2 activity and attenuates ischemia-induced cardiac pathophysiology. *Hypertension* 2013; 62:746-52.
- 5) Santisteban MM, Zubcevic J, Baekey DM, **Raizada MK**. Dysfunctional brain-bone marrow communication: a paradigm shift in the pathophysiology of hypertension. *Curr Hypertens Rep* 2013; 15:377-89.
- 6) Shan Z, Zubcevic J, Shi P, Jun JY, Dong Y, Murça TM, Lamont GJ, Cuadra A, Yuan W, Qi Y, Li Q, Paton JF, Katovich MJ, Sumners C, **Raizada MK**. Chronic knockdown of the nucleus of the solitary tract AT1 receptors increases blood inflammatory-endothelial progenitor cell ratio and exacerbates hypertension in the spontaneously hypertensive rat. *Hypertension* 2013; 61:1328-33.
- 7) de Kloet AD, Krause EG, Shi PD, Zubcevic J, **Raizada MK**, Sumners C. Neuroimmune communication in hypertension and obesity: a new therapeutic angle? *Pharmacol Ther* 2013; 138:428-40.
- 8) Shenoy V, Gjymishka A, Jarajapu YP, Qi Y, Afzal A, Rigatto K, Ferreira AJ, Fraga-Silva RA, Kearns P, Douglas JY, Agarwal D, Mubarak KK, Bradford C, Kennedy WR, Jun JY, Rathinasabapathy A, Bruce E, Gupta D, Cardounel AJ, Mocco J, Patel JM, Francis J, Grant MB, Katovich MJ, **Raizada MK**. Diminazene attenuates pulmonary hypertension and improves angiogenic progenitor cell functions in experimental models. *Am J Respir Crit Care Med* 2013; 187:648-57.
- 9) Zubcevic J, Jun JY, Lamont G, Murça TM, Shi P, Yuan W, Lin F, Carvajal JM, Li Q, Sumners C, **Raizada MK**, Shan Z. Nucleus of the solitary tract (pro)renin receptor-mediated antihypertensive effect involves nuclear factor- $\kappa$ B-cytokine signaling in the spontaneously hypertensive rat. *Hypertension* 2013; 61:622-7.
- 10) Jarajapu YP, Bhatwadekar AD, Caballero S, Hazra S, Shenoy V, Medina R, Kent D, Stitt AW, Thut C, Finney EM, **Raizada MK**, Grant MB. Activation of the ACE2/angiotensin-(1-7)/Mas receptor axis enhances the reparative function of dysfunctional diabetic endothelial progenitors. *Diabetes* 2013 62:1258-69.

- 11) Jun JY, Zubcevic J, Qi Y, Afzal A, Carvajal JM, Thinschmidt JS, Grant MB, Mocco J, **Raizada MK**. Brain-mediated dysregulation of the bone marrow activity in angiotensin II-induced hypertension. *Hypertension* 2012; 60:1316-23.
- 12) Yellowlees Douglas J, Bhatwadekar AD, Li Calzi S, Shaw LC, Carnegie D, Caballero S, Li Q, Stitt AW, **Raizada MK**, Grant MB. Bone marrow-CNS connections: implications in the pathogenesis of diabetic retinopathy. *Prog Retin Eye Res* 2012; 31:481-94.
- 13) Waki H, Gouraud SS, Maeda M, **Raizada MK**, Paton JF. Contributions of vascular inflammation in the brainstem for neurogenic hypertension. *Respir Physiol Neurobiol* 2011; 178:422-8.
- 14) Zubcevic J, Waki H, **Raizada MK**, Paton JF. Autonomic-immune-vascular interaction: an emerging concept for neurogenic hypertension. *Hypertension* 2011; 57:1026-33.
- 15) Shi P, Diez-Freire C, Jun JY, Qi Y, Katovich MJ, Li Q, Sriramula S, Francis J, Sumners C, **Raizada MK**. Brain microglial cytokines in neurogenic hypertension. *Hypertension* 2010; 56:297-303.

#### **D. Research Support:**

##### **ACTIVE**

**R01 HL33610-28** (Raizada & Sumners, Co-PIs) 08/13 – 08/18  
NIH/NHLBI

“Dysfunctional neural-bone marrow communication in hypertension”

Our overall objective in this application is to investigate the hypothesis that the brain-bone marrow communication and activation of microglial cell in the autonomic brain regions plays important role in the development and establishment of neurogenic hypertension. We also propose to conduct clinical studies to determine if attenuation of microglia activation and inhibition of brain inflammation by minocycline would result in the beneficial outcomes in drug resistant hypertensive patients.

**R01 HL56921-16** (Raizada & Katovich, Co-PIs) 09/11 – 07/15  
NIH/NHLBI

“CVD protection mechanisms involving ACE2/Ang-(1-7) axis”

The overall objective of this study is to use gene transfer technology to regulate the expression of various components of the RAS for long-term control of hypertension.

**HL102033-03** (Raizada & Katovich, Co-PIs) 05/10 – 04/15  
NIH/NHLBI

“ACE2 in vascular endothelial function”

The overall objective of this application is to investigate the role of ACE2 in pulmonary hypertension therapeutics.

**UF Opportunity Funds** (Raizada) 06/14 – 05/16  
UF Division of Sponsored Research

“Oral Delivery of ACE2 for pulmonary hypertension therapeutics”

Overall objective of this proposal is to develop transplasmic plant derived oral delivery system for ACE2 is for the treatment of pulmonary hypertension.

##### **OVERLAP**

There is no scientific or budgetary overlap.