BIOGRAPHICAL SKETCH

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NAME: Kenney, Michael

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

POSITION TITLE: Professor and Associate Dean for Research

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
Luther College, Decorah, IA	B.A.	05/1981	Health
University of Iowa, Iowa City, IA	Ph.D.	12/1988	Exercise Physiology
Michigan State University, East Lansing, MI	Post-doc	08/1990	Neurophysiology

A. Personal Statement

I have been an independent, extramurally-funded investigator for 25 years and my laboratory has been focused on determining how sympathetic nerve regulation is altered by aging, environmental and immune stress, and pathophysiological conditions. We have combined central and peripheral electrophysiological methods, molecular biological techniques, and frequency-domain and big-data analytics to study integrative mechanisms regulating central sympathetic outflow. Central sympathetic neural networks regulate the basal level of activity and the sympathetic nerve discharge (SND) bursting pattern, as well as the acute responsivity of the sympathetic nervous system. The aim of our current NIH grant is to determine how advancing age alters central mechanisms regulating SND under basal conditions and in response to acute physical stress. I have extensive experience mentoring postdoctoral fellows, graduate students and professional students, and have been a Principal Investigator on two NIH training grants. I addition, I am currently the research mentor for K01 and F99 awards and served as a mentor for the 2016 National Research Mentoring Network (NRMN) Grantwriting Workshop.

In December 2015 I became the Associate Dean for Research in the College of Science at the University of Texas at El Paso (UTEP), with a goal of enhancing interdisciplinary research endeavors by building bridges between departments and colleges, as well as between UTEP and community partners, as a platform for addressing research questions related to the Hispanic demographic. The current application provides a strategic integration of basic, clinical and behavioral research initiatives with a focus on Hispanic cancer health disparities. In my role as Deputy Director of Cores in the current U54 proposal, I will oversee the processes and functions associated with each of the designated cores: Research Infrastructure, Investigator Development, Community Engagement and Recruitment. My familiarity with the research infrastructure and processes both at the College and University levels, my experience overseeing major equipment purchases and space allocation processes, as well as training students and mentoring junior faculty will be critical to the successful completion of the proposed aims. The combination of my scientific, administrative and collaborative experiences position me to provide guidance and oversight to the dynamic procedures required for the successful expansion and sustainability of the BBRC.

B. Positions and Honors

Positions and Employment

1984-1986	NIH Graduate Research Assistant, Digestive Disease Core Center, College of Medicine, University of Iowa, Iowa City, IA
1986-1988	NIH Graduate Research Assistant, Cardiovascular Research Center, College of Medicine, University of Iowa, Iowa City, IA
1988-1990	Postdoctoral Research Associate, Department of Pharmacology and Toxicology, Michigan State University, East Lansing, MI
1990-1992	Adjunct Assistant Professor, Department of Physiology and Biophysics, College Of Medicine, University of Tennessee, Memphis, TN
1990-1992	Assistant Professor, Department of Biology, Rhodes College, Memphis, TN
1992 -1996	Assistant Professor, Dept. of Anatomy & Physiology, Kansas State University, Manhattan, KS
1996-2002	Associate Professor, Dept. of Anatomy & Physiology, Kansas State University, Manhattan, KS
2002-2015	Professor, Dept. of Anatomy & Physiology, Kansas State University, Manhattan, KS
2011-2013	Interim Head, Dept. of Anatomy & Physiology, Kansas State University, Manhattan, KS
2013-2015	Department Head, Dept. of Anatomy & Physiology, Kansas State University, Manhattan, KS
Dec 2015-	Associate Dean for Research in the College of Science and Professor of Biological Sciences, University of Texas at El Paso, El Paso, TX

Other Experience and Professional Memberships

American Physiological Society
American Heart Association

C. Contributions to Science

1. My early line of inquiry focused on understanding mechanisms regulating the pattern and frequency-domain components of sympathetic nerve discharge (SND). The dynamic nature of sympathetic neural circuits during acute physical stress is revealed not only by changes in the level of sympathetic nerve activity but also by alterations in the pattern of sympathetic nerve discharge bursts. The SND bursting pattern represents the signature output of central sympathetic neural circuits. Our publications determined that the paraventricular nucleus of the hypothalamus is an important component of the central neurocircuitry regulating the SND bursting pattern, identified SND pattern transformation as an important strategy for mediating sympathoexcitation in response to acute physical stress, and have contributed to the novel hypothesis that neural rhythmicity is important for coordinating activity in different sympathetic nerves. I served as the primary investigator in these studies.

    a. Kenney MJ. Frequency characteristics of sympathetic nerve discharge in anesthetized rats. *Am. J. Physiol*. 267 (*Regulatory Integrative Comp. Physiol*. 36): R830-R840, 1994.
    b. Kenney MJ, Claassen DE, Bishop MR, and Fels RJ. Regulation of the sympathetic nerve discharge bursting pattern during heat stress. *Am. J. Physiol*. 275 (*Regulatory Integrative Comp. Physiol.* 44): R1992-R2001, 1998.
    c. Kenney MJ, Weiss ML, Patel KP, Wang Y, and Fels RJ. Paraventricular nucleus bicuculline alters frequency components of sympathetic nerve discharge bursts. *Am. J. Physiol.* 281 *(Heart Circ. Physiol.)*: H1233-H1241, 2001.
    d. Barman SM and Kenney MJ. Methods of analysis and physiological relevance of rhythms in

sympathetic nerve discharge. *Clin Exp Pharmacol Physiol* 34: 350-355, 2007.

2. The sympathetic nervous system (SNS) plays a critical role in regulating physiological responses to acute stress, and it has generally been considered that the SNS functions independent of other adaptive systems. However, recent lines of inquiry have expanded the functional repertoire of the SNS by establishing an important role for this system in regulating and integrating processes between diverse physiological systems, including the immune system. Understanding mechanisms that mediate physiological relationships between the nervous and immune systems is critical for understanding chronic disease development. Our publications in this burgeoning area of research have demonstrated that central cytokines can contribute to activation of the SNS and have established that splenic sympathoexcitation modulates splenic cytokine gene expression. These findings support the novel hypothesis that activation of central sympathetic neural circuits and subsequent increases in splenic nerve outflow can modulate peripheral immune responses. These studies have contributed to an enhanced understanding of mechanisms mediating neural-immune interactions. I served as the primary investigator in these studies.

    a. Ganta CK, Lu N, Blecha F, Ganta RR, Zheng L, Fels RJ, and Kenney MJ. Central angiotensin II-enhanced splenic cytokine gene expression is mediated by the sympathetic nervous system. *Am J Physiol Heart Circ Physiol* 289: H1683-H1691, 2005.
    b. Helwig BG, Craig RA, Fels RJ, Blecha F, and Kenney MJ. Central nervous system administration of interleukin-6 produces splenic sympathoexcitation. *Autonomic Neuroscience: Basic and Clinical* 141: 104-111, 2008.
    c. Kenney MJ and Ganta CK. Autonomic Nervous System and Immune System Interactions. *Comprehensive Physiology* 4(3): 1177-1200, 2014.
    d. Balivada S, Pawar HN, Montgomery S, and Kenney MJ. Effect of ghrelin on regulation of splenic sympathetic nerve discharge. *Autonomic Neuroscience: Basic and Clinical*. 201: 68-71, 2016.

3. Accompanying the persistent growth in the world's population is a marked increase in the number of aged persons. The SNS is critically involved in the genesis and modulation of diseases and dysfunction in key organ systems in aged subjects. The incidence of many chronic disease conditions increases with advancing age and many adult Americans between the ages of 60-79 suffer from cardiovascular-related diseases. Understanding age-dependent alterations in mechanisms regulating SND is pertinent for understanding relationships between chronic disease development and age-associated changes in SNS function. Our contributions support the hypothesis that SNS responsiveness to acute stress is attenuated with advancing age, changes associated with altered GABAergic regulation in the rostral ventral lateral medulla. These findings have contributed to understanding the effect of advanced age on mechanisms regulating the SNS. I served as the primary investigator in all of these studies.

    a. Kenney MJ. Dexmedetomidine and regulation of splenic sympathetic nerve discharge in Aged F344 rats. *Autonomic Neuroscience: Basic and Clinical*. 190: 53-57, 2015.
    b. Pawar HN, Balivada S, and Kenney MJ. Does aging alter the molecular substrate of ionotropic neurotransmitter receptors in the rostral ventral lateral medulla? *Experimental Gerontology*. 91: 99-103, 2017.
    c. Balivada S, Ganta CK, Zhang Y, Pawar HN, Ortiz RJ, Becker KG, and Kenney MJ. Microarray analysis of aging-associated immune system alterations in the rostral ventral lateral medulla of F344 rats. *Physiological Genomics*. 49(8): 400-415, 2017.
    d. Pawar HN, Balivada S, and Kenney MJ. Does acute heat stress differentially-modulate expression of ionotropic neurotransmitter receptors in the RVLM of young and aged F344 rats? *Neuroscience Letters*. 687: 223-233, 2018.

4. The acute responsivity of the sympathetic nervous system (SNS) is a primary regulatory feature of this nervous system and plays a key role in maintaining physiological homeostasis in response to episodes of acute physical stress. Hyperthermia is a potent activator of sympathetic nerve outflow, and SNS dysfunction and cardiovascular regulatory alterations contribute to the pathophysiological consequences of heat stroke. Our recent studies have focused on understanding rostral ventral lateral medullary (RVLM) mechanisms mediating visceral sympathetic activation to acute heat stress. These publications demonstrate that maintenance of sympathetic activation during heating is dependent on the integrity of RVLM neural circuits,

and highlight the dynamic nature and intrinsic regulatory complexity of RVLM sympathetic neural circuits. These studies provide fundamental information that may contribute to understanding central neural mechanisms mediating altered SND regulation in heart failure patients and aged subjects. I served as the primary investigator in all of these studies.

a. Hosking KG, Fels RJ, and Kenney MJ. Role of the rostral ventral lateral medulla in mediating visceral sympathoexcitation to acute heat stress. *Autonomic Neuroscience: Basic and Clinical*. 150: 104-110, 2009.
b. Kenney MJ, Meyer CN, Hosking KG, and Fels RJ. Is visceral sympathoexcitation to heat stress dependent on activation of ionotropic excitatory amino acid receptors in the rostral ventral lateral medulla? *Am J Physiol Regulatory Integrative Comp Physiol* 301(2): R548-R557, 2011.
c. Kenney MJ, Ganta CK, and Fels RJ. Disinhibition of RVLM Neural Circuits and Regulation of Sympathetic Nerve Discharge at Peak Hyperthermia. *J Appl Physiol* 115: 1297-1303, 2013.
d. Kenney MJ. Medullary regulation of visceral sympathetic nerve discharge at peak hyperthermia in aged F344 rats. *Autonomic Neuroscience: Basic and Clinical*. 186: 32-37, 2014

D. Additional Information: Research Support and/or Scholastic Performance

Ongoing Research Support

5R01AG041948-04 Kenney (PI) 09/15/2012-05/31/2019
NIH/NIA
Sympathetic Neural Regulation and Aging: Medullary Mechanisms and Strategies
The objective of the present Research Plan is to determine how advancing age, and the transition from a healthy aged state to senescence, alters medullary mechanisms regulating SND under basal conditions and in response to acute physical stress.
Role: PI

2T32OD011169 Kenney (PI) 09/30/2015-05/31/2020
NIH/Office of the Director
BRITE Veterinary Student Program
The BRITE veterinary student program is designed to expose DVM students to hypothesis-driven research activities, methodologies involved in design and execution of laboratory experiments and ethical issues pertinent to biomedical research, at a formative stage of their veterinary education. The training grant has been transferred to other K-State personnel.
Role: PI

Completed Research Support

2R15HL108329-02A1 Poole (PI) 08/01/2015-07/31/2017
NHLBI
Heart Failure & Aging: Mechanistic Bases of Muscle Vascular Dysfunction
The objective of the research is to employ a novel multi-systems approach to address the global hypothesis that, in aged CHF rats, SNS and cardiac dysfunction coalesce within the muscle microcirculation to impair perfusive and diffusive O_2 conductance thereby reducing blood-myocyte O_2 flux and exercise tolerance.
Role: Collaborating Investigator

5T35OD010979 Kenney & Davis (Co-PIs) 04/01/2010-03/31/2018
NIH/Office of the Director
Short-Term Training in Health Professional Schools
This project attracts veterinary students into biomedical research careers by exposing them hypothesis-driven research activities and ethical issues pertinent to biomedical research. The training grant has been transferred to other K-State personnel.
Role: Co-PI