CURRICULUM VITAE

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Position: Professor and Director
Institution: School of Kinesiology, University of Minnesota Twin Cities
Location: 1900 University Avenue, Minneapolis, MN 55455, USA

Education:

East China Normal University	B. S.	07/1976	Physical Education
University of Wisconsin-Madison	M.S.	05/1982	Exercise Physiology
University of Wisconsin-Madison	Ph.D.	05/1985	Exercise Physiology
University of Wisconsin-Madison	Postdoctor	08/1987	Biochemistry, Nutrition

Representative Careers:

Postdoctoral Researcher, Institute for Enzyme Research, University of Wisconsin-Madison	1985-1987
Assistant/Associate Professor, Department of Kinesiology and Division of Nutritional Science, University of Illinois at Urbana-Champaign	1987-1993
Associate Professor, Department of Kinesiology and Interdisciplinary Graduate Program of Nutritional Science, University of Wisconsin-Madison	1993-1997
Professor, Department of Kinesiology and Interdisciplinary Graduate Program of Nutritional Science, University of Wisconsin-Madison	1997-2011
Visiting Professor, Karolinska Institute of Medicine, Stockholm, Sweden	2000
Adjunct Professor, Institute on Aging, Cardiovascular Research Center, UW-Madison	1994-2011
Chairman, Department of Kinesiology, University of Wisconsin-Madison	1994-1997, 2003-2010
Director, School of Kinesiology, University of Minnesota-Twin Cities	2011-2017
Director, Laboratory of Physiological Hygiene and Exercise Science, Univ. of Minnesota	2011-present
Adjunct Professor, Tianjin University of Sport, Jiao Tong University, China	2012-2017
Visiting Professor, University of Valencia Medical College, Spain	2017

Specialty & Present Interest:

Most of my academic career has been occupied by research on free radicals-antioxidants homeostasis which plays a critical role in life. Numerous diseases, health disorders and aging are now identified to be related to oxidative stress caused by imbalance of the two. A central paradigm of my research is to study how this delicate balance can be ameliorated by physical activity and aging. It is known that muscle disuse atrophy shares some common mechanisms with sarcopenia, the age-related loss of muscle mass and function. My laboratory has demonstrated that overexpression of PGC-1α, the master nuclear cofactor, ameliorates mitochondrial biogenesis, autophagy/ mitophagy and fusion/fission dynamics thus improving crosstalk among key signaling pathways and reducing muscle protein loss. We also showed that certain phytochemicals such as oat avenanthramides can modulate redox processes by inhibiting inflammatory pathways thus ameliorating cellular homeostasis and organ health.

Representative papers:

- Kang, C., C.A. Goodman, T. A. Hornberger, L. L. Ji. PGC-1α Over-expression by in vivo transfection attenuates mitochondrial deterioration of skeletal muscle caused by immobilization. FASEB J. 29:4092-2015.
- Kang, C. and L. L. Ji. PGC-1α over-expression via local transfection attenuates mitophagy pathway in muscle disuse atrophy. Free Rad. Biol. Med. 93:32-40, 2016.
- Kang, C., D. Yeo, L. L. Ji. Muscle Immobilization Activates Mitochondrial Autophagy and Disrupt Mitochondrial Dynamics. Acta Physiol. 218(3):188-197, 2016.
- Kang, C., D. W. S. Shin, D. Yeo, W. Lim, T. Zhang and L. L. Ji. Anti-inflammatory effect of avenanthramides via NF-κB pathways in C2C12 skeletal muscle cells. Free Rad. Biol. Med. 2018 Mar;117:30-36. doi: 10.1016/j.freeradbiomed.2018.01.020. Epub 2018 Jan 31. PMID: 29371164
- Yeo, D., C. Kang, M. C. Gomez-Aberar, J. Vina and L. L. Ji. Intensified mitophagy in skeletal muscle with aging is downregulated by in vivo PGC-1α overexpression. Free Rad. Bio. Med. 2018 Nov 2. pii: S0891-5849(18)31333-9. doi: 10.1016/j.freeradbiomed.2018.10.456. PMID:30395971