BIOGRAPHICAL SKETCH

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NAME Abraham D. Lee, Ph.D.,P.T.	POSITION TITLE Associate Professor, Dept. of Rehabilitation Sciences, Physical Therapy Program, The University of Toledo
eRA COMMONS USER NAME (credential, e.g., agency login) ABRAHAMLEE	

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

INSTITUTION AND LOCATION			
Kyungpook National Univ, Daegu, South Korea	B.S.	1975	Physical Education
Yonsei University, Seoul, South Korea	M.S.	1981	Physical Education
Northeastern Illinois University, Chicago, IL	M.S.	1986	Exercise Physiol.
Arizona State University, Tempe, AZ	Ph.D.	1991	Exercise Science
Texas Woman's University, Houston, TX	M.S.	1998	Physical Therapy
Washington University School of Medicine, St. Louis, MO	Post-doctoral training	1991-1995	Muscle glucose transport

PROFESSIONAL EXPERIENCE:

1976-1977 Teacher, Buhang Junior High School, Kyungpook, South Korea
1982-1986 Instructor, Rena Inc., Chicago, IL
1986 (April-August) Intern, Cardiac Rehab, Swedish Covenant Hospital, Chicago, IL
1986-1991 Graduate Research Assistant, Arizona State University, Tempe, AZ
1991-1995 Postdoctoral Fellow, Washington University School of Medicine, St. Louis, MO
1995-1996 Instructor, Dept. of Biological Sciences, Ohio University, Athens, OH
1996-1998 Research Associate, School of Physical Therapy, Texas Woman's University, Houston, TX
1999-2000 Physical Therapist (Part time), MCO Hospital & MCO Works,, Toledo, OH
1999-2007 Assistant Professor, Dept. of Physical Therapy, The University of Toledo, Toledo, OH.
2008-present Associate Professor, Dept. of Rehabilitation Sciences, the University of Toledo, Toledo, OH

HONORS:

1988-1989 Arizona State University Regents Graduate Academic Scholarship
1988 Arizona State University Graduate College Student Advisory Board Travel Grant
1989 Arizona State University Graduate College Student Advisory Board Travel Grant
2003 Summer Research Travel Award by The Federation of American Societies for Experimental Biology MARC Program
2003 Certificates of Recognition by Ohio Physical Therapy Association for contribution to Physical Therapy Research with peer-reviewed publication, national & local level presentations.
2004 Certificates of Recognition by Ohio Physical Therapy Association for contribution to Physical Therapy Research with peer-reviewed publication, national & local level presentations.
2004 Award for best platform presentation abstract by Ohio Physical Therapy Association.
2005 The Federation of American Societies for Experimental Biology and the Minority Access to Career Research: Travel Award for the Grantsmanship Training Program, Tucson, AZ, June 23-25
2006 The Federation of American Societies for Experimental Biology and the Minority Access to Career Research: Summer Research Travel Award for the meeting entitled, "AMPK: Impact on Mammalian Metabolism and Disease" Snowmass Village, CO, August 12-17
2007 Keynote speaker, "New concept of metabolic syndrome for physical therapy," in Taipei, Taiwan, School of Physical Therapy, National Taiwan University, Taipei, Taiwan, November 12-13
2011 Invited speaker, "Rehabilitation strategies for patients with diabetes mellitus and associated complications: treatment and prevention", American Physical Therapy Association, Combined Section Meeting Chicago, IL

2014 Invited speaker, “Effects of exercise intervention on metabolic abnormalities associated with metabolic syndrome”, American Physical Therapy Association, Combined Section Meeting, Las Vegas, NV
2016 Invited speaker, “Obesity-induced vascular dysfunction: pathophysiology and treatment with exercise”, American Physical Therapy Association, Combined Section Meeting, Anaheim, CA

C. CONTRIBUTION TO SCIENCE:

1. The PI has been interested in the effect of exercise on metabolism. During his post-doctoral training in the Holloszy laboratory at Washington University in St Louis, the PI investigated effects of hormonal and cellular signaling molecules on the role of insulin action in the regulation of glucose transport in skeletal muscle.

a. *Abraham D. Lee*, Eric A. Gulve, May Chen, and John O. Holloszy, (1995) Effects of Ca^{2+} ionophore ionomycin on insulin-stimulated and basal glucose transport in muscle. *Am. J. Physiol*. 268: R997-R1002.

b. *Abraham D. Lee*, Polly A. Hansen and John O. Holloszy, (1995) Wortmannin inhibits insulin-stimulated glucose transport but not contraction-stimulated glucose transport in skeletal muscles. *FEBS letters* 361: 51-54.

c. *Abraham D. Lee*, Polly A. Hansen, Jane Schluter, Eric Gulve, Jiaping Gao and John O. Holloszy, (1997) The effect of epinephrine on insulin-stimulated glucose uptake and GLUT-4 phosphorylation. *Am. J. Physiol*. 273: C1082-C1087.

2. The major focus of the Lee’s laboratory is to investigate the mechanisms underlying exercise training-induced-improvement in insulin action in the whole body and in isolated insulin target tissues. To this end, the laboratory follows two approaches. **First,** developing an animal genetic model to determine the extent of genetic contribution to insulin action. The Lee’s laboratory has recently observed that training improves insulin action in rats with heterogenetic backgrounds, and that this response is a heritable phenotype with heritability of ~25%. **Second,** utilizing available insulin-resistant animal models to determine the role of contraction-mediated pathways in modulating insulin action.

a. *Abraham D. Lee* and Abram Katz, (1989) Transient increase in glucose 1,6-bisphosphate in human skeletal muscle during isometric contraction. *Biochem. J*. 258: 915-918

b. Yasuo Kida, Abram Katz, *Abraham D. Lee* and David M. Mott, (1989) Contraction-mediated inactivation of glycogen synthase is accompanied by inactivation of glycogen synthase phosphatase in human skeletal muscle. *Biochem. J.* 259: 901-904

c. Koch LG, Green CL, *Lee AD,* Hornyak JE, Cicila GT, Britton SL., “Test of the principle of initial value in rat genetic models of exercise capacity” *Am. J. Physiol* 288:R466-72, 2005

d. Thomas A. Bowman, Sadeesh K. Ramakrishnan, Meenakshi Kaw, Sang Jun Lee, Payal R. Patel, Varun K. Golla, Raymond E. Bourey, Per Magnus Haram, Lauren G. Koch, Steven L. Britton, Ulrik Wisløff, *Abraham D. Lee,* and ***Sonia M. Najjar***, “Caloric Restriction Reverses Hepatic Insulin Resistance and Steatosis in Rats with Low Aerobic Capacity” *Endocrinology* 151: 5157–5164, 2010.

3. The Lee’s team has recently begun collaborations with Dr. McLoughlin in the University of Toledo, focusing on the role of FoxO1 in regulating glucose and fat metabolism using a mouse model in which FoxO1 is overexpressed in skeletal muscle. The team has found that overexpressing FoxO1 in skeletal muscle plays a minimal role in glucose metabolism but it causes a drastic decrease in triglyceride level and fatty acid oxidation in skeletal muscle. The collaborative team is actively engaged in investigating the underlying mechanisms.

a. *Abraham D. Lee,* Aaron M. Al-sorghali, Carrie E. Woods, Anthony J. Rampulla, and Thomas J. McLoughlin, Hepatic glycolytic capacity decreases in mice with a FoxO1 overexpression in skeletal muscle (Abstract), *The FASEB Journal* 27:1202.23, 2013

4. The Lee's team has collaborated closely with the Najjar team since 2000, sharing laboratory space and equipment in the last 10 years. The PI is very familiar with the line of research pursued in the Najjar laboratory and has worked very closely with her, co-mentoring several of her pre-doctoral students and post-doctoral trainees. Thus, he is well positioned to serve as an interim PI on her NIH grant during the time she transitions out into a sister institution, the Ohio University.

In collaboration with the Najjar laboratory, the Lee team has observed a strong role for hepatic CEACAM1 in regulating overall insulin action. Based on a recent observation that overexpressing hepatic CEACAM1 plays a key role in preventing the development of insulin resistance in skeletal muscle in response to high-fat intake, the Lee team is pursuing research on how hepatic CEACAM1 can mediate changes in insulin action after endurance exercise training. To this end, the Lee laboratory will continue to use the genetically modified mouse models of insulin resistance that the Najjar laboratory has generated, per NIH support.

a. M.N. Poy, Y. Yang, M.A. Fernstrom, *A.D. Lee*, Y. Kido and ***S.M. Najjar*****,** "CEACAM1 regulates insulin clearance in liver" *Nature Genetics* 30:270-276, 2002.

b. Tong Dai, Qusai Al-Share, Yan Yang, Mats A. Fersstrom, *Abraham D. Lee*, Lawrence Sweetman, Antonio Amato, Marzia Pasquale, Gary D. Lopaschuk, Sandra K. Erikson, and ***Sonia M. Najjar***, (2004) "Interaction between altered insulin and lipid metabolism in CEACAM1-inactive transgenic mice" *J. Biol. Chem.* 279:45155-45161

c. Garrett Heinrich, Sumona Ghosh, Anthony M. Deangelis, Jill M. Schroeder Gloeckler, Payal R. Patel, Tamara R. Castaneda, Shane Jeffers, Abraham D. Lee, Dae Young Jung, zhiyou Zhang, Darren M. Opland, Martin G. Myers Jr., Jason K. Kim, and ***Sonia M. Najjar***, "Carcinoembryonic Antigen-Related Cell Adhesion Molecule 2 Controls, "Energy Balance and Peripheral Insulin Action in Mice" *Gastroenterology* 139:644–652, 2010

d. Qusai Y. Al-Share, Anthony M. DeAngelis, Sumona Ghosh Lester, Thomas A. Bowman, Sadeesh K. Ramakrishnan, Simon L. Abdallah, Lucia Russo, Payal R. Patel, Meenakshi K. Kaw, Christian K. Raphael, Andrea Jung Kim, Garrett Heinrich, *Abraham D. Lee*, Jason K. Kim, Rohit N. Kulkarni, William M. Philbrick, and ***Sonia M. Najjar***, Forced hepatic overexpression of CEACAM1 curtails diet-induced insulin resistance, *Diabetes* 64:2780-2790, 2015

D. RESEARCH SUPPORT/GRANT:

1999-2013 **Abraham D. Lee** (P.I.) $100,000
Ohio Challenge Fund from Medical College of Ohio, Toledo, OH

Sept,2005 – Aug, 2007 **Abraham D. Lee** (10% effort, co-investigator) $383,522
Sonia M. Najjar (Co P.I.), and Marcia F. McInerney (Co P.I.)
US Department of Agriculture
"Dietary and genetic risk factors in obesity and diabetes"

2012-2013 **Abraham D. Lee** (P.I.) $12,893
Judith Herb College of Education, Health Science, and Human Service, The University of Toledo,
"Does the Overexpression of Foxo1 in Skeletal Muscle Alter Insulin Sensitivity of the Body?"