RUTGERS
School of Graduate Studies

Office of Student Affairs and Admissions
SGS at New Jersey Medical School
Rutgers, The State University of New Jersey
185 South Orange Avenue, MSB C-696
Newark, NJ 07103

http://njms.rutgers.edu/gsbs/
p. 973-972-4511
f. 973-972-7148

YOU ARE INVITED TO ATTEND THE

DEFENSE OF THE DOCTORAL

DISSERTATION

# "Adiponectin via AMPK enhances aerobic respiration through activating succinate dehydrogenase in cardiac myocytes"

by

Yong Heui Jeon
Cell Biology, Neuroscience and Physiology Program

MS 2012, Yonsei University College of Medicine, South Korea
BSc. 2009, Catholic University of Korea, South Korea

Thesis Advisor:

Maha Abdellatif, MB.ChB., Ph.D.
Department of Cell Biology and Molecular Medicine

Wednesday, October 9th, 2019
12:00 P.M.
MBS, H609B

# ABSTRACT

Adiponectin (Adn) is one of the most abundant circulating hormones, which through adenosine monophosphate-activated protein kinase (AMPK), enhances fatty acid and glucose oxidation, and exerts a cardioprotective effect. However, its effects on cellular bioenergetics have not been explored. We have previously reported that AMPK enhances mitochondrial respiration through a succinate dehydrogenase (SDH or complex II)-dependent mechanism in cardiac myocytes, leading us to predict that Adn would exert a similar effect via activating AMPK. Our results show that Adn increased basal mitochondrial oxygen consumption rates (OCR), ATP production, and spare respiratory capacity (SRC), which were all abolished by inhibition of SDH assembly, via the knockdown of the SDH assembly factor 1 (SDHAF1), inhibition of SDH activity, or inhibition of AMPK. Additionally, Adn alleviated hypoxia-induced reductions in OCR and ATP production, in a SDHAF1-dependent manner, whereas overexpression of SDHAF1 confirmed its sufficiency for mediating these effects. Importantly, the levels of holoenzyme SDH under the various conditions correlated with OCR. We also show that the effects of Adn, AMPK, SDHAF1, as well as, SDH assembly all required sirtuin 3 (Sirt3). In conclusion, Adn potentiates mitochondrial bioenergetics via promoting SDH assembly in an AMPK-, SDHAF1-, and Sirt3-dependent fashion in cardiac myocytes.