Organelle Stress and Metabolic Diseases

Fall, 2017 (106/9/11 updated)

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Time: Wed. 4:10~6:00pm, Rm#7023 (7F 教室)

Grading Policy: Presentation/Report 50%, Discussion/Participation 50%

This is a one-semester two-credit course. We will introduce the concept of the metabolic diseases and select several critical topics related to organelle stress on the metabolism-related diseases from the top journals. The course will include lecture and presentation/discussion from students. Students will be divided into 7 groups. Each group should introduce the ***background*** related to the current topic in organelle stress & metabolic disease, ***experimental design***, ***key methodology and tools***, and finally the ***assigned article***.

Date	Topic	Lecturer
9/20	Course orientation	蔡曜聲
9/27	Clinician view on metabolic diseases	吳至行
10/11	Post-translational modification	呂佩融
10/18	Autophagy	徐麗君

10/25 Post-translational modification (article discussion) 蔡曜聲 (Group 1)

Mitochondrial ROS regulate thermogenic energy expenditure and sulfenylation of UCP1. Nature. 2016 Apr 7;532(7597):112-6.

11/1 Autophagy (article discussion) 蔡曜聲 (Group 2)

Autophagy in the CNS and Periphery Coordinate Lipophagy and Lipolysis in the Brown Adipose Tissue and Liver. Cell Metab. 2016 Jan 12;23(1):113-27.

11/8 Innate immunity to inflammation 張志鵬

11/15 Inflammasome (article discussion) 蔡曜聲 (Group 3)

NOX4-dependent fatty acid oxidation promotes NLRP3 inflammasome activation in macrophages. Nat Med. 2016 Sep;22(9):1002-12.

11/22 ER stress 洪建中

11/29 ER stress in metabolic diseases (article discussion) 蔡曜聲 (Group 4)

Targeting ABL-IRE1α Signaling Spares ER-Stressed Pancreatic β Cells to Reverse Autoimmune Diabetes. Cell Metab. 2017 Apr 4;25(4):883-897.

12/6 Hypoxia 林世杰

12/13 Oxidative stress 顏賢章

12/20 Hypoxia in metabolic diseases (article discussion) 蔡曜聲 (Group 5)
Mitochondrial Protein Lipoylation and the 2-Oxoglutarate Dehydrogenase Complex Controls HIF1α Stability in Aerobic Conditions. Cell Metab. 2016 Nov 8;24(5):740-752.

12/27 Oxidative stress (article discussion) 蔡曜聲 (Group 6)
Mitochondrial calcium uptake underlies ROS generation during aminoglycoside-induced hair cell death. J Clin Invest. 2016 Sep 1;126(9):3556-66.

1/3 Mitochondrial stress 李佳榮
1/10 Mitochondrial stress (article discussion) 蔡曜聲 (Group 7)
Control of mitochondrial function and cell growth by the atypical cadherin Fat1. Nature. 2016 Nov 24;539(7630):575-578. [Supplement required]

Gordon Research Conference
Stress Proteins In Growth, Development & Disease
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All organisms are exposed to harsh conditions. These harsh conditions include environmentally imposed stresses such as elevated temperature and irradiation, physiological stresses such as rapid cellular proliferation, oxidative stresses due to metabolic reactions, and pathophysiological stresses such as pharmacological agents, infection, and *inflammation*. Even normal developmental or *nutritional changes* exert stresses as systems temporarily depart from and try to re-establish homeostasis. If unmitigated, stress can lead to protein misfolding and aggregation, and cell death. Recent studies suggest that the ability to sense and respond to stress is critical for normal cell growth and development, and helps protect against diseases that include cancer, cardiovascular disease, *metabolic disease (e.g., diabetes)* and liver disease, and protein folding diseases such as Alzheimer's, Huntington's and prion-based disease. Studies in model systems have helped establish these principles and suggest a correlation between *longevity* and the ability to mount stress responses. There is also an increasing appreciation that the stress response can be pharmacologically modulated, and thus diseases that arise from these phenomena might be selectively targeted.