FBSセミナーのお知らせ

日時：2013年10月21日（月）
15:00〜16:00

場所：ナノバイオロジー棟3階セミナー室

講演者：山野 晃史 先生
（NIH, Postdoctoral Fellow）

演題：Molecular mechanism for Parkin/PINK1-mediated selective mitochondrial autophagy

山野晃史先生は、ミトコンドリア・ダイナミクス研究分野の最先端拠点のひとつであるRichard Youle研究室（National Institute of Neurological Disorders and Stroke, National Institutes of Health, USA）に所属しており、選択的オートファジーによるミトコンドリア品質管理機構の研究を行なっておられます。この仕組みの鍵分子であるParkinとPINK1は、神経変性疾患であるパーキンソン病の原因因子として広く知られています。これらのタンパク質がどのように機能しているかを解き明かすことにより、病態の理解と治療法の開発につながるものと期待されています。本セミナーでは、選択的ミトコンドリア・オートファジーの新規因子に関する未公表データにも触れられる予定です。この機会に、「フロンティア研究の興奮」と「NIHで奮闘する若手研究者の情熱」をお見逃しなきよう、是非ご来聴ください。

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Title
Molecular mechanism for Parkin/PINK1-mediated selective mitochondrial autophagy

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Abstract
Mitochondria are essential organelles of eukaryotic cells that produce energy in the form of ATP. However, as highly reactive oxygen species are continually generated as a byproduct of electron transport during oxidative phosphorylation, the cells have to equip sophisticated quality control systems to eliminate damaged mitochondria. We have identified a dedicated pathway involved in the selective clearance of damaged mitochondria (mitophagy) composed of the proteins Parkin and PINK1, and have been investigating the molecular mechanism that underlies this process. Parkin and PINK1 are known as the gene products mutated in autosomal recessive forms of familial Parkinson's disease. Therefore, it attracts increasing attention not only from a field of basic research, but also from clinical and therapeutic aspect.

PINK1 is a mitochondrial serine/threonine kinase whose protein level is kept very low in healthy mitochondria. In sharp contrast, when mitochondria sustain damage that leads to a loss of membrane potential, PINK1 stabilizes on the outer membrane where it gets activated and recruit E3 ubiquitin ligase Parkin from the cytosol onto the mitochondria. This triggers ubiquitination of a broad range or mitochondrial outer membrane proteins and recruitment of autophagy related proteins followed by the elimination of damaged mitochondria by autophagy.

In this seminar, I am going to describe 1) how PINK1 is rapidly degraded in healthy mitochondria and 2) how PINK1 accumulates on the outer membrane of the damaged mitochondria. Finally, I will show you newly identified mitophagy-specific factors that are important for encapusulation of mitochondria by the autophagosome.