

## 令和7年度 第8回

## 対面で実施

## 応用動物科学セミナー

## Mitochondrial peptide MOCCI finetunes Complex IV to modulate infection outcomes Dr. Cheryl Lee

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使用言語·Language:英語·English

● 2025年11月14日(金) 16:00 ~フードサイエンス棟講義室

● 本セミナーは動物科学のフロンティア(修士課程)/ 動物科学フロンティア(博士課程)の講義1回分と しても認定されます。履修者は、セミナーの 内容についてレポートを作成の上(様式は自由)、 セミナー終了後2週間以内にUTOLで提出すること。 なお、弥生キャンパス外に所属する学生など、 現地参加がどうしても困難な場合に限り、 Zoomでの参加を認めます。希望者は、 前日までに下記担当教員に連絡すること。

担当教員:

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Mito-SEPs are small open reading frame-encoded peptides that localize to the mitochondria. Through ribosome profiling and RNA-sequencing of human aortic endothelial cells, we observed that the expression of mito-SEPs is negatively correlated with inflammation. Using a pipeline developed in the lab to identify novel mito- SEPs, we identified mito-SEPs upregulated during inflammation, termed i-mito-SEPs We report the discovery and deorphanization of Modulator of Cytochrome C oxidase during Inflammation (MOCCI), a 83 aa mito-SEP that is specific to the inflamed state. MOCCI is a paralog of NADH: Ubiquinone Oxidoreductase Complex Assembly Factor 4 (NDUFA4), the 14th subunit of complex IV (CIV) in the electron transport chain (ETC). During inflammation, MOCCI replaces NDUFA4 in CIV, which leads to repressed CIV activity, lower membrane potential and reduced ROS production. Interestingly, knocking out of MOCCI in mouse monocytes results in altered mitochondrial metabolism, suggesting a novel ETC in monocyte activation. We propose that mito-SEPs are important immunomodulators, of which one of the mechanisms is through altering mitochondrial ETC activity.