## 大学院特別講義

(医歯学先端研究特論) (生命理工学先端研究特論)(医歯理工学先端研究特論)

下記により大学院特別講義を行いますので、多数ご来聴下さい。

記

- 講 師: Paul Fraser, Professor, University of Toronto
- 演題: Modelling Genetic Modifiers of Neurodevelopment and Neurodegeneration
- 日 時: 平成 26 年 12 月 9 日 (火) 19 時 00 分~21 時 00 分
- 場 所: M&D タワー 21 階 大学院講義室1

## 抄 録:

Genome-wide association studies (GWAS) have identified a number of at-risk genes for several neurodegenerative disorders including Alzheimer and Parkinson disease. These gene candidates are associated with cellular pathways involving protein trafficking, endocytosis, cholesterol management and immune responses. Their impact on disease pathogenesis has been investigated in transgenic and knockout mouse models to determine how they contribute to protein misfolding and aggregation. Alzheimer disease (AD) is caused by a number of different factors and one of the principal pathological endpoints is the deposition of amyloid- $\beta$  (A $\beta$ ) as senile plaques. We have generated new models to investigate further the AD-related GWAS risk factors and their impact on amyloid formation and clearance. These studies reveal a complex relationship that affects both the uptake of amyloid aggregates as well as changes in A $\beta$  processing and production. Examination of this next generation of animal models will provide a more complete understanding of AD and provide valuable tools for the development and validation of new therapeutic approaches.

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