# 令和 5 年度　第 48 回　大学院セミナー

令和 5 年 9 月 26 日

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| 分 野 名<br>Area of Research<br>(責任者名)(内線) | バイオメディカルモデル動物学 分野<br>責任者名( 小林　篤史 )　　内線( 7132 ) |
| 演　　題<br>Title | Determining the role of glial cells in CNS prion disease pathogenesis |
| 講 師 等<br>Presenter | Prof. Neil A. Mabbott<br>The Roslin Institute & Royal (Dick) School of Veterinary Sciences<br>University of Edinburgh |
| 概要<br>Abstract | Prion diseases, also known as transmissible spongiform encephalopathies, are sub-acute neurodegenerative diseases of humans and certain mammalian species. Infectious prions are considered to comprise entirely from $PrP^{Sc}$, misfolded isoforms of the host cellular prion protein. During disease the build-up of prion-specific $PrP^{Sc}$ in the brain leads to the development of spongiform pathology and neurodegeneration, and coincides with extensive microglial and astrocytic activation in targeted regions. The microglia are the resident macrophages of the CNS, and microgliosis is a prominent histopathological feature during the development of CNS prion disease. Microglia have been attributed essential functions in CNS development and homeostasis, but their activation during some CNS disorders can lead to the development of neuropathology. The partial ablation or partial deficiency in microglia has been shown to enhance the accumulation of prions in the brain and accelerates the onset of clinical disease. This has led to the suggestion that microglia provide neuroprotection during CNS prion disease by engulfing and destroying prions. Astrocytes are also important glial cells in the brain that provide homeostatic support to neurons in the steady state, but can undergo neurotoxic reactive activation following brain injury, during some neurodegenerative disorders and aging. Prion diseases also induce extensive neurotoxic reactive astrocyte activation, and the level of activation inversely correlates with disease duration. I will present data to show that the microglia provide neuroprotection independently of $PrP^{Sc}$ clearance during prion disease and restrict the harmful activities of reactive astrocytes. Infections with prions ultimately cause chronic neurodegenerative diseases to which there are no treatments. Since astrocytes can contribute to both prion replication and synaptic loss in infected brains, identifying the microglia-derived factors that help to prevent these activities would have therapeutic potential during CNS prion disease and other important neurodegenerative disorders. |
| 開 催 日 時<br>Date and Time | 令和 5 年　10 月　30 日（月）<br>16:00 ～ 17:00 |
| 開 催 方 法<br>Online/Face to face | ポンペ会館セミナー室<br>Seminar Room, Pompe Hall 1F |
| 備　　考<br>Notes | 共催 ： 長崎大学大学院医歯薬学総合研究科　脳科学ユニット |

■先端医療科学特論（基礎編）　□先端医療科学特論（臨床編）
□先端新興感染症病態制御学特論　□先端放射線医療科学特論
□日本語（Japanese）　■英語（English）
■対面（Face to face）　□オンライン(Online)