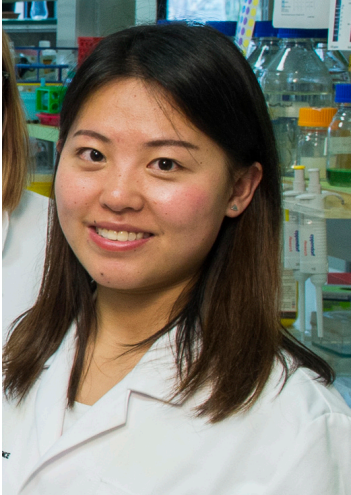


2017 Seminar Series – PhD Oration



**Wednesday 6th of September
12-1pm**

**Bio21 Institute Auditorium
30 Flemington Road, Parkville**

Wenting Zhao

Hill Laboratory,
Department of Biochemistry and Molecular
Biology, The University of Melbourne
& LIMS, La Trobe University

Investigating the functional roles of miR-29b/miR-146a in Prion disease

Neurodegenerative diseases such as Alzheimer's, Parkinson's and the prion diseases are closely related with specific gene and protein dysfunction. Prion diseases, also known as transmissible spongiform encephalopathies (TSE), are characterized by the structural transformation of PRNP encoding prion protein (PrP^C) to abnormal form (PrP^{Sc}).

MicroRNAs (miRNAs) are a class of small non-coding RNAs that regulate target gene or protein expression by targeting mRNAs and triggering either translational repression or mRNA degradation. Distinct miRNA signatures, including miR-29b and miR-146a, have been detected in various biological fluids and tissues from Prion disease patients, and in both cell and animal models. They could be potential diagnostic biomarkers of Prion disease, and investigating miRNA roles and miRNA-target regulation pathways will improve our understanding of disease regulation networks.

CRISPR/Cas9 system, miRNA mimics, and next-generation sequencing techniques are employed in this project to investigate miR-29b and miR-146a functions and regulations in Prion disease. Both miR-29b and miR-146a are shown to regulate prion protein expression, through different ways, however, they potentially share a partially similar mechanism involving miRNA functional complex. miR-29b and miR-146a can also impact the generation of abnormal form of prion protein in prion disease cell models, suggesting the therapeutic potential of these two miRNAs in treating Prion disease.