Role of PIWI/piRNA complex in neuronal differentiation and microglial activation

For PhD Qualifying Examination (PQE) "Oral Component" – Open Seminar

Neurodegeneration is characterized by loss of neurons in brain and spinal cord. Cell replacement therapy and regulation of microglia-mediated chronic inflammation are the two major approaches to treat neurodegenerative disorders. piRNAs are a class of small non-coding RNAs that bind to PIWI proteins. The exact role of the complex in brain is still unknown. Thus we aim to study the role of PIWI/piRNA complex in neuronal differentiation and in excessive neuroinflammation. During the course of differentiation, a particular PIWI homolog, is found to be increasing. Silencing of that specific PIWI protein suppressed early neuronal markers even in the presence of all-trans retinoic acid (RA), a neuronal differentiation-inducing agent. We have shown the interaction of PIWI with few histone modifying enzymes. Thus, a possible mechanism by which PIWI controls gene expression in neuronal differentiation could be through epigenetic regulation. We also observed that overexpression of a particular piRNA restored the expression of stemness-related genes in RA treated cells. This implies that PIWI/piRNA complex plays a significant role in neuronal differentiation.

During microglial activation, a particular PIWI homolog is increased. With the knockdown of PIWI protein in lipopolysaccharide (LPS) – stimulated microglial cell line, several pro-inflammatory genes were significantly down-regulated with decreased nitric oxide production and reduced neuronal cell death. This shows that PIWI could act as a pro-inflammatory factor. We have also identified a piRNA that was regulated with microglial activation. Thus, by understanding the role of PIWI/piRNA complex, we will be able to enhance neuronal differentiation and neuroprotection under pathological conditions and benefit patients suffering from neurodegenerative diseases.

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DEPARTMENT OF ANATOMY

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