Dr Parthiv Haldipur
Seattle Children’s Research institute, USA

Disrupted cerebellar development caused by aberrant mesenchymal signaling likely represents a unifying developmental mechanism for human Dandy-Walker malformation

Friday 3rd November 2017 1pm
June Lloyd room (PUW4), ICH

Abstract
Chondroitinase ABC (ChABC) promotes functional neuroplasticity after CNS injury by degrading extracellular matrix inhibitors. Although large scale ChABC delivery can be achieved via a gene therapy approach, it has not previously been possible to control gene expression, and prior experimental systems for gene regulation are likely to be incompatible with the non-resolving adaptive immune response known to occur following spinal cord injury. I will present data where we apply a novel immune-evasive dual vector system, in which the ChABC gene is also under a doxycycline inducible regulatory switch (dox-i-ChABC) and demonstrate control of ChABC gene expression in the adult rat injured spinal cord, revealing temporally-dependent therapeutic effects.

Biosketch
Parthiv Haldipur is a postdoctoral researcher working with Prof Kathleen Millen at Seattle Children's Research Institute and the University of Washington. During his PhD, at the National Brain Research Centre, in India, he studied the role of sonic hedgehog signaling in cerebellar development, including symmetric asymmetric divisions in granule cell precursors. He also studied cerebellar development in preterm infants. His interest in human cerebellar development led him to move to Seattle where he began studying the molecular and genetic basis of cerebellar development with particular emphasis on Dandy-Walker malformation (DWM), the most common cerebellar malformation in humans. His work has contributed to a growing body of evidence supporting a paradigm shift. The brain does not develop in isolation. Rather, the head mesenchyme exerts considerable influence on early embryonic brain development. More recently, he has embarked on studies of foetal pathology of cerebellar malformations. This study also led to the discovery of many features of normal human cerebellar development that have never been described and are not shared with model organisms. He has since initiated a collaboration with HDBR and Dr Paula Alexandre at DBC, UCL to carry out a parallel comprehensive study of normal human embryonic and foetal development.