

Seminar

Modelling human PI3K-related brain malformations and epilepsy – time, cause and treatment

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Mammalian development involves complicated cell morphogenesis and highly regulated interactions between transcription factors and signaling molecules. The phosphoinositide 3-kinase (PI3K) pathway is one such central player of intracellular signaling, crucial for cell metabolism, growth and death. Activating mutations in the PI3K pathway have long been linked to cancer. Strongly activating PI3K mutations found most commonly in cancer (hotspot mutations) also result in severe brain overgrowth and dysplasia; and can often cause intellectual disabilities, autism and intractable epilepsy.

Following a brief overview of my PhD thesis on early forebrain development, I will present my postdoctoral work that identified basic cellular mechanisms and critical developmental periods behind PI3K-related brain overgrowth and dysplasia in conditional mouse models. In addition, I will discuss new drug trials for acute anti-epileptic treatment and our ongoing investigation on the promising new therapeutic avenues for intractable pediatric epilepsy patients.

Monday, Sep 11th 2017

11:00 AM (Tea/Coffee at 10:45 AM)

Auditorium, TIFR-H (FReT-B)