



INDIAN INSTITUTE OF TECHNOLOGY GUWAHATI  
PhD-17 SHORT ABSTRACT OF THESIS

Name of the Student : VIJAY SINGH BOHARA

Roll Number : 196106107

Programme of Study : Ph.D.

**Thesis Title: Mechanistic understanding of glycolysis in Japanese encephalitis virus infected neuronal cells with special reference to hypoxia-inducible factor-1 $\alpha$  and pyruvate Kinase M2**

Name of Thesis Supervisor(s) : Prof. Sachin Kumar

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**SHORT ABSTRACT**

Viruses impose substantial metabolic stress on the host cells, driving the increased biosynthesis of nucleotides, lipids, and proteins required for supporting rapid viral replication in the infected cells. In the current study, we have shown that Japanese encephalitis virus (JEV) profoundly reprograms neuronal metabolism by stimulating glycolysis. This glycolytic reprogramming was mediated by the Hypoxia-inducible factor-1 (HIF-1 $\alpha$ ) dependent upregulation of glycolytic enzymes. Mechanistically, JEV infection is associated with reactive oxygen species (ROS) accumulation in the infected cells which contributes to the stabilization of HIF-1 $\alpha$ , thereby inducing the transcription of glycolytic genes and sustaining glycolytic flux. We further identify pyruvate kinase isoform M2, as a novel host factor regulating JEV replication in neuronal cells. PKM2 regulates JEV replication through STAT3 activation, leading to modulation of proinflammatory cytokines such as TNF- $\alpha$  and IL-1 $\beta$ . Additionally, we demonstrate the cellular co-localization and interaction of PKM2 with the NS1 protein of JEV, although the functional implication of this interaction remains to be elucidated. Collectively, the study provides insight into the mechanism underlying the JEV-induced glycolytic reprogramming in neuronal cells and highlight key metabolic enzymes as potential targets for antiviral therapeutic strategies.