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BRIEF SUMMARY OF THE RESEARCH (CITATION)

I hereby certify that the research work entitled “**Understanding the mechanism of host deacetylases SIRT1 and SIRT3 in the modulation of *Salmonella* pathogenesis**” has been conducted by **Ms. Dipasree Hajra** as a part of her doctoral thesis under my supervision in my **Molecular Pathogenesis Laboratory** at the **Department of Microbiology and Cell Biology, Indian Institute of Science**.

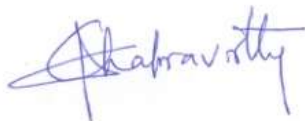
Ms. Dipasree Hajra, have tried to address, the role of Sirtuins in the modulation of the immune metabolism about Salmonellosis which is largely unknown. Here, Dipasree investigated the role of two important Sirtuins, SIRT1 and SIRT3 in the modulation of *Salmonella* pathogenesis and showed that *Salmonella*-induced modulation of SIRT1 and SIRT3 is crucial for governing immune-metabolic switch in the host and this switch influences the metabolic profile of

intracellular *Salmonella* thereby impacting bacterial intracellular replication. Her study indicated the ability of the live *Salmonella* Typhimurium to differentially regulate the levels of SIRT1 and SIRT3 for maintaining the high glycolytic metabolism and low fatty acid metabolism in *Salmonella*. Upon SIRT1 or SIRT3 knockdown or inhibition, the metabolism in intracellular *Salmonella* switched to high fatty acid oxidation and low glycolysis.

Dipasree further demonstrated that SIRT1 and SIRT3 played a role in modulating mitochondrial bioenergetics and dynamics during *Salmonella* infection in macrophages and further skews the intracytoplasmic pH of both host and the intracellular bacteria. She showed that there is a decline in mitochondrial bioenergetics, and the *S. Typhimurium* infected macrophages depict alteration in mitochondrial dynamics with increased mitochondrial fission and mitophagy alongside decreased mitochondrial fusion dynamics.

Date-12-08-24

Regards,



Prof. Dipshikha Chakravorty

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