Mahapa, A.; Samanta, G. C.; Maiti, K. G. Chatterji, D.; Jayaraman, N., 2019, "Mannopyranoside glycolipids inhibit mycobacterial growth, biofilm and potentiate isoniazid inhibition activities in *M. smegmatis*", *ChemBioChem*., *20*, 1966 – 1976.

Persistence against harsh environmental conditions is a survival strategy in the life cycle of a pathogenic mycobacterium. A consequence of the adaptation of a mycobacterium to changing environments is the multicellular aggregation and the formation of a biofilm. Biofilm represents a phenotype change and is physiologically distinct from planktonic cells. Biofilm grown mycobacterial cells are resistance to antibiotics, unlike the planktonic cells. Sustained efforts are on-going to unravel the biofilm phase of a mycobacterium and to ameliorate pathogenicity arising from the biofilm grown cells that are harder to overcome by drugs known for freely-swarming planktonic mycobacterium. In a sustained research over a decade, we have established that synthetic glycolipids that have essential components of lipoarabinomannan are efficacious inhibitors of not only mycobacterial growth, but also the biofilm growth. Lipodomics profiling has shown a down-regulation of many cell wall components, such as, phohphatidylinositol mannosides, alpha- and keto-mycolic acids. Arabinan as the only sugar components in the glycolipids did not elicit considerable inhibition activities in comparison to arabinomannan glycolipids. The effect of mannan glycolipids is undertaken in the present study. A graded number of mannan moieties is installed in the glycolipids and for comparison, an arabinomannan glycolipid is also undertaken. Chemical syntheses of these glycolipids are accomplished initially, followed by subjecting them to mycobacterial inhibition studies, in both growth and biofilm phases. From the series of studies, we observe that the mannan glycolipids surpass the arabinomannan glycolipid to inhibit the mycobacterial growth, whereas, arabinomannan glycolipids are more efficacious biofilm inhibitors. Thus, we observe an inhibition efficiency altering between the mannan and arabinomannan glycolipids in mycobacetial growth and biofilm phases. Further, we also undertook drug supplementation of an antibiotic along with the glycolipids. Antibiotic isoniazid, which exhibits mycobacterial biofilm inhibition concentration of 80 – 100 μg mL^{-1}, enhances the inhibition efficiency when supplemented with the glycolipids in both the mycobacterial phases. A direct consequence is the significantly reduced inhibition concentration of the antibiotic in the presence of the glycolipids. With these extensive continuing studies, we are able to establish a new class of glycolipid inhibitors, providing further promise to develop them as inhibitors towards communicable pathogens, such as, *M. tuberculosis*.