

Event Date

January 19-21, 2026

Venue

SP Jain Auditorium
University of Delhi
South Campus

Let's create an enriching scientific experience, share research, and strengthen collaborations

SUPPORTED BY

Department of BioTechnology, Government of India

Anusandhan National Research Foundation

CSIR-INDIA

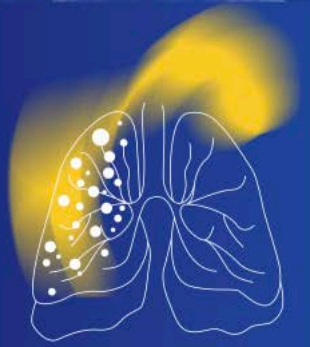
ABSTRACT BOOK

International TB Conference 2026

TURNING THE TIDE: NEW FRONTIERS IN TB THERAPEUTICS, VACCINES AND DIAGNOSTICS

Organised by:

University of Delhi South Campus
and
All India Institute of Medical Sciences, New Delhi



Phosphoregulation at the Crossroads: STPK Control of Toxin–Antitoxin Systems in *Mycobacterium tuberculosis* Persistence

Dr. Vandana Malhotra
Sri Venkateswara College, University of Delhi
New Delhi, India



Abstract

Persistence of *Mycobacterium tuberculosis* (*M. tb*) is a major barrier to tuberculosis eradication and is believed to be closely linked to the activity of toxin–antitoxin (TA) systems. Type II TA systems comprise of a toxin component and an antitoxin counterpart that neutralizes the toxin. Under stressful environments, the antitoxin is degraded, releasing the toxin which then acts to halt cellular growth. While TA modules are traditionally viewed as transcriptionally regulated stress-responsive elements, accumulating evidence suggests that post-translational control may play a critical role in tuning their function during persistent states. Recent findings from our laboratory have established Serine/Threonine protein kinases (STPKs) as key regulators operating at the intersection of global signaling and TA-mediated growth control in *M. tb*. Using a combination of biophysical, biochemical and computation biology methods, we demonstrate that STPK-dependent phosphorylation of RelJK TA cassette modulates toxin activity, alters toxin–antitoxin interaction resulting in inhibition of growth. We show that phosphorylation of Thr77 residue of the RelK toxin results in significant changes in its secondary structure, interfering with its neutralization by the RelJ antitoxin. Substitution of Thr77 with alanine or glutamate in RelK toxin resulted in poor binding to the RelJ antitoxin, allowing a partial rescue of cells co-expressing wild type RelJ antitoxin, and RelK.