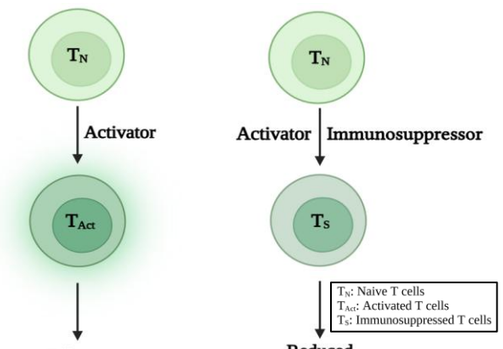
IOCBI 2026 Conference
The 1st International Online Conference on Biology
10–12 February 2026 | Online
MDPI

Intracellular Mechanisms of Experimental Immunosuppression: A Comparative Study in T Cells and Macrophages

Mrinalinee Pandit
National Institute of Science Education and Research (NISER)
an Off-campus Centre (OCC) of Homi Bhabha National Institute, India

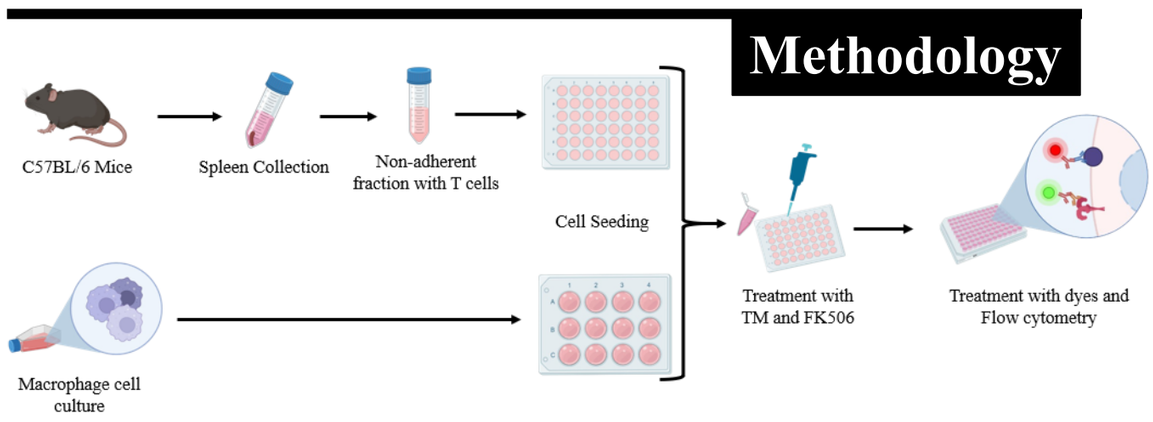
Immunosuppression, characterized by a diminished ability of the immune system to combat infections, plays major role in modulating immune functions. Experimentally induced immunosuppression using pharmacological agents, is instrumental in unraveling key immune regulatory mechanisms and identifying potential therapeutic targets. In this study, we investigated the immunosuppressive effects of two drugs, Telmisartan (TM) and Tacrolimus (FK506) on metabolic markers of T cell and macrophage function, intracellular calcium, nitric oxide (NO), reactive oxygen species (ROS), and mitochondrial membrane potential (MMP). Telmisartan, an angiotensin II type 1 receptor blocker primarily prescribed for hypertension, has demonstrated emerging immunosuppressive properties. FK506, a calcineurin inhibitor, is widely used as a potent immunosuppressant. Despite advances in understanding the mechanisms of immunosuppression, metabolic alterations in immune cells under these conditions remain insufficiently characterized. Calcium signaling is essential for T cell activation, whereas NO is known to inhibit T cell proliferation, oxidative stress and mitochondrial integrity have emerged as critical indicators of immune status, as observed in various studies. These findings aim to elucidate the mechanisms through which TM and FK506 mediate immunosuppression and contribute to the development of therapeutic strategies targeting these pathways.



Hypothesis

There will be differential expression of Calcium levels, NO levels, Reactive Oxygen Species and mitochondrial membrane potential in T cells and macrophages during experimental immune suppression.

Methodology



Results

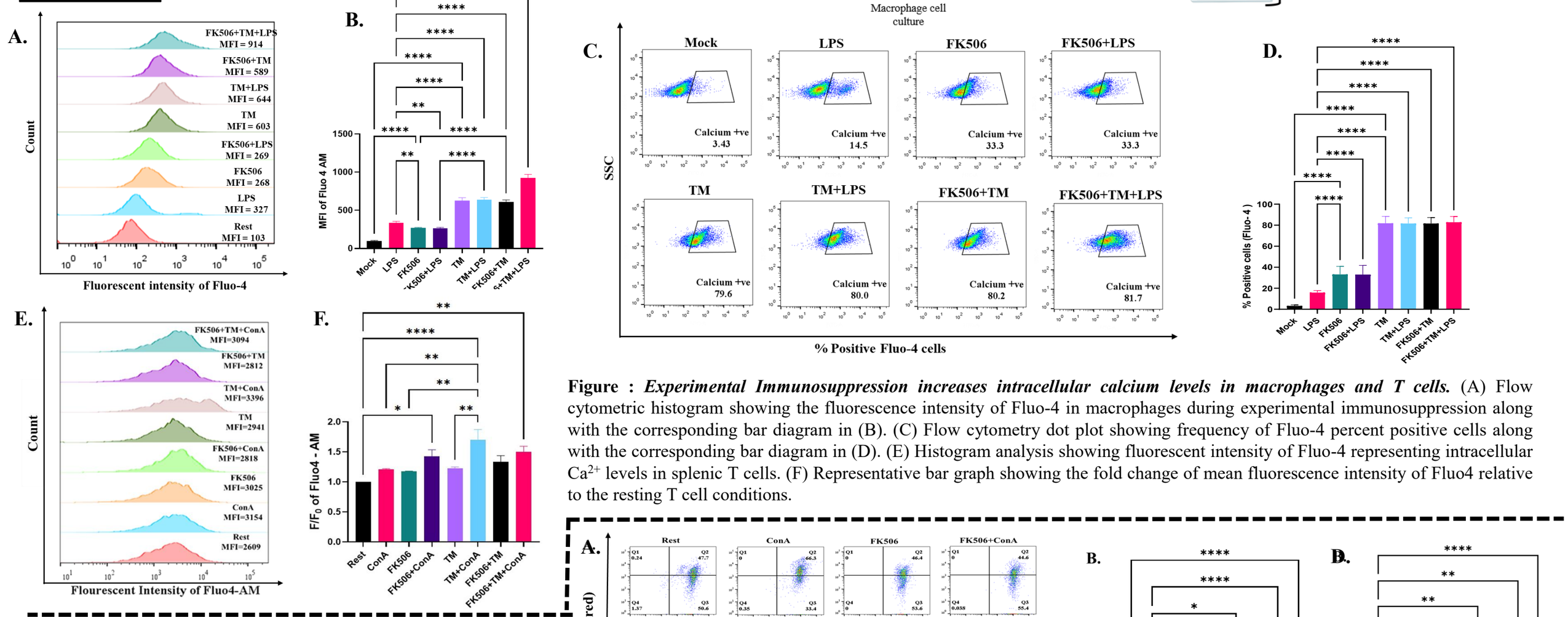


Figure : ***Experimental Immunosuppression increases intracellular calcium levels in macrophages and T cells.*** (A) Flow cytometric histogram showing the fluorescence intensity of Fluo-4 in macrophages during experimental immunosuppression along with the corresponding bar diagram in (B). (C) Flow cytometry dot plot showing frequency of Fluo-4 percent positive cells along with the corresponding bar diagram in (D). (E) Histogram analysis showing fluorescent intensity of Fluo-4 representing intracellular Ca^{2+} levels in splenic T cells. (F) Representative bar graph showing the fold change of mean fluorescence intensity of Fluo4 relative to the resting T cell conditions.

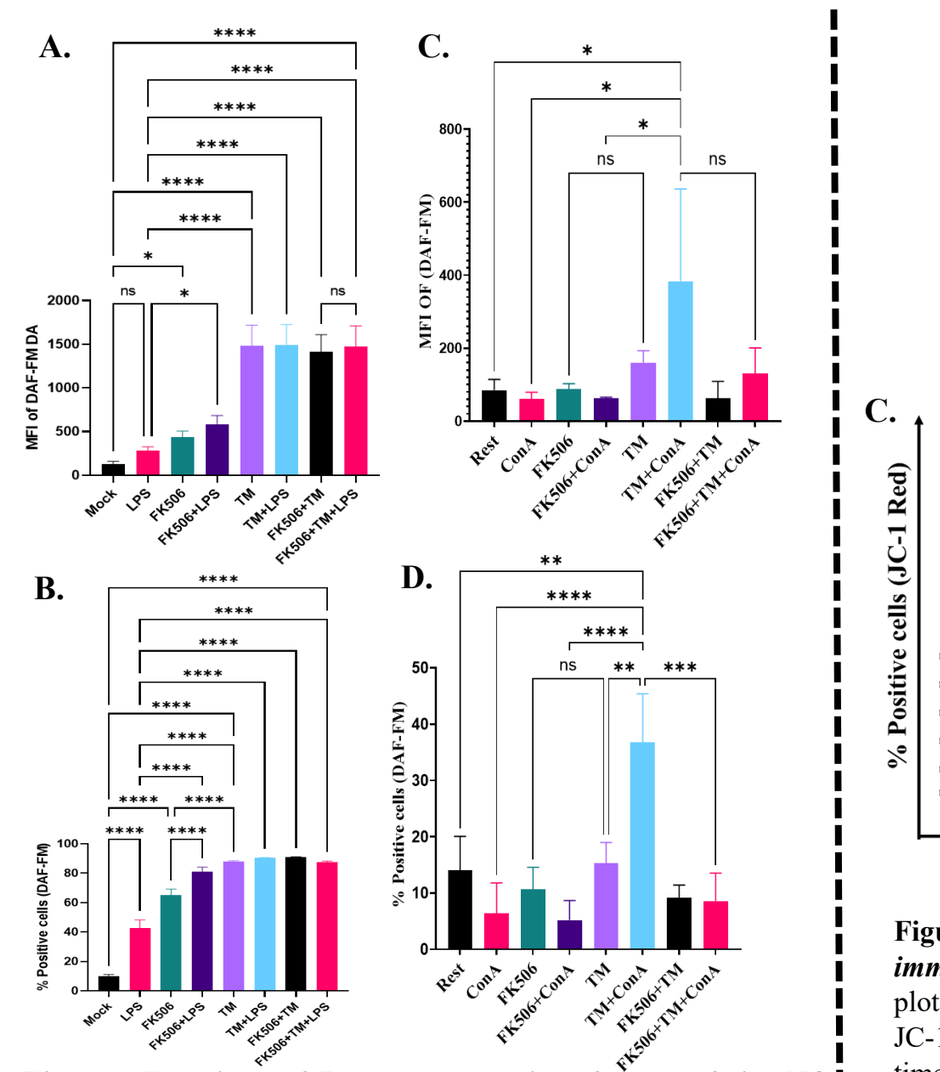


Figure : ***Experimental Immunosuppression elevates relative NO levels in macrophages and T cells.*** (A) Bar diagram showing the fluorescence intensity of DAF-FM in macrophages and (C) in T cells during experimental immunosuppression. (B) Representative bar graph showing the frequency of DAF-FM positive macrophages and (D) T cells during experimental immunosuppression.

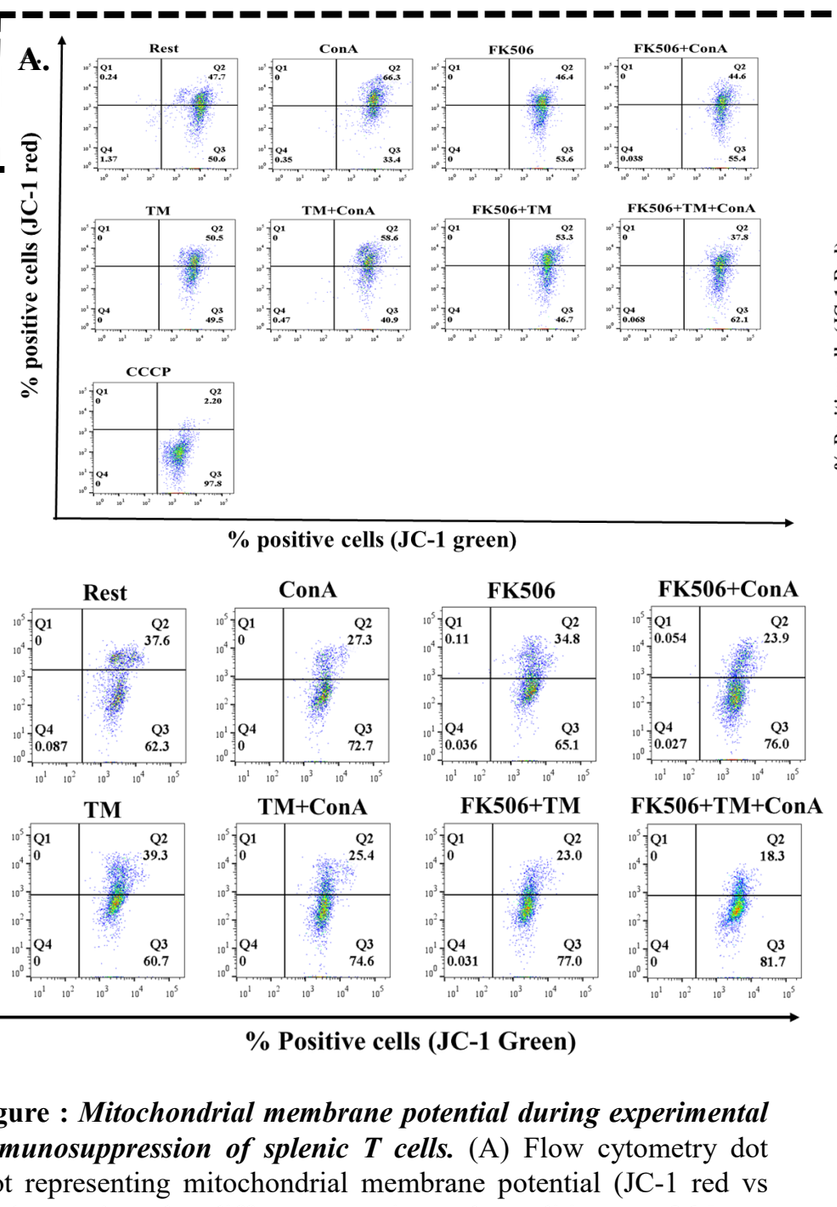
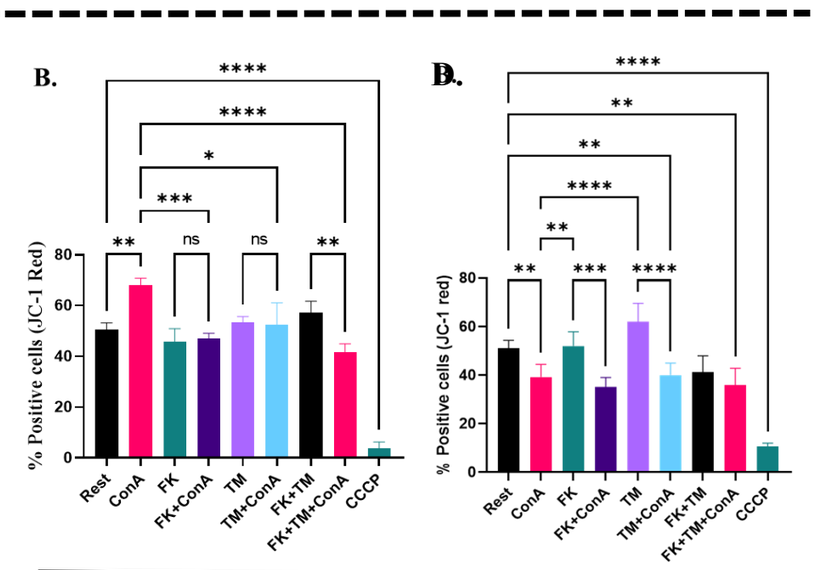


Figure : ***Mitochondrial membrane potential during experimental immunosuppression of splenic T cells.*** (A) Flow cytometry dot plot representing mitochondrial membrane potential (JC-1 red vs JC-1 green) under different experimental conditions at **36-hour** time point (B) Representative bar graph showing percent positive JC-1 red cells at **36 hours**. (C) Flow cytometry dot plot representing mitochondrial membrane potential (JC-1 red vs JC-1 green) under different experimental conditions at **48-hour** time point (D) Representative bar graph showing percent positive JC-1 red cells at **48 hours**



Summary

Our results found, intracellular calcium levels increased in activated splenic T cells and macrophages during immunosuppression. NO levels were reduced in experimentally activated T cells, but telmisartan increased NO production, while tacrolimus showed a mild effect. In macrophages, activated cells showed increased NO levels, with telmisartan further enhancing NO production supporting its role in NO mediated antihypertensive effects. In T cells, MMP showed time dependent changes. Experimental activation induced MM polarisation, however drug treatment decreased polarization, indicating restoration of MMP via immunosuppression Our findings highlight that immunosuppressive drugs reprogram immune cell metabolism in a cell type specific manner rather than causing uniform immune suppression.