Mycobacterium tuberculosis (Mtbb) kills 1.7 million people annually. The Th1 paradigm does not explain TB-driven cavitation. Current treatment is lengthy with many adverse effects. The Interleukin-23/Th17 axis plays a critical role in early Mtbb containment. Respiratory treatment is lengthy with many adverse effects. The Interleukin-23/Th17 paradigm does not explain TB-driven cavitation. Current treatment is lengthy with many adverse effects.

Summary MMPs are key mediators of tissue damage in human pulmonary TB and are regulated in a cell- and stimulus-specific manner. IL-17 and IL-22 drive MMPs but suppress MMP-9 in airway epithelium. The PI3Kα/p110α/p70S6K pathway is a crucial target and its immuno-modulation (eg, rapamycin) is a potential adjunctive therapy to limit tissue destruction and shorten chemotherapy in TB.
PI3K/p110a/p70S6K cascade mediated by Th-17 cytokines and the destruction in Human Tuberculosis (TB) is Matrix metalloproteinase-driven tissue T1

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