

Supporting Information

Mycobacterium tuberculosis Infection Induces BCSFB Disruption but No BBB Disruption In Vivo: Implications in the Pathophysiology of Tuberculous Meningitis

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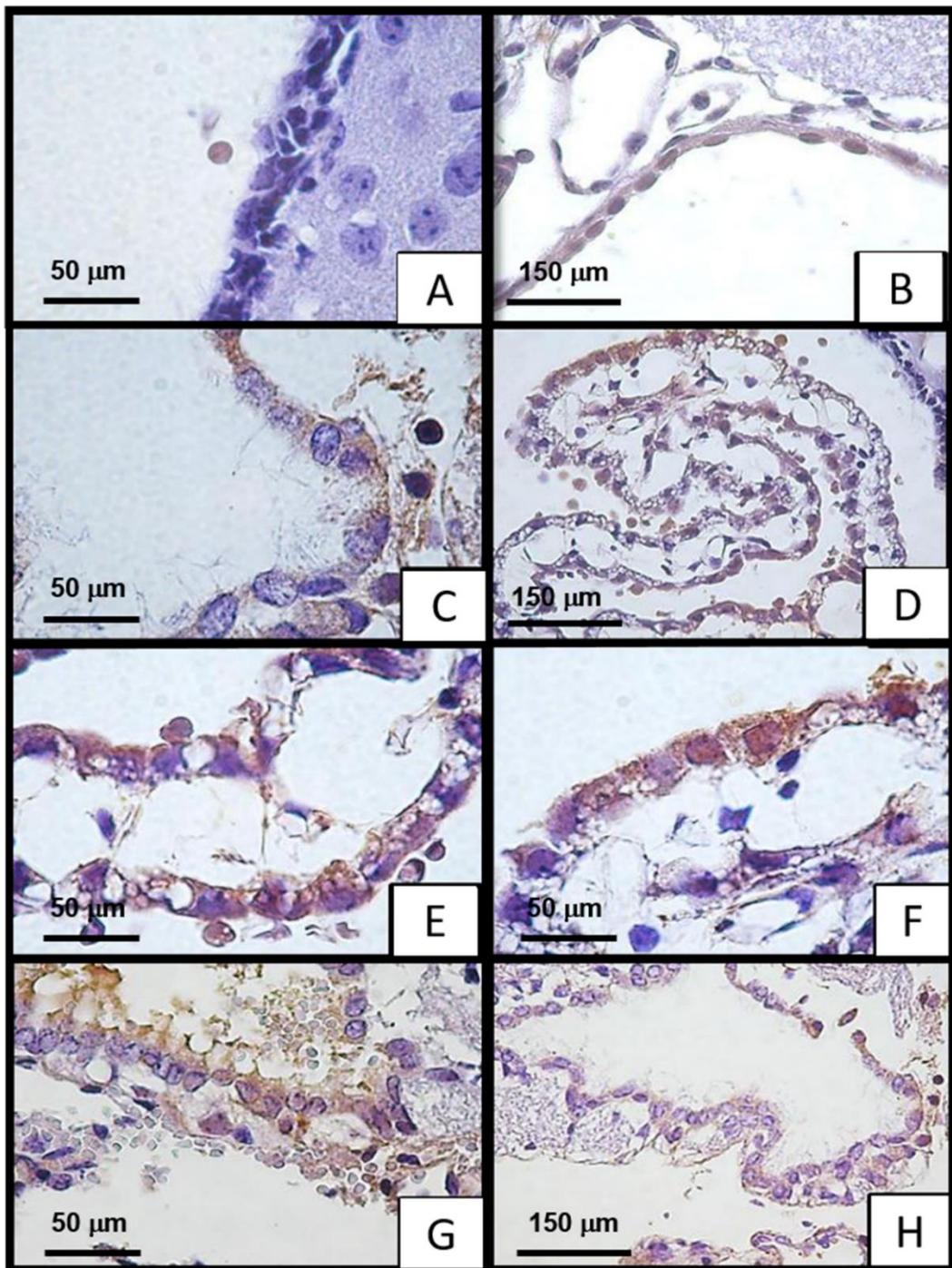


Figure S1. Histological micrographs of ependymal cells from Balb/C mice infected with *M. tuberculosis* LAM3/N15 immunoreactive to different cytokines by IHC. (A) Hyperchromatic, cytoplasmatic retraction, and pseudostratification of ependymal cells from the lateral ventricle (1000X). (B) Piamadre, IFN- γ at 60 DPI (400X). (C) Detached ependymal cells, IFN- γ positive with loss of organization at 60 DPI (1000X). (D) Choroid plexus, with loss of organization TNF- α at 120 DPI (400X). (E) Choroid plexus, with degeneration and vacuolization. TNF- α at 120 DPI (1000X). (F) Detached ependymal cells, TNF- α positive with loss of organization at 120 DPI(1000X). (G) Detached ependymal cells, IFN- γ positive with loss of organization at 120 DPI (1000X). (H) Brain cortex endothelial cells are positive for TNF- α with loss of organization and degenerative changes (400X).

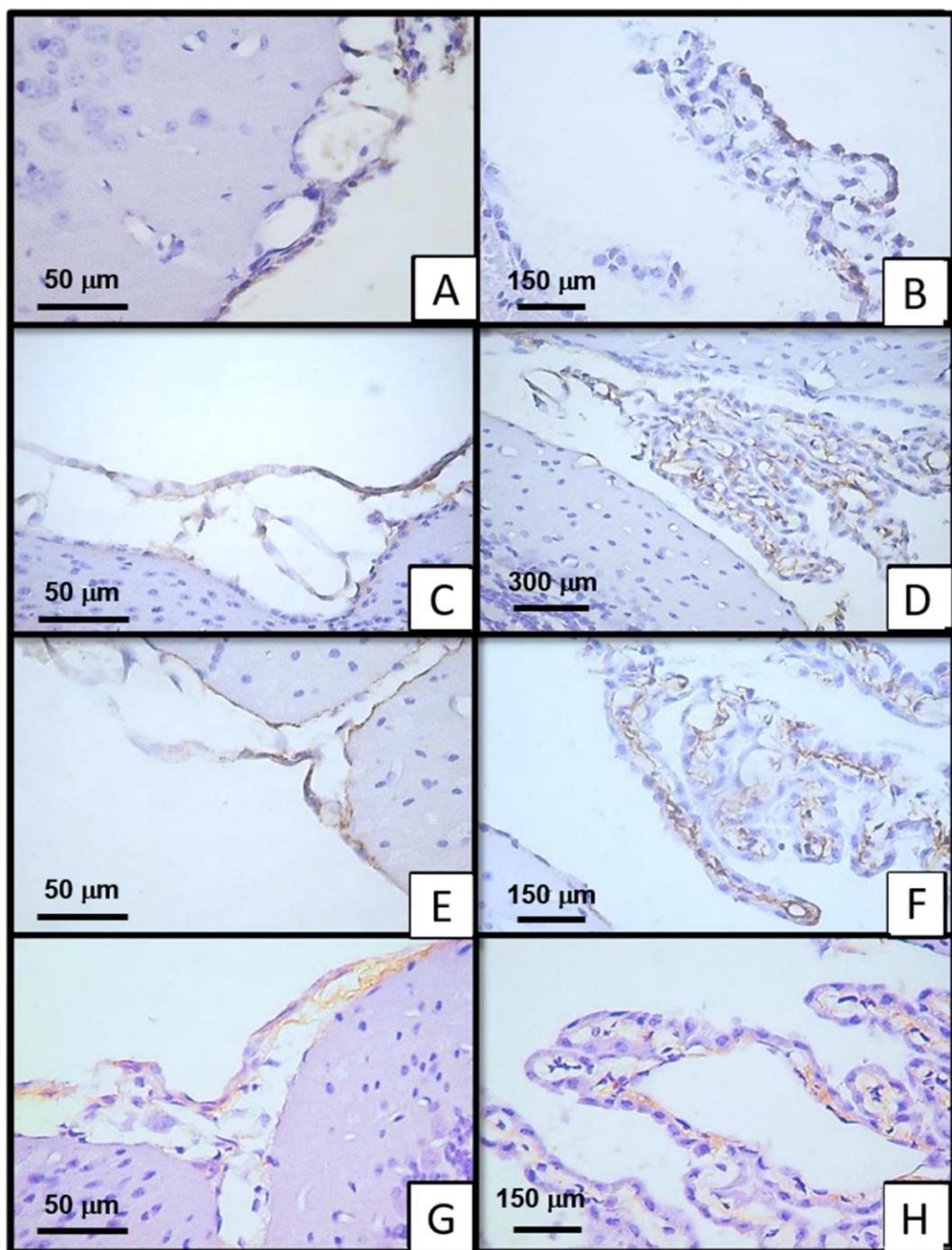


Figure S2. Histological micrographs of ependymal cells from Balb/C mice infected with *M. tuberculosis* H37Rv are immunoreactive to the presence of different cytokines by IHC. (A) Brain cortex, TNF- α at 28 DPI (400X). (B) Choroid plexus, TNF- α at 60 DPI (100X). (C) Brain cortex, IL-1 β at 60 DPI. (D) Choroid plexus, IL-1 β at 120 DPI (40X). (E) Brain cortex, IL-4 at 120 DPI. (F) Choroid plexus, IL-4 at 120 DPI (100X). (G) Brain cortex, TGF- β at 120 DPI. (H) Choroid plexus, TGF- β at 120 DPI (100X).