

Figure S1: Pathogen-specific therapies minimizes cytokine levels in cerebrospinal fluid of patients with meningitis. Patients with *C. neoformans* meningitis or Tuberculous meningitis were treated with standard pathogen-specific therapies (anti-fungal therapy or anti-tuberculous therapy), cytokine levels in cerebrospinal fluid before and after anti-fungal therapy (A) or anti-tuberculous therapy (B) were evaluated by ELISA. *0.01<p<0.05.

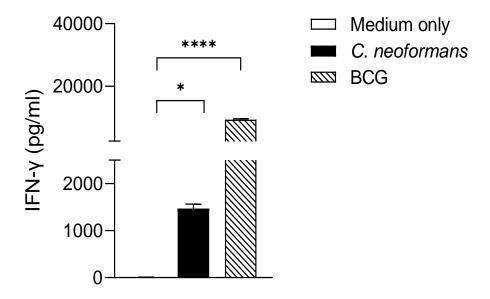


Figure S2: *C. neoformans* and BCG differentially stimulate IFN- γ expression by PBMCs. PBMCs were stimulated with or without *C. neoformans* or BCG for 3 days, supernatant IFN- γ was detected by ELISA, data was shown as mean \pm SE of three individual experiments. *0.01<p<0.05,****p<0.0001.

Figure S3

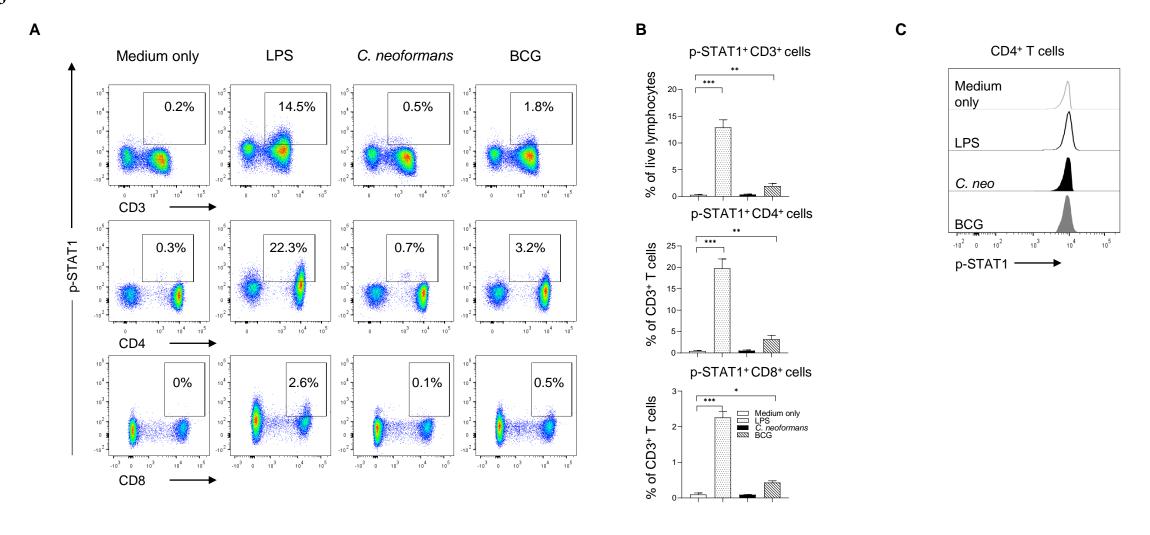


Figure S3: *C. neoformans* **stimulates phosphorylation of STAT1 in CD4**⁺ **T cells.** PBMCs were stimulated for 30 minutes with LPS , *C. neoformans*, or BCG, phosphorylation of STAT1 in T cell subsets (A and B) and its fluorescence intensity was analyzed by flow cytometry (C). *0.01<p<0.05, **0.001<p<0.01, ***<0.0001<p<0.001.

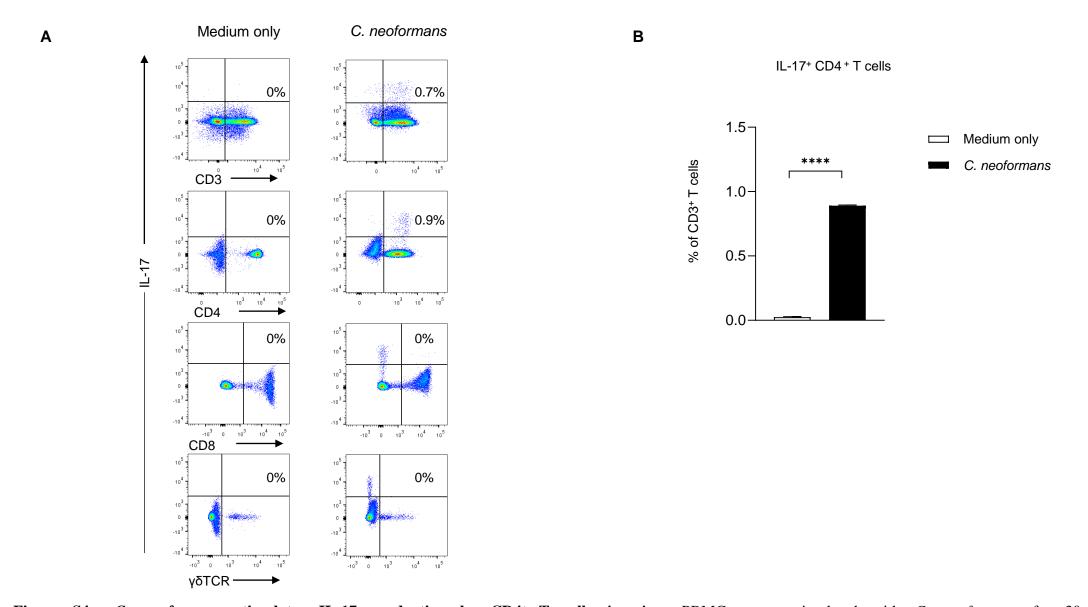


Figure S4: *C. neoformans* stimulates IL-17 production by CD4⁺ T cells *in vitro*. PBMCs were stimulated with *C. neoformans* for 30min plus protein transport inhibitor cocktail for 5 hours before collected cells, IL-17-producing cells were analyzed by flow cytometry: representative data of IL-17⁺ T cells in αβTCR⁺ T cells (CD3⁺γδTCR⁻ cells) and γδTCR⁺ T cells (A), and percentages of IL-17⁺ CD4⁺T cells in live CD3⁺ cells (B). **** p < 0.0001.