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Differential expression of Pattern Recognition Receptors in Neurotuberculosis patients with and without HIV infection

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Background: Neurotuberculosis (neuroTB) is one of the commonest opportunistic infections of the central nervous system associated with HIV. However, neuroTB cases without HIV are also seen. Cross-talk between HIV, Mycobacterium tuberculosis and host immune responses in a co-infected individual may differentially alter expression levels of the repertoire of pattern recognition receptors (PRRs), including Toll-Like Receptors (TLRs), thereby modulating dissemination and disease progression. The objective of this study was to elucidate the role innate immune mechanisms, especially PRRs in neuroTB patients with and without HIV.

Materials and Methods: Individuals with neuroTB with and without HIV co-infection (n=20 in each group) were studied with healthy subjects as controls. RNA was extracted from PBMC and converted into cDNA, which was used for Real Time SYBR Green PCR to study expression of TLRs 1-9, RIG-I and MDA-5. GAPDH served as the housekeeping gene. Fold change in the mRNA levels of the TLRs was calculated by the comparative CT method.

Results and Conclusion: We observed a significant upregulation of MDA-5 in the HIV neuroTB group as compared to neuroTB patients. MDA-5 is a major inducer of interferons, thus recruiting CD8 cells, which play a crucial role in controlling HIV replication. There was significant downregulation of TLR-6 expression in patients with HIV-neuroTB co-infection as compared to neuroTB alone. Interestingly, TLR-6 has been associated with recognition of mycobacterial antigens and its downregulation may contribute to neuropathogenesis of TB. TLRs 1, 3 and 9 expression was significantly downregulated in all patients with neuroTB, irrespective of HIV co-infection suggesting a protective role for these TLRs in host response to TB. Further studies on TLRs and other pattern recognition receptors in HIV-TB co-infected individuals would help understand pathogenesis and dissemination of the disease and may contribute to development of better therapeutic strategies.