



Upon brain ischemia, MOG₃₅₋₅₅-reactive CD4⁺ T cells enter the circulation and extravagate into the brain. Neuroantigens released after brain ischemia are phagocytosed, processed and presented by APCs including brain-resident microglia to MOG₃₅₋₅₅-reactive CD4⁺ T cells. Thereby, the MOG₃₅₋₅₅-reactive CD4⁺ T cells are activated by APCs (a), and then undergo *in situ* proliferation (b), or clonal expansion (c). These newly emerged T cell clones together with MOG₃₅₋₅₅-reactive T cells can be activated by APCs and produce pro-inflammatory factors that aggravate BBB disruption, inflammation and neuronal damage (d). a: Activation; b: Proliferation; c: Clonal expansion; d: neuronal damage.