Role of extracellular vesicles derived from HIV-infected T cells and microglial cells in neuroinflammation and neuropathogenesis using a blood-brain barrier and cerebral organoid co-culture model

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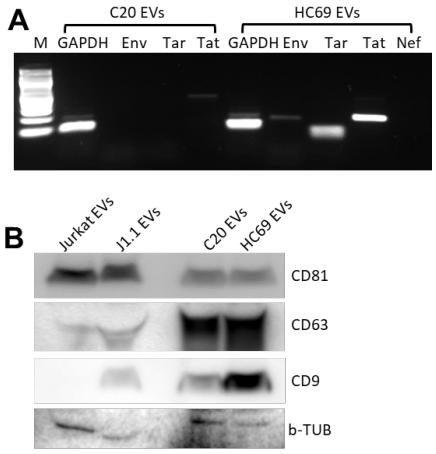
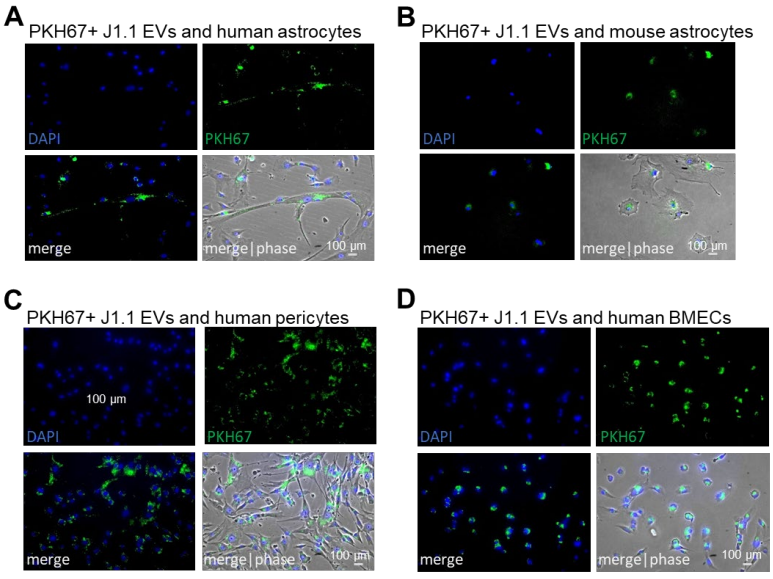
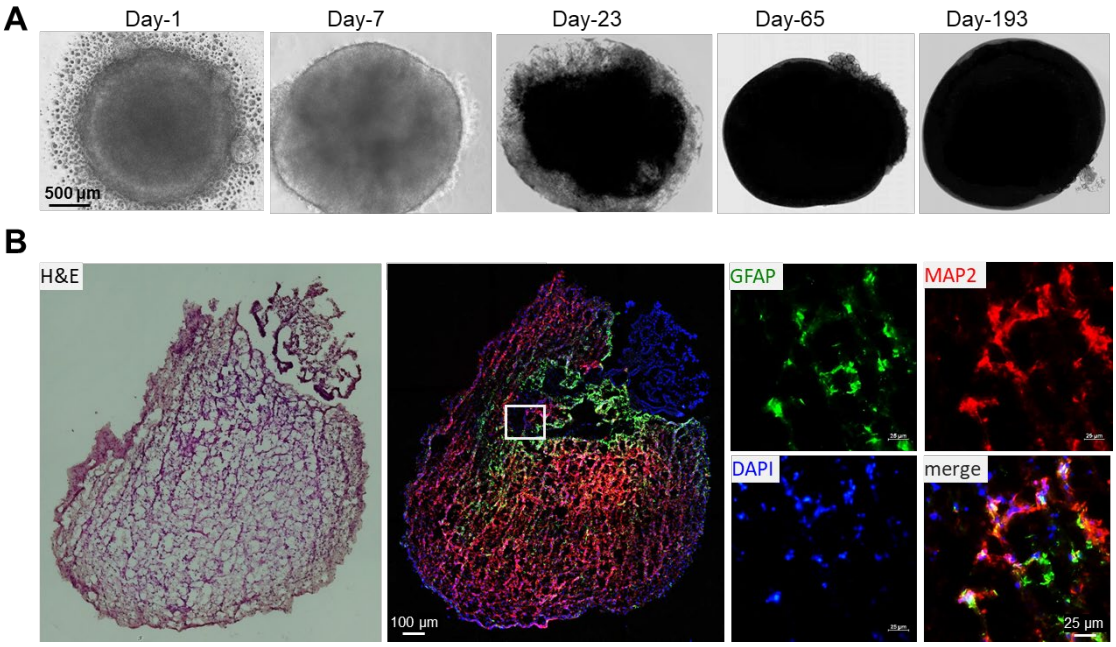
Supplementary Figures



Supplementary fig S1. A) RT-PCR of EV RNA derived from control C20 microglia and HIV-1-infected HC69 microglial cells to detect HIV TAR, Env, Tat, and Nef RNAs. B) Immunoblotting of exosome markers CD81, CD63, and CD9 in proteins extracted from EVs. Beta-tubulin was used a loading control.



Supplementary fig. S2. EV entry into human primary astrocytes, pericytes, and BMECs as well as mouse astrocytes. EVs were labeled with PKH67, followed by incubation with cells for 4 hr. EV entry was determined by microscopy. Nucleus, DAPI (blue). Representative images are shown at 20× magnification.



Supplementary fig. S3. A) Establishment of cerebral organoids using human iPSCs. Organoids from day 1 to day 193 were shown (top, representative images are shown at 20× magnification). **B)** Immunofluorescence staining (IFS) detected neurons (MAP2, red) and astrocytes (GFAP, green) in organoids. 95-day organoids were fixed and cyrosectioned for IFS. Nucleus, DAPI (blue). Representative images are shown at 20× and 40× magnification (enlarged panels).