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Passive and Active Cellular Immune Surveillance (CIS) of Central Nervous System (CNS) of healthy Humans revealed with the Marburg Cerebrospinal Fluid Model

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Transfer of blood leukocytes into cerebrospinal fluid (CSF) seems to be impossible in CNS of healthy humans: CNS (central nervous system) blood capillaries are completely locked by blood-brain-barrier (bbb); choroid plexus, producer of CSF into CNS ventricles, is locked for blood cells by blood-CSF-barrier (bCSFb); but proteins (albumin>imunoglobulins) are secreted here from blood into ventricular CSF. Literature studies reveal no bbb and leaky ependymal surfaces at 8 brain nuclei of circumventricular organs (CVOs) in CNS of healthy humans: 2 area postrema, 2 median eminence, neurohypophysis with infundibulum, organum vasculosum of lamina terminalis, pineal gland, subfornical organ. Blood pressure is the main force which presses blood leukocytes through leaky capillaries into CVO stroma; leaky ependyma at CVOs paves the way of blood leukocytes into CSF of 3rd and 4th ventricles, representing 0-3/µl leukocytes in suboccipital CSF (SOP-CSF) of healthy humans. Depending on blood pressure, transfer rates per min of blood leukocytes through CVOs into CSF increases from 0/µl to 32 leukocytes/µl SOP-CSF: Smaller blood lymphocytes (>90%) transfer easier than larger monocytic cells (<10%) into ventricular CSF, where some inactive blood leukocytes can transfer into the brain through naked ventricle walls (without ependyma) to perform passive CIS (common integration site) in human CNS. Active CIS is performed with about one HLA-DR+-activated lymphocyte of the 32 transferred blood leukocytes, which secrete proteases to pave the way actively through whole CNS. HLA-DR+-lymphocytes, when activated to CNS constituents in the body and so being increased, can induce destructive-inflammatory processes in human CNS, e.g. multiple sclerosis.

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