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# Abstracts

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T14-015A

**Increased density and redistribution of multivesicular bodies into perivascular astrocytic endfeet in sepsis-associated encephalopathy**T. Shuliatnikova*Pathological Anatomy and Forensic Medicine, Zaporizhzhia State Medical University, Zaporizhzhia, UA*

Sepsis associated encephalopathy (SAE) manifested by impaired perception, consciousness and cognition, results from multifactorial events, including high levels of systemic cytokines, microbial components and endotoxins, which damage the brain barriers, instigate neuroinflammation and cause homeostatic failure. The ultrastructure of astrocytes in the cortex and sub-cortical white matter of the parietal lobe in cecum ligation and puncture sepsis model established in Wistar rats was examined with transmission electron microscopy. Analysis of brain samples of SAE model animals at 24 hours after surgery revealed ultrastructural pathology represented by disintegration and destruction of nuclear and cytoplasmic structures in neurones and neuroglial cells. In comparison with the control group the processes and pericarya of astrocytes showed 4 - 5 times increase in the number of multivesicular bodies (MVBs) which appear as large (0.4 - 1  $\mu\text{m}$  in diameter) vacuolar structures packed with 2 to 30 vesicles with diameters of 50 to > 500 nm. Accumulation of MVBs in astrocytes in SAE reflects increased endocytosis and possibly phagocytosis, both likely revealing elevated phagocytic activity of astroglia. Astroglial MVBs are highly polymorphic: some of them are densely packed with vesicles whereas others display relatively large vesicle-free zones, which sometimes contain loose electron-dense material or lamellar structures including myelin-like bodies. The MVBs contribute to the sorting, degradation, trans-cellular transport and recycling of various intracellular molecules and are linked to the formation of exosomes involved in cell-to-cell communications. Increased of the density and redistribution of MVBs into perivascular astroglial endfeet plausibly reflects adaptive astroglial response to sepsis-associated pathology, manifested in particular, by elimination and export of cytosolic molecular debris linked to cellular damage.

T14-016A

**Crif1 deficiency in endothelial cells induces BBB disruption accompanying myelin damage**M.J. Lee<sup>1,2</sup>, Y. Jang<sup>1,2</sup>, J. Han<sup>1,2</sup>, S.J. Kim<sup>1,2</sup>, X. Ju<sup>1</sup>, Y.L. Lee<sup>1,2</sup>, J.H. Son<sup>2</sup>, J. Cui<sup>1</sup>, M.J. Ryu<sup>2,3</sup>, S.-Y. Choi<sup>6</sup>, W. Chung<sup>7</sup>, C. Heo<sup>8</sup>, Y.H. Huh<sup>9</sup>, G.R. Kweon<sup>1,2,3</sup>, J.Y. Heo<sup>1,2,5</sup><sup>1</sup>*Department of Medical science, Chungnam National University School of Medicine, Daejeon, KR*<sup>2</sup>*Department of Biochemistry, Chungnam National University School of Medicine, Daejeon, KR*<sup>3</sup>*Research Institute for Medical Science, Chungnam National University School of Medicine, Daejeon, KR*<sup>4</sup>*Brain research institute, Chungnam National University School of Medicine, Daejeon, KR*<sup>5</sup>*Infection Control Convergence Research Center, College of Medicine, Chungnam National University, Daejeon, KR*<sup>6</sup>*Department of Pathology, Chungnam National University School of Medicine, Daejeon, KR*<sup>7</sup>*Department of anesthesiology and pain medicine, Chungnam National University Hospital, Daejeon, KR*<sup>8</sup>*Center for Neuroscience Imaging Research, Institute for Basic Science, Suwon, KR*<sup>9</sup>*Electron Microscopy Research center, Korea Basic Science Institute, Cheongju, KR*

Endothelial cells (ECs) in Blood-brain barrier (BBB) have higher volume of mitochondria than endothelial cells of peripheral capillaries. Cerebral endothelial cells have junctional proteins to maintain BBB integrity by restricting toxic substances and peripheral immune cells. Although it is known that mitochondrial inhibitors cause BBB