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VASCULAR COMPONENT OF NEUROINFLAMMATION IN EXPERIMENTAL ALZHEIMER'S DISEASE

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We assessed RAGE- and CD147-mediated mechanisms of hippocampal damage caused by the accumulation of beta-amyloid, development of local inflammation, metabolic disorders, and damage to the blood-brain barrier using

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two experimental models of Alzheimer's disease *in vivo*. We studied the new effects of A β in the tissue of the hippocampus during chronic neurodegeneration of the Alzheimer's type, characterizing disorders of neuroplasticity, angiogenesis, structural and functional integrity of the blood-brain barrier, the development of local neuroinflammation in relation to the patterns of RAGE and CD147 expression. Early neurodegenerative changes in the hippocampus associated with accumulation of A β suggest induction of neoangiogenesis and establishment of aberrant intercellular contacts in endothelial cells in certain subregions of hippocampus as well as development of local neuroinflammation. Along the progression of neurodegeneration, neoangiogenesis is further suppressed in hippocampus.

Keywords: Alzheimer's disease, angiogenesis, beta-amyloid, neuroinflammation, blood-brain barrier