"Role of Homocysteine in the Pathophysiology of Alzheimer’s Disease"

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Recent epidemiological and clinical data suggest that elevated serum homocysteine levels may increase the risk of developing Alzheimer's disease (AD), but the underlying mechanisms are unknown. We tested the hypothesis that high homocysteine concentration may increase amyloid beta-peptide (Aβ) levels in the brain and could therefore accelerate AD neuropathology. For this purpose, we developed a new in vivo model for studying the effect of hyperhomocysteinemia in brain amyloidosis. This model helped us to demonstrate for the first time an unequivocal correlation between Aβ and plasma homocysteine levels in vivo. We also found evidence that the increase in Aβ may be in part by dysregulation of a key enzyme for Aβ degradation, insulin degrading enzyme (IDE). Finally, we obtained data, which unexpectedly suggested the presence of a yet not identified Aβ-degrading enzyme that it is also inhibited by homocysteine.

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8th Floor Neuroscience Center Conference Room
LSU Lion’s Building,
2020 Gravier Street