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## Is Alzheimer's disease type $\mathbf{3}$ diabetes?

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Alzheimer's disease (AD) is characterized by cellular, molecular and biochemical abnormalities, including cell loss as well as pathological inclusions derived from hyper-phosphorylation of tau and amyloid-beta over-production. Importantly, impaired energy metabolism is an early and key aspect in AD linked to a range of genes and pathways associated with metabolic function, namely increased oxidative stress and mitochondrial dysfunction amongst others. Currently, increasing interest has also been placed on the role of insulin deficiency and insulin resistance as mediators of AD associated neurodegeneration. Nevertheless, whether these changes are secondary to life-style/obesity associated conditions, such as the metabolic syndrome or Type II diabetes or rather due to brain-specific disturbances in brain insulin and insulin-like growth factor (IGF) signaling mechanisms remains an important research question. To this end, peripheral hormone therapies that are addressed at regulating insulin release and improving insulin sensitivity have been shown to be beneficial for cognitive function and AD associated pathology. On the other hand, insulin-associated signaling mechanisms can account for the majority of molecular, biochemical changes observed in the AD brain and are known to directly regulated amyloid-beta, the main histopathological entity of AD. As such, the goal of the proposedsession would be to bring together key investigators in the field of metabolism in AD in order to explore the concept of Alzheimer's Disease as a form of brain-specific Diabetes.

