In search for alternative strategies for the treatment of dementia: the sweet trail to neuroprotection…

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By 2050, health care costs related to treatment of Alzheimer's disease (AD) patients are projected to put at risk many economies around the world. To date, the only available therapies for AD are based on drugs that are unable to modify the course of the disease. We are in great need of alternative and/or additional approaches to counteract this incoming health care crisis. In that respect, a fruitful approach is offered by the investigation of the intersection between glucose dysmetabolism and brain impairment. Type 2 Diabetes Mellitus (T2DM), a disorder of peripheral glucose regulation, is associated with cognitive decline. Aging, a key contributing factor for AD development, greatly enhances the negative impact of T2DM on cognitive functions. Interestingly, emerging evidence in the past two decades has shown that T2DM and AD share common pathogenic mechanisms such as impaired glucose metabolism, increased oxidative stress, insulin resistance, and amyloidogenesis. In the brain, insulin signaling is not exclusively involved in metabolic processes but also plays a role in modulating neurotrophic and neuroendocrine functions. In the talk, we are going to discuss studies that have investigated effects of antidiabetic drugs that have been found to affect cognition, synaptic plasticity, and BDNF-related signaling in preclinical models of AD (3xTg-AD) and aging.