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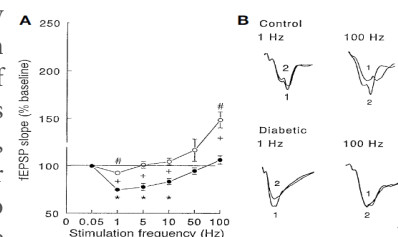


Amer H Kamal Al Ansari

Arabian Gulf University, Bahrain

Defective brain cognitive function in diabetes mellitus: A model for accelerated ageing process

The aim of the present study was to examine learning and hippocampal synaptic plasticity in ageing and diabetes, based on the hypothesis that the effects of diabetes and ageing on the brain could interact. This hypothesis stems from clinical observations that the effects of diabetes on the brain are most pronounced in the elderly. Moreover, many of the processes which have been implicated in the pathogenesis of brain ageing, in particular oxidative stress, micro-vascular dysfunction, non-enzymatic protein glycation and disturbed intracellular calcium homeostasis, are also implicated in the development of diabetic complications. To find the possible interaction between diabetes and ageing, we investigated Morris water maze performance and examined hippocampal synaptic plasticity *ex vivo* in young adult and aged diabetic and non-diabetic rats. Because the study aimed to examine the additive effects of diabetes and ageing on the brain, an experimental protocol was chosen in which each of these two conditions in isolation produces only moderate deficits. Rats were examined after 2 months of diabetes, which produces half-maximum deficits in synaptic plasticity in young adult rats. Aged rats were examined at 2 years of age, when they have developed moderate changes in synaptic plasticity due to aging alone. Significant learning impairments were observed in young adult diabetic rats compared with controls. These impairments were even greater in aged diabetic animals. In hippocampal slices from young adult diabetic animals long-term potentiation was impaired compared with controls. In contrast, induced long-term depression was enhanced in slices from diabetic rats compared with controls. It is concluded that both diabetes and ageing affect learning and hippocampal synaptic plasticity. The cumulative deficits in learning and synaptic plasticity in aged diabetic rats indicate that the effects of diabetes and ageing on the brain could interact. Relative fEPSP slopes after different conditioning stimuli in hippocampal slices from young adult (Left) and aged animals (right). Low frequency conditioning stimuli induced depression of the fEPSP whereas HFS induced potentiation. Diabetic animals in both groups show enhanced LTD and depressed LTP expressions when compared to the controls. Young diabetic animals had comparable defects to the aged control group indicating that DM acts like an accelerated ageing process.



Recent Publications

1. Kamal A, Ramakers G M J, Gispen W H, Biessels G J (2013) Hyperinsulinemia in rats causes impairment of spatial memory and learning with defects in hippocampal synaptic plasticity by involvement of postsynaptic mechanisms. *Exp Brain Res.*; 226(1): 45-51.
2. Gardoni F, Kamal A, Bellone C, Biessels G J, Ramakers G M J, Cattabeni F, et al. (2002) Effects of streptozotocin-diabetes on the Hippocampal NMDA receptor complex in rats. *J Neurochem.*; 80: 438-47.

References

1. Saedi E, Gheini M R, Faiz F, Arami M A (2016) Diabetes mellitus and cognitive impairments. *World J Diabetes.*; 7: 412-22.
2. Akinola O B (2016) Sweet old memories: a review of the experimental models of the association between diabetes, senility and dementia. *Metab Brain Dis.*; 31(5): 1003-10.
3. Ramos-Rodriguez J J, Jimenez-Palomares M, Murillo-Carretero ML, Infante-Garcia C, Berrocoso E, Hernandez-Pacho F, et al. (2015) Central vascular disease and exacerbated pathology in a mixed model of type 2 diabetes and Alzheimer's disease. *Psychoneuroendocrinology.*; 62: 69-79.

Biography

Amer H Kamal Al-Ansari has spent about 15 years in teaching and research activities in Rudolf Magnus Institute for Neurosciences in the Netherlands. His work was devoted to explore the effects of diabetes mellitus on the higher brain function, namely memory and behavior. He has then moved to Arabian Gulf University as a Professor and Chairperson of Physiology Department.

amerha@agu.edu.bh