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DIABETES AND COGNITIVE BRAIN FUNCTION: IS DIABETES AN ACCELERATED FORM OF AGEING?

Amer Kamal Al Ansari
Arabian Gulf University, Bahrain

The aim of the present study was to examine learning and hippocampal synaptic plasticity in ageing and diabetes. Many of the processes which have been implicated in the pathogenesis of brain ageing are also involved in the development of diabetic complications. We investigated Morris water maze performance and examined hippocampal synaptic plasticity ex vivo in young adult and aged diabetic and non-diabetic rats. 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). 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.