Introduction: Diabetes mellitus is a state of chronic hyperglycaemia. In late stages of the disease, especially if it is not regulated well, chronic complications may occur, dominating the clinical picture. Osteoporosis is characterized by bone loss per volume unit leading to microarchitectonics disorder of the bone. Connection between diabetes and osteoporosis is very complex.

Methods: Medical documentation collected at daily hospital of Clinic of endocrinology, diabetes and metabolic disorders is used in this study. Sample includes 60 patients which have been diagnosed with diabetes mellitus, with or without complications, who underwent densitometry measurement (DEXA). Glycosylated hemoglobin (HbA1c), fasting glucose and postprandial glucose are used as parameters of glicoregulation.

Results: Average duration of diabetes is 15.61 ± 9.63 years. Average value of HbA1c is 8.5 ± 1.79%, average value of fasting glucose is 9.23 ± 2.94 mmol/l and average value of postprandial glucose is 11.35 ± 4.27 mmol/l. 67% of patients have one or more complications. Bone mineral density (g/cm²) of femoral neck and total have significant negative correlation with HbA1c (p < 0.01). Bone mineral density of lumbosacral spine and femoral neck (g/cm², T-score) have light negative correlation with postprandial glucose.

Conclusion: Bone mineral density and parameters of glicoregulation have negative correlation. Statistically significant correlations between bone mineral density and chronic degenerative complications of diabetes were not found.

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PS237

Polymorphism of Kibra gene in patients with terminal renal insufficiency

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Aim: The aim of this study was to determine whether there is a difference in frequencies of genotypes and alleles of KIBRA gene polymorphism, rs17070145 between patients with terminal renal insufficiency and normal population.

Introduction: KIBRA gene has a role in signal transmission that regulates apoptosis, proliferation, and movements of the cytoskeleton of cells. Due to its most common expression in kidney and brain, the name of this protein is Kibra (Kidney, BRAin). Polymorphism rs17070145 (substitution of thymine with cytosine in the ninth intron of the gene) is associated with Alzheimer’s disease and memory, while its connection with kidney’s diseases has not been tested yet. It is thought that allele C is the factor of predisposition in TRI.

Methods: Polymorphism rs17070145 was analyzed with Real Time PCR method using TaqMan probes and 50 people with TRI were involved. Results of gene analysis for the control group were taken from previous research. Frequencies of genotypes and alleles between patients with TRI and healthy examinees was compared with χ² (chi-square) test.

Results: The frequency of CC genotype among patients with TRI is 76%, CT genotype 22% and TT genotype 2%. Based on frequencies of genotypes, we found that frequency of C allele is 87%, while the frequency of T allele is 13%.

Conclusion: Results of χ² test show extremely statistically significant difference in frequencies of genotypes and alleles in patients with TRI in comparison with healthy people (P < 0.0001). These results indicate that C alleles on locus rs17070145 in KIBRA gene are probably the significant factor of predisposition in the pathogenesis of TRI.

References


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Neurosciences Poster Session
Thursday, September 14th, 16h00

PS088

D-Galactose high-dose administration and oral epigallocatechin-3-gallate effects on the dendritic trees of developing neurons of young male rats

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Aim: In the present study, we aimed to explore the effect of d-galactose administration and epigallocatechin-3-gallatte (EGCG) on the dendritic trees of developing granule cells of the hippocampal formation (HF) of young male rats.

Introduction: The model of accelerated senescence with the administration of d-galactose is used in anti-aging studies. However, reports have questioned its effectiveness. To clarify this issue we used high-dose d-galactose on young rats and studied the immature granule cells stained with the neurogenesis marker doublecortin (DCX). We also used EGCG, a green tea catechin, to verify if there are neuroprotective effects in the d-galactose-treated animals.

Methods: At 4 weeks of age, male Wistar rats were allocated to a control group (n = 7), a d-galactose group (300 mg/kg body weight, intraperitoneally) (n = 5; GAL) and to a d-galactose + EGCG (oral solution, 2 grams/L) group (n = 5; gal + EGCG) during 4 weeks. After this period DCX immunocytochemistry was performed. The dendritic trees of immature granule cells were drawn with the aid of a camera lucida and a metric analysis of the dendritic segments of the dendritic trees was performed.

Results: No differences in all parameters quantified were found when controls and gal rats were compared. However, the results show that the total dendritic length of the dendritic trees of gal + EGCG rats was significantly reduced when compared with controls (p < 0.03). There were no differences in the others dendritic parameters quantified.

Conclusion: d-Galactose did not induce disturbance of the neurogenesis as shown by the absence of alterations in the dendritic trees confirming our previous studies. Surprisingly, the addition of EGCG led to a reduced total dendritic length. This unexpected effect can be explained if we consider that the addition of the catechin acted as a second aggression leading to a disturbed dendritic tree of the immature neurons.

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PS205

The bioactive compounds from elderberry to modulate mitochondrial dysfunctions underlying Alzheimer’s disease

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Aim: The specific objective of this work is to establish a correlation between the physical–chemical properties of the aqueous extract of elderberry (Sambucus nigra L.) and its ability to tune the cell redox state and to overcome mitochondrial dysfunctions, which are pathological events with high relevance in Alzheimer’s disease (AD).

Introduction: Currently, there is no effective medicine to prevent or delay the progressive brain degeneration underlying cognitive decline and dementia that characterize AD. Previous works support the idea that the loss of mitochondrial functionality, connected with the decline of complex I activity, is able to promote AD phenotype through the activation of multiple pathophysiological pathways, including oxidative stress, neuroinflammation, and also tau and amyloid-beta pathologies. Thus, multi-targeted