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 $8.5 \pm 1.79\%$, 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/cm^2) of femoral neck and total have significant negative correlation with HbA1c ($p < 0.01$). Bone mineral density of lumbosacral spine and femoral neck (g/cm^2, *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.

http://dx.doi.org/10.1016/j.pbj.2017.07.061

PS022

Effect of autologous stem cell transplantation in patients with hematological malignancies

A. Kovačić*, M. Džeba

Medical Faculty, University of Novi Sad, Serbia
E-mail address: alex.ak.bb@gmail.com
(A. Kovačić).

Aim: The aim of this study is to analyse avaliable medical data of patients diagnosed with multiple myeloma (MM), lymphoma Hodgkin (MB) and non-Hodgkin (NHL) and acute leukemia (AL), who underwent ASCT, and to compare the results with the results from other scientific works.

Introduction: Autologous stem cell transplantation (ASCT) with high dose chemotherapy is effectible and safe approach in the treatment of different hematological malignancies. Nowdays, it is the standard therapy for multiple myeloma, lymphomas and acute leukemias.

Methods: Retrospective study included 84 patient diagnosed with MM, MH, LNH and AL who underwent ASCT in the period from 2004 to 2016. Data are presented in table and charts.

Results: In relation to the underlying disease, the distribution of respondents was as follows: 35 patients with MM, 24 with NHL, 20 with MH and 6 with AL. Large volume apheresis procedure had to 75 patients (89.3%), and 9 patients (10.7%) had conventional two-day procedure. The mean value of processed blood volume amounted to 13050 ml. The average number of MNC in the apheresis product was 7.8×108/kg bw, a CD34+ cells was 12.11×106 kg bw. After the application of conditioning regimens, depending on the underlying disease, neutrophils engraftment occurs at 11 day and platelets engraftment at 14 day.

Conclusion: Analyzing data of the patients with hematological malignancies and ASCT conducted, we conclude that the mentioned procedure is successful method of treatment, with low transplant mortality and complications caused by the mentioned procedure.

http://dx.doi.org/10.1016/j.pbj.2017.07.062

PS237

Polymorphism of Kibra gene in patients with terminal renal insufficiency

N. Paovica*, D. Bajovic, I. Novakovic

Institute of Human Genetics, Faculty of Medicine, University of Belgrade, Serbia
E-mail address: 2carry.out2@gmail.com
(N. Paovica).

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 χ^2 (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 χ^2 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.[1–3]

References

1. Kremerskothen J, Plaas C, Buther K, et al. Characterization of KIBRA, a novel WW domain-containing protein. Biochem Biophys Res Commun. 2003;300:4.
2. Dunning K, Schurek EM, Schluter M, et al. KIBRA modulates directional migration of podocytes. J Am Soc Nephrol. 2008:19.
3. Maksimovic N. Analysis of polymorphisms in APOE, BDNF, BCHE and KIBRA genes and their correlation with memory performance in the population of students (Doctoral dissertation). NaRDuS – National Repository of Dissertations in Serbia.

http://dx.doi.org/10.1016/j.pbj.2017.07.063

Neurosciences Poster Session
Thursday, September 14th, 16h00

PS088

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

F. Barreto[1,2,*], R. Rodrigues[1,2], A. Cardoso[1,2], J.P. Andrade[1,2]

[1] *Department of Biomedicine – Unit of Anatomy, Faculty of Medicine, University of Porto, Alameda Prof. Hernâni Monteiro, 4200-319 Porto, Portugal*
[2] *Center of Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Rua Dr. Plácido da Costa, 4200-450 Porto, Portugal*
E-mail address: fbarreto44@hotmail.com
(F. Barreto).

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.

Acknowledgements: This article was supported by ERDF through the operation POCI-01-0145-FEDER-007746 funded by the Programa Operacional Competitividade e Internacionalização – COMPETE2020 and by National Funds through FCT - Fundação para a Ciência e a Tecnologia within CINTESIS, R&D Unit (reference UID/IC/4255/2013).

http://dx.doi.org/10.1016/j.pbj.2017.07.064

PS079

Analysis of variations in the F5, F2 and ACE genes among Latvian patients with ischemic stroke

Anna Inese Tutāne

Rīga Stradiņš University, Latvia
E-mail address: annatutane@gmail.com.

Aim: Evaluate thrombophilia causing genetic variants and ACE gene I/D variant impact on patients with ischemic stroke.

Introduction: Every year, 15 million people worldwide suffer a stroke that is the second leading cause of disability. Genetic variants in Leiden factor coding gene (F5) and in prothrombin gene (F2) cause inherited thrombophilia which is associated with increased risk of intravascular thrombosis, thromboembolism and cerebral stroke. Angiotensin-converting enzyme (ACE) coding gene I/D variant is discussed among numerous conditions including stroke.

Methods: In the study there were included 115 patients with mean age 70.3 ± 11.0 years, with diagnosed ischemic stroke. Control group for F5 and F2 gene variations consisted of 124 individuals with mean age 55.6 ± 14.6 years. And for ACE gene variation 248 individuals with mean age 56.8 ± 11.4 years. DNA was extracted from peripheral blood using standard phenol-chloroform method. Genotyping of F5 gene variant G1691A and F2 gene variant G20210A was performed using PCR-RFLP. ACE gene I/D variant genotyping were performed using PCR. Statistical analysis was performed using Fisher's exact test and SPSS v22.0 software.

Results: F2 gene variant were more frequent in patient group. Frequency in patients were 0.017 and in control group 0 ($p = 0.038$). F5 gene variant frequency in both patients and control group were 0.012 ($p > 0.05$). Seven patients (5.6%) had one variant in one of coagulation factors encoding genes comparing to three in control group (2.4%) ($p > 0.05$). Mean age for patients with identified variations in F2 or F5 was not significantly different comparing to other patients ($p > 0.05$). ACE gene I/D genotypes and allele frequencies in stroke patients were not significantly different from controls – I allele frequencies were 0.452 in patients versus 0.470 in controls ($p > 0.05$).

Conclusion: Prothrombin encoding gene variant G20210A could be risk factor for ischemic stroke. F5 and ACE gene I/D genotypes are not associated with ischemic stroke.

http://dx.doi.org/10.1016/j.pbj.2017.07.065

PS205

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

Dina Neves [1,*], João Bernardo [1], Patrícia Valentão [1], Maria C. Oliveira [2], David M. Pereira [1], Paula B. Andrade [1], Romeu A. Videira [1]

[1] *REQUIMTE/LAQV, Laboratório de Farmacognosia, Departamento de Química, Faculdade de Farmácia, Universidade do Porto, Rua de Jorge Viterbo Ferreira, Nº 228, 4050-213 Porto, Portugal*
[2] *Centro de Química de Vila Real (CQ-VR), Departamento de Química; Escola de Ciências da Vida e do Ambiente, Universidade de Trás-os-Montes e Alto Douro (UTAD), P.O. Box 1013, 5001-801 Vila Real, Portugal*
E-mail address: up201302607@ff.up.pt
(D. Neves).

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