deoxynucleotide pool size, while exposing cells to nutrient starvation caused reduced dNTP amounts. We are now in the process of further investigating this phenomenon by determining the mutation rates and mutational pattern arising from the applied genotoxic stress.

P.09-050-Tue
Low intensity exercise prevents disturbances in insulin regulation of α subunits of Na+/K+-ATPase in the heart of fructose-fed female rats

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Na^+/K^+-ATPase is an enzyme essential for regular functioning of the heart, regulated by insulin. Since estrogen deficiency combined with fructose rich diet provokes cardiac insulin resistance we hypothesized that sodium/potassium transport would be deteriorated accordingly and exercise training can prevent this disturbance. To test our hypothesis, we used fructose rich diet as animal model of insulin resistance in ovariectomized (OVX) female rats. OVX female Wistar rats were divided into three groups: sedentary control and sedentary and exercise groups submitted to fructose diet (10% fructose for 9 weeks). We analyzed biochemical parameters relevant for insulin action. Expression, phosphorylation and/or subcellular localization of cardiac insulin receptor (IR), insulin receptor substrate 1 (IRS1), protein kinase B (Akt) and Na^+/K^+-ATPase in basal and insulin-stimulated conditions were evaluated. Fructose diet did not change blood glucose level, but it increased plasma insulin level as well as homeostasis model assessment index, indicating insulin resistance. Exercise reversed these parameters to the control level. Fructose diet didn't have effects on cardiac IR, Akt (Thr308) and IRS1 (Tyr632) phosphorylation but it reduced Akt (Ser473), increased inhibitory IRS1 (Ser307) phosphorylation and increased expression and plasma membrane level of α1 and α2 subunits of Na^+/K^+-ATPase. Exercise returned Akt phosphorylation at Ser473 and increased at Thr308, without effect on IRS1 phosphorylation (Ser307), but returned total and plasma membrane level of α1 and α2 subunits of Na^+/K^+-ATPase. In conclusion, exercise prevents disturbances in cardiac sodium/potassium transport in fructose-fed OVX rats suggesting that low intensity physical activity might be important nonpharmacological treatment for cardiac insulin resistance.

P.09-051-Wed
Resolvin E1 regulates inflammatory mediators and oxidative stress enzymes of the cementoblasts

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Resolvin E1 (RvE1) has been effective in treatment of periodontitis and regenerated lost periodontal apparatus including new cementum formation in animals. The aim of this study was to investigate the action of RvE1 on the gene expression of inflammatory mediators and oxidative stress enzymes in cementoblasts. Murine immortalized cementoblasts (OCCM.30) were treated with different concentrations of RvE1 (0–1,000 ng/mL). The impact of RvE1 on OCCM.30 proliferation was determined for 360 h. RvE1 had a dose-dependent and bimodal impact on the expression of cytokines and enzymes. There was a statistically significant downregulation of IL-1β and IL-6 and a an upregulation in IL-10 in response to lower concentrations (0.1–1 ng/mL) of RvE1 on days 1 and 6 compared to control/untreated cells ($P < 0.01$). The elevated IL-1β/IL-10 and IL-6/IL-10 ratios indicated increased activity of a resolution of the inflammatory process in the early stage of the experiment. Higher concentrations (10–1,000 ng/mL) of RvE1 failed to stimulate the proliferation and expression of tested molecular targets. In contrast, expression of IL-1β, IL-6, IL-8 was significantly increased in response to higher doses of RvE1 while IL-10 expression was reduced ($P < 0.01$). We determined that the increase of IL-1β/IL-10, IL-6/IL-10, IL-8/IL-10 ratios with higher doses of RvE1 on days 1 and 6. RvE1 dose-dependently induced the expression of SOD and GPX in OCCM.30 ($P < 0.01$). These findings suggested that cementoblast function was regulated by the RvE1 where lower doses stimulate a resolution of inflammatory process in parallel with an increased enzymatic activity, which would favor a regenerative process and higher doses favor an activation of inflammation, which would be key for the resorptive role for the cementoblast-induced tissue turnover during the periodontal regeneration (Selcuk University Scientific Research Projects BAP-17401164).

P.09-052-Mon
The anti-neuroinflammation of erinacine A on LPS-induced Parkinson's disease in vivo and in vitro

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Hericium erinaceus (*H.E.*) is a kind of medicinal mushrooms in Asia, and has anti-oxidative, anti-diabetics and anti-hypertensive effects. Erinacine A (EA), the main active compound of *H.E.*, is demonstrated that it could inhibit the inflammatory cytokine expression in the model of stroke rats. However, few of researches discussed the preventive potencies of EA in neurotoxicity which is induced by inflammatory response. The previous studies found that neuroinflammatory response is induced by activated microglia which cause neurotoxicity and promote deteriorating of Parkinson's syndrome. This study aimed to investigate the neuroprotective effects of EA on LPS-induced neuroinflammation in the microglia cell, astrocyte, differentiated neuro-2a and animal model of semi-Parkinson's disease. Results demonstrated EA and *H.E.* could improve the physical coordination in the rotation test and dramatically decrease the expression of inflammatory cytokines, TNF-a, iNOS and IL-1b, *in vivo*. In addition, pretreated EA significantly suppressed iNOS expression and NO production in LPS-induced activated BV-2 cells. Moreover, pretreated EA could decrease TNF-a mRNA expression in astrocyte which was stimulated by LPS. Additionally, EA and *H.E.* could increase TH but decrease p-NF-kB, p-JNK expression on BV-2 conditioned medium in differentiated neuro-2a. Those results show that, *H.E.* and EA could ameliorate LPS-induced the semi-Parkinson's syndrome, inhibit the iNOS expression in activated BV-2 cells and also prevented the cell death from BV-2 conditioned medium in differentiated neuro-2a. Therefore, *H.E.* and EA may possess potent anti-inflammatory activity and improvement of Parkinson's disease.

FEBS Open Bio **8** (Suppl. S1) (2018) 107–496 DOI: 10.1002/2211-5463.12453