

(33 versus 38 days, respectively). Puberty onset was delayed in the rats receiving kisspeptin antagonist (41 days). Combination of kisspeptin and peptide 234 resulted in puberty onset similar to sham rats (37 versus 38 days, respectively). Pubertal weight was found to be lower in the kisspeptin injected rats compared to sham group (66.7 ± 3.8 and 86.4 ± 5.4 g, respectively). In conclusion, this novel kisspeptin antagonist peptide 234 appears to modulate the effects of kisspeptin on puberty onset in female rats.

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Decreased insulin sensitivity in multiple sclerosis

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Introduction: Multiple sclerosis (MS) is autoimmune neurological disease characterized by demyelination, leading to various neurological symptoms including movement impairment, vision problems, loss of sensitivity and others. Less is known about metabolic alterations during MS, so the aim of our study was to assess glucose metabolism status in MS patients.

Methods: We examined 19 patients with MS and 19 healthy controls matched for sex, age and body mass index (BMI) (9 males/10 females, age 30.4 ± 7.1 and 28.7 ± 6.7 years; BMI 23.7 ± 4.5 and 24.4 ± 5.3 kg/m² respectively). MS patients were newly diagnosed; first occurrence of MS symptoms was treated by short term methylprednisolone therapy. Examinations were performed 2-3 months after that and patients were currently in remission without any therapy. We used standard oral glucose tolerance test (oGTT), blood was drawn in 15 minute intervals for 2 hours. Glucose, insulin and GLP-1 and lipid parameters were measured. Insulin sensitivity indices (ISI) were calculated.

Results: Fasting plasma glucose was similar in both MS patients and controls (5.2 ± 0.3 vs. 5.0 ± 0.4 mmol/l, $p=0.05$) with similar levels of insulin (5.6 ± 5.2 vs. 3.9 ± 2.6 mIU/l, $p=0.216$), resulting in comparable index of insulin resistance IR-HOMA (1.33 ± 1.28 vs. 0.90 ± 0.62 , $p=0.197$). During oGTT glucose levels tended to be higher in MS group, but not significant ($p=0.076$). However we found clearly increased levels of insulin in MS group ($p=0.022$) during oGTT. Insulin sensitivity was significantly lower in MS group compared to control group [ISI(Matsuda) 6.95 ± 3.44 vs. 10.60 ± 4.81 , $p=0.011$ and ISI(Cederholm) 49.9 ± 15.3 vs. 61.3 ± 16.3 , $p=0.032$]. Levels of GLP-1 were comparable at the baseline and during oGTT in both groups. We did not find any difference in total, HDL and LDL cholesterol as well as in triglycerides levels between groups.

Conclusions: We found decreased insulin sensitivity in patients with MS compensated by hyperinsulinemia. This

could predispose MS patients for future Type 2 diabetes mellitus development.

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The effects of hyperbaric oxygen therapy (HBOT) on blood viscosity and erythrocyte aggregation in diabetic patients

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There are only a few studies about hyperbaric oxygen's effect on hemorheological parameters and their results showed an increase in blood viscosity and RBC aggregation both in vivo and in vitro. Some many other studies showed abnormal hemorheological parameters in diabetics and so, this would suggest more complications after HBO therapy however; reality is not consistent with this suggestion. Therefore, in this study, the effects of hyperbaric oxygen therapy on blood viscosity and erythrocyte aggregation have been investigated.

After the approval of local ethical committee, 11 diabetic ulcer patients aged between 42 and 82 were taken to our study. 100% oxygen was applied at 2.4 ATA for two hours in three cycles of 25 minutes of oxygen- 5 minutes air break. Treatments were carried on five days a week. Blood that was collected before the initial HBO therapy was accepted to be control. Samples were also collected after the initial therapy and twentieth one to be evaluated. Corrected whole blood viscosity was measured using a cone/plate viscometer with a hematocrit of 45%. RBC aggregation was measured using a Myrenne aggregometer in both autologous plasma and dextran70 solution.

Our results showed that there were no significant changes in corrected blood viscosity between the samples collected before and after the first and twentieth HBO treatments. Also RBC aggregation in both autologous plasma and dextran70 solution after the first and twentieth HBO treatments were not significantly different than the control samples.

These results were in contrast with the previous experimental studies. The reason of these contradictory results may be caused by experimental method and HBO application differences and/or different reactions of humans and animals. Still, this topic needs further studies to clear such an important effect.