BS07

The effect of chronic oral d-galactose administration on colon redox homeostasis in a rat model of sporadic Alzheimer's disease

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INTRODUCTION/OBJECTIVES: Parenteral administration of d-galactose is commonly used to model aging in rodents. Cognitive deficit observed in the model is explained by d-galactose induced oxidative stress. Conversely, chronic oral administration of d-galactose prevented cognitive deficit in the streptozotocin-induced rat model of Alzheimer's disease (STZ-icv). Hence, the study aims to assess the effect of chronic oral d-galactose on redox homeostasis in the colon of STZ-icv and control rats.

MATERIALS AND METHODS: Three-month-old male Wistar rats (N=40) were split into two groups treated bilaterally by intracerebroventricular injection of either streptozotocin (STZ-icv,3 mg/kg) or vehicle (CTR). Animals were further assigned into a group receiving daily oral galactose solution (200 mg/kg) or vehicle (tap water). After two months, rats were euthanized and colons (N=20) dissected and stored at -80°C for further analysis. Total antioxidant capacity (TAC) was evaluated by nitrocellulose redox permanganometry (NRP) and ABTS. Lipid peroxidation was assessed by thiobarbituric acid reactive substances (TBARS), catalase activity was assessed indirectly by quantification of the carbonato-cobaltate (III) complex, low molecular weight thiols (LMWT) and total protein sulfhydryls (SH) were measured with 5,5′-dithio-bis(2-nitrobenzoate).

RESULTS: STZ-icv group had a higher concentration of LMWT and lower TBARS compared to the control group. D-galactose treatment increased LMWT, decreased TBARS and catalase activity in both STZ-icv and CTR groups. Colon TAC and SH levels were decreased only in the galactose-treated STZ-icv group.

CONCLUSION: Colon redox homeostasis is altered in the STZ-icv rat model. Chronic oral d-galactose may exert beneficial effects by shifting redox homeostasis toward a more reductive/antioxidant state.

BS08

Stable Gastric Pentadecapeptide BPC 157 Counteracting Effects on Postsplenectomy Complications in Rats

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Keywords: BPC 157, Postsplenectomy complications, Splenectomy, Thrombosis

INTRODUCTION/OBJECTIVES: We focused on Stable gastric pentadecapeptide BPC 157 counteracting effects on complications taking place after splenectomy in rats, including portal vein, superior mesenteric vein, lienal vein and inferior vena cava thrombosis, severe venous hypertension, abdominal aorta hypotension, liver damage and subsequent cerebral edema.

MATERIALS AND METHODS: Wistar rats were deeply anaesthetized and underwent complete laparotomy followed by splenectomy. Immediately after splenectomy, portal vein was clamped using a vascular clamp for 15 minutes to induce thrombosis of portal vein and its contributories. Medication (BPC 157 (10 µg/kg) (treated group) or saline (5 mL) (control group)) was applied as an abdominal bath immediately after the vascular clamp removal. Rats' vessels, organs and brains were filmed 10 minutes and 24 hours after medication application using USB microcamera. Furthermore, blood pressure was measured via intravascular cannulation and thrombi were extracted and weighed. Using ImageJ software and with our knowledge of Square-cube law, we were able to express relative brain volumes and then graphically display data.

RESULTS: Blood pressure values (portal vein and inferior vena cava hypertension and aortic hypotension) showed significant differences between control and treated groups already after 10 minutes and those differences became even more distinct after 24 hours. Similar pattern is seen with thrombosis, therefore BPC 157 treated rats showed reduced thrombi weights. Macroscopically, control group presented with portal vein congestion and thrombosis, liver congestion and damage and cerebral edema, whereas the treated group showed none of the above.

CONCLUSION: These findings suggest that BPC 157 may be therapeutic solution for postsplenectomy portal venous system thrombosis.