2-B-01 Influence of neuropeptide W on feeding action by voluntary exercise

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Purpose: Neuropeptide W (NPW) has roles in the regulation of feeding behavior and energy metabolism. Administration of NPW decreases food intake (FI) in rodents. Several types of stress suppress feeding behavior through the hypothalamus-pituitary-adrenal gland (HPA) axis. NPW promotes to secrete both ACTH and corticosterone. Generally voluntary wheel running reduces the activation of HPA axis to stimuli. We hypothesized that NPW mediates a decrease in food intake via the HPA axis.

Methods: Normal diet-fed and diet-induced obese (DIO) mice were icv administered vehicle or NPW (2 nmol). Body weight (BW), FI, water intake (WI) and locomotor activity(LA) were recorded for 24 hours. BW was measured at 24h after the injection. The voluntarily exercising mice were administered vehicle or NPW. Running milage was recorded for 24 hours. BW, FI, WI were measured at 24h after the injection. Moreover, immunohistochemistry with anti-cFos antibody was performed on cryosections from the NPW-treated mice brain with or without voluntary exercise.

Results: NPW significantly decreased both FI and WI in the normal diet-fed mice, NPW unchanged FI, WI, and LA in DIO mice. Voluntary exercise canceled the NPW-induced hypophesia and decreased NPW-induced c-Fos expression in the hypothalamic paraventricular nucleus (PVN).

Discussion: Because of high plasma level of corticosterone in the DIO mice, it is possible that feedback system for PHA-axis is already failed. NPW-induced activation of neurons in the PVN by NPW administration were attenuated by voluntarily exercise. These results suggest that NPW decreases FI via the HPA axis.

Key Words: Neuropeptide W (NPW), voluntary exercise, feeding behavior, hypothalamus-pituitary-adrenal gland

2-B-02 Exercise has preventive effects on cognitive impairment in senescence -accelerated mice

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Purpose: In the present study, we focused on exercise as preventive tools for cognitive impairment, and searched for novel molecules, which may relate to prevention of cognitive impairment, in the hippocampus. **Methods:** Two-month old senescence-accelerated mouse prone-8 (SAMP8) mice were subjected to voluntary exercise training on running wheel for 4 months, and then assigned a conditioned fear memory test. Moreover, various mRNA levels in the hippocampus were examined by DNA analysis and real-time PCR. **Results and Discussion:** Contextual fear memory in SAMP8 control mice was significantly impaired as compared with that in non-senescence mice. Exercise training definitely attenuated such cognitive impairment. The results on real-time PCR analysis made following DNA array analysis in the hippocampus revealed that, compared with SAMR8 control mice, the expression levels of some mRNAs, such as brain-derived neurotrophic factor and leucine zipper transcription factor-like 1 (Lztfl1), were significantly higher in SAMP8 mice subjected to exercise training. In addition, overexpression of Lztfl1 promoted neurite outgrowth in Neuro2a cells. These results suggest that exercise has preventive effects on cognitive impairment in SAMP8 mice, and exercise-induced increases in Lztfl1 were thought to implicate neurite outgrowth.

Key Words: exercise training, SAMP8 mice, cognitive impairment, hippocampus, Lztfl1