**2AD5** Influence of exercise habits on nocturnal heart rate variability in young men -Practical use employing Lorez-plot-

Kisato KANOH1, Kiyotaka SATO1, Noriko ABE1, Jun SATOMI2, Goken SAKAMOTO2, Sachio KAWAI2
1Institute of Health and Sports Science, Chuo Univ., 2College of Science and Engineering, Ritsumeikan Univ., 3School of Health and Sports Science, Juntendo Univ.

**Purpose:** This study was designed to compare autonomic nervous system activity in healthy young men with different exercise habits. In order to obtain reliable indices for practical use, we also tested the consistency of the new methods employing Lorez-plot.

**Methods:** Nocturnal heart rate variability (HRV) was measured in healthy young students with (Group-A) and without (Group-N) a habit of physical training. All of them were free from any known diseases, nonsmokers, and were taking no drugs. Daily activities were recorded with a questionnaire, and they were also instructed to refrain from exercises on the day of measurement. The data were analyzed by Mem-Calc(GMS) and the cardiac vagal function index (CVI: Log\(_{10}(L 	imes T)\)), the sympathetic index (CSI: L/T) were calculated according to the method of Toichi et al. **Results and Discussion:** CVI and CSI were found to be closely related with the frequency indices of spectral analysis, such as HF (High-frequency power) reflecting parasympathetic activity, and the ratio of LF/HF, indicating sympathetic functions (p < 0.01, p < 0.05 respectively). As an example, the effects of training on HRV could not be clearly found out in this study. This may be partly due to the homogeneity of our subjects and the lack of previous training control in A-Group. Based on the evaluation of automatic nervous system activity, obtained from simple figure, we will study further on its usefulness.

**Key Words:** Heart rate variability, Lorenz plot, Exercise habit

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**2AD6** MAPK activation response to an acute bout of exercise in untrained and trained rat hearts

Motoyuki IEMITSU1,2, Seiji MAEDA1,2, Takeshi OTSU1,2 and Takashi MIYACHI1,2
1Institute of Health and Sport Sciences and 2Center for Tsukuba Advanced Research Alliance (TARA), University of Tsukuba

**Purpose:** Mitogen-activated protein kinase (MAPK) signaling pathways, such as extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), and p38 MAPK, play a critical role in cell hypertrophy. As exercise training causes cardiac hypertrophy, and a single bout of exercise induces mechanical stress to the heart, the present study aimed to characterize the activation patterns of multiple MAPK signaling pathways in the heart after a single bout of exercise or chronic exercise.

**Methods:** The hearts of untrained rats received 5, 15, and 30 min treadmill running exercise (Ex5, Ex15, Ex30) and rested for 0.5, 1, 3, 6, 12, and 24 h (PostEx0.5, PostEx1, PostEx3, PostEx6, PostEx12, PostEx24, each group of N = 6) before subjecting them to the following different experiments. Additionally, to investigate MAPK activation response to an acute bout of exercise in the heart of chronically exercise-trained rats, the trained rats received exercise training were randomly divided into two groups: resting control and 30-min exercise after 4, 8, or 12 weeks exercise training. **Results:** Activation of MAPKs (ERK, JNK, and p38) increased immediately after acute exercise in a time-dependent manner, with ERK, JNK, and p38 peaking at Ex15, Ex15, and Ex30, respectively. The elevated levels of MAPKs declined to the resting levels within 24 h after exercise. In another set of experiments, following a 4-, 8-, and 12-week exercise training, the rats exhibited significant cardiac hypertrophy by week 12. Left ventricular weight and myocyte surface area were significantly higher in the 12-week-trained rats compared to the age-matched sedentary rats, while unchanged in 4- or 8-week exercise-trained rats. Activation of MAPKs in the 4-week trained rats increased after 30-min single bout of exercise, but decreased in the 8-week group. Finally, the activity of MAPKs signaling in the 12-week trained rats exposed to an acute bout of exercise was unaltered.

**Conclusion:** We conclude that exercise induces the activation of multiple MAPK (ERK, JNK, and p38) pathways in the heart, an effect that gradually declines with the development of exercise-induced cardiac hypertrophy.

**Key Words:** Cardiac hypertrophy, ERK, JNK, p38