Elevated Cardiac Vagal Tone in Hypoestrogenic Active Premenopausal Women with Functional Hypothalamic Amenorrhea

Emma O'Donnell[1], Jack M Goodman[1,2], Beverly L Morris[3], John S Floras[2,3], and Paula J Harvey[4]

[1]Cardiovascular Research Laboratory, Department of Exercise Sciences, University of Toronto, Toronto, Ontario, Canada, and Division of Cardiology at the [2]Mount Sinai Hospital, [3]Toronto General Hospital, and [4]Women's College Hospital, University of Toronto, Ontario, Canada.

Compared with eumenorrheic women, exercise-trained women with functional hypothalamic amenorrhea (ExFHA) have a low heart rate (HR), absent reflex activation of their renin-angiotensin-system, and augmentation of the normal increase in muscle sympathetic nerve burst incidence during orthostatic stress, suggesting concurrently altered autonomic HR modulation. To test this hypothesis, three age-matched (pooled mean, 24±1 years; mean±SEM) groups of women were studied: ExFHA (n=11), exercise-trained and eumenorrheic (ExOv; n=17), and sedentary and eumenorrheic (SedOv; n=17). Blood pressure (BP), HR, and HR variability (HRV) in the frequency domain were measured at supine rest and during simulated orthostatic stress induced by graded lower body negative pressure (LBNP; -10, -20, and -40mmHg). Very low (VLF), low (LF) and high (HF) frequency power spectra (ms2) were determined, and due to skewness, were $\log_{10}$ transformed. LF/HF ratio and Total power (VLF+LF+HF) were calculated. At baseline, HR and systolic BP were lower ($p<0.05$), and HF and Total power higher ($p<0.05$) in ExFHA than eumenorrheic women. In all groups, LBNP decreased ($p<0.05$) systolic BP, HF and Total power, and increased ($p<0.05$) HR, and LF/HF ratio. However, in ExFHA, HF and Total power remained higher ($p<0.05$), and HR, systolic BP

and LF/HF ratio lower ($p<0.05$) than in eumenorrheic women, in whom measures did not differ ($p>0.05$). In conclusion, ExFHA women demonstrate augmented vagal HR modulation, whereas sympathetic HR modulation is unchanged, both at rest and during orthostatic stress. Less central angiotensin II may play a role.

Abstract Sponsor: Dr. John S Floras

Sponsor email: jfloras@mtsinai.on.ca

Sponsor telephone number: 416-586-8704