

## **Antioxidants Attenuate the Exercise Induced Resetting of the Arterial Baroreflex in Healthy Human Subjects: Implications for Exercise Induced Hypertension**

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### ABSTRACT

Patients with Exercise-induced-Hypertension (EiHT) exhibit exaggerated increases in arterial pressure at the onset of exercise which may prevent EiHT patients from participating in exercise training programs. EiHT is thought to occur due to dysregulated resetting of the arterial baroreflex (ABR). Prior studies in animal models demonstrate that reactive oxygen species (ROS) generated in the brainstem scavenge the sympathoinhibitory function of central Nitric Oxide (NO) and, thereby enable ABR resetting of the operating point (OP) pressure and hypertension. We tested the hypothesis that a centrally and peripherally active antioxidant cocktail (CT; composed of Vitamin E and C with Co-Q10) will attenuate the exercise induced resetting of the ABR's centering point (CP) and OP pressures compared to the same exercise intensity performed with a vehicle placebo (PL). Seven healthy human subjects were recruited and performed 700 back-supported semi-recumbent dynamic leg exercise at moderate (HR at 120 beats per minute: e120) and heavy (HR at 150 beats per minute: e150) intensities. Mean arterial pressure (MAP) was continuously recorded using photoplethysmography at the finger, while HR was recorded via a three lead electrocardiogram (ECG). On experimental day 1, subjects were either given the CT or PL 1 hr. (time of peak plasma concentrations) prior to the start of exercise. On a separate experiment day 2, the subjects repeated the same exercise intensity protocol with the other test article (CT or PL) in a randomized repeated measures design. During exercise with the PL ingestion, the CP of the ABR was reset to higher MAPs from rest to e120 ( $100 \pm 3$  mm Hg to  $121 \pm 3$  mm Hg,  $P < 0.02$ ) but not e150 ( $113 \pm 3$  mm Hg,  $P = 0.15$ ). The absence of resetting at the higher work intensity was likely due to cardiovascular drift (decreasing MAP). Ingestion of the CT prior to the exercise protocols prevented the increase of the CP to higher MAPs from rest to e120 and e150 (rest:  $97 \pm 3$  mm Hg, e120:  $106 \pm 3$  mm Hg, e150:  $106 \pm 3$  mm Hg,  $P > 0.21$ ). Furthermore, the OP- pressure of the ABR was attenuated with CT ingestion compared to PL at e120 (placebo e120:  $116 \pm 0.8$  mm Hg, CT e120:  $111 \pm 0.8$  mm Hg,  $P = 0.04$ ). These data: (a) confirm that centrally derived ROS contribute to exercise induced ABR resetting; and (b) indicate that EiHT could be treated by ingestion of an anti-oxidant cocktail prior to the start of exercise.