Heat, Hydration & Women

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Tori Stone, Ph.D., The John B. Pierce Laboratory, Yale School of Medicine, Department of Obstetrics, Gynecology & Reproductive Sciences, New Haven, CT 06519

Cardiovascular and Temperature Regulation during Exercise Heat Stress

Cardiovascular strain is greater during exercise in the heat versus a temperate environment. Generally, the thermoregulatory system takes priority over maintaining cardiovascular performance in hot conditions—it is more important to maintain core temperature at a safe level.

High core temperature (T_c), i.e., hyperthermia, increases heart rate (HR) intrinsically and through augmented sympathetic activity, both of which result in decreased ventricular filling time and thereby stroke volume (SV)[1]. Hyperthermia leads to increased sweating, which is an important cooling mechanism during exercise but results in dehydration that augments increased HR and the reductions in SV sustained during prolonged exercise heat stress. Progressive increases in HR and decreases in SV characterize the cardiovascular drift (CV drift) phenomenon that is expected during constant-rate, submaximal aerobic exercise. The magnitude of CV drift is greatest during exercise in the heat and associated with proportional decrements in maximal oxygen uptake ($\dot{V}O_{2max}$)[2]. Compared to other factors related to the CV drift-$\dot{V}O_{2max}$ relationship (e.g., hydration[3], exercise mode[4]), hyperthermia results in greater CV drift and reductions in $\dot{V}O_{2max}$[5]. Given the profound impact of hyperthermia, the inclusion of women in studies investigating CV drift-$\dot{V}O_{2max}$ relationship has been a concern.

Investigating the CV drift-$\dot{V}O_{2max}$ Relationship in Women

Peak progesterone levels in the luteal phase (LP) of the menstrual cycle increase T_c ~ 0.2–0.5 °C versus the follicular phase (FP) when levels are relatively low. Previous research would suggest that the phase-related difference in T_c is significant in terms of hyperthermia related to CV drift; however, we recently published empirical findings showing that the CV drift-$\dot{V}O_{2max}$ relationship is similar between the FP and LP[6]. Absolute HR was similar between phases, despite higher T_c in the LP (+0.3 °C vs. FP) that maintained over the 45-min exercise bout. Interestingly, the decrement in SV was greater in the LP vs FP. Absolute SV was higher in the LP prior to the onset of CV drift, which could be related to estrogenic effects on blood volume, and decreased to a similar absolute level as the FP after 45 min. Similar reductions in $\dot{V}O_{2max}$ between phases suggested that level of absolute SV rather than magnitude of reduction may primarily determine the decrements in performance associated with CV drift.

Women have also been excluded from exercise studies due to perceived sex differences in fitness level. Our recent findings (unpublished) suggest that women and men could potentially be in the same study if exercise is prescribed using a fixed level of metabolic heat production. It is common practice to use a relative percentage of $\dot{V}O_{2max}$ in exercise studies because it assures the same relative intensity among subjects. This method is fine for a repeated-measures design but problematic for independent comparisons if groups are unmatched for fitness level—high-fit subjects exercise at a greater absolute intensity than low-fit subjects to achieve the same given percentage of $\dot{V}O_{2max}$, leading to greater levels of metabolic heat production and thermal strain[7]. We compared the CV drift-$\dot{V}O_{2max}$ relationship between groups of high- (HI) and low-fit (LO) women using 60% of $\dot{V}O_{2max}$ (REL) and a fixed level of metabolic heat production (FIXED). In the REL condition, greater metabolic heat production in the HI group lead to greater hyperthermia, and thereby CV drift and reduced $\dot{V}O_{2max}$ compared to the LO group, but these differences were not present in the FIXED condition.

If the goal is to include men and women in the study, it is important to use a level of metabolic heat production below the sudomotor threshold for women. Previous studies found that sweat production plateaus during exercise performed at a high level of metabolic heat production (500 W), resulting in greater increases in T_c than men exercising at the same level[8]. Based on preliminary findings, it seems that greater increases in T_c resulting from sudomotor sex differences do not result in statistically different magnitudes of CV drift and decrements in $\dot{V}O_{2max}$ during exercise performed this particular, high level of metabolic heat production. Further interruption is needed, but these findings may support our previous connotation that considerably large differences in T_c are required to modulate the CV drift-$\dot{V}O_{2max}$ relationship.

Sex Hormones and Hydration

Another aspect of consideration in the data we do have in women, is how that data was collected in terms of hormonal status. Many studies seeking to investigate sex differences between men and women controlled the studies in such a way that the women were tested in the early follicular phase of the menstrual cycle, when sex hormones are characteristically low. Analyses in this phase only doesn't allow for understanding of the role sex hormones play. From vasculature, temperature regulation, to fluid regulation, female sex hormones can impact a wide variety of physiological functions, so it is important to have a comprehensive understanding of those effects.

Specific to hydration, estrogen and progesterone, the primary female sex hormones that vary drastically between phases of the menstrual cycle, are closely related to potent fluid regulatory hormones. Specifically, estrogen is closely related to arginine vasopressin (AVP) via lowering the osmotic threshold for AVP release[9-11] which stimulates the retention of water through the aquaporin receptors at the kidney; and progesterone is closely linked to aldosterone[11, 12] which stimulates sodium and water reuptake in the kidney. When estrogen and progesterone cycle, these fluid regulatory hormones cycle as well. This alters fluid reuptake, retention, possible edema, and potential fluid needs depending on hormonal concentrations.

Another factor specific to women is contraception. A majority of women who utilize contraception use oral contraceptive pills (OCPs) which have synthetic exogenous hormones, ethinyl estradiol and progestin, that interact with the body differently than endogenous estrogen and progesterone. Women utilizing OCPs have increased concentrations of the exogenous hormones than would be present in naturally cycling women[13]. While we do not have comprehensive knowledge of differences in OCP users and naturally cycling women, we do know that OCP users have a lower osmotic threshold for ACP secretion and thirst stimulation[14], but this is specific to monophasic OCPs and may not represent all OCPs that have varying concentrations of exogenous hormones or implantable contraceptive devices (intrauterine devices, implantable bar).

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