

## EFFECTS OF HYPOHYDRATION ON NEUROMUSCULAR RESPONSES AFTER CYCLING IN THE HEAT

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### SUMMARY

This study evaluated the effect of hypohydration induced by cycling in the heat on neuromuscular responses of knee extensors muscles. Ten male subjects exercised in the heat in two separate sessions: the first session (HY) was aimed at reaching 2% body weight (BW) loss, while the second (EU) no BW loss. Maximal isometric knee extensors torque, EMG activity and vastus lateralis muscle architecture (muscle thickness, pennation angle and fascicle length) were assessed before and after both sessions. Urine specific gravity (USG) was assessed pre- and post-exercise to check hydration status. Rectal temperature was maintained below 38°C during the neuromuscular evaluations. Pre-exercise USG (<1.020) indicated that subjects were euhydrated prior to both sessions, and it increased only in the HY condition. A 15.8% reduction ( $p < 0.05$ ) in maximal isometric knee extensors torque was observed in the HY compared to 2.98% ( $p > 0.05$ ) in the EU. EMG activation reduced in the same magnitude in both sessions, while muscle architecture parameters were unaffected by the protocols. Hypohydration induced by cycling in the heat impaired maximal knee extensor muscles strength production, but vastus lateralis neural and structural changes cannot explain this phenomenon.

### INTRODUCTION

It is still uncertain whether hypohydration impairs neuromuscular function [1]. Some studies have demonstrated a muscle strength reduction after inducing hypohydration [2,3], while others [4,5] found no changes. As reported by Judelson et al. (2007) [1], inconsistent results might be explained by the different methods used to achieve hypohydration and to the magnitude of the hypohydration induced. Depending on the hypohydration protocol, different factors may play a role in the determination of strength loss, such as hyperthermia and muscle fatigue. This study controlled the main intervenient factors on neuromuscular function in order to investigate the effect of hypohydration on the knee extensors neuromuscular responses after exercising in the heat on a cycle ergometer.

### METHODS

Ten healthy physically active men (age =  $22.7 \pm 2.2$  years old, body mass =  $77.9 \pm 7.3$  kg, height =  $176 \pm 6.4$  cm, body fat =  $18.9 \pm 3.0\%$ ) came to the laboratory in two sessions. The first session was designed to induce hypohydration (HY) (2% body weight loss), while in the second session subjects were kept euhydrated (EU). With the exception of

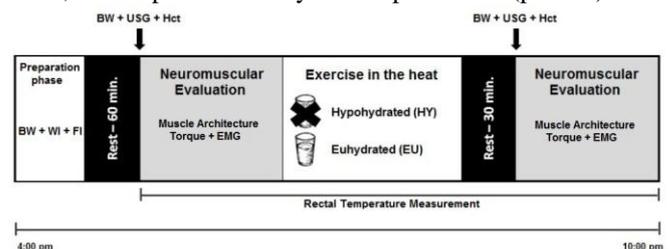
fluid ingestion during the exercise in EU, participants underwent the same protocol as follows (Figure 1): (1) a preparatory phase consisting of body weight assessment, water intake (6 ml/Kg), standard meal (161 Kcal – 77% CHO) and 60-min rest in supine position, (2) neuromuscular assessment, (3) exercise in the heat, (4) bath and a 30-min rest in supine position, and (5) neuromuscular assessment as performed before exercise.

Subjects cycled on a cycle ergometer at a constant load of 100 W, between 80-90 rpm in the heat (36-37°C - 45% relative humidity) inside an environmental chamber. Every 20 minutes, subjects interrupted pedaling and urinated (whenever was possible), dried sweat and unclothed body weight was obtained. In this first session, these procedures were repeated until body weight loss from the initial value achieved ~2%. No fluid ingestion was allowed.

USG and Hct was assessed before and immediately after exercise in the heat, and hypohydration was considered when  $USG > 1.020$  and  $Hct > 52\%$ . Rectal temperature ( $T_{re}$ ) was assessed during the entire protocol and neuromuscular assessments were performed with  $T_{re} < 38^\circ\text{C}$  [6].

The neuromuscular evaluation consisted of: (1) ultrasonography of vastus lateralis (VL) muscle to assess muscle architecture parameters (i.e., muscle thickness, pennation angle and fascicle length); (2) three five-second maximal voluntary contractions (MVCs) at 60° of knee flexion (0° = full extension) with 2-min rest between consecutive MVCs to assess peak torque; and (3) electromyography (EMG) of VL muscle during MVCs to assess muscle activation through RMS values.

A two-way ANOVA [session (HY and EU) x time (pre-exercise and post-exercise)] was used to examine the global effect of hypohydration on muscle architecture, knee extensor torque and VL EMG responses, as well as values of BW, USG and Hct. When between factors interaction was found, a LSD post-hoc analysis was performed ( $p < 0.05$ ).



**Figure 1.** Study design. BW: body weight; WI: water intake; FI: food intake; USG: urine specific gravity; Hct: hematocrit; EMG: electromyography.

## RESULTS AND DISCUSSION

A significant session-time interaction was observed for BW ( $p < 0.001$ ) and USG ( $p < 0.001$ ), but not for Hct ( $p = 0.891$ ). BW changes and the target hydration level was achieved in the HY session ( $p < 0.001$ ). Moreover, subjects started both sessions euhydrated ( $USG < 1.020$ ) and USG increased only in the HY session ( $p < 0.001$ ).

A significant session-time interaction was found for the knee extension torque ( $p = 0.001$ ). Baseline knee extensors torque was similar between sessions ( $p = 0.736$ ). However, HY session decreased the torque values in 15.8% ( $p < 0.001$ ), while non-significant reduction (2.98%;  $p = 0.058$ ) was observed in the EU session (Table 1).

No significant session-time interaction was observed in VL EMG responses ( $p = 0.661$ ), but a significant time effect ( $p = 0.009$ ) suggested a decrease of EMG signal in both HY and EU situations (Table 1).

No significant session-time interaction was observed in VL muscle thickness ( $p = 0.222$ ), pennation angle ( $p = 0.076$ ) and fascicle length ( $p = 0.103$ ) (Table 1).

We found two studies similar to ours, which used cycling to assess the hypohydration effects on the muscular strength [2,4]. Saltin et al (1964) [4] observed no changes in maximal quadriceps strength before and after a 90-min cycling protocol in the heat (36-38.5°C), suggesting that knee extensor strength impairment in the present study was relatively higher. Vallier et al (2005) [2] reported a significant reduction in knee extensors maximum isometric torque after a 180-min cycling in a thermoneutral environment, when hypohydration reached ~4.0% of BW loss. Although the subjects from Vallier et al (2005) [2] reached twice the level of hypohydration than that of our study, the maximal strength drop was similar (~16%), which may be related to the individual characteristics of the subjects, the intensity and volume of the exercise protocol, and the resting period between the exercise protocol and the strength measurements.

A suggested neuromuscular component to explain muscle strength impairment due to hypohydration is a reduction in the number of recruited motor units [1-3]. However, our results do not agree with this suggestion, because HY and EU decreased the VL RMS values compared to the pre-exercise condition. Vallier et al (2005) [2] also found a reduction in the VL RMS values after two hours of cycling exercise with and without water intake (18% and 26% respectively), in agreement with the results of this study.

Thus, the reduction on VL RMS values might be explained by neuromuscular fatigue induced by cycling in the heat and not by the hypohydration itself.

Evidence showed that muscle tissue water content was reduced due to hypohydration [7], and that could have generated structural and functional responses [8]. Although the effect of hypohydration on muscle structure in humans is unknown, changes in pennation angle or fascicle length could change the muscle mechanical properties and, consequently, strength capacity. Although no significant change in muscle architecture was observed, our study seems to be the first to evaluate muscle architecture changes after hypohydration. Additional studies are needed to determine possible structural responses in skeletal muscle due to water loss.

## CONCLUSION

In summary, our findings demonstrated that hypohydration induced by cycling in the heat impaired maximal knee extensors strength, but VL neural and structural changes cannot explain this phenomenon.

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**Table 1:** Maximal isometric knee extensors torque, EMG activation and Muscle architecture parameters before and after exercise in both sessions (Mean  $\pm$  SD).

	Hypohydrated		Euhydrated	
	Before	After	Before	After
<b>Torque (N.m)</b>	294.27 $\pm$ 44.82	247.16 $\pm$ 40.54* <sup>&amp;</sup>	291.99 $\pm$ 48.37	281.74 $\pm$ 38.65 <sup>&amp;</sup>
<b>EMG (mV)</b>	0.30 $\pm$ 0.17	0.19 $\pm$ 0.09*	0.31 $\pm$ 0.17	0.19 $\pm$ 0.07*
<b>Muscle thickness (cm)</b>	2.33 $\pm$ 0.42	2.22 $\pm$ 0.43	2.36 $\pm$ 0.46	2.35 $\pm$ 0.48
<b>Fascicle length (cm)</b>	5.09 $\pm$ 1.56	5.97 $\pm$ 1.70	5.00 $\pm$ 1.26	4.98 $\pm$ 1.68
<b>Pennation angle (°)</b>	22.03 $\pm$ 4.89	19.12 $\pm$ 4.14	22.11 $\pm$ 4.89	22.50 $\pm$ 4.57

\* different from pre-exercise ( $p < 0.05$ ); & different between sessions ( $p < 0.05$ )