Water is required for multiple processes related to muscle contraction from ATP hydrolysis at the myosin head to Krebs cycle reactions. In addition, water forms in muscle during the process of glycolysis and at the final step of mitochondria electron transport chain. Despite the utmost relevance of water for muscle energetics, it is largely unknown if humans incur into muscle water deficit to the extent to compromise contractile function or muscle energetics. It is however relevant to define the role of muscle water on fatigability and exercise tolerance since those are the main barriers for achieving health benefits from exercise.

Costill and associates studied muscle water changes during and after prolonged dehydrating exercise. They found that if muscle tissue is collected after 15 min of exercise, muscle does not decrease but rather increases its water content. Sjøgaard and Saltin also reported increases in active muscle water when sampling muscle right after three bouts of supramaximal cycling exercise. Likely, the increase in blood perfusion pressure and capillary recruitment with exercise results in a transient rise in muscle fluid content. However, Costill and co-workers were able to measure reductions in muscle water content when body weight decreased 5.8% by dehydration. After exercise, hydrostatic pressure rapidly declines to resting values allowing muscle water to shift to other fluid spaces and replenish their fluid losses. We have recently shown that whole body dehydration (4.2%) reduces muscle water content when 1 h of recovery is allowed to balance between body fluids. In fact, the reduction in muscle water content coincided with the recovery of plasma fluid suggesting the predominance of regaining cardiovascular stability after marked dehydration.

Skeletal muscle participation in whole body dehydration could have undesirable metabolic consequences. Haussinger and co-workers have found in the liver of rats that water deficit in this tissue causes reduced glucose transport and increased glycogenolysis. Based on this animal data it could be hypothesized that muscle water deficit by itself could increase glycogen use. Experiments in humans also point in that direction, since rehydration during exercise reduces in muscle water content when body weight decreased 5.8% by dehydration. After exercise, hydrostatic pressure rapidly declines to resting values allowing muscle water to shift to other fluid spaces and replenish their fluid losses. We have recently shown that whole body dehydration (4.2%) reduces muscle water content when 1 h of recovery is allowed to balance between body fluids. In fact, the reduction in muscle water content coincided with the recovery of plasma fluid suggesting the predominance of regaining cardiovascular stability after marked dehydration.

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Back in 1930’s, experiments in the liver of rabbits led to suggest that there was a relationship between water and glycogen storage in this tissue. Confirming this relationship in rat myotubes, glycogen synthesis is reduced when cell water is reduced by exposure to a hyperosmotic media. Furthermore, glycogen synthesis is increased after swelling induced by exposure to hyposmotic media. In horses, fluid ingestion after exercise affects glycogen restoration. Weller and co-workers found that glycogen recovery is accelerated in Standardbred horses after depleting-dehydrating exercise when an amount of water and electrolytes similar to the volume and composition of sweat lost during exercise was administered. In contrast in humans, Neuffer and co-workers were first to find that water deprivation does not prevent normal muscle glycogen resynthesis. We partially corroborate their findings, although we found a nearly significant reduction for glycogen stored when subjects remained dehydrated (P = 0.15). One important difference between our studies is that Neufers' recovery period was 15 hours while we studied the 4 hours after exercise. We studied the 4 h post-exercise period because it is the period with the higher glycogen resynthesis. It is possible that muscle dehydration affects glycogen resynthesis in the short but not in the long term.

Finally, the effect of training in muscle water content is another area of interest. Aerobic training expands intravascular water after a few sessions purportedly to allow better cardiac function and sweat gland fluid supply. It is unclear if muscle water is also expanded with aerobic training. Data in old women supports a water expansion effect after 3 months of aerobic training. However, this effect has not been confirmed in a subsequent study with
young and old men. Furthermore, competitive runners display lower muscle water content when compared with recreational runners. However, it is unclear if the repeated deficits in body water incurred during training may be behind these responses. In summary, researchers are currently unveiling the consequences of muscle dehydration on metabolism and muscle function.

Intensity aerobic performance. Those studies demonstrate that hypohydration does not alter aerobic performance in cold conditions; often impairs aerobic performance in temperate conditions, but consistently impairs aerobic performance in warm-to-hot conditions. Hypohydration begins to consistently impair submaximal aerobic performance when skin temperatures exceed 27°C, and even warmer skin exacerbated the impaired aerobic performance (additional -1.5 % impairment or each 1°C skin temperature elevation above 27°C). Elevated skin temperatures are associated with increased skin blood flow / volume and cardiovascular strain.

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