Water: The fountain of strength

See related article: Farhat, F., Grosset, J. F., Canon, F. 2018. Water deprivation decreases strength in fast twitch muscle in contrast to slow twitch muscle in rat. *Acta Physiol* 223, e13072.

Water is indispensable for life. It is the medium in the cell required for biochemical reactions, a main source of evaporative cooling and a solute for waste products in our urine. Water is also the main component of blood that is crucial for the transport of oxygen, hormones and substrates to, and removal of heat and metabolites from, body cells. In this issue, Farhat et al[1] observed that water deprivation for 96 hours (4 days) in rats caused a decrease in strength and fibre size of their fast-twitch extensor digitorum longus (EDL), but remarkably an increased strength and unaltered fibre size of their slow-twitch soleus muscles. In this carefully conducted study, the authors suggested a role for aquaporin 4 (AQP4), a water channel that helps the influx of water during times of hyperosmotic stress.[2] Surprisingly, however, the protein content of AQP4 was higher in the fast- than slow-twitch muscle before dehydration. Perhaps even more astounding is that during water deprivation, the AQP4 was reduced in the EDL, at a time when water influx is needed the most. The study thus comes with intriguing data that are counterintuitive and future work will be necessary to explain the differential regulation of dehydration across skeletal muscle fibre types.

Given the importance of hydration for skeletal muscle function and temperature regulation, one can imagine that desert animals are faced with an enormous challenge, particularly when the limited, or even absence of, water is combined with high environmental temperatures.[3] Indeed, the Kangaroo rat, *Dipodymos merriani*, living in the deserts of North America, often lives solely on water derived from the metabolic break down of dry seeds, supplemented by feeding on insects (that contain 70% water) and green succulent plants (90% water). To preserve water, desert animals produce highly concentrated urine and dry faeces and do not sweat.[3] Although these are suitable adaptations to prevent water loss, body temperature is hard to maintain and may even reach lethal levels during exercise.

Even man, who do sweat, can succumb to dehydration and overheating when performing prolonged intense exercise at high temperature, emphasizing the need for adequate hydration. Besides problems with temperature regulation, dehydration will also increase blood viscosity that will increase the work of the heart. Together with the reduced force generating capacity of particularly fast muscles, as observed by Farhat et al,[1] this will undoubtedly reduce both power and endurance performance of athletes.

Interestingly, the force in the slow-twitch soleus was increased, but that in the fast-twitch EDL reduced after water deprivation,[1] despite a similar amount of protein loss. There may be, however, a qualitative difference in dehydration-induced protein loss between the soleus and EDL. Where the proportion of myofibrillar protein to total protein remained the same in the EDL (46%—suggesting equal loss of non-myofibrillar and myofibrillar proteins), the increase in this proportion from 32% to 41% in the soleus must have been due to preferential dehydration-induced loss of non-myofibrillar proteins.[1] As the soleus has a higher mitochondrial volume density than the EDL, we suggest that the increased proportion of myofibrillar proteins in the soleus is the result of a preferential dehydration-induced loss of mitochondrial proteins. If so, this would impair muscle endurance performance, despite a potentially increased force generating capacity, something in fact observed in dehydrated muscle of volunteers.[4]

If there is indeed a preferential dehydration-induced loss of mitochondrial proteins, it will have a significant impact on muscle energy metabolism. Metabolic adaptations to dehydration have, however, been poorly described. Rapid breakdown of proteins and, in particular, glycogen[5] increases the osmolyte concentration that minimizes water loss from the cell. If anything, however, the larger amount of glycogen in fast- than slow-twitch muscles provides a larger source of osmolytes and hence a better potential to prevent an excessive loss of water from fast- than slow-twitch muscle. Maybe the hypertonic-induced breakdown of the mitochondrial network[6] in the soleus leads to even more osmolytes than the controlled breakdown of proteins and glycogen in the EDL, thereby paradoxically protecting the soleus more than the EDL from dehydration-induced loss of strength. Clearly, future research will be needed to further understand the metabolic effects of dehydration.

Dehydration may have a particularly severe impact on people who already suffer from muscle weakness, such as elderly people in care homes. In fact, cellular dehydration is associated with a greater prevalence of sarcopenia in elderly people[7] and muscle protein wasting in extremely ill patients.[2] Observations on voluntary dehydration in athletes competing in weight classes suggest that dehydration-

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induced muscle damage[8] may be another factor that triggers muscle weakness. This relationship is rarely considered, and the work by Farhat et al[1] emphasizes the importance of adequate hydration for the maintenance of muscle mass and function. Future research will undoubtedly shed light into the molecular effects of cellular dehydration and the consequences for skeletal muscle function in older people and chronically ill patients.

CONFLICT OF INTEREST

We have no conflict of interests.

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