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EDITORIAL

Hydration, dehydration, underhydration, optimal hydration: are we barking up the wrong tree?

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Dear Editor,

According to the Medical Subject Headings of the US National library of medicine "dehydration is the condition that results from excessive loss of water from a living organism" [1]. Even though dehydration describes the state of body water deficit, some scientists have suggested that dehydration refers to the process of losing water, while hypohydration is the state of water deficit, and rehydration is the process of gaining water from a hypohydrated state towards euhydration [2].

To define dehydration or hypohydration in a laboratory setting, scientists have been using acute changes of body weight as the gold standard [3]. For instance, if someone weighs 70 kg in a euhydrated state, the acute loss of − 1.4 kg is equivalent to dehydration of − 2% of body weight (− 1.4 kg/70 kg × 100%). Unfortunately, outside of laboratories where experimentally-induced dehydration is controlled, we rarely have a recent baseline euhydrated body weight to be able to accurately examine the presence and the degree of water deficit. For this reason, different blood, urine, and clinical biomarkers have been used to assess hydration status [4].

The majority of research on water homeostasis and its effects on the human body has focused on how water deficit impacts exercise performance, mainly in hot environments [5]. Edward Adolf in his classic work "Physiology of Man in the Desert" was one of the first to study the effect of water intake on thermoregulation and performance [6]. He also introduced the term voluntary dehydration when he observed that during "rapid sweating", humans do not drink enough to maintain body water. He concluded that: "…when he is active and needs much water his thirst sensations are inadequate". During the last 30 years we have learned that even a mild degree of dehydration (< 2% of body weight) can impair exercise performance and increase heat strain, especially in the heat [5, 7]. The degree of exercise-induced dehydration often ranges between 2 and 5% of body weight and it is accompanied by elevated plasma osmolality, decreased plasma volume, and increased urinary biomarkers (i.e. urine osmolality) [5]. Influenced by this observation and based on the mathematical symmetric property stating that if A = B, then B = A, we have mistakenly assumed that the backward association is also true. Thus, if exercise-induced dehydration leads to increased urine biomarkers, then elevated urinary biomarkers should correspond with water deficit and dehydration. So, when we read data indicating that a majority of children, adults, and athletes have elevated levels of urinary osmolality or specific gravity we mistakenly conclude that a large portion of the population is dehydrated [8–11]. Furthermore, when we read data indicating that a majority of people across the world do not meet the dietary guidelines for water intake we also conclude that most people are dehydrated. Is it possible that people with free access to water when they do not meet the water intake guidelines or when they have elevated urinary biomarkers are dehydrated? Probably not.

Let's examine the data from the National Health and Nutrition Examination Survey (NHANES) in the US. If we compare the 10th (1694 mL/day) and the 90th (7934 mL/day) percentile of water intake distribution in the US we will notice that they have nearly identical plasma osmolality (279 and 280 mmol/kg, respectively) [12]. Similarly, people who chronically consume either low (low-drinkers) or high (high-drinkers) amounts of water have similar plasma osmolality, but low-drinkers have greater vasopressin [13]. In 2015, Johnson and his colleagues published a study that identified low- and high-drinkers through an initial screening

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[14]. Then, they converted the low-drinkers to high-drinkers by increasing their water intake and the high-drinkers to low-drinkers by decreasing their water intake. Interestingly, body weight and plasma osmolality remained unaffected by the change in water intake in both groups. Nevertheless, urine volume and urine osmolality changed remarkably in both groups due to the intervention. These data suggest that drinking more or less water impacts the levels of vasopressin (AVP) as well as the volume and concentration of urinary output but does not affect total body water. How is this possible? Our water homeostasis is mainly regulated by the hormone vasopressin and thirst. AVP is very quickly activated by a slight water deficit and induces nearly maximal renal water conservation in low plasma concentrations well before activation of thirst [15]. Moreover, during dehydration, thirst sensation can be quickly terminated prior to full water restoration (rehydration) as a response to the act of swallowing via activation of the oropharyngeal receptors' [16]. In that case, we could be in a state of elevated AVP even in the absence of thirst. The important question: is low water intake, in the absence of dehydration, associated with negative health or performance outcomes.

Epidemiological data indicate that low water intake or elevated hydration biomarkers are associated with several adverse health outcomes [17]. Low water intake and elevated AVP, assessed by its surrogate marker copeptin, is linked to chronic kidney disease and diabetes [18, 19]. Also, increased water intake, even in patients with third stage chronic kidney disease, is well tolerated and suppresses high copeptin levels [20]. Drinking more water has also been found to dramatically decrease the recurrence of urinary tract infections [21], and to improve glucose regulation in people with low water intake or elevated copeptin [22]. Additionally, children with very concentrated urine (an indication of high AVP) can improve cognitive [23] and endurance performance [24] by drinking more water. These data suggest that low water intake is associated with negative health and performance outcomes.

Therefore, low water intake or chronically elevated urinary biomarkers does not mean by default dehydration, since total body water is maintained, and blood osmolality is typically unaffected. In the literature, scientists have referred to the hydration state of elevated urinary hydration biomarkers as either mild-dehydration, insufficient hydration, suboptimal hydration, or pre-dehydration [11, 25, 26]. Instead, I propose the term underhydration that may better capture the nature of this phenomenon, encompassing low water intake (consuming less than the reference values), in the absence of total body water deficit, thirst or elevated plasma osmolality, while the water homeostatic mechanism has been activated as indicated by elevated vasopressin and urine biomarkers (Fig. 1). It is time to differentiate the term dehydration from underhydration and refrain from using dehydration when someone only has elevated urine osmolality or does not meet the dietary water intake reference values.

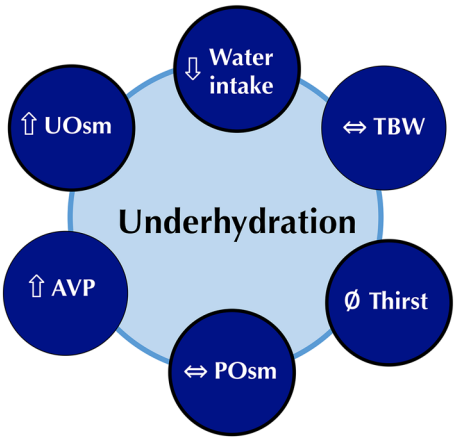

Fig. 1 Characteristics of state of underhydration. *TB*W total body water, *POsm* plasma osmolality, *AVP* arginine vasopressin; UOsm, urine osmolality

Of course, the topic of hydration and health is new and under researched [27]. At this point, we probably have more questions than answers and theories on potential mechanisms associating low water intake with various unexplored pathologies, including cancer and longevity [28, 29]. However, it is time to concentrate our efforts on the health implications of being a low-drinker rather than examining the acute effects of dehydration (water deficit). We need large scale studies and randomized control trials to investigate how increased water intake impacts health and well-being.

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Compliance with ethical standards

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