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Adverse functional outcomes associated with body water deficits

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Humans have intricate regulatory mechanisms to sustain hydration, however, body water deficits can occur. Acute body water deficits are common due to high rates of fluid losses and insufficient intake. Chronic water deficits are rare and often associated with pathologies and/or medications. This lecture examines the adverse functional outcomes possibly associated with body water deficits. Functional outcomes are measurable changes in physiological strain, health/wellness and performance. Hydration physiology and evidence regarding five possible adverse functional outcomes will be reviewed: 1) cardiovascular strain, 2) thermoregulation, 3) aerobic performance, 4) mood/cognition, and 5) diseases.

Hydration Fundamentals: Total Body Water (~76 % of Lean Body) is tightly regulated with normal daily variation of <1 % of body mass. Body water deficits >2 % of body mass are defined as hypohydration and can be either hyperosmotic (from under-drinking and high sweat loss) or iso-osmotic (diuresis from cold, hypoxia or loop diuretics). Hyperosmotic hypovolemia elicits a plasma volume reduction proportionate to the water deficit; whereas, iso-osmotic hypovolemia results in greater plasma loss for a given water deficit due to reduced osmotic gradient to draw water from the intra-cellular to extra-cellular space. Both plasma hyperosmolality and plasma volume reduction stimulate arginine vasopressin (AVP) release that acts on the kidneys to reabsorb water. Plasma hyperosmolality, plasma volume reduction and elevated AVP have all been postulated to contribute to adverse functional outcomes from acute and chronic body water deficits. In addition, since daily water losses vary greatly between persons due to physical activity, environmental exposure, health and genetics there is no one fluid consumption rate that ensures full hydration for all.

Cardiovascular Strain: Body water deficits reduce plasma volume and thus cardiac filling and stroke volume, making it more difficult to maintain blood pressure during exposure to orthostatic, environmental and exercise stressors. Hypohydration reduces cerebral blood flow during orthostatic stress causing fainting. During rest and aerobic exercise, hypohydration will increase heart rate in proportion to the magnitude of water deficit. During aerobic exercise with moderate heat stress, hypohydration decreases cardiac output (compared to performing the exercise task when euhydrated). During aerobic exercise with severe heat stress, hypohydration can reduce skeletal muscle blood flow. Likewise, the cardiovascular strain imposed by hypohydration increases the risk of heat exhaustion.

Thermoregulation: Body water deficits increase core temperature during exercise at a given metabolic rate in temperate and warm-hot conditions. As the magnitude of water deficit increases, there is a concomitant graded elevation of core temperature, which is accentuated in warm-hot conditions. The elevated core temperature results from a decrease in heat loss from both sweating and skin blood flow responses (elevated threshold temperatures and decreased sensitivity) for a given core temperature. Both plasma hyperosmolality and hypovolemia contribute (with plasma hyperosmolality contributing more consistently) to the regulated increase in core temperature. In addition, body water deficits enhance the fever response that is likely due to increased production of pyrogenic cytokines.

Aerobic Performance: Aerobic performance can be quantified as maximal intensity (e.g., maximal aerobic power) or submaximal intensity (time-trial or time-to exhaustion). Maximal intensity studies are relatively few, but demonstrate that hypohydration generally impairs VO2max. The earliest studies of hypohydration and submaximal intensity exercise capacity in the 1940's found a reduced work capacity when fluid replacement was restricted in warm-hot environments. Subsequently, many laboratory studies have examined hypohydration and submaximal 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 27ºC, and even warmer skin exacerbated the impaired aerobic performance (additional -1.5 % impairment or each 1ºC skin temperature elevation above 27ºC). Elevated skin temperatures are associated with increased skin blood flow / volume and cardiovascular strain.

Mood/Cognition: Mood degrades with hypohydration in proportion to the body water deficit. Early investigators reported strong associations between hypohydration level and impaired cognitive performance; however, many subsequent studies have not replicated those initial findings and suggest a more complex scenario. Recently, studies have demonstrated that body water deficits can alter brain structure and function (increased neural activity and recruitment) as well as impair cognitive-motor tasks. Thus, body water deficits are more likely to impair cognitive performance during or shortly after stressful psychological-motor tasks or in compromised (e.g., elderly, dementia) populations.

Diseases: Possible relationships between hydration and disease morbidities are difficult to establish due to an absence of a single valid "gold standard" biomarker of body water deficits. As a result studies of possible chronic dehydration and disease morbidities must rely on imprecise hydration biomarkers. Recently, copeptin a biomarker for arginine vasopressin (AVP) has been suggested as a hydration biomarker; however, several other factors (besides hydration status) can elevate these hormones. Regardless, interesting data are suggesting possible associations between elevated copeptin (thus AVP) and/or osmotic stress with diabetes/metabolic syndrome, renal and heart diseases in vulnerable populations.

Conclusion: The following hypohydration-mediated adverse functional outcomes are currently supported by the scientific literature: 1) increased cardiovascular strain and reduced orthostatic tolerance; 2) increased core temperature during physical exercise and fever; 3) impaired aerobic performance in warm-hot conditions with an increased risk of heat exhaustion; 4) degraded mood, with likely impairments of cognitive-motor performance.

Key words: *aerobic performance, body water deficits, cognition, dehydration, health, hypohydration, mood, orthostatic tolerance*.

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