

## Role of Hydration Status on the Pathogenesis and Management of Obesity

Sara H. ALRosan<sup>1\*</sup> , Hayder A. AL-Domi<sup>1</sup>  and Mohammad J. Alkhatatbeh<sup>2</sup> 

<sup>1</sup>Department of Nutrition and Food Technology, School of Agriculture, The University of Jordan, Amman 11942, Jordan.

<sup>2</sup>Department of Clinical Pharmacy, Faculty of Pharmacy, Jordan University of Science and Technology, Irbid 22110, Jordan.

Received on 5/6/2024 and Accepted for Publication on 11/12/2025.

### ABSTRACT

Emerging evidence suggests a strong association between hydration status and the etiopathogenesis of obesity. This critical review highlights the possible role of hydration status in preventing, or at least ameliorating, the prevalence of obesity, a chronic, low-grade proinflammatory disease. PubMed, Google Scholar, ScienceDirect, and World Health Organization (WHO) databases were searched for literature published in English, using the following keywords search terms 'water', 'water intake', 'hypohydration' or 'hydration' and 'energy intake', 'obesity', 'appetite', 'satiety', 'food consumption', and 'Body Mass Index (BMI)' for the period between 2003 and 2023. The current review included data from clinical trials, cross-sectional studies, cohort studies, published reviews, and case-control studies investigating the role of the hydration status in the development of obesity as well as prevention mechanisms. Rehydration by a moderate increase of daily water intake could be an effective, costless lifestyle modification for individuals at risk of developing obesity. Hypohydration could be one of the most important predisposing risk factors in both obesity pathogenesis and management, and its comorbidities via several mechanisms, mainly fat metabolism, thermogenesis, energy expenditure, food consumption, energy intake, hormonal balance, appetite regulation, and satiety regulation. Appropriate hydration status could help prevent or at least delay weight gain. Further clinical trials are required to verify the proposed mechanisms. The figure was created with BioRender.com.

**Keywords:** Hypohydration, Hydration, Water, Obesity, Appetite, Satiety.

### INTRODUCTION

Obesity is a growing global health challenge resulting in serious non-communicable diseases (NCDs), including, but not limited to, insulin resistance (IR), type 2 diabetes mellitus (T2DM), cardiovascular diseases (CVDs), and certain types of cancer (Al Haj Ahmad & Al-Domi, 2017). Obesity is associated with a low-grade subclinical pro-inflammatory state leading to altered adipocytokine secretions, including but not limited to elevated serum levels of bona fide adipokines, namely:

leptin, (Adamska-Patrano *et al.*, 2019) tumor necrosis factor-alpha (Freitas *et al.*, 2018), adiponin (Vasilenko *et al.*, 2017), and decreased levels of adiponectin (Szabová *et al.*, 2012).

Obesity is typically a consequence of the chronic imbalance between energy intake and energy expenditure (Adamska-Patrano *et al.*, 2019), which leads to chronic failure in body weight control mechanisms. Thus, to combat obesity, strategies are required to help individuals reduce energy intake and/or increase energy expenditure (Blundell, 2018). There are numerous ways to decrease energy intake, including surgical techniques and

\* Corresponding author. E-mail : [saraalrosan@hotmail.com](mailto:saraalrosan@hotmail.com)



medications, which promote satiety; yet these procedures could have adverse side effects ranging from feelings of nausea to an increase in mortality risk (Bruschi Kelles *et al.*, 2014).

A few reports suggested that water intake could be a potential goal for obesity prevention and management (Maffeis *et al.*, 2016; Rosinger *et al.*, 2016; Padrão *et al.*, 2017). Decreased food intake due to increased water intake has led to body weight loss (Tate *et al.*, 2012; Corney *et al.*, 2016; Fresán *et al.*, 2016).

Increased evidence indicated a role for hypohydration status in the development of obesity and obesity-related cardiometabolic complications (Chang *et al.*, 2016; Maffeis *et al.*, 2016; Rosinger *et al.*, 2016; Padrão *et al.*, 2017). Hypohydration is widely observed among individuals diagnosed with obesity, which indicates that obese individuals could have a higher water requirement (Chang *et al.*, 2016). In addition, obesity is typically associated with increased food intake that is accompanied by relatively low water intake, leading to hypohydration (Rosinger *et al.*, 2016).

Hydration status is defined as a parameter indicative of total fluid composition (Puga *et al.*, 2019). Having normal total body water is known as euhydration (Cheuvront, Kavouras & Kenefick, 2016), whereas hypohydration is a body water deficit greater than normal daily fluctuation and is clinically defined as body water losses of >2% of body mass (Cheuvront & Kenefick, 2014; Sawka *et al.*, 2015). On the other hand, dehydration is a deficit in total body water (Cheuvront & Kenefick, 2014).

Hydration status could affect the natural history of the development of obesity via several mechanisms. Hydration status could alter metabolism (Keller *et al.*, 2003), food consumption (Corney *et al.*, 2016; Pérez-Luco *et al.*, 2019), energy intake (Corney, Horina, *et al.*, 2015; Corney, Sunderland, *et al.*, 2015; Carroll *et al.*, 2019), hormonal balance (Larnkjaer *et al.*, 2015; Carroll *et al.*, 2019), appetite and satiety regulation (Corney, Sunderland, *et al.*, 2015). It is worth mentioning that hypohydration and dehydration have similar mechanisms in the development of obesity and obesity-related

cardiometabolic complications (Thornton, 2016). Hence, the objective of this critical review was to highlight the possible role of hydration status in preventing, or at least ameliorating, obesity as a chronic low-grade proinflammatory disease.

## Method

The PubMed database, google scholar, ScienceDirect, and WHO database were searched for literature published in English for the period between 2003 to 2023, using the following keywords search terms 'drinking water', 'water', 'water intake', 'hypohydration' or 'hydration' and 'energy intake', 'obesity', 'appetite', 'satiety', 'food consumption', and 'Body Mass Index (BMI)'.

## Selection of Published Studies:

The current critical review included data collected from clinical trials, cross-sectional, cohort studies, published reviews, commentaries, letters, and case-control studies investigating hydration in relation to obesity development and prevention mechanisms. Full-text articles of clinical and preclinical studies on humans and animals that have been published within 25 years were selected and included in this review. Additional sources were searched, including the reference lists of recent relevant reports and reviews and the reference list of the selected original papers to identify further eligible studies. Based on the research inclusion criteria, 65 appropriate articles were selected from around 200 articles that were examined. Selected articles were categorized in main headings "Factors Affecting the Hydration Status", "Effect of hypohydration on Obesity", and "Mechanisms of Obesity Development in Relation to Hydration Status". After the final screening, 16 articles were excluded for specific reasons, including insufficient information (n=4), were related to dehydration in clinical practice related to kidney dysfunction (n=5), were studying dehydration effect on certain types of sports (n=2), and were non-mechanistic reviews (n=5). The exclusion criteria were non-English language studies, conference proceedings, and studies published before 2003.

### Data Collection

Findings were extracted from articles by one researcher and independently checked for accuracy by two researchers. Data were summarized in one table titled “Studies Investigated Hydration Status in Relation to Obesity” and were divided into a group of columns entitled as follows: Author, Year, Study Design, Study Sample, Age group, and Findings. Also, a figure titled “Hypothesized mechanisms linking hydration to obesity”.

### Obesity: An Overview

Obesity has been identified as a costly global pandemic chronic disease. The prevalence of which has risen extremely in the last decade. In 2022, 2.5 billion adults aged 18 years and older were either overweight or obese, a minimum of 890 million of them were diagnosed with obesity (World Health Organization, 2024). The complexity of the etiopathogenesis of obesity represents a major challenge in the disease prevention and management (World Health Organization, 2021).

Globalization of “Westernized” nutrition behaviors has led to the reduction of poverty and food insecurity (Lafontan *et al.*, 2015) even though poverty remains high worldwide (World Health Organization, 2022). These altered nutrition behaviors are also coupled with a negative impact on health and food consumption behaviors, including increased consumption of sugar-sweetened beverages, increased portion sizes, calorie-dense food, sodium intake, and low-cost high-energy food and drinks of poor nutritional quality, besides low water intake that contributes to obesity development (Lafontan *et al.*, 2015). This multifactorial pandemic is characterized by an alteration in body composition, which is emphasized by an increase in fat mass as compared to fat-free mass, and a high ratio of extracellular fluids (ECF)/intracellular fluids (ICF) (Stookey *et al.*, 2007).

Obesity is a complex, heterogeneous, multifaceted disease developed predominantly from a polygenic multifactorial trait (Tan & Mitra, 2020). It develops from a chronic failure of complex interrelated homeostasis mechanisms that firmly control body-weight due to a complex interplay between both endogenous risk factors

including genetic predisposition, and exogenous risk factors mainly passive overconsumption of foods and the universal sedentary state described as a “sloth syndrome”, which act by means of mediators of energy input and energy output (Al-Domi, 2015). It could promote serious obesity-related cardiometabolic complications (Al Haj Ahmad & Al-Domi, 2017; Al-Domi & Al Haj Ahmad, 2017) that could alter water requirements, hydration, and metabolic processes including hypertension, kidney function, insulin resistance (IR), T2DM, CVDs, and certain types of cancer (Rosinger *et al.*, 2016). It is worth noting that obesity is a sterile chronic low-grade systemic pro-inflammatory state that leads to increased serum levels of the bona fide adipokines in the pro-inflammatory state (Roth *et al.*, 2011). This could be affected by hydration status, including ghrelin, and leptin, which stimulate and inhibit fat intake, respectively (Beck & Max, 2007; Mathai *et al.*, 2008). The two peptides have been found to be affected by the same hormones that are involved in the regulation of hydration status, including angiotensin-converting enzyme (ACE) and vasopressin (Beck & Max, 2007; Mathai *et al.*, 2008).

### Factors Affecting the Hydration Status

Human water requirements vary depending on age, gender, physical activity levels, ambient temperature, humidity, clothing, cultural dietary patterns, body composition, body size, and pathophysiological situations (Sawka *et al.*, 2005; Agostoni *et al.*, 2010; Goodman *et al.*, 2013; Rosinger & Tanner, 2015). These contributing factors have a significant role in water drinking, as different populations have different levels of water intake (Sawka *et al.*, 2005; Goodman *et al.*, 2013). Water intake varied widely among different populations; a part of some populations have been found to drink less than four cups of water daily (Goodman *et al.*, 2013) while in other populations, water needs could reach up to about 6 L for active adults living in a warm environment (Sawka *et al.*, 2005).

### Effect of hypohydration on Obesity

A few reports, including randomized controlled trials and epidemiological studies, investigated the correlation between hypohydration and obesity. Table 1 summarizes studies that investigated the association between hydration status and obesity according to age groups. A few reports indicated that increasing water intake could be a potential goal for obesity prevention and treatment (Maffeis *et al.*, 2016; Rosinger *et al.*, 2016; Padrão *et al.*, 2017); yet other reports linked childhood obesity with greater hydration (Haroun *et al.*, 2005). Obese children have been found to have lower hydration status than normal weight peers; in addition, 32% of obese children vs 20% of non-obese peers had negative free water reserve (Haroun *et al.*, 2005). BMI z-score and water intake from fluids correlated with free water reserve (Haroun *et al.*, 2005).

Stookey *et al.* (2014) examined the effect of hydration on weight status in pediatrics, and they found that participants with urine osmolality below 500 mmol/kg were complaining of significant weight loss (Stookey *et al.*, 2014). On the other hand, an observational matched case-control study found that obese children had greater hydration and reduced density of fat-free mass (FFM) (Haroun *et al.*, 2005).

A few reports have examined the relationship between obesity and hydration status among adults (18-64 years) (Stookey *et al.*, 2007; Vij & Joshi, 2013; Chang *et al.*, 2016; Rosinger *et al.*, 2016) and elderly ( $\geq 65$  years) (Marra *et al.*, 2016; Padrão *et al.*, 2017). Chang *et al.* (2016) combined data from the National Health and Nutrition Examination Survey (NHANES) 2009-2010 and 2011-2012 surveys (Chang *et al.*, 2016). The study found that hydration status was significantly associated with BMI. On average, inadequately hydrated adults had a mean BMI of 1.32 kg/m<sup>2</sup> greater than that of adequately hydrated adults (Chang *et al.*, 2016). In addition, inadequately hydrated adults had higher odds of being obese (OR = 1.59) compared with hydrated adults (Chang *et al.*, 2016). Furthermore, NHANES cross-sectional data

(2009-2012) were analyzed in males and non-pregnant females aged  $\geq 20$  years (Rosinger *et al.*, 2016). The cross-sectional study reported that the mean total water intake was ~3.1 L (Rosinger *et al.*, 2016). In addition, obese adult females significantly consumed more water (238.5 mL) as compared to that consumed by underweight or normal weight females (Rosinger *et al.*, 2016). Also, urine osmolality and the prevalence of hypohydration were increased with higher weight status (Rosinger *et al.*, 2016).

Findings of a report demonstrated that osmolality was increased with increasing BMI categories in both women and men (Padrão *et al.*, 2017). For men, being in the third osmolality tertile (highest; 530 mOsm/kg) was only associated with a higher risk of being obese (OR = 1.97), while no significant association between being in the second or the third (highest) osmolality tertiles and overweight or obesity in women were observed after adjustment of confounding variables (Padrão *et al.*, 2017). Stookey *et al.* (2007) have investigated the relationship between weight status and plasma tonicity and found that overweight and obese individuals were significantly more hypertonic than their normal weight counterparts (Stookey *et al.*, 2007).

Marra *et al.* (2016) undertook a cross-sectional analysis of baseline data on long-term care (LTC) elderly residents, and they found that inadequate total water intake and dehydration were prevalent among all BMI categories in LTC residents (Marra *et al.*, 2016). Nevertheless, total water intake was found to increase with increasing BMI, with significantly higher total water intake in the overweight and obese participants compared to the underweight participants (Marra *et al.*, 2016).

The limited number of epidemiological surveys and clinical trials on hydration status and body weight cannot ascertain a causal relationship with obesity. Current studies are conducted either on children or geriatrics, and results are scarce. So, further randomized clinical trials on young adults are recommended to establish such a relationship.

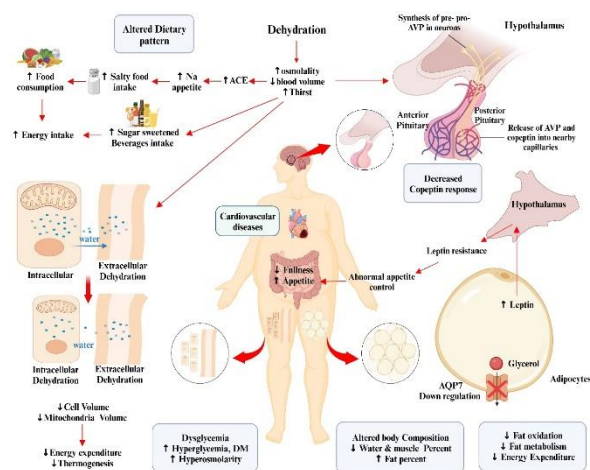
**Table 1:** Summary of studies investigating hydration status in relation to obesity

Author, Year	Study Design	Study Sample	Age group	Findings
<b>Children</b>				
Maffeis <i>et al.</i> , (2016)	Case-control study	86 obese (45 male, 41 Female) and 89 normal weight (40 male, -49 female) children	7–11 yrs.	Obese children have a lower hydration status than their normal weight peers.
Stookey <i>et al.</i> (2014)	Randomized intervention	25 children	9–12 yrs.	Participants with urine osmolality below 500 mmol/kg were significantly associated with weight loss
Haroun <i>et al.</i> (2005)	Observational matched Case-control study	28 obese children (13 boys, 15 girls) and 22 nonobese children (10 boys, 12 girls).	7–14 yrs.	The study found that according to the three-component model (divides weight into fat mass, water, and fat-free dry tissue) and four-component model (which divides weight into fat mass, water, protein, and mineral), obese children had greater hydration and reduced density of FFM.
<b>Adults and the Elderly</b>				
Padrão <i>et al.</i> , (2017)		1315 (563 male-755 female)	65–94 yrs.	- Osmolality was increased with BMI categories, both in women and men - The third tertile (highest) of osmolality was associated with a higher risk of being obese.
Chang <i>et al.</i> (2016)	Data from the NHANES 2009-2010 and 2011-2012 surveys	9,528	18–64 yrs.	Hydration status was significantly associated with BMI and other variables.
Rosinger <i>et al.</i> (2016)	NHANES cross-sectional data (2009-2012)	9601 (4854 male-4747 female)	≥20 yrs.	Adults who were obese consumed 238.5 mL more water, on average, or ~1 cup, than were consumed by adults who were underweight or of normal weight. Urine osmolality and the prevalence of hypohydration all increased with higher weight status.
Marra <i>et al.</i> , (2016)	Cross-sectional analysis of baseline data on long-term care residents' elderly	247 (53 male-194 female)	82.9 ± 11.3 yrs.	Inadequate total water intake and dehydration were prevalent in LTC residents over all BMI categories
Vij and Joshi, (2013)	Interventional	50 Overweight girls (BMI 25-29.9 kg/m <sup>2</sup> )	18–23 yrs.	Drinking 1.5 L of water above usual water intake resulted in a significant reduction in body weight, body mass index, and body composition scores
Stookey <i>et al.</i> , (2007)	NHANES III Cross-sectional data	1285	40–59 yrs.	Overweight and obese individuals in the study sample were significantly more hypertonic than their normal weight counterparts, independent of plasma glucose level.

## Mechanisms of Obesity Development about Hydration Status

### Food Consumption, Energy Intake, and Hydration Interplay

Studies on human have linked hydration status and water intake to food consumption (Corney *et al.*, 2016; Pérez-Luco *et al.*, 2019), energy intake (Corney, Horina, *et al.*, 2015; Corney, Sunderland, *et al.*, 2015; Carroll *et al.*, 2019), metabolism (Keller *et al.*, 2003), fullness, appetite (Corney, Sunderland, *et al.*, 2015), and satiety that could play a key role in obesity development. Figure 1 shows hypothesized mechanisms linking hydration to obesity. A number of epidemiological studies found that plain water intake was associated with reduced daily total energy intake, sugar intake, saturated fat intake, and sodium intake (An, 2016), and lower total energy from beverages (Nissensohn *et al.*, 2016), (Figure 1).



**Figure 1:** Hypothesized mechanisms linking hydration to obesity.

ACE: Angiotensin-converting Enzyme, AQP: Aquaporin, AVP: Arginine Vasopressin, CVD: Cardiovascular diseases, DM: Diabetes mellitus, Na: Sodium. Created with BioRender.com.

Hypohydration is widely spread among obese individuals. Several mechanisms have been proposed to clarify the hydration status effect in obesity development

and obesity outcomes. The development of hypohydration results in increased osmolality and decreased blood volume. Extracellular dehydration results in water movement from the intracellular to extracellular compartment, leading to intracellular dehydration, which results in decreased cell volume and decreased mitochondrial volume, in addition to altered sodium appetite. Mitochondrial dysfunctions decrease energy expenditure and decrease thermogenesis, resulting in a decrease in fat metabolism. Obesity was found to be associated with lower copeptin response. The increase in appetite toward sodium increases salty food intake, which results in increased food consumption and increased energy intake. Also, hypohydration affects the hypothalamus control of thirst and appetite, which could result in abnormal appetite control and altered leptin action. Higher intake of sugar-sweetened beverages instead of water due to thirst leads to higher energy intake. Hypohydration in adipose tissue could be associated with an increase in leptin secretion, also downregulation of aquaporin mainly 7, which might reduce glycerol release from adipocytes which impairing fat oxidation and decreasing energy expenditure. Increased food consumption and energy intake, and decreased fat metabolism, fat oxidation, and energy expenditure result in fat accumulation and obesity development. Obesity results in the development of cardiovascular diseases, Dysglycemia, altered body composition, and altered dietary patterns, which furthermore could affect hydration status.

On the other hand, a study on Zucker rats found that meal size in obese Zucker rats was 200% larger than that in lean rats during normal hydration, while after dehydration, meal size was 300% larger in obese than in lean Zucker rats, despite dehydration-induced anorexia in both groups (Fetissov & Meguid, 2020). Controversy, extreme fluid restriction resulted in decreased acute food intake after exercise (Pérez-Luco *et al.*, 2019).

Plain water has been suggested as an excellent replacement for sugar-sweetened beverages since water is

a hypotonic solution and has a zero glycemic index (Tate *et al.*, 2012; Fresán *et al.*, 2016). The substitution of one serving of beer and sweetened sugar beverages with one serving of water in a four-year period was associated with a lower incidence of obesity (Fresán *et al.*, 2016). Likely, the replacement of caloric beverages with water as a weight-loss strategy resulted in an average weight reduction of 2% (Tate *et al.*, 2012). In addition, the consumption of water before a meal reduces energy intake (Corney *et al.*, 2016). Controversially, a trial found that mild hypohydration produced by inadequate fluid intake and fluid availability during eating does not influence ad-libitum energy intake of a semi-solid breakfast in healthy young males (Corney, Horina, *et al.*, 2015). For the ad-libitum breakfast subjects, they were instructed to eat until they were 'comfortably full and satisfied' (Corney, Horina, *et al.*, 2015). The abovementioned findings indicate that plain water could be an excellent replacement for sugar-sweetened beverages to avoid beverage calories, improving overall hydration, decreasing food consumption, and decreasing total energy intake, with the goal of promoting a healthy weight status.

### **Effect of Hydration Status on Metabolism**

Water has a critical role in regulating metabolic processes, including cell volume, energy expenditure, thermogenesis, fat metabolism, and hormonal regulation (Keller *et al.*, 2003; Boschmann *et al.*, 2007; Carroll *et al.*, 2019). Water is a hypotonic solution with an osmolality level of <20 mmol/kg, which is far below that of blood (280 mmol/kg) (Stookey, 2016). This allows water to move following local osmotic gradients into cells, which acutely swell cells and mitochondria, and that in turn activates organ function and metabolism, leading to greater fat oxidation (Stookey *et al.*, 2014).

Normal decrease in total body water, which most likely refers to hypohydration (Cheuvront & Kenefick, 2014; Sawka *et al.*, 2015), leads to intra- and extracellular dehydration (Zhang *et al.*, 2021). Intra and extracellular dehydration stimulate the release of hormones involved in the regulation of body fluid, including Arginine vasopressin (AVP) or vasopressin, also known as anti-

diuretic hormone (ADH), renin, and angiotensin II (AngII) (Thornton, 2016). In addition to these hormones, copeptin, which is the C-terminal part of pro-AVP, is released together with AVP during processing of the precursor peptide (Nickel *et al.*, 2012). AVP and copeptin are produced in the hypothalamus and secreted from the posterior lobe of the pituitary gland upon osmotic or hemodynamic stimuli (Nickel *et al.*, 2012).

Findings of a report suggested that hypohydration leads to a decrease in cell volume, leading to a decrease in mitochondrial volume that results in a lower mitochondrial metabolism and lower energy production (Thornton, 2011). Increased water intake resulted in inhibition of the renin–angiotensin system, producing a marked decrease in adiposity (Mathai *et al.*, 2008). This decrease in adiposity is explained by the inhibition of the renin–angiotensin system, which unblocks fat metabolizing mechanisms, or the increase in water intake enhances cell hydration and fat metabolism regulation (Thornton *et al.*, 2009). In addition, a recent study on humans found that after each dehydration condition, participants with obesity reported feeling less thirsty and had decreased copeptin response and a higher urinary sodium concentration when stressed (Chang *et al.*, 2022). However, Angiotensin II, aldosterone, and atrial and brain natriuretic peptides concentrations did not differ by adiposity group and failed to justify the altered thirst or copeptin responses in participants with obesity. Also, the role of ACE in obesity development, which was directly linked to hydration status, has been studied (Bordoni *et al.*, 2017). ACE polymorphism was found to significantly enhance the effects induced by hydration on body composition parameters (Bordoni *et al.*, 2017).

Another hypothesized mechanism of the hydration effect on obesity involved a subclass of aquaporin (AQP) water channels, named aquaglyceroporins mainly 3, 7, and 9, which can transport glycerol and possibly urea and other small solutes (Hara-Chikuma & Verkman, 2006). Marrades *et al.* (2006) suggested that downregulation of AQP7 in adipose tissue in obesity might result in reduced glycerol release from adipocytes in obese compared to lean individuals (Marrades *et al.*, 2006). These complex

metabolic alterations, which are associated with hydration status, could have a critical role in fat metabolism and accumulation, and obesity development.

### **Effect of Hydration Status on Appetite and Satiety**

Hydration status might alter appetite and satiety by several suggested mechanisms. Water has been reported to be used as a common appetite suppressant (Langdon & Dennee-Sommers, 2010). The exact mechanism of the effect of hydration status on appetite and satiety regulation is not ascertained yet. Consumption of water preload before a meal was found to increase fullness and satisfaction, and to reduce energy intake, hunger (Corney *et al.*, 2016), appetite score, body weight, BMI, and sum of skinfold thickness (Vij & Joshi, 2013).

Other investigations, including human studies (Brown *et al.*, 2019; Chang *et al.*, 2022) and animal models (Beck & Max, 2007; Mathai *et al.*, 2008) have linked several hormones related to appetite and satiety regulation with hydration status or hormones that control body fluid. Normally, leptin has anorexigenic effects on the hypothalamus, however, leptin levels were found to be higher with resistance to the actions of the hormone among obese individuals, which results in abnormal control of the appetite (Adamska-Patrano *et al.*, 2019). Findings indicated that the inhibition of ACE in animal models resulted in selective reduction in body fat mass and plasma leptin, though treated rats drank twice the amount of water as the non-treated controls (Mathai *et al.*, 2008). In the Brattleboro animal model, where the homozygous *di/di* condition does not produce any vasopressin, and can drink and excrete up to 250 ml /day (Thornton *et al.*, 2009) The growth of these animals was slower than controls, lean, and had low leptin levels (Beck & Max, 2007). A study on humans found that leptin was correlated with copeptin response in lean individuals during the hypertonic saline infusion (dehydration condition). However, the association was diminished in those with obesity (Chang *et al.*, 2022).

Recently, studies have investigated the role of leptin action versus dehydration on lateral hypothalamic area neurotensin neurons (Brown *et al.*, 2019; Kurt *et al.*,

2022). A study has identified two molecularly- and projection-specified subpopulations of lateral hypothalamic area neurotensin neurons that were activated either by leptin or dehydration, and which possibly could regulate feeding vs. drinking behavior (Brown *et al.*, 2019). Nevertheless, a recent study found that the activation of the lateral hypothalamic area neurotensin neurons projecting to the lateral preoptic area is sufficient to mediate drinking behavior but does not suppress feeding, as detected after generally activating all lateral hypothalamic area neurotensin neurons (Kurt *et al.*, 2022). The previous studies supported the hypothesis that appetite might be affected by hydration status, which may affect either leptin levels or leptin action in obese individuals (Beck & Max, 2007; Mathai *et al.*, 2008; Brown *et al.*, 2019).

In addition to the hydration status correlation with leptin and its effect on appetite, hydration status was linked to sodium appetite. Reports demonstrated that deprivation in water using the water deprivation-partial repletion protocol resulted in elevated salt appetite (De Luca *et al.*, 2002). Such a relation was established to distinguish thirst from sodium appetite (De Luca *et al.*, 2002). Although thirst is an important behavior of water intake, it is not sufficient to correct dehydration caused by restricted fluid intake or water deprivation (De Luca *et al.*, 2010). Water is adequate to refill the intracellular fluid, while sodium and water are necessary to refill the extracellular fluid (De Luca *et al.*, 2010). Sodium appetite displays ingestion of sodium salts in response to extracellular dehydration (De Luca *et al.*, 2010) Thus, sodium could be a contributing factor in obesity development (Moosavian *et al.*, 2017; Zhang *et al.*, 2018).

### **Conclusion**

Hypohydration could be one of the most serious predisposing risk factors in the development and management of obesity as well as its comorbidities. The suggested mechanisms fall into several major pathways, including metabolism, food consumption, energy intake, hormonal balance, appetite regulation, and satiety regulation. Additional clinical trials are warranted to



ascertain the proposed mechanisms.

### Fund and grant.

This work was supported by a grant from the Deanship of Academic Research at the University of Jordan, Jordan (Grant number: 2/2018-2019 and 144/2019).

## REFERENCES

- Adamska-Patrano, E., Ostrowska, L., Goscik, J., Fiedorczuk, J., Moroz, M., Kretowski, A., & Gorska, M. (2019). The differences in postprandial serum concentrations of peptides that regulate satiety/hunger and metabolism after various meal intake, in men with normal vs. excessive BMI. *Nutrients*, 11(3). <https://doi.org/10.3390/nu11030493>
- Agostoni, C. V., Bresson, J.-L., Fairweather Tait, S., Flynn, A., Golly, I., Korhonen, H., Lagiou, P., Løvik, M., Marchelli, R., & Martin, A. (2010). Scientific opinion on dietary reference values for water. *EFSA Journal*, 8(3).
- Al Haj Ahmad, R. M., & Al-Domi, H. A. (2017). Complement 3 serum levels as a pro-inflammatory biomarker for insulin resistance in obesity. *Diabetes & Metabolic Syndrome*, 11(Suppl 1), S229–S232. <https://doi.org/10.1016/j.dsx.2016.12.036>
- Al-Domi, H. (2015). Paleolithic hunter-gatherers' dietary patterns: Implications and consequences. *African Journal of Food, Agriculture, Nutrition and Development*, 15(2), 9935–9948.
- Al-Domi, H. A., & Al Haj Ahmad, R. M. (2017). Association between complement component C3 and body composition: A possible obesity inflammatory biomarker for insulin resistance. *Asia Pacific Journal of Clinical Nutrition*, 26(6), 1082–1087. <https://doi.org/10.6133/apjcn.012017.02>
- An, R. (2016). Plain water and sugar-sweetened beverage consumption in relation to energy and nutrient intake at full-service restaurants. *Nutrients*, 8(5). <https://doi.org/10.3390/nu8050263>
- Beck, B., & Max, J. P. (2007). Hypothalamic galanin and plasma leptin and ghrelin in the maintenance of energy intake in the Brattleboro rat. *Biochemical and Biophysical Research Communications*, 364(1), 60–65. <https://doi.org/10.1016/j.bbrc.2007.09.092>
- Blundell, J. (2018). Behaviour, energy balance, obesity and capitalism. *European Journal of Clinical Nutrition*, 72(9), 1305–1309. <https://doi.org/10.1038/s41430-018-0231-x>
- Bordoni, L., Napolioni, V., Marchegiani, F., Amadio, E., & Gabbianelli, R. (2017). Angiotensin-converting enzyme Ins/Del polymorphism and body composition: The intermediary role of hydration status. *Journal of Nutrigenetics and Nutrigenomics*, 10(1–2), 1–8. <https://doi.org/10.1159/000458154>
- Boschmann, M., Steiniger, J., Franke, G., Birkenfeld, A. L., Luft, F. C., & Jordan, J. (2007). Water drinking induces thermogenesis through osmosensitive mechanisms. *The Journal of Clinical Endocrinology and Metabolism*, 92(8), 3334–3337. <https://doi.org/10.1210/jc.2006-1438>
- Brown, J. A., Wright, A., Bugescu, R., Christensen, L., Olson, D. P., & Leininger, G. M. (2019). Distinct subsets of lateral hypothalamic neurotensin neurons are activated by leptin or dehydration. *Scientific Reports*, 9(1), 1873. <https://doi.org/10.1038/s41598-018-38143-9>
- Bruschi Kelles, S. M., Diniz, M. F. H. S., Machado, C. J., & Barreto, S. M. (2014). Mortality rate after open Roux-in-Y gastric bypass: A 10-year follow-up. *Brazilian Journal of Medical and Biological Research*, 47(7), 617–625. <https://doi.org/10.1590/1414-431x20143578>
- Carroll, H. A., Templeman, I., Chen, Y. C., Edinburgh, R., Burch, E. K., Jewitt, J. T., Povey, G., Robinson, T. D., Dooley, W. L., Buckley, C., Rogers, P. J., Gallo, W.,

### Conflict of Interest

The authors declare no conflict of interest. The figure was created with BioRender.com.

- Melander, O., Thompson, D., James, L. J., Johnson, L., & Betts, J. A. (2019). Hydration status affects thirst and salt preference but not energy intake or postprandial ghrelin in healthy adults: A randomised crossover trial. *Physiology & Behavior*, 212, 112725. <https://doi.org/10.1016/j.physbeh.2019.112725>
- Carroll, H. A., Templeman, I., Chen, Y.-C., Edinburgh, R. M., Burch, E. K., Jewitt, J. T., Povey, G., Robinson, T. D., Dooley, W. L., Jones, R., Tsintzas, K., Gallo, W., Melander, O., Thompson, D., James, L. J., Johnson, L., & Betts, J. A. (2019). Effect of acute hypohydration on glycemic regulation in healthy adults: A randomized crossover trial. *Journal of Applied Physiology*, 126(2), 422–430. <https://doi.org/10.1152/jappphysiol.00771.2018>
- Chang, D. C., Penesova, A., Bunt, J. C., Stinson, E. J., Kavouras, S. A., Gluck, M. E., Paddock, E., Walter, M., Piaggi, P., & Krakoff, J. (2022). Water intake, thirst, and copeptin responses to two dehydrating stimuli in lean men and men with obesity. *Obesity*, 30(9), 1806–1817. <https://doi.org/10.1002/oby.23520>
- Chang, T., Ravi, N., Plegue, M. A., Sonnevile, K. R., & Davis, M. M. (2016). Inadequate hydration, BMI, and obesity among US adults: NHANES 2009–2012. *Annals of Family Medicine*, 14(4), 320–324. <https://doi.org/10.1370/afm.1951>
- Cheuvront, S. N., & Kenefick, R. W. (2014). Dehydration: Physiology, assessment, and performance effects. In *Comprehensive Physiology* (pp. 257–285). <https://doi.org/10.1002/cphy.c130017>
- Cheuvront, S. N., & Kenefick, R. W. (2016). Am I drinking enough? Yes, no, and maybe. *Journal of the American College of Nutrition*, 35(2), 185–192. <https://doi.org/10.1080/07315724.2015.1067872>
- Corney, R. A., Horina, A., Sunderland, C., & James, L. J. (2015). Effect of hydration status and fluid availability on ad-libitum energy intake of a semi-solid breakfast. *Appetite*, 91, 399–404. <https://doi.org/10.1016/j.appet.2015.04.075>
- Corney, R. A., Sunderland, C., & James, L. J. (2015). The effect of hydration status on appetite and energy intake. *Journal of Sports Sciences*, 33(8), 761–768. <https://doi.org/10.1080/02640414.2014.962578>
- Corney, R. A., Sunderland, C., & James, L. J. (2016). Immediate pre-meal water ingestion decreases voluntary food intake in lean young males. *European Journal of Nutrition*, 55(2), 815–819. <https://doi.org/10.1007/s00394-015-0903-4>
- De Luca, L. A. Jr, Pereira-Derderian, D. T., Vendramini, R. C., David, R. B., & Menani, J. V. (2010). Water deprivation-induced sodium appetite. *Physiology & Behavior*, 100(5), 535–544. <https://doi.org/10.1016/j.physbeh.2010.02.028>
- De Luca, L. A. Jr, Xu, Z., Schoorlemmer, G. H., Thunhorst, R. L., Beltz, T. G., Menani, J. V., & Johnson, A. K. (2002). Water deprivation-induced sodium appetite: Humoral and cardiovascular mediators and immediate early genes. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, 282(2), R552–R559. <https://doi.org/10.1152/ajpregu.00295.2000>
- Fetissov, S. O., & Meguid, M. M. (2020). Food intake and meal pattern in response to hyperosmotic-induced dehydration in obese and lean Zucker rats. *Nutrition*, 70, 100011. <https://doi.org/10.1016/j.nutx.2020.100011>
- Freitas, W. R. J., Oliveira, L. V. F., Perez, E. A., Ilias, E. J., Lottenberg, C. P., Silva, A. S., Urbano, J. J., Oliveira, M. C. J., Vieira, R. P., Ribeiro-Alves, M., Alves, V. L. S., Kassab, P., Thuler, F. R., & Malheiros, C. A. (2018). Systemic inflammation in severe obese patients undergoing surgery for obesity and weight-related diseases. *Obesity Surgery*, 28(7), 1931–1942. <https://doi.org/10.1007/s11695-017-3104-9>
- Fresán, U., Gea, A., Bes-Rastrollo, M., Ruiz-Canela, M., & Martínez-Gonzalez, M. A. (2016). Substitution models of water for other beverages, and the incidence of obesity and weight gain in the SUN cohort. *Nutrients*, 8(11). <https://doi.org/10.3390/nu8110688>

- Goodman, A. B., Blanck, H. M., Sherry, B., Park, S., Nebeling, L., & Yaroch, A. L. (2013). Behaviors and attitudes associated with low drinking water intake among US adults, Food Attitudes and Behaviors Survey, 2007. *Preventing Chronic Disease*, 10, E51. <https://doi.org/10.5888/pcd10.120248>
- Hara-Chikuma, M., & Verkman, A. S. (2006). Physiological roles of glycerol-transporting aquaporins: The aquaglyceroporins. *Cellular and Molecular Life Sciences*, 63(12), 1386–1392. <https://doi.org/10.1007/s00018-006-6028-4>
- Haroun, D., Wells, J. C., Williams, J. E., Fuller, N. J., Fewtrell, M. S., & Lawson, M. S. (2005). Composition of the fat-free mass in obese and nonobese children: Matched case-control analyses. *International Journal of Obesity*, 29(1), 29–36. <https://doi.org/10.1038/sj.ijo.0802834>
- Keller, U., Szinnai, G., Bilz, S., & Berneis, K. (2003). Effects of changes in hydration on protein, glucose and lipid metabolism in man: Impact on health. *European Journal of Clinical Nutrition*, 57(Suppl 2), S69–S74. <https://doi.org/10.1038/sj.ejcn.1601904>
- Kurt, G., Kodur, N., Quiles, C. R., Reynolds, C., Eagle, A., Mayer, T., Brown, J., Makela, A., Bugescu, R., Seo, H. D., Carroll, Q. E., Daniels, D., Robison, A. J., Mazei-Robison, M., & Leininger, G. (2022). Time to drink: Activating lateral hypothalamic area neurotensin neurons promotes intake of fluid over food in a time-dependent manner. *Physiology & Behavior*, 247, 113707. <https://doi.org/10.1016/j.physbeh.2022.113707>
- Lafontan, M., Visscher, T. L., Farpour-Lambert, N., & Yumuk, V. (2015). Opportunities for intervention strategies for weight management: Global actions on fluid intake patterns. *Obesity Facts*, 8(1), 54–76. <https://doi.org/10.1159/000375103>
- Langdon, S. W., & Dennee-Sommers, B. (2010). Exploring the relationships between self-objectification, rationales, and the use of water as a strategy for appetite suppression. *Psychology, Health & Medicine*, 15(1), 17–25. <https://doi.org/10.1080/13548500903431519>
- Larnkjaer, A., Arnberg, K., Michaelsen, K. F., Jensen, S. M., & Mølgaard, C. (2015). Effect of increased intake of skimmed milk, casein, whey, or water on body composition and leptin in overweight adolescents: A randomized trial. *Pediatric Obesity*, 10(6), 461–467. <https://doi.org/10.1111/ijpo.12007>
- Maffei, C., Tommasi, M., Tomasselli, F., Spinelli, J., Fornari, E., Scattolo, N., Marigliano, M., & Morandi, A. (2016). Fluid intake and hydration status in obese vs normal weight children. *European Journal of Clinical Nutrition*, 70(5), 560–565. <https://doi.org/10.1038/ejcn.2015.170>
- Marra, M. V., Simmons, S. F., Shotwell, M. S., Hudson, A., Hollingsworth, E. K., Long, E., Kuertz, B., & Silver, H. J. (2016). Elevated serum osmolality and total water deficit indicate impaired hydration status in residents of long-term care facilities, regardless of low or high body mass index. *Journal of the Academy of Nutrition and Dietetics*, 116(5), 828–836.e2. <https://doi.org/10.1016/j.jand.2015.12.011>
- Marrades, M. P., Milagro, F. I., Martínez, J. A., & Moreno-Aliaga, M. J. (2006). Differential expression of aquaporin 7 in adipose tissue of lean and obese high-fat consumers. *Biochemical and Biophysical Research Communications*, 339(3), 785–789. <https://doi.org/10.1016/j.bbrc.2005.11.080>
- Mathai, M. L., Naik, S., Sinclair, A. J., Weisinger, H. S., & Weisinger, R. S. (2008). Selective reduction in body fat mass and plasma leptin induced by angiotensin-converting enzyme inhibition in rats. *International Journal of Obesity*, 32(10), 1576–1584. <https://doi.org/10.1038/ijo.2008.126>
- Moosavian, S. P., Haghighatdoost, F., Surkan, P. J., & Azadbakht, L. (2017). Salt and obesity: A systematic review and meta-analysis of observational studies. *International Journal of Food Sciences and Nutrition*, 68(3), 265–277.

- Nickel, C. H., Bingisser, R., & Morgenthaler, N. G. (2012). The role of copeptin as a diagnostic and prognostic biomarker for risk stratification in the emergency department. *BMC Medicine*, 10, 7. <https://doi.org/10.1186/1741-7015-10-7>
- Nissensohn, M., Sánchez-Villegas, A., Ortega, R. M., Aranceta-Bartrina, J., Gil, Á., González-Gross, M., Varela-Moreiras, G., & Serra-Majem, L. (2016). Beverage consumption habits and association with total water and energy intakes in the Spanish population: Findings of the ANIBES study. *Nutrients*, 8(4), 232. <https://doi.org/10.3390/nu8040232>
- Padrão, P., Sousa, A. S., Guerra, R. S., Álvares, L., Santos, A., Borges, N., Afonso, C., Amaral, T. F., & Moreira, P. (2017). A cross-sectional study on the association between 24-h urine osmolality and weight status in older adults. *Nutrients*, 9(11). <https://doi.org/10.3390/nu9111272>
- Pérez-Luco, C., Díaz-Castro, F., Jorquera, C., Troncoso, R., Zbinden-Foncea, H., Johannsen, N. M., & Castro-Sepulveda, M. (2019). Fluid restriction decreases solid food consumption post-exercise. *Nutrients*, 11(6). <https://doi.org/10.3390/nu11061209>
- Puga, A. M., Lopez-Oliva, S., Trives, C., Partearroyo, T., & Varela-Moreiras, G. (2019). Effects of drugs and excipients on hydration status. *Nutrients*, 11(3). <https://doi.org/10.3390/nu11030669>
- Rosinger, A., & Tanner, S. (2015). Water from the fruit or the river? Examining hydration strategies and gastrointestinal illness among Tsimane' adults in the Bolivian Amazon. *Public Health Nutrition*, 18(6), 1098–1108. <https://doi.org/10.1017/S1368980014002158>
- Rosinger, A. Y., Lawman, H. G., Akinbami, L. J., & Ogden, C. L. (2016). The role of obesity in the relation between total water intake and urine osmolality in US adults, 2009–2012. *The American Journal of Clinical Nutrition*, 104(6), 1554–1561. <https://doi.org/10.3945/ajcn.116.137414>
- Roth, C. L., Kratz, M., Ralston, M. M., & Reinehr, T. (2011). Changes in adipose-derived inflammatory cytokines and chemokines after successful lifestyle intervention in obese children. *Metabolism*, 60(4), 445–452. <https://doi.org/10.1016/j.metabol.2010.03.023>
- Sawka, M. N., Cheuvront, S. N., & Carter, R. (2005). Human water needs. *Nutrition Reviews*, 63(suppl\_1), S30–S39.
- Sawka, M. N., Cheuvront, S. N., & Kenefick, R. W. (2015). Hypohydration and human performance: Impact of environment and physiological mechanisms. *Sports Medicine*, 45(1), 51–60. <https://doi.org/10.1007/s40279-015-0395-7>
- Stookey, J. D., Barclay, D., Arieff, A., & Popkin, B. M. (2007). The altered fluid distribution in obesity may reflect plasma hypertonicity. *European Journal of Clinical Nutrition*, 61(2), 190–199. <https://doi.org/10.1038/sj.ejcn.1602521>
- Stookey, J. D., Del Toro, R., Hamer, J., Medina, A., Higa, A., Ng, V., TinajeroDeck, L., & Juarez, L. (2014). Qualitative and/or quantitative drinking water recommendations for pediatric obesity treatment. *Journal of Obesity & Weight Loss Therapy*, 4(4), 232. <https://doi.org/10.4172/2165-7904.1000232>
- Stookey, J. J. (2016). Negative, null, and beneficial effects of drinking water on energy intake, energy expenditure, fat oxidation and weight change in randomized trials: A qualitative review. *Nutrients*, 8(1). <https://doi.org/10.3390/nu8010019>
- Szabová, M., Jahnová, E., Horváthová, M., Ilavská, S., Pružincová, V., Nemessányi, T., Tulinská, J., Wsólóvá, L., & Volková, K. (2012). Changes in immunologic parameters of humoral immunity and adipocytokines in obese persons are gender dependent. *Human Immunology*, 73(5), 486–492. <https://doi.org/10.1016/j.humimm.2012.02.006>
- Tan, P. Y., & Mitra, S. R. (2020). The combined effect of polygenic risk from FTO and ADRB2 gene variants, odds of obesity, and post-Hipcref diet differences.

- Lifestyle Genomics*, 13(2), 84–98.  
<https://doi.org/10.1159/000505662>
- Tate, D. F., Turner-McGrievy, G., Lyons, E., Stevens, J., Erickson, K., Polzien, K., Diamond, M., Wang, X., & Popkin, B. M. (2012). Replacing caloric beverages with water or diet beverages for weight loss in adults: Main results of the Choose Healthy Options Consciously Everyday (CHOICE) randomized clinical trial. *The American Journal of Clinical Nutrition*, 95(3), 555–563.  
<https://doi.org/10.3945/ajcn.111.026278>
- Thornton, S. N. (2011). Angiotensin inhibition and longevity: A question of hydration. *Pflügers Archiv - European Journal of Physiology*, 461(3), 317–324.  
<https://doi.org/10.1007/s00424-010-0911-4>
- Thornton, S. N. (2016). Increased hydration can be associated with weight loss. *Frontiers in Nutrition*, 3, 18.  
<https://doi.org/10.3389/fnut.2016.00018>
- Thornton, S. N., Even, P. C., & Van Dijk, G. (2009). Hydration increases cell metabolism. *International Journal of Obesity*, 33(3), 385.
- Vasilenko, M. A., Kirienkova, E. V., Skuratovskaia, D. A., Zatolokin, P. A., Mironyuk, N. I., & Litvinova, L. S. (2017). The role of production of adipisin and leptin in the development of insulin resistance in patients with abdominal obesity. *Doklady. Biochemistry and Biophysics*, 475(1), 271–276.  
<https://doi.org/10.1134/s160767291704010x>
- Vij, V. A., & Joshi, A. S. (2013). Effect of “water induced thermogenesis” on body weight, body mass index, and body composition of overweight subjects. *Journal of Clinical and Diagnostic Research: JCDR*, 7(9), 1894–1896.  
<https://doi.org/10.7860/jcdr/2013/5862.3344>
- World Health Organization. (2021). *Obesity and overweight*. Retrieved on February 26, 2023, from <http://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>
- World Health Organization. (2022). *The latest State of Food Security and Nutrition Report shows the world is moving backwards in efforts to eliminate hunger and malnutrition*. Retrieved on February 26, 2023, from <https://www.who.int/news/item/06-07-2022-un-report--global-hunger-numbers-rose-to-as-many-as-828-million-in-2021>
- World Health Organization. (2024, March 1). *Obesity and overweight*. Retrieved on June 5, 2024, from <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>
- Zhang, J., Zhang, N., Du, S., Liu, S., & Ma, G. (2021). Effects of water restriction and water replenishment on the content of body water with bioelectrical impedance among young adults in Baoding, China: A randomized controlled trial (RCT). *Nutrients*, 13(2).  
<https://doi.org/10.3390/nu13020553>
- Zhang, X., Wang, J., Li, J., Yu, Y., & Song, Y. (2018). A positive association between dietary sodium intake and obesity and central obesity: Results from the National Health and Nutrition Examination Survey 1999–2006. *Nutrition Research*, 55, 33–44.  
<https://doi.org/10.1016/j.nutres.2018.04.008>

## دور حالة الإمالة في التسبب في السمنة وعلاجها

ساره حكمت الروسان<sup>1\*</sup>، حيدر عبدالله الدومي<sup>1</sup>، محمد جميل الخطاطبة<sup>2</sup>

<sup>1</sup> الجامعة الأردنية، عمان، الأردن.

<sup>2</sup> جامعة العلوم والتكنولوجيا، اربد، الأردن

تاريخ استلام البحث: 2024/6/5 وتاريخ قبوله: 2025/12/11.

### ملخص

تشير الأدلة الناشئة إلى وجود ارتباط قوي بين حالة الإمالة والسمنة ذات المسببات المرضية. تسلط هذه المراجعة النقدية الضوء على الدور المحتمل لحالة الإمالة في الوقاية من السمنة، أو على الأقل تخفيف معدل انتشارها، وهي مرض التهابي مزمن منخفض الدرجة. تم البحث في قواعد بيانات PubMed و Google Scholar و ScienceDirect ومنظمة الصحة العالمية (WHO) عن الأدبيات المنشورة باللغة الإنجليزية، باستخدام مصطلحات البحث بالكلمات الرئيسية التالية "الماء" و "تناول الماء" و "نقص الإمالة" أو "الإمالة" و "استهلاك الطاقة" و "السمنة" و "الشهية" و "الشبع" و "استهلاك الطعام" و "مؤشر كتلة الجسم (BMI)" للفترة بين عامي 2003 و 2023. شملت المراجعة الحالية بيانات من التجارب السريرية، والدراسات المقطعية، ودراسات الأتراب، والمراجعات المنشورة، ودراسات الحالات والشواهد التي تبحث في دور حالة الإمالة في تطور السمنة وكذلك آليات الوقاية منها. الإمالة عن طريق زيادة معتدلة في استهلاك المياه اليومي يمكن أن يعتبر تعديلاً فعالاً غير مكلف لنمط الحياة للأفراد المعرضين لخطر للإصابة بالسمنة. نقص الإمالة قد يكون أحد أهم عوامل الخطر المهيمنة في كل من نشأة السمنة وعلاجها، ومضاعفاتها من خلال عدة آليات أهمها التمثيل الغذائي للدهون، وتوليد الحرارة، وإنفاق الطاقة، واستهلاك الغذاء، واستهلاك الطاقة، والتوازن الهرموني، وتنظيم الشهية، وتنظيم الشبع. يمكن أن تساعد حالة الإمالة المناسبة في منع أو على الأقل تأخير زيادة الوزن. هناك حاجة إلى مزيد من التجارب السريرية للتحقق من الآليات المقترحة. تم إنشاء الشكل باستخدام BioRender.com.

الكلمات الدالة: نقص الإمالة، الإمالة، الماء، السمنة، الشهية، الشبع.

\* الباحث المعتمد للمراسلة: [saraalrosan@hotmail.com](mailto:saraalrosan@hotmail.com)