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Surgical Research

Water Intoxication, Hyponatremia, and the Rise of Digital Hydration Misinformation: A Comprehensive Narrative Review of Physiology, Clinical Evidence, and Social Media–Driven Public Health Risks

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ABSTRACT

Background: *Water intoxication — the clinical syndrome arising from ingestion of fluid volumes exceeding renal excretory capacity — produces dilutional hyponatremia with potentially catastrophic neurological consequences, including cerebral edema, herniation, and death. Despite decades of documented fatalities across diverse clinical contexts, public awareness remains critically inadequate. The proliferation of social media platforms since the early 2010s has introduced a novel and underappreciated exposure pathway: digital health misinformation that actively encourages excessive water consumption through competitive challenges, influencer-promoted hydration rituals, and viral wellness content.*

Objectives: *This narrative review synthesizes the published literature on water homeostasis, the pathophysiology of hyponatremia and cerebral edema, documented clinical cases of water intoxication across all etiological categories, and the emerging evidence base for social media–mediated risk. Epidemiological, toxicological, neurological, and public health dimensions are considered in an integrated framework.*

Methods: *A systematic search of PubMed, MEDLINE, Embase, and grey literature databases was conducted through August 2025. Search terms included: water intoxication, hyponatremia, exercise-associated hyponatremia, psychogenic polydipsia, cerebral edema, osmotic demyelination syndrome, social media health challenges, gallon challenge, WaterTok, and digital health misinformation. Case reports, cohort studies, clinical guidelines, systematic reviews, meta-analyses, and coroner reports were evaluated.*

Results: *The maximal renal excretory capacity for free water is approximately 0.8–1.0 L/hour; ingestion beyond this threshold produces dilutional hyponatremia that may precipitate cerebral edema within hours. Documented fatalities span radio contest participants (KDND-FM, 2007), military recruits, marathon runners, psychiatric patients with psychogenic polydipsia, MDMA users, and individuals attempting rapid rehydration. Exercise-associated hyponatremia affects 7–15% of marathon competitors and carries a documented case fatality rate. Social media platforms — particularly TikTok (#gallonchallenge, #WaterTok), YouTube, and Facebook — have normalized and gamified excessive water intake, creating new exposure risks that exceed historical baseline incidence. The treatment of symptomatic acute hyponatremia with hypertonic (3%) saline is established and life-saving; however, overcorrection precipitates osmotic demyelination syndrome with devastating neurological sequelae.*

Conclusions: *Water intoxication constitutes a preventable public health hazard whose incidence is being amplified by digital misinformation. Coordinated responses are urgently required from public health authorities, social media platforms, nutrition professionals, and clinicians. Regulatory frameworks governing health-related content on digital platforms are inadequate and require reform.*

Keywords
Water intoxication, Hyponatremia, Cerebral edema, Exercise-associated hyponatremia, Psychogenic polydipsia, Osmotic demyelination syndrome, Social media health challenges, Digital health misinformation, WaterTok, Gallon challenge, Public health.

Introduction
Water is biologically indispensable. Its centrality to mammalian physiology has rendered it simultaneously ubiquitous and paradoxically underestimated as a toxicological agent. Yet the principle that any substance consumed in sufficient quantity becomes toxic applies without exception to water. When fluid intake surpasses the kidneys' maximum free-water excretory capacity of approximately 0.8–1.0 liters per hour, plasma osmolality falls, sodium is diluted, and an osmotic gradient is established that drives water into cerebral cells. The resulting cerebral edema, constrained within the rigid calvarium, rapidly becomes life-threatening. This pathological sequence — denominated water intoxication or dilutional hyponatremia — is not a theoretical curiosity but a documented cause of morbidity and death across multiple clinical contexts [1].

The epidemiology of water intoxication has historically encompassed four primary populations: endurance athletes engaging in hyper-hydration during competition; psychiatric patients with psychogenic polydipsia (PPD), a compulsive water-drinking behavior occurring in up to 20% of institutionalized patients with schizophrenia [2]; recreational users of MDMA (3,4-methylenedioxymethamphetamine) who combine drug-stimulated antidiuretic hormone hypersecretion with deliberate fluid loading [3] and individuals subjected to organized water-drinking contests or hazing rituals [4]. More recently, a fifth and underappreciated exposure category has emerged: the algorithmically amplified hydration culture of social media.

Beginning in the early 2010s and accelerating through the COVID-19 pandemic era, digital platforms including TikTok, Instagram, YouTube, and Facebook have hosted a succession of viral trends normalizing extreme water intake. Challenges bearing hashtags such as #gallonchallenge, #gallonofwateraday, and #WaterTok, attracting tens of millions of views, have promoted consumption volumes — typically defined as one US gallon (approximately 3.8 liters) per day — that substantially exceed evidence-based recommendations and that, consumed rapidly, can produce fatal hyponatremia. Influencer testimonials, misinformation about hydration thresholds, and the gamification of water intake have contributed to a cultural environment in which drinking excessive water is portrayed as a health virtue [5].

Despite documented fatalities and repeated public health warnings, no comprehensive review to date has integrated the clinical, physiological, and social media–driven dimensions of water intoxication into a unified analytical framework. The present article addresses this gap. Drawing upon case series, epidemiological data, experimental physiology, clinical guidelines, and qualitative analysis of digital health content, it synthesizes the evidence on water homeostasis, hyponatremia pathophysiology, clinical case documentation, social media influences, and preventive strategies, with the aim of informing clinicians, nutrition scientists, public health professionals, and policymakers.

Methods
Search Strategy
This narrative review was conducted following the general principles applicable to narrative synthesis, with additional systematic features to ensure comprehensive coverage. Electronic databases searched included PubMed/MEDLINE, Embase, Cochrane Library, and Web of Science. Grey literature was retrieved from government public health portals, coroner reports, regulatory agency publications, and verified news archives. Social media–related content was identified through platform-native search tools and published infodemiology studies.

The following MeSH terms and free-text keywords were used in combination: "water intoxication", "dilutional hyponatremia", "hyponatremia", "cerebral edema", "exercise-associated hyponatremia" (EAH), "psychogenic polydipsia", "osmotic demyelination syndrome", "pontine myelinolysis", "antidiuretic hormone", "arginine vasopressin", "social media health challenge", "gallon challenge", "WaterTok", "TikTok health misinformation", "overhydration", "water poisoning", "polydipsia", and "MDMA hyponatremia". The search was conducted through August 2025 without language restriction, though final inclusion prioritized English-language publications.

Inclusion and Exclusion Criteria
Included publications comprised: (1) peer-reviewed case reports and case series documenting water intoxication or hyponatremia; (2) prospective and retrospective cohort studies; (3) systematic reviews and meta-analyses; (4) clinical guidelines from major nephrology, emergency medicine, and sports medicine bodies; (5) infodemiology studies examining social media hydration content; and (6) verified media reports of deaths attributed to water intoxication, cross-referenced with coroner findings where available. Excluded were: studies confounding hyponatremia with primary endocrinological or pharmacological causes unrelated to water intake; studies with unverifiable data sources; and case reports of uncertain diagnostic attribution.

Physiology of water Balance
Total Body Water and Compartmental Distribution
Total body water (TBW) constitutes approximately 60% of lean body mass in adult males and 50–55% in adult females, with substantial variability attributable to adipose tissue content, which is metabolically inert with respect to water. In a 70-kilogram adult, TBW approximates 42 liters, partitioned between the intracellular compartment (~28 L, 67%) and the extracellular compartment (~14 L, 33%). The extracellular compartment is further subdivided into the intravascular (plasma, ~3 L) and interstitial (~11 L) fractions [6]. The relative impermeability of cellular membranes to solutes, combined with active transport mechanisms, maintains the osmotic gradient sustaining this compartmental arrangement. The Gibbs-

Donnan equilibrium and the sodium-potassium ATPase pump are central determinants of intracellular tonicity; sodium constitutes the principal extracellular cation and the primary determinant of plasma osmolality.

Plasma osmolality is maintained within a remarkably narrow physiological range of 275–295 mOsm/kg H2O. Deviations as small as 2 mOsm/kg trigger hypothalamic osmoreceptor responses that modulate both arginine vasopressin (AVP) secretion and thirst [7]. This exquisite sensitivity to osmotic perturbation underscores the inherent fragility of water homeostasis when overwhelmed by volumes beyond the system's buffering capacity.

Renal Free-Water Excretion and Its Physiological Limits

Under conditions of maximal suppression of AVP — the operational state prevailing in healthy individuals during a water load — the kidneys can excrete approximately 0.8 to 1.0 liter of electrolyte-free water per hour, contingent upon adequate renal function and sufficient solute delivery to the collecting duct [8]. This capacity is determined by the renal solute load (predominantly urea and electrolytes), the glomerular filtration rate, and the diluting capacity of the loop of Henle and distal tubule. The clinically critical implication is unambiguous: any individual who ingests water at a rate exceeding approximately one liter per hour for a sustained period risks progressive plasma dilution, regardless of baseline health status.

The kidneys' diluting mechanism operates principally in the thick ascending limb of the loop of Henle and the distal convoluted tubule, where active sodium chloride reabsorption without concomitant water movement generates a dilute tubular fluid. In the virtual absence of AVP, aquaporin-2 (AQP2) channels remain internalized from the apical membrane of collecting duct principal cells, rendering the collecting duct impermeable to water; dilute urine is therefore produced [9]. AVP binds to V2 receptors on basolateral membranes, triggering a cyclic AMP–mediated signaling cascade that inserts AQP2 channels into the apical membrane, enabling water reabsorption [10]. Physiological suppression of AVP thus represents the primary protective mechanism against water intoxication; conditions that maintain or paradoxically elevate AVP secretion in the setting of hypo-osmolality — including non-osmotic stimuli such as nausea, pain, exercise-induced volume contraction, and pharmacological agents — dramatically reduce this protective capacity.

Thirst Regulation and Its Limitations

The thirst response is mediated by hypothalamic osmoreceptors located in the organum vasculosum of the lamina terminalis (OVLT) and the subfornical organ, which lie outside the blood-brain barrier and are consequently directly responsive to plasma tonicity [11]. Thirst is stimulated when plasma osmolality rises above approximately 285–290 mOsm/kg or when there is a significant reduction in effective circulating volume. However, this physiological feedback system has critical limitations as a safeguard against overhydration: thirst suppression occurs only once adequate oral intake has restored plasma osmolality to its set-point, yet this inhibitory signal may be overridden by habit, social pressure, competitive incentives, misperceived dehydration, pharmacological alteration, or psychiatric compulsion. The contemporary cultural narrative — amplified by social media — that thirst is an unreliable and already-delayed hydration signal requires critical scrutiny; in healthy individuals without pharmacological interference, thirst constitutes an efficient and appropriate guide to fluid intake.

Water Intoxication and Hyponatremia: Pathophysiology

Definition and Classification of Hyponatremia

Hyponatremia is defined as a serum sodium concentration below 135 mmol/L and represents the most commonly identified electrolyte disturbance in clinical practice, occurring in 20–35% of hospitalized patients [12]. Its classification is fundamental to clinical management and reflects distinct pathophysiological mechanisms. The traditional volumetric classification distinguishes hypovolemic (total body sodium and water depleted, with sodium deficit exceeding water deficit), euvolemic (total body sodium near-normal with excess total body water, the paradigmatic category in primary water intoxication), and hypervolemic (excess of both total body sodium and water, with a proportionately greater excess of water) hyponatremia [13].

From an osmolality perspective, hypotonic hyponatremia — the category relevant to water intoxication — involves genuine reduction in plasma osmolality and accounts for the overwhelming majority of clinically significant cases. Isotonic or hypertonic hyponatremia, by contrast, results from specific etiologies such as pseudohyponatremia in lipemic states or translocational hyponatremia induced by hyperglycemia, and are not products of water excess [14].

Temporal classification is of paramount prognostic importance: acute hyponatremia, developing within 48 hours, carries a dramatically higher risk of cerebral edema than chronic hyponatremia of equivalent magnitude, because the brain's adaptive volume-regulatory mechanisms — the extrusion of intracellular organic osmolytes including taurine, glutamine, glutamate, myoinositol, and creatine — require 48–72 hours to fully compensate [15]. Water intoxication from acute, rapid fluid loading classically produces acute hyponatremia with a compressed time frame for cerebral adaptation and consequently a high risk of fatal cerebral herniation.

Mechanisms of Dilutional Hyponatremia

In pure water intoxication, the pathogenesis follows a direct and quantifiable sequence. Ingestion of water in excess of the renal excretory capacity produces net free-water retention, which distributes across total body water compartments. Because sodium is predominantly extracellular, the dilutional effect on plasma sodium concentration is proportional to the volume of water retained relative to pre-existing total body water. A 70-kg individual with a TBW of 42 L and a serum sodium of 140 mmol/L who retains an excess 3 liters of water will sustain a serum sodium reduction to approximately 130 mmol/L — already within the

range of symptomatic hyponatremia. Rapid ingestion of 5–6 liters in excess of renal clearance capacity can reduce serum sodium to levels below 120 mmol/L within hours [16].

The physiological scenario is often compounded by non-osmotic AVP stimulation. During vigorous exercise, AVP is released in response to volume contraction, hyperthermia, and nausea, independent of plasma osmolality; this prevents the free-water diuresis that would otherwise partially compensate water overload [17]. In MDMA-intoxicated individuals, the drug directly stimulates AVP secretion through serotonergic and oxytocin-mediated mechanisms, simultaneously compounding thirst stimulation from hyperthermia [18]. In psychiatric patients with PPD, disordered thirst regulation, antipsychotic medication effects, and hypothalamic dopaminergic dysregulation all converge to sustain compulsive intake [19].

Cerebral Edema and Neurological Consequences

The neurological sequelae of acute hyponatremia arise from water movement into cerebral cells, driven by the osmotic gradient between the hypotonic extracellular fluid and the relatively hypertonic intracellular compartment. Brain cells swell as a consequence, and because neuronal tissue is encased within the rigid calvarium, there is minimal capacity for volumetric accommodation. Intracranial pressure rises, cerebral perfusion pressure falls, and transtentorial herniation may occur [20].

The clinical progression of acute hyponatremic encephalopathy follows a characteristic trajectory. Mild symptoms — nausea, headache, and malaise — emerge when serum sodium falls below 125–130 mmol/L. As sodium declines below 120 mmol/L, altered mental status, confusion, and behavioral changes appear. At concentrations below 115–120 mmol/L, seizures, respiratory distress, and loss of consciousness are common; at sodium levels below 110–115 mmol/L, respiratory arrest and death from transtentorial herniation may occur with little further warning [21]. Postmortem findings consistently include diffuse cerebral edema with astrocytic swelling, herniation patterns, pulmonary congestion, and in forensic cases reported by Pini et al. (2025), ubiquitin-positive axonal swellings and reactive astrogliosis reflecting the profound ionic disruption preceding death [22].

An important and clinically relevant sex difference has been identified: premenopausal women are substantially more vulnerable to hyponatremic encephalopathy at equivalent serum sodium concentrations compared to men. Estrogen inhibits the extrusion of brain osmolytes during the adaptive response and augments non-osmotic AVP release, reducing the safety margin available before cerebral herniation occurs [23]. The syndrome of severe hyponatremia with pulmonary and cerebral edema occurring disproportionately in premenopausal women — termed the Varon-Ayus syndrome — was first documented in marathon runners and remains an important consideration in risk stratification [24].

Osmotic Demyelination Syndrome and the Paradox of Treatment

The management of severe hyponatremia is complicated by a counter-intuitive therapeutic hazard: overly rapid correction of chronic hyponatremia can precipitate osmotic demyelination syndrome (ODS), previously denominated central pontine myelinolysis (CPM), a potentially devastating neurological complication characterized by non-inflammatory demyelination of the central pons and, in extrapontine forms, the basal ganglia, thalamus, and cerebellum [25]. The pathophysiological mechanism involves the disruption of endothelial tight junctions and astrocyte dysfunction when the rapid rise in plasma osmolality overwhelms the brain's capacity to re-accumulate the organic osmolytes previously extruded during chronic hyponatremic adaptation, resulting in osmotic stress and myelin damage [26].

A 2024 meta-analysis by See et al. identified seven cohort studies encompassing 6,032 patients with severe hyponatremia, reporting an ODS incidence of 0.48%; rapid sodium correction (exceeding 8–12 mmol/L per 24-hour period) was associated with a 3.91-fold increased relative risk of ODS (95% CI, 1.17–13.04) compared to controlled correction [27]. A larger systematic review with meta-analysis by Chiang et al. (2024) incorporating 26,710 patients demonstrated an overall ODS incidence of 0.23%, with rates of 0.73% and 0.10% for rapid and non-rapid correction groups, respectively (OR 3.16, 95% CI, 1.54–6.49) [28]. Current clinical guidelines from the European Society of Intensive Care Medicine and the American Society of Nephrology recommend correction rates not exceeding 8 mmol/L per 24 hours in high-risk patients (chronic hyponatremia, alcoholism, hypokalemia, malnutrition, advanced liver disease), with prompt intervention with hypertonic saline in acute symptomatic cases, where the urgency of reversing cerebral edema outweighs the ODS risk [29].

Documented Clinical Cases of water Intoxication

Competitive Water-Drinking Contests and Entertainment-Related Deaths

The most comprehensively documented fatality attributable to a water-drinking contest occurred on January 12, 2007, at Sacramento radio station KDND-FM (107.9, "The End"), Entercom Communications Corporation, Sacramento, California. The station's morning program conducted a promotional contest denominated "Hold Your Wee for a Wii," offering a Nintendo Wii gaming console as prize to the contestant able to consume the greatest volume of water without urinating. Approximately 20 competitors participated, drinking 250 mL aliquots of water at 15-minute intervals, with the volume subsequently increasing to 500 mL per round. Jennifer Strange, a 28-year-old mother of three children, consumed an estimated 7.5 liters of water over approximately three hours [30].

Strange left the station reporting severe headache, a hallmark prodrome of hyponatremic encephalopathy. She was found deceased in her home approximately six hours following the contest. Post-mortem examination confirmed water intoxication as cause of death [31]. Critically, during the broadcast, a nurse

practitioner called the station to warn of the lethal risks; disc jockeys dismissed the warning, and audiotape of the broadcast documented a female DJ asking, "Can't you get water poisoning and, like, die?" — a warning ignored in real time [32]. Entercom Communications was found liable in the wrongful death proceedings; the jury awarded the Strange family USD $16.5 million in compensatory damages. All ten station staff directly involved in the contest were terminated [33]. The case established that the hazard of organized water-drinking contests was foreseeable and that failure to heed expert warnings constitutes actionable negligence.

A preceding institutional precedent is provided by the death of Matthew Carrington, a 21-year-old California State University, Chico student who died in February 2005 during fraternity hazing at Chi Tau fraternity. Carrington was compelled to consume large volumes of water alternating with calisthenics performed in a cold basement. He died of hyponatremia-induced cerebral edema. Convictions were secured under California's anti-hazing statute [34]. The KDND radio station personnel were audiotaped referencing Carrington's death — as a subject of mockery — during the very broadcast that would kill Jennifer Strange [32].

Exercise-Associated Hyponatremia

Exercise-associated hyponatremia (EAH) was first formally characterized by Timothy Noakes and colleagues in 1985 following observations of collapsed ultra-marathon runners in the Comrades Marathon, South Africa, who presented with clinical hyponatremia attributable to excessive fluid ingestion during competition [35]. The condition has since been documented across marathon running, ultra-marathon running, triathlon, hiking, and military training; it constitutes the single numerically largest category of water intoxication-related morbidity.

The pathophysiology of EAH is distinct from simple water overload. During prolonged endurance exercise, non-osmotic stimuli — including hypovolemia, hyperthermia, nausea, and direct musculoskeletal injury-related cytokine release — stimulate AVP secretion independently of plasma osmolality, impeding the normal free-water diuresis that would otherwise compensate hypo-osmolality produced by hypotonic fluid ingestion [36]. The net effect is retention of ingested free water despite dilutional plasma hypo-osmolality. Runners who over-drink — often because of pre-race hydration advice urging maximal fluid intake — and who run slowly enough to generate sustained non-osmotic AVP secretion are at highest risk.

Epidemiological data from Klingert et al. (2022), a narrative review of EAH among marathon runners, reported prevalence estimates of 7–15% for combined symptomatic and asymptomatic EAH. In the landmark Boston Marathon 2002 study by Almond et al., 13% of runners who provided post-race samples had hyponatremia (serum sodium < 135 mmol/L), with 0.6% demonstrating critical hyponatremia (< 120 mmol/L); weight gain during the race — reflecting net fluid retention — was the strongest predictor [37]. A review of seven outdoor endurance activities by Hoffman and Stuempfle documented 220 EAH field cases and identified upright weight-bearing exercise (marathon, ultramarathon, hiking) as accounting for 89.2% of all reported cases [38]. Regarding sex differences, a higher percentage of women than men develop EAH, consistent with estrogen-mediated vulnerability discussed in Section 4.3.24.

Fatalities from EAH are documented but uncommon in absolute terms, though their occurrence is predictable given the prevalence of the condition and the delay in recognition. Eight deaths attributable to EAH have been documented since 1985 [39]. Fatal cases typically involve the development of hyponatremic encephalopathy with cerebral herniation, pulmonary edema, or respiratory arrest at finish lines or in the hours following race completion, when athletes have stopped sweating but continue to retain previously ingested water. The Varon-Ayus syndrome — pulmonary and cerebral edema with fatal outcome — has been documented disproportionately in younger women competing in endurance events [23].

Psychogenic Polydipsia in Psychiatric Populations

Psychogenic polydipsia (PPD) is a chronic, compulsive pattern of excessive water drinking that occurs in an estimated 6–20% of institutionalized psychiatric patients, with the highest prevalence in chronic schizophrenia [40]. The etiology of PPD remains incompletely understood; proposed mechanisms include hypothalamic-dopaminergic dysregulation altering the thirst set-point, antipsychotic medication effects modifying renal water handling (particularly antipsychotics with significant anticholinergic activity, which provoke dry mouth and secondary polydipsia), and delusional or psychotic ideation that incorporates water consumption [41].

The clinical consequences of PPD are severe. In a retrospective study reviewed by Gill et al. (2015), 18.5% of patients who died before the age of 53 in a psychiatric institution were found to have died from complications of water intoxication [42]. Patients with schizophrenia and comorbid polydipsia have a 13% shorter life expectancy than non-polydipsic schizophrenic patients [43]. Pini et al. (2025), publishing in Cureus, reported two forensic autopsy cases illustrating the full clinical-pathological spectrum of fatal PPD: the first, a 42-year-old woman with chronic schizophrenia, developed gradual-onset severe hyponatremia (< 120 mmol/L) that allowed partial therapeutic intervention; the second, a 28-year-old woman with postpartum psychosis who harbored religious delusions compelling compulsive water consumption, deteriorated to cardiopulmonary arrest within hours of hospital admission. Both cases demonstrated diffuse cerebral edema, pulmonary congestion, and diffuse axonal injury with reactive astrogliosis on post-mortem neuropathological examination [22].

Case reports from Evanson and Espiridion (2023) and from the CHEST journal (2022) further document the spectrum of hyponatremic injury in psychiatric patients: one case involved severe hyponatremia with seizures requiring intubation,

hypertonic saline correction, and transition to DDAVP protocol to avoid overcorrection; another involved a 32-year-old male with schizophrenia in whom sodium fell to 100 mEq/L, precipitating rhabdomyolysis and acute kidney injury requiring urgent renal replacement therapy [44].

Drug-Associated Hyponatremia: MDMA and Related Substances

MDMA (3,4-methylenedioxymethamphetamine, "ecstasy") occupies a unique position in the pharmacological epidemiology of water intoxication by combining two synergistic mechanisms: direct stimulation of AVP secretion via serotonergic pathways, and drug-induced hyperthermia with associated thirst stimulation and fluid-seeking behavior at raves and dance events where advice to drink large amounts of water is commonly given as harm-reduction guidance [18]. The paradox of this advice is now well-established: in a person with pharmacologically elevated AVP, even moderate water intake can produce fatal hyponatremia because the kidneys are rendered incapable of excreting the excess.

The death of Leah Betts on November 16, 1995, in Chelmsford, Essex, United Kingdom, remains the most widely known MDMA-related water intoxication fatality. Betts, 18 years old, ingested an MDMA tablet during her birthday celebration and subsequently drank approximately 7 liters of water within a 90-minute period. She collapsed into coma four hours later and was declared brain dead five days subsequently; the inquest concluded that water intoxication producing fatal cerebral edema was the cause of death [45]. The case generated extensive public health controversy: the initial narrative attributed her death to "one ecstasy tablet," obscuring the central role of excessive water intake and pharmacologically impaired AVP suppression, a misrepresentation with significant implications for drug harm-reduction messaging that persists to the present.

In a military context, a 20-year-old basic military trainee described in a 2021 Cureus case report (PMC8484258) developed acute symptomatic hypotonic hyponatremia after ingesting five to six liters of water prior to a mandatory urine drug test — a pattern of presentation also documented in civilian populations attempting to adulterate drug-screening results through dilutional protocols [46].

Contemporary Non-Athletic Civilian Cases

Ashley Summers, a 35-year-old mother of two from Indiana, United States, died on July 6, 2023, following a period of intense rehydration after boating at Lake Freeman, Monticello, during Fourth of July weekend. Her family reported that, in the context of perceived severe dehydration after outdoor heat exposure, she consumed approximately 64 ounces (approximately 1.9 liters) of water within 20 minutes. She subsequently developed headache, lightheadedness, and progressive obtundation; she collapsed in her garage and was transported to Indiana University Health Arnett Hospital, where she never regained consciousness. The official cause of death documented by the White County Coroner was "cerebral edema and herniation with anoxic brain injury due to electrolyte imbalance "[47]. The Summers case, widely reported by national and international media, was significant in introducing the concept of water toxicity to broad public awareness, including through social media, where her brother Devon Miller posted warnings that accumulated millions of views.

In September 2023, actress Brooke Shields, 58, suffered a grand mal (tonic-clonic) seizure that she subsequently attributed to water intoxication. Shields, preparing for a one-woman theatrical performance at Café Carlyle, New York City, reported that she had been consuming "so much water" in response to perceived dehydration from the demands of rehearsal, singing, and performance, and had not been aware that her serum sodium had fallen to a critically low level. In her published account in Glamour's 2023 Women of the Year interview, she described loss of consciousness, tonic-clonic convulsions, perioral cyanosis, and ambulance transport; hospital assessment confirmed hyponatremia [48]. The Shields case received global media coverage and generated substantial public discussion about the risks of overhydration, including from physicians who subsequently noted that her case also likely reflected inadequate dietary sodium intake compounding the dilutional effect.

Social Media Challenges and Digital Misinformation

The Digital Amplification of Hydration Culture

The cultural valorization of high water intake predates social media, with its roots in 20th-century marketing by beverage corporations, the evolution of sports hydration science, and the promotion of "8×8" (eight 8-ounce glasses per day) hydration guidelines whose evidence base has been challenged in the peer-reviewed literature [49]. However, the advent of social media platforms fundamentally altered the velocity and reach of hydration-related messaging, enabling misinformation to propagate from individual content creators to audiences of millions within days. The incentive structures of platform algorithms, which reward engagement over accuracy, and the absence of robust regulatory mechanisms for health-related user-generated content, have created an environment in which dangerous hydration advice can achieve viral reach without scrutiny.

A scoping review of risky social media challenges published in PMC (2025) examined the broader landscape of online behavioral challenges spanning 2000–2024, identifying a consistent pattern in which challenges of dubious or negative health value — the Tide Pod Challenge, cinnamon challenge, and various dietary extremes — achieve rapid dissemination through platforms including TikTok, Instagram, Facebook, and YouTube before public health countermeasures can be deployed.50 Water-related hydration challenges exemplify this pattern, with the additional hazard that, unlike challenges with immediately aversive sensory properties, consuming large amounts of water carries no immediate deterrent signal to naive participants.

The Gallon Challenge and Related Hydration Challenges

The "Gallon Challenge" or "Gallon-a-Day Challenge" (hashtags

#gallonchallenge, #gallonofwateraday, #gallonaday) constitutes one of the most persistent hydration-related trends on social media. The challenge prescribes consumption of one full US gallon (approximately 3.8 liters) of water per day for a defined period — typically 30 days — often while excluding other beverages. Content creators document their daily intake, report skin, energy, and weight changes, and invite followers to participate. TikTok and YouTube hosts under hashtags including #30daywaterchallenge have accumulated tens of millions of aggregate views [5].

The physiological risk profile of the gallon-a-day challenge is context-dependent. For most adults engaged in moderate activity under temperate conditions, 3.8 liters per day — when consumed across 24 hours — is unlikely to exceed maximal renal clearance capacity, as healthy kidneys can excrete the equivalent in excess of 20 liters per day when appropriately distributed. The genuine risk is threefold: first, participants are encouraged to consume this quantity rapidly, often in large morning doses; second, certain vulnerable populations (elderly individuals with reduced glomerular filtration rate, individuals on SSRI or antipsychotic medications, individuals with hypothyroidism or adrenal insufficiency) lack the renal reserve to safely excrete even moderate water loads; third, the challenge normalizes supra-physiological intake as a wellness ideal, distorting the reference frame against which further escalation is judged. The promotion of gallon-per-day intake in conjunction with low-solute diets, fasting, or diuretic use represents a genuine water intoxication risk.

WaterTok: Flavored Water Culture and Disordered Hydration

WaterTok (#WaterTok) emerged as a distinct social media trend approximately 2022–2023, primarily on TikTok, in which creators prepared and shared "water recipes" — combinations of water with flavored sugar-free syrups, powdered drink supplements, and colorants, served in large-volume insulated tumblers. The trend merged hydration culture with beverage aesthetics, generating content that simultaneously promoted high daily water intake and provided consumption structures around which identity and community could be organized [51].

Health experts issued public warnings regarding WaterTok on grounds that extended beyond the overhydration risk. Martha Williams, a specialist at Beat (a UK eating disorder charity), identified a plausible pathway from WaterTok to water loading behavior — the practice of consuming large volumes of water to manipulate body weight measurements, a practice associated with disordered eating — in media commentary [52]. From a biochemical perspective, the flavoring systems used in many WaterTok preparations contain artificial sweeteners and additives whose interactions with hydration physiology are poorly characterized, and the systematic replacement of dietary fluids with sweetener-containing water preparations may alter gastrointestinal microbiome composition and appetite regulation in ways not yet empirically studied.

Influencer-Promoted Extreme Hydration and Celebrity Amplification

The influence of celebrities on public hydration behavior merits specific analysis. Attributions of celebrities' reported consumption of 1 gallon of water per day — circulated on social media platforms — function as powerful normative signals, particularly among populations for whom celebrity behavior constitutes a significant reference standard. The journalistic trope "Celebrity X drinks a gallon of water per day: why shouldn't you?" exemplifies the logical inversion that social desirability bias introduces into health messaging [53].

Brooke Shields' 2023 seizure generated an unusual countervailing effect: a celebrity's near-fatal water intoxication received global media coverage, effectively communicating — in terms accessible to a general audience — the previously abstract risk of overhydration. Published analyses of media response suggest that this type of personal testimonial, delivered with emotional immediacy, can generate short-term increases in health information-seeking behavior [48]. However, the mechanism is inherently dependent on dramatic adverse events, constituting reactive rather than preventive public health communication.

Influencer-promoted salt water trends, documented on TikTok in 2023–2024, represent an illustrative counter-current: content creators advocating addition of table salt in quantities of up to two teaspoons per glass — equivalent to approximately 3,360 mg sodium — to "enhance hydration" by creating a hypertonic beverage [54]. Registered dietitian Lisa Young and other nutrition professionals publicly characterized this practice as having "no benefit whatsoever" while posing independent cardiovascular risks. The trend underscores the degree to which social media health content exists in a landscape of competing physiological claims, most of which have little or no empirical validation.

Platforms, Algorithms, and Regulatory Considerations

The platforms most implicated in the dissemination of excessive hydration content — TikTok, Instagram, YouTube, and Facebook — operate under regulatory frameworks principally designed to address hate speech, misinformation in the political domain, and exploitation of minors, rather than health misinformation with potentially delayed or individually variable harms. Health-related content moderation on these platforms relies heavily on automated systems and reactive community reporting, mechanisms that are fundamentally unsuited to the rapid detection and suppression of novel health challenges that generate engagement precisely because of their novelty.

Purushothaman et al. (2022), in an infodemiology analysis of nicotine poisoning ("Nic Sick") videos on TikTok, documented the platform's amplification of content depicting harmful self-experimentation and identified the lag between content proliferation and platform response as a structural vulnerability [55]. The same dynamic applies to hydration challenges: by the time platform moderation engages with a hashtag, the organic reach

of the challenge typically has already declined. This implies that preventive rather than reactive platform governance is required — a challenge the platforms have not yet systematically addressed for nutrition-related content.

Public Health Implications

Vulnerable Populations and Risk Stratification

Not all individuals face equivalent risk from excessive water intake. Risk stratification is essential for targeting preventive communication. The highest-risk populations identified in the literature include: (1) endurance athletes, particularly women, novice competitors, and those running at slower pace with prolonged course exposure [56]; (2) psychiatric patients with psychogenic polydipsia, particularly those with schizophrenia receiving antipsychotic medications [40]; (3) MDMA and stimulant users in settings providing unsafe hydration advice [57]; (4) infants under 12 months of age, in whom even modest water supplementation can produce hyponatremia due to immature renal concentrating mechanisms and small body mass [58]; (5) elderly individuals with reduced renal reserve and heightened non-osmotic AVP secretion; (6) individuals receiving thiazide or loop diuretics, SSRIs, or antipsychotic medications, which impair renal sodium excretion and free-water clearance [59] and (7) individuals consuming low-solute diets ("tea-and-toast" diet, beer potomania), whose limited solute excretion restricts free-water clearance even at normal urine osmolality.

Diagnostic Challenges and Clinical Misattribution

A significant public health challenge is the non-specific clinical presentation of early hyponatremia, which overlaps with dehydration — the condition it biologically opposes. Nausea, headache, malaise, fatigue, and muscle weakness may be attributed to insufficient water intake in the community setting, leading to further water consumption that exacerbates rather than resolves the underlying hypo-osmolality [60]. Clinicians assessing athletes, festival attendees, or individuals arriving after outdoor heat exposure must maintain a high index of suspicion for hyponatremia even in the context of apparent dehydration complaints. Point-of-care serum sodium measurement should be immediately available in race medical tents, emergency departments, and psychiatric facilities where high-risk populations are concentrated.

The educational gap extends to emergency medicine clinicians. Military trainees, athletes, and individuals presenting with altered mental status and a history of high fluid intake may be misdiagnosed as having hyperthermia, hypovolemic shock, or toxicological syndromes, and may inadvertently receive hypotonic fluid administration that further worsens serum sodium. The critical importance of obtaining a fluid intake history and performing immediate serum electrolyte measurement in any patient presenting with neurological deterioration after apparent fluid consumption cannot be overstated.

Recommendations and Prevention Strategies

Clinical and Institutional Recommendations

For symptomatic acute hyponatremia with neurological manifestations (confusion, seizures, coma), immediate administration of hypertonic 3% saline is the evidence-based first-line treatment. Current guidance, including the European consensus statement and guidance from the American Society of Nephrology, recommends 100–150 mL boluses of 3% saline administered intravenously, repeated as needed, with the target of raising serum sodium by 4–6 mmol/L to relieve acute cerebral edema, and a maximum correction rate of 10–12 mmol/L in the first 24 hours and 18 mmol/L in 48 hours for patients not at high ODS risk [29]. Patients with risk factors for ODS (chronic hyponatremia, alcoholism, liver cirrhosis, hypokalemia, malnutrition) should have correction limited to 8 mmol/L per 24 hours, monitored by frequent serum sodium measurements [27].

For exercise-associated hyponatremia, the Wilderness Medical Society and other sports medicine bodies recommend oral hypertonic saline for mild-to-moderate EAH in ambulatory athletes, and intravenous 3% saline for moderate-to-severe cases. The critical guidance for race medical teams is that isotonic saline must not be administered to hyponatremic athletes, as it can worsen or fail to correct the hyponatremia. Body weight monitoring during ultra-endurance events — using pre-race weight as the reference — remains the most reliable field measure for detecting water overload, with any weight gain during a race representing net fluid retention rather than performance optimization [36].

In psychiatric facilities, institutional protocols should mandate monitoring of fluid intake and serum electrolytes in patients with known PPD; fluid restriction is the primary management strategy, supplemented by behavioral interventions, and in refractory cases by pharmacological agents including demeclocycline, clozapine (which has demonstrated particular efficacy in reducing polydipsic behavior in schizophrenia), and lithium [40].

Public Health and Nutrition Communication Recommendations

Evidence-based water intake guidance from the National Academy of Medicine specifies total water intake (from all dietary sources) of approximately 3.7 liters/day for men and 2.7 liters/day for women, noting that dietary food contributes approximately 20% of this total [61]. These figures represent population-level medians derived from survey data; they do not constitute minimal required intakes, and individuals satisfying thirst through normal dietary and drinking patterns are almost universally meeting their hydration needs. The promotion of intake substantially above these levels — as systematized by the gallon challenge — lacks evidential support and carries calculable risk.

Public health communication should explicitly address the "more is always better" fallacy as applied to water, using plain-language analogies appropriate for social media audiences. Nutrition educators, dietitians, and healthcare professionals active on digital platforms have an obligation to generate counter-content

that challenges viral hydration misinformation with accessible, accurate evidence-based messaging. Professional bodies including the Academy of Nutrition and Dietetics, the European Food Safety Authority, and the World Health Organization should develop official guidance specifically addressing digital hydration misinformation.

Race organizers, military commanders, and event managers should provide explicit pre-event briefings on EAH risk, emphasizing drink-to-thirst rather than pre-emptive aggressive hydration, and ensuring that medical personnel are equipped and trained to recognize and treat hyponatremia.

Platform Governance and Regulatory Recommendations

Social media platform governance regarding health content requires systemic reform. Specifically: (1) platforms should classify water intoxication risk as a health hazard category warranting proactive monitoring, not solely reactive user reporting; (2) content associated with documented high-volume water intake challenges should be subject to the same preventive warning framework applied to other documented self-harm risks; (3) influencer accounts making specific physiological claims about water consumption should be required to disclose the absence of medical qualification and to link to authoritative external resources; and (4) health regulatory agencies — including the US Food and Drug Administration, the European Food Safety Authority, and national-level equivalents — should develop frameworks for engaging with social media platforms on the regulation of nutrition-related health claims analogous to those applied to food labeling.

Gaps in the Literature and Future Research Directions

Several significant gaps limit the current evidence base. First, the epidemiology of water intoxication attributable specifically to social media–influenced behavior has not been systematically characterized. No published study to date has prospectively or retrospectively quantified the incidence or burden of hyponatremia hospitalizations associated with online hydration challenges, leaving the public health magnitude of this exposure pathway entirely undefined. Surveillance systems that capture etiology-specific data for hyponatremia admissions are urgently needed.

Second, the relationship between individual renal excretory capacity and risk from a given water intake rate has been characterized at the population level but not in individuals in real-world consumption contexts. The factors determining inter-individual variability in free-water excretion — including GFR, solute intake, ambient temperature, hormonal status, and pharmacological exposure — require integration into clinically applicable risk models.

Third, the dietary context of high water intake — particularly the interaction of low-sodium diets, intermittent fasting, and high water consumption volumes — has received limited attention. The emerging popularity of fasting regimens combined with high water intake, often promoted concurrently on social media, represents a plausible risk amplification factor warranting dedicated investigation.

Fourth, the effectiveness of various communication strategies in countering social media–driven hydration misinformation has not been studied in randomized or quasi-experimental designs. Infodemiology methods exist for characterizing content reach and engagement, but behavioral outcomes of counter-messaging campaigns are unstudied.

Fifth, the neurological outcome data for survivors of severe water intoxication — particularly those managed with hypertonic saline — remain sparse. Long-term cognitive sequelae of near-fatal hyponatremic encephalopathy and ODS are poorly documented in longitudinal follow-up studies.

Conclusion

Water intoxication and its principal biochemical consequence, dilutional hyponatremia, constitute a preventable public health hazard with a documented history of mortality spanning diverse clinical and social contexts. The physiological mechanisms are well established: renal free-water excretory capacity has a fixed ceiling of approximately 0.8–1.0 liters per hour, and ingestion beyond this threshold in a sustained manner produces osmotic dilution of plasma sodium, cerebral edema, and potentially fatal neurological compromise. The clinical spectrum ranges from athletic deaths at marathon finish lines to psychiatric ward fatalities, to competitive contest-related homicide-equivalent negligence, to spontaneous recreational overconsumption.

The emergence of social media–driven hydration culture represents a qualitatively new dimension of this established hazard. The normalization of supra-physiological water intake through platforms reaching hundreds of millions of users, the gamification of water consumption through challenges generating peer social rewards for compliance, and the systematic misrepresentation of evidence-based hydration science in influencer content collectively constitute a digital public health threat that has not yet attracted the regulatory and professional attention it warrants.

The response required is multi-sectoral. Clinicians must maintain heightened vigilance for water intoxication presentations across the full spectrum of clinical settings, ensuring that serum sodium is promptly measured in any patient with neurological deterioration and a history of high fluid intake, and that the evidence-based treatment with hypertonic saline is initiated without delay. Nutrition professionals and public health communicators must engage actively with digital platforms to produce and amplify evidence-based counter-content. Regulatory agencies must develop frameworks appropriate for the governance of health claims in social media environments. Platform operators must accept that algorithmic amplification of health misinformation with documented fatal consequences constitutes an addressable engineering and governance failure.

Water is not toxic at physiologically appropriate intake volumes —

it is essential. But no substance is without a toxic dose threshold, and the social media assertion that more water is unconditionally better represents a falsification of basic toxicological principle with demonstrably lethal consequences. Translating this message into effective, scalable, and culturally resonant public health communication is the central challenge confronting the nutrition and clinical medicine communities in the digital age.

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