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Research report

Dietary sodium, added salt, and serum sodium associations with growth and depression in the U.S. general population ☆

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ABSTRACT

It is not known why salt is so attractive to humans. Here, guided by hypotheses suggesting that the attraction of salt is conditioned by postingestive benefits, we sought to establish whether there are such benefits in a population by analyzing the National Health and Nutrition Examination Survey (NHANES) 2007–2008 database (n = ~ 10,000). We focus on two potential benefits supported by the literature, growth and moderation of depression, and examine their relationship to sodium, dietary, added at table, and serum. We find that during growth (<18 years), there is a specific increase in adjusted dietary sodium intake, independent of caloric or other electrolyte intakes. We find that adding salt and depression are related. In contrast, and in women only, dietary sodium and depression are inversely related. The relationships are correlational, but we speculate that this constellation may reflect self-medication for depression by adding salt, and that men may be protected by their higher dietary sodium intake. Additional findings are that women add more salt than men below age ~30, after which men add more, and below 40 years of age, serum sodium is lower in women than in men. It remains possible that small but beneficial effects of sodium could condition salt preference and thus contribute to population-wide sodium intake.

Introduction

Humans voluntarily ingest far more salt than any other animal, but unlike research on animal salt appetite, there is no understanding of the underlying causes for human salt-seeking (Beauchamp, 1987; Geerling & Loewy, 2008; Leshem, 2009, 2013; Mattes, 1997; Newson et al., 2013). Such knowledge is required because salt intake is a worldwide health concern (Al-Dahhan, Jannoun, & Haycock, 2002; Dietary Guidelines for Americans, 2012; Du, Neiman, Batis, Wang, Zhang, Zhang, and Popkin, 2014; He & MacGregor, 2010; Henney, Taylor, & Boon, 2010; IOM (Institute of Medicine), 2013; Moritz & Ayus, 2008; Taylor, Ashton, Moxham, Hooper, & Ebrahim, 2011).

Yet currently, the most extant hypothesis for high salt intake is that commercial products infiltrate sodium insensibly into our nutrition (He & MacGregor, 2010). It is arguable whether this is involuntary sodium intake (Shepherd, Farleigh, & Wharf, 1989), because it is not known why there is a hedonic preference for such foods in the first place, other than the tautological that people primarily choose their food for salt content or flavor (Beauchamp, 1987; Henney et al., 2010; Leshem, 2009; Liem, Miremadi, & Keast, 2011; Mattes, 1997).

Nevertheless, despite the lack of knowledge about the root causes of the human predilection for salt, the hypothesis is the one that shapes policy, and health authorities work to curtail sodium intake by reducing the salt content of commercially prepared food and to increase awareness of its sodium content (Dietary Guidelines for Americans, 2012; He & MacGregor, 2010; Hooper, Bartlett, Davey Smith, & Ebrahim, 2002; Henney et al., 2010; IOM (Institute of Medicine), 2013; Liem, Miremadi, Zandstra, & Keast, 2012; McCarron, Kazaks, Geerling, Stern, & Graudal, 2013; Webster, Li, Dunford, Nowson, & Neal, 2010). However, there is yet no consensus as to whether decreased sodium in commercially prepared food, increased dietary advice, and product ingredient labeling, are effective in reducing salt intake (Bernstein & Willett, 2010; Bolhuis et al., 2011; Brown, Tzoulaki, Candeias, & Elliott, 2009; Du et al., 2014; He & MacGregor, 2010; Henney et al., 2010; Hooper et al., 2002; Liem et al., 2012; Luft, Morris, & Weinberger, 1997; Millett, Laverty, Stylianou, Bibbins-Domingo, & Pape, 2012; Newson et al., 2013; Webster et al., 2010). Indeed, the hypothesis provides little insight into the causes of the similar worldwide high salt intake, where nonindustrialized food preponderates (Brown et al., 2009; Du et al., 2014; He & MacGregor, 2010; McCarron et al., 2013). Spontaneous reduction or increases in sodium intake may occur because of dietary

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trends, or "global uniformization" (Meneton et al., 2009), and sodium intake may also be stable despite significant changes and differences in dietary habits (Hoy, Goldman, Murayi, Rhodes, & Moshfegh, 2011; Kurlansky, 2003; Leshem, 2009; Leshem, Saadi, Alem, & Hendi, 2008; McCarron et al., 2013). Thus the greatest reduction in salt use (as preservative) followed the invention of the refrigerator, yet a century later we still relish the cured hams, salted beef, pork, salami, herring, anchovy, cheeses, biscuits, sauerkraut, brine-pickled vegetables, and other necessities of that bygone era (Kurlansky, 2003; Leshem, 2009). Indeed, in the 2007–2008 NHANES sample they are the largest contribution to sodium intake (>9%). We are ignorant of why such powerful preferences persist across generations.

Among the primary properties of salt driving its attraction is taste enhancement, bitterness and sour suppression, and its hedonic appeal, resulting in its supraphysiological ingestion (Breslin & Beauchamp, 1997; Wise & Breslin, 2011). Yet while the hedonics of the other basic tastes are believed to be adaptive in guiding intake by its energy (sweet), protein (umami) or toxic (bitter, sour) qualities, it is not known how sodium may be adaptive in the enormous quantities it is ingested relative to physiological need.

Sodium intake is a complex behavior, impacting all of the body's physiological systems, and a comprehensive understanding of salt use, its determinants, variants, and in particular its short-term effects, may focus intervention more effectively (Beauchamp, 1987; Gibson, 2008; Henney et al., 2010; Leshem, 2009; Mattes, 1997; Morris, Na, & Johnson, 2008; Newson et al., 2013).

A rational approach might consider whether restriction of sodium intake engenders compensatory intake in individuals or populations (Beauchamp, Bertino, & Engelman, 1987; Bolhuis et al., 2011; Shepherd et al., 1989), whether some benefits of sodium might be lost, such as for specific groups of healthy or ill people, whether, what, and among whom, measures might ameliorate the avidity. Little is known of these issues and the state of the art is such that the long-term consequences of salt intake are exhaustively studied while its primary causes are generally ignored (Hayes, Sullivan, & Duffy, 2010; IOM (Institute of Medicine), 2013; Leshem, 2009; Mattes, 1997).

An alternative hypothesis posits that the short-term benefits of sodium intake may condition the preference for salt. Such effects must be beneficial in the short-term and hence positively reinforcing, even if the intake they engender may be deleterious in the long-term (long-delayed effects cannot condition a preference or aversion). For example, there is evidence that the postingestive effects of sodium in hastening rehydration might condition its preference (Leshem, 2009; Leshem, Abutbul, & Eilon, 1999; Moritz & Ayus, 2008; Passe, Stofan, Rowe, Horswill, & Murray, 2009; Valentine, 2007; Wald & Leshem, 2003; Yeomans, Blundell, & Leshem, 2004).

One possibility is that salt intake is related to all-source water intake to maintain euhydration. It is clear that sodium promotes euhydration after exertion-induced dehydration (Leshem et al., 1999; Moritz & Ayus, 2008; Passe et al., 2009) and that this can condition a preference (Wald & Leshem, 2003); however, no relation of dietary sodium and all-source water ('moisture') intake was found in the NHANES population sample (Kant & Graubard, 2010; Kant, Graubard, & Atchison, 2009), and other detailed analyses of the relationship of dietary sodium and urinary water or total water intake when adjusted for BMI or energy (Gibson, 2008; Kant & Graubard, 2010; Kant et al., 2009).

Sodium is implicated in two more important issues, stress and growth, and it is these that this report examines.

The possibility that salt intake protects against stress is an enduring hypothesis deriving from the commonality of adrenal corticoids mediating salt appetite and stress, and the intertwining renin-angiotensin aldosterone system (RAAS) regulating sodium appetite, sodium concentration, fluid volume, and sympathetic tone including blood pressure and depression (Apostolopoulou et al., 2014; Henry, 1988; Krause et al., 2011b; Morris, Na, Grippo, & Johnson, 2006; Morris et al., 2008; Morris, Na, & Johnson, 2010; Murck, Schüssler, & Steiger, 2012; Rollnik, Mills, & Dimsdale, 1995; Weber et al., 2008). It is a bidirectional hypothesis, positing that stress may increase salt intake, and that sodium intake or loss may respectively reduce stress or aggravate it (Henry, 1988). The effects of stress on sodium appetite have been tested in experiments in humans and rats, and in a number of experiments with endocrine activation of the HPA axis in various species (Oliver, Wardle, & Gibson, 2000; Torres, Turner, & Nowson, 2010). In contrast, the effects of sodium intake or loss on stress have been studied only in rats, which by and large is the only species to show effects compatible with the hypothesis that sodium deficiency may engender stress (Bensi, Bertuzzi, Armario, & Gauna, 1997; Ely, Herman, Ely, Barrett, & Milsted, 2000; Grippo, Moffitt, Beltz, & Johnson, 2006; Henry, 1988; Howell et al., 1999; Krause et al., 2011a, 2011b; Leshem, 2011; Morris et al., 2006, 2010; Torres et al., 2010).

Animal experiments show that sodium deficiency or sodium hunger model depression (reduced exploration in the elevated maze and open field, reduced sweet hedonics and increased current for electrical brain self-stimulation), which is reversed by sodium intake (Grippo et al., 2006; Morris et al., 2006, 2010). Moreover, sodium deficiency as well as excess in animal models have been related to social interaction, respectively withdrawal or its prevention (Ely et al., 2000; Henry, 1988; Krause et al., 2011a).

Voluntary intake of sodium as a response to stress has not been shown convincingly in humans (Leshem, 2009; Torres et al., 2010) despite some collateral results (Miller et al., 1998; Oliver et al., 2000). Moreover, exogenous experimental endocrine activation of the hypothalamic–pituitary–adrenal axis in pigs and primates, including humans, does not increase sodium intake, although in rats and rabbits it often does (Shade et al., 2002; Wong et al., 1993).

Nevertheless, a systematic study of the relationship of depression to sodium in humans is lacking, and would need to be demonstrated before it can be shown to condition a salt preference.

The single most replicated determinant of sodium intake in humans is prenatal and neonatal sodium loss which contributes to increased intake of salt later in life (Crystal & Bernstein, 1995; Leshem, 1998; Shirazki, Weintraub, Reich, Gershon, & Leshem, 2007; Stein et al., 1996; Zinner, McGarvey, Lipsitt, & Rosner, 2002). A number of possible mechanisms for such long-term changes in salt preference have been suggested, and it may be related to early programming of metabolic changes, yet no precise mechanism for the effect has emerged (Alwasel, Barker, & Ashton, 2012). The obligate dehydration and hyponatremia of low-weight neonates is addressed by sodium supplementation to prevent developmental deficits (Al-Dahhan et al., 2002; Haycock, 1993). Such treatment may also alleviate discomfort in the neonates, and may therefore condition increased dietary sodium intake in the long term (Kochli, Tenenbaum-Rakover, & Leshem, 2005; Shirazki et al., 2007).

Even without the boost of perinatal sodium loss, babies divide into those averse to salt, avid or indifferent to it, but infants rapidly develop a preference that leads to salt intakes exceeding that of adults throughout childhood and well into adolescence. In part this could be due to the increased caloric requirement of growth, but there is also evidence that salt preference is high among children following on an early decline (Beauchamp, Cowart, & Moran, 1986; De Courcy, Mitchell, Simmons, & MacGregor, 1986; Desor, Greene, & Maller, 1975; Harris, Thomas, & Booth, 1990; Leshem, 2009; Stein et al., 2006; Verma, Mittal, Ghildiyal, Chaudhary, & Mahajan, 2007; Zinner et al., 2002). Taken together, if these effects indeed reflect need or benefit, one might expect conditioning of preference for sodium.

Here we use the National Health and Nutrition Examination Survey (NHANES) 2007–2008 data to dissect the relationship of dietary sodium intake, voluntarily added salt, and serum sodium concentration to short-term benefits suggested most robustly by the