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**Review Article**

**Does stress induce salt intake?**

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(Received 4 September 2009 – Revised 4 February 2010 – Accepted 24 February 2010 – First published online 26 April 2010)

Psychological stress is a common feature of modern day societies, and contributes to the global burden of disease. It was proposed by Henry over 20 years ago that the salt intake of a society reflects the level of stress, and that stress, through its effect on increasing salt intake, is an important factor in the development of hypertension. This review evaluates the evidence from animal and human studies to determine if stress does induce a salt appetite and increase salt consumption in human subjects. Findings from animal studies suggest that stress may drive salt intake, with evidence for a potential mechanism via the sympatho-adrenal medullary system and/or the hypothalamo–pituitary–adrenal axis. In contrast, in the few laboratory studies conducted in human subjects, none has found that acute stress affects salt intake. However, one study demonstrated that life stress (chronic stress) was associated with increased consumption of snack foods, which included, but not specifically, highly salty snacks. Studies investigating the influence of chronic stress on eating behaviours are required, including consumption of salty foods. From the available evidence, we can conclude that in free-living, Na-replete individuals, consuming Na in excess of physiological requirements, stress is unlikely to be a major contributor to salt intake.

**Acute stress: Chronic stress: Salt intake: Blood pressure: Eating behaviour**

Henry(1) suggested over 20 years ago that the ‘salt (NaCl) consumption of a society is a measure of the social stress to which it is exposed’, and that stress, through its effect on increasing salt intake, is an important factor in the development of hypertension. Henry’s hypothesis was initially based on ecological studies in human subjects, which suggested that salt intake may be driven by exposure to stress(1,2). These ecological studies relied on observational analyses, rather than on direct measures, and included a comparison of stress exposure in two different Polynesian communities: one which reflected a modern day, Westernised society that was likely to have higher stress exposure, had higher salt intakes and exhibited a blood pressure (BP) rise with age; and the other was the traditional community, with potentially low exposure to stress, and was associated with low salt intakes and minimal increases of BP with age(1–3).

Hypertension is a well-established risk factor for CVD(4,5), and is a contributing factor to the development of cerebrovascular disease, IHD, and cardiac and renal failure(6). There is overwhelming evidence that salt intake is linked to the development of hypertension(7–11). It also appears that stress plays a role in increasing risk of CVD(12). This raises the question – could stress actually induce a salt appetite and increase salt consumption in human subjects?

In this review, we will address the question – does stress increase salt intake in human subjects? Firstly, we describe salt appetite, salt preferences and the physiological responses to stress. Secondly, we assess the effect of stress on salt intake, reviewing evidence from animal models and human studies, which has been the recent focus of the present research. Thirdly, we discuss the physiological mechanisms that may link stress to salt intake using data from animal studies.

**Salt appetite and salt preference**

Salt appetite is the strong motivation for ingesting salt in situations of salt wasting(13), whereas salt preference is a liking for salt in a Na-replete state(14), both salt appetite and salt preference can be induced or innate, the latter may be determined by epigenetic influences on the foetus’s genotype(15). Herbivores characteristically exhibit a salt appetite, which is likely to be due to a low Na diet, but omnivores, such as human subjects, do not typically exhibit this behaviour due to a high salt diet(2). Of interest is the finding that taste cells expressing the epithelial Na channel, which mediates behavioural attraction to salt, have been identified in mice, and may be present in human subjects(16). Salt appetite in human subjects is rare, but it has been documented in
Stress and salt intake

Stressors of varying severity and duration have been imposed, and the intake of a test NaCl solution has been measured (Table 1). Eight studies reported an increase(34–41), one study reported no change(35) and two studies reported a decrease in salt intake(42–43). It appears that under these conditions, stress can increase salt intake.

Physiological mechanism for a stress-induced salt appetite

Any element of the physiological systems activated during stress may mediate the effects of stress on salt intake. When rats that exhibited a stress-induced increase in salt intake were given a drug that blocked sympatho-adrenal medullary system activity and were then subjected to stress, salt intake from both food and fluids decreased(34,41). One possible explanation is that sympatho-adrenal medullary system activity may increase urinary Na excretion, resulting in a Na-depleted state which may increase Na appetite(44,45).

Corticotrophin-releasing hormone decreased salt intake when administered subcutaneously to sheep(46) and baboons(47) and when administered directly into the lateral parabrachial nucleus of the brain in rats(48). In contrast, corticotrophin-releasing hormone increased salt intake after intracerebroventricular infusion in mice(37) and rabbits(49), and it had no effect after subcutaneous infusion in mice(37). Subcutaneous infusion of adrenocorticotropic hormone stimulated salt intake in mice(37), sheep(46), rabbits(49) and rats(50), although studies in baboons(47) and pigs(51) reported no effect of intramuscular injections of adrenocorticotropic hormone. Glucocorticoids may also influence salt appetite, but possibly only when co-administered with mineralocorticoids. Shelat et al.(52) found that glucocorticoids in isolation did not influence salt intake in rats, but when glucocorticoids were administered in combination with a mineralocorticoid, a salt appetite was induced.
and this may have been mediated by the direct effects of angiotensin II in the brain. In another study conducted in rats, glucocorticoid co-administered with a mineralocorticoid was found to increase salt intake, and this may have been due to increased urinary excretion of water and Na\(^+(53)\); a Na-induced appetite may have resulted from Na depletion\(^{(54)}\). It is not clear why administration of elements of the hypothalamo–pituitary–adrenal axis resulted in different outcomes in different studies. The route of administration may be important since salt appetite-regulatory pathways\(^{(55)}\) may be influenced in a stimulatory or inhibitory manner through different routes of administration of the hypothalamo–pituitary–adrenal axis.

### Table 1. Summary of the effects of stress on salt intake in animals\(^*\)

<table>
<thead>
<tr>
<th>Study</th>
<th>Stressor</th>
<th>Stressor severity</th>
<th>Stressor duration</th>
<th>Number of animals</th>
<th>Effect on salt intake (pre-v. post-stress)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ely et al.(^{(41)})</td>
<td>Intruder</td>
<td>Mild</td>
<td>Acute</td>
<td>Ten male SHR(^{†‡})</td>
<td>Increased (+138%)</td>
</tr>
<tr>
<td>Denton et al.(^{(37)})</td>
<td>Immobilisation</td>
<td>Severe</td>
<td>Chronic</td>
<td>Six stressed mice</td>
<td>Increased (values NR)</td>
</tr>
<tr>
<td>Leshem et al.(^{(40)})</td>
<td>Postnatal maternal separation</td>
<td>Moderate</td>
<td>Chronic</td>
<td>Five stressed 15-d-old rats(^§)</td>
<td>Increased (+20%) in adult rats</td>
</tr>
<tr>
<td>Bourjellil et al.(^{(34)})</td>
<td>Intruder</td>
<td>Mild</td>
<td>Chronic</td>
<td>Six to eight WKYR(^†)</td>
<td>Increased (1.8 v. 2.5 mmol/100 g (rat)/24 h) (+38%)</td>
</tr>
<tr>
<td>Kuta et al.(^{(38)})</td>
<td>Immobilisation</td>
<td>Mild</td>
<td>Chronic</td>
<td>Eight WKYR(^†)</td>
<td>Increased (+30%)</td>
</tr>
<tr>
<td>Denton et al.(^{(36)})</td>
<td>Attachment of jacket</td>
<td>Severe</td>
<td>Chronic</td>
<td>Six stressed mice(^‡)</td>
<td>Increased (+150%)</td>
</tr>
<tr>
<td>Howell et al.(^{(35)})</td>
<td>Restraint</td>
<td>Moderate</td>
<td>Acute</td>
<td>Six hamsters(^†‡)</td>
<td>Increased (5.1 v. 8.5 mmol/24 h) (+67%)</td>
</tr>
<tr>
<td>Niebyński et al.(^{(43)})</td>
<td>Immobilisation</td>
<td>Severe</td>
<td>Acute</td>
<td>Ten WKYR(^†‡)</td>
<td>Unchanged (58.0 v. 66.0 mmol/24 h)</td>
</tr>
<tr>
<td>Bensi et al.(^{(42)})</td>
<td>Immobilisation</td>
<td>Severe</td>
<td>Chronic</td>
<td>Nine stressed WKYR(^‡)</td>
<td>Decreased (29-0 ml/24 h in control rats v. 14.5 ml/24 h in stressed rats) (−50%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nine control WKYR(^‡)</td>
<td>Decreased (5.4 mmol/22 h in control rats v. 1.9 mmol/22 h in stressed rats) (−184%)</td>
</tr>
</tbody>
</table>

SHR, spontaneously hypertensive rat; WKYR, Wistar-Kyoto rats; NR, not reported.

* All differences are at \(P<0.05\) level.

† Own control.

‡ Intake measured after conclusion of stress.

§ Na depleted.

**Fig. 1.** Physiological systems activated in response to stress. * Any element of the physiological systems activated during stress may be involved in inducing a salt appetite, and there is evidence for some of these actions in animal studies (see text for details). Nevertheless, since human subjects are generally salt replete due to an excess of salt in the food supply, these mechanisms are not likely to be active in human populations. CRH, corticotrophin-releasing hormone; ACTH, adrenocorticotropic hormone.
hormones. There may also be species-specific differences related to the function of the hypothalamo–pituitary–adrenal axis hormones in salt appetite. Further studies would be needed to elucidate the precise mechanisms involved. A systematic approach in a single species would be best, with caution being exercised in the extrapolation of the findings to human subjects.

The mechanism of salt appetite involves two processes: central regulation and regulation by the renal system. Mechanisms of central regulation of salt appetite are not fully resolved, but they are thought to include input signals from aldosterone and angiotensin II, from sensory inputs via baroreceptors and from detection of intracerebroventricular Na concentrations \( ^{(55)} \). Many different brainstem and forebrain regions (such as lamina terminalis and amygdala) are involved in the integration of these signals. These brain regions are involved in inducing motor responses such as salt-ingestive behaviours in the case of Na deficiency \( ^{(55)} \). Glucocorticoids are thought to enhance the salt appetite-promoting actions of aldosterone by increasing the concentration of mineralocorticoid receptors in the brain \( ^{(56,57)} \). The renal system is also involved in the regulation of Na levels via the renin–angiotensin–aldosterone system \( ^{(58)} \). When Na levels fall, renin is secreted by the kidney. Renin catalyses the conversion of angiotensinogen to angiotensin I and then to angiotensin-converting enzyme. Angiotensin II results in the secretion of aldosterone from the adrenal cortex, which in turn increases Na reabsorption by the distal and collecting tubules of the kidney. Angiotensin II and aldosterone act directly on the lamina terminalis and amygdala to stimulate Na appetite.

**Stress-induced salt intake: evidence from human studies**

There are five laboratory studies conducted in human subjects, which allow close monitoring of food intake, that have examined the effect of stress on intake of salt and high salt foods (Table 2). One of these was a study that we conducted in men and women (n 20) with a mean age of 38·6 (SD 11·5) years and a mean BMI of 23·8 (SD 3·3) kg/m\(^2\), which investigated the effect of acute mental arithmetic stress induced in a laboratory setting on salt preference (Torres SJ & Nowson CA, unpublished results, 2008). Subjects were asked to indicate their preference for tomato juice with a range of salt concentrations (0, 109, 173, 240, 304, 370, 435 and 565 mmol/l) before and after acute mental stress. The mean perceived level of stress (range: 1 (no stress) to 10 (severe stress)) was 5·9 (SEM 0·5). The mental stress test caused a significant increase in systolic BP (+13·8 (SEM 2·2) mmHg), diastolic BP (+8·7 (SEM 1·5) mmHg) and pulse rate (+11·2 (SEM 7·9) beats per minute) \( (P<0·05 \text{ for all}) \). There was no significant difference in mean salt preferences in the non-stressed and post-stress states, 82 (SEM 16) v. 96 (SEM 13) mmol/l \( (P>0·05) \). All five laboratory studies reported significant increases in stress by either subjective or objective measures: three studies reported significant increases in BP and heart rate (Torres SJ & Nowson CA, unpublished results, 2008) \( ^{(59,60)} \); one study reported an increase in cortisol \( ^{(61)} \) and three studies reported increases in self-reported stress (Torres SJ & Nowson CA, unpublished results, 2008) \( ^{(59,62)} \). In summary, laboratory-based studies have found no effect of stress on salt intake in human subjects. Even though all the studies reported

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**Table 2. Summary of the effects of stress on salt intake in human subjects**

<table>
<thead>
<tr>
<th>Stressor</th>
<th>Stress duration</th>
<th>Food/fluid</th>
<th>Timing of eating</th>
<th>Subjects</th>
<th>Effect on salt intake (stress v. control group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torres &amp; Nowson†</td>
<td>Acute</td>
<td>Tomato juice</td>
<td>After stress</td>
<td>Twenty male and female adults‡</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Miller et al.</td>
<td>Acute</td>
<td>Salt added to salt-free soup</td>
<td>After stress</td>
<td>Thirty-seven low hostile‡§ Thirty-two high hostile‡§</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Zellner et al.</td>
<td>Acute</td>
<td>Potato chips and peanuts</td>
<td>After stress</td>
<td>Female undergraduate students§</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Oliver et al.</td>
<td>Acute</td>
<td>Potato chips and peanuts</td>
<td>After stress</td>
<td>Male undergraduate students§§</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Oliver &amp; Wardle(63)</td>
<td>Chronic</td>
<td>General food consumption</td>
<td>During stress</td>
<td>Fifty-nine premenopausal female adults†</td>
<td>Salted snack foods increased</td>
</tr>
</tbody>
</table>

TSST, Trier Social Stress Test.
- All differences are at \( P<0·05 \) level.
- Based on visual analog scale scores.
- Based on visual analog scale scores.
- Based on visual analog scale scores.
- Based on visual analog scale scores.
- Based on visual analog scale scores.
significant increases in stress, we may not see an effect on salt intake in laboratory studies as the response to acute stress induced may differ from chronic exposure to a stressful environment.

Naturalistic studies provide the opportunity to measure the effect of life stress on salt intake. The effect of self-reported stress on eating behaviour was examined in 212 undergraduate students (Table 2)(63). Snacking increased during periods of stress, and foods eaten in greater quantity included sweets and chocolate, cakes and biscuits, and savoury snacks (high salt foods). However, the observed increase in the consumption of savoury snacks in response to stress may be due to a drive for fat rather than for salt, or the perception that snacks are treats/rewards. In a previous review, we concluded that chronic life stress seems to be associated with a greater preference for energy- and nutrient-dense foods, namely those that are high in sugar and fat(64).

It has been suggested that cortisol, a key hormone secreted during stress, may be a critical factor in the drive for hedonic, highly palatable foods(65) such as foods containing a high content of salt(66). Cortisol may increase appetite by affecting leptin and neuropeptide Y, key hormones that reduce(67) and those that are high in sugar and fat(64).

Data from animal studies suggest that stress might be a driver for salt intake, with evidence for a potential mechanism by the sympatho-adrenal medullary system and/or hypothalamo–pituitary–adrenal axis. In contrast, laboratory studies conducted in human subjects have found no effect of acute stress on salt intake. However, one study which measured the effect of life stress found that intake of snack foods including highly salty foods did increase, although there are likely to be a range of drivers for this behaviour other than a craving for salt taste. Stress could induce a learned response to consume comfort foods during stress which could include high salt foods. The majority of studies in human subjects have investigated the effect of acute stress. Studies investigating the influence of chronic stress on eating behaviours are required, including consumption of salty foods. In the current environment where most human subjects are consuming Na well in excess of physiological requirements, acute stress is unlikely to increase salt intake.

Acknowledgements

S. J. T. performed the literature review and wrote the manuscript except for the sections on ‘Responses to stress’ and ‘Physiological mechanism for a stress-induced salt appetite’, which were written by A. I. T.; C. A. N. provided expert input and guidance. All authors have read and approved all sections of the manuscript, and participated in the decision to submit for publication. The authors have no conflict of interest. The present research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

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