BACKGROUND REPORT - VETERANS ' AFFAIRS CONSULTANCY

Justin Kenardy, PhD September, 1998.

I have been asked to provide input into determining if there is any evidence of a relationship between salt intake and addiction. In other words, if salt over-consumption can be construed as an addiction.

There are two issues I will address in covering this overall question-

1. What are the psychological and physiological processes that determine salt consumption?
2. Can salt over-consumption be viewed as a substance dependence?

A number of caveats must be stated at the outset of this report.

a. Firstly almost all research into the psychophysiology of salt has been done with analogue populations, that is animals.

b. Secondly almost all research has evaluated the effects of salt in isolation. Hence it may not be possible to absolutely distinguish the factors associated with macronutrient intake from salt intake in humans.

What are the psychological and the physiological processes that facilitate salt consumption, or salt-appetite?

Psychological Processes

Sensory or hedonic preferences for particular intensities of salt develop over a period of time. Ordinarily these preferences take about 2-4 months of consuming a particular quantity of salt on a daily basis to produce an enduring preference (Capaldi, 1996). These changes in salt preferences can be formed for both increases and decreases in daily salt intake and concentrations.

Experiments by Beauchamp and colleagues (Beauchamp,1987; Bertino, Beauchamp & Engelman, 1986) show that increases in salt intake following repeated exposure to higher salt diets indicate an underlying process that it purely sensory or hedonic (for the pleasure of the flavour) rather than because of a physiological need, or dependence on salt. Giving subjects a 10g/day increase of salt in the form of table salt on food, over six weeks, increased their preference for higher concentrations of salt on foods. However, subjects given an increase of 10g of salt per day in the form of a salt tablet (which does not carry the same taste qualities) did not increase their desire for higher salt intensities in soup or crackers.

This indicates that preferences for salt following high-salt diets are due to altered sensory experience with salt, and hedonic factors, not due to a physiological response to large amounts of sodium consumed (Bertino, et al., 1986).

Physiological Processes

There is also a physiologically driven appetite for salt. This salt appetite, unlike the increased hedonic preference due to exposure and learning, is the result of the body being in a state of sodium depletion.

Salt appetite resulting from sodium depletion has been demonstrated many times through experiments with rats. Bertino & Tordoff (1988) found that although rats normally do not prefer salted foods to unsalted foods (and conversely prefer dilute saline to unsalted water), sodium-depleted rats consumed more salted than unsalted sodium-deficient food when freely offered both. They also consumed more salt water than water. This indicates that a salt appetite is aroused in sodium depleted animals, even for salted foods, which they otherwise would not prefer.

In a study testing whether rats could associate flavours of food with its salt content (Coldwell & Tordoff, 1993) found that rats' preference for a salt-paired flavours were strongest when rats were severely sodium depleted, less strong after mild depletion and absent when sodium-replete. Berridge and Schulkin (1989) demonstrated that concentrated NaCl becomes more palatable during sodium-depletion and can act as an incentive.

Studies by Huggins, di Nicolantonio, and Morgan (1992) and Bertino et al (1986) which show that increases in untasted dietary salt do not increase (and may even reduce) salt preference, support these findings. Although salted foods are normally more preferable to humans than unsalted foods, and tasted salt increases salt preference, these preferences are reduced by high bodily levels of sodium.

It is definitely the sodium in salt, not the chloride, that drives the salt appetite. It has been demonstrated that chloride depletion does not increase NaC1 appetite.

Muntzel et al (1991) tested three groups of rats, (NaC1 depleted, Na+ depleted, and Cl- depleted), for their salt appetite, as measured by saline consumption when offered both saline and water. Both NaC1 and Na+ depletion in rats had stimulated salt appetite, whereas C1- depletion did not change the saline intake from baseline.

The salt appetite increase in sodium-depleted rats returns to normal when sodium levels are restored. If however a rat is depleted a second or subsequent time, the effect of the sodium depletion on the salt appetite is more drastic and results in permanently higher levels of need-free salt intake. This occurs even if the interval between first and second depletion is up to four months (Sakai, Fine, Epstein & Frankman, 1987). The depletion-driven consumption of salt after a subsequent depletion is more rapid and more robust than the intake to restore sodium levels after the first depletion (McCaughey, Giza & Scott, 1996; Sakai, et al., 1987; Stellar, 1993).

Central Nervous System

It is sodium depletion within the central nervous system (CNS) that activates a large salt appetite. Decreasing the concentration of sodium in the CSF (cerebrospinal fluid) in the brain by infusing 0.SM

NaC1 artificial CSF, causes a large reduction of sodium appetite in sodium-depleted sheep (Weisinger, et al., 1987). Equivalent increases of CSF osmotic pressure without infusing NaC1 (and thus decreasing CSF[Na]) doubles sodium appetite. Even in severely sodium-deplete animals, lowering CSF[Na] stimulates sodium appetite further. These dramatic consequences of manipulating concentrations of sodium in the central nervous system indicates that sodium serves an important function in the brain.

Origins of salt appetite in humans

The development of salt appetites in human infants is consistent with the evidence concerning the role of sodium in the CNS. Neonatal infants do not produce a response indicating preference or aversion, to the taste of salt as they do with sweet, sour and bitter tastes. The preference for a salty taste, indicated by facial expressions and sucking reflexes, develops between three and six months of age, which is thought to reflect postnatal maturation in central mechanisms (Mennella & Beauchamp, 1996). It seems

also that single sodium depletions very early in life can produce permanent. elevations in salt appetite (Frankmann, Dorsa, Sakai, & Simpson, 1986; Sakai, Fine, Epstein, & Frankmann, 1987).

Although not evident at birth, the sodium appetite is certainly an innate process (Stellar, 1993). Richter (1936), first showed the innate salt appetite in rats as an adrenalectomised rat kept itself alive by drinking hypertonic NaC1 solutions. Similarly, in humans, the innate desire for salt was illustrated by Wilkins and Richter (1940). They described a three-year-old boy who ate copious amounts of salt. He was admitted to hospital as his eating was otherwise poor, and was administered a low-salt diet. Within a week he died, and the autopsy revealed that his adrenal glands were destroyed by tumours, hence his innate need for copious amounts of salt.

CNS mechanism of salt-appetite modulation

Two of the major hormones involved in the appetite for salt seem to be Angiotensin II and Aldosterone. Depletion-induced sodium appetite in reduced by 50% by blocking either hormone in the brain, and is eliminated by blocking both hormones (Sakai, & Epstein, 1990; Stellar, 1993). Threats to body fluid homeostasis releases renin from the kidneys which catalyses the production of angiotensin. It acts peripherally, to promote vasoconstriction, release aldosterone from the kidneys, and to promote water and sodium retention. Centrally, angiotensin together with aldosterone stimulates sodium appetite (Stellar, 1993).

After a comprehensive review of the evidence concerning the physiology of salt appetite, Stellar (1993) concluded that repeated sodium depletions increase salt appetite due to altered sensitivity of the brain to further depletions by the activity of angiotensin and aldosterone; these increases in salt appetite also extends to need-free salt appetite (when sodium replete) and causes chronic excess salt intake; salt appetite is greater in females than in males, and that the effect of prior depletions is also greater.

In my review of the literature *there was no evidence found of CNS mechanisms that directly link stress hormone activity to salt consumption*.

Conclusion- there is a physiologically driven need for salt, and in some cases a need for excess salt, however this is not due to overconsuming salt. On the contrary, it is a result of current or prior sodium-depletions. The increase in salt intake after a period of consuming higher amounts of salt is not physiologically driven but is rather a hedonic preference for that amount and concentration of salt.

Can salt over-consumption be viewed as a substance dependence?

In order to determine whether salt over-consumption can be regarded as an addiction, we will examine the processes that facilitate salt consumption with regard to the DSM-IV (APA, 1994) definition of substance dependence. Please refer to the Appendix for the full criteria.

The definition of substance abuse could possibly be stretched to fit the case of severe repeated sodium-depletion. The substance is taken in larger amounts and over a longer period than was intended, due to the increased need-free salt-appetite. High consumption of salt may continue despite knowing of the possible detrimental effects of consuming salt in excess. This is again due to the increased need-free salt appetite brought on by risk of sodium-depletion. This may possibly be coupled with a persistent desire or unsuccessful attempts to cut down on salt consumption. No literature has been found which indicates any tolerance or withdrawal effects of salt (except that the extreme condition of being completely unable to retain sodium ie adrenalectomy or equivalent as lethal).

Although three of the seven criteria of substance dependence could be contrived to be indicators of salt dependence, the essence of the definition is that these features of addiction are the result of the use of a substance ("A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three of the following .."). In the case of sodium depletion, the resultant overconsumption is actually not a result of the use of the substance (salt), but rather a deprivation of the substance.

For argument's sake consider the case of overconsumption of salt occurring from a pattern of high salt consumption, rather than depletion.

Tolerance: There is no physiological need for an increased amount of sodium, Beauchamp' s (1987) study showed that humans given an extra 10g/day of untasted salt did not increase preferences for higher salt intensities; where as those who tasted the extra 10g/day did. Thus this salt appetite is an hedonic preference rather than a physiological need. There is no apparent evidence of a diminished physiological effect with continued consumption of salt.

Withdrawal: Completely going without salt would certainly increase the physiological salt appetite, as it would be dangerous. However, there is no evidence that reducing levels of salt consumption to an amount required by the body (ie no longer overconsuming) causes a withdrawal syndrome, as would be the case in reducing levels of drug use.

Loss of control (consuming substance in larger amounts and over a longer time than was intended): There is no evidence that overconsumption of salt, without depletion, is governed by a loss of control. Again, referring to Beauchamp's (1987) study, increasing consumption of salt does not always lead to later consuming higher amounts of salt (in the case of untasted salt).

There is no evidence of a persistent desire to cut down on salt by those who over-consume; nor that a great deal of time is spent in obtaining, using or recovering from the effects of salt; nor that social, occupational, or recreational activities are reduced due to salt. Salt consumption may remain high despite knowledge of a health risk in some people, as preferences for lower amounts of salt take 2-4 months to become enduring (Capaldi, 1996), but it would seem that this is not too difficult to achieve, as Beauchamp (1987) had subjects self-maintaining a low-salt diet over a six-month period for a study. Shepherd, Farleigh, and Wharf (1989) found that subjects who reported adding table salt to foods, failed to compensate completely for the lack of salt in foods with the amount of table salt they freely added, and hence easily coped with less salt than the usual preference (1.4 g versus 5.09 g of salt usually).

In conclusion - The overconsumption of salt following a pattern of high-salt diet cannot be construed as having an underlying mechanism of addiction. The physiological appetite for salt cannot be regarded as an addictive process either, as substance dependence is a result of the use of a substance, not the depletion of a substance in the body.

Appendix

DSM-IV criteria for Substance Dependence.

(APA, 1994)

Substance Dependence or addiction is defined by the DSM-IV (APA, 1994) as follows-

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as' manifested by three (or more) of the following, occurring at any time in the same 12-month period.

· Tolerance, either (a) a need for a markedly increased amounts of the substance to achieve intoxication or the desired effect, or (b) markedly diminished effect with continued use of the same amount of the substance.

· Withdrawal, either (a) the characteristic withdrawal syndrome for the substance, or (b) the same or closely related substance is taken to relieve or avoid the withdrawal symptoms.

- *The substance is often taken in larger amounts or over a longer period than was intended*
- *There is a persistent desire or unsuccessful efforts to cut down or control substance use*
- *A great deal o£ time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects*
- *Important social, occupational, or recreational activities are given up or reduced because of substance use*
- *The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.*

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