# 22. Sodium

## Physiology [1,2]

Sodium is the principal cation in extracellular fluid. Its physiological roles include the maintenance of (i) extracellular fluid volume (ECF), which is related closely to total body sodium content, (ii) extracellular fluid oncotic pressure, (iii) acid base balance, (iv) electrophysiological phenomena in muscle, neuromuscular and nerve impulse transmission, and (v) generation of transmembrane gradients essential for the energy-dependent carrier-mediated uptake of nutrients and substrates by cells, including hepatocytes and those in the intestinal mucosa and renal tubules.

A typical adult male has a total body sodium of 4 mol (92 g); of this 0.5 mol (11.5 g) is in the intracellular fluid at an activity concentration of 2 mmol/L (46 mg/L), and 1.5 mol (34.5 g) is sequestered in bone. About 2 mol (46 g) is in the ECF at a concentration of 135-145 mmol/L (3.1-3.3 g/L).

Net intestinal absorption of sodium occurs in the distal small intestine and colon: its concentration in the ECF is maintained by the kidneys. Daily 25 mol (575 g) sodium enters the glomerular filtrate; since daily dietary intakes (say 50-150 mmol; 1.15-3.45 g) represent only 0.2-0.6 % of this amount, almost all of this filtered sodium must be reabsorbed to maintain sodium homoeostasis.

Renal sodium reabsorption is highly efficient and adaptable. Regulation of the sodium content of the ECF is closely related to the systemic control of the ECF volume. If the body sodium burden is increased, water is also retained and ECF volume increases; conversely, if the body sodium burden falls the ECF volume decreases. The overall regulation of these changes is unclear [1]. Changes in ECF volume are detected by sensors of pressure and distension which are located in the cardiac atria and right ventricle, the pulmonary vasculature, the carotid arteries and the aortic arch. From these sensors afferent nerve pathways end in the medulla and hypothalamus. When ECF or blood volume falls retention is stimulated, sympathetic nervous activity increases, stimulating the nerves supplying the afferent renal arterioles to induce vasoconstriction and thereby producing a redistribution of renal blood flow which, by reducing glomerular filtration, increases sodium and water retention.

Additionally, sympathetic nervous stimulation of the juxtaglomerular apparatus increases production of renin. This in turn leads to an increase in circulating

angiotensin II, adrenal medullary secretion of noradrenaline and adrenaline, and pituitary release of adrenocorticotrophin (ACTH) and antidiuretic hormone (ADH). ACTH and angiotensin II induce adrenal cortical secretion of aldosterone and other mineralocorticoids which stimulate sodium retention and potassium loss by the kidneys and the distal bowel. Increased secretion of antidiuretic hormone promotes sodium reabsorption from the renal distal tubules and probably the colon.

Renal sodium excretion is increased by factors which include specific natriuretic hormone, and vasodilators, parathyroid hormone, prostaglandins and kinins. The adult kidney can regulate sodium excretion at between 0.5 and 10 % of its filtered load. Under normal circumstances virtually all dietary sodium is absorbed and daily urinary loss matches this amount closely after allowance is made for that which may be lost in sweat.

## Deficiency and excess

The role of higher sodium intakes in the pathogenesis of hypertension has generated much interest [3,4,5,6,7,8]. However other factors, such as low intakes of potassium (see chapter 23), little physical activity, mental stress, alcohol consumption, smoking and high body mass index, are contributory to the development of hypertension [4,5]. Indeed obesity is associated with an increased sensitivity to sodium-induced hypertension [9]. It has been suggested that absence of these adverse factors, rather than lower sodium intakes alone, is responsible for the lower blood pressures observed in economically less developed communities compared with those seen in developed societies. Nevertheless, after allowance is made for such confounders, a relationship still exists between urinary sodium excretion (assumed to be a marker of intake) and increasing blood pressure with age [4]. A recent meta-analysis of studies of the relationship between sodium intake and blood pressure strongly implies that the causal association has been underestimated [6,7]. When published epidemiological studies and clinical trials in economically advanced and non-advanced populations were analysed separately to minimise the socio-economic variables mentioned, there was apparent for both types of community, and amongst individuals within such communities, an association between sodium intake, and increasing systolic and diastolic blood pressures. This relationship was continuous, i.e. there was no threshold of sodium intake below which the effect did not exist: it affected all age groups and it increased progressively with age. This reanalysis then applied the regression equations of blood pressure against sodium intake derived from the initial pooled study to calculate for each age group the potential falls in blood pressure which would result from reductions in sodium intakes [8]. Since the major source of sodium in the diet is salt these predictions were compared with

observed falls in blood pressure arising from intervention trials of reductions of salt intake on blood pressure. For trials lasting five weeks or longer the predicted and observed reductions matched closely. From this it was deduced that dietary salt reduction would have an appreciable effect on reducing mortality from ischaemic heart disease and stroke [8].

## Requirements

### *Adults*

The determination of a meaningful PRI and Average Requirement for sodium intake is difficult and a range of intakes is proposed for adults.

Healthy adults maintain a sodium balance on intakes as low as 3-20 mmol/d (69-460 mg/d), and some healthy populations have customary daily intakes of less than 40 mmol (920 mg), possibly even as low as 10 mmol/d (230 mg/d) [3,4,6]. The latter intake requires maximal adaptation to conserve sodium; to allow for changes in physical activity and climate it is thought prudent to set a lower intake at 25 mmol/d (575 mg/d).

On the basis of current evidence intakes in excess of 200 mmol (4.6 g) sodium daily would be associated with a significant risk of high blood pressure, especially in older people [6,7,8]; ideally intakes should be lower than this to reduce or prevent hypertension and the attendant risk of cardiovascular and cerebrovascular disease in the population in general. On the basis of the recent analyses, it is proposed that an upper limit of 150 mmol/d (3.5 g/d) be set.

### *Children*

The evidence is insufficient to give any recommendations for children.

## Summary

*Adults:*

**Acceptable Range of Intakes** 575-3500 mg/d

**(25-150 mmol/d)**

## References


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