TOX/2013/31

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

POTASSIUM SALT REPLACERS IN VULNERABLE GROUPS

Background

1. The Department of Health (DH) has asked the Scientific Advisory Committee on Nutrition (SACN) to consider the current recommendations on potassium-based salt replacers in time for setting new Responsibility Deal salt targets at the end of the 2013. The aim of this is to reduce population salt intakes, thereby reducing blood pressure and the risk of stroke.

2. DH does not currently recommend the use of potassium-based salt replacers as a means of achieving salt reduction since their use would continue to maintain a higher salt flavour in food, when it is the general aim to gradually reduce salt in food products so that the palates of consumers become used to lower salt levels. In addition, it is also noted that increasing potassium levels in food could have potential adverse effects in some vulnerable groups including very young children, the elderly and individuals with kidney disease who could be at risk of hyperkalaemia due to immature or impaired kidney function. With regard to individuals with impaired kidney function, there are individuals with diagnosed problems, some of whom will have been advised to consume a low potassium diet and there will also be those whose problems have not been diagnosed and who might be adversely affected by increased levels of potassium in the diet.

3. Industry has been asking DH to reconsider this view as some producers would like to use, and may already be using, potassium chloride, potassium-based raising agents and other salt replacers to achieve sodium reduction in food. The products concerned are those where further salt reduction would not be possible by reformulation and includes items such as bakery goods (eg scones, scotch pancakes, crumpets) and meat products (eg sausages and bacon) largely where the sodium has a function such as a preservative or as a raising agent may as well as having flavouring properties. Potassium cannot totally replace sodium as it is considered to have a metallic aftertaste and it does not have the flavouring properties of sodium. It has been suggested that a maximum 25-30% of added sodium could be replaced by potassium.

4. Potassium chloride (E508) is a generally permitted food additive permitted in most processed foods but it can also be used as an ingredient without being subject to further regulation other than general food law. At present, potassium-based salt replacers and products containing potassium-based salt replacers are generally labelled with a warning that they contain potassium, but this is voluntary.

5. Due to the need for rapid advice on this issue, the secretariat have not reviewed all the available data but have attached the 2006 review by EFSA at annex A to provide the overall background and have summarised any new or key information in the paper below. Similarly, the numerous potassium supplementation trials, which investigate the potential beneficial effects of increased potassium intakes on blood pressure, have not been reviewed individually since the majority have not addressed the end points of concern which are markers of kidney function and serum potassium concentrations. However, some key papers and the findings of the relevant meta-analyses have been noted. Many of the available supplementation studies have measured urinary rather than serum potassium levels; this has not been considered in this paper since the population of concern is those with renal impairment, thus urinary potassium may not be informative in this group.

6. SACN are undertaking a risk-benefit consideration of the use of potassium-based salt replacers and have asked the COT to comment on the risk aspects of this. Members are asked to consider the possibility of adverse effects occurring in vulnerable groups from increased exposure to dietary potassium. The aim of this paper is to focus on adverse effects to the kidney and it should be noted that other endpoints have not been considered.

Serum/plasma potassium levels and their regulation.

7. Potassium is readily absorbed from foods.

8. The concentration of potassium in plasma is tightly regulated within a narrow concentration range of about 3.5-5 mmol/L (EFSA, 2005; Wang, 2004). The body is able to accommodate a high intake of potassium, without any substantial change in plasma concentration, by synchronised alterations in both renal and extra-renal handling, with potassium either being excreted in the urine or taken up into the cells. Thus the plasma or extracellular concentration of potassium does not give a clear indication of the body content of potassium. Both the renal and extra-renal mechanisms through which potassium homeostasis are achieved are complex and linked to the cellular handling of other minerals and water homeostasis. The main process through which the body content of potassium is regulated over extended periods of time is renal excretion. Most of the potassium which is filtered in the glomerulus is reabsorbed in the proximal tubule and loop of Henle. Regulated excretion is determined by the rate at which potassium is secreted in the distal tubule and collecting ducts. Healthy kidneys are able to adapt to high intakes of potassium by excreting potassium more rapidly.

9. The mechanisms which enable the body to cope with a wide range of potassium intake involve complex changes in the kidney, colon and muscle over the short and long term.

10. In response to a large increase in potassium intake, insulin mediated uptake into skeletal muscle (and probably liver) is increased. This transfer of potassium from the extra-cellular to the intracellular space minimises any rise in plasma potassium in

the short term. The potassium, which has been buffered by uptake into muscle, is released into the extra-cellular fluid during the post-prandial period and excreted through the kidney. There is a short term renal response to increased potassium in the diet, with stimulation of potassium secretion in the collecting duct within hours of a potassium rich meal.

11. The kidney responds to a sustained increase in potassium intake through a decrease in the absorption of potassium in the proximal tubules and adaptive changes in the collecting duct leading to prolonged enhancement of excretion. The combination of insulin mediated buffering in muscle and enhanced renal secretion in the short term, and more marked renal adaptive changes in the long term combine to ensure that plasma levels are maintained within narrow limits when potassium intake is increased. The uptake of potassium into muscle appears reduced in insulin resistant states such as obesity, and consumption of high fat diets. The EFSA NDA panel (2005) commented that presumably the capacity for muscle to hold potassium is finite and therefore if there is a sustained high intake of potassium, the ability to cope with the dietary intake will be determined by the maximal rate of renal excretion, plus any increase in loss through the distal colon. Colonic losses of potassium may achieve 10-20 mmol/day when glomerular filtration rates fall below 30 ml/min (from the normal 130 ml/min). Therefore the adverse effects of prolonged higher intakes of potassium are determined by a) local effects on the gastrointestinal tract, and b) metabolic effects determined by the maximum capacity for renal excretion, and to a lesser extent colonic excretion.

12. It has been argued (Gennari and Segal, 2002; Einhorn *et al*., 2009) that the elevated plasma potassium levels measured in individuals with impaired kidney function could be an adaptive response to promote potassium excretion.

Adverse effects of high potassium intakes

General adverse effects

13. Few adverse effects have been associated with excess potassium in the general population. However, in studies using potassium supplements, damage to the oesophagus has been reported as a result of the supplement causing physical damage as it moves through the gastrointestinal tract. In some cases this has only been detectable by endoscopy (Grimm *et al*., 1990). The severity of the effects observed may depend on the formulation of the supplement.

Hyperkalaemia

14. As noted previously, serum potassium is maintained over a narrow concentration range of 3.5-5 mmol/L. In comparison, the intracellular potassium concentration is 150 mmol/L. The intracellular to extracellular ratio (150:4 mmol/L) results in a voltage gradient across the cell membrane and plays a major role in establishing the resting cell membrane potential, particularly in cardiac and neuromuscular cells. Whilst changes in the large intracellular concentration would

have little effect on this ratio, even small changes in the extracellular concentration would have significant effects on this ratio, the trans-membrane potential gradient and thereby the function of neuromuscular and cardiac tissues (Schaefer and Wolford, 2005).

15. Hyperkalaemia is defined as serum blood potassium greater than 5.5 mmol/L. Hyperkalaemia and accompanying physiological changes can be further divided into minimal (5.5-6.5 mmol/L - minor electrocardiographic changes), moderate (6.6-8 mmol/L - ECG changes limited to peaking of T waves) and severe (>8 mmol/L - or any level with a widened QRS complex, atrioventricular block or ventricular dysrhythmia), However, it should be noted that the serious complications do not strictly correlate with a given potassium level and are related more to the rate of rise in the potassium level, the effect on cardiac conduction and the underlying cause of the hyperkalaemia (Schaefer and Wolford, 2005).

16. The organ systems affected by hyperkalaemia are cardiac, neuromuscular and gastrointestinal. Patients may complain of only vague feelings of not feeling well, gastrointestinal symptoms or generalised weakness. The most serious concern is impaired cardiac conduction with risk of sudden death from asystole or ventricular fibrillation. Neuromuscular signs and symptoms include muscle cramps, weakness, paralysis, paresthesia (tingling or numbness in the skin) and decreased deep tendon reflex (Schaefer and Wolford, 2005).

17. Usually, severe symptoms do not occur until potassium levels reach > 7 mmol/L and a rapidly rising level is more dangerous than a slowly rising level. The classic ECG changes associated with hyperkalaemia are well established. The earliest changes occur at concentrations of >6.5 mmol/L and are peaked on tented T waves which are most prominent in pre-cordial leads. With further rise in serum levels there is a diminished cardiac excitability manifested by flattening of the P-wave, PR interval lengthening and the eventual disappearance of the P wave. The QRS duration becomes prolonged, progressing to a sine wave appearance and finally ending in ventricular asystole and fibrillation with potassium levels of 8-10 mmol/L. Hyperkalaemia results from an imbalance of normal potassium handling, this can result from increased potassium loads, transcellular shifting of potassium, or decreased potassium elimination (Schaefer and Wolford, 2005).

18. In individuals with normal renal function, hyperkalaemia from excess potassium load is very uncommon. Possible causes include potassium supplement overdose, massive blood transfusion with hypoperfusion or accidental ingestion of potassium chloride crystals used in water softeners. Short term intakes of approximately 15 g/day potassium do not result in serum potassium levels being outside the normal range provided that fluid intake is sufficient and intake is spread over the day (Rabelink *et al*., 1990). In this balance study, 6 healthy volunteers aged 22-26 y were given 400 mmol (15.6 g) potassium in 4 equal meals, every 6 hours to investigate short term (72 hrs) and long term (20 d) potassium loading. Throughout the study, each meal was followed by an acute transient increase in plasma potassium and aldosterone and potassium excretion in the urine. Potassium balance was achieved in the second 24 hour period of loading. This was associated with

elevated plasma potassium, slightly negative sodium balance and stimulated plasma renin activity. At 20 days loading sodium loss had been compensated. Discontinuation of loading was followed by negative potassium balance lasting only 24 hours and by sodium retention lasting 72 hours. Mean plasma potassium was 3.75 mmol/L at days 1 and 2 of the control period, increasing to 4.25, 4.77, 4.17 and 4.22 mmol/L after 24h, 48h, 72h and 20 days respectively. In the de-adaptation period, levels had declined to 3.98 and 3.67 at 24 and 46 hours. These values were all in the normal range. However, in a study by Keith (1941) symptoms of increased T-wave ECG and paraesthesia of the hands and feet in parallel with marked or severe hyperkalaemia were observed within 2-3 hours in 2/7 apparently normal subjects given doses of 12.5 or 17.5g potassium chloride or potassium bicarbonate (6.5-6.8 g potassium).

19. The large majority of cases of hyperkalaemia (>80%) occur when potassium excretion is impaired by a medical condition or by the use of certain medications in a patient with some degree of underlying renal dysfunction. Dietary salt substitutes, potassium supplements, penicillin potassium therapy and drinking potassium softened water may also cause hyperkalaemia in the pre-disposed individual (Schaefer and Wolford, 2005).

20. Acute cases of toxicity are generally from deliberate or accidental overdose of potassium supplement tablets, but some relate to misuse of salt replacer products. Case reports of potassium toxicity are summarised in Table 1 below which has been expanded from the table included in EFSA (2005); in some case reports, the adverse effect of concern is gastrointestinal damage and bleeding rather than hyperkalaemia.

Table 1. Case reports of potassium toxicity

Subject	Symptoms	Dose	Comment	Reference
Acute				
Woman 62 y	Gastric distention, inflamed stomach, necrotic mucosal lining sloughed off.	94 g K as 300 KCL slow-release tablets	Suicide attempt.	Peters & van der Weef, 1998
Woman 52 y	Vomiting, sweaty, breathless, left ventricular failure, cyanosis, lung crepitations	0.63 g KCL x 20 (approx. 6.6 g K)	Bendrofluazide and phenylbutazone also taken.	Illingworth & Proudfoot, 1980
Man 26 y	Vomiting, fatal cardiac arrest	0.6 g KCL x 40 (approx. 12.5 g K)	Distalgesic also taken.	Illingworth & Proudfoot, 1980
Woman 32 y	Presented with diarrhoea, subsequently found dead.	47 KCl tablets (information on dose not available).		Wetli & Davis, 1978.
Boy, 2 mo	Listlessness, cyanosis, ceased breathing, fatal 28h later.	3 g KCl and 1.5g in breast milk on 2 subsequent days (approx. 1.56 and 0.78 g K)	KCl given after infant having colic.	Wetli & Davis, 1978.
Man 56 y	Hyperkalaemia, cardiac arrest after aortic valve replacement.	Potassium supplement 40 mmol (1.56g) after bicycle exercise test. Salt substitute 5.5 g/day 2 wk before test.	Existing heart disease. Digoxin, chlorthiazide. Low Na diet.	Hultgren *et al*., 1975
Man 58 y	Hyperkalaemia, cardiac arrest	Potassium supplement 40 mmol	Existing heart disease. . Low Na diet (1.5 g/day) 2 wks before. Moderate renal	Hultgren *et al*., 1975

		(1.56g) after exercise test.	dysfunction	
Man 53 y	Chest tightening, nausea, vomiting. Died of hyperkalaemia with asystole.	283 mmol (approx. 11 g K) as Nu salt.	Imipramine, beer also taken	Restuccio, 1992.
Infant 8 mo	Stiffness, eye rolling back, breathing difficulties, severe hyperkalaemia.	17.2 g S-alt substitute, equivalent to 26 mmol K/kg BW (approx. 0.66g K)	Mild upper respiratory infection causing emesis and diarrhoea.	Kallen *et al*., 1976
Man 52 y	Hyperkalaemia.	KCL solution, single oral dose, 32mmol (approx. 1.3 g)	Hypertension, hypoaldosteronism. Chlorthalidone taken. Low Na and K diet 3 days before	Perez *et al*., 1984
Man 49 y	Hyperkalaemia.	KCL solution, single oral dose, 47 mmol (approx. 1.8 g)	Diabetes mellitus, peripheral sensory neuropathy, hypoaldosteronism.	Perez *et al*., 1984
Child, 8y	Abdominal discomfort, vomiting, hyperkalaemia	KCl (as water softener crystals)- “handful” consumed		Mosely and Osborne, 2003
Repeat dose				
Woman 75 y	Shortness of breath, oedema, heart failure.	Salt substitute *ad lib* for 6 weeks	Previous myocardial infarction	Snyder *et al*., 1975
Patient	Near fatal hyperkalaemia.	Soup seasoned with salt substitute		Hoyt, 1986
Man 31 y	Ventricular tachycardia. Collapse due to myocardial infarction	5 g/day potassium supplements. Duration unknown.	Body builder. Subject also taking anabolic steroids, amphetamines and potassium sparing diuretics	Appleby *et al*., 1994
Woman 68 y	Nausea and abdominal cramps, stenosis of the small bowel, probably	X 10 mEq KCl tablets/day for several years (approx. 0.78g/day)	Hypertension treatment 50 mg hydrochlorthiazide.	Bronson & Gamellli, 1987

	caused by focal alteration.			
Man 63 y	Hyperkalaemia developed,	Salt substitute , 35 mmol (1.4g K) per ½ teaspoon. Duration not stated.	Existing cardiomyopathy.	McCaughan, 1984
Man 74 y	Cardiac arrhythmia, oedema, hyperkalaemia	Salt substitutes used liberally several days prior to diagnosis.	Chronic rheumatic valvular disease, digoxin, furosemide, spironolactone also taken.	Yap *et al*., 1976
2 men, 64 & 67y	Hyperkalaemia, loss of consciousness, vomiting	Salt substitute (approx. 70-133 mmol/day -2.7-5.2 g/day) > 1 week.	Hypertensive patients on Angiotensin Converting Enzyme inhibitors	Ray *et al*., 1999
Woman 29 y	Hyperkalaemia, cardiac arrest Post-hypoxic brain damage.	K-containing salt substitutes taken after period of diarrhoea as patient suspected hypokalaemia	Frusemide also taken	Schim van der Loeff *et al*., 1988
Boy 14 y	Hyperkalaemia, premature ventricular beats	Hydrosaline beverages approx. 5 g K/day for 2 months.	Football player	Parisi *et al*., 2002.
Man, 78y	Acute renal failure, hyperkalaemia	3-4L water containing 7mmol (0.27g) /L K per day. Equivalent to 0.82-1.1 g		Graves, 1998
Man, 65y	Dyspnea, muscle weakness, bradycardia, hypotension and acute respiratory failure	8 tsp/day (estimated to be 25.4 g) plus 2.34g K in a KCL supplement.		John, 2011
Man, 35y	Depressed alertness, bradycardia, hypotension, cardiac arrest	Intake estimated to be 8g/day from supplements and sports drinks		John, 2011

Man 38y	Abdominal distension, diarrhoea, faintness, tingling sensation, muscle weakness.	20g potassium citrate (approx. 7.25g K) in solution on empty after 3 wks on low K diet	Same effect produced the next day by the same dose, but after 9 mo on a normal diet, the same dose produced only discomfort and some diarrhoea.	Bedford, 1954
Woman 74y	Malaise, abdominal pain, bradycardia, asystole and required resuscitation. Serum potassium 9.2 mmol/L	Use of salt substitute doses unknown	Patient on maintenance haemodialysis	Doorenbos & Vermeij, 2003
Woman 74y	Mild renal insufficiency diagnosed serum potassium 5.7mm/L. 1 week later cardiac arrest required resuscitation. tachycardia, Serum potassium 9.7 mmol/L.	Use of salt substitute doses unknown	Nabumetone taken. Orange juice and bananas consumed	Pal *et al.*, 1995
Woman 69y	Fatal suicide	Consumption of liquid medication > 21g potassium.	Patient being treated for heart failure	Kaplan, 1969

Supplementation studies

21. As noted elsewhere, the majority of studies investigating potassium supplementation have not recorded changes in serum potassium levels or on renal function parameters. Studies where this has been done have been considered below.

22. In a randomised placebo-controlled study designed to establish whether supplemental potassium chloride reduced the need for anti-hypertensive medication, 142 men aged 45-68 y were given 96 mmol (0.27 g/day) potassium chloride while 147 were given a placebo (Grimm *et al*., 1990). The men were hypertensive and were on a restricted sodium diet and were followed for an average of 2.2 y after the withdrawal of their anti-hypertensive medication. Participants who were given supplemental potassium had significantly higher serum potassium levels and urinary potassium excretion (4.5 mmol/L and 42.5 mmol/8 hours) than those given the placebo (4.2 mmol/L and 20 mmol/8 hours) (P<0.001). However, this was within the normal range for serum potassium. Potassium intakes were not calculated in this study, but the 97.5% ile total potassium intake was estimated (by EFSA, 2005) to be 7-8 g/day calculated from baseline data on potassium excretion.

23. In a randomised, crossover study reported by Overlack *et al*, (1991)[1], 12 patients with essential hypertension were given 4.68 g K as potassium citrate or bicarbonate or placebo for 8 weeks. At the end of the trial, mean (SE) serum potassium was 4.33 (0.1) mmol/L in the treated group and 4.06 (0.1) mmol/L in the controls. These values are within the normal range.

24. Siani *et al*., 1987 conducted a 15 week RCT, in which 37 patients who had mildly increased blood pressure and normal dietary sodium intake received potassium supplements (1.87 g/day) or placebo. No significant change was found in plasma potassium, though urinary potassium was increased in the group that received the supplements. Dietary potassium was not assessed but participants were asked not to change their usual diet.

25. In a study by Bulpitt *et al* (1985) 33 patients with hypertension receiving drug treatment that included a normal diuretic[2] were given a 2.5 g supplement of potassium chloride (n=14) or placebo (n=19) for 3 months. Plasma creatinine[3] fell by 11% in the treatment group compared to a 6% rise in the control group (P<0.05). Mean creatinine levels changed from 94 ± 6 to 84 ± 5 µmol/L and 104 ± 8 to 110 ±9 µmol/L in the supplement and placebo groups respectively. When analysed within patient, the fall in plasma creatinine remained significant; at 3 months creatinine

[1] The details are taken from the abstract and from Dickinson et al., 2006 as the full paper has not been obtained.

[2] Most diuretics encourage potassium secretion and thus increased loss in the urine. Potassium sparing diuretics do not have this effects, hence the risk of hyperkalaemia associated with these.

[3] Increasing serum creatinine levels can indicate kidney damage.

clearance was lower in the supplemented group but this was not significant. Plasma potassium increased from 3.7 mmol/L at baseline to 3.8 mmol/L in the treated group and decreased to 3.5 mmol/L in the placebo group. The difference between the two groups at the end of the study was significant but the plasma concentrations were within the normal range. It was suggested that the fall in serum creatinine could be partly due to water retention since weight increased by a mean of 1.4 kg in the treated group. However it was also suggested that the supplements could be restoring depleted potassium levels and improving the glomerular filtration rate (GFR).

26. A double blind randomised, placebo controlled crossover study was conducted by Patki *et al* (1990). Patients (n=37) with mild hypertension received placebo or 2.34 g potassium alone or 2.34 g potassium with 20 mmol (0.49 g) magnesium for 3 x 8 weeks with a two week washout period between each treatment. At baseline, mean (SD) serum creatinine was 76.02 (11.44) µmol/L. At day 56 the levels were 75.14 (14.15), 73.38 (6.96) and 70.72 (10.06) µmol/L in the placebo, potassium and potassium plus magnesium groups respectively. The changes were not significantly different. Serum potassium was 3.6 (0.42) mmol/L at baseline changing to 3.6 (8.4), 3.7 (8.5) and 3.8 (8.6) in the placebo, potassium, and potassium plus magnesium groups respectively at day 56. Urinary potassium was significantly increased in the two treatment groups compared to the controls but was within the normal range.

27. Twenty patients with mild or moderate essential hypertension who were not receiving other drug treatment and who were moderately restricting their sodium intake were included in a double blind, randomised, placebo controlled crossover study to compare one month's treatment with potassium supplements to placebo (Smith *et al* ., 1985). Mean urinary potassium excretion increased with treatment but plasma potassium did not significantly change with mean (SEM) being 4.0 (0.1), 4.1 (0.1) and 3.9 (0.1) mmol/L with sodium restriction only, potassium supplementation or placebo respectively. Plasma creatinine was also unaffected with mean (SEM) being 91 (4.0), 89 (3.8) and 91 (3.2) µmol/L with sodium restriction only, potassium supplementation or placebo respectively.

Vulnerable groups

28. Elderly people may be more vulnerable to potassium toxicity due to reduced physiological reserve in renal function (Beck, 1998). Ageing is associated with a progressive loss of kidney volume and GFR fall with each decade. This decline and changes in, for example, renin release, leads to decreased capacity for potassium secretion and thus limits the ability to handle large potassium loads. The elderly are therefore more vulnerable to increased intake from the diet and/or supplements or due to drugs that affect potassium balance.

29. Individuals using certain drugs, such as potassium sparing diuretics, β-adrenergic blockers, Angiotensin Converting Enzyme (ACE) inhibitors, digitalis, non-

steroidal anti-inflammatory drugs (NSAIDs) may also be vulnerable to increased potassium levels. As noted above, these drugs are more likely to be used by older people, increasing their risk of hyperkalaemia as well as declining kidney function.

30. Infants may also be vulnerable to excessive potassium due to limited excretion and immature function (EFSA, 2005 quoting EVM, 2003). Several aspects of kidney function vary considerably in the first year of life and differ markedly from the equivalent values in the adult. GFR increases little, prior to the time an infant reaches the conceptional age of 34 weeks, the point in renal development at which the absolute GFR increases gradually to mature values when linear growth is completed during adolescence (Arant, 1987) . GFR corrected for body size is not comparable with adult normal values until after 12 months of age. Further details are given by DeWoskin and Thompson (2008) who noted that in full term neonates (< 1 month) GFR was about 30% of the adult level, subsequently approaching adult levels between 6 months and 1 year of age. Tubular secretion is around 25% of adult rates at birth and increases more slowly and variably than GFR not approaching adult rates until 1-5 years if age. Limited data on renal plasma flow indicate neonatal rates of only 10-20% of adult values that rapidly increase to 50% by 6 months and then approach adult levels by 1-2 years of age.

31. Although infants may be more susceptible to excess potassium, they may be less likely to consume foods in which salt-replacers might be used since these are likely to occur in processed foods and in foods such as bacon which would be unlikely to be consumed by young children. The use of additives in foods designed for children is restricted and parents are advised not to use salt in family foods given to children. The exception to this could be raising agents as young children could consume products such as scones or crumpets containing these.

Renal disease and potassium intake

32. Chronic Kidney Disease (CKD) is divided into a number of stages, according to the level of kidney damage and the ability of the kidney to filter blood, as below:

CKD stage	GFR[4] (ml/min/1.73 m^2)	Description
1	≥ 90	Normal or increased GFR, but with other evidence of kidney damage.
2	60-89	Slight decrease in GFR, with other evidence of kidney damage.
3A	45-59	Moderate decrease in GFR, with or without other

[4] GFR over 90 (generally 90-110) ml/min is considered to be normal unless there is other evidence of kidney disease.

3B	30-44	evidence of kidney damage.
4	15-29	Severe decrease in GFR, with or without other evidence of kidney damage.
5	<15	Established renal failure.

33. In a recent NHS Kidney Care report, it was noted that the quality and outcomes framework (QOF) register indicate that in 2009-10, 1,817,871 adults in England had stages 3-5 CKD, a diagnosed prevalence rate of 4.3% of the population over the age of 18 (NHS Kidney care, 2012). It is likely that the total prevalence is higher as there are thought to be a substantial number of undiagnosed cases of CKD in the population. Comparing data from health surveys with the number of individuals on the QOF register indicates that between 900, 000 – 1.8 million individuals may have undiagnosed stage 3-5 CKD (2.1-4.3%). There are no accurate time series data from England but it seems likely that the prevalence could be rising due to the ageing population and the increasing incidence of conditions such as obesity, type 2 diabetes and hypertension which are associated with kidney disease. This increase would be consistent with the findings of NHANES studies in the US. These data apply to England but it seems likely that the findings would be comparable elsewhere in the UK. Data from the Quality Improvement in CKD (QICKD) study quoted in the report estimated a prevalence rate of 5.41% of the population and thus a total of 2.81 million people with stage 3-5 CKD, 97% (2.73 million) of those affected were at stage 3. Data from the renal transport register quoted in the NHS Kidney care report indicate that in 2009, 40,962 individuals in England were receiving renal replacement therapy (either receiving haemodialysis or having received a transplant).

34. However, not all individuals with renal disease need to have a low potassium diet. It has been noted that patients are unlikely to need to restrict potassium intake until renal function is less than 40% of normal (WHO, 2009) which would be these individuals in categories 4-5, (some in 3B) however, this is still likely to be many thousands of individuals not all of whom will have been diagnosed as having CKD (3% of 2.8 million (the individuals not at stage 3 or below-see above), being 84, 000). However, the American Heart Association (Appel *et al*., 2006) state that individuals with a GFR of 60 ml/min or below (CKD stages 3-5) would be advised to restrict their potassium intake, suggesting that the numbers involved could be a lot higher. Gennari and Segal, (2002) noted that the incidence of hyperkalaemia in chronic kidney disease was difficult to assess because of the almost universal use of drugs which affect plasma potassium. In a random sample of 300 patients with serum creatinine levels of 1.5 -6 mg/dl taken from their clinic, excluding individuals with diabetes or those taking diuretics or ACE inhibitors, an incidence of hyperkalaemia (> 5 mmol/L) of 55% was found in the remaining sample (n =18). Based on these limited data, hyperkalaemia was as likely to occur in individuals with glomerular disease as those with tubulointerstitial disease. The authors argue that hyperkalaemia is potentially an adaptive response to promote urinary potassium excretion and needs further investigation.

35. To assess the frequency of hyperkalaemia, Einhorn *et al*., 2009 conducted a retrospective analysis of 2,103 422 records from 245 808 US veterans with at least 1 hospitalisation and 1 inpatient or outpatient serum potassium record during 2005. A total of 66, 259 hyperkalaemic events occurred which represented 3.2% of the records analysed. The veterans were 95.6% male and 79.6% white, 19.4% African-American. The mean age of the veterans was 61y in those individuals without CKD and 73y in those that had CKD. When analysed by potassium level, 212,171 had serum potassium ≤ 5.5 mmol/L, with 75.2, 21.6 2.3 and 1 % of them having no CKD, stage 3, 4 or 5 CKD respectively. Of the individuals with serum potassium ≥ 5.5 mmol/L, with 46.0, 35.6, 10.5 and 8 % having no CKD, stage 3, 4 or 5 CKD respectively. The risk of hyperkalaemia was elevated in individuals treated with renin-angiotensin-aldosterone blockers. Individuals with cancer and diabetes were also more likely to have elevated serum potassium compared to individuals without the condition; ORs 1.16 (1.13-1.19) and 1.51 (1.47-1.55) respectively were calculated for these conditions. Compared to individuals without CKD the OR (95% CI) for elevated potassium were 2.24 (2.17-2.30) 5.91 (5.63-6.20) and 11.00 (10.34-11.69) in those with stage 3, 4 or 5 CKD respectively. The occurrence of hyperkalaemia also increased the odds of mortality within 1 day of the hyperkalaemic event but the odd ratios were higher in those without CKD. For moderate hyperkalaemia (≥5.5 -<6 mmol/L) the ORs of death were 10.32, 5.35, 5.73 and 2.31 for no CKD, stage 3, 4 and 5 respectively. Whereas for severe hyperkalaemia (>6 mmol/L) the ORs of death were 31.64, 19.52, 11.56 and 8.02 for no CKD, stage 3, 4 and 5 respectively

Potassium intakes – see Annex B

36. Important sources of potassium include potatoes, fruit, berries, vegetables, milk products (excluding cheese) and nuts. Potassium occurs in foods mainly associated with weak organic acids. Potassium is also found in mineral, spring and table waters, although the concentrations are very variable (EFSA, 2005).

37. In the UK, the Reference Nutrient Intake (RNI) for potassium is 3.5 g/day for adults based on ensuring optimal sodium metabolism (DH, 1991). The EU Recommended Daily Amount for potassium is 3.1-3.5 g/day.

38. The US Institute of medicine (IOM) recommendation for an adequate intake of 4.7 g was established to maintain reduced blood pressure, reduce the adverse effects of sodium chloride on blood pressure levels, reduce the risk of recurrent kidney stones and possibly decrease bone loss.

39. It has been suggested that, as a guide, CKD patients who need to restrict potassium intake should not exceed an intake 1 mmol (39 mg) K per kg body weight (personal communication). For a 70 kg adult, this would be equivalent to 2.73 g/day. A potassium intake of 50-75 mmol K is recommended for patients with clinically significant renal failure (Graves, 1998). This is equivalent to 1.95-2.93 g potassium.

40. Individuals on a low potassium diet avoid or have reduced consumption of foods high in potassium (including many fruits and vegetable) and use cooking techniques such as boiling and discarding cooking water to remove potassium from foods such as vegetables.

41. Data from the National Diet and Nutrition Survey (NDNS) –rolling programme published in July 2012 (Bates *et al*., 2012) show that mean potassium intakes in the UK were generally less than the recommended amounts, being 78 and 64% of the RNI in boys and girls aged 11-18 years respectively, whereas in adults aged 19-64, intakes were 91 and 74% of the RNI in males and females respectively (3.18 and 2.56 g). In adults aged 65+ intakes were 90 and 74% of the RNI in males and females (3.14 and 2.59 g). The mean intakes in males would be higher than 2.73 g maximum estimated for a 70 kg individual on a restricted potassium diet.

42. Detailed data on the increase in potassium intakes if there was wide-spread use as a salt replacer are not available. However, as an approximate estimate it has been assumed that all the food categories in which sodium could be replaced by potassium, have been replaced a maximum level of 25 %. This quantity has then been added to known potassium intakes from NDNs, this suggests that for children aged 1.5-3 there could be an increase in potassium intakes of up to 328 mg and for adults (19-64) there could be a potential increase of up to 593 mg potassium representing 267 and 99 % of the RNI respectively. For adults aged 65-74 and over 75, who would be more likely to have impaired kidney function, there could be an increase of up to 557 and 485 mg potassium representing 101 and 88% of the RNI respectively. The largest contributors to the increased potassium intakes are the cereal and cereal products group and the meat and meat products group.

43. It should be stressed that these calculations are likely to be a substantial over-estimate as it has been assumed that all the sodium in the foods (not just the added sodium) has been partially replaced with potassium at the maximum likely level of use and that mean levels of all of the relevant foods are being consumed.

44. There are no maximum intake levels of potassium to compare these values against; however, in adults the intakes do not reach or significantly exceed the RNI and in toddlers the % of the RNI increases from 226% to 267% of the RNI. However, as noted earlier, current estimated intakes of potassium in adults (as well as the RNI itself) would exceed an intake of 1 mmol/kg bodyweight, assuming a 70 kg adult, and the replacement of sodium with potassium would increase this exceedance.

45. The potassium intakes and the estimated increase in potassium intakes are given in Figure 1 below.

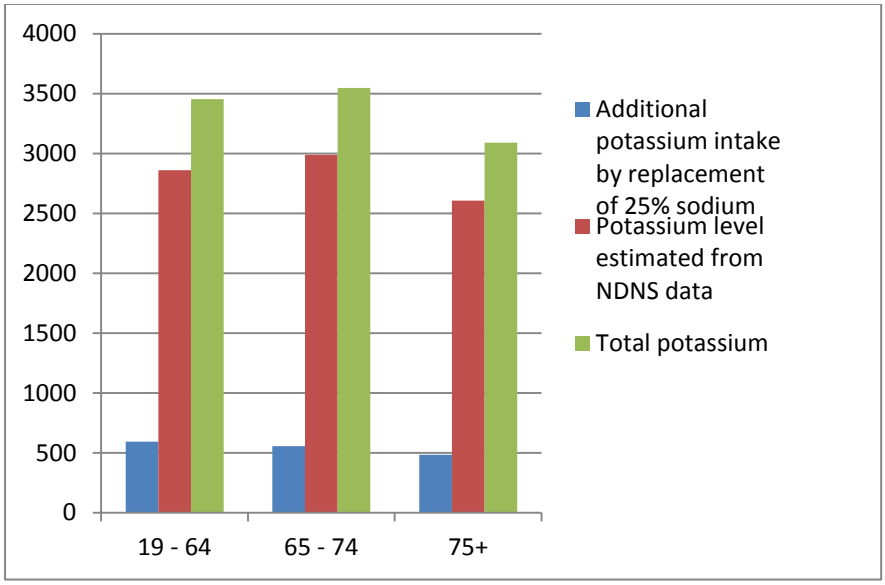


Figure 1: A comparison of potassium intakes among the three adult age groups (mg per person per day)

Previous assessments of potassium– Expert bodies

EVM

46. The Expert Group on Vitamins and Minerals (EVM, 2003) concluded that there were insufficient data to set a Safe Upper Level (SUL) for potassium, but noted that for guidance, supplements of up to 3.7 g/day potassium appeared to be without adverse effects but could be associated with gastro-intestinal lesions diagnosed by endoscopy. It was noted that patients with pre-existing hyperkalaemia, renal disease, acidosis, insulin deficiency or digitalis intoxication should not take potassium supplements without medical advice. A number of case reports of potassium poisoning from salt substitutes were noted.

EFSA

47. The EFSA panel on Dietetic Products Nutrition and Allergies (NDA) (EFSA, 2005) concluded that there were insufficient data to establish a Tolerable Upper Level of intake for potassium. However, it was noted that in subjects with reduced kidney function and reduced potassium excretion, doses as low as 1 g potassium in addition to food were associated with elevated plasma potassium and adverse heart effects[5]. They further noted that supplemental potassium doses of 5-7 g/day could cause adverse effects on heart function in apparently healthy subjects (this was based on the study by Keith *et al*., (1941)).

[5] It is unclear which reference this is referring to but it may be the study by Perez et al., 1984.

48. The EFSA panel stated that certain groups, particularly those with impaired kidney function, were sensitive to adverse effects of increasing potassium intake on heart function associated with increases of plasma potassium. These included subjects engaging in strenuous activities leading to dehydration, with diabetes mellitus, with impaired kidney function, on cardiovascular disease drug treatment or other metabolic disorders affecting potassium balance. Elderly people might be more vulnerable to adverse effects of potassium due to reduced kidney function or due to use of drugs affecting potassium balance. The EFSA panel commented that the available case reports "emphasize the potential risk of excessive use of salt substitutes and supplements, especially when used by those who are pre-disposed to retain potassium".

49. The effect of potassium supplements (particularly certain formulations) on the gastrointestinal tract was also noted.

US IOM

50. The US Institute of Medicine (IOM) did not make any recommendations on a Tolerable Upper Level of potassium intake, since they considered that intakes throughout the population were too low (IOM, 2005). However, it was noted that in individuals in whom urinary excretion of potassium was impaired, a potassium intake below the Acceptable Intake (AI) of 4.7 g/day (set on beneficial effects on blood pressure) was appropriate because of adverse cardiac effects (arrhythmias) from the resulting hyperkalaemia, but that such individuals were typically under medical supervision. Medical conditions that were associated with impaired urinary potassium excretion included diabetes, chronic renal insufficiency, end-stage renal disease, severe heart failure and adrenal insufficiency. Elderly individuals were at increased risk as they often had one or more of these conditions or were treated with medication that impaired potassium excretion. The health effects of potassium in infants were considered to be uncertain, so an AI was established based on a calculated mean potassium intake of infants fed human milk or human milk and complementary foods. For older children, AIs were set on the basis of energy requirements for children of different ages.

51. Based on studies by Textor *et al*., (1982) and Reardon *et al*., (1998) the IOM concluded that the AI would apply to healthy individuals on ACE therapy. These studies are described below:

52. Textor *et al*., (1982) reported a case series of 33 hypertensive patients. Ten patients with low levels of plasma renin activity had no change in aldosterone[6] excretion or serum potassium levels during the first week of ACE therapy. The 23 other patients had decreased average aldosterone excretion of 63% , associated with a rise in serum potassium from 3.6 to 4.4 mmol/L. Serum potassium levels during captopril therapy were inversely related to GFR (creatinine clearance) and

[6] Aldosterone is a mineral corticoid hormone which conserves sodium by encouraging sodium re-absorption and potassium secretion by the kidney, increasing water retention and maintaining blood pressure.

transiently exceeded 6 mmol/L in markedly azotemic[7] subects. IOM noted that serum potassium rarely rose to greater than 5 mmol/L unless the estimated GFR was less than 40 ml/min. In this study, patients consumed 2.7-3.1 g of potassium/day so it is unclear whether higher intakes would lead to hyperkalaemia. It was concluded that the risk of developing hyperkalaemia during ACE therapy increases as kidney function declines.

53. In a case-control study of 1,818 medical out-patients on ACE inhibitor therapy, severe hyperkalaemia (> 6 mmol/L) was uncommon in patients less than 70 years of age (Reardon *et al*., 1998). Data on potassium intakes was not collected in the study, though IOM considered it was likely that many of the patients would have intakes below the AI.

WHO 2012

54. The health effects of potassium in adults were reviewed in 2012 by WHO (2012a) who noted that lower potassium intake had been associated with elevated blood pressure, hypertension and stroke and higher levels of consumption could be protective against these conditions. Public health interventions to increase the potassium intake from food could be a cost- effective measure for reducing the burden of mortality from non-communicable diseases. However, the WHO also noted that the evidence for a potential beneficial effect on blood pressure and cardiovascular disease was not entirely consistent, with the available meta-analyses providing different results and without adverse effects being considered. Therefore, to inform the development of a guideline on potassium consumption, WHO commissioned a review on the effects of potassium intake, to compile results from studies in apparently healthy adults and children.

55. The review in adults (also published as Arbuto *et al*., 2013) considered RCTs only, 23 studies were included in the analysis, involving a total of 1606 participants and contributing 22 comparisons between potassium supplementation and a corresponding control group. The primary outcome measures were blood pressure (systolic, diastolic or both) and renal function. As secondary outcome measures, any other outcomes reported in the study were noted. Adverse effects were considered; these included increased total cholesterol, LDL, HDL, triglycerides, increased adrenaline or noradrenaline, or other adverse effects as reported. Changes in serum potassium levels were not reported. The studies involved intervention of at least 4 weeks duration and excluded studies where there was a concomitant intervention unless this was also conducted in the controls. Studies were considered in apparently healthy populations who may have been at risk of, or have had, hypertension, were known to have hypertension or were known to have normal blood pressure. Studies were excluded where they targeted those who were acutely ill, infected with human immunodeficiency virus or hospitalised.

[7] Azotaemia is the accumulation of nitrogenous products in the blood.

56. Three studies measured renal function by measuring serum creatinine (Bulpitt *et al*., 1985; Patki *et al*., 1990; Smith *et al*., 1985 – see paragraphs 24-26). These reported a non-significant decrease in serum creatinine of 4.86 µmol/L with increased potassium intake. The evidence that increased potassium intake did not affect renal function was considered to be of high quality.

57. The adverse effects were discussed in Arbuto *et al*., 2013. It was noted that increased potassium intake had been shown to be safe in people without renal impairment, but that in individuals with impaired urinary potassium excretion there could be a risk of hyperkalaemia. However, the risk was confined to those patients, who were largely under medical supervision and who were excluded from the review. It was noted that potassium intakes of 400 mmol (15.6 g)/day from food for several days or 115 mmol (4.49 g)/day for up to year were not associated with adverse effects (the studies concerned were Rabelink *et al*., 1990 and Siani *et al*., 1991). The authors further noted that none of the studies in the review reported increased side effects, minor complaints or major adverse effects in the increased potassium groups compared with the controls.

58. A review was also conducted in children, using the same inclusion criteria and an outcome measure of blood pressure (WHO, 2012b). Again, adverse effects were considered as an outcome; these included increased total cholesterol, LDL, HDL, triglycerides, increased adrenaline or noradrenaline, or other adverse effects as reported. Four studies were included in the meta-analysis, 2 RCTs, one non-randomised trial and one cohort study. However no studies meeting the inclusion criteria reported blood lipid or catecholamine levels, or, monitored adverse effects.

59. WHO recommended that potassium intake from food should be increased to reduce blood pressure and risk of cardiovascular disease, stroke and coronary heart disease in adults (this was considered to be a strong recommendation where the desirable effects of adherence outweigh the risks) (WHO, 2012c). The WHO further recommended a potassium intake of at least 90 mmol/day (3.51g/day) for adults. This was stated to be a conditional recommendation where the desirable effects of adherence probably outweigh the risks but that the group were not confident of the trade-off. For children, WHO made a conditional recommendation that the recommended potassium intake should be adjusted downwards based on the relative energy requirements of children and that individual countries should determine the requirements for the various age categories. It was noted that the recommendation did not apply to infants during a period of exclusive breastfeeding (0-6 months) or to the period of complementary feeding and continued breast feeding.

American Heart Association.

60. The American Heart Association also recommended a potassium intake of 4.7 g/day (Appel *at al*., 2006). They also agreed that a level lower than this would be appropriate for individuals with impaired potassium excretion who could be at risk of adverse cardiac effects (arrthymias) from hyperkalaemia. They noted that the available evidence was insufficient to identify the level of kidney function at which individuals with chronic kidney disease were at risk of hyperkalaemia, but noted that

an expert panel (the Kidney Disease Outcomes Quality Initiative) had recommended that individuals with stage 3 or 4 chronic kidney disease (GFR < 60 ml/min) should restrict their potassium intake.

Published reviews or meta-analyses

Cochrane reviews

61. A Cochrane review (Dickinson *et al*., 2006) evaluated the effects of potassium supplements and health outcomes and blood pressure in people with high blood pressure. The 5 included studies involved 425 participants with 8-16 weeks of follow up. It was noted that of the studies included in the analysis, only 2 (Overlack *et al*., 1991; Siani *et al*., 1987) reported blood potassium levels and when these were included in a meta-analysis, serum potassium was higher at the end of the study in the treated group compared to the controls (mean difference 0.20 mmol/L) but still within the normal range. Renal effects were not considered. It is not stated, but from the descriptions of the studies it appears that they were conducted in apparently healthy populations. It has been noted (nutrition evidence.com, 2006) that the inclusion criteria for this study were very restrictive.

Other meta-analyses

62. Earlier authors (Geleijnse *et al*., 2003; Whelton *et al*., 1997) had conducted meta-analyses investigating the relationship between potassium supplementation and blood pressure. These have included more studies than the Cochrane review, but have not considered adverse effects of high potassium intakes on either serum potassium or renal parameters.

Summary and discussion.

63. Potassium-based salt replacers (potassium chloride and potassium-based raising agents) have not previously been recommended as a means of reducing salt levels because there were concerns that the increased intake could increase the risk of hyperkalaemia and subsequent cardiac problems in individuals with reduced or impaired renal function, particularly as many individuals with kidney disease may not have been diagnosed. Other vulnerable groups include the elderly, as renal capacity diminishes with age, young children and individuals taking drugs which reduce potassium excretion such as ACE inhibitors and potassium sparing diuretic (these medications are more likely to be taken by older people.

64. Potassium is readily absorbed from food and the excess excreted in the urine. Serum potassium levels are tightly regulated within a narrow concentration range and the difference in concentration between intracellular and extracellular potassium results in a voltage gradient across cell membranes, helping to establish the resting cell membrane potential, particularly in cardiac and neuromuscular tissue. When serum potassium levels increase through impaired excretion, hyperkalaemia and

subsequent adverse physiological changes can occur, potentially leading to cardiac arrest. There are numerous case reports of potassium toxicity from supplements and salt substitutes in individuals with renal impairment.

65. The beneficial effects of potassium on blood pressure have been assessed in a number of trials but these have not generally assessed relevant endpoints such as serum/plasma potassium levels or renal endpoints such as serum creatinine levels. Where these have been measured, no adverse effects have been reported. The majority of such studies have also been conducted in healthy populations (or those with some degree of hypertension). It is therefore unclear whether any adverse effects would have been detected since the most at risk populations would not have been included.

66. At present intakes of potassium in the UK are lower than the current recommendations. As an approximate estimate of intake, it has been assumed that 25% of added salt had been replaced with potassium (real data have been used where appropriate) and the increase has been added to the current potassium intake. This suggests that potassium intakes could increase by a maximum of 557 mg potassium in adults.

Questions for the Committee

67. The committee are asked:

a) How much potassium could be consumed by either the normal population, by those with kidney impairment or by other vulnerable groups without a significant risk of adverse effects?

b) Whether the estimated increase in potassium from the use of salt-replacers is of concern to the normal population, to those with kidney impairment or to other vulnerable groups?

c) Whether they have any comments on the implications of increased potassium intakes for individuals with undiagnosed kidney impairment?

d) Whilst this review has largely focussed on individuals with impaired kidney, function, should the other vulnerable groups, notably very young children, be considered any further?

e) Any other comments they may have?

Secretariat

June 2013

Glossary

ACE- Angiotensin Converting Enzyme

AI- Adequate Intake

BW- body weight

CKD – Chronic kidney disease

DH- Department of Health

ECG Electrocardiogram

EFSA- European Food Safety Authority

EVM- Expert Group on Vitamins and Minerals

g -grams

GI - Gastrointestinal

HDL- High density lipoprotein

NSAID- Non Steroidal Anti-Inflammatory Drugs

IOM – Institute of Medicine

K-potassium

KCl- potassium chloride

L-litre

mmol- millimoles

mmol/L- millimoles/Litre

NDA- EFSA Panel on Dietetic Products, Nutrition and Allergies

NDNS- National Diet and Nutrition Survey

NHANES-National Health and Nutrition Examination Survey

nmol/L- nanomoles/Litre

QICKD – Quality Initiatives in Chronic kidney disease

QOF-Quality and Outcomes Framework

RCT- Randomised Controlled Trial

RNI- Reference Nutrient Intake

RDA- Recommended Daily Amount

SACN- Scientific Advisory Committee on Nutrition

SD- standard Deviation

SEM- Standard error of the mean

TUL- Tolerable Upper Level

UL - Upper Level

US-United Sates

WHO- World Health Organisation

y- years

REFERENCES

Aburto NJ, Hanson S, Gutierrez H, Hooper L, Elliott P & Cappuccio FP. 2013. Effect of increased potassium intake on cardiovascular risk factors and disease: systematic review and meta-analyses. *BMJ*; 346:f1378.

Appel LJ, Brands MW, Daniels SR, Karanja N, Elmer PJ, Sacks FM; 2006. American Heart Association Dietary approaches to prevent and treat hypertension: a scientific statement from the American Heart Association. Hypertension. 2006 Feb;47(2):296-308.

Appleby M, Fischer M, Martin M (1994). Myocarial infarction. Hyperkalaemia and ventricular tachycardia in a young male body-builder. Int J Cardiology 44: 171-174.

Arant BS (1987). Postnatal development of renal function during the first year of life. Pediatr Nephrol. 1:308-13.

Bates B, Lennox A, Prentice A, Bates C, & Swan G, eds. 2012. National Diet and Nutrition Survey. Headline results from Years 1, 2 and 3 (combined) of the rolling programme (2008/2009 – 2010/11). *Department of Health* http://transparency.dh.gov.uk/2012/07/25/ndns-3-years-report/

Beck LH (1998). Changes in renal function with aging. Clin Geriatr Med 14: 199-209.

Bedford PD (1954). Acute potassium intoxication. The Lancet, 267(6832):268-270.

Bronson DL and Gamelli RL (1987). Jejunal ulceration and stricture due to wax-matrix potassium chloride tablets and amitriptyline. J Clin Pharmacol 27: 788-789.

Bulpitt CJ, Ferrier G, Lewis PJ, Daymond M, Bulpitt PF, Dollery CT. (1985). Potassium supplementation fails to lower blood pressure in hypertensive patients receiving a potassium losing diuretic. Ann Clin Res.17:126-30.

Department of Health. 1991. Dietary reference values for food energy and nutrients in the UK. Report on health and social subjects 41. London: HMSO.

DeWoskin RS, Thompson CM. (2008) Renal clearance parameters for PBPK model analysis of early lifestage differences in the disposition of environmental toxicants. Regul Toxicol Pharmacol. Jun;51(1):66-86.

Dickinson HO, Nicolson D, Campbell F, Beyer FR & Mason J. 2006. Potassium supplementation for the management of primary hypertension in adults (review). *Cochrane Database of Systematic Reviews*, Issue 3. Art. No: CD004641. DOI:10.1002/14651858.CD004641.pub2.

Doorenbos CJ and Vermeij CG (2003). Danger of salt substitutes that contain potassium in patients with renal failure BMJ 326: 35-36.

EFSA 2005, Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Potassium (Request N° EFSA-Q-2003-018) The EFSA Journal (2005) 193, 1-19

Einhorn LM, Zhan M, Hsu VD, Walker LD, Moen MF, Seliger SL, Weir MR, Fink JC. (2009) The frequency of hyperkalemia and its significance in chronic kidney disease. Arch Intern Med. Jun 22;169(12):1156-62.

Expert Group on Vitamins and Minerals. 2003. Safe Upper Levels for Vitamins and Minerals. *Food Standards Agency*; http://cot.food.gov.uk/pdfs/vitmin2003.pdf

Geleijnse JM, Kok FJ, Grobbee DE (2003). Blood pressure response to changes in sodium and potassium intake: a metaregression analysis of randomised trials. J Hum Hypertens 17:471-480.

Gennari FJ, Segal *A.,* (2002). Hyperkalemia: An adaptive response in chronic renal insufficiency. Kidney International 62:1–9

Graves JW (1998). Hyperkalaemia due to a potassium based water softener. NEJM. 339: 1790.

Grimm RH Jr, Neaton JD, Elmer PJ, Svendsen KH, Levin J, Segal M, Holland L, Witte LJ, Clearman DR, Kofron P, *et al.* (1990). The influence of oral potassium chloride on blood pressure in hypertensive men on a low-sodium diet. N Engl J Med 322: 569-574.

Hoyt RE (1986). Hyperkalemia due to salt substitutes. JAMA 256: 1726.

Hultgren HN, Swenson R, Wettach G (1975). Cardiac arrest due to oral potassium administration. Am J Med 58: 139-142.

Illingworth RN and Proudfoot AT (1980). Rapid poisoning with slow-release potassium. Br Med J 281: 485-486.

IOM FNB (Food and Nutrition Board) (2004). Dietary Reference Intakes: water, potassium, sodium, chloride, and sulphate. Institute of Medicine. National Academy Press, Washington DC. USA.

John SK, Rangan Y, Block CA, Koff MD. (2011). Life-threatening hyperkalemia from nutritional supplements: uncommon or undiagnosed? Am J Emerg Med. Nov;29(9):1237

Kaplan M. (1969) Suicide by oral ingestion of a potassium preparation. Ann Intern Med. Aug;71(2):363-4.

Kallen RJ, Rieger CH, Cohen HS, Sutter MA, Ong RT (1976). Near-fatal hyperkalemia due to ingestion of salt substitute by an infant. JAMA 235: 2125-2126.

Keith NM, Osterberg AE, Burchell HB (1941). Some effects of potassium salts in man. Ann Int Med 16: 879-892.

McCaughan D (1984). Hazards of non-prescription potassium supplements. Lancet 1: 513-514.

McMahon FG, Ryan JR, Akdamar K, Ertan A (1984). Effect of potassium chloride supplements on upper gastrointestinal mucosa. Clin Pharmacol Ther 35: 852-855.

Mosely DS, Osborne B. (2003) Ingestion of potassium chloride crystals causes hyperkalaemia and haemorrhagic gastritis, Emergency medicine News, 25: 18-20.

NHS Kidney care 2012. Chronic Kidney Disease in England : the human and financial cost available from http://www.kidneycare.nhs.uk/resources_old/online_library/

Overlack A, Conrad H, Stumpe KO (1991). The influence of oral potassium citrate/bicarbonate on blood pressure in essential hypertension during unrestricted salt intake. Klin Wochenschr 69: 79-83.

Pal B, Hutchinson A, Bhattacharya A, Ralston A (1995) Cardiac arrest due to severe hyperkalaemia in patient taking nabumetone and low salt diet. BMJ. Dec 2;311(7018):1486-7.

Parisi A, Alabiso A, Sacchetti M, Di Salvo V, Di Luigi L, Pigozzi F (2002). Complex arrhythmia induced by overuse of potassium supplementation in a young male football player. Case report. J Sports Med Phys Fitness 42: 214-216.

Patki PS, Singh J, Gokhale SV, Bulakh PM, Shrotri DS, Patwardhan B (1990). Efficacy of potassium and magnesium in essential hypertension: a double-blind, placebo controlled, crossover study. BMJ. Sep 15;301(6751):521-3.

Peeters JWPM and van der Weef SDJ (1998). Gastric stenosis after potassium chloride ingestion. Endoscopy 30: S100-111.

Perez GO, Oster JR, Pelleya R, Caralis PV, Kem DC (1984). Hyperkalemia from single small oral doses of potassium chloride. Nephron 36: 270-271.

Rabelink TJ, Koomans HA, Hene RJ, Dorhout Mees EJ (1990). Early and late adjustment to potassium loading in humans. Kidney Int 38: 942-947.

Ray KK, Dorman S, Watson RDS (1999). Severe hyperkalaemia due to the concomitant use of salt substitutes and ACE inhibitors in hypertension: a potentially life threatening interaction. J Hum Hypertension 13: 717-720.

Reardon LC, Macpherson DS. (1998) Hyperkalemia in outpatients using angiotensin-converting enzyme inhibitors. How much should we worry? Arch Intern Med. Jan 12;158(1):26-32.

Restuccio A (1992). Fatal hyperkalemia from a salt substitute. Am J Emerg Med 10: 171-173.

Schaefer TJ, Wolford, RW (2005). Disorders of Potassium, Emerg Med Clin N. Amer., 23:723-747

Schim van der Loeff HJ, Strack van Schijndel RJM, Thijs LG (1988). Cardiac arrest due to oral potassium intake. Intensive Care Med 15: 58-59.

Siani A, Strazzullo P, Guglielmi S, Pacioni D, Giacco A, Iacone R, Mancini M. (1988). Controlled trial of low calcium versus high calcium intake in mild hypertension. J Hypertens. Mar;6(3):253-6

Smith SJ, Markandu ND, Sagnella GA, MacGregor GA. (1985). Moderate potassium chloride supplementation in essential hypertension: is it additive to moderate sodium restriction? Br Med J (Clin Res Ed). Jan 12;290(6462):110-3

Snyder EL, Dixon T, Bresnitz E (1975). Abuse of salt "substitute". N Engl J Med 292: 320.

Textor SC, Bravo EL, Fouad FM, Tarazi RC. 1982. Hyperkalemia in azotemic patients during angiotensin-converting enzyme inhibition and aldosterone reduction with captopril. Am J Med. Nov;73(5):719-25

Wang WH (2004). Regulation of renal K transport by dietary K intake. Annu Rev Physiol 66: 547-569.

Wetli CV and Davis JH (1978). Fatal hyperkalemia from accidental overdose of potassium chloride. JAMA 240: 1339.

Whelton PK and He J (1997). Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials. J Am Med Assoc 277: 1624-1632.

WHO (2009) Potassium in drinking–water. Background document for development of WHO *Guideline for Drinking-water Quality* WHO/HSE/WSH/09.01/7

WHO (2012a) Effects of increased potassium intake on blood pressure, renal function, blood lipids, and other potential adverse effects. WHO, Geneva. Obtainable from: http://apps.who.int/iris/bitstream/10665/79331/1/9789241504881_eng.pdf

WHO (2012b). Effects of increased potassium intake on blood pressure and potential adverse effects in children. WHO, Geneva. Obtainable from: http://apps.who.int/iris/bitstream/10665/79338/1/9789241504850_eng.pdf

WHO 2012 c Guideline: Potassium intake for adults and children, WHO, Geneva. Obtainable from: http://www.who.int/nutrition/publications/guidelines/potassium_intake_printversion.pdf

Yap V, Patel A, Thomsen J (1976). Hyperkalemia with cardiac arrhythmia. Induction by salt substitutes, spironolactone, and azotemia. JAMA 236: 2775-2776.

Annex A toTOX/2013/31

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

POTASSIUM SALT REPLACERS IN VULNERABLE GROUPS

Annex A

EFSA 2005, Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Potassium (Request N° EFSA-Q-2003-018) The EFSA Journal (2005) 193, 1-19

This paper can be downloaded from: http://www.efsa.europa.eu/en/efsajournal/doc/193.pdf

Secretariat

June 2013

Annex B toTOX/2013/31

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

POTASSIUM SALT REPLACERS IN VULNERABLE GROUPS

Current intakes of potassium from the diet and estimated changes to intakes from the use of potassium-based salt replacers.

Secretariat

June 2013

Potassium intakes from food and supplements

1. Potassium intakes were obtained from the National Diet and Nutrition Survey (NDNS), a programme of surveys designed to assess the diet, nutrient intake and nutritional status of the general population aged 1.5 years and over, living in private households in the UK. Since 2008 the NDNS has been a rolling programme for people aged 1.5 years and over.

2. Food consumption data (including supplements) and information on shopping and food preparation practices were collected from participants by means of a face-to-face Computer Assisted Personal Interview (CAPI) and completion of a four-day food diary. Portion sizes were estimated (not weighed) using household measures, weights from labels or photographs of 10 frequently consumed foods reproduced in the diary. Combined results from the rolling programme (2008/09 – 2009/10) for a sample of the UK population designed to be nationally representative is considered.

3. Potassium intakes were calculated using a set of nutrient composition data contemporaneous with the time of the survey. Therefore some apparent differences in intakes between population age groups may be due to changes in the nutrient composition data and reflect changes in the nutrient composition of manufactured foods over time.

Total intakes of Potassium

4. Intakes of potassium are reported in two ways: from foods only and from all sources, that is, including dietary supplements, as recorded in the four-day diary

5. Table 1 provides information on the absolute intakes of potassium by the UK population, classified by age and sex. Mean and median intake and the upper and lower end of the intake distribution (defined as upper and lower 2.5 percentiles respectively) are also given.

6. The average daily intake of potassium is also given as a percentage of the UK Reference Nutrient Intake (RNI)[8], by age and sex and the proportions of participants with intakes below the Lower Reference Nutrient Intake (LRNI)[9] is also shown. Table 2 gives the RNIs and LRNIs by sex and age.

[8] The RNI for a vitamin or mineral is the amount of the nutrient that is sufficient for about 97% of people in the group. If the average intake of the group is at the RNI, then the risk of deficiency in the group is judged to be very small. However, if the average intake is lower than the RNI then it is possible that some of the group will have an intake below their requirement.

[9] The adequacy of vitamin or mineral intake can be expressed as the proportion of individuals with intakes below the LRNI. The LRNI for a vitamin or mineral is set at the level of intake considered likely to be sufficient to meet the needs of only 2.5% of the population.

Table 2: Average daily intake of potassium (mg/day) and as a percentage of Reference Nutrient Intake (RNI) and the Lower Reference Nutrient Intake (LRNI), by age and sex *(aged 1.5 years and over; 2008/09 - 2009/10)*

			Boys		Men			Girls		Women					Total
	4-10	11-18	Total boys	19-64	65+	4-10	11-18	Total girls	19-64	65+	1.5-3	4-10	11-18	19-64	65+
Average daily intake of potassium from food sources only (mg/day)															
Mean	2222	2558	2410	3174	3139	2083	2120	2104	2558	2592	1807	2154	2345	2865	2832
Median	2195	2481	2335	3085	3046	2052	2127	2099	2542	2574	1762	2117	2275	2747	2753
sd	517	765	687	1052	940	496	574	541	737	626	519	511	713	958	823
Upper 2.5th percentile	3461	4332	3993	5773	5153	3212	3402	3299	3991	3900	2857	3299	3891	5128	4605
Lower 2.5th percentile	1321	1225	1245	1518	1268	1227	987	1164	1195	1504	971	1296	1078	1303	1465
Average daily intake of minerals from all sources (including dietary supplements) (mg/day)															
Mean	2222	2558	2410	3175	3143	2083	2120	2104	2560	2593	1808	2154	2345	2866	2834
Median	2195	2481	2335	3085	3046	2052	2127	2099	2554	2575	1762	2117	2275	2747	2753
sd	517	765	687	1053	943	496	574	541	738	628	520	511	713	959	826
Upper 2.5th percentile	3461	4332	3993	5773	5153	3212	3402	3299	4001	3900	2857	3299	3891	5128	4605
Lower 2.5th percentile	1321	1225	1245	1518	1268	1227	987	1169	1195	1504	971	1296	1078	1303	1465
Average daily intake of potassium from food sources only as a percentage of RNI (%)															
Mean	148	78	109	91	90	140	64	98	73	74	226	144	71	82	81
Median	138	77	95	88	87	127	65	79	73	74	220	133	69	78	79
sd	48	23	51	30	27	51	18	52	21	18	65	49	22	27	24
Average daily intake of potassium from all sources (including dietary supplements) as a percentage of RNI (%)															
Mean	148	78	109	91	90	140	64	98	73	74	226	144	71	82	81
Median	138	77	95	88	87	127	65	79	73	74	220	133	69	78	79
sd	48	23	51	30	27	51	18	52	21	18	65	49	22	27	24
Proportion of participants with average daily intakes of potassium from food sources only below the LRNI (%)															
	0	16	9	10	11	0	31	17	22	18	1	0	23	16	15
Proportion of participants with average daily intakes of potassium from all sources (including dietary supplements) below the LRNI (%)															
	0	16	9	10	11	0	31	17	22	18	1	0	23	16	15

Table 2: Reference Nutrient Intakes (RNIs) and Lower Reference Nutrient (LRNIs) for potassium (mg/d), by sex and age

Age group (years)							
		1-3	4-6	7-10	11-14	15-18	19-75+
Males							
	RNI	800	1100	2000	3100	3500	3500
	LRNI	450	600	950	1600	2000	2000
Females							
	RNI	800	1100	2000	3100	3500	3500
	LRNI	450	600	950	1600	2000	2000

7. Average intake of potassium from food was lowest for children aged 1.5 - 3 years and highest for males aged 19 - 64 years. The trend was for intake from food to increase with age until adulthood and decrease with age for older people aged 65 years and over. The contribution of supplements was very small and average intakes including supplements were similar to average intakes from food only.

8. Intakes from food only and all sources at the 97.5%ile were between 1.6 - 1.8 times the mean for all population groups. The mean intake of potassium from food sources and all sources for children aged 1.5 - 10 years exceeded the RNI for these groups. Mean intakes for young people aged 11-18 years, adults and people aged 65 years and over were below the RNI. The mean intakes of potassium for all age groups were above their respective LRNIs.

Sources of Potassium in the diet

9. Table 3 indicates the contribution made by different types of food to average intakes of potassium by different age groups. The main contribution to potassium (in all age groups) came from vegetables, potatoes and savoury snacks - the intake was 20 - 30% of the total potassium intake. Potatoes and potato products alone provided 12 - 23% of the total intake. This was followed by milk and milk products (11 - 32% of total potassium; this category includes cheese, yogurt and dairy desserts) and then cereal and cereal products (12 - 16%).

10. The main source of potassium for children aged 1½ - 3 years was milk and milk products (32%) followed by vegetables, potatoes and savoury snacks (20%).

The main sources of potassium were similar for people aged 19 - 64 and 65 years and over. Beverages contributed slightly more in these age groups.

Table 3: Contribution of food types to average daily intake of potassium

Food type	Contribution to potassium intake (mg/day)							% of total						
Age (years)	1.5 to3	4 to 10	11 to 18	19 to 49	50 to 64	65 to 74	75+	1 to3	4 to 10	11 to 18	19 to 49	50 to 64	65 to 74	75+
Cereal and cereal products	239	354	376	380	364	387	382	13%	16%	16%	14%	12%	13%	15%
- of which all breakfast cereals	*57*	*70*	*49*	*55*	*70*	*84*	*98*	*3%*	*3%*	*2%*	*2%*	*2%*	*3%*	*4%*
Milk and milk products	580	444	309	308	367	418	433	32%	21%	13%	11%	12%	14%	17%
- of which semi-skimmed milk	*123*	*162*	*146*	*146*	*170*	*169*	*206*	*7%*	*8%*	*6%*	*5%*	*6%*	*6%*	*8%*
Eggs and egg dishes	12	13	17	27	24	30	22	1%	1%	1%	1%	1%	1%	1%
Fat spreads	5	7	6	8	8	9	10	0%	0%	0%	0%	0%	0%	0%
Meat and meat products	122	230	359	409	356	352	296	7%	11%	15%	15%	12%	12%	11%
- of which chicken and turkey dishes	*43*	*87*	*141*	*164*	*103*	*104*	*69*	*2%*	*4%*	*6%*	*6%*	*3%*	*3%*	*3%*
Fish and fish dishes	39	52	41	78	122	133	129	2%	2%	2%	3%	4%	4%	5%
Vegetables, potatoes and savoury snacks	359	575	750	822	875	841	681	20%	27%	32%	29%	29%	28%	26%
- of which potatoes & potato products	*215*	*383*	*545*	*477*	*453*	*459*	*396*	*12%*	*18%*	*23%*	*17%*	*15%*	*15%*	*15%*
Fruits and nuts	260	210	146	207	307	342	269	14%	10%	6%	7%	10%	11%	10%
Sugar, confectionery and preserves	21	33	44	38	25	17	16	1%	2%	2%	1%	1%	1%	1%
Beverages	117	168	211	405	472	368	278	6%	8%	9%	15%	16%	12%	11%
Miscellaneous	52	67	88	110	94	92	88	3%	3%	4%	4%	3%	3%	3%
Total intake from food	1805	2153	2347	2793	3012	2989	2606	100%	100%	100%	100%	100%	100%	100%

Potassium intakes from supplements

11. A wide range of potassium supplements are available on the market both individually as well as a part of multivitamin/mineral formulations with daily doses ranging from 40 - 200 mg potassium. The counter ions are usually chloride, iodide, citrate, gluconate and amino acid chelates. The list of potassium salts that can be used in the manufacture of food supplements is set out in EC Directive 2002/46/EC as amended.

12. The number of consumers of dietary supplements containing potassium was very small and they provided zero or negligible contribution to mean intakes. The inclusion of prescribed supplements containing potassium had the effect of a very small increase on the average intake from all sources.

13. The use of supplements containing potassium was low in all population groups. Table 4 shows the number of consumers of dietary supplements containing potassium in each age group. The table also provides information on the mean and range of intakes of potassium from supplements for those who consumed them. The highest prevalence of potassium supplement use was in older people (aged 65 - 74).

Table 4: Potassium intake from supplements

Age/sex	Consumers of potassium supplements		Potassium intake from supplements (consumers only; mg/day)	
	Number (unweighted)	%	Mean	Range
Toddlers 1-3 yrs	3	1%	82	2 to 250
4 - 10 yrs	4	1%	4	1 to 16
11 - 18 yrs	3	1%	36	20 to 40
19 - 49	20	4%	34	2 to 60
50 - 64	14	5%	19	2 to 40
65 - 74	12	10%	41	5 to 175
75 +	2	1%	20	10 to 40

Potassium intake from drinking water

14. There is no WHO guideline value for potassium because the occurrence levels in drinking water are well below those that are of health concern. Consequently there is no EU or national standard and no recent monitoring results.

Under the former EU Drinking Water Directive (1980) there was a maximum acceptable concentration of 12 mg/L.

15. Levels of potassium in drinking water are low; reported mean levels range from 1.1 - 2.6 mg/L (Morton et al, 1979, Powell et al, 1986). The most recent data held dates from 2003, when the mean concentration of potassium in drinking water in England and Wales was about 2.9mg/L (DWI, personal communication). In a survey of bottled water, the levels ranged from <1 - 10.9 mg/l (Buckinghamshire County Council, 2005).

16. Assuming consumption of 2 litres of water daily, even at the highest level reported, drinking water would not make a significant contribution to potassium intake.

Replacement of sodium by potassium

17. In order to reduce intake of sodium from various sources in food, replacing common salt (sodium chloride) with potassium chloride has been considered. In addition, other sodium salts - for example in raising agents - could be substituted by their potassium equivalents. The effect of replacement of sodium salts in various categories of food by potassium salts on the total intake of potassium was modelled.

18. The industry has indicated that it is reasonable to assume that up to 25% of current salt levels could be replaced by potassium based salts. Although this might be an over estimation of potassium intakes (not all products would replace salt at 25%), this would be a worst-case scenario of increased potassium exposure from salt replacers.

19. This is a very approximate estimation where a quarter of the total sodium content of a particular food category is replaced by potassium. This additional potassium intake was then added on to the potassium intake for that food category as determined by the NDNS. Table 5.1 - 5.4 and figures 5.1 - 5.5 show the additional potassium from a 25% replacement of sodium and the resulting total potassium intake for various age groups.

20. It must be emphasised that these estimations are very approximate as there is not much actual data on potassium levels after replacement. This would very much depend on actual recipes and levels of substitution. The potassium intake for toddlers aged 1½ to 3 years could increase from 1805 mg/day (226% of RNI) to 2133 mg/day (267% of RNI). The increased intakes from potassium replacement for adults aged 19 - 65 years could bring it up to around the RNI (3500 mg/day). The intakes for adults aged over 75 years is slightly lower, with the proposed substitution estimated to be about 88% of the RNI.

Table 5.1: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for children aged 1.5 to 3

Food category	Current sodium level[10]	Additional potassium intake by replacement of 25% sodium[11]	Potassium level estimated from NDNS data	Total potassium	% of RNI
Cereal and cereal products	431	108	239	347	43
- of which all baked goods	*285*	*71*	*135*	*206*	*26*
- of which all breakfast cereals[12]	*54*	*14*	*57*	*70*	*9*
- of which pasta, rice and other cereals	*93*	*23*	*48*	*71*	*9*
Milk and milk products	228	57	580	637	80
- of which semi-skimmed milk	*33*	*8*	*123*	*131*	*16*
Eggs and egg dishes	24	6	12	18	2
Fat spreads	38	10	5	14	2
Meat and meat products	242	60	122	182	23
- of which chicken and turkey dishes	*39*	*10*	*43*	*53*	*7*
Fish and fish dishes	41	10	39	49	6
Vegetables, potatoes & savoury snacks	171	43	359	402	50
- of which potatoes & potato products	*77*	*19*	*215*	*234*	*29*
Fruits and nuts	14	4	260	263	33
Sugar, confectionery and preserves	9	2	21	23	3
Beverages	13	3	117	120	15
Miscellaneous[13]	100	25	52	78	10
Total intake from food	1311	328	1805	2133	267

[10] Sodium levels from both salt and non-salt sources.

[11] Assuming 0.25mg of potassium for every 1mg of sodium in food (including sodium not from salt).

[12] Baked goods comprise all types of bread, biscuits, buns, cakes, pastries, fruit pies & puddings.

[13] Miscellaneous foods include table salt, commercial toddlers foods and drinks, artificial sweeteners, soups, sauces, etc.

Figure 5.1: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for children aged 1.5 to 3

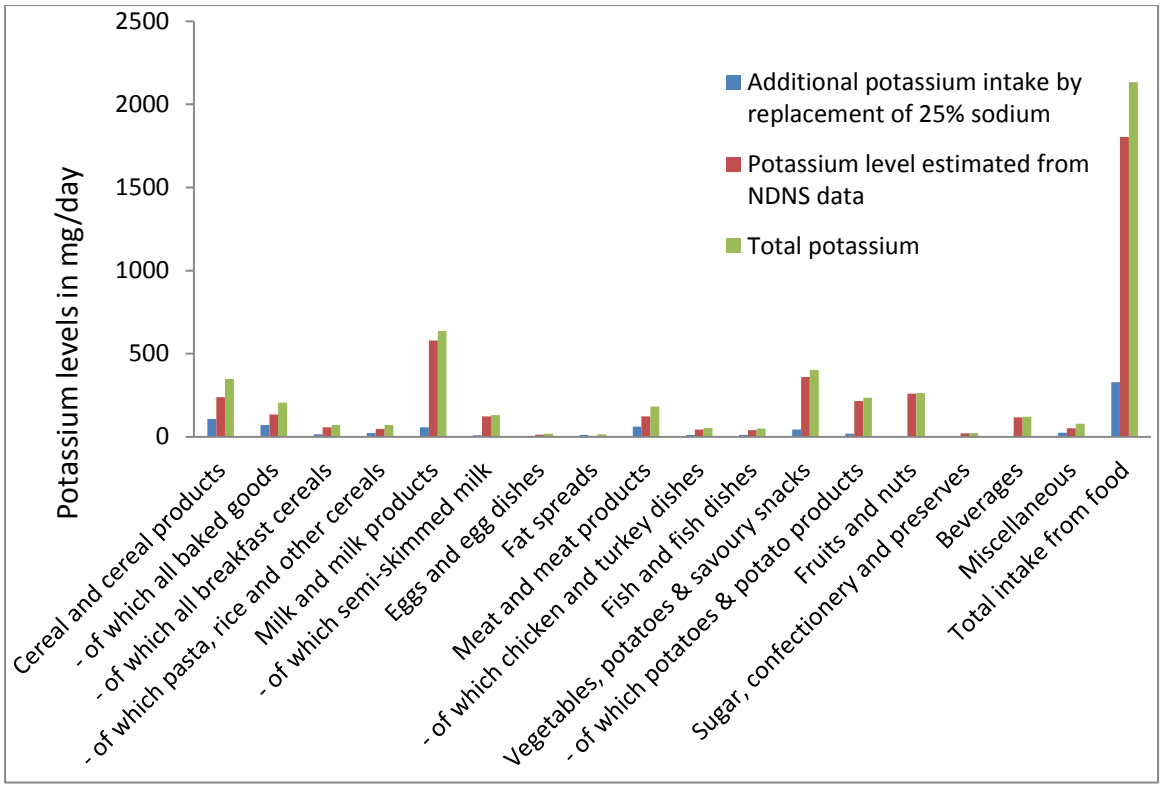


Table 5.2: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for adults aged 19 - 54

Food category	Current sodium level[14]	Additional potassium intake by replacement of 25% sodium [15]	Potassium level estimated from NDNS data	Total potassium	% of RNI
Cereal and cereal products	708	177	375	552	16
- of which all baked goods[16]	*521*	*130*	*235*	*366*	*10*
- of which all breakfast cereals	*54*	*14*	*59*	*73*	*2*
- of which pasta, rice and other cereals	*132*	*33*	*80*	*114*	*3*
Milk and milk products	208	52	326	378	11
- of which semi-skimmed milk	*42*	*10*	*153*	*164*	*5*
Eggs and egg dishes	47	12	26	38	1
Fat spreads	64	16	8	24	1
Meat and meat products	571	143	393	536	15
- of which chicken and turkey dishes	*94*	*23*	*145*	*168*	*5*
Fish and fish dishes	112	28	92	120	3
Vegetables, potatoes & savoury snacks	252	63	838	901	26
- of which potatoes & potato products	*92*	*23*	*469*	*492*	*14*
Fruits and nuts	22	5	237	243	7
Sugar, confectionery and preserves	12	3	34	37	1
Beverages	51	13	426	438	13
Miscellaneous[17]	324	81	105	186	5
Total intake from food	2370	593	2860	3453	99

[14] Sodium levels from both salt and non-salt sources.

[15] Assuming 0.25mg of potassium for every 1mg of sodium in food (including sodium not from salt).

[16] Baked goods comprise all types of bread, biscuits, buns, cakes, pastries, fruit pies & puddings.

[17] Miscellaneous foods include table salt, commercial toddlers foods and drinks, artificial sweeteners, soups, sauces, etc.

Figure 5.2: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for adults aged 19 - 54

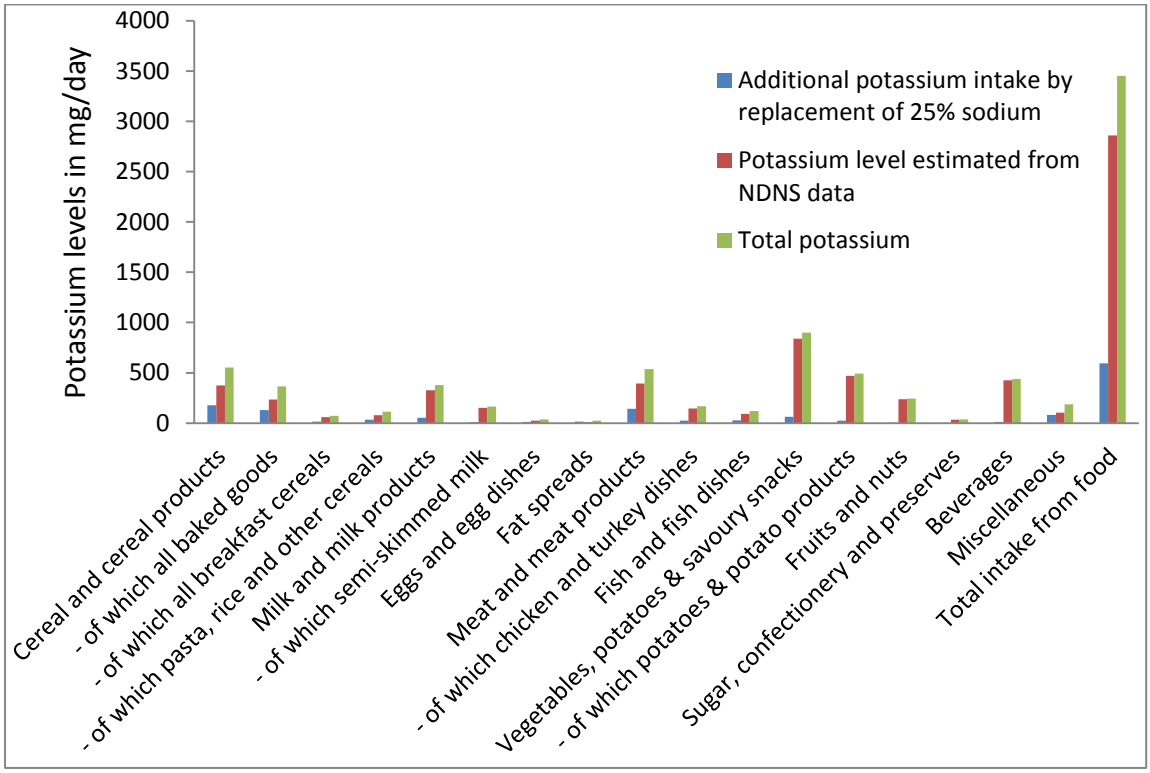


Table 5.3: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for adults aged 65 - 74

Food category	Current sodium level[18]	Additional potassium intake by replacement of 25% sodium[19]	Potassium level estimated from NDNS data	Total potassium	% of RNI
Cereal and cereal products	707	177	387	563	16
- of which all baked goods[20]	*565*	*141*	*270*	*411*	*12*
- of which all breakfast cereals	*94*	*24*	*84*	*108*	*3*
- of which pasta, rice and other cereals	*48*	*12*	*33*	*45*	*1*
Milk and milk products	240	60	418	478	14
- of which semi-skimmed milk	*46*	*11*	*169*	*180*	*5*
Eggs and egg dishes	46	11	30	42	1
Fat spreads	78	20	9	29	1
Meat and meat products	490	122	352	474	14
- of which chicken and turkey dishes	*57*	*14*	*104*	*118*	*3*
Fish and fish dishes	159	40	133	173	5
Vegetables, potatoes & savoury snacks	166	42	841	882	25
- of which potatoes & potato products	*48*	*12*	*459*	*471*	*13*
Fruits and nuts	24	6	342	347	10
Sugar, confectionery and preserves	5	1	17	18	1
Beverages	29	7	368	375	11
Miscellaneous[21]	284	71	92	164	5
Total intake from food	2229	557	2989	3546	101

[18] Sodium levels from both salt and non-salt sources.

[19] Assuming 0.25mg of potassium for every 1mg of sodium in food (including sodium not from salt).

[20] Baked goods comprise all types of bread, biscuits, buns, cakes, pastries, fruit pies & puddings.

[21] Miscellaneous foods include table salt, commercial toddlers foods and drinks, artificial sweeteners, soups, sauces, etc.

Figure 5.3: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for adults aged 65 - 74

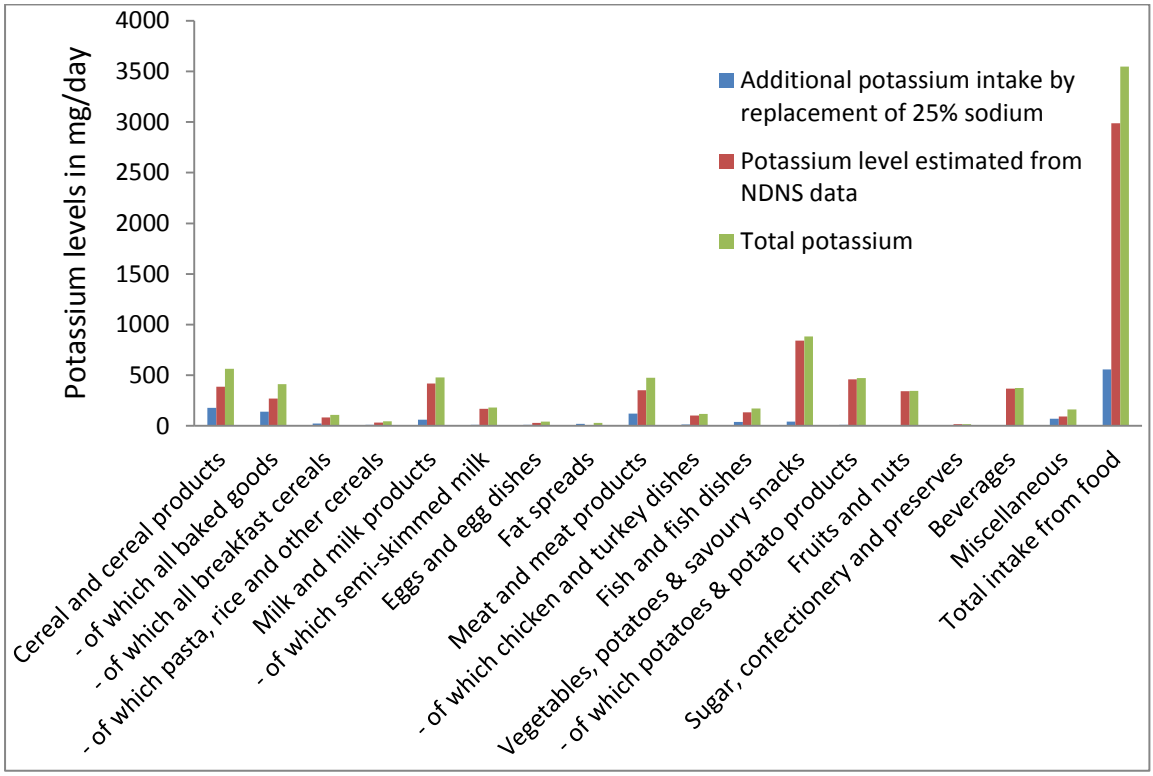


Table 5.4: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for adults aged 75+

Food category	Current sodium level[22]	Additional potassium intake by replacement of 25% sodium[23]	Potassium level estimated from NDNS data	Total potassium	% of RNI
Cereal and cereal products	624	156	382	538	15
- of which all baked goods[24]	*500*	*125*	*263*	*388*	*11*
- of which all breakfast cereals	*88*	*22*	*98*	*120*	*3*
- of which pasta, rice and other cereals	*36*	*9*	*21*	*31*	*1*
Milk and milk products	209	52	433	485	14
- of which semi-skimmed milk	*57*	*14*	*206*	*221*	*6*
Eggs and egg dishes	32	8	22	31	1
Fat spreads	87	22	10	32	1
Meat and meat products	458	115	296	411	12
- of which chicken and turkey dishes	*42*	*11*	*69*	*80*	*2*
Fish and fish dishes	106	26	129	156	4
Vegetables, potatoes & savoury snacks	132	33	681	714	20
- of which potatoes & potato products	*39*	*10*	*396*	*406*	*12*
Fruits and nuts	7	2	269	271	8
Sugar, confectionery and preserves	5	1	16	17	0
Beverages	18	4	278	282	8
Miscellaneous[25]	263	66	88	154	4
Total intake from food	1941	485	2606	3091	88

[22] Sodium levels from both salt and non-salt sources.

[23] Assuming 0.25mg of potassium for every 1mg of sodium in food (including sodium not from salt).

[24] Baked goods comprise all types of bread, biscuits, buns, cakes, pastries, fruit pies & puddings.

[25] Miscellaneous foods include table salt, commercial toddlers foods and drinks, artificial sweeteners, soups, sauces, etc.

Figure 5.4: Additional potassium from a replacement of 25% of sodium in various food categories (mg per person per day) for adults aged 75+

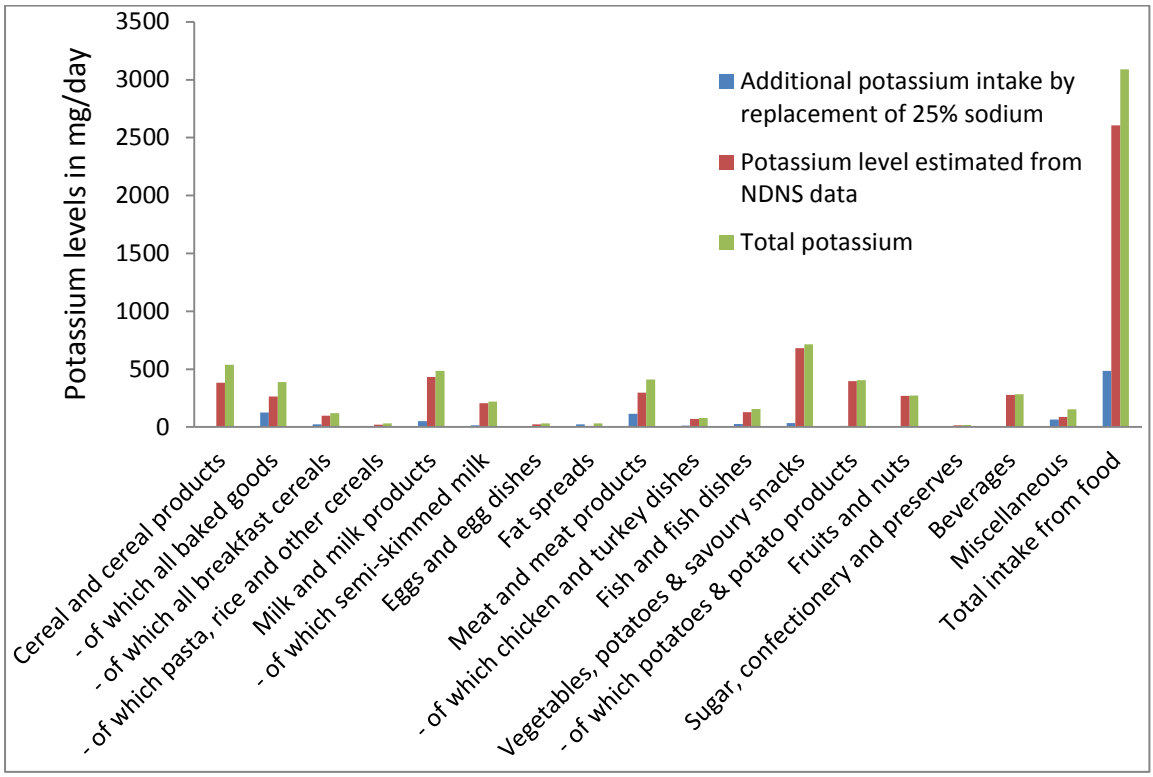
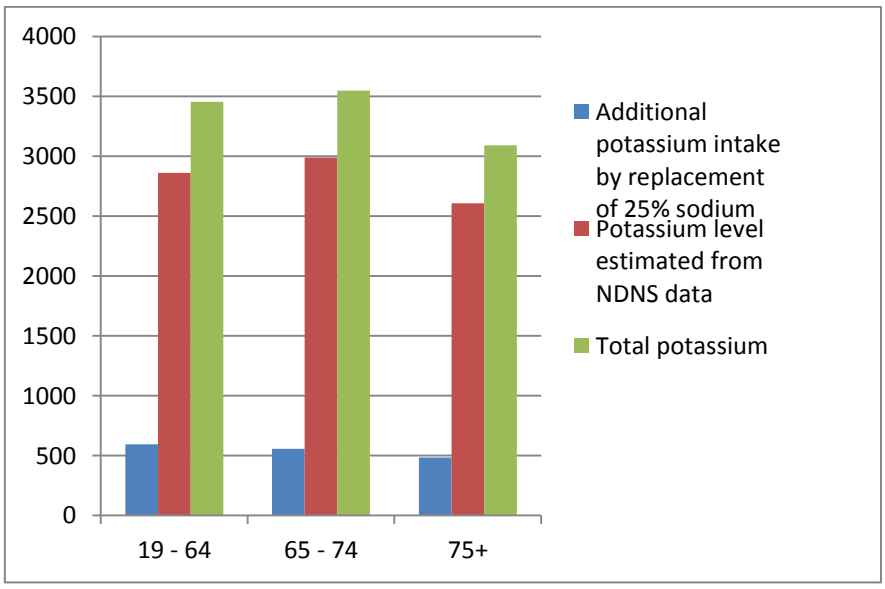


Figure 5.5: A comparison of potassium intakes among the three adult age groups (mg per person per day)



Data on actual potassium levels in products when replaced

21. Very limited data has been provided by the industry on actual levels of potassium based salt replacers. These seem to be primarily in baked goods where sodium bicarbonate is replaced by potassium bicarbonate. It has been suggested that in 'hotplate products' such as crumpets and pancakes, up to 50% sodium could be substituted by potassium. The resulting products could have a potassium level of anywhere between 100 - 450 mg/100 grams.

22. In an initial indication of actual potassium levels in these substituted or low-salt products, industry have provided values ranging from 203 - 344 mg/100 grams in products such as scones, cakes, crumpets and pancakes. If an individual were to consume low-salt two scones (2 x 50 grams) at 344 mg/100 gram, their potassium intake would be increased by 344 mg.

Secretariat
June 2013

References:

Buckinghamshire County Council (2005) Bottled Water Survey. Available at http://www.buckscc.gov.uk/media/137448/food_water_survey.pdf (accessed on 13 June 2013).

Morton MS, Elwood PC, Abernethy M. (1976) Trace elements in water and congenital malformations of the central nervous system in South Wales. Br J Prev Soc Med.30:36-39.

Powell P, Bailey RJ, Jolly PK (1987) Trace elements in British tap-water supplies. Swindon, WRc (Report PRD 706-M/1). Available at http://dwi.defra.gov.uk/research/completed-research/reports/dwi0007.pdf; accessed on 13 June 2013).

WRc (1998). Investigation of Drinking water Quality Enforcement Procedures in Member States of the European Union (Report No: DWI 4419/1). Available at http://dwi.defra.gov.uk/research/completed-research/reports/dwi0800.pdf (accessed on 13 June 2013).