The development of a dietary intervention to modify cation content of foods and the evaluation of its effect on blood pressure in hypertensive black South Africans

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Brief description of thesis content

Black South Africans are at high risk of hypertension, stroke and blood pressure-related target-organ damage. In South Africa, the limited resources at primary health care level allocated to the prevention, early diagnosis and management of hypertension necessitate a non-pharmacological population-based approach to curb the escalating burden of cardiovascular disease, for which raised blood pressure is an important major contributory risk factor.

The series of five studies included in the thesis provide a systematic approach to developing an appropriate nutritional population-based approach to lowering blood pressure in a high risk population. Firstly, valid, reliable, and updated information was obtained to identify habitual intake of sodium, potassium, magnesium and calcium in the target population, using the gold standard method of assessing sodium intake, namely 24-hour urinary excretion collections (Chapter 3). This information was necessary to inform the levels of sodium and other cation modification required in order to obtain a physiologically relevant change in blood pressure. As well as quantitative data on levels of sodium intake, the food sources that are the most important contributors to overall non-discretionary salt intake, and the pattern of intake of these foods, is described (Chapter 4). This data allowed identification of commonly consumed foods that could be targeted for modification of their cation content.

The collection of 24-hr urinary samples to determine sodium intake is not feasible on a large scale in a developing country such as South Africa due to cost constraints, methodological difficulties and generally low education levels. Since dietary surveys are time-consuming and costly, a rapid questionnaire that is able to estimate salt intake in the local population and that is useful for both research and clinical purposes was developed and validated (Chapter 5). Together with secondary analyses of previously published dietary surveys, data obtained in the dietary and urinary studies in this body of work were used to develop such a tool.

Next, in partnership with the food industry, the identified important commonly consumed food sources of salt were altered in cation content and tested for sensory properties, product quality and nutritional content (Chapter 6). Finally, the blood-pressure lowering effect of substitution of these novel food products for the standard varieties were investigated in an 8-week, randomised double-blind controlled community-based intervention (Chapter 7) in 80 black hypertensive patients aged 50 - 75 years. It is noteworthy that the effectiveness of the health benefits of the products were evaluated in the context of a community-based dietary intervention in which subjects were free-living within their usual socioeconomic living conditions.

This group of studies which culminated in a randomised controlled trial showed that free-living black hypertensive patients that are taking anti-hypertensive medication can lower their blood pressure further by consuming their usual foods which have been modified to contain lower levels of sodium and higher levels of potassium, magnesium and calcium. These findings have shown, for the first time in South Africa, that the improvement of commonly consumed foods can lower blood pressure. An imperative need for South African food companies to improve staple food products in the country, particularly bread, has been identified. The recommended public health strategy approach also identifies a need for the Department of Health to ensure that the necessary regulations are in place to enforce such improvements in the food chain in order to reduce blood pressure in the South African population.

Detailed abstracts

Study 1

Objectives: (1) To determine habitual urinary excretion and dietary intake of sodium, potassium, magnesium and calcium in three South African ethnic groups; (2) To assess the proportion of salt intake which is discretionary; (3) To investigate whether the BP-cation association varies, according to ethnic status; (4) To assess inter- and intra-individual variability in BP and urinary Na excretion; (5) To determine renin and aldosterone status across ethnic and hypertensive groups.

Design: A cross-sectional study of 325 black, white and mixed ancestry men and women, conveniently sampled in Cape Town.

Methods: 24-hr urine samples were collected on three separate occasions for assessment of urinary cations, and three 24-hour dietary recalls for the corresponding urine collection times were administered by two trained fieldworkers. Para-amino benzoic acid was used as a marker of completeness of urine collection. Blood pressure was measured on 3 occasions using an Omron automated device. BMI was measured, and plasma renin and aldosterone concentrations determined. Inter-individual variability was calculated as the coefficient of variation (CV = SD./mean), expressed as a percentage, between subjects for an average of 3 measurements. Intra-individual CV is calculated as the variation in each subject between the 3 repeated measurements.

Results: Mean urinary Na values equate to a daily salt (NaCl) intake of 7.8g, 8.5g and 9.5g in black, mixed ancestry and white subjects, respectively. Between 33 % and 46 % of total Na intake was discretionary. In normotensive subjects, both black and mixed ancestry subjects had significantly lower median urinary Na concentrations than white subjects, but these differences were not evident between black and white hypertensive subjects. No ethnic differences were found for urinary potassium, except for mixed ancestry normotensives having a lower excretion than white normotensives. Urinary Mg excretion did not differ across ethnic groups. In both normotensives and hypertensives, urinary calcium concentrations differed between all three ethnic groups, with black subjects having the lowest values of all groups, approximately less than half that of white subjects. No association was found between any of the urinary cations and BP. Inter-individual CV was 15 % and 13 % for systolic and diastolic BP, respectively, while intra-individual CV was 5.2 and 5.3 %, respectively, and was higher in hypertensives compared to normotensives. For urinary Na, inter-individual CV was 41.5 % and intra-individual CV was 33.7 %. Black and mixed ancestry hypertensives had lower plasma renin and aldosterone:renin ratio values compared to their normotensive counterparts, and a low renin status was more common in the black sample. An inverse association

between renin and systolic and diastolic BP was found, controlling for ethnic group, age, sex and BMI.

Conclusion: White South Africans have higher habitual intakes of sodium, but also higher calcium intakes, than their black and mixed ancestry counterparts. All ethnic groups had sodium intakes in excess of 6 g salt/day, while potassium intakes in all groups fell below the recommended 90 mmol/day. Dietary differences may contribute to ethnic-related differences in blood pressure.

Study 2

Objectives: To identify which food sources are the major contributors to sodium, potassium, calcium and magnesium intake in the South African population. **Research methods**: Cross-sectional study of 324 black, white and mixed ancestry hypertensive and normotensive subjects. Three repeated 24-hr urine samples were collected for assessment of urinary Na, and 3 corresponding 24-hour dietary recalls were administered by trained fieldworkers. Additionally, secondary analyses was performed on existing dietary databases obtained from 4 regional surveys undertaken on adults in South Africa.

Results: Between 33 % and 46 % of total Na intake was discretionary, while of the non-discretionary sources, bread was the single greatest contributor to Na intake in all groups. Urban/rural differences exist regarding sources of dietary Na, with over 70 % of total non-discretionary Na being provided by the bread and cereals food group in rural black South Africans, compared to 49 - 54 % in urban dwellers. Bread also provided the most K and Mg and, after *maas* (fermented milk), Ca in black subjects. Ethnic differences in calcium intake were evident, with black subjects having particularly low intakes.

Conclusion: The cereals food group is the largest contributor to non-discretionary Na intake and bread is the single food item which provides the highest proportion of Na in the diets of South Africans, particularly in the black population. It is recommended that the food industry be lobbied to lower the sodium content of bread, while simultaneously increasing K, Mg and possibly Ca, and that the impact of this intervention on blood pressure be tested in randomized controlled trials.

Study 3

Objectives: To develop and validate a short, food frequency questionnaire to assess habitual dietary salt intake in South Africans and to allow classification of individuals

according to intakes above and below the maximum recommended level of 6 g salt per day.

Design: A cross-sectional validation study in 324 black, white and mixed ancestry men and women, conveniently sampled in Cape Town.

Methods: 24-hr urine samples were collected on three separate occasions for assessment of urinary Na, and three 24-hour dietary recalls for the corresponding urine collection times were administered by two trained fieldworkers. All individual food items consumed by more than 5% of the sample and which contributed ≥ 50 mg Na/serving were included in the questionnaire and collapsed into 42 separate categories. The questionnaire was scored by calculating the Na content of an index food in each category (divided by 50 and rounded to the nearest integer) and multiplying it by a frequency factor (1 - 6) calculated from subjects' repeated dietary recall data. Internal consistency of the final questionnaire was assessed in a new sample of 80 black hypertensive South Africans using the same data collection methodology.

Results: Positive and significant correlations were found between the Na content of 35 of the 42 food groups in the questionnaire and total Na intake, calculated using the 24-hr recall data. A positive association was found between total Na content of the questionnaire and Na estimations from the 24-hr recalls (Spearman r = 0.750; P <0.0001; N = 328) and urinary Na excretion (r = 0.152; P = 0.0105; N = 284). r = 0.750 (P <0.0001) (N = 328). Comparing tertiles of questionnaire Na score, urinary Na was significantly higher for subjects in tertile 3 than those in tertile 1. Stanines of questionnaire Na content were combined into three groups (1+2+3; 4+5+6; 7+8+9) which yielded significant differences in urinary Na between Groups 1 and 3 (P <0.05). Two groups were created for questionnaire Na content: < and ≥ 2 400 mg (6 g NaCl)/day which equated to a reference cut-off score of 48. According to this categorisation, a significant difference in urinary Na was found (144.8 (68.4) mmol/day vs 176.4 (99.4) mmol/day; P<0.05). Sensitivity and specificity against urinary Na ≥ and < 100 mmol/day was 14.2 % and 92.4%, respectively. Positive and negative predictive value was 86.1 % and 24.6 %, respectively. In a new sample, the questionnaire Na score was correlated with Na intake estimated from repeated 24-hr dietary recall (r = 0.5606; P<0.0001) but not with urinary Na.

Conclusion: A 42-item food frequency questionnaire has been shown to have content, construct and criterion-related validity, as well as internal consistency, with regard to categorising individuals according to their habitual salt intake. Due to its high specificity, the questionnaire can identify individuals with an excessive salt consumption. The instrument will be useful in research and clinical settings. The devised categorical scoring system needs to show improved sensitivity. Further validation studies of the

instrument should be undertaken in different geographical areas (i.e. urban and rural) where local communities are known to have different eating patters with regard to processed foods and salt use.

Study 4

Objectives: To favourably modify the cation content (Na, K, Mg, Ca) of food items which are commonly consumed by the black urban South African population. **Methods**: Partnerships were forged between academics from two institutions (Medical Research Council and University of Cape Town) and three food industry members. The experimental bread was compared against standard brown bread, after a drop test (to assess whether bread quality deteriorates with abuse) and after usual baking practices for baking properties (volume, crust colour, crumb colour and cell structure), sensory properties and nutritional composition. Plant production feasibility was evaluated in an industrial plant. Breads produced there were subjected to sensory evaluation using triangulation tests in a panel of 122 consumers. Twenty-four samples of both the standard and the experimental bread were laboratory-analysed for Na, K, Mg and Ca content using standard methods laboratory. A reduced salt version of Knorr soup mix, Knorrex stock cubes, Aromat (monosodium-glutamate based flavour enhancer) and a brick margarine was developed by Unilever Foods South Africa. Two types of salt replacements were used in the development of these products.

Results: A 32.3 % reduced Na brown bread was developed which was acceptable in terms of baking qualities, appearance, texture and taste. Potassium, magnesium and calcium content of the bread were increased by 55.2 %, 69.0 %, and 34.8 %, respectively. Sodium was reduced by 68.8 % in the soup mix, 23.6 % in the stock cube, 51.1 % in Aromat, and 62.1 % in margarine. All products were similar to the standard versions in terms of appearance, structure and taste (evaluated by expert research and development staff).

Conclusion: Reduced Na versions of brown bread, brick margarine, soup mix, stock cube and a flavour enhancer (Aromat) were successfully developed. The impact on blood pressure of substitution of these items for the regular varieties requires testing in a randomised controlled trial.

Study 5

Objective: To assess the impact of a food-based intervention on blood pressure in treated hypertensive individuals.

Design: Randomized, double blind, parallel group controlled trial.

Setting: Community-based, peri-urban area of Cape Town, South Africa. **Participants**: Free-living black South African men and women aged 50 - 75 y, with drug-treated mild-to-moderate hypertension (Systolic BP ≤ 160 mmHg and Diastolic BP ≤ 95 mm Hg), were recruited from a church-based luncheon club and by advertising in a local community newspaper. Subjects were randomly allocated to a control group (n = 40) or a reduced sodium, increased potassium, magnesium and calcium diet group (n = 40) in a 2-staged design.

Intervention: 8-week provision of 6 commonly consumed food items (salt replacement (Solo™), bread, margarine, stock cubes, soup mixes, Aromat (flavour enhancer)) which had a modified cation content (reduced in Na, and increased in potassium, magnesium and calcium content). In addition, 500 ml/day of maas (fermented milk) was provided. The control diet provided the same quantities of these foods to subjects, but which were of standard commerical composition. Instead of maas, 500 ml/day of artificially sweetened cooldrink was given. Compared to control foods, intervention foods provided 41 % less Na and 826 %, 388 %, 368 % more K, Ca and Mg, respectively.

Main outcome measures: Between-diet difference in change in office blood pressure (Omron automated BP monitor) and 24h ambulatory blood pressure monitoring (ABPM) between baseline and mean of 4-week and 8-week intervention readings ("Post").

Results: The intervention effect, as measured by the Omron automated monitor, for systolic BP is a reduction of 6.19 (SEM = 2.64) mmHg (95% CI: -11.44 to -0.94 mmHg) (P=0.021) for systolic BP and a non-significant reduction of 0.60 (1.22) mmHg (95% CI: -3.02 to 1.83 mmHg) for diastolic BP. Mean 24-h ABPM between-diet change was a reduction in both systolic BP of 4.53 (2.27) mmHg (95 % CI = -9.05 to -0.006 mmHg; P = 0.050) and diastolic BP of 2.49 (1.34) mmHg (95 % CI = -5.16 to 0.17; P = 0.066). The largest change in 24-h ABPM was demonstrated for wake systolic BP (-5.14 (2.40) mmHg; 95 % CI = -9.93 to -0.35; P = 0.036) and wake diastolic BP (-2.66 (1.46) mmHg; 95 % CI = -5.56 to 024; P = 0.072).

Conclusions: An 8-week dietary intervention, in which high salt foods with a modified Na, K, Mg and Ca content are provided, together with a fermented milk drink, resulted in clinically significant reductions in systolic BP in treated South African hypertensive patients from a low socioeconomic setting who receive hypertension care from the public health sector.

Chapter 1

Literature review: Effect of dietary factors on blood pressure

1.1 Overview of hypertension in South Africa: prevalence, treatment status and risk factors

The estimated leading cause of death in South Africa is non-communicable diseases, which accounted for 37 % of all deaths in the country in 2000, followed by HIV/AIDS (30 % of deaths) and pre-transitional causes (communicable diseases, maternal causes, perinatal conditions and nutritional deficiencies) (21 %).[1] Regarding specific cause of death, cardiovascular disease is the second leading cause of death after HIV/AIDS, and accounts for 17 % of all deaths.[1] Approximately 6 million of the total population of 41 million South Africans are estimated to be hypertensive (< 140/90 mmHg),[2] placing them at high risk for developing cerebral haemorrhage, malignant hypertension, kidney disease leading to uraemia and congestive heart failure.[3,4] Coronary heart disease (CHD) is relatively uncommon in the black population, in contrast to it being the major outcome related to hypertension in the white and Indian communities.[3] Diagnosis and management of hypertension is poor. Local studies suggest that only about half of all hypertensive patients in South African communities are known to health services; only half of those who are diagnosed are treated; and only between 16 % to 35 % of treated hypertensives are "controlled" (<160/95 mmHg).[5,6,7] The comprehensive Demographic and Health Survey (DHS), conducted in almost 14 000 adults in 1998, reported that in the hypertensive (BP ≥140/90 mmHg[8]) urban black population, less than a fifth (17.5 %) of hypertensive men and 35.6 % of hypertensive women were receiving treatment for their condition, while only 8.2 % and 18.7 %, respectively, had controlled BP levels (< 140/90 mmHg).[9]

Hypertension poses a health and economic burden on South Africans by virtue of its morbidity, costly complications, and predisposition to premature mortality.[10] The impact of hypertension on mortality was assessed in an African population in 1996 by Kaufman *et al.*[11] The risk of death increased by 60% with an increase of 20 mmHg in diastolic blood pressure (BP) in rural Nigeria and the authors estimated that the population attributable risk (the reduction in total mortality that would have been observed if hypertension were not present) was 7%. Available evidence suggests an escalating risk for not only hypertension but also for a deterioration of the global CVD risk factor profile of black South Africans. Data from a birth cohort study conducted in the greater Johannesburg area (Birth-to-Ten study) has shown that black children aged as young as 5 years already have significantly higher BP levels than Indian and white children of the same age.[12]

The consistent finding of a higher prevalence of hypertension and its related sequelae in US blacks compared to whites has led to speculation that African-origin populations are particularly susceptible to this condition.[13,14] This conventional wisdom has been challenged in a recent analysis of data from 8 white and 3 black populations that has demonstrated a wide variation in hypertension prevalence among both ethnic groups.[15] The rates among black populations are not unusually high when viewed internationally. Instead, the analysis suggests that the impact of environmental factors among both ethnic groups may have previously been under-estimated. The gradient in hypertension prevalence among black populations from Nigeria, Jamaica and the US is consistent with transition to an industrialized lifestyle.[15] In this regard, the age-adjusted prevalence of hypertension of 23.5 % and 25.0 % for South African black men and women, respectively, could possibly be expected to rise by 25 % if a typical Westernized lifestyle is adopted by both urban and rural dwellers alike.[16] In South Africa, as in other countries experiencing economic transition from underdeveloped to more developed such as Brazil and China, increased rates of obesity accompany the nutrition transition taking place.[17] The already alarmingly high overweight and obesity rates in urban black South African women (62 %)[18] probably explain why hypertension prevalence has exceeded that of white women.

In addition to obesity being a major risk factor for hypertension, dietary factors other than excessive energy intake, are also important determinants of raised blood pressure in African communities. Data from the THUSA study has identified factors related to hypertension in a black sample undergoing the health transition in the North West province of South Africa.[19] Factors related to urbanisation were positively associated with hypertension in this population. Steyn *et al.*[20] reported similar findings whereby the duration of urbanisation independently predicted the presence of hypertension in the black community of Cape Town. However, other authors have not found an urban/rural difference in the prevalence of hypertension in the black South African population.[21] In the THUSA study, blood pressure correlated positively with age, level or urbanisation, waist:hip ratio and tobacco smoking. Additional factor analyses of the data found that, of the clusters of risk factors relating to hypertension, the most important was the malnutrition cluster which included high intakes of saturated fat, animal protein, sodium, vitamin A and B_6.[22] A second cluster identified had characteristics of the metabolic syndrome. A third cluster consisted of an hypercholesterolaemic and obesity group of factors that included ageing, total, LDL cholesterol, tryglycerides, high body mass index and central obesity.

The reported dietary patterns of the majority of South Africans indicate a low carbohydrate, low dietary fibre, and marginal micronutrient intake.[23,24] Consumption of fruits and vegetables (and therefore potassium) is inadequate and falls far below the internationally recommended guideline of at least 5 portions (400g) of fruits and vegetables per day.[25] A meta-analysis of cohort studies has provided evidence that an even greater intake of fruit and vegetables may reduce the risk of both ischaemic and haemorrhagic stroke.[26] Compared with individuals who had less than three servings of fruit and vegetables per day, the relative risk of stroke was 0.89 (95% CI = 0.83 - 0.97) for those with three to five servings per day, and 0.74 (95 % CI = 0.69 - 0.79) for those with more than five servings per day.

It is estimated that South African adults and children aged 10 + years consume, on average, 93g/day of vegetables and 61 g/day of fruit.[27] Black urban dwellers are reported to eat fruits and vegetables in small amounts, usually one small portion twice a day.[23,28,29,30] Some studies have reported negligible fruit and vegetable intakes in this group, such as in Cape Town where 29% of black adults in a peri-urban setting reported eating no fruit or vegetables in the previous 24 hour period.[31]

Reliable information on habitual dietary intakes of sodium in the South African population is sparse. Elderly residents of West Coast fishing villages were found to have a 24-hour urinary sodium excretion which corresponded to a dietary intake of about 9g of salt per day,[32] which exceeds the recommended value of 6g or less per day of the US National High Blood Pressure Education Program.[33]

1.2 Concept of salt sensitivity and its role in blood pressure regulation

Blood pressure is a function of cardiac output and peripheral vascular resistance. The kidneys, which excrete almost all ingested electrolytes and much of the water consumed daily, are responsible for managing the electrolyte and water content in the body. Volume content is tightly controlled by the regulation of sodium (and thereby chloride) excretion. Almost all people living in westernized societies ingest a high-sodium diet, however not all individuals respond similarly to a high-salt intake. A relationship between renal salt and water excretion and blood pressure can be created for any level of blood pressure and is termed the renal pressure-natriuresis or diuresis relationship, first described by Guyton.[34] According to his hypothesis, the pressure–natriuresis curve has always to be affected in hypertension, whatever the

cause initiating the hypertensive process. All forms of hypertension in animal models tested to date feature a shift in the pressure-natriuresis relationship to the right, so that a higher level of pressure is required to excrete any given amount of salt and water. In normotensive individuals the relationship between salt and water intake (and excretion) is very steep, so that little change in blood pressure occurs when salt and water intake (and excretion) are modified over a large range. Conversely, a fairly flat pressure-natriuresis curve indicates a sensitivity to salt. Kimura and Brenner[35] have extended this approach and described the various pressure–natriuresis curves in sodium-sensitive and sodium-resistant forms of secondary hypertension. They have proposed three major renal mechanisms leading to the development of hypertension, namely an increased pre-glomerular vascular resistance, a decrease in whole kidney ultrafiltration, and an increase in tubular sodium reabsorption. They suggest that pre-glomerular vasoconstriction leads to a salt-resistant hypertension whereas a reduced nephron mass and alterations of renal sodium handling result in the development of salt-sensitive forms of hypertension. Johnson and Schreiner[36] emphasize the role of microvascular injury and tubulointerstitial fibrosis in shifting the pressure–natriuresis curve to the right, to result in the development of salt-sensitive hypertension. These authors have shown that transient angiotensin II[37] and phenylephrine infusions[38] can induce renal microvascular injury and tubulointerstitial fibrosis and salt-dependent hypertension even when the hyperactivity of the sympathetic or renin–angiotensin system is no longer engaged. These various hypotheses, together with the results of renal transplantation experiments in animals[39] and humans,[40] identify the kidney to be the key determinant of the blood pressure response to salt. Yet salt sensitivity is a multifactorial phenomenon involving extrinsic, essentially hormonal, as well as intrinsic renal tubular and haemodynamic mechanisms, possibly modulated by genetic factors.[41]

In most individuals a very wide range of salt intake is accompanied by only a modest and transient change in arterial blood pressure, as would be expected from the pressure–natriuresis relationship that dominates the long-term control of blood pressure.[34] The concept of salt sensitivity and salt resistance in humans was first described by Kawasaki *et al.*[42] and later by Weinberger et al.,[43] in an attempt to explain the heterogeneity of the blood pressure response to salt. Salt sensitivity was initially defined as an increase in mean arterial pressure greater than 10% when a high-salt diet was administered, compared with a low-salt period.[44] The methodology submitted subjects to extreme changes in sodium intake (from 10 mmol/day to 250 mmol/day) for a period of one week.[43] Subsequently, various different experimental

protocols have been used to test the salt sensitivity of blood pressure in humans, including an acute protocol in which patients are salt-loaded with an intravenous infusion of saline and salt-depleted with the administration of frusemide.[43] It has been suggested that acute changes in sodium balance may not adequately reflect long-term changes in dietary sodium intake and that ambulatory blood pressure monitoring may be a more reliable way of measuring the salt-induced changes in blood pressure.[45,46] As well as the route of sodium administration, other methodological discrepancies between investigators include the level of salt restriction and/or salt loading required to elicit a response, as well as the number of days on which subjects should be salt loaded or depleted. In addition, the validity of salt sensitivity as a clinical concept has been questioned with regard to its usefulness to dichotomize a population, when in fact salt-induced changes in blood pressure are normally, rather than bimodally distributed.[41] Yet another major criticism of the concept of salt sensitivity relates to its low reproducibility,[47,48,49] however this is contested by Campese and colleagues who maintain that salt sensitivity is a reproducible and persistent phenomenon over time when assessed according to rigorous protocols.[50]

Since there is no quick and easy way to predict whether an individual is salt sensitive, the classification has remained in the research domain rather than being of practical or clinical importance. However, despite seemingly arbitrary and varied definitions of salt-sensitivity in the literature, several findings are consistently observed: hypertensive patients are more frequently salt sensitive than normotensive subjects and the prevalence of salt sensitivity is increased in older individuals, black populations, and patients with a low-renin hypertension such as diabetics.[43]

Putative mechanisms for salt sensitivity are: alterations in circulating levels of (or renal responses to) atrial natriuretic factor, kallikrein, prostaglandins, and nitric oxide; increased levels of norepinephrine; abnormal suppression of both renin and aldosterone; genetic mechanisms; and acquired renal microvascular and tubular injury.[51,52]

In the US, the greater susceptibility of African-Americans than whites to hypertension and pressure-related target-organ damage has been linked to a higher prevalence of salt sensitivity, as well as lower urinary potassium excretion, lower plasma renin activity, and higher circulating levels of immunoreactive parathyroid hormone and 1,25 dihydroxyvitamin D.[53] Salt sensitivity has been reported to be present in 73% of

African American hypertensives, compared to 56% of Caucasian hypertensives but in the normotensive population, the frequency of salt sensitivity among blacks (36%) is similar to that seen among whites (29%).[54] Similar observations of a greater frequency of salt sensitivity among blacks have been reported in other studies.[55,56] A more recent study has demonstrated the prevalence of salt sensitivity to be similar in both races when subjects are closely matched, although the magnitude of BP increase is greater in African American hypertensives.[57] Studies conducted in hypertensive patients, in both Johannesburg[58] and Cape Town,[59] South Africa, have also suggested diminished activity of the sodium-potassium ATPase pump in black hypertensives. The clinical observation that hypertension in black South Africans and African-Americans manifest higher average BP responses to calcium antagonists than to ACE inhibitors supports the hypothesis that hypertension amongst these groups may be salt sensitive.[60,61]

1.3 Blood pressure and lifestyle modification: overview and introduction

In patients with hypertension, consideration of the degree or stage of the condition is required, together with an assessment of the presence of other major cardiovascular disease risk factors, target organ damage, or clinical cardiovascular disease, in order to decide on a first line of management (Table 1). A new classification, "prehypertension" (120-139/80-89 mm Hg), has been included in the most recent version of the Joint National Council (JNC7) guidelines of the US National Heart Lung and Blood Institute,[33] for which lifestyle modification is recommended. This identifies a need for increased education of health-care professionals and the public to reduce blood pressure levels and thus prevent the development of hypertension in the general population.

Lifestyle modification (Table 2) is recommended for all patients with hypertension as well as for those with pre-hypertension, regardless of whether or not drug therapy is initiated.[33]

Table 1
Classification and management of blood pressure for adults* (JNC 7 Guidelines)[33]

BP Classification	Systolic BP* (mmHg)	Diastolic BP* (mmHg)	Lifestyle modification	Initial drug therapy	
				Without compelling indication	With compelling indications**
Normal	< 120	And < 80	Encourage	No antihypertensive drug indicated.	Drug(s) for compelling indications.‡
Prehypertension	120 - 139	Or 80 - 89	Yes		
Stage 1 Hypertension	140 - 159	Or 90 - 99	Yes	Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB or combination.	Drug(s) for compelling indications.‡ Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.
Stage 2 Hypertension	≥ 160	Or ≥ 100	Yes	Two-drug combination for most† (usually thiazide-type diuretic and ACEI or ARB or BB or CCB).	

Drug abbreviations: ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; BB = beta-blocker; CCB = calcium channel blocker.

* Treatment determined by highest BP category.

** Compelling indications include heart failure, postmyocardial infarction, high coronary disease risk, diabetes, chronic kidney disease, or recurrent stroke prevention.

† Initial combined therapy should be used cautiously in those at risk for orthostatic hypotension.

‡ Treat patients with chronic kidney disease or diabetes to BP goal of <130/80 mm Hg.

Table 2
Lifestyle modifications for hypertension prevention and management (JNC 7 guidelines)[33]

Modification	Recommendation	Average Systolic BP reduction range †
Weight reduction	Maintain normal body weight (BMI = 18.5 - 24.9 kg/m^2)	5 - 20 mm Hg/10kg
DASH eating plan	Adopt a diet rich in fruits, vegetables and low fat dairy products with reduced content of saturated and total fat.	8 - 14 mm Hg
Dietary sodium restriction	Reduce dietary sodium intake to ≤ 100 mmol/day (2.4 g sodium or 6 g sodium chloride).	2 - 8 mm Hg
Aerobic physical activity	Regular aerobic physical activity (eg. Brisk walking) at least 30 minutes/day, most days of the week.	4 - 9 mm Hg
Moderation of alcohol consumption	Men: limit to ≤ 2 drinks*/day. Women and lighter weight persons: limit to ≤ 1 drink/day.	2 - 4 mm Hg

† Effects are dose and time dependent.
* 1 drink = 1/2 oz or 15 mL ethanol (eg. 12 oz beer, 5 oz wine, 1.5 ox 80-proof whiskey).

It is noteworthy that all of the recommended lifestyle modification measures are dietary related, with the exception of increased physical activity. However, physical activity, through its integral role in the energy balance equation together with energy intake, helps regulate body weight.

The publication of the findings of the landmark Dietary Approaches to Stop Hypertension (DASH) clinical trial[62] signalled a move by health professionals away from a preoccupation with salt to the role of a holistic, composite diet in the prevention and management of hypertension. The rest of this literature review will consider the evidence relating to the influence of individual nutrients on blood pressure, and then review the role of various dietary patterns, as well as other behavioural factors.

1.4. Diet and blood pressure

1.4.1. Sodium

Salt restriction as a form of treatment for hypertension was introduced at the beginning of the century when chloride could first be measured. Interestingly almost 100 years later, the merits of the salt hypothesis and the utility of its application are still being debated. The medical community is seldom as bewildered and polarized about a public health policy issue as it is regarding the role of salt in health and disease. In 2000, two opposing arguments (for[63] and against[64]) regarding the appropriateness of the current US dietary guideline for sodium (Na), which recommends less than 6g sodium chloride (or < 2.4 g Na) per day, were published back-to-back in the *American Journal of Clinical Nutrition*.

During the 1930s and 1940s, it was shown by Grollman *et al.* (1953)[65] that if renal mass and blood flow were reduced in experimental animals, blood pressure increased predictably. At the same time, Kempner (1948)[66] demonstrated that malignant hypertension associated with renal insufficiency could be greatly attenuated by an extremely low sodium diet, consisting predominantly of rice. In the early 1950s, Tobian and Binion[67] observed that the sodium content of blood vessels correlated with arterial pressure, however other investigators at the time who tried to reproduce salt-induced hypertension found that extreme manipulation of vertebrate physiology and nutritional intake was required to observe such an effect. Typically, salt intakes needed to be increased by 10 – 20 times in rodent models (even in Dahl and colleagues' genetically salt sensitive rat), renal mass reduced to less than half, and mineralocorticoid hormones administered in pharmacological doses.[68] Metabolic

studies of the haemodynamic and hormonal effect of short term manipulations of salt over the physiological range were not conducted in either normotensive or hypertensive subjects until the late 1970's, over twenty years since the first reported observations between salt and blood pressure.[69,70]

The salt-blood pressure hypothesis states that excessive salt intake [in physiological terms] leads to an increase in blood pressure [BP] in genetically susceptible persons and, if the high intake is maintained over the long-term, ultimately leads to sustained hypertension.[71] The hypothesis regards salt as the essential pathogenic factor, however, the influence of supraphysiological salt intake on blood pressure interacts with both genetic predisposition as well as other environmental factors.[72,73,74,75]

The INTERSALT ecological study,[76] conducted in over 10,000 individuals from 52 centres around the world, demonstrated that, across populations, level of blood pressure, increment in blood pressure with age (See Figure 1), and the prevalence of hypertension are related to salt intake, measured using 24-hr urinary sodium excretion. However, the within-population analyses indicated that wide variation in the blood pressure-salt relationship occurs throughout a population.

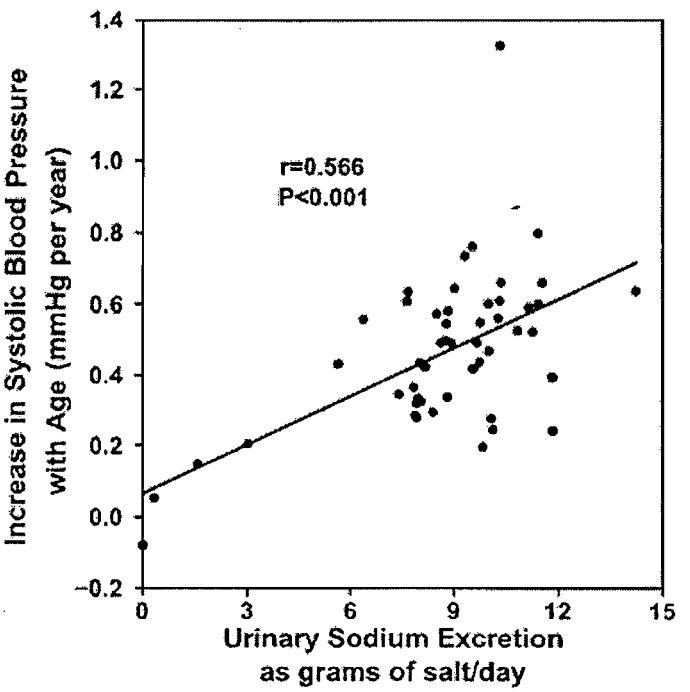

Figure 1
The relation of salt excretion to the slope of the rise in systolic blood pressure with age in 52 centres in the INTERSALT study (Taken from ref[77], originally adapted from ref[78]).

The main observations in the INTERSALT study are that: (i) for individuals, a difference of 100 mmol/d of sodium (6 g NaCl) intake is associated with an average difference of between 3 and 6 mm Hg in systolic blood pressure, and (ii) for populations, a 100 mmol/d lower sodium intake is associated with attenuation of the age-associated rise in systolic blood pressure by 10 mm Hg in persons aged 25–55y.

It is noteworthy that in four remote INTERSALT study sites who consumed less than 3 grams of salt per day, both sodium excretion and blood pressure were low, there was little or no upward slope of blood pressure with age, and little or no hypertension. Similarly, there are over 40 studies in unacculturated human populations who at one time consumed, or continue to consume, less than 3 grams of salt per day.[79] In these populations, blood pressure did not and does not rise with age. For example, the Yanomamo Indians from southern Venezuela consume a diet that is very low in salt (1 mmol Na/day) as well as saturated fat and that is high in fruit, vegetables and roots, yielding a potassium intake of 200 - 300 mmol/day. In this tribe, blood pressure does not rise with age, and vascular disease is not found, despite the fact that much of their time is spent fighting under stressful conditions.[80,81] However, if this group migrate to a Venezuelan or Brazilian town and adopt a western lifestyle, they, like native Americans, become overweight and develop diabetes and premature vascular disease.

Several systematic reviews have reported that restricting sodium intake in people with hypertension reduces their blood pressure.[82,83,84,85,86] Generally, a 100 mmol/day reduction in sodium intake is associated with an average reduction in diastolic blood pressure of 1.4 to 2.5 mm Hg, which could result in a 15 % reduction in stroke risk.[87] However, most of the studies included in these reviews have been short in duration and have not followed subjects up over the long term to assess whether dietary changes are sustainable.

Most of the large dietary intervention blood pressure trials have been conducted in the Unites States or Europe. However, a recently published study has shown that short-term Na restriction can be achieved in two developing countries, namely Jamaica and Nigeria.[88] Normotensive subjects randomly followed a low salt and a high salt diet for 3 weeks each, with a 2-week washout in between. Subjects were counselled on dietary Na sources and on behavioural techniques to lower dietary Na. In Nigeria, rock salt (potash), stock cubes and fermented locust beans (iru) are the main Na sources and very little processed food is eaten. In Jamaica, participants

were provided with pre-packaged, low sodium spices for cooking and had the option of purchasing low sodium food at a local vendor. The BP reduction associated with the low salt intake was similar in both groups, at approximately 5 mmHg for systolic BP. This study suggests that the efficacy of sodium reduction in developing countries equals those noted in more affluent cultures.

As well as human studies which show a salt-blood pressure relationship, there are also numerous studies in the rat, dog, chicken, rabbit, baboon and chimpanzee, all of which demonstrate an increase in blood pressure in the presence of a prolonged increase in salt intake.[89] In all forms of experimental hypertension, whatever the animal model, a high salt intake is essential for the blood pressure to rise. A study conducted in chimpanzees, man's closest relative, showed that when salt intake was increased from their usual intake of < 0.5 g/day to 15g/day, the blood pressure rose slowly, to a significant increase after one year, and continued to rise thereafter.[90] Blood pressure returned to normal when salt intake was reduced to pre-trial levels.

Is dietary sodium intake a modulator between hypertension and end organ damage?

In addition to its influence on arterial pressure, there is some evidence that dietary sodium may exert some nonpressure-related effects on left ventricular mass in experimental models of hypertension,[91] as well as in human essential hypertension.[92,93,94] As long ago as 1972, it was observed that the addition of salt to food before tasting (proxy for high salt intake) was associated with an increase in the prevalence of electrocardiographic evidence of left ventricular hypertrophy (LVH) in hypertensive men.[95] Recently, it has been demonstrated that dietary sodium amplifies the effect of arterial pressure on target organ damage (microalbuminaemia and left ventricular mass), even in normotensive humans, thus it can be considered to be an independent factor of cardiovascular risk.[96] Further, moderate sodium restriction in patients with mild-to-moderate essential hypertension has been shown to decrease LVH.[97]

Is dietary sodium intake associated with cardiovascular disease risk and all-cause mortality?

It has been argued for decades that a high salt intake may not be detrimental in subjects who are not sensitive to the blood pressure elevating–effects of sodium. However, a prospective study of 1173 men and 1263 women in Finland found that coronary heart disease, cardiovascular disease and all-cause mortality all rose

significantly with increasing 24-hour urinary sodium excretion, independently of other cardiovascular risk factors, including blood pressure.[98] In another 19-year cohort study, high reported dietary sodium intake at baseline was strongly and independently associated with an increased risk of cardiovascular disease and all-cause mortality in overweight persons.[99]

Dietary sources of sodium and recommended levels of salt restriction

Sodium chloride is approximately 40% sodium and 60% chloride. The mean daily sodium intake of Americans adults is 3290 mg (equivalent to a salt intake of about 8 grams),[100] which greatly exceeds the estimated minimum requirements of healthy nonpregnant, nonlactating adults of about 500 mg.[101] It is estimated that about three-quarters of sodium intake comes from food processing, about 15% is discretionary (half of which is contributed by table salt and half by added salt in cooking), 10-11% is naturally occurring (inherent) in foods, while less than one percent is provided by water.[102,103,104,105] The largest single source of sodium in the American diet comes from grain products, including bread, which contributes about a quarter of total intake.[105]

Cultural practices may contribute to salt intake patterns as the following example illustrates. A 10-fold difference in sodium intake has been reported between two populations of the Solomon Islands which has been attributed to the practice by one group's of steaming foods with fresh water whereas the other group cooks with sea water.[106]

In South Africa, the habitual sodium intake of the population is not known. Many dietary surveys have been conducted in various sub-groups of the population,[23,24] however it is notoriously difficult to accurately assesses sodium intake from dietary reports, since added salt (i.e. discretionary) usage cannot be easily quantified. Undoubtedly, the gold standard method for assessment of salt intake is repeated analyses of 24-hr urinary sodium. Studies conducted in black and white normotensives in Johannesburg in 1982,[107] demonstrated ethnic differences in 24-hr urinary sodium excretion, with black subjects having significantly lower values than their white counterparts (127 ± 55 mmol/day and 167 ± 63 mmol/day, respectively). The urinary Na estimations equate to a daily salt intake of 7.5g and 9.8g in black and white South Africans, respectively. However, dramatic lifestyle-related changes associated with urbanisation and rapidly increasing economic growth have resulted in demographic and nutritional transition in the black population.[31,108] The increasing

rise in overweight and obesity, and accompanying prevalence of non-communicable diseases, such as coronary heart disease, stroke and diabetes mellitus, has been shown to be associated with these dietary and lifestyle changes.[109] Updated, reliable information on amounts and sources of dietary salt intake is thus required in order for population-wide non-pharmacological interventions to be developed and successfully implemented.

The Joint National Council (JNC) 7 guidelines of the US National Heart Lung and Blood Institute[33] recommends a maximum sodium intake of 100 mmol (2400 mg) per day, which equates to about 6 g salt (Table 1). If an individual is prepared to give up salt added to food and to avoid eating salt-rich processed foods, salt intake can be reduced from the average intake of around 9 g (144 mmol or 3310 mg sodium) to about 6 g per day. This is the level of sodium restriction which is usually referred to as "No added salt" regimen (i.e., 80–100 mmol/d). The World Health Organization's (2003) dietary goals for the prevention of cardiovascular disease, including the prevention of hypertension and stroke, recommend more stringent restrictions of sodium, namely an optimal intake of 70 mmol per day (4g sodium chloride).[110] That body does however advise that a more realistic population goal is 5 g salt per day. Minimal consumption of other forms of Na consumption, such as food additives and preservatives (eg. monosodium glutamate) is cautioned.

Can excessive salt restriction have adverse health effects?

It may be argued that, providing widespread advice to the general public to reduce salt intake may be warranted if health benefits are gained by at least those individuals who are "salt sensitive," while at the same time those who are salt-resistant experience neither benefit nor risk. There is a school of thought that proposes an increased morbidity to be associated with salt restriction. In this regard, a cohort study of 2 937 mildly and moderately hypertensive men which investigated the effects of a low salt intake on cardiovascular disease over an average of 3.8 years of follow-up, demonstrated a significant, inverse association between baseline 24 h urinary sodium excretion (anithypertensive therapy was discontinued for 3 to 4 weeks before providing a urine collection) and myocardial infarction, independent of several known coronary heart disease risk factors.[111] The downfalls of observational studies, with regard to unmeasured confounder variables and the imprecision of the potential confounders which are measured, are well known and may have contributed to the occurrence of this surprising finding.[112]

A 20-year follow-up of the first National Health and Nutrition Examination Survey (1971 - 1975) baseline sample (N = 20 729 US adults aged 25-75 y) examined mortality in gender-specific quartiles of sodium intake in 11 346 persons.[113] The inverse association found between sodium intake at baseline and both all-cause and cardiovascular mortality was no longer apparent when sodium intake was expressed as a function of energy intake. Instead, a direct association between sodium intake and mortality was found

Feasibility of salt restriction

A systematic review of the long term effects of advice to restrict dietary sodium in adults, with and without hypertension, reported only small reductions in blood pressure (-1.1 mmHg (95 % CI = -0.4 to -1.8 mm Hg) for systolic BP and – 0.6 mm Hg (0.3 to -1.5 mmHg) for diastolic BP) at 13 to 60 months of follow-up.[114] The disappointing long-term results are undoubtedly due to subjects' inability to comply with dietary advice to reduce salt intake. As follow-up in trials became longer, the difference in 24-hr urinary sodium excretion between intervention and control subjects became progressively smaller (reduction of 49 mmol/d at 6–12 months of follow-up, compared to 35 mmol/d at 13 to 60 months, and 10 mmol/d in trials which reported follow-up of over 60 months). The results of the review reflect the best-case scenario since the cited trials all used intensive interventions, which are probably unsuited to primary care or population-based prevention programs. However, it was concluded that advice to reduce sodium intake may help people on antihypertensive medication to stop taking their drugs while maintaining good blood pressure control. In this regard, moderate dietary sodium restriction (from 206 to 109 mmol urinary Na/24 h) has been shown to be as effective as the addition of a thiazide diuretic agent, as combination therapy, to the prescription of an ACE inhibitor.[115] The particular advantage of sodium restriction over diuretic therapy is that urinary potassium excretion is not increased in the former.

A randomized controlled trial in 47 elderly people aged 60 – 78 years who were not receiving anti-hypertensive medication found that a modest salt restriction from 10 g per day to 5g per day resulted in a reduction of systolic and diastolic blood pressure by 7.2 and 3.2 mm Hg, respectively, over a four-week period.[116] Importantly, unlike studies in younger subjects, similar falls in blood pressure were seen for both normotensive and hypertensive subjects. A low salt bread was provided to subjects which greatly improved compliance with the reduced sodium regimen.

The findings are consistent with the predictions of Law and colleagues, who estimate that a reduction in sodium intake of 50 mmol/day (about 3 g salt) in older people would lower the population's systolic blood pressure by an average of 5 mm Hg.[117] This magnitude of blood pressure reduction is similar to trials of drug therapy with thiazide diuretics in this age group, in which a 36 % reduction in the 5-year incidence of stroke has been estimated.[118] To prevent one vascular event (particularly stroke) over a given period of time, four times as many younger people need to be treated than those over the age of 60. These studies provide convincing motivation for universal sodium restriction in all older people.

The transduction of the salty taste involves passage of sodium through a specific ion channel in the apical membrane of receptor cells. The channel can be blocked with the drug amiloride, a potassium-sparing diuretic, and is specific; lithium, which can pass through readily, tastes salty, whereas other cations, such as potassium, which do not fit, do not taste salty. This specificity explains the difficulty in finding an acceptable salt substitute. There is some evidence that long-term adherence to a diet low in sodium can lead to a change in taste perception whereby both normotensive[119] and hypertensive[120] persons develop an increased acceptance of foods with a reduced sodium content, presumably because the salt taste receptors become more sensitive, and a lower sodium concentration provides the same salty taste as previously.

Over two decades ago, it was suggested that a relatively easy way to lower habitual sodium intakes in Australians would be to encourage the food industry to use less sodium in bread.[121] An example of a successful partnership in Australia between the food industry and professional bodies in lowering sodium content of foods has recently been reported.[122] In response to a 1982 government report in Australia in which lower levels of sodium intake were recommended for the general population, together with national dietary guidelines in the country and the accreditation criterion of the National Heart Foundation's *Pick the Tick* programme (i.e < 400 mg Na per 100g food), Kellogg's reformulated 12 of their breakfast cereal products to comprise, on average, 40 % less sodium. As a result, 235 tonnes of salt were removed annually from the Australian food supply. Importantly, consumer food appeal was not affected. In many countries, the food industry has been largely resistant to the call by public health advocates and food lobby groups to lower the salt content of processed foods. MacGregor and de Wardener[72] have graphically depicted the economic importance of salt use in processed foods (Figure 2)

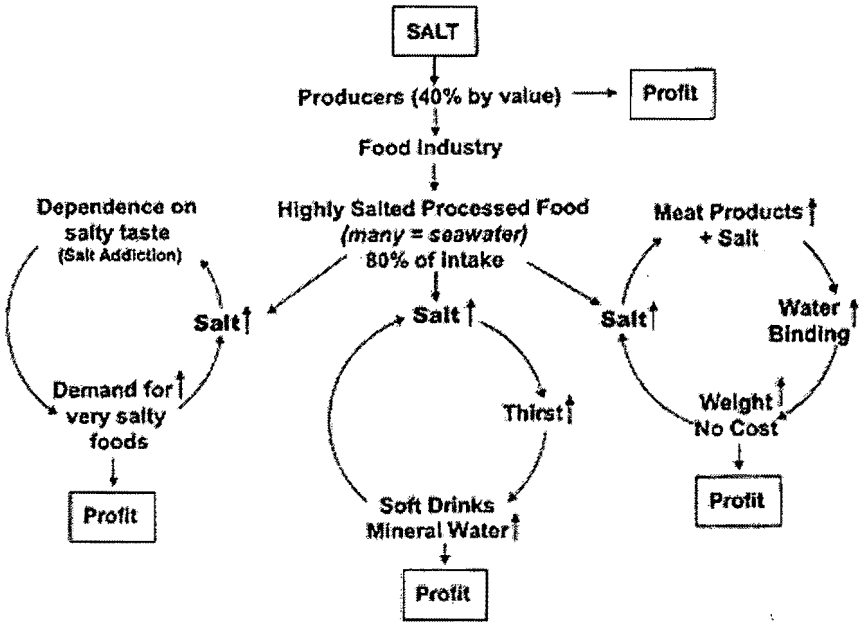

Figure 2
The commercial importance of salt in processed foods, as depicted by MacGregor and de Wardener[72]

There are some encouraging examples of community-based interventions which have been successful in lowering salt intake. The North Karelia project in northern Finland, an area where the incidence of cardiovascular disease was very high, was able to reduce salt and fat intake, increase fruit and vegetable consumption and reduce cigarette smoking through a government-backed campaign which involved the food industry.[123,124] These dietary and lifestyle changes resulted in a reduction in blood pressure and significant falls in stroke and coronary heart disease mortality. Three-quarters of the fall in coronary heart disease and two-thirds of the fall in stroke mortality have been attributed to the change in risk factors.

A Portugese study of two villages has demonstrated that salt intake can be reduced by approximately half by providing a population with information on how to reduce salt intake, particularly from processed sources, and providing processed foods with a reduced salt content.[125] Over two years, the intervention and control (no salt reduction) villages had a significant and substantial difference in blood pressure.

1.4.2 Potassium

Mechanisms of the blood pressure-lowering effect of potassium include its vasodilator activity,[126,127] an increased loss of water and sodium,[128] suppression of secretion of renin and angiotensin,[129] stimulation of the sodium-potassium pump, and

reduction in adrenergic tone.[130] Data from over 60 reports of the blood pressure-lowering effects of potassium supplementation, and from a meta-analysis of randomized clinical trials,[131] suggest that potassium supplementation should be considered more widely in therapeutic and preventive strategies for hypertension, particularly in certain subgroups. A mean increase in urinary potassium excretion of 53 mmol/d resulted in a decrease in systolic and diastolic blood pressure of 3.1 and 2.0 mm Hg, respectively. In almost all the trials potassium was given as a chloride salt supplement, but both the JNC 7 and the 2003 World Health Organization (WHO)[110] dietary guidelines recommend an increased intake of **foods** rich in potassium. The latest JNC 7 guidelines do not specify a recommended intake of potassium, however the earlier version (JNC 6, 1997)[8] recommended an intake of 90 mmol of potassium per day. The WHO report recommends a potassium intake which will keep the sodium:potassium ratio close to one (i.e., a potassium intake of 70–80 mmol/d, if their guidelines on sodium restriction are achieved).[132] The WHO suggest that this potassium intake can be achieved through the adequate daily consumption (400 – 500 g per day) of fruit and vegetables (including beries, green leafy and cruciferous vegetables and legumes), as well as the use of K-enriched low sodium salt substitutes.

The findings of a metabolically controlled study of 38 normotensive men (24 African-American, 14 whites) found that in normotensive black, but not white, men an elevated blood pressure response to sodium occurred when dietary potassium was even marginally deficient, but was dose-dependently suppressed when dietary potassium was increased within its normal range.[132] The study participants were given a basal diet low in sodium (15 mmol/day) and marginally deficient in potassium (30 mmol/day) for six weeks; 250 mmol/day of sodium chloride was added to the diet during the last four weeks, accompanied by potassium supplementation ($KHCO_3$) to either mid- or high-normal (70, or 120 mmol/day, respectively) levels throughout the last three weeks. On the low potassium intake, salt loading induced a mean increase in blood pressure only in black men. Even in the presence of moderate potassium supplementation, salt sensitivity was attenuated similarly in black and white subjects, while supplementation to 120 mmol/day suppressed the frequency and severity of salt sensitivity to levels similar to those in whites.

In studies in which dietary potassium was controlled at normal intakes ranging from 60 to 100 mmol/d throughout the dietary salt loading period, dietary intakes of salt as high as 400 – 600 mmol/d have failed to induce a mean pressor effect in groups of

either black or white normotensive men.[133,134,135,136] Similarly, a marginally deficient dietary potassium intake has been shown to result in an enhanced vasopressor responsiveness to sympathetic stress, induced either by experimental cold or mental stress in normotensive African-American black men who were salt sensitive, but not in normotensive white men who were salt-resistant.[137]

The level of dietary potassium intake which would confer the greatest benefit with regard to protection against sodium-induced pressor effects in different groups has yet to be described. Morris and colleagues[129] have proposed that a "normal" dietary intake of potassium may not be sufficient to suppress expression of salt sensitivity in a substantial number of normotensive black individuals and in fewer normotensive white subjects. In the US, dietary intake of potassium has been found to be lower in inner-city black groups compared to their white counterparts.[138,139]

1.4.3 Calcium

Resnick and colleagues[140] hypothesize that salt sensitivity interacts with calcium metabolism in patients with essential hypertension. A high sodium intake has been shown to be associated with higher blood pressure levels among persons consuming low-calcium diets.[141] The inverse relationship between calcium intake and blood pressure is more convincing at low levels of calcium consumption, i.e. 300 – 600 mg/day.[142] In at least three animal models of hypertension, calcium supplementation has produced a significant decrease in blood pressure, while in human studies the most consistent pressure-lowering effect of calcium has been seen in subjects during a high sodium intake.[143,144,145] Studies performed in normotensive offspring of hypertensive subjects demonstrated altered calcium metabolism when given a high salt diet.[146]

Two meta-analyses of controlled trials have shown that calcium supplementation (1000 to 2000 mg/day) results in small, but significant, reductions in systolic (between –1.7 and -4.3 mmHg) but not diastolic blood pressure.[147,148] Weinberger and colleagues[149] have confirmed a heterogeneity of blood pressure responses of both normotensive and hypertensive humans to calcium supplementation. Among hypertensives, calcium supplementation is more likely to reduce blood pressure in older or black subjects.[150,151,152,153,154] Hypertensives have been shown to have an increase in urinary calcium excretion despite a lower calcium intake,[155] which has been referred to as a renal calcium "leak".

1.4.4 Magnesium

There is evidence, albeit indirect, from both experimental and metabolic studies, to suggest that an increased level of magnesium may be beneficial in lowering blood pressure in otherwise healthy free-living individuals. In vitro studies have shown that magnesium influences cell membrane sodium pump activity, which in turn affects sodium-potassium transport across cell membranes, and subsequently vascular tone and reactivity.[156] Clinical studies have demonstrated significant blood pressure reductions with parenteral high-dose magnesium in patients with eclampsia and glomerulonephritis.[157,158]

In addition, observational epidemiological studies have reported an inverse association between dietary magnesium intake and blood pressure.[159] However, the imprecision of dietary intake reporting, together with the colinearity of magnesium intake with other dietary components that affect blood pressure, limit the interpretation of epidemiological data. Since 1983, many magnesium supplementation trials have been conducted in humans.[160,161,162] Results of these trials have been inconsistent, maybe due to small sample sizes or other design limitations.

A meta-analysis of 20 randomized clinical trials was conducted by Jee and colleagues to determine whether magnesium supplementation reduces blood pressure, to identify the dose-response relationship, and to determine trial characteristics associated with the greatest reductions in blood pressure.[163] The pooled estimate of the effect of magnesium supplementation on systolic blood pressure was a small reduction of 0.6 mm Hg (95 % CI: -2.2 to 1.0 mm Hg). There was a net decrease of 0.8 mm Hg for diastolic blood pressure, but this was not significant (95 % CI: -2.1 to 0.5). A dose-dependent effect of magnesium was found, with reductions of 4.3 rnm Hg and 2.3 mm Hg in systolic and diastolic blood pressure, respectively, for each 10 mmol/day increase in magnesium dose. However, few of the studies were conducted in the higher dose range of magnesium (20 to 40 mmol per day) and the authors of the meta-analysis recommended that properly designed and adequately powered trials at higher dose ranges be performed in order to confirm the inverse dose-response relationship that was detected. The median amount of magnesium administered in the trials included in the meta-analysis was 15 mmol/day (range = 10 – 40 mmol/day), which results in about a doubling of usual magnesium intake derived from the diet (median = 15.6 mmol or 375 mg/day for men

in the United States). This dosage may not, however, be sufficiently large enough to produce a clinically relevant effect.

Another limitation identified by the authors of the meta-analysis was the lack of data on dietary intake of magnesium. In studies that enrolled participants diagnosed with hypertension, subjects may have already increased intake of magnesium-rich foods as a result of advice given by health professionals. In summary, the meta-analysis[77] demonstrated only a small overall reduction in blood pressure associated with magnesium supplementation, but identified an apparent dose-dependent effect of magnesium.

In South Africa, a study published in 1987 reported on the relationship between serum and erythrocyte electrolytes, specifically magnesium and calcium, and blood pressure in 296 urbanized black male labourers in Johannesburg.[164] A significant, inverse association was found between both serum and erythrocyte magnesium and blood pressure, as well as between both serum calcium and potassium and blood pressure. Of all the electrolytes assessed, magnesium (either serum or erythrocyte) correlated most closely with blood pressure. The authors concluded that body magnesium status, and its interactions with calcium, sodium and potassium, may play an important role in the development and maintenance of elevated blood pressure in the South African black population.

1.4.5 Composite dietary interventions to prevent and manage hypertension

The Dietary Approaches to Stop Hypertension (DASH) randomized controlled trial provided unequivocal evidence that non-pharmacological methods can reduce blood pressure as much as some anti-hypertensive drugs.[62] Subjects fed a diet rich in fruit and vegetables for 8 weeks significantly reduced systolic and diastolic blood pressure by 2.8 and 1.1 mm Hg more, respectively, than subjects on a typical American control diet. Subjects randomized to the DASH diet, rich in fruit, vegetables and low fat dairy products, and with a reduced saturated and total fat intake (Table 3), had an even greater reduction in both systolic and diastolic blood pressure (5.5 and 3.0 mm Hg, respectively). It was estimated that a population-wide reduction in systolic or diastolic blood pressure of the magnitude observed with the DASH diet would reduce incident coronary heart disease by approximately 15 % and stroke by about 27 %. It is noteworthy that the effects of the 8-week DASH diet were greatest in the hypertensive

African-American sub-group, in which a blood pressure reduction of 13.2/6.1 mmHg was demonstrated.[165] Increased efficacy of the DASH diet among African Americans supports other data suggesting racial differences in blood pressure response to diet. For example, African Americans tend to consume less potassium than their white counterparts which may explain some of the increased efficacy of an increased fruit and vegetable intake, together with an increased dairy food (calcium) intake in this segment of the population.[166]

Table 3
The Dietary Attempts to Stop Hypertension (DASH) diet[62]

Food group	Daily servings	Serving sizes	Examples and notes	Significance to the DASH diet pattern
Grains and grain products	7-8	1 slice bread ½ cup dry cereal ½ cup cooked rice, pasta or cereal	Wholewheat bread, muffin, pita bread, bagel, cereals, oatmeal	Major sources of energy and fibre
Vegetables	4-5	1 cup raw, leafy vegetables ½ cup cooked vegetables 6 oz vegetable juice	Tomatoes, potatoes, carrots, peas, squash, broccoli, turnip greens, kale, spinach, artichokes, green beans, sweet potatoes	Rich sources of potassium, magnesium and fibre
Fruits	4-5	1 medium fruit ¼ cup dried fruit 6 oz fruit juice ½ cup fresh, frozen or canned fruit	Apricots, bananas, dates, oranges, orange juice, grapefruit, grapefruit juice, mangoes,melons, peaches, pineapples, prunes, raisins, strawberries, tangerines	Important sources of potassium, magnesium and fibre
Low-fat or non-fat dairy foods	2-3	8 oz milk 1 cup yoghurt 1.5 oz cheese	Skim or low fat (2 %) milk, skim or low fat buttermilk, nonfat or low-fat yoghurt, nonfat or low fat cheeses	Major sources of calcium and protein
Meats, poultry, and fish	≤ 2	3 oz cooked meats, poultry or fish	Select only lean meats; trim away visible fats; broil, roast, or boil, instead of frying; remove skin from poultry	Rich sources of protein and magnesium
Nuts, seeds and legumes	4-5/wk	1.5 oz 1/3 cup nuts 1/2 oz or 2 Tbsp seeds, ½ cup cooked legumes	Almonds, mixed nuts, peanuts, walnuts, sunflower seeds, kidney beans, lentils, split peas	Rich sources of energy, magnesium, potassium, protein, fibre

The DASH eating plan shown above is based on 2000 kcal a day (8 400 kJ/d). Depending on energy needs, the number of daily servings in a food group may vary from those listed.
*Table adapted from the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.[33]

The follow-up DASH Sodium study investigated the additional benefits of salt restriction over and above the merits of the DASH diet.[167] Reducing sodium intake from a high (150 mmol/day) to either an intermediate (100 mmol/day) or low (65 mmol/day) intake resulted in a stepwise reduction in blood pressure, which was approximately twice as great in subjects on the control than on the DASH diet (Figure 3).

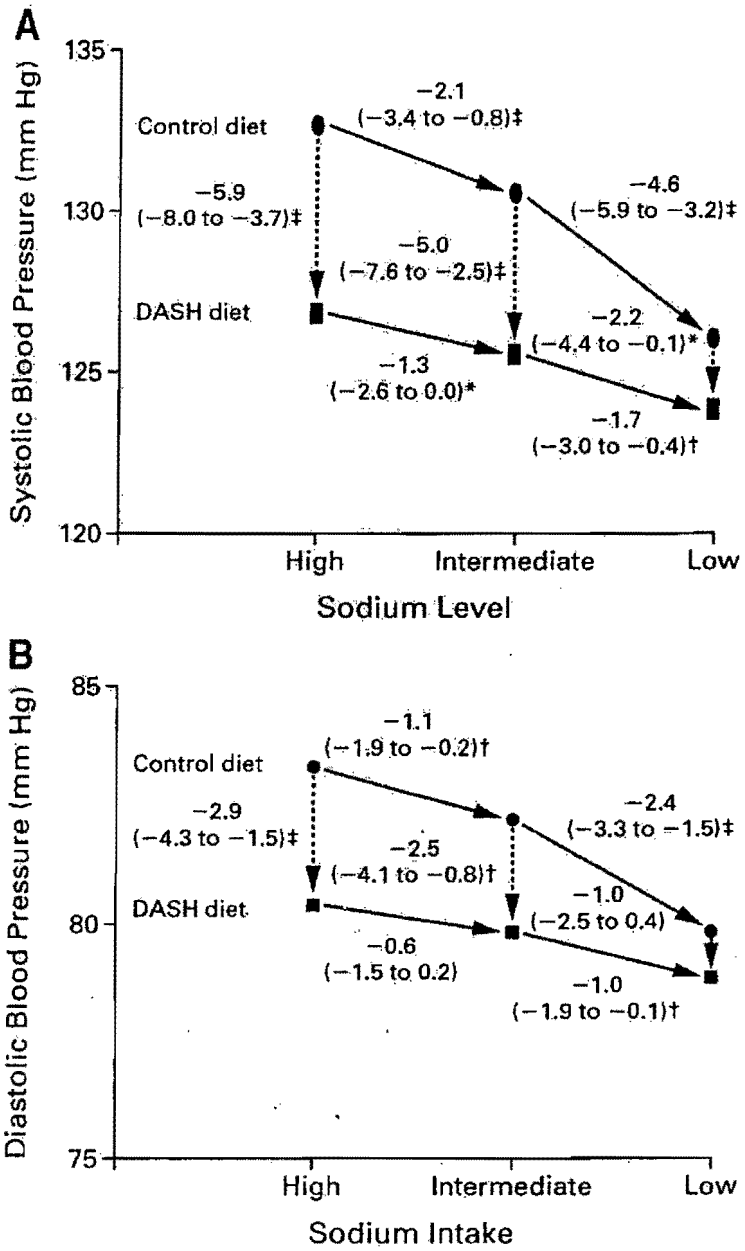

Figure 3
The effect on systolic blood pressure (panel A) and diastolic blood pressure (panel B) of reduced sodium intake and the DASH diet (Taken from Sacks *et al.*, *N Engl J Med* 2001;344:4.[167]).

Asterisks (P<0.05), daggers (P<0.01), and double daggers (P<0.001) indicate significant differences in blood pressure between groups or between dietary sodium categories.

Sodium restriction from high to low intake in those following the DASH diet resulted in a relatively small additional drop in blood pressure of 3.0 and 1.6 mm Hg for systolic and diastolic blood pressure, respectively. It may be concluded that the greatest benefits in sodium restriction are seen in those with a poor diet (ie. typical "American" high-fat, low nutrient-dense diet), and that subjects who include a large amount of fruit and vegetables, together with low fat diary products, may be able to tolerate higher amounts of salt. This compelling evidence further supports the shifting paradigm in provision of nutrition messages to the (often confused) public, in that a holistic approach needs to be taken, rather than the targeting of individual messages.

In a subsequent study, the Diet, Exercise and Weight loss Intervention Trial (DEW-IT),[168] overweight hypertensives demonstrated a net change of 9.5/5.3 mm Hg in ambulatory blood pressure, using a low-calorie version of the DASH diet, in combination with weight loss. In a fourth dietary intervention trial (PREMIER) which included the DASH diet, the effects of combining the DASH diet with "established" lifestyle modification recommendations (weight loss, exercise, and restriction of Na and alcohol) was investigated.[169] Surprisingly, the addition of the DASH diet to lifestyle modification resulted in an incremental decrease of blood pressure of only 0.6/0.9 mmHg (1.7/1.6 mmHg in hypertensive individuals).

Pickering, in an editorial which appeared in the same issue of the *JAMA* as the PREMIER results,[170] offers possible explanations why the effects of combined interventions on blood pressure do not appear to be additive. Patients in the PREMIER study were not following the DASH diet as closely as in the three earlier DASH studies. PREMIER is the first study to intentionally investigate the effects of the DASH diet when patients are required to purchase their own food, rather than be provided with carefully prepared meals. In the original DASH study, patients were provided with 9.6 daily servings of fruits and vegetables, whereas in the PREMIER study, the intake increased from 4.8 servings at baseline to 7.8 servings. This was evident in the difference in urinary potassium excretion in the two studies, whereby there was a 105 % increase in the original DASH study, but only a 28 % increase in the PREMIER study. Similarly, the effects of sodium restriction in the DASH Sodium follow-up study greatly exceeded the changes observed in almost all other studies in which participants prepared their own low salt meals, presumably due to greater compliance with a low sodium regimen.

The authors of the PREMIER study suggest a "subadditivity of intervention effects." In other words, the combination of two or more interventions has a smaller effect on blood

pressure than the sum of the effects of the individual interventions. In the DEW-IT study, in which obese hypertensives were given a hypocaloric version of the DASH diet and a mean weight loss of 5.5 kg was achieved, the net reduction in blood pressure was comparable to the effects of the DASH diet alone. In the Trials of Hypertension Prevention II (TOHP II) study, the effect of adding sodium restriction to weight loss produced no further decrease of blood pressure, even though moderate sodium restriction alone produced a modest, but significant, decrease.[171] Similarly, the DASH Sodium trial demonstrated that although the DASH diet had an additive blood-pressure lowering effect when combined with sodium restriction, the combined effect was not as great as that estimated on the basis of strict additivity.[167]

It is often assumed that most individuals are not capable of changing more than one lifestyle factor at a time. However, it is probable that participants in the DASH Sodium trial were diligently consuming the prescribed diet since all food was provided. Similarly, in the Trials of Hypertension Prevention Trial (TOHP) II trial,[171] which comprised three intervention groups (weight loss, sodium restriction, and combination), the magnitude of weight loss and reductions in urinary Na excretion were only marginally less in the combined group than the other two single intervention groups. This indicates that subjects were able to simultaneously achieve both lifestyle changes.

An alternative explanation for a lack of additive effects is that different lifestyle interventions may act through the same physiological interventions, thus resulting in a nonlinear dose-response relationship.[170] Much the same way as doubling the dose of anti-hypertensive drugs produces only a small further decline in blood pressure,[172] a combination of lifestyle modifications (such as weight loss, increase in physical activity patterns and altering the cation composition of the diet) may all be operating through the same physiological pathway. However, much remains to be learned regarding the mechanisms by which obesity and other lifestyle factors increase blood pressure.[173]

It has consistently been shown, in intervention studies conducted in the United States, that African-American individuals with raised blood pressure benefit the most from non-pharmacologial interventions, such as dietary changes and weight loss. Evidence of the impact of such interventions on blood pressure is awaited from other ethnic populations. It is likely that the DASH diet cannot be applied to ethnically diverse populations in a "one size fits all" manner. As with all food-based dietary

guidelines, recommendations for dietary change need to take into account affordability, accessibility, and sustainability, as well as cultural acceptability of the dietary patterns being promoted. To date, no dietary intervention to lower blood pressure has been conducted in the South African black population.

1.5 Alcohol and blood pressure

Epidemiological studies over the past two decades have firmly established a relationship between regular, excessive alcohol consumption and hypertension. This association has been found in both sexes and in those of differing ethnicity, and is independent of the type of alcoholic beverage, adiposity, education, smoking, salt intake, and several other factors.[174] It has been shown that a habitual intake of alcohol greater than 30 to 60 g per day (i.e., about 2.5 - 5 alcoholic drinks) results in blood pressure elevation in both men and women. A rule of thumb can be derived: for subjects who consume an habitual intake of 30 g (about 2.5 drinks) or more of alcohol per day, an increment of 10 g alcohol per day increases systolic blood pressure by an average of 1-2 mm Hg and diastolic blood pressure by 1 mm Hg. As well as its direct effect on blood pressure, alcohol can cause resistance to antihypertensive therapy[175] and is a risk factor for stroke.[176] The US National High Blood Pressure Education Program (NHBPEP) recommends that in those who drink alcohol, an intake of no more than 25 g of alcohol (2 drinks) be consumed a day for men and no more than 15 g of alcohol per day for women and lighter-weight people (see Table 2).[33]

1.6 Weight reduction and blood pressure

Weight reduction has been the single most effective component utilized in large-scale lifestyle approaches to the reduction of blood pressure, such as in the Trials of Hypertension Prevention (TOHP) Phases I[177] and II[171] studies and the Trial of Antihypertensive Interventions and Management (TAIM) study.[178] A systematic review of all randomized trials of nonpharmacological interventions that included at least 6 months of follow-up revealed that net blood pressure reductions were greatest for trials of the effects of weight loss, averaging a 5.2 mm Hg reduction in systolic blood pressure.[179] It has been estimated that for every 1kg decrease in body weight, obese hypertensive patients can expect a decrease in blood pressure of 2.4/1.5 mm Hg.[180] A subsequent meta-analysis reported that a loss of 3 to 9% of body weight in overweight hypertensive subjects is associated with blood pressure reductions of approximately 3 mm Hg in both systolic and diastolic blood pressure.[181]

Weight reduction may decrease dosage requirements for antihypertensive medications. It is not yet clearly established whether weight reduction is superior to sodium restriction and/or potassium supplementation and/or exercise in reducing blood pressure. It is unlikely that weight loss alone will achieve blood pressure control in patients with Stage 2 hypertension, or in those who are not sufficiently motivated to lose weight.

Various explanations have been proposed to describe the mechanisms between obesity, increased sensitivity to dietary sodium and increased blood pressure levels. Obesity has been linked with suppressed kallikrein levels, an enzymatic precursor of bradykinin and nitric oxide formation,[182,183] as well as with increased serum angiotensin converting enzyme (ACE) or kinnase II activity. Compared with their nonobese counterparts, obese Jamaicans (BMI >31 kg/m^2) have been shown to have higher serum ACE activity and angiotensinogen levels than their non-obese counterparts.[184] It is hypothesized that obese persons have more renin substrate (i.e. angiotensinogen) and maybe greater conversion of angiotensin I to angiotensin II, along with accelerated breakdown of bradykinin to inactive metabolites, leading to impaired nitric oxide (NO) production.

There is limited evidence regarding the genotype-phenotype association of obesity and hypertension in black South Africans. Angiotensinogen (AGT) is expressed in adipose tissue, and obesity is a powerful risk factor for hypertension. Tiago and colleagues[185] investigated whether some AGT gene variants may influence blood pressure in our local population. Five hundred and twenty one black hypertensive South Africans with a high BMI were compared with 547 normotensive black control subjects. The AGT promoter region variant gene was present with a highly significant Odds Ratio in those black hypertensives with a BMI of over 27 Kg/m^2. The AGT gene expressed in adipose tissue would directly accelerate the production of angiotensin II, as well as increase leptin production, The sympathetic nervous system would thus be stimulated, resulting in vasoconstriction and increased plasma volume, leading to hypertension. Promising work that suggests that genetic determinants of hypertension may be located at the adipocyte level and influence local renin-angiotensin production is increasing. However, the complex, multifactorial nature of hypertension probably involves many gene polymorphisms, interacting at different levels.

Adiposity has been shown to be associated with central sympathetic overactivity (which is a recognized intermediate phenotype for incident hypertension) in young, normotensive African American women.[186] Adiposity accounted for one third of the inter-individual variability in sympathetic nerve discharge rates. In lean African-American men, on the other hand, sympathetic nerve discharge rate is 20 – 40 % higher than in lean white men or in either lean black or white women, and the sympathetic overactivity is independent of body mass index.[186] Thus, there is microneurographic evidence of differences in sympathetic nerve activity (at least in the skeletal muscle vasculature) between black and white Americans, however similar studies have yet to be performed in the South African context.

Another emerging body of evidence relates to the low birthweight hypothesis, whereby an adverse intrauterine environment programs the foetus to an increased susceptibility in later life to components of the metabolic syndrome, such as hypertension. An inverse association between birthweight and blood pressure was first described by Barker and colleagues,[187] and this has now been confirmed in many other studies around the world.[188,189,190,191,192] In developing countries, one possible method of primordial prevention of hypertension may be to ensure adequate weight gain and appropriate nutrient intake in women during pregnancy.

Obesity has been linked with sensitivity to sodium. Findings from phase 1 of the Trials of Hypertension Prevention (TOHP) study, a randomized non-pharmacological trial conducted in 2 182 normotensive adults aged 35-54 y, have shown that weight loss and sodium reduction were well-tolerated and produced significant declines in systolic and diastolic blood pressures (-2.9/-2.4 and -2.1/-1.2 mmHg for weight loss and sodium restriction, respectively).[193] Data from the Coronary Artery Risk Development in Young Adults [CARDIA] study indicate that African-American women have a 2.7 kg/m^2 higher age-adjusted mean BMI than white American women, a significantly higher energy intake, lower levels of physical activity and lower physical fitness (assessed according to duration on exercise treadmill test).[194] Similarly, black South African women have been shown to have the highest prevalence of obesity (BMI ≥ 30) compared to other population groups in the country (34 % in the 15 - 64 y age group, rising to 59 % in adults aged 45-54 y).[18]

As well as differences in the prevalence of overweight or obesity in African-American and black South African women compared to their white counterparts, differences in body composition have been shown. African-Americans have a greater density of fat-

free mass because of a heavier and denser skeletal mass and denser muscle mass.[195,196] Mueller[197] found that African-American children have a more central pattern of fat deposition than do white children from pre-school age through adolescence and into adulthood, and that these trends in fat deposition patterns were independent of fitness, socioeconomic status and age. It is well established that upper body obesity, and particularly visceral adipose tissue (VAT), is associated with increased metabolic and cardiovascular risk in white women.[198] However, little is known about patterns of intra-abdominal deposition of VAT and subcutaneous adipose tissue (SAT) in both African-American women and black South African women, and more specifically, how regional body fat composition affects blood pressure response to dietary manipulations.

A study by Lovejoy *et al.*[199] has shown that in a sample of 37 African-American and 22 white women, matched for age and BMI, visceral fat area (measured by computed tomographic scans) was smaller in the African-American women, despite a similar waist-to-hip ratio. Visceral fat correlated with metabolic risk factors, such as insulin resistance, but subcutaneous abdominal fat sensitivity was significantly correlated with insulin resistance and fasting insulin only in African-American subjects, suggesting that total adiposity rather than intra-abdominal fat stores influence metabolic risk in this population.

1.7 Physical activity and blood pressure

To complete the discussion on lifestyle modification to reduce blood pressure, the effects of physical activity is briefly reviewed (Table 2). The JNC 7[33] guidelines on lifestyle modification provide guidelines on intensity and duration of recommended activity levels for the control of blood pressure. It is well established that lack of physical activity is associated with at least a 1.5-2.0 fold higher risk of hypertension and coronary heart disease.[200] A meta-analysis which included only trials in which the exercise intervention had lasted 4 wk or longer concluded that exercise reduced systolic blood pressure by 4.7 mm Hg and diastolic blood pressure by 3.1 mm Hg.[201] In another meta-analyses of over 68 studies, representing more than 1500 patients, the effect of exercise training on blood pressure in normotensive subjects has been shown to average about 3.0 and 1.7 mmHg for systolic and diastolic blood pressure, respectively.[202] Changes in blood pressure associated with exercise training are even more marked in those who are already hypertensive (7.8 and 5.8 mmHg for systolic and diastolic, respectively). After 8 weeks of exercise training in hypertensive

patients, it was found that blood pressure response to an increasing dose of physical activity was sigmoidal with a peak effect at 90 minutes, after which there was no further improvement.[203] Mechanisms which have been implicated in the effect of chronic exercise on blood pressure control include reductions in vascular resistance secondary to neurohumoral and structural adaptations.[204]

Ideal exercise intensity for blood pressure-lowering effects ranges between 40-70% of maximal, age-predicted heart rate.[205] There is little or no evidence for extra benefit from higher-intensity exercise (>70% maximal oxygen uptake) or from more than three bouts of exercise per week. However, regarding acute effects of exercise on the attenuation of post-exercise blood pressure, higher intensity (75% maximum) exercise has been shown to be associated with a more marked and prolonged reduction in blood pressure in the post-exercise window, compared to lower intensity exercise (50% maximum).[206] In the acute post-exercise period, the reduction in blood pressure has been linked to a sympathetic inhibition and increased release of vasodilator substances.[207]

The challenge for most individuals is to maintain long-term compliance with exercise programmes. In this regard, the encouragement of low-intensity activities which can be incorporated into everyday lifestyle (such as walking instead of using the car), rather than structured high-intensity aerobic exercise programs, shows more promise and appears to result in comparable reductions in blood pressure.[208] For previously sedentary individuals, a prudent approach would be to commence exercise cautiously and at low intensity (40–50% of maximal oxygen uptake). In high-risk individuals, an initial, thorough medical evaluation, including an electrocardiogram is advised prior to beginning an exercise program.

For both the prevention and management of hypertension, the current JNC 7 guidelines are based on those of the American College of Sports Medicine (ACSM).[209] Moderately intense physical activity (40–60% maximal oxygen uptake) is recommended on most or preferably all days of the week, for greater than 30 minutes of continuous or accumulated moderate physical activity per day. For maximum benefit, the type of exercise for hypertensives should be predominantly endurance physical activity including walking, jogging, cycling, swimming, or dancing, supplemented by resistance exercise, prescribed according to the ACSM or American Heart Association guidelines.[210]

1.8 Conclusions

Undoubtedly, nonpharmacological lifestyle changes, mostly diet related, represent a safe and effective approach to blood pressure reduction, and offer broad advantages for lowering cardiovascular risk. Furthermore, using this approach, the need for antihypertensive medications can be decreased in subjects with established hypertension. Dietary advice for the prevention and management of hypertension needs to address changes in dietary patterns as a whole, rather than focusing on one or more nutrients. It appears that the most beneficial dietary pattern is a DASH-type diet that is low in total and saturated fat and alcohol (and reduced in total energy, if the subject is obese), and high in fibre, potassium, calcium, and magnesium, and moderately high in protein. In terms of foods, this translates into a diet rich in fruit and vegetables and low-fat dairy foods. Regarding sodium restriction, the greatest benefits are seen in those with a diet of poor quality (high in fat and low in nutrient density), However various subgroups, such as elderly hypertensives, obese individuals, and subjects of African descent, may particularly benefit from reducing dietary sodium intake.

In order to empower and encourage individuals to make sustainable, lifestyle changes, a systematic team approach is required, utilizing health-care professionals and community resources wherever possible, so as to provide necessary education, support, and follow-up. Nutrition professionals and legislators involved in food policy development need to work closely with the food industry to develop and market products which are reduced in sodium, or which contain an optimal combination of cations known to be beneficial to blood pressure reduction, namely potassium, magnesium, and calcium.

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Chapter 2

Rationale, study objectives and overview of study design

2.1 Rationale

Black South Africans are at high risk of hypertension, stroke and blood pressure-related target-organ damage. In South Africa, the limited resources at primary health care level allocated to the prevention, early diagnosis and management of hypertension necessitate a non-pharmacological population-based approach to curb the escalating burden of cardiovascular disease, for which raised blood pressure is an important major contributory risk factor.

It has been shown, in other populations, that dietary changes are able to lower blood pressure in both subjects with and without hypertension. The most important dietary component influencing blood pressure is dietary sodium. Law and colleagues estimate that a reduction in sodium intake of 50 mmol/day (about 3 g salt) in older people would lower a population's systolic blood pressure by an average of 5 mm Hg.[1] This magnitude of blood pressure reduction is similar to trials of drug therapy with thiazide diuretics in this age group, in which a 36 % reduction in the 5-year incidence of stroke has been estimated.[2] Other dietary factors which may contribute to lowered blood pressure levels (if their intakes are increased) are potassium, magnesium and calcium.

The effect of lowering dietary sodium intake, while simultaneously increasing potassium, calcium and magnesium intake, on blood pressure is presently unknown in the black South African population. The identification of a dietary pattern which involves relatively few behavioural changes and which is based on commonly consumed food items that are affordable and culturally acceptable to the poorer section of the black South African population is paramount to the long-term sustainability and thus effectiveness of diet-related strategies to lower blood pressure in this target group.

The series of studies included in this thesis provide a systematic approach to developing an appropriate nutritional population-based approach to lowering blood pressure in a high risk population. Firstly, valid, reliable, and updated information is required to identify habitual intake of sodium, potassium, magnesium and calcium in the target population, using the gold standard method of assessing sodium intake, namely 24-hour urinary excretion collections. This information is necessary to inform the levels of sodium and other cation modification required in order to obtain a physiologically relevant change in blood pressure. As well as quantitative data on levels of sodium intake, information is also required on which food sources are the most important contributors to overall non-discretionary salt intake, and the pattern of intake of these foods. This data will allow

identification of commonly consumed foods that can be targeted for modification of their cation content.

The collection of 24-hr urinary samples to determine sodium intake is not feasible on a large scale in a developing country such as South Africa due to cost constraints, methodological difficulties and generally low education levels. Since dietary surveys are time-consuming and costly, the development of a rapid questionnaire that is able to accurately assess salt intake in the local population would be useful for both research and clinical purposes. Together with secondary analyses of previously published dietary surveys, data obtained in the dietary and urinary studies in this body of work will be used to develop such a tool.

Next, in partnership with the food industry, the identified important commonly consumed food sources of salt will be altered in cation content and tested for sensory properties, product quality and nutritional content. Finally, the blood-pressure lowering effect of substitution of these novel food products for the standard varieties will be investigated in a randomised controlled human feeding trial. The effectiveness of the health benefits of the products will be evaluated in the context of a community-based dietary intervention in which subjects are free-living within their usual socioeconomic living conditions.

Results of the studies included in this thesis which have been planned and partly funded by two large food companies will be used to lobby the food industry to develop new products and to reformulate existing products which will improve blood pressure profiles in the country. It is anticipated that the food industry will embrace the opportunity to use the scientific data to develop widespread marketing campaigns in this regard, and provide a competitive edge in the market, while at the same time, enabling them to be socially responsible in terms of promoting health.

2.1 Overall aim

To identify patterns of food consumption in the black South African population in order to improve the cation content of commonly consumed food items and to evaluate the impact on blood pressure of these foods when substituted for the standard varieties. The ultimate aim is to lower blood pressure on a population level through a food-based public health strategy.

2.2 Objectives

The thesis comprises five separate studies, the content of which will be published as individual manuscripts in peer-reviewed indexed scientific journals. The data presented in Chapters 3 and 4 has already been published in international peer-reviewed journals,[3,4] in a shorter, modified format. In both these papers, Karen Charlton was the principal author who was responsible for the study design and conceptualization, fieldwork execution, data analyses and write-up of the work. Co-authors of the papers included doctoral supervisors, fieldworkers and a consultant statistician.

The titles and objectives of the studies are outlined below:

Study 1

Ethnic differences in dietary intake and urinary excretion of sodium, potassium, calcium and magnesium in South Africans

(1) To quantify the habitual dietary intake and urinary excretion of sodium, potassium, calcium and magnesium in three South African ethnic groups;

(2) To investigate whether cation intake and excretion differs according to ethnic and hypertensive status;

(3) To investigate whether cation excretion is a predictor of BP according to ethnic and hypertensive status.

Study 2

The contribution of foods to total sodium, potassium, calcium and magnesium intake in the South African diet

(1) To identify which food sources are the major contributors to sodium, potassium, calcium and magnesium intake;

(2) To perform secondary analyses on existing dietary databases of South African adults in order to identify ethnic and urban:rural differences in sources of sodium intake.

Study 3

Development and validation of a questionnaire to assess sodium intake

(1) To develop a food frequency-type short questionnaire to assess habitual dietary salt intake in South Africans, using secondary dietary data analyses and data from a new, multi-ethnic study.

(2) To determine a simplified scoring system for the questionnaire which will allow classification into two categories of intake (desirable and excessive).

(3) To demonstrate criterion validity of the questionnaire by comparing it with 24-hr urinary Na excretion data and reported Na intake obtained from repeated 24-hr dietary recalls;
(4) To demonstrate internal consistency and reliability of the questionnaire.

Study 4

Development of foods with altered cation content: bread, margarine, stock cubes, soup mix and monosodium glutamate-based flavour enhancer (Aromat)

(1) To develop reduced sodium (with increased potassium, magnesium and calcium content) varieties of five commonly consumed food items that are major contributors to overall non-discretionary sodium intake in the diet of the black South African population.
(2) To evaluate the sensory properties and physical qualities of the products with a modified cation content, compared to the standard varieties thereof.

Study 5

The impact of dietary manipulation of sodium, potassium, magnesium and calcium on blood pressure in hypertensive black South Africans: a randomised controlled trial

(1) To investigate the impact on blood pressure in black hypertensives of a randomised, parallel arm, double blind controlled trial of 8 weeks duration, in which a dietary intervention of reduced sodium and simultaneous increase of potassium, magnesium and calcium is achieved by providing commonly consumed foods with a modified cation content.

Each of the papers is described in entirety in a separate chapter (Chapters 3 - 7) in the thesis, and a combined conclusion of the main results is provided, in the context of their public health impact, in Chapter 8. Abstracts of the papers have been combined in a single Executive Summary at the beginning of the thesis. Challenges facing the various sectors are also outlined in Chapter 8, as are recommendations regarding policy development and other appropriate strategies.

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Chapter 3

Ethnic differences in dietary intake and urinary excretion of sodium, potassium, calcium and magnesium in South Africans

Introduction

It is estimated that approximately 6 million of the total population of 41 million South Africans are hypertensive.[1] Black hypertensive patients in South Africa are prone to cerebral haemorrhage, malignant hypertension, kidney disease leading to uraemia and congestive heart failure, whereas coronary heart disease (CHD) is relatively uncommon.[2] This contrasts with the situation in the white and Indian communities, where CHD is the major outcome related to hypertension. The prevalence of hypertension (age-adjusted to the world population) in the adult black population (24.4 %) is almost as common as in the white population (27.6 %).[3] However, the absolute number of hypertensives is almost four times higher in the black compared to white population in the country,[3] which places a major burden on health services in the public sector. Not only is hypertension common in the black population, but diagnosis and management of the condition in this group is particularly poor.[3,4,5,6]

Regarding lifestyle-related risk factors for hypertension, the Kenyan Luo migration study of Poulter *et al.*[7] demonstrated that migration of people living in traditional rural villages on the shores of Lake Victoria to the urban settings of Nairobi was associated with an increase in BP. Urban migrants had higher body weights, pulse rates and urinary sodium/potassium ratios than those who remained in the rural areas. This data suggests a marked change in the diet of the new arrivals in Nairobi, namely a higher salt and kilojoule intake together with a reduced potassium intake due to the consumption of less fruit and vegetables.[4,8] Duration of urbanisation independently predicted the presence of hypertension in black peri-urban communities in Cape Town,[9] but other authors have not found urban/rural differences in the prevalence of hypertension in black South Africans.[10] More recently, the THUSA study conducted in the North West province of South Africa found that the highest level of BP was observed in the group of newcomers to the urban setting and that factors related to urbanisation were positively associated with hypertension.[11] Factor analyses of the data demonstrated that a high sodium intake, together with high intakes of saturated fat, animal protein, vitamin A and B_6, was identified as the most important cluster of risk factors relating to hypertension in this population.[12]

Steyn and colleagues[13] have highlighted the importance of adjusting for differences in age, gender and socioeconomic factors when considering whether or not ethnic differences exist regarding the risk of developing hypertension. Logistic regression modeling of the large, nationally representative Demographic and Health Survey dataset

demonstrated that, apart from a lower prevalence of hypertension in rural African participants, no other ethnic differences were apparent after the influence of gender, education, urbanisation (in the African group), BMI, family history and excessive alcohol use were included. This is consistent with the between-population epidemiological studies of Cooper *et al.* which have demonstrated a wide variation in hypertension prevalence among both white and black ethnic groups internationally.[14] A gradient in the prevalence of hypertension among black populations from Nigeria, Jamaica and the US provides evidence of the association between blood pressure levels and transition to an industrialized lifestyle.[14] The authors suggest that the impact of environmental factors among both ethnic groups may have previously been under-estimated and that African-origin populations are not inherently at increased risk of developing hypertension. This shift in conventional wisdom highlights the importance of promoting a healthy lifestyle in an attempt to halt the deterioration of the global cardiovascular disease risk factor profile of populations undergoing transition.

The literature review of this thesis (Chapter 1) has provided extensive evidence that dietary factors are related to hypertension, particularly a high sodium intake, a low potassium intake (mostly due to too few fruits and vegetables) and an excessive alcohol and kilojoule intake. Seedat[15] suggests that black people of African origin have an abnormal transport mechanism of sodium, accompanied by a low renin activity. Studies conducted in hypertensive patients in South Africa[16,17] have found diminished activity of the sodium-potassium ATPase pump in black hypertensives. The clinical observation that black South African[18] and African-American[19] hypertensives manifest higher average BP responses to calcium antagonists than to ACE inhibitors supports the hypothesis that hypertension amongst these groups is often salt-sensitive.

Meta-analyses of intervention trials of either Na restriction[20,21,22,23,24,25] or K supplementation[26] have provided evidence that dietary manipulation of these two factors is key in the BP-diet relationship. In order to plan appropriate public health interventions to prevent and manage hypertension in South Africa, information is required on the habitual intake of sodium, potassium and other nutrients which are important determinants of blood pressure. The main objectives of the study were (1) to determine whether the habitual urinary excretion and dietary intake of sodium, potassium, calcium and magnesium differs in three South African ethnic groups; (2) to assess the proportion of salt intake which is discretionary; (3) to investigate whether the BP-cation association varies, according to ethnic status. Other secondary objectives were to assess inter- and intra-individual variability in BP and urinary Na excretion (for the purpose of sample size

determination in dietary intervention trials), and to determine renin and aldosterone status across ethnic and hypertensive groups.

Methods

Subjects and sampling

Sample size was determined using published data (1982) on mean urinary Na excretion values in black normotensive South Africans.[27] Assuming a mean Na excretion value of 126.8 (SD = 55) mmol/day, a desired standard error of 5.47 mmol/day (acceptable margin of error in expected mean is 30 mmol/day) and a precision of 95 %, sample size was estimated as $n = sd^2/e^2$ where s^2 is the between-subject variance and e is the desired standard error (a measure of precision required for the estimate of the mean).[28] A sample size of 100 was required. In order to compare urinary Na excretion across ethnic groups, 100 subjects per group was required. In total, three hundred and twenty five normotensive and hypertensive (BP ≥140/90 mm Hg and/or on antihypertensive medication) men and women (black, mixed ancestry and white), aged 20 - 65 years, were recruited from their place of work using a stratified, convenience sampling method. The study was designed so that there were approximately 50 people in each ethnic and sex group stratum, and that equal numbers of hypertensive and normotensive subjects would be recruited in each of the 6 strata. The sampling frame comprised employees of the Cape Town City Council, which includes a total of about 4 000 people. Eligible volunteers were invited to attend for blood pressure screening. Compliance with the study protocol was improved by having two fieldworkers (nurses) housed within the in-house clinic facility of the office building where all data collection took place. Approval to conduct the study was obtained from the management of City Council, as well as from representative trade unions of employees. The Research and Ethics Committee of the University of Cape Town approved the study protocol, and written informed consent was obtained from all participating subjects.

To determine habitual urinary Na, K, Ca and Mg excretion, subjects were required to collect three 24-hour urinary volumes over a consecutive 3-week period. As a marker of completeness of collection, 3 tablets (450 mg/day) of non-metabolizable para-aminobenzoic acid (PABA; Laboratories for Applied Biology, London) were given to the subjects, to be taken with meals during the collection period.[29] Urinary electrolyte concentration was measured using flame photometry and PABA measured calorimetrically. Urine collections were excluded from the analyses for that day of collection if the volume was ≤ 500 ml (n = 9 samples), or if either (1) urinary creatinine

values <0.2 mmol/kg/day and PABA ≤ 97 % or (2) urinary creatinine values = 0.2 – 0.3 mmol/kg/day and PABA ≤ 75 % (n = 24).[30] Three complete collections were obtained in 44.3 %; two in 27.8 %; and one in 16 % of subjects. Twelve percent of subjects had no usable urinary data.

Dietary intake was assessed using an interviewer-administered 24-hour recall method, for each of the three urinary collection periods. Standard household measuring utensils, rulers and food photographs of typical South African foods (developed and validated by Venter *et al.*, 2000[31]) were used to quantify food portion sizes. The recorded quantities of food consumed were converted to gram weights using the Medical Research Council (MRC) Food Quantities Manual.[32] Average daily nutrient intake was calculated using the Foodfinder III computerised dietary assessment programme, based on MRC Food Composition Tables.[33,34] Discretionary salt use was not quantified in the dietary assessments. Regarding added salt consumption, subjects were asked whether salt was usually added to their food during cooking, whether salt was usually added to food before tasting and whether they liked their food to taste "*very salty*," "*a little salty*" or "*not at all salty.*"

Blood pressure was measured on each of the three occasions when urine samples were returned to the data collection office, using an automated Omron M1 electronic blood pressure manometer (Omron Life Science Co. Ltd, Tokyo, Japan). Blood pressure measurements were taken three times from the left arm, with the palm upward, resting on a table or support at the level of the heart, after the participant had been seated for at least 5 min. A large cuff size was used for subjects with a mid-arm circumference ≥ 33 cm. Weight was recorded on a calibrated scale, to the nearest 100g, in duplicate, and standing height recorded as the vertical distance from the floor to the vertex of the head, with the subject barefoot. Body Mass Index was calculated as weight (kg)/ (height (m)).^{2} Anthropometrical measurements were taken in duplicate, at the first visit only, by two trained professional nurses. Weight was measured to the nearest 0.5 kg on a portable electronic scale and height was measured, barefoot, with head in the Frankfort plane.[35] Body Mass Index (BMI) calculated as weight (kg)/(height (m))^2. Waist circumference was measured at the level of the umbilicus. Whole body bio-electrical impedance was measured at 50 kHz using a standard tetrapolar bioimpedance monitor (Bodystat 2000,™ Douglas, British Isles), with the subject lying supine. Equations derived from a South African population, and validated by the Dunn Nutritional Centre, Cambridge, UK, were used to estimate % body fat (Anthony Bunn, personal communication).

Active renin was measured in EDTA plasma samples using a Nichols Institute Diagnostics two-site immunoradiometric assay. Plasma aldosterone was measured using a Diagnostic Products Corporation coated tube kit. Samples were frozen at -20°C and batch analyses of renin and aldosterone were performed.

Subjects were asked, in open-ended unprompted questions how they controlled their blood pressure and whether they thought that salt was good or bad for them. Verbatim responses were transcribed and summarized according to themes.

Statistical analyses

For each of the urinary and dietary variables, values are expressed as the mean of three separate measurements. Differences between ethnic groups for numerical variables were assessed using either One-Way ANOVA (parametric data) or the Kruskal-Wallis test (non-parametric data). For categorical data, ethnic differences were assessed using the X^2 test. For differences between hypertensives and normotensives, independent t-tests or Mann-Whitney tests were used. General linear modelling was performed to assess ethnic differences in Na excretion, while co-varying for BP and BMI. Spearman correlation coefficients were calculated to investigate univariate relationships between BP and urinary and dietary cations. Multiple regression modelling was used to investigate predictors of systolic and diastolic blood pressure, while controlling for age, sex, ethnicity and BMI. Multiple regression models were performed for each ethnic group, to assess the relationship between urinary Ca and urinary Na, controlling for systolic BP. All statistical analyses were considered significant at the $P<0.05$ level.

For the variables BP and urinary Na excretion, inter- (between-subject) and intra- (within-subject) individual variation is calculated as the coefficient of variation (CV = (SD/mean)) and expressed as a percentage, according to ethnic group and hypertensive status. Intra-individual CV is calculated as the variation within each subject between the three repeated measurements for each of the variables. Analyses on variability were conducted only in subjects for whom data from all three of the repeated measurements was available.

Results

Mean urinary excretion and reported dietary intake of electrolytes are shown, according to ethnic group, in Table 1. As expected from the study design, subjects in each of the

ethnic groups were comparable, in terms of blood pressure and proportion of men to women, however white subjects were significantly older than either black or mixed ancestry subjects. Black women had higher BMI values than white and mixed ancestry women and a greater percentage were obese (BMI ≥ 30). In men, the only inter-ethnic difference in anthropometrical status was a lower BMI and a lower proportion of subjects who were obese in white, compared to other, men.

Table 1
Anthropometric characteristics of subjects, and urinary excretion and dietary intake of cations, according to ethnic group: mean (SD)

	Black (N=110)	Mixed ancestry (N=112)	White (N =103)
Age*	39.7 (10.5)	39.4 (10.5)	46.9 (9.5) [b,c]
Sex (M/F)	54/56	51/61	54/49
Normotensive/Hypertensive subjects	51/59	48/64	46/57
BMI (kg/m^2)			
Men	28.0 (4.9)	29.8 (5.3)	27.6 (4.6) [c]
Women**	33.5 (7.4)	28.5 (6.1) [a]	30.5 (7.9) [b]
% Subjects obese (BMI ≥ 30)‡			
Men*	37.7	47.1	18.5
Women**	67.9	34.4	36.7
Waist circumference (cm)			
Men	92.6 (13.3)	97.4 (13.4)	96.0 (13.2)
Women	92.4 (20.0)	84.1 (16.7) [a]	92.8 (18.8) [c]
% Fat			
Men	24.3 (6.7)	24.0 (6.8)	22.8 (5.9)
Women	42.6 (8.6)	37.6 (7.6) [a]	40.0 (8.8)
Dietary energy intake (kJ/day)			
Men	8 098 (2908)	9 158 (3241)	9 031 (2567)
Women	6 654 (2154)	6 567 (1689)	6 850 (2609)
Sodium			
Dietary Na (mg/day)**	1 467 (891)	1 761 (885) [a]	1 922 (911) [b]
Dietary Na/4 200 kJ (mg/day)**	808 (347)	952 (287) [a]	1 019 (324) [b]
Urinary Na (mmol/day) *	135.3 (50.1)	147.5 (73.5)	164.8 (91.0) [b,c]
Potassium			
Dietary K (mg/day)**	2 058 (831)	2 092 (648)	2 513 (903) [b]
Urinary K (mmol/day)	55.6 (23.4)	54.3 (26.9)	61.9 (23.1)
	53.8 (29.3) †	47.2 (40.0)	59.9 (32.0) [b,c]
Na:K ratio			
Urinary Na:K ratio‡	2.66 (1.00)	3.14 (1.83) [a]	2.85 (1.43)
Magnesium			
Dietary Mg (mg/day)*	261 (110)	228 (105) [a]	285 (122) [c]
Urinary Mg (mmol/day)	21.8 (18.6)	23.2 (19.6)	22.9 (23.1)
Calcium			
Dietary Ca (mg/day)**	436 (332)	464 (215)	577 (274) [b,c]
Urinary Ca (mmol/day)**	1.47 (1.26)	2.38 (2.16) [a]	3.28 (5.0) [b,c]
PABA (%)[1]	74.8 (17.8)	76.2 (19.3)	80.3 (15.5) [b]

*P<0.05; ** P<0.001; ‡ P = 0.0536: ANOVA for differences between ethnic groups ([a] Black vs Mixed ancestry; [b] Black vs White; [c] Mixed ancestry vs White; Post-hoc analyses).
† Median (IQR); Kruskall-Wallis test performed.
[1] Mean PABA value calculated for the "complete" urine collections which were used in the final analyses only.
‡ X^2 test performed.

Urinary and dietary cations

Sodium

Mean reported dietary Na intake (ie. non-discretionary sources - sodium inherent in foods or added in processed foods)) was significantly lower in black subjects than either mixed ancestry or white subjects (Table 1), even accounting for differences in energy intake across groups. Normotensive white subjects had significantly higher urinary Na excretion and reported dietary Na intake than black normotensives, but in hypertensives, only dietary Na levels differed between the ethnic groups (Table 2). In both black and white subjects, reported Na intake was significantly ($P<0.05$) lower in hypertensives compared to normotensives (1 263 (972) vs 1 675 (805) mg, respectively, for black subjects and 1 729 (833) vs 2 048 (971) mg, respectively, for white subjects). However Na intake remained significantly higher in white than black subjects, regardless of blood pressure.

The ratio of urinary excretion to reported dietary intake of Na was 2.93 (4.80), and differed between ethnic groups (3.89 (7.44), 2.26 (1.39) and 2.55 (2.81) in black, mixed ancestry and white subjects, respectively; $P<0.05$ (ANOVA); $P= 0.051$ for black vs white (Bonferroni test)). The discrepancy between reported dietary Na intake and urinary excretion of Na (urinary Na taken as proxy for total Na intake) suggests that salt added to food at the table and in cooking (ie. discretionary) makes up 45.5 %, 32.8 % and 42.2 % of total sodium intake in black, mixed ancestry and white subjects, respectively. This equates to a daily added salt (NaCl) amount of 4.08 g, 4.15 g, and 4.76 g in black, mixed ancestry and white subjects, respectively.

Urinary Na excretion was significantly higher in white subjects, compared to the other two groups (Table 1). Mean urinary Na excretion equates to a daily salt (NaCl) intake of 7.8g, 8.5g and 9.5g in black, mixed ancestry and white subjects, respectively. Twenty-three percent of subjects had urinary Na concentrations < 100 mmol/day[36] and this proportion did not differ between ethnic groups (Table 3). Urinary Na was associated with BMI in men and women (Spearman $r = 0.196$ and $r = 0.176$; $P<0.05$, respectively). General linear modelling found that ethnicity (ie. black versus white; $P = 0.005$) was associated with lower urinary Na excretion, independently of blood pressure, and co-varying for BMI (whole model adjusted $r^2 = 0.0545$; $P = 0.0014$). No differences were found between mixed ancestry subjects and the other two ethnic groups.

Table 2
Urinary excretion and dietary intake of electrolytes, according to ethnic and hypertensive status: mean (SD)

	Black		Mixed ancestry		White	
	Hypertensives	Normotensives	Hypertensives	Normotensives	Hypertensives	Normotensives
N	51	59	48	64	46	57
Age	47.1 (8.2)	33.3 (7.7)	46.6 (7.0)	34.0 (9.5)	50.7 (5.9) [b,c]	43.9 (10.8) [b,c]
Sex (Men/Women)	27/24	27/32	23/25	28/36	24/22	30/27
Systolic BP (mm Hg)*	135 (19)	114.1 (11.9)	142 (21) [a]	117.4 (12.0)	137 (14)	117.6 (12.5)
Diastolic BP (mm Hg)	84.6 (10.0)	73.4 (7.1)	89.0 (10.7)	75.6 (7.6)	86.2 (8.8)	75.4 (7.7)
Mean Arterial Pressure (mmHg)	102 (12)	86.5 (8.1)	107 (13)	89.9 (9.0)	103 (10)	88.9 (91.7)
Sodium						
Dietary Na intake (mg/day)*	1 263 (972)	1 675 (805)	1 760 (1 082) [a]	1 778 (723)	1 729 (833) [b]	2 048 (971) [b]
Urinary Na (mmol/day)	140 (44)	131.0 (56.0)	162 (78)	135.1 (67.6)	163 (114)	166.1 (63.1) [b,c]
	134 (42)†	128 (65)†	156 (83)	127 (80)	126 (84)	154 (85) [b,c]
Potassium						
Dietary K intake (mg/day)*	1 849 (621)	2 269 (982)	2 152 (691) [a]	1 981 (533) ‡	2 370 (825) [b]	2 601 (943) [c]
Urinary K (mmol/day)	60.1 (25.2)	51.2 (20.9)	62.2 (27.9)	47.6 (24.0)	64.8 (23.2)	59.0 (22.1) [c]
	55.3 (34.9)†	46.8 (31.4)†	69.9 (41.8)	42.8 (26.2)	64.5 (32.3)	56.0 (33.6) [c]
Na:K ratio						
Dietary Na:K ratio	0.66 (0.42)	0.78 (0.32)	0.82 (0.37)	0.89 (0.35)	0.75 (0.31)	0.84 (0.40)
Urinary Na:K ratio	2.57 (0.93)	2.74 (1.08)	3.04 (1.54)	3.23 (2.07)	2.62 (1.35)	3.07 (1.48)
	2.37 (0.97)†		2.74 (2.02)		2.62 (1.39)	
Calcium						
Dietary Ca intake (mg/day)**	331 (183)	534 (413)	460 (210) [a]	471 (225)	477 (202) [b]	650 (285) [c]
Urinary Ca (mmol/day)**	1.45 (1.40)	1.49 (1.12)	2.60 (2.65) [a]	2.19 (1.61)	2.64 (1.36) [b]	3.89 (6.78) [b,c]
	1.06 (1.34)†	1.27 (1.55)†	1.73 (2.47) [a]	1.73 (1.92) [a]	2.45 (1.63) [b]	2.67 (2.32) [b,c]
Magnesium						
Dietary Mg intake (mg/day)	263 (89)	263 (131)	253 (134)	210 (72) [a]	253 (103)	314 (135) [b,c]
Urinary Mg (mmol/day)	18.9 (17.2)	24.7 (19.6)	18.8 (17.2)	27.0 (20.9)	24.2 (21.5)	21.7 (24.7)

*P<0.05; ** P<0.001; ***P<0.0001: ANOVA test for differences between ethnic groups.
Post-hoc analyses: [a] Black vs Mixed ancestry; [b] Black vs White; [c] Mixed ancestry vs White; ‡ P = 0.055 for Black vs Mixed ancestry.
† Median (IQR); Mann-Whitney test for differences between ethnic groups: [a] Black vs Mixed ancestry; [b] Black vs White.

Table 3
Proportion of sample with urinary Na and K concentrations (mean of three 24hr collections) which meet the JNC[34,35] dietary guidelines, according to ethnic group: % (n)

	Black (N=101)	Mixed ancestry (N=97)	White (N =87)	Total (N =285)
Urinary Na < 100 mmol/day				
Men	22.9 % (11)	23.3 % (10)	15.6 % (7)	20.6 % (28)
Women	20.8 % (11)	33.3 % (18)	21.4 % (9)	25.5 % (38)
Total	21.8 % (22)	28.9 % (28)	18.4 % (16)	23.2 % (66)
Urinary K ≥ 90 mmol/day				
Men	10.4 % (5)	14.0 % (6)	11.1 % (5)	11.8 % (16)
Women	5.7 % (3)	5.6 % (3)	11.9 % (5)	7.4 % (11)
Total	7.9 % (8)	9.3 % (9)	11.5 % (10)	9.5 % (27)

No significant differences between ethnic groups for any of the categories.

Significantly more black and mixed ancestry subjects reported adding salt to food in cooking than their white counterparts (98.2 %, 93.8 % and 83.4 %, respectively; P<0.05), more of the black subjects (50.4 %) than either the mixed ancestry (25.0 %) or white (31.0 %) subjects reported that they added salt to their food before tasting it (P<0.005), and more black subjects (20.2 %) than either mixed ancestry (6.3 %) or white (11.7 %) subjects reported that they liked their food to taste "very salty" (P<0.05). Within each ethnic group, neither urinary Na excretion nor reported dietary Na intake differed across categories of responses to the questions on salt taste preferences and the practice of adding salt to food before tasting it (data not shown).

Potassium

Reported dietary K intake was significantly lower in black compared to white subjects (P<0.001) (Table 1), but aggregated according to hypertensive status, this ethnic difference was only evident for hypertensive subjects (Table 2). Median urinary K was significantly lower in black and mixed ancestry groups, compared to white subjects (Table 1). Few subjects (9.5 %) had urinary K values which met or exceeded the reference optimal dietary intake value of 90 mmol/day[37] (Table 3). According to hypertensive status, no ethnic differences were found for urinary potassium, except for mixed ancestry normotensives having a lower excretion than white normotensives (Table 2). In black subjects only, reported K intake was lower in hypertensives compared to normotensive subjects (P<0.05).

Na:K ratio

Urinary Na:K ratio was significantly higher in the mixed ancestry group compared to the black subjects (Table 1). Urinary Na:K ratio did not differ between ethnic groups, in either

normotensive or hypertensive subjects (Table 2). Comparisons are made in Table 4 between our data and findings from a 1982 study of black and South Africans in Johnannesburg, for urinary Na, K and Na:K ratio.[27,38]

Table 4
Urinary Na and K in normotensive black and white South Africans, and in hypertensive black South Africans: comparison with other studies: mean (SD)

	Urinary Na (mmol/24h)	Urinary K (mmol/24h)	Na:K ratio	Equivalent salt intake (g NaCl/day)
Normotensives				
Cape Town, 2002				
Present study				
Blacks (N = 51)	131.0 (56.0)*	51.2 (20.9)	2.7 (1.1)	7.5
Whites (N = 45)	166.1 (63.1)	59.0 (22.1)	3.1 (1.5)	9.5
Johannesburg, 1982				
Barlow *et al.*[27]				
Blacks (N = 71)	126.8 (55)	30.9 (13)*	4.3 (1.0)*	7.5
Whites (N = 34)	166.8 (63)	59.3 (19)	2.9 (0.8)	9.8
Hypertensives - black subjects only				
Cape Town, 2002				
Present study (N = 49)	140 (44)	60 (25)	2.6 (0.9)	8.0
Johannesburg, 1982	112 (42)	35 (16)	3.2	6.6
Cohen *et al.*[38] (N = 33)				

* P<0.05. Independent t-test for differences between black and white subjects in the same study.

Calcium

Mean dietary calcium was higher in white subjects compared to either the black or mixed ancestry groups, however in all groups calcium intake was low (about half of Dietary Reference Intake (DRI)[39] of 1000 mg/day) (Table 1). Inadequate (< 67 % DRI) calcium intakes were evident in 84.4 %, 88.4 % and 78.6 % of black, mixed ancestry and white subjects, respectively. Calcium intake was significantly (P<0.005) lower in hypertensive than normotensive subjects, for both black and white ethnic groups, and more hypertensive compared to normotensives had inadequate dietary intakes of calcium (92 % vs 78 %; P<0.05) (Figure 1).

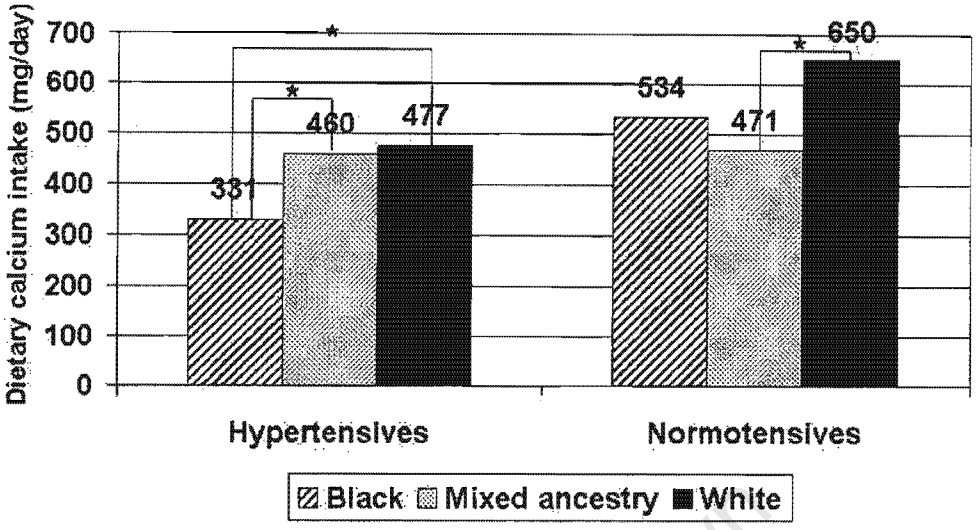

Figure 1
Mean daily dietary calcium intake, according to ethnic and hypertensive status (*P<0.05 for differences between ethnic groups)

Urinary Ca excretion was significantly higher in white, compared to either black or mixed ancestry, subjects (Table 1). In normotensives, urinary calcium concentrations differed between all three ethnic groups, with black subjects having the lowest values of all groups (median = 1.06 (IQR = 1.34) mmol/day), approximately less than half that of white subjects (median = 2.45 (1.63) mmol/day) (Table 2). Similarly, inter-ethnic differences were found for urinary calcium in hypertensive subjects (Table 2).

A positive association was found between reported dietary protein intake and urinary calcium for white subjects only ($r = 0.266$ ($P<0.01$)), who had a higher protein intake than black subjects (70.8 (25.4) g/d) and 62.2 (28.5) g/d, respectively ($P<0.05$)). In black and mixed ancestry subjects, urinary Ca was significantly and positively associated with urinary Na ($r = 0.422$ ($P<0.0001$) and $r = 0.632$ ($P<0.0001$), respectively), however no association was found for white subjects. Regression modeling was conducted by ethnic and sex group, since these were confounding variables in the urinary Ca-Na relationship. In models which included systolic BP and dietary Na intake, urinary Na was significantly and independently associated with urinary Ca in black and mixed ancestry men and women, and in white men only (Table 5). A 100 mmol increase in 24-h urinary Na predicted an increase in average daily urinary Ca of between 1.03 mmol (black women)

and 1.76 mmol (mixed ancestry men). No association was found between urinary and dietary Ca intake in any ethnic group.

Table 5
Relationship of daily urinary Ca excretion (mmol/24-h) with urinary Na, controlling for systolic BP in each ethnic group

	Men			Women		
	ß	SE (ß)	P-value	ß	SE (ß)	P-valu
Blacks						
Adjusted r^2 of model (P-value)	0.1799 (0.0048)			0.1226 (0.0142)		
Intercept	-1.4125	1.496	0.3501	0.9626	1.1521	0.407
24-h Urinary Na	0.0108	0.0032	0.0014	0.0103	0.0037	0.007
Systolic BP	0.0110	0.0103	0.2917	-0.0081	0.0086	0.348
Mixed ancestry						
Adjusted r^2 of model (P-value)	0.3215 (0.0002)			0.3673 (<0.0001)		
Intercept	0.4282	3.2524	0.8959	-0.8105	0.9970	0.420
24-h Urinary Na	0.0176	0.0039	<0.0001	0.0169	0.0030	<0.00(
Systolic BP	-0.0030	0.0256	0.9061	0.0034	0.0073	0.642
Whites						
Adjusted r^2 of model (P-value)	0.1844 (0.0052)			-0.0283 (0.6415)		
Intercept	-2.3492	2.0663	0.2620	1.9180	1.2555	0.134
24-h Urinary Na	0.0116	0.0036	0.0023	-0.0011	0.0096	0.482
Systolic BP	0.0276	0.0141	0.0571	0.0053	0.0096	0.587

[a] Multiple regression models, performed by ethnic and sex group.
Abbreviations: BP= blood pressure; ß = beta; SE = standard error.

Magnesium

Reported dietary magnesium intake did not differ between black and white subjects, but was significantly lower in mixed ancestry subjects (Table 1). More of the mixed ancestry, compared to black or white subjects, had Mg intakes below 67 % DRI[38] (67.9 %, 51.8 %, and 43.7 %, respectively; $P<0.05$). In normotensives, mixed ancestry and black subjects had significantly lower reported dietary Mg intakes than white counterparts, but this difference was not evident in hypertensive subjects (Table 2). Urinary Mg excretion did not differ across ethnic groups (Table 1), nor across ethnic groups aggregated according to hypertensive or normotensive status (Table 2).

Treatment status effect on urinary electrolytes

Both treated and untreated hypertensives were included in this study. Ethnic differences in urinary excretion and dietary intake of cations were investigated in hypertensives, according to whether or not they were taking medication that may affect electrolyte excretion (i.e. diuretics and ACE inhibitors). Thirty percent (n = 44) of hypertensives were being treated with these classes of drugs. No differences in either urinary or dietary

electrolytes, were found between treated and untreated hypertensives for both black and mixed ancestry hypertensives, with the exception that in mixed ancestry hypertensives, urinary calcium was significantly lower in treated individuals (Table 6). In the white group, treated subjects had significantly higher values than untreated hypertensives for urinary Na (258 (180) vs. 130 (51) mmol/day) and K (83.8 (22.8) vs. 58.3 (21.0) mmol/day). In both black and white treated hypertensives, plasma renin concentrations were higher in treated versus untreated subjects, while the aldosterone:renin ratio was significantly lower.

Table 6
Urinary cations and aldosterone and renin concentrations of hypertensives, according to ethnic and treatment status (mean (SD))

	Black		Mixed ancestry		White	
	Treated[1]	**Not treated**	**Treated[1]**	**Not treated**	**Treated[1]**	**Not treated**
N	16	35	17	31	11	35
Systolic BP (mm Hg)	140 (19)	133 (19)	148 (23)	139 (20)	137 (15)	139 (16)
Diastolic BP (mm Hg)	85.6 (9.2)	84.3 (10.7)	92.1 (13.4)	87.4 (9.0)	84.0 (7.9)	86.7 (9.5)
Urinary Na (mmol/day)	137 (56)	132 (50)	152 (66)	151 (96)	258 (180)[b,c]	130 (51)*
Urinary K (mmol/day)	53.8 (25.7)	61.2 (26.6)	64.9 (28.5)	54.7 (32.2)	83.8 (22.8)[b]	58.3 (21.0)*
Na/K ratio	2.72 (0.80)	2.45 (0.93)	2.68 (1.33)	3.27 (1.34)	3.05 (2.04)	2.42 (0.93)[c]
Urinary Ca (mmol/day)	1.46 (2.0)	1.40 (1.07)	2.35 (2.01)	2.75 (3.01)[a]	2.58 (1.67)	2.68 (1.21)[b,c]
Renin (uU/ml)	89.4 (122.7)	24.8 (37.0)†	25.6 (27.9)	43.1 (62.4)	126.4 (140.0)[c]	42.4 (59.7)*
Aldosterone (pmol/ml)	258 (316)	231 (155)	278 (224)	253 (172)	272 (157)	278 (232)
Aldosterone:renin ratio	14.6 (26.7)	19.0 (19.7)*	16.6 (11.6)	14.8 (14.3)	7.8 (10.7)	15.9 (15.9)‡

[1]Treated = prescription of diuretics and/or ACE inhibitors.
* P<0.05; † P = 0.086; ‡ P = 0.0685; Difference between treated and not treated for that ethnic group (Independent t-test). For variables not normally distributed (renin, aldo, urinary Ca in blacks; urinary Na and renin in mixed ancestry; and urinary Na and renin in whites), the Mann Whitney test assessed differences between treatment groups.
[a] Black vs Mixed ancestry; [b] Black vs White; [c] Mixed ancestry vs White; Post-hoc analyses (Bonferroni test) for differences between ethnic groups for that treatment group (P<0.05).

In untreated hypertensives (n=101), ethnic differences (P<0.05) were found for the following urinary variables: K higher in black compared to white subjects; Na:K ratio higher in mixed ancestry than in white subjects; Ca higher in mixed ancestry and white subjects than in black subjects.

Predictors of blood pressure

Neither urinary nor dietary Na, K, or Mg were associated with either systolic or diastolic blood pressure. Results did not change when white treated hypertensives were excluded from the analyses. In the normotensive group only, urinary calcium was positively (P<0.05) associated with both systolic (Spearman r = 0.214) and diastolic (r = 0.185) blood pressure.

In white subjects, reported dietary calcium intake was negatively associated with both systolic ($r = -0.224$ ($P<0.05$)) and diastolic blood pressure ($r = -0.254$ ($P<0.01$)) (Figure 2). A similar inverse trend was seen for the other ethnic groups, but this did not reach significance. In the total group combined, reported dietary calcium intake was inversely associated with both systolic and diastolic BP ($r = -0.127$ for each; $P<0.05$).

Controlling for age, sex and ethnic group in regression modes, neither Na, K, Ca or Mg excretion predicted blood pressure in either normotensive or hypertensive subjects. Controlling for age, ethnic group, sex, and BMI, it was found that renin was inversely associated with systolic and diastolic BP:

Systolic BP = 98.648 - 0.614 (ethnic group) - 9.794 (sex) +0.534 (BMI) -0.032 (renin) + 0.711 (age). Adjusted $r^2 = 0.286$; model is significant at $P<0.0001$.

Diastolic BP = 63.838 + 0.392 (ethnic group) - 5.376 (sex) +0.413 (BMI) -0.022 (renin) + 0.305 (age). Adjusted $r^2 = 0.257$; model is significant at $P<0.0001$.

In both models, sex (1 = men; 2 = women), BMI, age and renin are significant ($P<0.01$), while ethnicity (1 = black, 2 = mixed ancestry; 3 = white) was not.

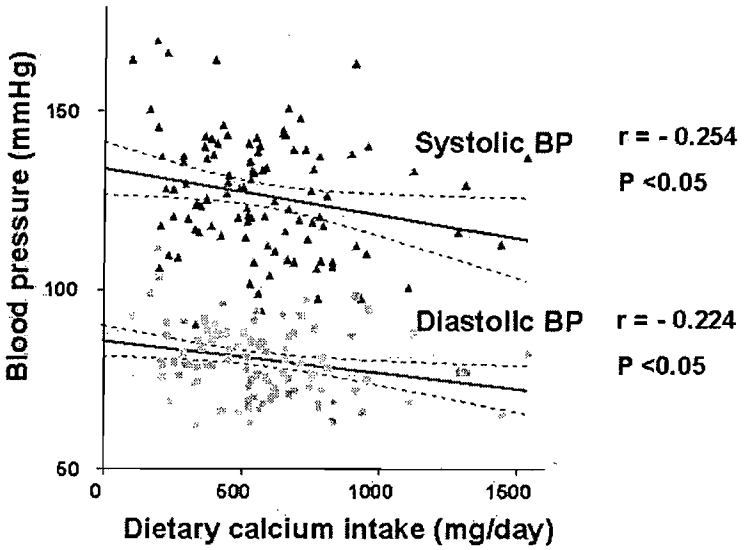

Figure 2
Association between blood pressure and mean daily reported calcium intake in white South Africans (purple triangles denote systolic BP; yellow circles denote diastolic BP).

Inter- and intra-individual variability in blood pressure and urinary Na excretion

For the total group, inter-individual CV for systolic and diastolic BP was 15.95 % and 13.17 %, respectively. Inter-individual CV was similar across ethnic groups for mean systolic blood pressure (13.18 % to 16.07 %) and diastolic blood pressure (12.14 % to 13.86 %) (Table 7). Intra-individual CV for BP for the three repeated days of measurements was 5.15 % and 5.27 % for systolic and diastolic BP, respectively (total group) and was similar between ethnic groups, according to hypertensive status. Systolic BP intra-individual CV was higher in hypertensive compared to normotensive subjects (6.08 % and 4.64 %, respectively; P<0.0001). Similarly, intra-individual variation in diastolic BP was higher in hypertensive than normotensive subjects (was 6.03 % and 4.64 %, respectively; P<0.0001).

Inter-individual CV in mean 3-day urinary Na excretion for the total group (N = 143) was 41.5 %. Inter-individual CV, by ethnic group, was 31.5 %, 29.8 % and 52.4 % for black (n = 50), mixed ancestry (n = 45) and white (n =48) subjects, respectively. In the total group, intra-individual variation in Na excretion over the three separate 24-hour collection periods was 33.7 % (95 % CI = 30.6 - 36.8 %). Within-subject variation in urinary Na was similar between ethnic groups (31.7 %, 34.0 % and 35.6 % in black (n = 50), mixed ancestry (n = 45) and white (n = 48) subjects, respectively. Normotensive subjects (n = 74) tended to have a higher intra-individual CV than hypertensives (n = 69) (36.9 % and

30.3 % respectively, P= 0.073). The ratio of intra- to inter-individual variation was 1.01, 1,14 and 0.68 for black, mixed ancestry and white subjects, respectively.

Table 7
Inter- and intra-individual variation in blood pressure for three repeated measurements, according to ethnic group and hypertensive status: mean (SD) coefficient of variation (CV)[a]

	Black N = 108	Mixed ancestry N = 112	White N = 102	Total N = 322
Systolic BP (mm Hg)*	123 (22)	128 (21)	127 (17)	126 (19)
Diastolic BP (mm Hg)	78 (13)	81 (11)	80 (10)	80 (10)
INTER-INDIVIDUAL CV (%)				
Hypertensives	**N = 51**	**N = 48**	**N = 46**	**N = 145**
CV Systolic BP	13.73	14.86	10.51	13.31
CV Diastolic BP	11.84	12.00	10.17	11.52
Normotensives	**N = 57**	**N = 64**	**N = 56**	**N = 177**
CV Systolic BP	10.43	10.48	10.43	10.51
CV Diastolic BP	9.96	10.30	10.05	10.17
Total				
CV Systolic BP	15.21	16.07	13.18	14.95
CV Diastolic BP	13.22	13.86	12.14	13.17
INTRA-INDIVIDUAL CV (%)				
Hypertensives	**N = 51**	**N = 48**	**N = 46**	**N = 145**
CV Systolic BP	5.99	6.27*	5.98*	6.08**
CV Diastolic BP	6.32 *	5.87*†	5.87*	6.03**
Normotensives	**N = 57**	**N = 64**	**N = 56**	**N = 177**
CV Systolic BP	4.68	4.09	4.45	4.40
CV Diastolic BP	4.95	4.60	4.38	4.64
Total				
CV Systolic BP	5.30	5.02	5.14	5.15
CV Diastolic BP	5.60	5.14	5.05	5.26

[a] CV = (SD/mean) * 100
* P<0.05; Mann-Whitney test for differences between hypertensive and normotensive subjects, for that ethnic group.
** P<0.0001; Mann-Whitney test for differences between hypertensive and normotensive subjects, for combined ethnic groups.

Renin and aldosterone concentrations

Renin and/or aldosterone data was missing for 19 subjects (6 black; 7 mixed ancestry and 6 white subjects). Fifty-one subjects (17.5 %) had low renin (≤ 10uU/ml) concentrations. In each of the ethnic groups, significantly more of the hypertensive subjects had low renin status, compared to normotensives (29.5 % vs % 8.4 %, respectively; P<0.05). There was a trend for more of the black, compared to mixed

ancestry or white, hypertensives to have a low renin status (40.4 %, 22.0 % and 24.4 %, respectively), however this difference did not reach significance (P = 0.114). Three subjects (2 hypertensive black; 1 normotensive mixed ancestry) had suspected primary aldosteronism (i.e. aldosterone > 500 pmol/ml and aldosterone:renin ratio >=70).[40] Forty-five subjects (15.5 %) had low aldosterone concentrations (<111 pmol/ml), while four subjects (1.4 %) had aldosterone levels > 860 pmol/ml, and the remainder (83.2 %) had levels in the normal range of 111 – 860 pmol/l. Eleven subjects (3.8 %) had both low renin (≤ 10 uU/ml) and low aldosterone (<111 pmol/l) concentrations, of whom 8 subjects were black (8 % of total black sample), and the remaining 3 subjects were white (3.3 % of white sample) (Table 8).

Table 8
Active renin and aldosterone concentrations in normotensive and hypertensive subjects in Cape Town: n (% total sample)

Renin concentrations	Aldosterone			Total
	Low (< 111 pmol/ml)	Normal range (111 – 860pmol/ml)	High (>860 pmol/ml)	
Low (≤ 10 uU/ml)	11 (3.8 %)	40 (13.8 %)	0	51 (31.3 %)
> 10 uU/ml	34 (11.7 %)	202 (69.4 %)	4 (1.4 %)	240 (82.4 %)
Total	45 (15.5 %)	242 (83.2%)	4 (1.4 %)	291

Six of the eight black subjects with low renin/low aldosterone levels were hypertensive (13.3 % of all hypertensive blacks), while all 3 of the white subjects were hypertensive (7.5 % of total white hypertensive group). Using the less stringent criteria for low aldosterone levels described by Rayner and colleagues[41] (i.e. < 225 pmol/L), 26.7 % of black hypertensive subjects in our study had low renin and low aldosterone levels, compared to 10.9 % of black normotensive subjects. In the hypertensives, significantly more black than either white (7.5 %) or mixed ancestry subjects (7.5 %) had low renin/low aldosterone concentrations in this range (P<0.05).

A significantly lower plasma renin concentration and a higher aldosterone:renin ratio was found in hypertensives compared to normotensives in the black and mixed ancestry (P<0.005) groups, but not in white subjects (Table 9). The only ethnic group difference was a lower plasma aldosterone concentration in black normotensives, compared to either their mixed ancestry or white normotensive counterparts (Mann-Whitney test; P<0.005). In the hypertensive group, 29 % of black, compared to 20 % of mixed ancestry and 17.5 % of white subjects had low renin hypertension (low renin and normal aldosterone concentrations).

Table 9
Active renin and aldosterone in normotensive and hypertensive subjects from three ethnic groups in Cape Town

	Active renin (uU/ml)	Aldosterone (pmol/L)	Renin:aldo Ratio	Mean Arterial Pressure (MAP) (mm Hg)
Normotensives				
Black (N = 56)	68.7. (63.5)	213.0 (258)	7.2 (10.9)	85.4 ± 13.9
Median (IQR)	39 (103) ††	165 (102) [a, b]	3.9 (7.2) ††	
Mixed ancestry (N = 62)	84.5 (70.1)	264 (181)	8.8 (11.1)	89.6 ± 8.9
Median (IQR)	41.5 (121) ††	213 (152)	4.2 (10.5) ††	
White (n = 52)	89.6 (90.6)	256 (145)	8.9 (11.0)	89.5 ± 8.6
Median (IQR)	36.5 (145)	219 (166)	4.0 (9.4)	
Total (N = 170)	80.4 (78.1)	245 (201)	8.3 (11.0)	88.2 ± 10.8
Median (IQR)	38 (120)†	193 (158)	4.0 (8.4)†	
Hypertensives				
Black (N = 48)	48.8 (83.7)	254 (220)	18.3 (22.1)	101.4 ± 12.5
Median (IQR)	18 (39)	225 (180)	8.4 (15.5)	
Mixed ancestry (N = 43)	44.1 (55.5)	304 (178)	15.7 (13.3)	106.7 ± 13.3
Median (IQR)	21 (48)	236 (239)	11.4 (16.9)	
White (N = 45)	71.6 (94.8)	285 (223)	14.3 (15.1)	103.3 ± 9.9
Median (IQR)	25.5 (82.8)	251 (195)	10.6 (17.7)	
Total (N = 136)	54.6 (80.2)	280 (208)	16.2 (17.4)	103.8 ± 12.1
Median (IQR)	21 (52)	232 (196)	10.3 (17.6)	

† P<0.0001; Mann-Whitney test for differences between the total hypertensive and normotensive groups (all ethnic groups combined in each group).
†† P<0.005; Mann-Whitney test for differences between the hypertensive and normotensive groups, according to that ethnic group.
[a] P<0.005; Mann Whitney test for differences between black and mixed ancestry normotensives.
[b] P<0.005; Mann Whitney test for differences between black and white normotensives.

Use of health facilities for hypertension care

More of the black hypertensives (66.7 %), compared to either their mixed ancestry (27.7 %) or white (34.1 %) counterparts attended a public sector clinic for hypertension care, while fewer black hypertensives (25 %) than the other two ethnic groups (56.8 %) attended a private doctor. The remaining subjects either attended a clinic in the workplace, or were not currently receiving treatment.

Self-reported strategies to control blood pressure in hypertensives

Regarding dietary factors which were identified by hypertensives as being important in blood pressure control, "avoidance of salt and salty foods" was the most common response (20.6 %), followed by "avoidance of fatty foods" (16.5 %), and consumption of "a balanced diet" (6.2 %) (open-ended, unprompted responses). Fewer black hypertensives (9.8 %) than mixed ancestry (22.9 %) or white (26.1 %) hypertensives reported that they avoided salt/salty foods (P<0.05). Few subjects (4.1 %) reported an increased intake of vegetables, and none mentioned fruit, as being a method of blood pressure control. In a separate, open-ended, unprompted question asking whether

subjects thought that salt was good or bad for them, it was evident that there was a fairly low awareness regarding the salt-BP association. Of the 195 subjects (60.2 %) who reported that salt was bad for them, 46.7 % (n = 91) identified that salt raised blood pressure. Other responses were water retention (10.3 %, n = 20), salt is unhealthy if eaten in excess (14.9 %, n = 29), causes heart disease (8.2 %, n = 16), causes dehydration (5.1 %, n = 10), spoils taste of food (5.1 %, n = 10), and medical advice (5.1 %, n = 10). More of the white subjects (72.8 %), compared to either mixed ancestry (58.9 %) or black (48.2 %) subjects reported that salt was bad for them. However, responses linking salt to raised blood pressure were similar across ethnic groups (22.7 %, 29.5 % and 31.1 % of the black, mixed ancestry and white samples, respectively (28 % of total sample)).

Discussion

Similar to findings of studies conducted in black and white normotensives in Johannesburg in the early 1980's[38,27] (see Table 4), ethnic differences exist in urinary sodium excretion, with black subjects having significantly lower values than their white counterparts. In our study, black and mixed ancestry subjects have excretion values indicative of a daily salt intake of 7.8 g and 8.5 g, respectively, compared to 9.5 g in the white sub-sample. Urinary sodium does not appear to have changed over time in either black or white groups, compared to the earlier Johannesburg study.[27] The salt intake of the white subjects is similar to that reported in the adult American[42] and British population.[43]

The finding that ethnic differences in urinary Na concentrations were evident only in normotensive subjects suggests that in the hypertensive subjects, there may have been confounding between ethnic groups, with regard to access to dietary advice previously given by a health professional to reduce salt intake after diagnosis of the condition. White hypertensives may have had greater access to nutrition education since they were more likely than black hypertensives to receive medical care at private, rather than public, health facilities. In this regard, a quarter of white and mixed ancestry hypertensives, compared to only a tenth of black hypertensives reported the avoidance of salt and salty foods in their diet, but despite this, these groups still had a significantly higher reported Na intake than their black counterparts with hypertension. Only in the white sub-sample was urinary Na excretion significantly lower in hypertensive (those not being treated with agents that affect urinary electrolyte excretion) compared to normotensive subjects, which indicates that the lower reported dietary Na intake does

reflect a change in their dietary patterns. Our data suggests that under-reporting of food sources of Na (excluding salt added to foods at the table and in cooking) may have occurred in both black and white hypertensives, but that actual dietary changes in terms of sodium reduction were evident only in the white group.

The contribution of discretionary salt to total Na intake (33 - 46 %) in the South African population appears to be much higher than that reported in affluent western countries, in which 75 – 85 % of salt is estimated to come from processed foods.[44,45,46] This finding suggests a need to educate South Africans to add less salt to foods and in cooking. It is noteworthy that in all three ethnic groups, there was a low awareness of salt as being pathogenic in the development of hypertension (28 % of total sample reported (unprompted) that high blood pressure was related to an excessive salt intake). The use of dietary data to estimate actual sodium intake across populations is limited by the inability to accurately quantify discretionary (added) salt usage. Our data suggests that, as a rule of thumb, reported dietary sodium intake data should be doubled to account for salt added to food at the table and in cooking, at least in black South Africans living in urban areas. However, caution needs to be exercised as under-reporting of non-discretionary sources of Na would falsely elevate our estimation of added salt in the diet. Other studies have demonstrated that hypertensive patients tend to underestimate their sodium intake by between 22 %[47] and 50 %[48] using 24-hour dietary recall methods. A Japanese study which used a self-administered diet history questionnaire, rather than repeated 24-hour dietary recalls, reported ratios of urinary excretion to dietary intake of Na close to one (0.84 to 0.97).[49] In the present study, the higher ratios (2.26 to 3.89) suggest a greater degree of under-reporting of habitual dietary Na intake.

Within subject variation in urinary Na excretion is evident, even in individuals provided with a constant dietary Na intake. A Dutch study in which subjects were provided with a constant Na intake for 60 days while 24-h urine samples were collected daily found a relatively high intra-individual coefficient of variability (CV) in Na excretion of 16 %.[50] The level of CV did not improve when the excretion was expressed on the basis of urinary creatinine. The ratio of intra- to inter-individual variation in urinary Na provides some indication of the number of 24-h urine specimens required to obtain accurate assessments of habitual Na excretion. The smaller the ratio, the lower the number of 24-h urine samples required.[51] The ratio of 1.01 and 1.14 for black and mixed ancestry subjects is considerably lower than that reported for Chinese (1.48)[50] populations, and similar to that reported for a sample of Italian men (1.12).[52] The Chinese and Italian studies calculated that between four and five 24-h urine collections were necessary to

reduce to 10 - 15 % the diminution of the correlation coefficient between urinary Na and another related physiological variable.[50,51] In our study, an even lower ratio of intra- to inter-individual ratio was found for white subjects (0.68), which suggests that in all ethnic groups, three repeated 24-hour urine collections is sufficient to appropriately characterize dietary Na intake.

Urinary potassium excretion in black subjects in the present study has almost doubled since those reported in 1982[38] and, surprisingly, we did not find a difference in urinary excretion of potassium between black and white subjects (either normotensive or hypertensive) in the present study. As a result of this increase in potassium excretion, the previously reported higher urinary sodium/potassium ratio in black, compared to white South Africans,[27] no longer exists.

Optimal reference values for urinary excretion of Na and K are not available and since more than 98 % and 85 % of the daily ingested amount, respectively, is excreted in the urine,[53] our results are compared with optimal recommended dietary intakes. In all ethnic groups, urinary sodium excretion exceeded the recommended maximum dietary intake value of 100 mmol per day (2.4 g sodium or 6g sodium chloride)[36] and less than a quarter of subjects had urinary sodium values below this level. In addition, in all ethnic groups, potassium excretion values fell far below the recommended dietary intake of 90 mmol per day[37] and less than ten percent of subjects met this recommendation. The most recent dietary guidelines of the World Health Organization recommend even more stringent restrictions of sodium for the prevention of cardiovascular disease (coronary heart disease, hypertension, and stroke), namely 70 mmol per day (4 g sodium chloride). These guidelines suggest a potassium intake which will keep the sodium:potassium ratio close to one, i.e. 70 – 80 mmol/day, if sodium recommendations are followed.[54] It has been demonstrated in hypertensive black South African women (with a baseline K excretion similar to that reported in black subjects in the present study) that supplementation with 65 mmol/day of potassium chloride reduces blood pressure by an average of 7/3 mmHg.[55]

The use of anti-hypertensive medication which affects electrolyte excretion (e.g. diuretics and ACE inhibitors) is a potential effect modifier. No differences in 24-hour urinary electrolyte excretion were found in either black or mixed ancestry hypertensives when analyses were performed according to whether or not they were prescribed these types of drugs. However, in white subjects, both urinary sodium and potassium values were higher in those prescribed such medication. This suggests that white hypertensives may

have either been more compliant with taking prescribed medications or that the prescribing pattern of these medications (i.e. dosage and frequency) differed in this group. However, analyses of hypertensive subjects who were not being treated with such medications still demonstrated no difference in Na excretion between black and white subjects, suggesting a lack of an ethnic-related difference in dietary intake patterns in hypertensives.

Intracellular calcium is an important determinant of arteriolar tone and it has been suggested that low dietary calcium intakes may predispose susceptible individuals to the pressor effect of sodium. The findings of the DASH study convincingly showed an additional benefit on blood pressure in subjects consuming a diet rich in fruit, vegetables and low fat dairy products compared to those on the high fruit and vegetables diet alone.[56] In the present study, we found that, of all the urinary and dietary cations investigated, the strongest (inverse) association with blood pressure was found for dietary calcium intake, and, in each ethnic group, hypertensives had a significantly lower reported intake of calcium than normotensives. Low reported calcium intakes in black subjects in the present study are consistent with data from dietary surveys conducted in black South Africans since 1975.[57]

Meta-analyses of randomized controlled trials in which the relationship between calcium supplementation and BP have been investigated have demonstrated only small reductions in systolic BP (-0.53 to -1.68 mm Hg) and diastolic BP (zero change to -0.84 mm Hg).[58,59] To date, no interventions of simultaneous dietary manipulation of calcium and potassium have been undertaken in South Africans. Extrapolations from interventions conducted in populations with vastly different eating habits may not be appropriate.

It has previously been reported that a higher urinary calcium excretion (and consequent secondary increase in parathyroid activity) is evident in essential hypertension.[60] However, in our study, no difference in urinary Ca was found between hypertensive and normotensive subjects in any of the three ethnic groups. The underlying mechanism of hypertension was not investigated in our hypertensive subjects, and the lack of data on parathyroid hormone levels further limits interpretation in this regard. In both hypertensive and normotensive groups, black subjects had the lowest urinary calcium, followed by mixed ancestry and then white subjects. Urinary calcium excretion is variable and is not a marker of habitual calcium intake within an individual.[61] In our study, 24-h urinary Na was associated with urinary Ca excretion in black and mixed ancestry South

Africans, and in white men, but not white women. The urinary Na-Ca relationship has previously been studied extensively[62] and exists in different ethnic groups.[63] Blackwood and colleagues (2001)[62] demonstrated lower urinary Ca excretion in black, compared to South Asian or white, hypertensives in the UK. Blood pressure, salt intake and ethnic origin were independent predictors of urinary Ca in this multi-ethnic population. The authors concluded that ethnic differences in renal tubular handling may exist, rather than differences in Ca intake or intestinal Ca absorption since the relationships remained in subjects who had undergone an overnight fast. The lack of an association between urinary Na and Ca in white women in our study, even after controlling for reported Na intake and BP, as well as for treatment with diuretic agents (data not shown), cannot be explained and warrants further investigation.

Regarding magnesium status, no differences in either urinary magnesium excretion or dietary magnesium intake were found between black and white subjects in the present study, however in all ethnic groups, intakes were low. A significant, inverse association between both serum and erythrocyte magnesium and blood pressure has been reported in urban black male labourers in Johannesburg.[64] The authors concluded that body magnesium status, and its interactions with calcium, sodium, and potassium, may play an important role in the development and maintenance of elevated blood pressure in the South African black population.

It has been demonstrated that hypertensive black South Africans have intracellular Na and Ca overload, accompanied by intracellular Mg depletion.[65] These findings are in contrast to published studies of reported dietary intake patterns of the South African black population, in which a similar Na and Mg intake between black and white subjects, accompanied by a lower K and Ca intake, is generally demonstrated.[56] Milne[66] hypothesizes that the difficulty in reconciling dietary intake data with the marked intracellular changes (i.e. increased Na and Ca, decreased Mg) may be due to an inherited abnormality of one or all three of the cation pumps. In this regard, a decrease in both platelet and erythrocyte membrane activity has been found for Na-K-ATPase, Ca-ATPase and Mg-ATPase in hypertensive blacks compared to either white hypertensives or normotensive blacks.[62] The authors reported an inverse association between platelet membrane ATPase activity and blood pressure in black subjects, and the lowest activities were found in those with malignant phase hypertension. Alternatively, over-expression of endothelin, a vasoactive peptide, has been shown in African-American hypertensive patients[67] and this may influence pump activity, resulting in an altered cation status.

In our study, more hypertensives than normotensives had a low renin status (i.e. active renin <10 uU/ml) and there was a trend for low renin concentrations to be more common in black subjects. The trend for lower renin concentrations in black, compared to either mixed ancestry or white, hypertensive subjects suggests salt and water overload as a pathogenetic mechanism and renin-angiotensin-aldosterone suppression as a normal physiological response. In addition, a higher aldosterone:renin ratio was found in black hypertensive, compared to normotensive subjects, but not in white subjects. We identified that over a quarter of our sample (29 %) of black hypertensives had low renin hypertension (LRHT) (low renin and normal aldosterone concentrations) which is consistent with other studies that have described this entity to be common in black hypertensives elsewhere.[68,69]

Our findings are similar to those of Cohen *et al.*[37] who reported that black South Africans with mild-to-moderate hypertension had significantly lower mean plasma renin levels than their normotensive counterparts. In the Cohen study, the severely hypertensive group had low renin levels, and only in the group with malignant hypertension and advanced renal failure did renin rise. In the present study, plasma renin concentrations did not differ across stages of hypertension in black subjects, however none of our subjects had blood pressure measurements higher than the classification of stage 2 hypertension (data not shown). A study of urban and rural Xhosas[70] found that in the urban, as well as rural normotensive and hypertensive subjects, the plasma renin concentrations were significantly lower than those reported for white populations.

The epithelial sodium channel (ENaC) is the principal site for regulating the amount of sodium reabsorbed by the kidney, and increased activity of this channel is the final common abnormality in several forms of low-renin hypertension: primary aldosteronism, glucocorticoid remediable aldosteronism, Liddle's syndrome, and 11-β-hydroxysteroid-2 dehydrogenase deficiency.[71] Activating mutations of either the β or γ ENaC subunits can result in Liddle's syndrome,[72,73] where constitutive reabsorbtion of Na leads to hypertension, low plasma levels of renin and aldosterone, and hypokalemia. Rayner and colleagues have demonstrated an association between the R563Q mutation in the β-subunit of the ENaC and low-renin, low-aldosterone hypertension in black and mixed-ancestry South African patients.[40] They studied patients from different ethnic groups who had a tightly defined low renin (PRA < 0.5 ng/ml/hr) and low aldosterone (< 225 pmol/L) profile. These highly selected patients were contrasted to subjects with normal or high renin hypertension. A new mutation, R563Q, of the beta subunit of ENaC was found

more frequently in low renin hypertensive (LRHT) black patients than in LRHT white patients. Within the black group the gene mutation was found in 28.6% of the LRHT group compared to 5.5% of the normal or high renin hypertensives. None of the 103 black normotensive controls, nor any of the 136 white hypertensives, had the mutation. The same authors have subsequently reported a strong and unequivocal association of this mutation with apparent essential hypertension, but not Liddle's syndrome, in family members.[74]

In the present study, very few subjects had both a low renin and low aldosterone (< 111 pmol/L) status (n = 11; 3.8 %), however most (8/11) of these subjects were black. Using the less stringent criteria for defining a low aldosterone concentration (< 225 pmol/L) used by Rayner *et al.*, [40] over a quarter (27 %) of black hypertensives, compared to a tenth of black normotensives, had a low renin/low aldosterone status.

A number of factors limit the interpretation of our data. Antihypertensive therapy may have influenced the renin and aldosterone levels.[75] Indeed, as expected, we found higher plasma renin and aldosterone:renin concentrations in hypertensive black and white subjects being treated with either diuretics or ACE inhibitors, compared to those that were not using these agents. It is impractical and inappropriate in a non-clinical setting to withdraw antihypertensive medication due to potential unacceptable risks. Further, since the primary objective of this study was to determine habitual intakes of Na, no standardization of salt intake was undertaken.

Regarding the influence of being overweight on blood pressure, BMI and waist circumference (data not shown) were positively and significantly associated with blood pressure in this sample of multi-ethnic South Africans. Similar to previously reported data from both urban and rural Zulu South Africans,[76] a positive association was demonstrated between BMI and urinary Na excretion. Adipocytes locally synthesize components of the renin-angiotensin system; obesity is a volume overloaded state and the resultant hypertension could be due to abnormal polymorphisms of the local renin-angiotensin system, or of the angiotensinogen gene. In this regard, a study by Tiago *et al.*[77] found that the angiotensinogen (AGT) promoter region variant gene was present with a highly significant Odds Ratio in black hypertensive South Africans with a BMI of over 27 kg/m^2 compared to normotensive controls. The AGT gene expressed in adipose tissue would directly accelerate the production of angiotensin II as well as increase leptin production, thus stimulating the sympathetic nervous system with vasoconstriction and increased plasma volume leading to hypertension. Data from Jamaica has demonstrated

that, compared with their non-obese counterparts, obese Jamaicans (BMI >31) have higher serum ACE activity and angiotensinogen levels (the latter is expressed in adipose tissue).[78]

There are methodological limitations to the cross sectional nature of the study that need to be considered. Observational studies that consider risk factors (in this case, the exposure is considered to be urinary Na excretion) for certain diseases (in this case, raised BP) are distinct from randomised controlled trials because the researcher does not manipulate the exposure. The exposure is merely measured and its association with the outcome is analysed. In a cross sectional study such as the present one, data on exposure and outcome are measured at the same point in time, on the same individuals. The major limitation of cross sectional study designs is that they are unable to confirm causality.[79] However, data obtained from observational studies may still play an important role in describing trends and generating hypotheses about associations.

The type of non-random sampling used (ie. convenient, stratified) may have introduced selection bias which, together with sample size impacts on the generalizability of the findings. For example, significant differences in the mineral intakes between individuals who responded and those that did not may be present. However, it is not possible to answer this question retrospectively since non-responders were not followed up further. Every effort was made to encourage eligible subjects to participate in the study. Fieldworkers set up a research facility within the workplace clinic and remained in regular contact with subjects, either through visits or telephonic contact.

The results are applicable to economically active adults working in Cape Town. The sampling frame (ie. Cape Town City Council offices) was purposively selected in order to obtain sufficient numbers of subjects from the three ethnic groups who had a similar socioeconomic status. The sample size was calculated using the only available published data on urinary Na excretion values in normotensive black South Africans. The relatively small numbers for comparison in the ethnic/hypertension strata may have resulted in the demonstrated differences being due to chance. However, the conventional statistical significance level of $P = 0.05$ was used and, where indicated, potential confounding variables were controlled for in regression analyses.

In order to improve the validity of the study findings, the gold standard measure for assessment of habitual sodium intake, namely 24-hr urinary Na assessment, was used. As a marker of completeness of collection, the method of Bingham *et al.*[29] whereby non-

metabolizable para-aminobenzoic acid is administered during the collection period was used. Three repeated 24-hr urine collections measurements may not have been sufficient to accurately estimate habitual Na excretion. Indeed, Simpson *et al*[80] estimated that fifteen 24-hr urine collections are required to characterize an individual's sodium output with 95 % accuracy. It was not possible in the present study to increase the number of repeated urine collections due to logistical limitations and respondent burden associated with the procedure in free-living subjects. A more detailed discussion of the validity of both dietary and urinary estimations of Na intake is provided in Chapter 5.

Despite these limitations, the findings of this study have identified that black South Africans have a dietary pattern which is low in Ca, K, and Mg, but high in Na. Against this context, together with a possible predisposition to inherited abnormalities in cation pump activity, an investigation of the impact on blood pressure of dietary modification of the cation content of commonly consumed foods appears warranted. The low awareness of dietary factors being influential in the development of hypertension further indicates a need for population-based approaches. In this regard, this study has provided useful information on inter- and intra-individual variation in blood pressure, as well as urinary Na excretion and reported dietary Na intake across three ethnic groups. Such data is required in order to calculate sample size requirements in intervention studies.

Conclusions

White normotensive subjects in Cape Town have higher habitual intakes of sodium, but also higher calcium intakes, than their black or mixed ancestry counterparts In all three ethnic groups investigated, sodium intake exceeded the recommended maximum of 6 g/day, while potassium intake fell far below the recommended minimum of 90 mmol/day. These findings indicate a need for population-based approaches to change dietary behaviour in order to both prevent the development of hypertension and to improve blood pressure control in hypertensives.

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Chapter 4

The contribution of foods to total sodium, potassium, calcium and magnesium in the South African diet

Introduction

The causes of hypertension are complex and multifactorial, however the adoption of an unhealthy lifestyle is recognized as being a major risk factor in its development.[1] As outlined in detail in the literature review of this thesis (Chapter 1), the nutrients of most interest in the diet-blood pressure relationship, apart from energy and alcohol, are sodium, potassium, magnesium and calcium.

The Dietary Approaches to Stop Hypertension (DASH) randomized controlled trial provided evidence that adoption of a dietary pattern that is rich in fruit, vegetables and low fat dairy products can reduce blood pressure as much as some anti-hypertensive drugs.[2] Increased efficacy of the DASH diet among African Americans[3] supports other data suggesting ethnic differences in blood pressure response to diet, which may be related to differences in habitual dietary patterns.[4] The follow-up DASH Sodium study demonstrated additional blood-pressure lowering benefits of salt restriction over and above the merits of the DASH diet[5] although the greatest benefits in sodium restriction are seen in those with a high fat and micronutrient-poor diet (i.e. typical American control diet).

In South Africa, a diversity of ethnic and cultural groups exists, each with different eating patterns and at different stages in the nutrition transition.[6] The reported dietary patterns of the majority of South Africans indicate a marginal micronutrient intake and an inadequate dietary fibre intake.[7,8] Due to high levels of food insecurity that exist in the poor sector of the population[9] and the very low intake of fruit, vegetables and dairy products by the majority of the population,[8] promotion of the DASH eating plan in the South African context is unrealistic at this time.

Regarding salt intake, in the early 1980's urinary excretion studies in Johannesburg demonstrated a lower sodium intake in black normotensive and hypertensive South Africans, compared to their white counterparts. [10,11] However, at that time, mean sodium excretion of the black groups exceeded the current international recommendation of a maximum of 6 g salt per day. [12] Potassium excretion of black subjects was low, suggesting an inadequate intake of potassium-rich foods, such as fruit and vegetables. [10,11]

In many other countries, the food industry has gradually reduced the amount of salt in many of their products, as a result of collaboration between food manufacturers and their trade bodies, governmental organizations, retailers, the catering and food service sector, academia, and consumer groups. In order to facilitate a population-wide strategy to lower salt intake in South Africa, it is necessary to identify the major food sources of

sodium in the local diet. Further, identification of food sources of potassium, magnesium and calcium in different ethnic groups would enable nutrition education activities to be culturally appropriate in terms of the types of foods that could be promoted to improve blood pressure levels.

Thus, the present study was undertaken to identify which food sources are the major contributors to sodium, potassium, calcium and magnesium intake.

Methods

Subjects and sampling

Three hundred and twenty five men and women from three different ethnic groups (black, mixed ancestry and white), aged between 20 and 65 years, were recruited from their place of work, Cape Town City Council offices in central Cape Town, South Africa. Equal numbers of hypertensive (BP ≥140/90 mm Hg and/or on antihypertensive medication) and normotensive (BP <140/90mm Hg) men and women were planned (n = 150 in each group; 50 from each ethnic group). Approval for the study was granted from the Research and Ethics Committee of the University of Cape Town and written informed consent was obtained from all participating subjects.

Dietary intake

Dietary intake was assessed using an interviewer-administered 24-hour recall method, for each of the three urinary collection periods. Fieldworkers were trained in the 24-hour recall procedure and standard household measuring utensils, rulers and validated food photographs of typical South African foods[13] were used to quantify food portion sizes. The recorded quantities of food consumed were converted to gram weights using the Medical Research Council (MRC) Food Quantities Manual.[14] Average daily nutrient intake was calculated using the Foodfinder III computerised dietary assessment programme, which is based on the MRC Food Composition Tables.[15,16] Added salt intake was not quantified in the 24-hour dietary recall assessments, therefore data on sources of sodium intake refer only to non-discretionary sources (i.e. naturally inherent Na in food and drink, and/or salt added in food processing).

Foods and beverages were classified into food categories according to the GEMS/Food (Global Evaluation Monitoring System/ Food Contamination Monitoring and Assessment Programme) commodities of the World Health Organization,[17] namely: cereals; sugars and honey; nuts and oilseeds; vegetable oils and fats; stimulants; spices; pulses; roots

and tubers; vegetables; fish and seafood; eggs; fruit; milk and milk products; meat and offal; animal oils and fats. New groups were created for alcoholic beverages, dietary supplements and soup mixes. Detailed methodology is described elsewhere.[18]

Contribution of intakes from the various food groups towards total Na, K, Mg and Ca intake was assessed, by ethnic group. Thereafter, the contribution of individual food items to mean total daily intake of Na, K, Mg and Ca was determined, by ethnic group, by ranking foods in descending order for cation content of mean group intake of that food item.

For comparative purposes, secondary data analyses of other dietary surveys undertaken in adult South Africans, which utilized a 24-hour recall method, were conducted to assess quantities and food sources of sodium intake. The databases included two studies of rural black subjects (Lebowa (1998; n = 292; age = 10 - 25 yrs)[19,20] and Dikgale (1992; n = 209; 19 + yr)[21,22], a study of urban black residents in Cape Town (BRISK study: 1990; n = 1 243; 10 - 89 yr)[23,24] and a study of rural white subjects in the Western Cape (1989; n = 1 784; 15 - 99 yr).[25,26,27] The same dataset, together with the Cape Town study, was used to assess the frequency of intake of servings from the main food groups of the DASH diet in an attempt to assess the proportion of the South African population who have intakes in line with the DASH diet recommendations.

Statistical analyses

For each of the dietary variables, values are expressed as means of the three separate measurements (or the maximum number of valid measurements obtained for each subject). Differences between ethnic groups for numerical variables were assessed using either the ANOVA or the Krusal-Wallis ANOVA (non-parametric data) tests. The inter- (between-subject) and intra- (within-subject) individual variation in reporting of dietary Na, K, Mg and Ca intake is calculated as the coefficient of variation (CV = (SD/mean)) and expressed as a percentage. Intra-individual CV is calculated as the variation within each subject between the three repeated dietary assessments for each of the variables. Analyses on variability were conducted only in subjects for whom data from all three of the repeated measurements was available.

Results

Characteristics and dietary cation and energy intake of sample

A total of 110, 112 and 103 subjects were recruited from black, mixed ancestry and white ethnic groups, respectively. Subjects in each of the ethnic groups were comparable, in

terms of proportion of hypertensives and proportion of men to women, however white subjects were significantly older than either black or mixed ancestry subjects (Table1). It has been reported elsewhere in this thesis (Chapter 3) that mean urinary Na excretion in this sample equates to a daily salt (sodium chloride) intake of 7.8g, 8.5g and 9.5g in black, mixed ancestry and white subjects, respectively, and that 23 % of the total sample had urinary Na concentrations which met the JNC 7 guidelines (ie. < 100 mmol/day).[28] Mean reported dietary intake of Na, K, Mg and Ca is shown in Table 1, according to ethnic group.

Table 1
Mean reported daily dietary intake of cations, according to ethnic group: mean (SD)

	Black (N=110)	Mixed ancestry (N=112)	White (N =103)
Age*	39.7 (10.5)	39.4 (10.5)	46.9 (9.5) [b,c]
Sex (M/F)	54/56	51/61	54/49
Normotensives/Hypertensives (n)	51/59	48/64	46/57
Dietary Na (mg/day)**	1 467 (891)	1 761 (885) [a]	1 922 (911) [b]
Dietary K (mg/day)***	2 058 (831)	2 092 (648)	2 513 (903) [b]
Dietary Mg (mg/day)*	261 (110)	228 (105) [a]	285 (122) [c]
Dietary Ca (mg/day)**	436 (332)	464 (215)	577 (274) [b,c]
Dietary energy (kJ/day)	7 363 (2 641)	7 747 (2 820)	7 994 (2 798)

*P<0.05; ** P<0.001; ***P<0.0001; ‡ P = 0.0536: ANOVA for differences between ethnic groups. Post-hoc analyses:
[a] Black vs Mixed ancestry; [b] Black vs White; [c] Mixed ancestry vs White.

Dietary sources of sodium

The contribution of food groups (i.e. non-discretionary salt intake) to total sodium intake is shown in Table 2, by ethnic group. In all three sub-samples, cereals were the main contributor to total reported dietary sodium intake (45.9 – 48.6 %), followed by meat and meat products (20.3 – 23.6 %) and milk and dairy products (6.3 – 8.1 %).

The top twenty individual food items which contributed to total percentage Na intake are shown, for each ethnic group, in Tables 3 a - c. In all groups, bread was the major source of dietary sodium (25.2 - 40.5 %), while meat products such as processed meats (*polony* (a pork-based product), vienna sausages, salami, ham, other sausages) and commercial meat pies, and margarine (brick type) were important sources.

Table 2
Percentage contribution of food groups and bread to total Na intake in South African adults

Ethnic group	Black urban (present study)	Black Urban (BRISK)	Black Rural	Black Rural	White Urban (present study)	White Rural (CORIS)	Mixed ancestry Urban (present study)
Place and year of study	Cape Town 2002	Cape Town 1990	Dikgale 1998	Lebowa 1992	Cape Town 2002	Western Cape province 1983	Cape Town 2002
N	110	1 243	209	292	103	1 784	112
Age (years)	20 - 65	10 - 89	19 +	10 - 25	20 - 65	15 - 99	20 - 65
Total reported Na intake (mg/day)	1 459	1 258	759	1 070	1 922	2 293	1 761
Cereal and cereal products	48.6	53.9	74.8	70.3	45.9	37.2	45.9
Bread (All types)	40.5	51.9	73.1	66.4	25.2	24.6	30.7
Bread (Brown)	17.3	18.9	57.3	46.6	5.7	9.7	6.7
Bread (White)	22.3	32.6	15.8	19.6	15.0	12.0	22.0
Meat and meat products	20.3	19.9	10.2	10.4	23.6	28.9	22.6
Milk and milk products	6.3	7.8	0.9	3.3	7.0	12.3	8.1
Soups	5.9	2.7	1.8	0.4	2.5	1.5	3.4
Roots	4.5	1.4	0.4	0.3	5.1	2.8	3.7
Fats and oils	4.3	6.3	1.3	2.9	3.9	6.7	3.9
Fish and seafood	2.1	1.9	1.3	5.1	4.3	2.6	2.4
Sauces, seasonings and flavourings*	1.9	0.09	0.08	-	0.5	1.2	2.3
Eggs	1.2	1.6	1.6	2.2	1.1	1.7	1.1
Legumes and legume products	1.1	0.7	2.3	0.6	1.3	0.7	0.3
Vegetables	0.9	0.8	2.0	2.8	1.4	1.6	1.7
Stimulants	0.7	0.6	1.9	0.7	1.1	1.2	1.6
Sugar, syrups and sweets	0.7	1.1	0.09	0.1	1.0	0.9	1.1
Fruit	0.6	0.2	0.07	0.06	0.3	0.3	0.4
Nuts and seeds	0.6	0.5	1.1	0.9	0.6	0.3	0.7
Alcohol	0.3	0.6	0.3	0.02	0.3	0.3	0.7
Supplements	-	-	-	0.04	-	0.05	-

* Includes Marmite, sandwich spread, chutney.

Table 3 (a)
Contribution of top 20 individual food items to total non-discretionary Na intake[a] - black subjects ((n = 110)

Ranking	Food item	% Consumers	Mean portion size (g) (Consumers)	Mean Na (mg/day) (Consumers)	% Total Na intake (Group)
	Bread – all types	93.6	134.6	631.9	40.54
1	**Bread/rolls - white**	74.6	89.0	435.9	22.26
2	**Bread/rolls - brown**	59.1	94.6	426.5	17.27
3	**Beef sausage - Boerewors**	12.7	59.1	475.3	4.15
4	**Steak and kidney pie (commercial)**	13.6	71.8	366.1	3.42
5	**Soup powder (reconstituted)**	10.9	90.8	391.5	2.93
6	**Margarine - brick/hard**	57.3	9.2	73.8	2.90
7	**Polony**	20	18.2	185.0	2.53
8	**Maas/sour milk**	26.4	189.9	134.9	2.44
9	**Potato chips/french fries**	28.2	57.9	114.6	2.21
10	**Milk - full cream, fresh**	53.6	120.4	57.8	2.12
11	**Potato crisps**	12.7	22.4	224.3	1.96
12	**Popcorn, plain**	1.8	58.3	1131.7	1.41
13	**Salami, pork/beef (Russian)**	4.6	23.8	442.7	1.38
14	**Sausage roll (commercial)**	7.3	64.4	276.4	1.38
15	**Breakfast cereal - All Bran flakes**	3.6	61.1	491.1	1.22
16	**Soup - vegetable (canned)**	3.6	140.4	478.8	1.19
17	**Vienna sausage (canned)**	6.4	27.4	260.9	1.14
18	**Chicken pie (commercial)**	4.6	68.3	363.9	1.13
19	**Aromat**	3.6	1.2	453.3	1.13
20	**Bread/rolls - wholewheat**	9.1	42.7	162.1	1.01

a Arranged in descending order of % total Na intake (group).Together, these 20 food items make up 75.2 % of total reported Na intake for the group.

Table 3 (b)
Contribution of top 20 individual food items to total non-discretionary Na intake[a] - mixed ancestry subjects (n = 112)

Ranking	Food item	% Consumers	Mean portion size (g) (Consumers)	Mean Na (mg/day) (Consumers)	% Total Na intake (Group)
	Bread – all types	97.3	117.7	555.7	30.70
1	**Bread/rolls - white**	87.5	90.5	443.4	22.03
2	**Bread/rolls - brown**	30.4	86.0	387.8	6.68
3	**Beef sausage - Boerewors**	23.2	62.4	502.1	6.62
4	**Breakfast cereal - Cornflakes**	10.7	39.4	476.7	2.90
5	**Potato crisps**	24.1	19.9	199.1	2.73
6	**Polony**	26.8	14.0	143.0	2.17
7	**Bread/rolls - wholewheat**	17.9	51.7	196.3	1.99

Ranking	Food item	% Consumers	Mean portion size (g) (Consumers)	Mean Na (mg/day) (Consumers)	% Total Na intake (Group)
8	**Cheese - cheddar**	42.0	16.6	80.6	1.92
9	**Milk - full cream, fresh**	72.3	96.5	46.3	1.90
10	**Margarine - brick/hard**	65.2	6.4	51.1	1.89
11	**Potato chips/french fries**	34.8	42.1	83.4	1.65
12	**Savoury snack - corn chips (eg. Niknaks)**	17.0	14.9	158.7	1.53
13	**Breakfast cereal - All Bran flakes**	5.4	61.8	497.1	1.51
14	**Steak and kidney pie (commercial)**	10.7	41.5	211.5	1.29
15	**Fish biltong (salted, dried cod)**	0.9	33.3	2342.3	1.19
16	**Vienna sausage (canned)**	12.5	16.9	161.1	1.14
17	**Chicken pie (commercial)**	7.1	52.1	277.6	1.13
18	**Baked beans (canned)**	17.0	26.5	105.2	1.01
19	**Salami, pork/beef (Russian)**	1.8	51.7	961.0	0.97
20	**Sausage roll (commercial)**	7.1	52.0	223.3	0.91

a Arranged in descending order of % total Na intake (group).Together, these 20 food items make up 63.2 % of total reported Na intake for the group.

Table 3 (c)
Contribution of top 20 individual food items to total non-discretionary Na intake[a] - white subjects (n = 103)

Ranking	Food item	% Consumers	Mean portion size (g) (Consumers)	Mean Na (mg/day) (Consumers)	% Total Na intake (Group)
	Bread – all types	96.1	109.8	503.5	25.18
1	**Bread/rolls - white**	76.7	76.3	376.0	15.24
2	**Bread/rolls - brown**	37.9	64.6	291.4	5.74
3	**Bread/rols - wholewheat**	35.0	60.8	231.0	4.20
4	**Breakfast cereal - All Bran Flakes**	9.7	103.2	829.5	4.19
5	**Breakfast cereal - Corn Flakes**	7.8	62.6	758.4	3.06
6	**Vienna sausage (canned)**	13.6	37.9	360.8	2.55
7	**Beef sausage - Boerewors**	16.5	35.1	282.5	2.43
8	**Sausage - pork**	7.8	36.9	477.2	1.93
9	**Cheese - cheddar**	53.4	13.9	67.8	1.88
10	**Pizza**	8.7	67.8	386.3	1.76
11	**Steak and kidney pie (commercial)**	9.7	65.9	335.9	1.70
12	**Milk - full cream, fresh**	68.9	90.4	43.4	1.56
13	**Potato chips/french fries**	27.2	55.5	109.8	1.55
14	**Margarine - brick/hard**	52.4	6.9	55.5	1.51
15	**Soup - vegetable**	5.8	145.8	497.3	1.51
16	**Ham – cokked/canned**	20.4	9.4	124.4	1.32
17	**Potato crisps**	14.6	17.2	171.8	1.30
18	**ProVita crackers**	12.6	27.8	197.5	1.30
19	**Bacon - fried, lean**	4.9	22.0	501.6	1.27
20	**Low fat spread - polyunsaturated**	26.2	10.3	87.5	1.19

a Arranged in descending order of % total Na intake (group).Together, these 20 food items make up 57.0 % of total reported Na intake for the group.

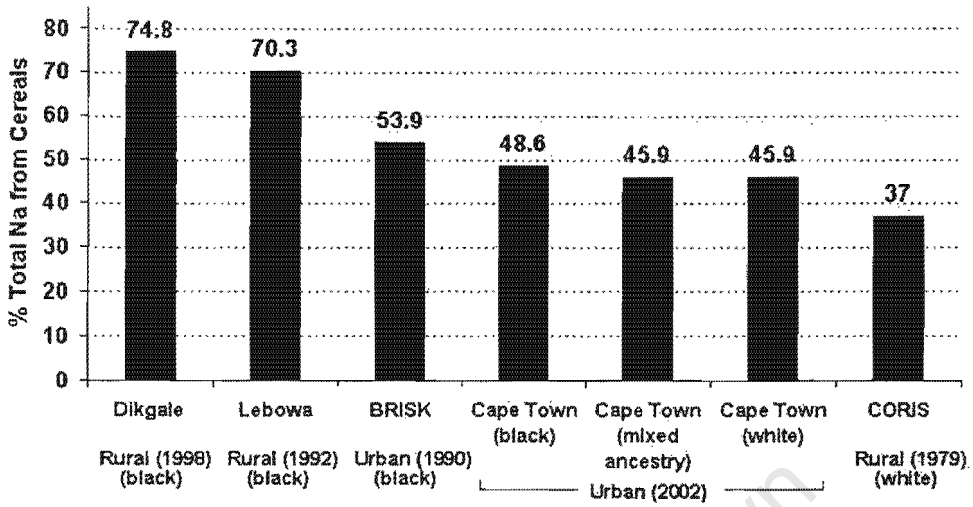

Figure 1
Proportion of Na provided by cereals food group in South Africans

Potassium

Only 8 % of subjects had reported K intakes which met or exceeded the recommendation of 90 mmol/day[29] (3 330 mg), with significantly more ($P<0.001$) white (16.5 %) than black (3.6 %) or mixed ancestry (4.5 %) subjects meeting this goal. Reported dietary K intake was significantly ($P<0.0001$) lower in black compared to white subjects (Table 1). In black subjects only, reported K intake was lower in hypertensives compared to normotensive subjects ($P<0.05$) (data not shown).

The cereals food group contributed the highest proportion of total dietary K intake (ie. 17.1 % - 26.8 %) (Tables 6a - c) across all ethnic groups, and provided a higher absolute amount of K in black subjects (548 mg/day), compared to mixed ancestry (358 mg) or white subjects (435 mg/day). Fruit provided a similar amount of K across ethnic groups (mean = 210 - 259 mg/day; 10.0 - 12.6 % total K), but in white subjects the vegetables food group provided almost double that of black subjects (189 and 97 mg/day, respectively). Potatoes were included in the roots food group, which provided 11.3 - 13.9 % of K (mean = 260 - 291 mg/day), while milk and dairy foods contributed about 11 % of K intake. Stimulants, which include coffee and tea, contributed 17.2 % of K intake in white subjects, compared to 12.7 % and 8.9 % in mixed ancestry and black subjects, respectively.

Table 6 (a)
Contribution of food groups to total K, Mg and Ca intake in black subjects (N = 110)

Food group	K intake of group		Mg intake of group		Ca intake of group	
	% total K	Mean (mg/day)	% total Mg	Mean (mg/day)	% total Ca	Mean (mg/day)
Cereal and cereal products	26.8	548.0	54.5	141.2	29.2	126.4
Meat and meat products	12.9	263.5	8.4	21.9	5.2	22.6
Roots	12.7	259.9	5.4	14.0	1.9	8.1
Milk and milk products	11.4	232.4	7.1	18.3	46.7	201.8
Fruit	12.6	258.1	5.5	14.2	3.1	13.2
Vegetables	4.7	96.5	2.8	7.3	3.2	14.1
Fish and seafood	2.6	52.5	1.2	3.2	3.5	15.1
Stimulants	8.9	181.8	6.3	16.3	1.2	5.4
Eggs	0.7	14.0	0.5	1.2	1.3	5.8
Pulses (Legumes and legume products)	2.5	50.2	2.9	7.4	1.1	4.8
Vegetable fats and oils	1.3	26.0	0.08	0.2	0.3	1.2
Nuts and seeds	0.9	18.5	1.8	4.5	0.4	1.7
Soups	0.7	14.8	0.3	0.8	0.8	3.4
Sugar, syrups and sweets	0.4	8.9	0.9	2.4	1.4	6.2
Alcohol	0.9	18.6	2.2	5.7	0.5	2.4
Condiments (Sauces, seasonings and flavourings)	0.2	3.2	0.2	0.4	0.08	0.3
Spices	-	-	-	-	-	-
Total intake (mg/day)		**2046.8**		**259.0**		**432.5**

Table 6 (b)
Contribution of food groups to total K, Mg and Ca intake in mixed ancestry subjects (N = 112)

Food group	K intake of group		Mg intake of group		Ca intake of group	
	% total K	Mean (mg/day)	% total Mg	Mean (mg/day)	% total Ca	Mean (mg/day)
Cereal and cereal products	17.1	358.1	40.9	93.3	26.7	124.0
Meat and meat products	17.5	366.9	12.8	29.1	5.1	23.7
Roots	13.9	290.9	6.9	15.8	2.2	10.0
Milk and milk products	10.6	221.1	8.3	18.9	49.1	228.1
Fruit	10.0	209.8	5.3	12.0	2.1	9.7
Vegetables	6.2	129.5	4.1	9.4	2.6	11.9
Fish and seafood	5.3	111.0	3.3	7.5	3.5	16.4
Stimulants	12.7	266.0	10.6	24.2	2.4	11.0
Eggs	0.7	14.3	0.5	1.2	1.3	6.2
Pulses (Legumes and legume products)	1.5	30.5	1.5	3.3	0.8	3.7
Vegetable fats and oils	1.4	29.1	0.2	0.4	0.6	2.7
Nuts and seeds	0.9	18.1	1.9	4.2	0.3	1.6
Soups	0.5	10.3	0.2	0.4	0.3	1.6
Sugar, syrups and sweets	0.4	8.7	1.3	3.0	2.2	10.2
Alcohol	1.2	26.0	2.3	5.2	0.6	2.9
Condiments (Sauces, seasonings and flavourings)	0.08	1.8	0.1	0.2	0.1	0.3
Spices	0.01	0.27	0.02	0.04	-	-
Total intake (mg/day)		**2092.4**		**228.2**		**464.1**

Table 6 (c)
Contribution of food groups to total K, Mg and Ca intake in white subjects (N = 110)

Food group	K intake of group		Mg intake of group		Ca intake of group	
	% total K	Mean (mg/day)	% total Mg	Mean (mg/day)	% total Ca	Mean (mg/day)
Cereal and cereal products	17.3	434.6	40.4	115.3	27.4	147.8
Meat and meat products	13.8	346.6	9.8	27.9	4.0	21.7
Roots	11.3	284.8	5.4	15.5	1.9	10.3
Milk and milk products	10.9	273.1	8.3	23.6	47.9	258.2
Fruit	10.3	259.1	5.7	16.2	2.8	14.9
Vegetables	7.5	189.2	4.5	12.8	3.2	17.1
Fish and seafood	3.0	76.3	1.8	5.1	2.2	12.0
Stimulants	17.2	431.0	14.2	40.4	3.7	20.1
Eggs	0.7	16.6	0.5	1.4	1.3	6.9
Pulses (Legumes and legume products)	0.8	20.2	1.3	3.8	0.4	2.1
Vegetable fats and oils	0.9	21.3	0.2	0.5	0.6	3.3
Nuts and seeds	0.7	17.7	1.4	4.1	0.3	1.5
Soups	0.5	0.6	0.2	0.6	0.4	2.2
Sugar, syrups and sweets	0.7	16.7	1.1	1.8	2.0	10.6
Alcohol	3.8	94.9	4.8	13.5	1.7	8.9
Condiments (Sauces, seasonings and flavourings)	0.6	14.4	0.4	1.1	0.1	0.7
Spices	0.07	1.9	0.1	0.1	0.1	0.7
Total intake (mg/day)		**2512.3**		**285.2**		**539.1**

The top twenty individual food items which contributed to total percentage K intake are shown, for each ethnic group, in Tables 7a - c.

Table 7a
Contribution of top 20 individual food items to total K intake[a] - black subjects (n = 110)

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean K (mg/day) (Consumers)	% Total K intake (Group)
	Bread - all types	93.6	134.6		10.94
1	Bread/rolls, brown	59.1	94.6	209.9	6.06
2	Coffee, brewed/instant	68.2	309.3	167.0	5.56
3	Potato chips/french fries	28.2	57.9	398.9	5.49
4	Samp and beans (1:1)	22.7	263.3	484.5	5.38
5	Milk, full fat, fresh	53.6	120.4	189.0	4.95
6	Maas/sour milk	26.4	189.9	360.9	4.65
7	Maize meal, cooked, stiff porridge	56.4	277.5	166.5	4.58
8	Bread/rolls - white	74.6	89.0	122.8	4.47
9	Potato (boiled)	49.1	50.2	140.0	3.36
10	Tea	54.6	321.8	119.1	3.17
11	Banana	29.1	64.1	154.4	2.19
12	Potato crisps	12.7	22.4	324.5	2.02
13	Samp and beans (1:2)	5.5	220.4	615.3	1.64
14	Peach	14.6	107.5	216.2	1.54
15	Grapes	12.7	110.0	236.5	1.47
16	Beef, steak, grilled	9.1	87.0	294.8	1.31
17	Rice, white, cooked	76.4	89.2	34.8	1.30
18	Non-dairy coffee creamer	37.3	8.4	68.0	1.24

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean K (mg/day) (Consumers)	% Total K intake (Group)
19	**Beef, braised topside**	5.5	139.7	430.3	1.15
20	**Orange**	9.1	128.2	225.6	1.00

[a] Arranged in descending order of % total K intake (group).Together, these 20 food items make up 62.5 % of total reported K intake for the group.

Table 7 b
Contribution of top 20 individual food items to total K intake[a] - mixed ancestry subjects (n = 112)

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean K (mg/day) (Consumers)	% Total K intake (Group)
	Bread - all types	97.3	117.7		8.96
1	**Coffee, brewed/ instant**	70.5	380.2	205.3	6.92
2	**Milk, full fat, fresh**	72.3	96.5	151.5	5.24
3	**Bread/rolls - white**	87.5	90.5	124.9	5.22
4	**Tea**	74.1	397.2	147.0	5.21
5	**Potato chips/french fries**	34.8	42.1	290.3	4.83
6	**Potato crisps**	24.1	19.9	288.2	3.32
7	**Bread/rolls, brown**	30.4	86.0	190.9	2.77
8	**Fish, medium fat, fried**	24.1	51.4	228.3	2.63
9	**Banana**	26.8	66.2	159.6	2.04
10	**Potato (boiled)**	40.2	28.2	78.7	1.51
11	**Grape**	17.0	81.4	175.0	1.42
12	**Milk, low fat, fresh**	23.2	83.8	127.4	1.41
13	**Sausage, boerewors**	23.2	62.4	122.3	1.36
14	**Non-dairy coffee creamer**	28.6	11.3	92.0	1.26
15	**Chicken, roasted**	17.0	48.3	129.8	1.05
16	**Mutton, loin chop, grilled**	22.3	29.7	97.2	1.04
17	**Potato (sauteed)**	14.3	41.9	146.1	1.00
18	**Rice, white, cooked**	74.1	71.5	27.9	0.99
19	**Mutton stew**	12.5	78.9	161.0	0.96
20	**Bread/rolls - wholewheat**	17.9	51.7	110.1	0.94

[a] Arranged in descending order of % total K intake (group).Together, these 20 food items make up 51.1 % of total reported K intake for the group.

Table 7 c
Contribution of top 20 individual food items to total K intake[a] - white subjects (n = 103)

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean K (mg/day) (Consumers)	% Total K intake (Group)
	Bread - all types	96.1	109.8		7.42
1	**Coffee, brewed/ instant**	86.4	629.5	293.7	11.69
2	**Tea**	60.2	508.5	113.3	4.51
3	**Potato chips/french fries**	27.2	55.5	103.9	4.14
4	**Milk, full fat, fresh**	68.9	90.4	97.9	3.90
5	**Bread/rolls - brown**	76.7	76.7	81.2	3.23
6	**Milk, low fat, fresh**	37.9	107.9	62.1	2.47
7	**Wine - red/white/rose**	26.2	253.0	59.0	2.35
8	**Bread/rolls - white**	37.9	64.6	54.3	2.16
9	**Breakfast cereal - All Bran Flakes**	9.7	103.2	49.9	1.99

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean K (mg/day) (Consumers)	% Total K intake (Group)
10	**Bread/rolls - wholewheat**	35.0	60.8	45.3	1.80
11	**Potato crisps**	14.6	17.2	36.2	1.44
12	**Potato (boiled)**	27.2	43.8	33.2	1.32
13	**Tomato, raw**	51.5	27.2	32.3	1.29
14	**Fish, medium fat, fried**	12.6	55.3	31.0	1.23
15	**Banana**	26.2	46.7	29.5	1.17
16	**Milk, skim, fresh**	11.7	147.6	28.6	1.14
17	**Avocado**	15.5	27.1	24.5	0.98
18	**Beer**	19.4	363.2	23.3	0.93
19	**Orange juice**	6.8	165.5	22.5	0.90
20	**Breakfast cereal, muesli**	9.7	60.7	20.3	0.81

[a] Arranged in descending order of % total K intake (group).Together, these 20 food items make up 49.4 % of total reported K intake for the group.

In all three ethnic groups, coffee and tea, bread, milk and potatoes (french fries, crisps or boiled) were important sources of overall K intake. Notably different to the other groups was the appearance of alcoholic drinks (beer and wine) and breakfast cereals (All Bran flakes, muesli) in the top 20 list in white subjects. Consistent with the food group analysis (Table 6a - c), few fruits or vegetables (with the exception of banana, grapes and orange/juice) were in the top 20 list of contributors to total K intake.

Magnesium

Reported dietary magnesium intake did not differ between black and white subjects, but was significantly lower in mixed ancestry subjects (Table 1). More black and mixed ancestry, compared to white, subjects had Mg intakes below 67 % DRI[27] (51.8 %, 67.9 % and 43.7 %, respectively; $P<0.05$). In normotensives, mixed ancestry and black subjects had significantly lower reported dietary Mg intakes than their white counterparts, but this difference was not evident in hypertensive subjects (data not shown). The cereals food group provided the majority of Mg intake (40.4 - 54.5 %, followed by the meat (8.4 - 12.8 %), milk (7.1 - 8.3 %) and roots (5.4 - 6.9 %) food groups (Tables 6 a-c). Stimulants contributed 6.3 - 14.2 % of total Mg intake. The top twenty individual food items which contributed to total percentage Mg intake are shown, for each ethnic group, in Tables 8 a-c.

Table 8 a
Contribution of top 20 individual food items to total Mg intake[a] - black subjects (N = 110)

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean Mg(mg/day) (Consumers)	% Total Mg intake (Group)
	Bread - all types	93.6	134.6	67.8	24.50
1	Bread/rolls - brown	59.2	94.6	70.0	15.97
2	Maize meal, cooked, stiff porridge	56.4	277.5	49.9	10.87
3	Bread/rolls - white	74.6	89.0	25.8	7.43
4	Samp and beans (1:1)	22.7	263.3	76.4	6.70
5	Coffee	68.2	309.3	15.5	4.07
6	Rice, white, cooked	76.4	89.2	11.6	3.42
7	Milk - full fat, fresh	53.6	120.4	14.5	2.99
8	Maas/sour milk	26.4	189.9	26.6	2.71
9	Potato chips/french fries	28.2	57.9	22.6	2.46
10	Samp and beans (1:2)	5.5	220.4	100.4	2.11
11	Tea	54.6	321.8	9.7	2.03
12	Breakfast cereal, Weetbix	15.5	26.9	31.2	1.86
13	Breakfast cereal, All Bran Flakes	3.6	61.1	111.2	1.56
14	Banana	29.1	64.1	13.5	1.51
15	Beer	5.5	717.2	64.6	1.36
16	Potato, boiled	49.1	50.2	6.0	1.14
17	Bread/rolls - wholewheat	9.1	42.7	31.6	1.11
18	Potato crisps	12.7	22.4	18.8	0.93
19	Orange juice	10.9	196.5	19.6	0.83
20	Peanut butter, smooth	16.4	8.3	13.1	0.82

[a] Arranged in descending order of % total Mg intake (group).Together, these 20 food items make up 71.9 % of total reported Mg intake for the group.

Table 8b
Contribution of top 20 individual food items to total Mg intake[a] - mixed ancestry subjects (N = 112)

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean Mg (mg/day) (Consumers)	% Total Mg intake (Group)
	Bread - all types	97.3	118.0	50.7	21.61
1	Bread/rolls - white	87.5	90.5	26.2	10.06
2	Bread/rolls - brown	30.4	86.0	63.6	8.46
3	Coffee	70.5	380.2	19.0	5.88
4	Tea	74.1	397.2	11.9	3.87
5	Milk - full fat, fresh	72.3	96.5	11.6	3.67
6	Rice, white, cooked	74.1	71.5	9.3	3.02
7	Bread/rolls - wholewheat	17.9	51.7	38.2	2.99
8	Breakfast cereal, All Bran Flakes	5.4	61.8	112.5	2.64
9	Potato chips/french fries	34.8	42.1	16.4	2.51
10	Breakfast cereal, Weetbix	26.8	17.1	19.8	2.33
11	Beer	5.4	870.0	78.3	1.84
12	Potato crisps	24.1	19.9	16.7	1.77
13	Banana	26.8	66.2	13.9	1.63
14	Oats/oatmeal - cooked	9.8	128.8	30.9	1.33
15	Milk - 2 %, low fat, fresh	23.2	83.8	10.1	1.02
16	Fish, medium fat, fried	24.1	51.4	9.3	0.98
17	Chicken, roasted	17.0	48.3	12.5	0.93
18	Maize meal, cooked, stiff	3.6	327.5	59.0	0.92

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean Mg (mg/day) (Consumers)	% Total Mg intake (Group)
	porridge				
19	Cheese, cheddar type	42.0	16.6	5.0	0.91
20	Peanut butter, smooth	24.1	5.3	8.3	0.88

[a] Arranged in descending order of % total Mg intake (group).Together, these 20 food items make up 57.7 % of total reported Mg intake for the group.

Table 8c
Contribution of top 20 individual food items to total Mg intake[a] - white subjects (N = 103)

Ranking	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean Mg (mg/day) (Consumers)	% Total Mg intake (Group)
	Bread - all types	96.1	111.9	54.4	18.32
1	Coffee	86.4	629.5	31.5	9.54
2	Breakfast cereal, All Bran Flakes	9.7	103.2	187.8	6.39
3	Bread/rolls - brown	37.9	64.6	47.8	6.35
4	Bread/rolls - white	76.7	76.7	22.3	5.99
5	Bread/rolls - wholewheat	35.0	60.8	45.0	5.51
6	Tea	60.2	508.5	15.3	3.22
7	Milk - full fat, fresh	68.9	90.4	10.9	2.62
8	Wine, all types	26.2	253.0	25.3	2.33
9	Beer	19.4	363.2	32.7	2.23
10	Breakfast cereal, muesli	9.7	60.7	61.9	2.11
11	Potato chips/french fries	27.2	55.5	21.6	2.06
12	Breakfast cereal, Weetbix	19.4	22.6	26.2	1.79
13	Milk - 2 %, low fat, fresh	37.9	107.9	13.0	1.72
14	Rice, white, cooked	62.1	60.5	7.9	1.71
15	Banana	26.2	46.7	9.8	0.90
16	Cheese, cheddar type	53.4	13.9	4.2	0.78
17	Potato crisps	14.6	17.2	14.4	0.74
18	Milk - skim, fresh	11.7	147.6	16.2	0.66
19	Chicken, boiled	22.3	39.5	8.3	0.65
20	Oats/oatmeal - cooked	6.8	111.4	26.7	0.64

[a] Arranged in descending order of % total Mg intake (group).Together, these 20 food items make up 57.9 % of total reported Mg intake for the group.

In all three groups, bread contributed the greatest proportion of total Mg intake, ranging from 18.3 % in white subjects to 24.5 % in black subjects (all bread types combined). In the black group, the staple foods maize meal porridge and dishes and samp (maize kernels) were important contributors to Mg intake (11.8 % and 7.7 % of total Mg intake, respectively). Coffee and tea, milk and maas (sour milk), white rice, and breakfast cereals were major sources of Mg common to all three ethnic groups.

Calcium

Mean dietary calcium was higher in white subjects compared to either the black or mixed ancestry groups, however in all groups calcium intake was low (about half of DRI of 1000 mg/day)[30] (Table 1). In black, mixed ancestry and white subjects, 84.4 %, 88.4 % and

78.6 %, respectively had inadequate (< 67 % DRI) calcium intakes. Calcium intake was significantly (P<0.005) lower in hypertensive than normotensive subjects, for both black and white ethnic groups (data not shown), and more hypertensive compared to normotensive subjects had inadequate (< 67 % DRI) dietary intakes of calcium (89.0 % vs 73.9 %; P<0.05).

The contribution of food groups to total Ca intake was similar across ethnic groups. The milk and milk products group provided the majority of Ca (46.7 - 49.1 % total Ca), followed by the cereal and cereal products group (26.7 - 29.2 % Ca) (Tables 6a - c). The top twenty individual food items which contributed to total percentage Ca intake are shown, for each ethnic group, in Tables 9 a - c. Notably, in the black sample, maas (sour milk) provided the most calcium. The milk and milk products food group provided 46.7 %, 49.1 % and 47.8 % of total Ca in black, mixed ancestry and white groups, respectively. Bread (all types combined) was also an important source of Ca, providing 16.2 %, 13.8 % and 10.6 % in black, mixed ancestry and white subjects, respectively.

Table 9 (a)
Contribution of top 20 individual food items to total Ca intake[a] - black subjects (n = 110)

Rank-ing	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean Ca (mg/day) (Consumers)	% Total Ca intake (Group)
	Bread - all types	93.6	134.6	75.8	16.20
1	**Maas, sour milk**	26.4	189.9	307.7	18.76
2	**Milk, full fat, fresh**	53.6	120.4	144.5	17.92
3	**Bread/rolls, white**	74.5	89.0	49.8	8.59
4	**Bread/rolls, brown**	59.1	94.6	52.0	7.11
5	**Cheese, cheddar-type**	13.6	13.7	107.7	3.40
6	**Breakfast cereal, ProNutro Great Start**	1.8	150.0	630.0	2.65
7	**Samp & beans (1:1)**	22.7	263.3	39.5	2.08
8	**Milk, low fat, fresh**	10.9	64.9	79.1	2.00
9	**Rice, white**	76.4	89.1	9.8	1.73
10	**Muffin, plain**	4.5	98.0	137.2	1.44
11	**Pilchards in brine**	7.3	20.0	72.0	1.21
12	**Steak and kidney pie, commerical**	13.6	71.8	38.0	1.20
13	**Mahewu/magou (traditional beer)**	4.5	266.7	98.7	1.04
14	**Cabbage, sauteed in sunflower oil**	21.8	46.2	20.3	1.03
15	**Cold drink, carbonated**	41.8	252.2	10.1	0.98
16	**Coffee**	68.2	309.3	6.2	0.98
17	**Orange**	9.1	128.2	38.5	0.81
18	**Offal**	9.1	54.8	37.3	0.78
19	**Milk, skim, fresh**	2.7	90.0	110.7	0.70
20	**Breakfast cereal, ProNutro**	1.8	35.7	164.1	0.69

a Arranged in descending order of % total Ca intake (group).Together, these 20 food items make up 75.1% of total reported Ca intake for the group.

Table 9 (b)
Contribution of top 20 individual food items to total Ca intake[a] - mixed ancestry subjects (n = 112)

Ranking	Food item	% Consumers	Mean portion size (g) (Consumers)	Mean Ca (mg/day) (Consumers)	% Total Ca intake (Group)
	Bread - all types	97.3	118.0	66.0	13.84
1	**Milk, full fat, fresh**	72.3	96.5	115.8	18.05
2	**Cheese, cheddar-type**	42.0	16.6	130.4	11.80
3	**Bread/rolls, white**	87.5	90.5	50.7	9.56
4	**Milk, low fat, fresh**	23.2	83.8	102.2	5.11
5	**Bread/rolls, brown**	30.4	86.0	47.3	3.09
6	**Yoghurt, fruit, low fat**	13.4	63.9	92.7	2.68
7	**Cheese, Gouda/Edam/Swiss-type**	7.1	18.5	149.1	2.30
8	**Muffin, plain**	10.7	58.6	82.0	1.89
9	**Cold drink, carbonated**	63.4	304.0	12.2	1.66
10	**Breakfast cereal, ProNutro**	0.9	166.7	766.7	1.48
11	**Rice, white**	74.1	71.5	7.9	1.26
12	**Coffee**	70.5	380.2	7.6	1.16
13	**Bread/rolls, wholewheat**	17.9	51.7	29.5	1.13
14	**Fish, medium fat, fried**	24.1	51.4	21.1	1.09
15	**Ice cream, commercial**	8.9	39.0	51.5	0.99
16	**Dairy fruit juice mix**	3.6	139.6	110.3	0.85
17	**Milkshake, vanilla, purchased**	1.8	135.0	197.1	0.76
18	**Doughnut, plain**	17.0	25.9	19.7	0.72
19	**Milk, skim, fresh**	7.1	37.9	46.6	0.72
20	**Milk, powder, low fat**	0.9	30.0	357.3	0.69

a Arranged in descending order of % total Ca intake (group).Together, these 20 food items make up 67.0% of total reported Ca intake for the group.

Table 9 (c)
Contribution of top 20 individual food items to total Ca intake[a] - white subjects (n = 103)

Ranking	Food item	% Consumers	Mean portion size (g) (Consumers)	Mean Ca (mg/day) (Consumers)	% Total Ca intake (Group)
	Bread - all types	96.1	111.9	63.4	10.56
1	**Milk, full fat, fresh**	68.9	90.4	108.5	12.96
2	**Cheese, cheddar-type**	53.4	13.9	109.7	10.15
3	**Milk, low fat, fresh**	37.9	107.9	131.6	8.64
4	**Bread/rolls, white**	76.7	76.7	43.0	5.71
5	**Milk, skim, fresh**	11.7	147.6	181.6	3.67
6	**Rusk, commercial, buttermilk**	3.9	83.3	448.3	3.02
7	**Bread/rolls, brown**	37.9	64.6	35.5	2.33
8	**Ice cream, commercial**	13.6	88.6	90.3	2.13
9	**Bread/rolls, wholewheat**	35.0	60.8	34.6	2.10
10	**Pizza, cheese and tomato**	8.7	67.8	128.8	1.95
11	**Coffee**	86.4	629.5	12.6	1.88
12	**Cheese, Gouda/Edam/Swiss-type**	7.8	16.2	130.6	1.76
13	**Muffin, plain**	10.7	56.5	79.1	1.46
14	**Yoghurt, fruit, low fat**	8.7	56.7	82.2	1.24
15	**Cold drink, carbonated**	53.4	292.2	11.7	1.08
16	**Rusk, commercial, white**	4.9	21.7	118.5	1.00
17	**Wine, all types**	26.2	253.0	20.2	0.92

Rank-ing	Food item	% Cons-umers	Mean portion size (g) (Consumers)	Mean Ca (mg/day) (Consumers)	% Total Ca intake (Group)
18	**Breakfast cereal, All Bran Flakes**	9.7	103.2	51.6	0.87
19	**Sweets, chocolate coated bar**	12.6	28.6	38.6	0.84
20	**Milkshake, vanilla, purchased**	1.9	163.3	238.5	0.80

a Arranged in descending order of % total Ca intake (group).Together, these 20 food items make up 64.5% of total reported Ca intake for the group.

Inter- and intra-individual variation in dietary intakes

Inter-individual coefficient of variation (CV) in the reporting of mean 3-day sodium intake for the total group was 52.8 %. Inter-individual CV, by ethnic group, was 59.2 %, 50.2 % and 47.0 % for black, mixed ancestry and white subjects, respectively. As expected, the inter-individual CV in Na intake was markedly higher if each of the single days of dietary reporting was considered in isolation (68.3 % - 77.6 for the total group; 69.9 % - 96.5 % for black subjects; 60.7 % - 85.7 % in mixed ancestry subjects; and 51.6 % - 71.3 % in white subjects). In the total group, intra-individual variation in reported Na intake over the three separate 24-hour recall periods was 44.4 % (95 % CI = 41.5 - 47.3 %). Black subjects had the highest between-subject variation in reported Na intake (CV = 50.9 %, 44.4 % and 37.6 % in black, mixed ancestry and white subjects, respectively ($P<0.005$)).

The inter-individual CV for reporting of potassium, magnesium and calcium intake within ethnic groups was 45.9 - 46.7 %, 43.0 - 45.9 % and 70.6 - 74.6 %, respectively. Intra-individual CV in 3-day reporting of potassium, magnesium and calcium intake was 35.7 %, 30.5 % and 54.3 %, respectively.

Proportion of the South African population consuming recommended number of servings in food groups, according to the DASH diet

Further secondary analyses of the 4 other South African dietary datasets demonstrated that the mean number of servings from the cereals, vegetables, fruit and dairy product food groups was low in the black populations sampled, particularly in the rural studies (Table 7). For these food groups, no rural black subjects had intakes that met the DASH diet recommendations, except for 6 % of the Lebowa sample meeting the dairy recommendation of at least 2 servings per day. In the BRISK urban black sample, more dairy foods were consumed, however less than a third of this group had 2 or more servings per day. Notably, over a quarter of Dikgale subjects were consuming one or more servings per day of the legumes, nuts and pulses food group. Negligible

proportions of any of the samples consumed 4 or more servings of vegetables and of fruit per day, except for the case of the two white groups.

Table 7
Mean number of times (SD) per day that items were consumed from the various food groups in the DASH diet, and proportion (%) of the sample consuming recommended number of servings[a] according to DASH diet†

Food group	Dikgale Black rural, 1998[21,22]	Lebowa Black rural, 1992[19,20]	BRISK Black urban, 1990[23,24]	CORIS White rural, 1979[25-27]	Cape Town (current study) Urban, 2002		
					Black	Mixed ancestry	White
Cereals/cereal products	2.30 (0.86)	3.37 (0.88)	2.55 (0.98)	3.83 (2.20)	2.61 (1.03)	3.17 (1.16)	2.84 (1.17)
% ≥ 7 servings	0	0.3	0	11.1	0	0.9	0
Vegetables[b]	0.84 (0.92)	1.09 (0.95)	1.48 (1.35)	3.09 (2.21)	1.33 (1.04)	1.92 (1.24)	2.22 (1.43)
% ≥ 4 servings	0.5	1	8.1	35.7	0.9	8.9	21.4
Fruits	0.16 (0.44)	0.21 (0.53)	0.43 (0.80)	1.51 (1.71)	0.79 (0.89)	0.83 (0.91)	1.04 (1.16)
% ≥ 4 servings	0	0.3	0.9	10.1	0.9	2.7	7.8
Dairy foods	0.11 (0.34)	0.28 (0.59)	1.06 (1.15)	4.32 (3.09)	0.88 (1.01)	1.89 (1.24)	3.10 (1.88)
% ≥ 2 servings	0.9	5.8	29.5	69.9	15.4	57.1	75.7
Meats, poultry, fish	0.67 (0.79)	0.84 (0.80)	1.38 (1.08)	2.36 (1.62)	1.12 (0.66)	1.73 (0.79)	1.54 (0.74)
% ≤ 2 servings	98.6	100	86.1	65.6	96.4	85.7	90.3
Nuts, seeds, legumes[c]	0.34 (0.63)	0.14 (0.44)	0.05 (0.25)	0.27 (0.57)	0.19 (0.33)	0.33 (0.37)	0.25 (0.35)
% ≥ 1 serving	27.3	12.7	4.4	22.3	5.5	10.7	6.8

[a] One serving taken as each reported frequency of consumption of items from that food group per day.
†The DASH eating plan shown above is based on 2000 kcal a day (8 400 kJ/d). Depending on energy needs, the number of daily servings in a food group may vary from those listed.
[b] Potatoes and root vegetables included in this group.
[c] DASH diet recommends 4-5 servings per week, but 24-hr recall methodology used in South African surveys allows only mean number of servings **per day** to be calculated therefore recommended serving of once per day was used in calculations.

Discussion

Data obtained from a multi-ethnic, economically active sample of South Africans in Cape Town, demonstrates that the bread and cereals food group is by far the largest contributor of non-discretionary dietary sodium intake (45.9 - 48.6) in this population. Bread was identified as the single food item that provided the greatest contribution to total dietary (non-discretionary) sodium intake, particularly in the black sub-sample. Other important food sources of sodium in black subjects included meat products, such as commercial meat pies, beef sausage (*boerewors*), and processed meats (polony, vienna, salami, ham, other sausages), as well as soup powders and margarine (brick). Together, these food items made up 64 % of total non-discretionary salt intake. In white subjects, a much more varied intake of individual food items was found, demonstrated by the much lower percentage of Na intake contributed by 20 foods (57.0 %), compared to mixed ancestry (63.2 %) and black subjects (75.2 %). Important differences in this group were that breakfast cereals made a substantial contribution to Na intake (fourth and fifth top foods), as did cheese, while crackerbreads, bacon and pizza also appeared in the

top twenty foods list. Dietary information such as this provides the basis for the targeting of specific commonly eaten foods in population-based non-pharmacological approaches to lowering blood pressure.

Secondary analyses of four regional dietary datasets from other South African populations, reported here for the first time, have demonstrated an urban/rural difference with regard to the contribution of food groups to total, non-discretionary Na intake in the diets of the black population. The bread and cereals food group contributes over 70 % of Na intake in rural areas,[19-22] while in black South Africans in urban Cape Town (present study and BRISK study[23,24]) this food group provides between 48.6 and 53.4 % of total Na intake. This suggests that rural dwellers have a less varied diet than do their urban counterparts. The meat and meat products food group provides double the amount of total non-discretionary salt intake in urban, compared to rural black subjects (10 % and 20 %, respectively), which probably reflects a higher purchasing power of these more expensive food items in the urban centres. No clear time trend in dietary patterns of salt intake from food sources is evident in black urban dwellers. The proportion of sodium provided by the" meat and meat products" and the "milk and milk products" groups was higher in white rural South Africans in the CORIS study conducted in 1979[25-27] (41.2 %), compared to the present (2002) sub-group of white subjects (30.6 %).

In terms of absolute amounts of habitually ingested sodium from non-discretionary sources, the lowest reported intakes are found in rural black subjects (759 - 1 070 mg/day), followed by urban black subjects (1 258 - 1459 mg/day), mixed ancestry subjects (1 761 mg/day) and then white subjects (1 922 - 2 293 mg/day). This finding suggests a greater reliance on processed foods by white South Africans, compared to the black population, in which maize meal (naturally low in sodium) remains the staple food item.

Subjective reporting of dietary intake is the tool most frequently used by the dietitian to determine baseline habitual intakes and to assess compliance of patients with dietary advice. Fifty-three percent variation was found, on average, between individuals for reported sodium intake, whereas in an individual, his/her reported dietary intake of sodium over a three-day period differed on average by 44 %. Variations are similar for calcium intake, but lower for reported potassium and magnesium intake. In large epidemiological surveys, a single 24-hr dietary recall is commonly used to assess nutrient intake. In this regard, our data demonstrate that the inter-individual variation in reporting of sodium intake, using a single 24-hr recall, can be as much as 71 % in white, 86 % in mixed ancestry and 97 % in black subjects. Clearly, repeated 24-hr recalls are

required in order to obtain a reliable estimate of usual Na intake. Indeed, it has been estimated that 81 days of dietary recording would be required to estimate an individual's intake within 10 % of the observed mean.[31] Our data is similar to previously reported estimates of high intra- (45 %) and inter- (45 – 56 %) subject variability for reporting of non-discretionary sources of Na (i.e. salt intake which excludes table salt and salt added in cooking).[31] Simple questions on habitual salt intake which are often included in studies as proxy measures for the true salt intake (e.g. salt taste preferences and the practice of adding salt to foods before tasting) were not found to be useful in distinguishing between either urinary Na excretion or reported dietary Na intake values in the present sample (see Chapter 3 - page 56).

As well as sodium intake, this study also investigated food sources of other nutrients known to be important in the diet-blood pressure relationship, namely potassium, calcium and magnesium.

In order to achieve a daily K intake of 4 700 mg per day, the DASH diet[2] recommends the consumption of 4 - 5 servings of fruit (providing about 620 - 780 mg K/day), as well as 4 - 5 servings of vegetables per day (860 - 1070 mg K/day). In the present study, the amount of K provided by fruits equates to only 1 - 2 servings daily, while potassium consumption from the vegetables group equates to less than one vegetable serving daily. Mean intake of root vegetables, including potatoes, contributed an additional vegetable serving to K intake, but overall K intake remained less than half of the amount included in the DASH diet.[1] Foods which were the single-most main contributors to total K intake across all three ethnic groups were coffee and tea, bread, milk and potatoes. The only published South African intervention study of potassium supplementation, conducted in 32 hypertensive black women, found that an additional 65 mmol/day of potassium chloride (i.e. 2 535 mg K - the amount which would make up the DASH diet deficit) reduced blood pressure by 7/3 mmHg over a 6-week period.[32]

Regarding calcium intake, our findings are similar to those reported in a study of older Chinese vegetarians who were habitually consuming a diet rich in sodium but deficient in calcium, and who had similar reported dietary calcium intakes to the black group in the present study.[33] Calcium intakes across all three ethnic groups were much lower than the recommended daily intakes.[34] Intake of dietary salt, as well as protein and caffeine, aggravate obligatory urinary calcium loss.[35] It is noteworthy that the addition of oral potassium citrate (90 mmol/day) to a high-salt diet (225 mmol Na/day) has been shown to prevent an increased urinary calcium excretion in posmenopausal women.[36] Our

findings suggest that the benefits of sodium restriction, together with an increased intake of potassium-rich fruits and vegetables, on blood pressure may be greatest in the black and mixed ancestry South African populations, in light of their extremely low calcium intakes.

An alternative approach would be to encourage an increased intake of calcium-rich foods. As expected, the main source of calcium intake in the present study was the milk and milk products food group, and a similar contribution to total calcium intake from this food group was found across all three ethnic groups. Nationally representative estimates of usual food consumption of the South African population have been undertaken by Steyn and colleagues using data from existing dietary surveys.[18] The authors reported that 30.6 % of the adult population consume items from the milk and dairy products food group, and average consumption (of consumers) is 239 g per day which equates to approximately one serving, providing around 280 mg Ca per day.[18]

In order to promote an increased intake of dietary calcium, it is necessary to identify which foods are rich sources thereof in the target population. This is in keeping with the food-based dietary guidelines approach proposed by the Food and Agricultural Organization and the World Health Organization (1998),[37] and recently adopted by South African Department of Health for the purpose of nutrition education in the country.[38,39] Maas (fermented, sour milk - commercially available) was the highest single contributor of calcium in our black sample and was consumed by over a quarter of the sample. As well as milk and cheese, bread was an important contributor of calcium intake in all three ethnic groups (13.8 % - 16.2 %).

In our analyses (including secondary analyses of other dietary datasets) of the proportion of individuals consuming the recommended number of servings from the various food groups according to the DASH diet principles,[2] we found a marked difference in dairy product intake between ethnic groups in the country. Mean frequency of intake from this food group was 3.1 - 4.3 times per day in white samples, compared to 0.9 to 1.1 times a day in black urban dwellers, and less than once a day, on average, in black rural groups. Over 70 % of the white samples consumed dairy products two or more times a day, but this number dropped to 15.4 - 29.5 % in black South Africans in urban areas, and was 0.9 - 5.8 % for black communities in rural areas. A limitation of our analyses is that in the secondary analyses of only reported frequency of intake was considered, rather than calculation of average number of servings according to pre-determined portion sizes. An example of inherent problems with this methodology is demonstrated in the case of the

cereals food group. The black sample in Cape Town had an average intake of 499 g/day, compared to 280g per day for their white counterparts, however average reported frequency of consumption was similar between groups (2.61 and 2.80, respectively). This is probably explained by larger portions of maize meal (staple food) being consumed by the black population than bread, rice, pasta or breakfast cereals in the white group. The analysis does however provide insight into dietary trends across area of residence (urban/rural) and according to ethnic and cultural preferences.

The role of magnesium in the prevention of hypertension is considered to be small and probably not of public health importance. A meta-analysis of trials of magnesium supplementation found a pooled estimate effect of a reduction in systolic BP of only 0.6 mm Hg (95 % CI: -2.2 to 1.0 mm Hg), and no effect on diastolic blood pressure.[40] However, few of the studies were conducted in the higher dose range of magnesium (20 to 40 mmol per day). An inverse dose-response was observed; each 10 mmol/day increase in magnesium dose was associated with reductions of 4.3 mm Hg and 2.3 mm Hg in systolic and diastolic blood pressure, respectively. In South Africa, a significant, inverse association between both serum and erythrocyte magnesium and blood pressure has been reported in urban black male labourers in Johannesburg.[41] To date, no supplementation trials of magnesium have been conducted locally.

In all ethnic groups in the present study magnesium intake was low (43.7 - 67.9 % had < 67 % DRI) and, on average, only about half of the amount of 500 mg/day which is achieved by the DASH diet.[2] No differences in magnesium intake were found between black and white subjects, however in black subjects, a greater proportion of dietary magnesium is provided from the bread and cereals group than in either of the other two ethnic groups. The most important individual food items in this regard, in the black sub-sample, are bread (24.5 % total Mg) and maize porridge and dishes (19.5 %).

Since compliance with advice to restrict dietary sodium in adults, over the long term, appears to be poor,[42] it has been proposed that the only effective way to lower salt intake on a population level is through the reduction of the sodium content of processed foods. The findings of the present study have identified that bread is the obvious food to be targeted in order to achieve a reduction in total sodium intake in the South African population.

Conclusions

The findings of this study demonstrate that the cereals food group is the largest contributor to non-discretionary Na intake, and bread is the single food item which provides the highest proportion of Na in the diets of South Africans, particularly in the black population. It is recommended that the food industry be lobbied to lower the sodium content of bread, while simultaneously increasing K, Mg and possibly Ca, and that the impact of this intervention on blood pressure be tested in randomized controlled trials.

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Chapter 5

Development and validation of a questionnaire to assess sodium intake

Introduction

Epidemiological studies demonstrate that the prevalence of hypertension and its associated cardiovascular consequences are directly related to the level of dietary salt intake in societies throughout the world in whom the intake is above a level of 50–100 mmol/day.[1] It has been estimated, in a meta-analysis of the contributions of behavioural factors to the prevalence of hypertension in Finland, Italy, the Netherlands, United Kingdom and USA, that a high sodium (Na) intake makes the second largest contribution to hypertension (after being overweight), with population attributable risk percentages (PAR%) of between 9 and 17 %.[4]

In order for advice to reduce salt intake to be targeted to those with excessive intakes, reliable estimations of habitual intake are required. Accurate assessments of salt intake are also necessary in epidemiological surveys and clinical trials in which diet-blood pressure associations are being investigated. The INTERSALT study demonstrated that in order to assess diet-blood pressure relationships, high-quality dietary information is required, together with controlling of multiple confounding variables, modern multivariate methods of data analyses, and correction of observed associations for within-person variation in intake.[2]

Measurement of dietary sodium, either on a population or individual level, is fraught with methodological difficulties. High intra- (45 %) and inter- (45 – 56 %) subject variability for reporting of non-discretionary sources (i.e. salt intake which excludes table salt and salt added in cooking) has implications for the reliability of food record estimates. It has been estimated that 81 days of dietary recording would be required to estimate an individual's intake within 10 % of the observed mean.[3] For this reason, the gold standard for assessment of salt intake is considered to be analysis of repeated 24-hour urinary Na estimations. However this method is not useful for large, community-based studies since it is time-consuming, inconvenient to the individual performing the collections, and under-collections of urine are commonplace. In addition, urinary Na estimations will not identify specific dietary sources of salt. A simple method to estimate population mean levels of 24-h urinary Na excretion from spot urine specimens collected at any time has been developed by Japanese investigators.[4] This method may be useful for comparing dietary Na intakes between different populations, as well as indicating annual trends of a particular population, but is not appropriate to estimate individual intakes.

There have been various attempts at developing short questionnaires for classifying persons according to their use of salt.[5,6,7] Other authors have shown that self-reported

abstinence from use of table salt is strongly correlated with actual behaviour[8] but this is only useful in identifying practices relating to discretionary salt use. The unique dietary features of a population group limits the applicability of an instrument developed in another ethnic group, in which food availability and food preferences may substantially differ. In developing countries, reliance on processed foods may be relatively less than in more developed countries, a factor which would further affect total salt intake estimations.

This aim of this study was to develop and validate a short, food frequency-type questionnaire to assess habitual dietary salt intake in South Africans and to enable classification of individuals into desirable and excessive categories of intake.

Methods

A systematic 8-step approach was undertaken to develop and validate a questionnaire-based instrument to assess habitual sodium intake. Approval for all components of the study was granted by the Research and Ethics Committee of the University of Cape Town, and written informed consent was obtained from all participating subjects.

Step 1: *Identification of food categories to be included in the salt intake questionnaire*

To ensure construct validity of a questionnaire to assess dietary salt intake in the South African population, reported dietary intake of a multi-ethnic sample was used as the basis for identification of food categories to be included in the questionnaire. Three repeated 24-hr dietary recalls were conducted in a sample of 325 men and women from three different South African ethnic groups (black, mixed ancestry and white), aged between 20 and 65 years, who were recruited from their place of work at the Cape Town City Council offices in central Cape Town. Since a salt intake questionnaire will most likely be used either in clinical practice to determine intake in people who are hypertensive, or for preventive health promotion purposes on a public health level, it was necessary to include both hypertensive and normotensive individuals. The study design aimed to recruit equal numbers of hypertensive (BP ≥140/90 mm Hg and/or on antihypertensive medication) and normotensive (BP <140/90mm Hg) men and women (n = 150 in each group; 50 from each ethnic group). A total of 180 hypertensive and 145 normotensive subjects were included in the final sample. Subjects were required to collect three 24-hour urinary volumes over a consecutive 3-week period, to correspond with the dietary recall reporting periods. As a marker of completeness of collection, 3

tablets (450 mg/day) of non-metabolizable para-aminobenzoic acid (PABA; Laboratories for Applied Biology, London) were given to the subjects, to be taken with meals during the collection period.[9] Urinary electrolyte concentration was measured using flame photometry and PABA measured calorimetrically.

All individual food items which were consumed by more than 5% of the sample and which contributed at least 50 mg Na per serving of that item (i.e. average portion of consumers) were included in the draft questionnaire. Food items were combined into 42 categories which included both food sources with inherent Na, such as milk, as well as those food items with a high added salt content, such as processed meat. The remaining items which fitted the inclusion criteria were combined into an "other" category.

Step 2: *Determination of portion sizes of foods included in food categories*

Average serving sizes of the various items included in the 42 food categories was determined using data obtained from the three repeated 24-hr recalls, as well as from data previously obtained from secondary analyses of other dietary surveys undertaken in adult South Africans.[10,11] The surveys included in the secondary analyses all utilized a 24-hour recall method, and included two studies of rural black subjects (Lebowa (1998; n = 292; age = 10 - 25 yrs)[12,13] and Dikgale (1992; n = 209; 19 + yr)[14,15]), a study of urban black residents in Cape Town (BRISK study: 1990; n = 1 243; 10 - 89 yr)[16,17] and a study of white subjects in the Western Cape (CORIS study:1989; n = 1 784; 15 - 99 yr).[18,19,20] In certain cases, common standard serving sizes of foods was assessed using the MRC Food Quantities Manual.[21]

Step 3: *Calculation of daily Na intake from questionnaire*

After perusal of many different food frequency questionnaire layouts, and due to the fact that some food items which are relatively low in sodium may be consumed very frequently (ie. more than once a day) and thus significantly contribute to overall Na intake, a possible range of 6 frequency responses were included in the questionnaire: Never; 1- 3 times per week; 4 - 6 times per week; once per day; twice per day; and 3 + times per day. Retrospective analyses were conducted on the repeated 24-hr recall data in order to assign one of these frequency factors to each of the 42 food categories for each subject. The average number of times ("times") a specific food is consumed daily per subject, calculated from data obtained in the three 24-hr recall periods was calculated as: Times = (times1 + times2 + times3)/3.

The factors used to determine intakes, according to the frequency categories used in the final questionnaire, was calculated from the intake data obtained by the three repeated 24hr recalls, as shown in Table 1 below:

Table 1
Average number of times that foods categories were consumed over three days, according to frequency categories used in the questionnaire

Category of frequency of consumption in questionnaire	No. average times consumed by subject in 3 repeated 24 hr recalls ("times")
Never	0
1 - 3 times per week	> 0 and <0.5
4 - 6 times per week	≥ 0.5 and < 0.9286
Once per day	≥ 0.9286 and < 1.5
Twice per day	≥ 1.5 and < 2.5
Three or more times per day	≥ 2.5

Absolute amounts of Na per serving size used for a single representative food in each of the 42 categories included in the final questionnaire were calculated from MRC Food Composition Tables.[22] This amount (in mg) was multiplied by the frequency factor that each individual reported, in order to arrive at a total daily Na intake for each subject.

Step 4: *Reliability of the questionnaire*

Alternative-form reliability: This is assessed by measuring the same objects by two instruments that are designed to be as nearly alike as possible. A low degree of response similarity may reflect either an unreliable instrument or non-equivalent forms. To further investigate construct validity with regard to the grouping of food items in the 42 food categories and the portion sizes used for the reference food items in each category, Spearman correlation coefficients were calculated between Na intake of individual food categories in the draft questionnaire, reported Na intake from repeated 24-hr recalls and 24-hr urinary Na excretion. The correlation between the overall Na content of the questionnaire (total of 42 food categories) was compared with total reported Na intake from the 24-hr recall data (using 43 food groupings, including the 'other' category).

Internal consistency/Internal-comparison reliability: This involves comparing the responses among the various items on a multiple-item index designed to measure a homogeneous concept. This is estimated by the inter-correlation among the scores of the items on a multiple-item index. All items on the index must be designed to measure the same thing. The Cronbach Alpha test (coefficient alpha) was also conducted for Na content of the various categories included in the questionnaire. This test produces the mean of all possible split-half coefficients resulting from different splittings of the

measurement instrument. Results can range from 0 to 1; a value of 0.6 or less is usually viewed as unsatisfactory. This indicates how the 42 food groupings perform in relationship to each other.

Step 5: *Ensuring criterion validity of the questionnaire*

Concurrent validity, a form of criterion validity, is the extent to which one measure of a variable can be used to estimate an individual's current score on a different measure of the same, or closely related variable.[23] To demonstrate concurrent validity, habitual urinary Na excretion was compared across tertiles of dietary Na intake, estimated using the questionnaire. Stanines (i.e. 9 categories) of Na intake, assessed from the questionnaire, were also calculated and mean daily urinary Na was compared across various combinations of stanines.

Step 6: *Determination of a scoring system*

The questionnaire uses the actual Na content value for each reference food item (according to its corresponding average serving size) in the 42 food categories. The Na content is multiplied by the frequency factor. The complexity of this scoring system would probably limit its widespread use by clinicians and academics, therefore a simpler scoring system, based on rounded integers for each food category, was devised.

Step 7: *Inter-rater reliability of the questionnaire*

A reference cut-off value which equated to greater or less than 6 g salt/day was assigned to the questionnaire score. Using the cut-off scores for the questionnaire, and comparing these categories with 24-hr urinary Na concentrations of either <= 2 400 mg Na/day and > 2 400 mg Na/day, a kappa statistic was calculated. Sensitivity and specificity of the questionnaire was determined, as well as positive and negative predictive values.

Step 8: *Validation of the questionnaire in another sample*

The objective of this step was to ensure reliability by means of internal consistency on an independent sample of the target group. The questionnaire was administered in a convenience sample of 80 black hypertensive South African subjects with mean age of 61.7 (7.9) years, ranging from 50 - 76 years. All subjects were taking anti-hypertensive medication and were attending primary care facilities in Langa (a peri-urban township of Cape Town) for treatment of their hypertension. Subjects completed the salt intake questionnaire on one occasion and completed three interviewer-administered 24-hr dietary recalls and collected three 24-hr urinary samples for urinary Na analyses, each

one week apart, according to the same methodology as described above for the original validation sample.

Results

Determination of food items and food groupings to be included in the questionnaire

The various food items which were included in each of the 42 categories, the reference food item for each category, together with the accompanying serving size and Na content, are shown in Table 2. Throughout the results, the Na content of the questionnaire = sum of absolute Na intake per day for reported frequency of intake of food items from each of the 42 food categories. In order to simplify the scoring system of the questionnaire for future use in clinical practice or in dietary surveys, absolute amounts of Na per serving for each food category were divided by 50 and rounded to the nearest integer.

In order to demonstrate that the food categories derived from the 24-hr recall data were valid for the purpose of estimating habitual Na consumption, internal consistency was assessed using Spearman correlations between the Na content of each of the food groups and total Na intake of the 24-hr recall data (Table 3).

It is important to note that actual reported serving sizes of food items within the groupings was used for the purpose of this analysis (not the single assigned serving size of one reference food per category, as in the questionnaire). Positive and significant correlations were found all of the food groups except the following: minimally processed breakfast cereal; crackers; roti/samoosa/springroll/doughnut; pizza; battered dipped chicken/ chicken patties; gravy; maas; yoghurt; tinned fish; canned vegetables/baked beans; chutney; savoury sauces and marmite/bovril. A possible reason for a low correlation may be that some of these groups, such as breakfast cereal, maas and yoghurt may be associated with a healthier diet. Thus, individuals who consume large quantities of these food items may consume less of the foods that are higher in Na (such as bread, cookies, pies etc). Alternatively, few subjects may be consuming these items, contributing to a weak correlation.

Table 2
Food categories, index food items, serving size and Na content of each category included in the questionnaire

Food category	Index food	Serving size	Serving (g)	Na content/ 100g	Na content/ serving	Na Score
BREAD AND GRAIN PRODUCTS						
1.White bread or rolls/croissants/pita bread/ bread crumbs	White bread	3 slices	75	490	367.5	7
2.Brown & wholewheat bread or rolls/health bread	Brown bread	3 slices	90	451	405.9	8
3.Breakfast Cereal: cornflakes/rice crispies/ all bran/hi bulk fibre bran/pro nutro/frosties/puffed corn/special K	Cornflakes	1 large bowl	40	1211	484.4	10
4.Minimally processed Breakfast cereal: weetbix, muesli, puffed wheat	Weetbix	2 weetbix	50	165	82.5	2
5.Provita/ crackers/rye bread and crispbread/matzos	Pro Vita	5 x 6g crackers	30	710	213	4
6.Cookies, rusks	Commercial plain	3 x 10g biscuits	30	410	123	2
7.Cake/scone/ muffin/ puddings(baked and instant)/ pancake / tarts/sweet breads and buns/semolina/ koeksister/	Muffin, plain	1 unit	70	130	91	2
8.Roti/ samoosa /springroll / doughnut/ savoury tart/ dumplings	Doughnut, plain	long, 130 mm	90	230	207	4
9.Pizza	Pizza	½ unit	170	570	969	19
10.Pasta /noodles dishes with cheese (lasagne/ macaroni cheese/noodle salad/spaghetti bolognaise)	Macaroni & Cheese, white sauce type	2 ladles	150	168	252	5
11.Popcorn	Popcorn, plain, salted	2 cups	40	1940	776	16
12.Potato crisps/ Niknaks/Chipkins	Potato Crisps	Small packet	30	1000	300	6
MEAT AND MEAT PRODUCTS						
13.Sausage – boerewors	Boerewors	Average thick piece	100	805	805	16
14. Processed, smoked, cooked and canned meat (Polony/salami/ham/canned corned meat/vienna/bacon/frankfurter/luncheon meat)	Polony	Homecut slice	60	1019	611.4	12
15.Meat or chicken pies, sausage rolls	Steak and Kidney pie	Commer-cial pie	140	460	644	13
16.Chicken burger patties and fried, battered chicken dipped in batter dip (KFC etc.)	Kentucky fried chicken	Thigh	100	292	292	6
17.Meat and meat dishes (minced beef, cottage pie, meatballs, stew, chicken stew)	Meatballs	Ladle	105	97	101.85	2
18.Gravy, made with stock or gravy powder	Brown gravy powder, reconstituted	Level ladle	35	4893	1712.55	34
19.Biltong(beef, game, fish), dry beef sausage	Biltong	Short piece	60	2213	1327.8	27
DAIRY PRODUCTS / EGGS						
20.Milk (all types, dairy fruit juice, malted milk, milk shakes, drinking chocolate, evaporated and condensed milk)	Full cream milk	½ cup	120	48	57.6	1
21.Maas/sour milk/ buttermilk	Maas	Small carton	500	71	355	7
22.Cheese, including processed cheese, feta, cottage	Cheddar Cheese	½ cup	40	487	194.8	4
23.Yoghurt	Low fat sweetened	Small carton	175	74	129.5	3

Food category	Index food	Serving size	Serving (g)	Na content/ 100g	Na content/ serving	Na Score
24.Eggs (any preparation - boiled, fried, scrambled, omelette)	Egg fried in sun oil	1 egg	50	120	60	1
FISH						
25.Tinned fish (pilchards, tuna, salmon, mackerel)	Tuna canned in water	½ cup	100	338	338	7
26. Other fish and seafood (shrimp, perlemoen, calamari, oyster, mussel, crab, fish cake, battered fish, fish fingers, fish paste)	Fish, medium fat, fried in sun oil	Medium piece	120	94	112.8	2
VEGETABLES/PULSES						
27.French fries and potato salad	French Fries	1.5 household serving	120	198	237.6	5
28.Baked beans, canned vegetables, tomato paste, olives (canned)	Beaked beans in tomato sauce	Heaped ladle	100	397	397	8
29.Soup (all types)	Average soup	Large mug	250	431	1077.5	22
VEGETABLE OILS						
30.Salad dressing/mayonnaise	Mayonnaise	Level dessert-spoon	15	755	113.25	2
31.Ice cream (sorbet or dairy)	Soft Serve (13% Fat)	Large serving	150	61	91.5	2
32.Margarines, all types, butter, Butro	Brick margarine	Heaped teaspoon	10	805	80.5	2
CONDIMENTS, SPREADS AND OTHER						
33.Chutney / atchar/ worcester sauce	Fruit chutney	Heaped Tbs	30	811	243.3	5
34.Savoury sauces (mushroom, pepper, cheese, white)	Mushroom sauce	2 level Tbs	40	575	230	5
35.Tomato sauce	Tomato sauce	Level Tbs	20	582	116.4	2
36.Salt	Iodised salt	Pinch	1	38850	388.5	8
37.Aromat / seasoning /mustard	Aromat	1 serving	1	24030	240.3	5
38.Peanuts (salted, unsalted, raw)	Salted Peanuts	Handful	30	433	129.9	3
39.Peanut butter	Peanut butter	Heaped teaspoon	12	478	57.36	1
40.Marmite / Bovril/Fray Bentos	Marmite	5ml teaspoon	5	4500	225	5
41.Chocolate sweets/chocolate sauce	Chocolate Bars	Average bar	40	165	66	1
42.Beer / cider	Commercial Beer	2 cans	680	10	68	1

Table 3
Association between average Na content per individual food category and average total Na content (all items consumed) of 3 x 24-hr recalls

Food category	Spearman Correlation coefficient (r)
1. White bread/rolls	0.341***
2. Brown bread/rolls,	0.142*
3. Breakfast Cereal (highly processed)	0.295***
4. Breakfast Cereal (weetbix, muesli)	-0.038
5. Crackers (ProVita etc)	0.082
6. Cookies, biscuits, rusks	0.160**
7. Cake/scone/ muffin/ puddings/pancake/fruit pie/koeksister	0.151*
8. Roti/ samoosa/springroll/ /doughnut	0.057
9. Pizza	0.092
10. Pasta/noodle dishes with cheese sauces (macaroni cheese, lasagne, noodle salad etc.)	0.113*
11. Popcorn	0.119*
12. Crisps (Simba and Niknaks etc.)	0.179**
13. Sausage (wors)	0.253***
14. Polony/salami/bacon/salami/pork suasages (processed meat, cooked, smoked and canned)	0.411***
15. Meat or chicken pies/sausage rolls	0.242***
16. Chicken - battered (KFC etc). and chicken burger only	0.074
17. Meat and meat dishes (steaks, minced meat, cottage pie, mince, meatballs, stew, bobotie, etc.)	0.123*
18. Gravy, made with stock or gravy powder	0.023
19. Biltong/dry wors/bokkems	0.130*
20. Milk (all types, also dairy fruit juice, malted milk, milk shakes)	0.226***
21. Maas	-0.030
22. Cheese	0.255***
23. Yoghurt	0.035
24. Eggs	0.203**
25. Tinned fish (pilchards/tuna, etc.)	0.101
26. Other fish and seafood	0.169**
27. Potato chips/french fries and potato salad	0.123*
28. Canned vegetables, incl. Baked beans, tomato paste, sweetcorn.	0.063
29. Soup (all types)	0.130*
30. Salad dressing/mayonnaise	0.233***
31. Ice cream (all types)	0.184**
32. Margarines, all types, also butter and Butro	0.468***
33. Chutney/ atchar/chakalaka / worcester sauce	0.086
34. Savoury sauces (mushroom, monkey gland, white,cheese)	0.059
35. Tomato sauce	0.106*
36. Salt - Not included in 24-hr data	
37. Aromat / Fondor /mustard	0.180**
38. Peanuts	0.174**
39. Peanut butter	0.152**
40. Marmite/Bovril	0.081
41. Chocolate sweets and sauce	0.199**
42. Beer and cider	0.109*
43. All other foods (not included in final questionnaire)	0.272***

***P<0.0001; **P<0.005; * P<0.05.

Reliability of the questionnaire

Alternative-form reliability

Table 4 shows Spearman correlation coefficients between Na intake of individual food categories in the questionnaire (using the determined serving size of the single reference food item per category as shown in Table 2), reported Na intake from repeated 24-hr recalls and mean daily urinary Na excretion. The very high correlation coefficients indicate a similar behaviour between the questionnaire and the actual 24-hr recalls. Only 8 of the food categories were significantly associated with urinary Na (cookies; popcorn; processed meats; meat and meat dishes, fish (not tinned fish), canned vegetables; Aromat; and peanuts).

Table 4
Spearman correlation coefficients between Na intake of individual food categories in questionnaire, reported Na intake from repeated 24-hr recalls and 24-hr urinary Na excretion

Individual food category	Questionnaire vs repeated 24-hr recalls (per food category)	Questionnaire vs urinary Na	Repeated 24-hr recalls vs urinary Na
1. White bread/rolls	0.915***	0.011	-0.013
2. Brown bread/rolls,	0.966***	0.020	0.025
3. Breakfast Cereal (processed)	0.983***	0.069	0.069
4. Breakfast Cereal (weetbix, muesli)	0.989***	0.036	0.032
5. Crackers (ProVita etc)	0.997***	0.037	0.042
6. Cookies, biscuits,rusks	0.988***	0.132*	0.129*
7. Cake/scone/ muffin/ puddings/pancake/fruit pie/koeksister	0.977***	0.014	0.024
8. Roti/ samoosa/springroll/ /doughnut	0.992***	-0.071	-0.071
9. Pizza	0.999***	0.080	0.080
10. Pasta/noodle dishes with cheese sauces (macaroni cheese, lasagne, noodle salad etc.)	0.999***	0.110	0.110
11. Popcorn	0.999***	0.128*	0.127*
12. Crisps (Simba and Niknaks etc.)	0.992***	-0.079	-0.075
13. Sausage (wors)	0.995***	-0.005	-0.006
14.Polony/salami/bacon/salami/pork suasages (processed meat, cooked, smoked and canned)	0.955***	0.122*	0.119*
15. Meat or chicken pies/sausage rolls	0.995***	-0.010	-0.008
16. Chicken - battered (KFC etc). and chicken burger only	0.999***	0.036	0.033
17. Meat and meat dishes (steaks, minced meat, cottage pie, mince, meatballs, stew, bobotie, etc.)	0.760***	0.121*	0.102
18. Gravy, made with stock or gravy powder	0.999***	0.088	0.087
19. Biltong/dry wors/bokkems	0.999***	0.112	0.113
20. Milk (all types, also dairy fruit juice, malted milk, milk shakes)	0.781***	-0.011	0.016
21. Maas	0.999***	0.022	0.025
22. Cheese	0.953***	0.077	0.047
23. Yoghurt	0.997***	0.043	0.044
24. Eggs	0.981***	0.003	0.004
25. Tinned fish (pilchards/tuna, etc.)	0.994***	0.071	0.076
26. Other fish and seafood	0.983***	0.118*	0.125*
27. Potato chips/french fries and potato salad	0.977***	-0.036	-0.026

Individual food category	Questionnaire vs repeated 24-hr recalls (per food category)	Questionnaire vs urinary Na	Repeated 24-hr recalls vs urinary Na
28. Canned vegetables, incl. Baked beans, tomato paste, sweetcorn, etc.	0.993***	0.120*	0.120*
29. Soup (all types)	0.996***	-0.040	-0.042
30. Salad dressing/mayonnaise	0.986***	0.056	0.050
31. Ice cream (all types)	0.998***	0.083	0.086
32. Margarines, all types, also butter and Butro	0.897***	-0.019	-0.007
33.Chutney/atchar/chakalaka/worcester sauce	0.999***	0.020	0.027
34. Savoury sauces (mushroom, monkey gland, white,cheese)	0.998***	0.035	0.035
35. Tomato sauce	0.999***	0.045	0.046
36. Salt	-	-	-
37. Aromat / Fondor /mustard	0.999***	-0.124*	-0.125*
38. Peanuts	0.999***	0.128*	0.131*
39. Peanut butter	0.995***	0.008	0.012
40. Marmite/Bovril	0.999***	0.067	0.068
41. Chocolate sweets and sauce	0.994***	0.030	0.028
42. Beer and cider	0.999***	0.095	0.096

***P<0.0001; **P<0.005; * P<0.05.

Internal consistency/internal comparison reliability

Spearman correlations were conducted between the Na content of each of the food categories and the overall Na content of the total questionnaire (Table 5). The Spearman correlation coefficient for the Na content of the total questionnaire (N = 42 categories) and the repeated 24-hr Na data was r = 0.683 (P <0.0001) (N = 328). Regarding urinary Na excretion, the association with total questionnaire Na was r = 0.173 (P = 0.0034) (n=284). The 24-hr recall data, which included the remaining reported food items in a very large "other" food group, did not perform better against the urinary Na data (r = 0.141 (P = 0.0174); N = 284). Spearman correlation coefficients for the questionnaire score (ie. Na content divided by 50 and rounded to nearest integer) vs repeated 24-hr recall Na data was r = 0.684 (P<0.0001) and for questionnaire score vs urinary Na was r = 0.171 (P = 0.0039).

The overall standardized Cronbach-α coefficient between the total questionnaire Na content and that calculated from the mean of 3 repeated 24-hr recalls was less than acceptable (ie. < 0.6) at 0.443. Cronbach-α coefficients for each of the individual food categories is shown in Table 5. Nine food categories had undesirable Cronbach-α coefficients which exceeded the overall coefficient of 0.443. Four of these nine categories were also not significantly correlated with total Na content of 3 x 24-hr recalls (see Table 3): fried, battered chicken/chicken patties; gravy; maas; and marmite/bovril.

Table 5
Internal consistency of questionnaire: Na content of individual food categories compared to repeated 24-hr dietary recall values

Individual food category	Spearman correlation with total Na intake of questionnaire†	Cronbach-α coefficient between food categories in questionnaire and 24-hr recalls
1. White bread/rolls	0.081	0.439
2. Brown bread/rolls,	-0.017	0.452*
3. Breakfast Cereal (processed)	0.012	0.448*
4. Breakfast Cereal (weetbix, muesli)	0.131	0.432
5. Crackers (ProVita etc)	0.064	0.441
6. Cookies, biscuits,rusks	0.045	0.444*
7. Cake/scone/muffin/puddings/pancake/fruit pie/koeksister	0.124	0.433
8. Roti/ samoosa/springroll/ /doughnut	0.184	0.424
9. Pizza	0.073	0.440
10. Pasta/noodle dishes with cheese sauces (macaroni cheese, lasagne, noodle salad etc.)	0.206	0.421
11. Popcorn	0.111	0.435
12. Crisps (Simba and Niknaks etc.)	0.069	0.440
13. Sausage (wors)	0.065	0.441
14. Polony/salami/bacon/salami/pork sausages (processed meat, cooked, smoked and canned)	0.115	0.434
15. Meat or chicken pies/sausage rolls	0.034	0.445*
16. Chicken - battered (KFC etc). and chicken burger only	0.004	0.449*
17. Meat and meat dishes (steaks, minced meat, cottage pie, mince, meatballs, stew, bobotie, etc.)	0.096	0.437
18. Gravy, made with stock or gravy powder	0.035	0.445*
19. Biltong/dry wors/bokkems	0.076	0.439
20. Milk (all types)	0.184	0.424
21. Maas	-0.187	0.474*
22. Cheese	0.163	0.427
23. Yoghurt	0.072	0.440
24. Eggs	0.071	0.440
25. Tinned fish (pilchards/tuna, etc.)	0.057	0.442
26. Other fish and seafood	0.229	0.418
27. Potato chips/french fries and potato salad	0.140	0.431
28. Canned vegetables	0.059	0.442
29. Soup (all types)	-0.064	0.458*
30. Salad dressing/mayonnaise	0.092	0.437
31. Ice cream (all types)	0.095	0.437
32. Margarines, all types, also butter and Butro	0.222	0.419
33. Chutney / atchar/chakalaka / worcester sauce	0.046	0.443
34. Savoury sauces (mushroom, white, cheese)	0.138	0.431
35. Tomato sauce	0.180	0.425
36. Salt	Not included	
37. Aromat / Fondor /mustard	0.150	0.429
38. Peanuts	0.082	0.439
39. Peanut butter	0.054	0.442
40. Marmite/Bovril	0.025	0.446*
41. Chocolate sweets and sauce	0.130	0.432
42. Beer and cider	0.053	0.443
43. Other foods (not included in final questionnaire)	0.325	0.404

† Excluding Na content of that food category.
*Cronbach Coefficient Alpha with deleted variable larger than Cronbach Coefficient Alpha of all variables (ie. > 0.443), using standardised variables (i.e. undesirable coefficients)

No difference was found between Na intake, assessed using the questionnaire and that reported in the three repeated 24-hr recalls, when using non-parametric measures (Sign test: P = 0.2040; Sign-rank test: P = 0.7425).

Factor analysis identified a number of diet pattern groupings, with regard to intake of the food items included in the questionnaire (Table 6).

Table 6
Factor analysis of dietary patterns identified by salt intake questionnaire responses

Factor	Diet pattern
Factor 1	White bread, margarine, beef sausage (boerewors), eggs, soups
Factor 2	Popcorn, aromat/fondor, roti etc., potatoes
Factor 3	Tomato sauce, pasta/noodle dishes, peanuts
Factor 4	Cheese, breakfast cereal (weetbix/muesli type), crackers, "other" food category, biltong.
Factor 5	Brown bread, milk, peanut butter, marmite/bovril
Factor 6	Yoghurt, savoury sauces, ice cream, other fish and seafood, potato crisps
Factor 7	Canned vegs, battered/fried chicken, chutney, processed meat, chocolate sweets, meat dishes, mayonnaise, maas
Factor 8	Pizza, cake/cookies, beer/cider, tinned fish
Factor 9	Breakfast cereals, meat pies
Factor 10	Cookies, gravy
Final communality est. = 18.09; 18.09/43 categories = 42 %	

Sodium intake, estimated from both the questionnaire (1221 (641); 1853 (589); and 1873 (663) mg/day) and the repeated 24-hr recalls (1459 (890); 1761 (884); and 1922 (911) mg/day) differed significantly (P<0.0001) between the black, mixed ancestry and white ethnic groups, respectively, included in the study.

Criterion validity of the questionnaire

Both Na intake from 24-hr recall data and urinary Na were assessed, according to tertiles of the Na content of the questionnaire (Table 7). Urinary Na was significantly higher for subjects in tertile 3, compared to those in tertile 1 (Bonferroni, P=0.0312; Kruskal-Wallis, P=0.0635). However, dietary Na intake (24-hr recall data) differed significantly across all three tertiles (Bonferroni, P<0.05; Kruskal Wallis p<0.0001). Questionnaire Na score also differed significantly between black, mixed ancestry and white subjects (24.2 (12.8); 36.6 (11.6); and 37.2 (13.3), respectively; P<0.0001).

Table 7
Mean reported daily Na intake and 24-hr urinary Na excretion, according to tertiles of Na content of questionnaire

	Tertile of Na content of questionnaire		
	Tertile 1 < 1 255 mg/day	**Tertile 2 1259-1931 mg/day**	**Tertile 3 > 1 935 mg/day**
N	108	108	112
Mean Na score (Mean (SD))	17.3 (6.1)	32.0 (4.2)	47.6 (7.6)
Range	0 – 25.7	25.0 – 39.3	38.0 – 76.7
Dietary Na intake (24-hr dietary recall) (mg/day)			
Mean (SD)	1 015 (548)	1 693 (739)	2 382 (836)*
Range (Min - Max)	54 – 4 007	782 – 5 409	1 084 - 6 114
Urinary Na excretion (mg/day)			
Mean (SD)	3 049 (1 182)	3 514 (1 659)	3 670 (2 039)**
Range (Min - Max)	1 004 – 6 745	297 – 9 090	1 097 -14 173
Mean urinary Na in salt (NaCl) equivalent: g/day	7.62	8.78	9.17**

*P<0.05: Difference between Tertiles 1, 2 and 3, using General Linear Models (Bonferroni test).
**P<0.05: Difference between Tertiles 1and 3, using General Linear Models (Bonferroni test); Kruskal Wallis p=0.0635).

Stanines (i.e. 9 categories) of Na intake, assessed from the questionnaire, were also calculated. Mean daily urinary Na was compared across the combination of stanines 1, 2 and 3 together (Group 1); 4, 5 and 6 together (Group 2); and 7, 8 and 9 together (Group 3). A significant difference between Groups 1 and 3 was found for urinary Na, using General Linear Modelling (F = 3.95; P=0.0203) (Table 8).

Table 8
Difference in urinary Na according to three categories created using combined categories of stanines (1+2+3; 4+5+6; 7+8+9) of dietary Na intake, assessed from questionnaire

Category (combined stanines)	Difference between mean urinary Na (mmol/day)	95 % Confidence Intervals of differences
3 vs 2	24.20	-2.47 to 50.85
3 vs 1	35.55	4.35 to 66.74*
2 vs 1	11.36	-14.12 to 36.83

* P< 0.05 for difference between categories 1 and 3 (Bonferroni test: F=3.95; P=0.0203; Wilcoxon test; P=0.1003).

Since the first group differed significantly from the third group, but no difference was found between the first and second groups, or the second and third groups, the questionnaire Na intake value corresponding to cut-off point of stanine 6 (upper limit) was identified to be 2 133 mg. (Stanine 7 lies between 2 133 and 2 478 mg Na). Since

added salt intake (discretionary) was not quantified in the 24-hr recall data (from which the questionnaire food categories were developed), it was decided to account for this by increasing the cut-off value of the questionnaire from 2 133 mg to 2 400 mg. This is the value that equates to the current international dietary guideline for the maximum recommended amount of salt intake per day (i.e. 6 grams NaCl per day).[24] This categorisation yielded a significant difference in urinary Na, equivalent to 145 (68) mmol/day (3 335 (1 564) mg/day) vs 177 (103) mmol/day (4 071 (2 369) mg/day) (P=0.0225) (Table 9).

Table 9
Mean (SD) urinary Na according to two categories of dietary Na intake, assessed from questionnaire

Grouping category	Mean (SD) urinary Na	95 % Confidence Intervals
Group 1 (< 2 400 mg/day)	144.9 (68.1) mmol/day	136.4 to 153.3 mmol/day
N = 252	3 333 (1 566) mg/day	3 137 to 3 526 mg/day
Group 2 (≥ 2 400 mg/day)	177.5 (102.9)* mmol/day	140.4 to 214.6 mmol/day
N = 32	4 082 (2 367)* mg/day	3 229 to 4 936 mg/day
Mean difference in urinary	-32.7 (72.7) mmol/day	-59.5 to -5.8 mmol/day
Na between Groups	752 (1 672) mg/day	-1 369 to -133 mg/day

* P=0.0225 for difference between categories (one-sided Wilcoxon approximation for t-test)
(P=0.0450 for two-sided Wilcoxon approximation t-test)

In keeping with the simplified scoring system (see earlier), the reference value of 2400mg Na per day (ie. 6 grams of NaCl/day) was divided by 50, yielding a value of 48 to indicate a cut-off score for desirable versus excessive Na intake. Both reported Na intake and urinary Na excretion differed significantly according to this classification (Table 10).

The urinary Na excretion in terms of salt (NaCl) equivalents, according to this cut-off value was 8.33 g and 10.21 g per day. This indicates that the two-category scoring system of the questionnaire is able to differentiate between excessive salt users and those with intakes closer to the desired reference of < 6 g/day, thus demonstrating construct validity.

Table 10
Daily Na intake and excretion, according to two categories of Na intake estimated by questionnaire, using cut-off scores[a]

	Group 1 Score < 48†	Group 2 Score ≥ 48
N (questionnaire)	288	40
N (urinary Na)	253	31
Dietary Na intake		
Questionnaire score (Mean (SD))	28.7 (11.1)	55.4 (6.3)***
Questionnaire dietary Na intake (mg/day) Mean (SD)	1 453 (556)	2 788 (317)***
24-hr recall dietary Na intake (mg/day) Mean (SD)	1 553 (808)	2 798 (862)***
Urinary Na excretion (mmol/day)		
Mean (SD)	144.9 (67.9)	178.4 (104.5)*
95 % CI	136.5 - 153.3	140.1 - 216.7
Salt (NaCl) equivalent: g/day	8.33	10.26

[a] Score = sum of absolute Na intake per day for each food category divided by 50, and rounded to nearest integer.
† Score < 48 equates to a daily Na intake of < 2 400 mg/day.
* P <0.05; ** P< 0.0001; Wilcoxon t-test for differences between score groups.

Inter-rater variability

A kappa statistic of 0.0318 was found between the questionnaire cut-off scores of (< 48 and ≥ 48) and 24-hr urinary Na concentration categories (< 100 mmol and ≥ 100 mmol) (N = 284).

Sensitivity and specificity of questionnaire

Sensitivity is the proportion of positive results that are correctly identified by the test (in this case, questionnaire). The questionnaire, using the cut-off score of ≥ 48 to indicate an excessive Na intake ("positive test result") has a sensitivity of 12.4 % (27/218) against 24-hr urinary Na values of ≥ 100 mmol/day ("disease") (Table 11). Specificity is the proportion of negative results that are correctly identified by the test (in this case, questionnaire). The questionnaire, using the cut-off score of < 48 ("negative test result") has a specificity of 93.9 % (62/66) against 24-hr urinary Na values of < 100 mmol/day ("not diseased").

Positive predictive value is 87.1 % (27/31), while negative predictive value is 24.5 % (62/253).

Table 11
Frequency table of questionnaire score and reference urinary Na cut-off values (Cape Town City Council sample; N = 284)

Questionnaire score	Urinary Na (mmol/day)		Total
	≥ 100	< 100	
≥ 48	27	4	31
<48	191	62	253
Total	218	66	284

Internal consistency of the final questionnaire in a new sample of 80 black hypertensives

Three complete repeated 24-hr urinary Na excretion values were available in 79 of the 80 subjects sampled, while data for the repeated dietary recalls and questionnaire were available in 80 subjects. Mean daily sodium intake, estimated using the questionnaire, is compared with intake estimated from 3 repeated 24-hr dietary recalls in Table 12. A positive Spearman correlation was found between the questionnaire Na score and (1) total Na intake calculated from the 24-hr dietary recall data ($r = 0.3618$; $P = 0.0010$) and (2) Na intake calculated from the 24-hr recall data for only the 42 food item categories included in the questionnaire ($r = 0.5606$; $P<0.0001$). No association between questionnaire Na score and 24-hr urinary Na was found ($r = -0.0004$; $P = 0.985$).

Table 12
Comparison of mean daily sodium intake, estimated from repeated 24-hr recalls and 24-hr urinary excretion, and salt questionnaire estimates (black hypertensives; N = 79)

	Mean (SD)	Range
Questionnaire (N = 80)		
Total Na intake (mg/day)	1489.6 (512.3)	448.8 – 3038.1
Na score	41.9 (17.0)	8.57 - 59.86
24-hr Dietary recall (N = 80)		
Total Na intake (mg/day)	1 798 (836)	426 - 4 093
Na score†	29.4 (10.1)	8.6 - 59.8
24-hr Urinary Na (N = 79)		
mmol/day	172.5 (52.7)	83.5 - 344.7
mg/day	3 968 (1212)	1 920 - 7 928

† Estimated using only the 42 food item categories that were included in the salt questionnaire.

Using the cut-off score of ≥ 48 to indicate an excessive Na intake ("positive test result"), the questionnaire had a positive predictive value of 80 % (4/5) and a negative predictive value of 6.8 % (5/74), compared to urinary Na reference cut-off values (Table 13).

Table 13
Frequency table of questionnaire score and reference urinary Na cut-off values (black hypertensives; N = 79)

Questionnaire score	Urinary Na (mmol/day)		Total
	≥ 100	< 100	
≥ 48	4	1	5
<48	69	5	74
Total	73	6	79

Regarding performance against the repeated 24-hr recalls, the overall standardized Cronbach-α coefficient between the total questionnaire Na content and that of the recall data was not acceptable (ie. < 0.6) at 0.35. The Kappa statistic between questionnaire score categories (<48 or ≥ 48) and 24hr recall categories of Na (<2 400 or ≥ 2400 mg/day) is 0.3333. Comparing the two methods, PPV was 100 % (n = 5/5) (i.e. all 5 subjects who scored ≥ 48 on the questionnaire also had Na intakes ≥ 2400 mg from the 24-hour recall data), while NPV was 80 % (n = 60/75).

Discussion

Accurate measurement of sodium intake is difficult due to extensive sodium distribution in foods and the widespread use of sodium compounds in food processing,[25,26,27] the extensive use of sodium chloride as table salt[28] and the presence of sodium compounds in drinking water, either as a natural phenomenon or due to some types of water conditioning.[29] In Europe and the United States, it has been shown that about three-quarters of Na intake comes from food processing, 10 – 11 % is naturally occurring (inherent) in foods, about 15 % is discretionary (half of which is contributed by table salt and half by added salt in cooking) while less than one percent is provided by water.[5,27,28,29,30]

We have developed a simplified food frequency-type questionnaire to assess habitual salt intake using representative dietary data from three ethnic groups of the South African population, and from secondary analyses of dietary datasets from other large surveys in the country. The final version of the questionnaire was further tested for internal consistency in a sample of older black hypertensive patients attending primary care facilities for blood pressure control. As well as being able to quantify sodium intake, as would be required for the purpose of epidemiological surveys and clinical trials, a rapid scoring system was developed to enable its use in public health-related activities. Black South African hypertensive patients mainly receive dietary advice from nurses at

primary care clinics but there is a lack of health promotion tools to assist clinic staff in empowering patients to consume a diet which is low in sodium and high in potassium.[31] Despite hypertensive patients having a good knowledge of the role of salt intake in the development of hypertension,[31] few are consuming diets comprising less than 6g salt per day.[32] The availability of an instrument that does not require detailed dietary records may be used as a motivational tool to quantify salt intake and to set targets for lifestyle changes within a clinic setting.

A significant, but poor, positive correlation was found between reported Na intake, estimated from either the questionnaire or the repeated 24-hr dietary recall data, and urinary Na excretion. The discrepancy between the questionnaire estimations of sodium and the urinary excretion values highlights the difficulty in quantifying discretionary (ie. added) salt intake in dietary surveys. In this study, the average of three repeated 24-hr recall dietary assessments were used as the basis for identifying food items and food categories which were significant contributors to overall salt intake in South Africans. The obvious under-reporting of discretionary salt intake using this method is problematic.

Low correlations between dietary reports and urinary estimations of Na excretion have been reported by other authors. In a cross-over study, participants were provided with either a 2 000 mg or 3 500 mg sodium diet for 7 days and sodium intake was estimated from seven 24-hr urinary Na collections per diet period.[5] The urinary Na analyses were significantly associated with duplicate chemical food analysis ($r = 0.61$), but not with sodium intake estimated from food composition tables ($r = 0.05$). Thus, even under strictly controlled conditions, whereby any food not provided by the research centre was obtained in duplicate and accounted for, where monitoring of intake and wastage took place daily, and where added salt intake was carefully measured, dietary analyses did not correlate with urinary Na excretion. These findings suggest that dietary assessment methods that rely on food composition tables are unable to accurately calculate the sodium content of foods, probably due to the large variation in the sodium content of processed foods.

In terms of reliability of the questionnaire, only 8 of the individual 42 food categories were significantly associated with urinary Na (cookies; popcorn; processed meats; meat and meat dishes, fish (not tinned fish), canned vegetables; Aromat; and peanuts). The questionnaire has been designed and validated as a composite measure and should be

used in its entirety. In assessing sodium intake, both the sodium density of various foods as well as the frequency of consumption of those foods in the population of interest needs to be ascertained. We included all individual food items that were consumed by more than 5% of the sample and which contributed at least 50 mg Na per serving of that item in the questionnaire. Thus, some foods, such as popcorn and salted peanuts, which are consumed by few individuals but which are very high in salt may have skewed the relationship.

Criterion validity of the questionnaire has been demonstrated however internal consistency is low. A possible reason why the Cronbach-α coefficient of the questionnaire is low could be related to the way in which the food choices of individuals in the sample are grouped together. For example, factor analysis identified that white bread consumption was associated with margarine, beef sausage (*boerewors*), eggs and soup intake, whereas consumers of brown bread were more likely to have peanut butter or marmite/bovril, together with milk.

The point of developing an instrument to measure habitual dietary Na intake is to use it to distinguish between individuals with high and low salt intakes. The current scoring system categorises individuals into either a desirable or excessive salt intake category and has been shown to have a very high specificity (94 %) but a poor sensitivity (12 %), compared to 24-hr urinary Na excretion. Only four of the 66 subjects (6.1 %) with urinary Na values which fell below the maximum recommended intake of 6 grams of salt per day (100 mmol urinary Na/day) had scores ≥ 48 on the new instrument.

It is necessary to determine what the probability is of the instrument giving the correct assessment. In this regard, it is useful to know the positive and negative predictive values of the instrument. Positive predictive value (PPV) is the proportion of subjects with "positive" results using the instrument (i.e. score ≥ 48) who are correctly determined. The PPV of the questionnaire indicates that, given a positive result (i.e. score ≥ 48), there is 87.1 % chance that an individual will have a urinary Na concentration above 100 mmol/day. Conversely, the negative predictive value (NPV) is the proportion of subjects with "negative" results using the instrument (i.e. score < 48) who are correctly determined. The NPV is low – given a score of < 48 on the instrument, there is 24.5 % chance that the urinary Na concentration will be less than 100 mmol/day. The instrument, using the current reference cut-off scores, is thus much more useful to determine high salt intakes rather than identifying people with habitually low/desirable salt intakes. The low negative predictive value demonstrates that classifying individuals

according to the proposed scoring system will result in a large number of people being falsely classified as having desirably low salt intakes when, in fact, their urinary Na concentrations indicate otherwise.

Agreement between categorical assessments is usually considered as a problem of comparing the ability of different observers or methods to classify subjects into one of several categories. The kappa statistic has a maximum value of 1.00 when agreement between two methods is perfect and a value of zero indicates no agreement better than chance. A kappa value of <0.20 is generally considered to reflect poor agreement while one of >0.60 and > 0.80 indicates good and very good agreement, respectively. The low kappa statistic between urinary Na reference values and questionnaire score categories indicates that the scoring system needs further refinement.

The two-category scoring system of the questionnaire is able to differentiate a significant difference in urinary Na excretion, thus demonstrating a degree of construct validity. However, the corresponding urinary values are much higher than the reference cut-off value of either greater or less than 6g salt per day. Data in Chapter 3 of this thesis show that estimated added salt intake is 4.08 g per day or 45.5 % of total Na intake. If this value is used as a proxy in our study, corresponding urinary Na excretion values related to only non-discretionary salt intake would be 4.34 and 5.92 g per day according to reference score categories of < 48 and ≥ 48 on the questionnaire (compared to the total 24-hr values of 8.33 and 10.30 g/day that were obtained). These values are much closer to the mean estimated Na content of the questionnaire that corresponds to these cut-off scores, namely 3.65 and 6.95 g/day.

We further attempted to account for discretionary salt intake by extrapolating responses obtained from a set of qualitative questions included in the same sample (data not shown). Subjects were asked about the use of salt and flavour enhancers (Aromat or Fondor) in food preparation (yes/no response); whether they usually add salt to their food before tasting it (yes/no response); and about their preference for a saltiness taste in foods (very salty/a little salty/not at all salty). If either salt or Aromat were used in food preparation, then an additional 389 mg Na (score = 8) or 240 mg (score = 5), respectively was added to the composite Na content of the questionnaire. If subjects also reported that they add salt before tasting food, then the salt and/or Aromat estimation was further multiplied by a factor of 2. If subjects liked their food to taste either "very salty" or "a little salty," these amounts were multiplied by a factor of 2 and 1.5, respectively. For example, a Na content of 778 mg (score = 16) was assigned to subjects

if they used salt in cooking and if they had a preference for a "very salty" taste. However, the addition of this data to the questionnaire score did not improve sensitivity of the questionnaire (data not shown), nor improve the kappa statistic. It is reported elsewhere in this thesis (Chapter 3) that such questions are not useful to determine between high and low salt consumers, and that their inclusion in epidemiological surveys, as a proxy for more detailed information on salt intake, is not warranted.

Internal consistency of the questionnaire was re-tested in a new sample of 80 older black people with hypertension in Cape Town, all of whom were receiving drug treatment for blood pressure control. The most commonly prescribed antihypertensive medication was thiazide-type diuretics which may have affected (increased) urinary Na excretion, irrespective of dietary intake. However, after reaching equilibrium, urinary sodium excretion should reflect the dietary intake of salt. Urinary Na is much higher, indeed more than double, that estimated either from the 24hr recall data or questionnaire assessment of Na. Using the same scoring system as in the original sample, in this group the PPV was 80 %, however few individuals (n = 5) scored ≥ 48 on the questionnaire. Of concern is the very low negative predictive value (6.8 %). It is important to note that the original sample in which the questionnaire was developed and the scoring categories devised included economically active subjects, both hypertensive and normotensive, from three ethnic South African groups. In Chapters 3 and 4 of this thesis it has been shown that the habitual reported dietary Na intake of black subjects (mean = 1 467 (891) mg/day; N = 110) is significantly lower than either their mixed ancestry or white counterparts. Our data suggests that the reference cut-off score will need to be lowered for use in this group. Thus, the current proposed scoring system requires further testing in larger, more representative samples of the black population in order to improve its ability to categorize between high and low salt consumers.

Limitations of the study need to be considered. The main benefits of the salt questionnaire are that it is simple, requires little participant time and effort, and is easy to score. The questionnaire reflects sodium intake over the past 7-day period which includes weekend days when sodium consumption patterns may differ. However, only a single nutrient is being measured. The current version of the questionnaire does not allow provision for the testing of hypotheses about other nutrients, such as potassium, calcium or magnesium, either singly or interactively with sodium, in the blood pressure-diet relationship. The more food a person consumes, the more likely they are to have a higher intake of sodium, unless the diet is a traditional with no access to processed foods. Another potential limitation is that the instrument did not account for total energy

intake nor did it consider sodium intake as a function of estimated energy requirements, as other methods have attempted to do.[5]

As with all food frequency questionnaires, the checklist of included food items may not necessarily be inclusive of all the important sources of sodium in another sample. Further, the serving sizes depicted in the questionnaire were based on a combination of average reported intakes in the present study and data previously obtained from secondary analyses of other dietary surveys undertaken in adult South Africans, including two rural populations.[10] Where possible, the serving sizes were adapted to represent convenient portion sizes. The instrument may require modification for sub-populations whose food habits differ substantially from the group of urban, economically active adults that were included in our study.

Consideration needs to be given to the validity of using three 24-hr urinary collections as the gold standard measure against which sodium intake using the questionnaire is assessed. Two decades ago, Luft and colleagues cautioned against the use of single or occasional 24-hour urine collections to identify biologic correlations due to the presence of considerable intra-individual variability.[33] Liu *et al.* suggested that 5 to 10 overnight collections are needed to characterize the urinary patterns of children.[34] Several factors may account for low correlations between the two measures. Intra-individual variability was high for both the measures against which the instrument was being tested, namely urinary Na (Coefficient of variation (CV) = 33.7 %) and 24-hr dietary recall Na estimates (CV = 44.4 %). The use of only three repeated measurements each of dietary recalls and urinary collections may not have been sufficient to accurately characterise the individual's usual sodium intake.

Conclusion

A short food frequency questionnaire to assess habitual Na intake has been developed using repeated 24-hour dietary from a multi-ethnic, economically active South African sample. The questionnaire demonstrates acceptable internal consistency and criterion validity against the gold standard indicator of repeated 24-hr urinary Na concentrations. It performs as well as three repeated 24-hr recalls against urinary sodium excretion and an acceptable correlation was demonstrated between the questionnaire and the repeated 24-hr recalls. However, the questionnaire considerably underestimates the dietary intake of Na in the studied population, presumably due to the large proportion of salt intake that is provided from salt added by individuals. The devised categorical scoring system needs to show improved sensitivity. Further validation studies of the instrument should be

undertaken in different geographical areas (i.e. urban and rural) where local communities are known to have different eating patters with regard to processed foods and salt use. The questionnaire may be used to monitor dietary compliance in research studies but in its current format cannot be used to estimate habitual dietary salt intake.

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Chapter 6

Development of foods with altered cation content: bread, margarine, stock cubes, soup mix and flavour enhancer (Aromat)

Introduction

The nutrition transition which has accompanied the rapid urbanisation in South Africa over the past few decades[1] has resulted in the displacement of more traditional diets in favour of dietary patterns which include more meat, fish, eggs, milk, cheese, bread, margarine, salt, oil, sugar and other sweets, often in the form of convenience foods.[2] Such changes in eating patterns typically lead to increased intakes of total fat, animal protein, sugar and salt, and decreased intakes of plant protein, dietary fibre and other complex carbohydrates.[3] Data on urinary Na excretion values reported elsewhere in this thesis (Chapter 3) indicates that the black population consume on average 7.8g of salt per day, which exceeds the JNC-7 guidelines for the management and prevention of hypertension of less than 6 g salt per day.[4] The urinary sodium:potassium ratio of the same group was 2.66 which indicates a substantially higher dietary intake of sodium compared to potassium.

Compliance with dietary advice to reduce salt intake is generally poor. Over the long term (13 to 60 months), disappointingly small reductions in blood pressure (average of 1.1/0.6 mm Hg) are associated with dietary Na restriction advice.[5] This information provides motivation for a population-wide reduction in salt in processed foods, in collaboration with the food industry. It has been demonstrated that non-personal health interventions, including government action to stimulate a reduction in the salt content of processed foods, are cost-effective ways to limit cardiovascular disease and could avert more than 21 million disability-adjusted life years per year worldwide.[6]

In order for diet-related public health attempts to reduce hypertension to be effective in a developing country such as South Africa, the recommended dietary pattern needs to involve relatively few behavioural changes, has to be affordable and sustainable long-term, and should be culturally acceptable. It is hypothesized that a reduction in the sodium content of various processed foods, together with a simultaneous increase in potassium (thereby reducing the sodium:potassium ratio) and other cations, would lead to an overall reduction in blood pressure levels in the South African population. In this regard, earlier work in this thesis has identified commonly consumed food items that are major contributors to overall non-discretionary sodium intake in the diets of the three ethnic South African groups.

As in many other countries,[7,15,8] bread is the single major contributor to dietary sodium intake in South Africa (see Chapter 4). Bread is a staple food commodity, eaten by the majority of the population in moderate to large quantities in South Africa. Brown bread

and white bread are the fourth and fifth most commonly consumed food items, respectively, by the adult population.[9] Fifty-five percent of South African adults consume brown bread (average serving size of consumers is 165 g/day), and 28 % consume white bread (average serving size is 163 g/day).[9] Bread is also an important staple food in the diets of South African children.[10] After maize, bread is the second highest contributor of total energy intake (14.8 % energy) and, prior to the mandatory fortification of maize and bread flour which was introduced in October 2003, was already an important source of micronutrients (15.6 % of iron; 16.9 % of zinc; 19.5 % of niacin; and 15.6% of thiamin intake) in children's diets.

In addition to bread, other food items which are important contributors to the sodium intake of the black South African population are brick margarine, and dry goods which are used to flavour dishes, such as soup mix, stock cubes, and Aromat (flavour enhancer, based on monosodium glutamate) (Chapter 4). The objectives of this study are (1) to develop reduced sodium (with increased potassium and magnesium content) varieties of these five food items and (2) to ensure that the technological aspects and the taste of the experimental products are acceptable. The novel food products will be used in a randomised controlled food-based trial of blood pressure reduction in the target population (Chapter 7).

Methods and Results

Methods and results are reported separately for (1) bread and (2) dry goods and maragrine. For bread, a two-stage approach was followed, namely "bread development" (Stage 1) and "bread evaluation" (Stage 2).

(1) Bread

Stage 1: Bread development

The third largest producer of bread in South Africa, Sasko Milling and Baking (Division of Pioneer Foods), partnered academics from the South African Medical Research Council, to develop a variation to their standard Sasko Sam brown bread (control), which contains 2% non-iodated sodium chloride (salt). The aim of the development was to reduce the sodium content by a minimum of 25 %, while simultaneously increasing the potassium, magnesium and calcium content. This level of Na reduction is in line with proposed new food labeling legislation in the country (which is currently being finalized before implementation), in which it is stated that a "reduced sodium" claim may appear on the

packaging of a product only if the sodium content of the product is at least 25 % lower than the regular variety.[11]

Based on information from the literature regarding flavour considerations, baking performance, physical properties and cost, the highest sodium reduction that could be achieved after trial and error, without compromising taste and functionality of the dough, was 36 %. Four different formulations for experimental breads with 36 % Na reduction (1.28% salt) were theoretically calculated (Table 1). One of the experimental breads had its salt content replaced with SOLO® Low Sodium sea salt, a salt replacement produced from Icelandic water, which provides 60 % less sodium than ordinary table salt (see Table 7 later). The remaining three bread variants included salt mixes that were developed in the Research and Development (R & D) laboratory of Sasko, in an attempt to provide a local, more cost-effective alternative mix of ion salts. In these breads, the 0.72% salt was replaced on a molecular basis with an equal quantity of other cations, including magnesium and potassium. The sources of magnesium and potassium, as well as the ratios of various mineral salts were evaluated by trial and error experimentation and the final compounds selected were based on expert discretion. A blend of magnesium chloride (6 hydrate), potassium chloride (dry) and magnesium sulphate (dry) was used to reach a target magnesium and potassium content in the final product (see Table 1). The experimental products also included additional calcium carbonate to increase the calcium content of the final product to a target calcium content of 200 mg/100g (Table 1). Other than the reduction of salt and increase of magnesium, potassium and calcium addition, the rest of the formulation was kept standard. Similar quantities of yeast and improvers were used in all formulations. The cereal base of Sasko brown bread consists of brown bread flour made up of approximately 87.5% white bread flour plus 12.5% bran.

Compared to the control brown loaf, all four experimental breads formulations were found to be acceptable in terms of taste and baking properties. This conclusion was reached after tasting by seven experienced bakers.

All four of the experimental breads were significantly more expensive than the control bread. Due to cost, the bread with the Solo salt replacement was eliminated as an option (deviance of 30.1 cents per loaf from standard brown bread). Out of the remaining breads, the one with the most favourable cation content, in terms of K and Mg, was variant 3. This experimental bread was taken to the next stage, where it was evaluated against the control bread.

Table 1
Theoretical determination of the cation content and cost of various bread formulations

Variation	Accept.	Nutritional information / 100g bread (39% moisture base)					Cost (cents)	
	Taste	Unit	Na	K	Mg	Ca	Per loaf	Deviance **
Standard Sasko Brown	Yes	mg	519.98339	213.25984	71.086614	132.2800		
(Control)		mmol	22.618034	5.4544531	2.9247733	3.3003992	92.294813	-
		% Increase*	-	-	-	-		
		% Reduction*	-	-	-	-		
Solo Brown 36%	Yes	mg	339.7574	390.2346	106.80421	52.8346		
		mmol	14.778634	9.9808584	4.3943308	1.3182297	122.39333	30.098
		% Increase*	-	82.985503	50.245179	-		
		% Reduction*	35.99	-	-	-		
R&D Brown 36% Var 1 [a]	Yes	mg	330.08809	423.76468	136.51619	210.7223		
		mmol	14.358042	10.838443	5.6167945	5.2575421	101.20448	8.910
		% Increase*	-	98.708147	92.042049	59.300188		
		% Reduction*	36.519493	-	-	-		
R&D Brown 36 % Var 2 [a]	Yes	mg	337.44771	320.85861	169.2997	214.2425		
		mmol	14.678168	8.2064594	6.9656328	5.3453727	108.47751	16.183
		% Increase*	-	50.454304	138.15975	61.961399		
		% Reduction*	35.104137	-	-	-		
R&D Brown 36 % Var 3 [a]	Yes	mg	330.15067	401.87301	150.16207	210.7522		
		mmol	14.360764	10.278529	6.1782379	5.2582889	101.18596	8.891
		% Increase*	-	88.442888	111.23818	59.322815		
		% Reduction*	36.507459	-	-	-		

** A deviance of 0.7 cents from the standard Sasko Brown (control) bread is considered to be of significant economical consequence.
[a] Var = Variant

Stage 2: Bread evaluation

After development, the experimental and control breads were compared, in terms of baking quality, sensory properties and nutritional profile.

- **Baking quality**

Methods

Sample Control and Experimental were baked in the Sasko R&D laboratory, according to the straight dough process, and compared for quality. Each batch consisted of 2.5kg flour, 2.5% compressed yeast (of flour mass) and standard quantity of improvers. One batch yielded four loaves. Six batches of each bread type were baked in random order per day, consecutively for 5 days, yielding a total of 30 batches (120 loaves) of each variation over the testing period.

Flour, yeast, mineral salts and improvers were all added in the mixing bowl and water added. The mixing time was defined as the time when the dough was optimally developed, as judged by a professional baker. Dough was left to rest 5 minutes,

thereafter it was divided into 4 x 780g dough portions. Each portion was moulded (formed), placed in a baking tin and proofed (fermented) for sixty minutes at 80% relative humidity and 38°C. After proofing, bread height was measured in each loaf and the mean height calculated for each batch (lower bread height indicates a reduced fermentation rate). Before baking, two of the four breads, per batch, were randomly selected and subjected to a drop test[12] to measure whether bread quality deteriorates with abuse. These breads were referred to as Control Abuse and Experimental Abuse while the remaining two breads were referred to as Control Normal and Experimental Normal. Variables which were measured and compared between experimental and control breads, for both normal and abused versions, included volume, crust colour, crumb colour and cell structure. The difference in loaf volume between the normal and abused loaves of the control and experimental variations provides an indication of how the bread will perform in an industrial plant where dough is moved on conveyer belts that knock pans against each other, leading to defects such as excessive increase or decrease in volume. All breads were baked for 26 minutes at 260°C. After baking, breads were left to cool and thereafter the volume measured by displacement of rape-seed, and a mean value of two measurements was calculated per loaf per treatment.

Finally bread was sliced and submitted for blind quality evaluation by a trained panel of five members, after which a consolidated decision on quality was made. Bread was scored on a scale from 1 to 10 for various quality characteristics, which included crust colour (the higher the score the lighter the bread), crumb colour (the higher the score the lighter the bread); and cell structure (the lower the ranking the finer the cell structure). Scores were converted to rankings for statistical analyses.

All statistical analyses were performed using the SAS statistical package.[13] Variables were tested for normality of distribution using the *Shapiro-Wilk W* test.[14] Non-normality may be due to kurtosis (measure of flatness or peakedness) or skewness (measure of asymmetry).[15] The normal distribution has a kurtosis value of three or less and a skewness value of zero. Kurtosis has no effect on the means and therefore allows standard statistical analysis.[11] Outliers were identified as values exceeding sample means by more than three standard deviations and were removed as a standard statistical procedure. General Linear Modelling (GLM) was used to construct analysis of variance (ANOVA) tables for the measured variables and interactions calculated where applicable. Thereafter, the least significant differences (LSD) were calculated according to the Student's t-LSD test[16] to compare treatment means. The level of statistical significance was taken as a P value of <0.05 for difference between the breads.

Results

All batches, irrespective of being control or experimental, had a mixing time of six minutes and required 49.49% water (of flour mass). The replacement of salt with a blend of mineral salts (experimental bread) had no effect on either the water absorption or on the time required to develop the dough. All measurements followed a normal distribution, except for volume difference between breads (P=0.0175) which was ascribed to kurtosis (Kurtosis = -1.0726), therefore ANOVA and LSD could still be applied.

Table 2
Comparison of baking properties between control and experimental breads: mean (SD)

Variable	Control		Experimental		LSD
	Normal	Abuse	Normal	Abuse	
Proof height (mm)	138.3 (7.6)[a]	N/A	137.6 (8.6)[a]	N/A	2.05
Volume (mm)	2913.5 (102.9)[b]	2828.5 (110.1)[d]	2966.0 (114.2)[a]	2874.8 (115.0)[c]	18.83
Volume difference (mm)† 95 % CI of difference	85.0 (60.4)[a] 62.3-107.5		91.2 (48.8)[a] 73.0 - 109.4		19.79

[a-d] Means with different superscripts in the same row differ at P≤0.05.
† (Normal - Abuse) for that bread type.

There was no difference in proof height between the control and experimental breads (Table 2). In a model which included bread type (control or experimental), treatment (normal or abused) and replicate (30 batches for each bread type), bread volume differed significantly according to type (P<0.0001) and treatment (P<0.0001), but there was no significant evidence of interaction between bread type and treatment (P = 0.7430). The experimental bread had a significantly higher loaf volume for both the normal and abused variations, compared to the control bread (Table 2). In both bread types, abuse (i.e. drop test) results in a significantly lower bread volume than in normal bread.

In GLM analyses, controlling for treatment and treatment*type interaction factors, both crust colour (P = 0.004) and crumb colour (P = 0.0387) of the experimental bread was lighter than the control bread. In similar models, cell structure did not differ between breads.

- **Sensory properties**

Methods

In order to test plant production feasibility, and for the purpose of sensory evaluation, bread was baked on one day in an industrial plant of Sasko located in Worcester. Six

consecutive batches of dough (three Control and three Experimental) were made from a single batch of flour. Each batch consisted of 130kg of brown bread flour, 3.2% (of flour mass) liquid yeast and a standard quantity of improvers. Each batch yielded approximately 420 loaves. After 24 hours, breads were subjected to sensory evaluation using triangle tests.[17,18] Triangle testing is designed to answer only one question: "Do the judges detect a difference between samples?" Three samples are presented simultaneously to each panellist - two of the samples are identical and the other is different ("odd"). Each panellist is asked to indicate which sample is the odd one or which two samples are most similar. The probability of making a correct selection by chance alone is thus 1 in 3 (33 %). Panellists were not asked to evaluate specific characteristics, but were told that they could consider any aspect related to bread texture, taste, colour, and aroma.

The study population was a consumer panel, made up of 122 volunteers (mean age = 36 (9.27) years), including factory workers from the Worcester bakery and personnel from the Worcester regional offices. Volunteers were recruited through posters and word-of-mouth, and tasting panels were performed during breaks in working hours. Sixty-eight percent of the volunteers were men (n = 83), whose ethnicity included black (n = 13), mixed ancestry (n = 51) and white (n = 19) subjects. The remaining 39 volunteers (32 %) were women, of which 12 were black, 14 were of mixed ancestry and 13 were white. The ethnic distribution of the volunteer sample reflects the ethnicity of the workforce employed by Sasko in the Western Cape.

Half of the volunteers tasted the Sample Control first and Sample Experimental second, while the other half tasted Sample Experimental first and Sample Control second, in a random fashion. Half of the volunteers were presented with Sample A being odd and the other half were presented with Sample B being odd, in a random fashion. The samples were presented in one of six random orders of presentation (AAB, ABA, BAA, BBA, BAB, ABB) and the adjusted chi-square test was performed.

Results

Of the 122 volunteers, 29 (23.8%) correctly identified the Sample Control, while 18 (14.7%) correctly identified the Sample Experimental (Table 3). In total, 47 of 122 sample sets (38.5%) were correctly identified.

Table 3
Results of triangle test for detection of control and experimental bread types

Triangle test (Sample A & B)			Demographic profile of total population					
Sample order	Total N	Correct N	Men			Women		
			White	Black	Mixed ancestry	White	Black	Mixed ancestry
AAB	21	11	4	2	8	2	4	1
BAA	21	9	4	4	6	4	1	2
ABA	20	9	5	1	8	3	2	1
Total (Odd B)	62	29	13	7	22	9	7	4
BBA	20	6	3	1	9	1	2	4
ABB	21	6	1	1	12	2	3	2
BAB	19	6	2	4	8	1	0	3
Total (Odd A)	60	18	6	6	29	4	5	9
TOTAL	122	47	19	13	51	13	12	14

The chi-square test yielded a calculated X^2 value of 1.4795 in comparison to the X^2-Distribution Table value of 2.706 required to prove significance at a probability level of 0.2239 (Table 4). Thus, overall, no significant deference between Control and Experimental breads (P>0.05), as judged by a consumer panel, were detected.

Table 4
Chi-square calculation for the pooled data and sub-data sets

Chi-square data set	Pooled data	Sample presented as odd	
		Odd B	Odd A
Observed (O1)	47.00	29.00	18.00
Observed (O2)	75.00	33.00	42.00
Expected (E1)	40.67	20.67	20.00
Expected (E2)	81.33	41.33	40.00
Calculated Chi-square	1.4795	5.0403	0.3000
P value	0.2239	0.0248	0.5839
Table value Chi-square	2.7060		

Respondents could however detect Sample B (experimental) when it was presented with two samples of Sample A (control) (see Table 4, Odd B, P = 0.0238). When Sample A was presented as being odd (see Table 4, Odd A), respondents could not detect a difference between samples (P = 0.5839).

- **Nutritional profile**

Methods

The experimental bread was developed for a food-based randomised controlled trial (RCT) of reduced Na foods (See Chapter 7). Throughout the trial, between March 2004 and July 2005, 24 samples of both the standard brown bread (Control) and the

experimental bread were analysed for Na, K, Mg and Ca content. Ten loaves were drawn at each of the 24 occasions and a composite prepared for analysis. At each occasion, samples of the pre-sliced bread, including crusts, were taken from all ten loaves, cross-sliced into smaller blocks and then hand-blended. Duplicate analyses were repeated in each of the 24 samples. Methods of analyses for Na, K, Mg and Ca content differed across the various laboratories used for the study. The CSIR used atomic absorption, according to the methods outlined by the South African National Scientific Programme of the CSIR (1981).[19] The Woolworths Laboratory used an ion chromatography method, as outlined by the *Metrohm Applications* Bulletin No. 257/1E.[20] Statistical tests for differences in cation content between the bread types were performed according to the methods described earlier. In the case of non-normally distributed data, outliers were identified and excluded from the analyses of differences between the means for experimental and control breads. Median values were also calculated, using all values.

Results

Sodium

After two sets of outliers were excluded, ANOVA indicated a significant difference for sodium content between breads (P<0.0001) (Table 5). Sodium content was reduced from a mean of 475 (75) mg/100g to 322 (62) mg/100g, which equates to a difference of 32.3 %.

Table 5
Laboratory analyses of sodium content of experimental and control breads

Laboratory*	Date	Sodium content (mg/100g)		
		Control	Experimental	% Reduction
Target (calculated)		**519.98**	**330.15**	**36.51**
CSIR	1-Mar-04	558.58	331.54	40.65
CVAC	4-May-04	366.95	192.05	47.66
CVAC	1-Oct-04	263.63	147.54	44.04
WW	1-Dec-04	422.78	373.74	11.60
WW	1-Dec-04	420.09	330.18	21.40
WW	1-Dec-04	561.14	387.72	30.91
WW	27-Jan-05	473.01	329.54	30.33
WW	3-Feb-05	*528.15†*	*702.90*	*-33.09*
WW	10-Feb-05	362.61	362.58	0.01
WW	17-Feb-05	544.56	306.04	43.80
WW	23-Feb-05	547.21	393.82	28.03
WW	3-Mar-05	439.41	362.87	17.42
WW	10-Mar-05	562.16	359.83	35.99
WW	16-Mar-05	463.25	353.62	23.66
WW	23-Mar-05	458.03	296.09	35.36
CSIR	26-May-05	558.91	286.48	48.74
CSIR	02-Jun-05	519.22	330.69	36.31
CSIR	09-Jun-05	492.79	285.34	42.10
CSIR	16-Jun-05	*488.43†*	*696.54*	*-42.61*
CSIR	23-Jun-05	499.06	390.91	21.67
CSIR	30-Jun-05	492.05	362.53	26.32
CSIR	07-Jul-05	482.07	264.17	45.20
CSIR	14-Jul-05	473.21	277.17	41.43
CSIR	18-Jul-05	496.92	357.22	28.11
Median		**490.24**	**342.58**	**30.62**
Mean (SD) (n=22)		**475.35** (75.34)[a]	**321.89** (62.50)[b]	**32.28**
Least Significant Difference (LSD)		**29.17**		

[a-b] Means with different superscripts in the same row differ at P ≤ 0.05.
* CVAC = CV Analytical Consultants (Pty) Ltd., P.O. Box 4936, Atlasville.
CSIR = Bio/Chemtek, Food, Biological and Chemical Technologies, 15 Lower Hope Road, Rosebank.
WW = Woolworths, Wooltru House, 93 Long Market Street, Cape Town.
† Outlier values (removed for calculation of mean, but not median)

Potassium

Bread potassium content was significantly higher (P<0.0001) in the experimental compared to the control bread (331 (84) mg/100g vs 213. (51) mg/100g; mean difference of 117.6 mg/100g). The mean increase in K content of 55.2% was considerably lower than the targeted increase of 88% (Table 6).

Table 6
Laboratory analyses of potassium content of control and experimental bread

Laboratory*	Date	Potassium content (mg/100g)		
		Control	Experimental	% Increase
Target (calculated)		**213.26**	**401.87**	**88.44**
CSIR	1-Mar-04	204.17	254.50	24.65
CVAC	4-May-04	131.48	240.29	82.76
CVAC	1-Oct-04	40.34	42.68	5.81
WW	1-Dec-04	216.35	473.57	118.90
WW	1-Dec-04	206.65	395.59	91.43
WW	1-Dec-04	255.87	443.02	73.14
WW	27-Jan-05	209.37	346.61	65.55
WW	3-Feb-05	260.98	254.94	-2.31
WW	10-Feb-05	344.16	349.50	1.55
WW	17-Feb-05	231.96	348.57	50.27
WW	23-Feb-05	226.87	368.81	62.56
WW	3-Mar-05	204.71	313.34	53.06
WW	10-Mar-05	231.42	320.92	38.68
WW	16-Mar-05	196.65	358.82	82.47
WW	23-Mar-05	200.69	340.11	69.47
CSIR	26-May-05	231.76	358.07	54.50
CSIR	02-Jun-05	200.27	330.60	65.08
CSIR	09-Jun-05	216.89	373.77	72.34
CSIR	16-Jun-05	215.54	217.46	0.89
CSIR	23-Jun-05	223.46	376.86	68.64
CSIR	30-Jun-05	211.02	353.77	67.65
CSIR	07-Jul-05	208.06	341.77	64.26
CSIR	14-Jul-05	210.01	361.70	72.23
CSIR	18-Jul-05	233.69	368.84	57.84
Median		**213.28**	**349.04**	**64.67**
Mean (SD) (n=24)		**213.02** (51.26)[a]	**330.59** (84.49)[b]	**55.20**
LSD		**27.61**		

[a-b] Means with different superscripts in the same row differ at P ≤ 0.05.
* CVAC = CV Analytical Consultants (Pty) Ltd., P.O. Box 4936, Atlasville.
CSIR = Bio/Chemtek, Food, Biological and Chemical Technologies, 15 Lower Hope Road, Rosebank.
WW = Woolworths, Wooltru House, 93 Long Market Street, Cape Town.

Magnesium

Magnesium content was significantly higher (P<0.0001) in the experimental compared to the control bread (53.6 (14.7) mg/100g vs 90.7 (29.3) mg/100g; mean difference of 37.0 mg/100g). The mean increase in magnesium content of 69 % was considerably lower than the targeted increase of 111 % (Table 7).

Table 7
Laboratory analyses of magnesium content of control and experimental bread

Laboratory*	Date	Magnesium content (mg/100g)		
		Control	Experimental	% Increase
Target (calculated)		**71.09**	**150.16**	**111.23**
CSIR	1-Mar-04	63.72	93.08	46.08
CVAC	4-May-04	54.11	122.06	125.55
CVAC	1-Oct-04	55.35	106.71	92.78
WW	1-Dec-04	32.76	98.27	199.95
WW	1-Dec-04	31.91	81.51	155.41
WW	1-Dec-04	45.71	68.36	49.55
WW	27-Jan-05	48.60	76.38	57.18
WW	3-Feb-05	58.73	51.61	-12.13
WW	10-Feb-05	61.35	68.90	12.31
WW	17-Feb-05	40.32	67.43	67.26
WW	23-Feb-05	42.84	63.56	48.35
WW	3-Mar-05	34.12	56.83	66.56
WW	10-Mar-05	33.28	43.46	30.59
WW	16-Mar-05	33.75	69.68	106.45
WW	23-Mar-05	37.07	55.03	48.45
CSIR	26-May-05	64.96	123.68	90.39
CSIR	02-Jun-05	61.54	117.26	90.54
CSIR	09-Jun-05	70.92	128.85	81.69
CSIR	16-Jun-05	66.32	66.70	0.58
CSIR	23-Jun-05	70.47	125.99	78.78
CSIR	30-Jun-05	67.19	115.39	71.74
CSIR	07-Jul-05	68.72	127.59	85.67
CSIR	14-Jul-05	67.64	123.35	82.35
CSIR	18-Jul-05	75.96	124.11	63.39
Median	-	**57.04**	**87.30**	**69.50**
Mean (SD) **(n=24)**	-	**53.65** (14.72)[a]	**90.66** (29.26)[b]	**69.02**
LSD	-	**9.22**		**-**

[a-b] Means with different superscripts in the same row differ at P ≤ 0.05.
* CVAC = CV Analytical Consultants (Pty) Ltd., P.O. Box 4936, Atlasville.
CSIR = Bio/Chemtek, Food, Biological and Chemical Technologies, 15 Lower Hope Road, Rosebank.
WW = Woolworths, Wooltru House, 93 Long Market Street, Cape Town.

Calcium

Five sets of outliers were removed from the analyses for calcium content. Calcium content was significantly higher (P<0.0001) in the experimental compared to the control bread (131 (29.6) mg/100g vs 176 (63) mg/100g; mean difference of 45.6 mg/100g). The mean increase in calcium content of 34.8 % was lower than the targeted increase of 59 % (Table 8).

Table 8
Laboratory analyses of calcium content of control and experimental bread

Laboratory	Date	Calcium content (mg/100g)		
		Control	Experimental	% Reduction
Target (calculated)		**132.28**	**210.75**	**59.32**
CSIR	1-Mar-04	58.68	76.94	31.12
CVAC	4-May-04	171.64	240.29	40.00
CVAC	1-Oct-04	107.89	142.90	32.45
WW	1-Dec-04	99.84	103.50	3.66
WW	1-Dec-04	95.74	119.41	24.73
WW	1-Dec-04	128.20	113.75	-11.27
WW	27-Jan-05	146.82	155.48	5.90
WW	3-Feb-05	167.82	198.20	18.10
WW	10-Feb-05	158.61	164.44	3.68
WW	17-Feb-05	*145.56*†	*116.19*	*-20.18*
WW	23-Feb-05	128.52	167.74	30.52
WW	3-Mar-05	125.10	126.69	1.27
WW	10-Mar-05	147.69	136.96	-7.27
WW	16-Mar-05	103.71	151.85	46.42
WW	23-Mar-05	100.08	142.02	41.91
CSIR	26-May-05	*150.47*†	*281.96*	*87.39*
CSIR	02-Jun-05	*134.71*†	*254.08*	*88.61*
CSIR	09-Jun-05	*156.67*†	*291.04*	*85.77*
CSIR	16-Jun-05	146.89	241.46	64.37
CSIR	23-Jun-05	155.46	273.09	75.67
CSIR	30-Jun-05	*144.67*†	*281.98*	*94.91*
CSIR	07-Jul-05	147.03	266.33	81.14
CSIR	14-Jul-05	148.66	270.47	81.94
CSIR	18-Jul-05	149.71	262.50	75.34
Median	-	**146.19**	**166.09**	**36.22**
Mean (n=19)	-	**130.95** [a]	**176.53** [b]	**34.81**
SD		*29.62*	*63.45*	
LSD	-	**22.41**		-

[a-b] Means with different superscripts in the same row differ at P ≤ 0.05.
* CVAC = CV Analytical Consultants (Pty) Ltd., P.O. Box 4936, Atlasville.
CSIR = Bio/Chemtek, Food, Biological and Chemical Technologies, 15 Lower Hope Road, Rosebank.
WW = Woolworths, Wooltru House, 93 Long Market Street, Cape Town.
† Outlier values (removed for calculation of mean, but not median)

(2) Dry goods and maragrine

Reduced sodium varieties of three dry goods (soup mix, stock cubes, flavour enhancer (Aromat)), were developed with the aim of maximum retention of a salty taste. Two types

of salt replacements were used in the development of the experimental food items, namely Solo® and Cerebos Low salt. Standard table salt (NaCl) contains 39.3 g sodium per 100g. One teaspoon (6g) of salt provides approximately 2 400 mg Na (6 g NaCl) per day. SOLO® Low Sodium sea salt is a UK-manufactured salt replacement, produced from Icelandic water, which provides 60 % less sodium than ordinary table salt. SOLO® comprises sodium chloride (41 %), potassium chloride (41 %), magnesium salts (17 %) and trace minerals (1 %). The following composition is provided (by the manufacturers) per 100g: Sodium = 16g; Potassium = 21.7g; Magnesium = 1g. Cerebos Low Salt, a locally manufactured product, comprises 48.5 % NaCl and 51.0 % KCl. Per 100g, it contains 19.4g Na, 27.03 g K, and only trace quantities of Ca (<0.05 %) and Mg (<0.10 %). The cost of SOLO is approximately R40 per kg, compared to approximately R14 per kg for Cerebos Low Sodium salt and 65c per kg for regular salt. The cation content of the two salt replacements are compared with regular salt in Table 9 below.

Table 9
Cation content of salt and salt replacements used in manufacture of products in the diet and blood pressure trial

	Cation content (mg/100g)			
	Na	**K**	**Mg**	**Ca**
Medium Food Grade salt[a]	>39 200	-	<0.01%	<0.01 %
Solo sea salt[b]	16 000	21 700	1 000	0
Cerebos Low Salt[c]	19 400	27 030	<0.10 %	<0.05 %

[a] Average Chemical analysis provided by Sun Salt Services, 7 Clarke Street South, Alberton (suppliers to Unilever Bestfoods Robertsons).
[b] Average Chemical analysis provided by Solo Low Sodium Sea Salt Co., 101-102 Palace Rd, Bromley, Kent, UK.
[c] Average Chemical analysis provided by Cerebos Limited.

A reduced salt version of both Knorr soup mix (Oxtail flavour), Knorrex stock cubes (Beef flavour), and Aromat was developed by Unilever Bestfoods Robertsons. SOLO® was used to replace regular NaCl in the soup mix (16.5 % salt) and Aromat (60.07 % salt) (Table 10). Both the soup mix and Aromat remained free-flowing. The taste perception was less salty and had a slight metallic aftertaste due to the mineral content of SOLO®.

Regarding the reduced Na Knorrex stock cubes (Beef flavour), during the development phase, the experimental cubes were first made up using Solo in place of regular salt. However, the cubes were too moist and this resulted in the density of the cubes being too "fluffy" and crumbly, compared to the more crystalline structure of the cubes made with regular salt. This altered texture would have adversely affected the forming and

packing characteristics of the cube mass. Thus, half of the salt level of regular cubes was replaced with Cerebos low sodium salt, resulting in a regular salt content of 28.3 % of product composition and a further 28.3 % comprising Cerebos Low Sodium salt (Table 10).

Table 10
Composition and proportion of salt used in regular and experimental dry food products (recipe formulation)

	% NaCl	% Cerebos Low Salt	% Solo
Aromat (regular)	60.07		
Aromat (experimental)			60.07
Knorr soup mix (oxtail) (regular)	16.5		
Knorr soup mix (oxtail) (experimental)			16.5
Knorrox Beef Cube (regular)	56.59		
Knorrox Beef Cube (experimental)	28.3	28.3	

Margarine

Brick (hard) margarine contributes 3 % of non-discretionary sodium intake in black subjects (data from baseline survey - Chapter 4). Fifty percent of the salt in normal brick *Rama* margarine was substituted with Solo salt replacement, providing an overall salt content of 28.9%. The method of introduction was to form a saturated brine with Solo and add this to the aqueous phase of the product. The structure of the product was not affected and no consumer trials were performed.

Both regular (control) and reduced Na varieties of the test foods were sent to independent testing laboratories for analyses of Na, K, Mg and Ca. Sodium was reduced by 51.1 % in Aromat; 68.8 % in the soup mix; 23.6 % in the stock cube; and 62.1 % in margarine (Table 11). Since the control versions of the foods did not contain K, increases of this cation in all four test foods exceeded 100 %.

Table 11
Laboratory analyses of cation content of control and experimental foods

Food item	Cation content (mg/100g)							
	Sodium		Potassium		Magnesium		Calcium	
	Theor-etical	Lab tests[1]	Theor-etical	Lab tests[2]	Theor-etical	Lab tests[3]	Theor-etical	Lab tests[3]
Aromat (control)	25 733	22 500	-	140	-	14	-	8.3
Aromat (experimental)	11 040	11 000	13035	10250	1 120	1 120	-	15
% Reduction/increase	**-57.1**	**-51.1**		**+7 221 %**		**+ 7 900 %**		**+80.7 %**
Knorr soup mix (oxtail) (control)	6 724	8 000	-	180	-	28	-	20.3
Knorr soup mix (oxtail) (experimental)	5 334	3 500	3581	2340	165	345	-	39.3
% Reduction/Increase	**-20.7**	**-68.8**		**+1200 %**		**+1 132 %**	**-**	**+93.6 %**
Knorrox Beef Cube (control)	24 940	22 250	-	80	-	19	-	20.3
Knorrox Beef Cube (experimental)	16 240	17 000	7650	7670	-	15	-	15.7
% Reduction/Increase	**-34.9**	**-23.6**		**+9 487 %**		**-21.1 %**		**-22.7 %**
Rama brick margarine (control)	805	Regular variety	not analysed by independent laboratory					
Rama brick margarine (experimental)	543	305	204	75.52	41			9.06
% Reduction/Increase (compared to recipe spec.)	**-32.5**	**-62.1**						

[1] Average of analyses from two laboratories: Chem Science cc (380 Umbilo Rd, Durban) and Echalaz & Osbourne (E & O) Laboratory (499 Sydney Rd, Congella, Durban); [2]Single analysis by E & O Laboratory; [3]Average of 3 analyses (single E & O Laboratory result and repeat tests on different days (23-03-2004 and 16-04-2004) by ChemScience); [4]Single analysis by CV Analytical Consultants (Pty) Ltd.

Discussion

We have developed a novel bread with a lowered sodium content, and an increased potassium, magnesium and calcium content. The bread has undergone extensive testing at the product development stage, with regard to baking properties and physical quality of the final product, required mixing times during dough production, sensory considerations, and ease of production in baking plants. Other products that were similarly modified in cation content include a brick margarine, an oxtail-flavoured soup mix, a beef stock cube, and a flavour enhancer (Aromat).

The laboratory-based nutritional analyses of the experimental bread demonstrated a Na reduction of 32 %, which is slightly lower than the recipe formulated amount of 36%. Food labeling legislation is currently in the process of being revised in South Africa. The draft guidelines allow a comparative claim (i.e. "*reduced sodium*") to appear on the packaging of a product only if that product is at least 25 % lower than the regular variety.[11] A "*low sodium*" nutritional claim cannot be made on bread, nor on any of the

other food items developed in the study, since a product must contain less than 120 mg sodium per 100g in order to qualify for this claim. This level of sodium content is not feasible in terms of sensory and physical properties of the food items of interest.

The Na content of our experimental bread (342mg/100g bread, independent laboratory analyses) is in line with targets set by the Food Standards Agency (FSA) in the United Kingdom (UK), which stipulate 350mg Na per 100g bread. In the UK, the FSA and the Department of Health are currently undertaking a programme of work with the food industry to reduce the level of salt in processed foods. A model has been developed, and revised in February 2005, to demonstrate the types of reductions that would need to be made in foods to ensure that the 6g salt/day population average target intake value was achieved by 2010.[21]

Sodium chloride is an important ingredient in bread due to its flavour enhancing properties. It also regulates fermentation, and strengthens the gluten (protein) component of the flour, thereby enhancing the malleability of the dough and making it more stable. Ions may theoretically enhance either protein association or dissociation. When sodium chloride is present, protein association is enhanced as water absorption is reduced, in addition to the strengthening of the dough.[22] The partial replacement of sodium chloride with other electrolytes instead of the mere reduction of sodium chloride in the dough recipe offers the opportunity to maintain the electrolyte concentration required for optimal baking outcomes, as well as being of potential benefit to health. A study in which either 20 %, 40 % or 100 % of the sodium chloride (2 % flour weight basis) was replaced with either an equivalent amount of potassium chloride, magnesium chloride, calcium chloride, magnesium acetate, magnesium sulphate or sodium sulphate was performed in Finland.[23] At replacement levels of 20 and 40 %, magnesium chloride and calcium chloride weakened the physical properties of the dough, whereas magnesium sulphate and sodium sulphate strengthened them. Replacement with potassium chloride or magnesium acetate had no significant effect on dough rheology. In the baking tests, provided that sufficient mixing times were used, no difficulties in baking performance were demonstrated at the level of 40 % replacement with potassium chloride, calcium chloride or magnesium salts. However, the flavour of these breads was poor. Other authors have also demonstrated that bread baked with potassium chloride instead of sodium chloride is unpalatable.[24]

In the present study, baking performance of the experimental bread was similar to that of Sasko standard brown bread. No difference was found for either bread proof height or

cell structure between the experimental and control breads. Bread volume differed significantly between the control and experimental loaves, however the magnitude of the difference is of no practical importance. Internal quality control measures at Sasko Milling and Baking stipulate that a volume difference of 125 mm and above is considered unacceptable. Baking performance of the experimental bread remained similar to that of control bread after dough had been subjected to a standard drop test and neither bread types exceeded the critical volume difference of 125 ml. This demonstrates that the experimental bread would perform adequately in an industrial plant setting and, indeed, was successfully mass-produced, for use in a randomised controlled trial between March 2004 and July 2005 (See Chapter 7).

The lighter crumb and crust colour in the experimental bread may be related to the presence of insoluble calcium carbonate in the experimental bread which would have been present in the dough as a white particle. The lower chloride content of the experimental bread, due to the partial replacement of sodium chloride with a combination of magnesium chloride hydrate, potassium chloride and magnesium sulphate, may have been expected to result in a darker, not lighter, bread.[12] It is important to note that, despite colour differences being detected by a highly trained panel of baking experts, no sensory differences were identified between the experimental and control breads when subjected to a large consumer panel of 122 individuals. Sensory testing was performed on breads 24 hours after having being baked according to standard protocol in an industrial bakery, thus providing further evidence of the viability of the experimental bread production on a large scale.

Food manufacturers are often resistant to reducing the sodium content of processed foods such as bread since the sodium content is largely perceived to be essential to the flavour and acceptability of the product.[25,26] Sudden, large reductions in salt content may make foods less palatable and unacceptable to consumers,[27] however small to moderate changes may be less noticeable.[28] Sustained reductions in sodium consumption may lead to a preference for a lower salt diet.[29] There is some evidence that long-term adherence to a diet low in sodium can lead to a shift in taste perception whereby both normotensive[30] and hypertensive[31] persons develop an increased acceptance of foods with a reduced sodium content. Presumably, the salt taste receptors become more sensitive, and a lower sodium concentration provides the same salty taste as previously.[11,12]

An Australian study has demonstrated that it is possible to deliver a one-quarter reduction in the sodium content of bread without detection by consumers if the reduction is gradual.[32] In that study, over six consecutive weeks, the sodium content of bread was reduced from 2g salt per 100g flour to 1.5g salt per 100g flour, with cumulating 5% reductions each week. The intervention group were no more likely than the control group to report a difference in the salt content of the bread from week to week. Similarly, scores for flavour or liking of the bread over the study follow-up period did not differ between groups. However, saltiness scores recorded on a visual analogue scale did significantly decline in the intervention group compared with the control group. Another study has demonstrated that the sodium content of bread can be lowered by about one third, with no significantly detectable disadvantage in bread taste, while the content of the nutritionally favourable potassium and magnesium can be increased by two-to three-fold, a level representing that of dark European-type breads.[33] The best results were found with the use of potassium chloride and magnesium chloride salts.

There are some limitations in the bread development process that need to be considered. The representivity of the consumer tasting panel warrants discussion. The panel comprised less than a quarter of individuals from the target black population. Acceptability of bread tastes may differ from population to population and may be dependent on the types of breads usually consumed, the total salt content of the diet and the manner in which the bread is usually consumed (e.g. with margarine, as a snack or as a staple food). Brown bread was chosen as the type to be modified since this is the most commonly consumed bread by the majority of the South African population.[9]

In South Africa, the level of added sodium chloride to bread has remained high for many decades (on average 2 % of flour weight [also referred to as bakers percentage] or 520 mg Na/100g bread), despite an international recommendation made in 1977 to reduce the sodium chloride content of bread.[34] The consumption of an average 150 g of bread per day[9] contributes 750 mg Na (1.9 g salt), which is almost a third of the maximum recommended intake of 6 g salt per day. It may therefore be assumed that consumers are used to the taste of bread with a high salt content. However, no taste differences were detected between the experimental bread compared to the standard variety in the consumer panel in the present study. Moreover, in a subsequent randomised controlled trial in which the reduced sodium bread was used, 77.5 % of subjects reported that they preferred the bread to their usual choice (Chapter 7).

Assessment of the feasibility of launching such a staple product to the mass market on a widespread level requires consideration of factors other than flavour considerations. These include the cost of the ion replacements, acceptability of the formulation by local bakers using existing production lines, as well as the perceived need for the product and the perceived health benefits thereof by the target population. The latter investigation would require social marketing research, using in-depth techniques.

Regarding cost considerations, the ingredient cost of a standard brown bread containing 2% salt is R0.92294. The additional cost of the reduced sodium bread developed in this study was calculated to be R0.0891 per loaf, which exceeds the deviation of R0.007 per loaf that is considered by the baking industry to be acceptable in terms of expected profit margins. If 25 % of salt were to be removed from the standard bread formulation without replacement with other ions, as occurred in the Australian study,[33] the cost increase per loaf would be negligible, at R0.00169 per loaf (ingredient cost of R0.92464), assuming a salt price of R0.47/kg. The retail-selling price of this type of reduced sodium bread would be R4.6232, compared R4.6142 for a standard loaf (cost calculations provided by Elizabeth MacGregor, Food Technologist at Sasko Milling and Baking, Pioneer Foods, Paarl). The cost of the experimental bread developed here may not be acceptable to the target population and investigations of other, more affordable types of reduced Na bread may be required in the South African context.

A further limitation of the study relates to the methodological variability of the laboratory nutrient analyses. Substantial variation was found in the cation analyses of both the experimental and control versions of the study foods, both between and within the different testing laboratories used. Notably, values for calcium content increased substantially after 23 May 2005, when analyses were performed by the CSIR laboratory. Considering that the minerals were added as a cocktail in one premix, it is unlikely that the higher calcium measurements observed after this date were due to processing or dosing errors. If the latter was the cause for the increased calcium measurements, the other mineral measurements should have been also expected to increase proportionally (this was not the case).

Despite stringent quality control processes being exercised during the production of the food items, laboratory values consistently differed from the recipe formulations. This poses the question of which values to use for the determination of the nutritional content of the food items. We have erred on the side of caution and taken the laboratory values that indicate a lower Na reduction and a lower increase in K, Ca and Mg in the

experimental food products than was calculated from theoretical formulations. In terms of marketing and food labelling of such products, the legislating body of the Directorate of Food Control within the Department of Health in the country will require evidence of chemical analyses in order for a claim to be made on the packaging. Our data demonstrates that standardisation of analytical methods used by accredited testing laboratories would be required prior to commercialisation of reduced sodium bread that carries a nutritional content claim.

As well as bread, we have also successfully developed reduced Na (and increased K, Mg, Ca) versions of a brick margarine, stock cube, soup mix, and flavour enhancer (Aromat). Brick margarine is the sixth most commonly consumed food in the diet of South Africans, consumed by 21 % of the total population, in average amounts of 19g per day.[15] In many countries, including South Africa, specialist brands of margarine have been marketed as a functional food for some time already. The plant sterol-enriched margarine (*Pro-Activ*, Unilever Foods, South Africa) is such an example, however this product retails at a vastly increased cost over its standard *Flora Light* low fat spread relative and is targeted specifically towards people with hypercholesterolaemia. Uptake by the majority, black population has been marginal (personal communication, Christine Broadhurst, Consumer Affairs Manager, Unilever South Africa Foods).

Aromat (monosodium glutamate-based product), together with stock cubes and soup mixes, is used as a flavour enhancer in many commonly consumed foods, particularly in maize-based dishes to provide a salty taste. In a study to determine nutrition knowledge and dietary practices of hypertensive adults attending blood pressure clinics at primary care facilities in the Cape Town metropole, a large percentage (34.1%) of participants believed that flavour enhancers such as Aromat could safely be used instead of table salt.[35] Thus, this is an appropriate food item to target for a modified cation content.

The UK Food and Drink Federation, under their Project Neptune initiative, achieved an overall reduction in the sodium content of sauces and soups of 20% and 6%, respectively, in 2003. Project Neptune members aimed to achieve an overall 30% reduction in soups and meal sauces over 3 years between 2003 and 2005. In the present study, through the substitution of regular salt for Solo™ (a salt replacement mineral mix), we were able to achieve a 69 % and 51 % Na reduction in soup mix and Aromat composition, respectively, without any adverse effects on the production or quality of the items. The 24 % reduction in Na content of the stock cube was achieved through replacement of half the regular salt content with Cerebos Low Salt™ (salt

replacement containing KCl) since the crystalline structure of the cube was altered with the use of Solo alone. A limitation of the study is that, unlike bread, the experimental dry goods and margarine did not undergo extensive consumer testing, which would be necessary prior to the commercialization of such products.

In other countries, the food industry has made good progress in lowering the salt content of many processed foods. This has been achieved, to a large extent, due to active lobbying from consumer groups and government agencies. In the UK, a Consensus Action on Salt and Health (CASH) group has been set up, headed by Professor Graham MacGregor of St George's Hospital Medical School in London.[36] Since 2005, CASH have co-ordinated an annual national Salt Awareness Day which is accompanied by extensive media coverage.

In September 2004. the UK Food Standards Agency launched a "Sid the Slug" salt campaign. A consumer survey conducted by the Agency showed an upsurge in consumers making an effort to cut down on how much salt they eat and to change their shopping behaviour. Between August 2004 and January 2005, there was a 32% increase in people claiming to be making a special effort to cut down on their salt intake; a 31% increase in those who look at labelling to find out salt content; and a 27% increase in those who say that salt content would affect their decision to buy a product "all the time".[37] This provides encouraging evidence of a rapidly achieved change in consumer awareness related to salt intake behaviour associated with a campaign that involved effective partnerships between members of the food industry, academics, consumer bodies, retailers, trade associations, professional and non-governmental organizations and the Department of Health. Further, the change appears to be sustained - between September 2004 and September 2005, an increase of nearly 6 million people or 12% of adults claimed to be trying to cut down the amount of salt they eat (total of 46% adult population).[38]

Until now, the availability of reduced sodium products in South Africa has been limited. This is probably due, in part, to the lack of consumer lobbying and consumer demand for such products in the country.

Conclusion

The sodium content of food items frequently consumed by the South African population can successfully be reduced by between 24 % and 69 %, and partially replaced with other cations such as K, Mg and Ca without adversely affecting palatability or product

quality. The blood pressure-lowering impact of substitution of these food items for the regular higher salt varieties needs to be tested in black South Africans in order to contribute to other non-pharmacological approaches to the management and prevention of hypertension. Thereafter, the challenge will be for the food industry to make such items cost competitive to the target population, and, in collaboration with the Department of Health, to implement effective social marketing techniques to increase awareness of the health benefits of switching to these products.

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Chapter 7

The impact of dietary manipulation of sodium, potassium, calcium and magnesium on blood pressure in hypertensive black South Africans: a randomised controlled trial

Introduction

Earlier studies included in this thesis have identified that ethnic differences exist in the habitual intake of dietary factors involved in blood pressure regulation. Mean urinary Na values equate to a daily salt (NaCl) intake of 7.8g, 8.5g and 9.5g in black, mixed ancestry and white subjects, respectively. In all three ethnic groups, sodium intake exceeded the recommended maximum of 6 g/day, while potassium intake fell far below the recommended minimum of 90 mmol/day. For calcium, both dietary and urinary parameters were lowest in black subjects. These data, together with findings from published dietary surveys conducted in black South Africans since 1975,[1] indicate a need for population-based approaches to change dietary behaviour in order to both prevent the development of hypertension and to improve blood pressure control in hypertensives.

Since compliance with advice to restrict dietary sodium in adults, over the long term, is generally poor,[2] it has been proposed that the only effective way to lower salt intake on a population level is through the reduction of the sodium content of processed foods.[3,4] It has been demonstrated elsewhere in this thesis (Chapter 4) that the largest contributor to non-discretionary Na intake in the diets of South Africans is the bread and cereals food group, contributing between 49 - 54 % in the urban black population and 70 - 75 % in the rural black population. Bread is the single food item providing the highest proportion of non-discretionary Na, ranging between 41 - 52 % and 66 - 73 % in urban and rural dwellers, respectively. Other important food sources of Na in this population include soup powders (fifth highest ranking) and margarine (6^{th}). Added salt intake was estimated to comprise 46 % of total sodium intake, which equates to a daily added salt (NaCl) amount of 4.1 g. This is a much higher proportion than that reported in affluent western countries, in which 75 – 85 % of salt is estimated to come from processed foods.[5,6,7]

We hypothesize that a moderate reduction in sodium intake, in the presence of an increased intake of potassium, magnesium and calcium, will reduce blood pressure levels in mild-to-moderate hypertensives by a clinically significant magnitude.

As a result of a partnership with members of the food industry, novel varieties of the identified important food sources of salt were produced (see Chapter 6), which had a reduced sodium, and increased potassium, calcium and magnesium content. A dietary intervention was designed, based on the cation manipulation of 6 commonly consumed

foods, namely salt, bread, margarine, stock cubes, soup mixes and Aromat (a popular flavour enhancer comprising monosodium glutamate) and the addition of a high calcium fermented milk product (maas). The impact of the intervention on blood pressure was tested in an 8-week randomised controlled trial in black South African hypertensives.

Methods

A randomized, parallel group controlled trial was undertaken to investigate the impact of an 8-week feeding study (in which sodium intake is decreased, and potassium, magnesium and calcium intake is increased) on blood pressure in mild–to-moderate hypertensive black South Africans who have an habitually inappropriate intake of these cations. Eligible subjects completed a 3-week run-in period, during which time baseline screening measurements were obtained. Thereafter, subjects were randomized to either the intervention or control diet for 8 weeks. The study was conducted in 2 consecutive Phases. The study design is shown graphically in Figure 1.

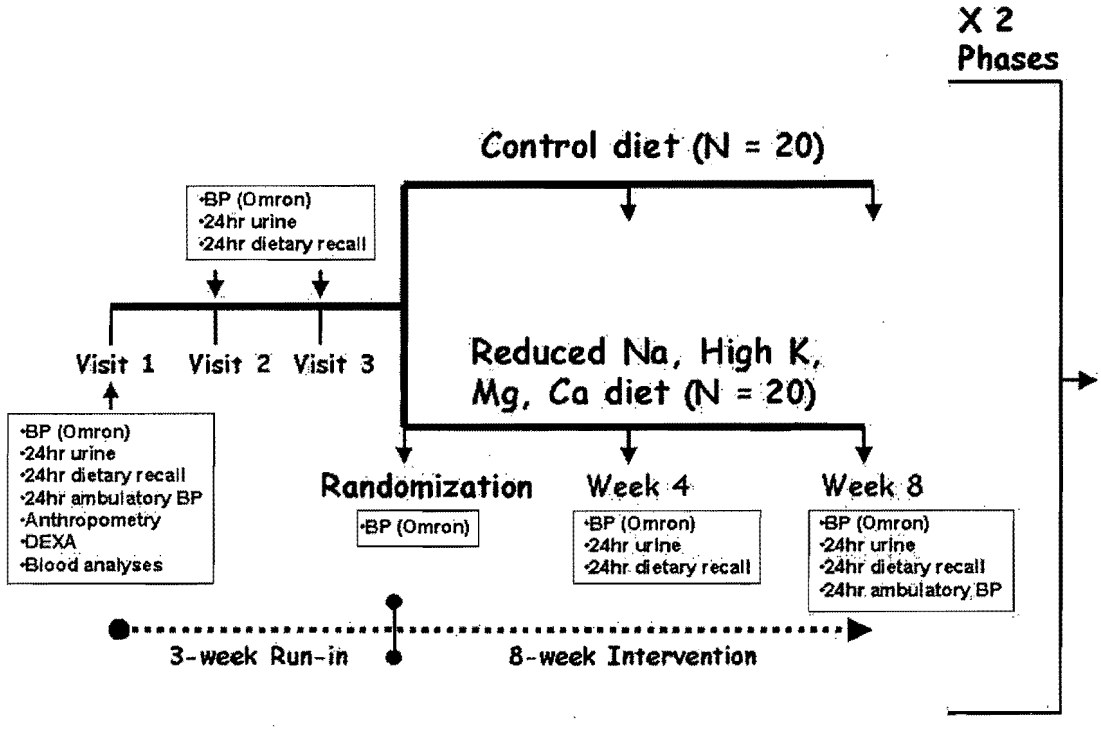


Figure 1
Scheme of study design

Subjects and sampling

Inclusion and exclusion criteria

Black male and female residents of Langa township on the outskirts of Cape Town, aged between 50 and 75 years, with drug-treated mild-to-moderate hypertension (BP: Systolic ≤ 160 mmHg and Diastolic ≤ 95 mm Hg) were eligible for participation in the study. Exclusion criteria included taking two or more diuretics for treatment of hypertension; prescription of furosemide (Lasix) (presumed to be prescribed for cardiac failure); previous cerebral infarction or hemorrhage; renal impairment (serum creatinine > 114.4 umol/l (1.3 mg/dl) and creatinine clearance < 50 ml/min) values; habitual consumption of > 3 alcoholic drinks per day; Type 1 diabetes mellitus; impaired cognitive function; and a Body Mass Index (BMI) > 45. Creatinine clearance (ml/min) was estimated using the Cockroft- Gault equation: {(140 - age) x weight (kg) x [1 - (0.15 x sex)]}/0.814 x serum creatinine (umol/l), where sex = 0 for male and 1 for female.[8] Cognitive function was assessed using a modified version of the six-item Cognitive Impairment test (6CIT).[9] The first three items assess orientation in time, while the latter three items test memory and concentration. The 6CIT excludes items about current affairs, in order to facilitate both linguistic and cultural translation, and does not require the subject to be literate. The 6CIT tool has been modified for use in African, Xhosa-speaking subjects and has been validated locally[10] against the Bristol Activities of Daily Living Scale (BADL), which is developed specifically for people with dementia.[11] A local address has replaced the original 5-item memory phrase ("Pumla/Zibi/Z69/Jama Road/ Khayelitsha"). Scoring on the test is weighted and inverse, and measures the number of errors made by the subject for each question, with a maximum score of 28, which indicates severe dementia. A score of ≥ 10 was considered to impaired cognitive function.[12]

Recruitment procedures

The recruitment drive was primarily undertaken at a church-based luncheon club, as well as a primary-care clinic that serves the residential area of Langa. In order to encourage participation in the study, an information session was given at the luncheon club, posters were placed at the clinic and an advertisement was published in the weekly local, free community newspaper. Two trained nurses measured the blood pressure of all volunteers who attended the study site (N = 225). At the recruitment drive stage, BP was taken according to study protocol (see below) using an Omron automated device and weight and height was measured to determine BMI. A questionnaire was administered which included information on age, diagnosis of hypertension, previous history of stroke, continence, and habitual alcohol intake. Cognitive function was assessed using the Cognitive Impairment test (6CIT) described above. Thereafter, eligible subjects were

invited to attend for the first of the three run-in visits. During this visit, as well as the measurements depicted in Figure 1, fasting blood was taken to assess creatinine clearance and detailed information on all prescribed medications was obtained from the drug packages which subjects were requested to bring to the visits.

Sample size

Sample size calculations were performed using the nQuery Advisor 5.0 program using the outcome estimate from a meta-analysis of RCTs of reduced dietary Na in hypertensives aged 45+ years (systolic BP change of -6.3 mm Hg (95 % CI = -8.4 to -4.1 mmHg)).[13] The trial was originally planned to be a two-armed cross-over design therefore sample size was calculated according to this methodology, assuming a common standard deviation of 12.0, and a two group t-test with a 0.050 two-sided significance level and 80 % power. The required 16 participants per cross-over arm (N = 32) was increased to 20 per arm (N = 40) to account for an expected 25 % drop-out. After eligibility testing and screening, 45 participants were randomized to either a low salt (n = 23) or control diet (n = 22) (See Figure 2). Four participants dropped out before the start of the trial and one participant was excluded during the trial due to hospitalization for a stroke, leaving 40 who completed the trial. At the completion of the first arm, the assumption that there would be no carry-over effects in the second arm (after wash-out) was not met, and the study design was subsequently adapted to a 2-phased randomised controlled (parallel group) trial. Using the same parameters as for the first cross-over arm, the sample size was re-calculated to reflect the parallel group design, indicating that 58 subjects per arm (N = 116 in total) were required. However, data obtained in the first cross-over arm demonstrated that the variability of blood pressure measurements (difference between pre- to post intervention) was less than assumed at the start (SD closer to 8 mmHg than 12 mmHg). Using the same expected intervention effect as original (-6.3 mm Hg) and a SD of 10mmHg for being conservative, a sample size of 41 subjects per arm (N = 82 in total) was required.

Data from the first cross-over arm (n = 40) was included as "Phase 1" data in the final analysis. Thus, in Phase 2 an additional 47 subjects were randomized to a low salt (n = 24) or a control (n = 23) diet (Figure 2). Of these, five refused to start the trial while another 2 subjects defaulted during the trial leaving 40 subjects who completed the trial. Results are available for 80 subjects (N = 40 per diet group).

In total, after screening of subjects deemed to be eligible for the trial (n = 113), a total of 26 subjects were excluded. Two subjects had impaired renal function, five had Type 1

diabetes (ascertained from drug information), three were taking furosemide, two were acutely ill at the beginning of the run-in, one subject lived outside Langa, and one subject was found to be normotensive (normal BP and not taking prescribed BP-lowering medication). An additional 12 subjects were excluded during the 3-week run-in period for either defaulting (n = 8) or for being unable to comply with instructions on urine collection (n = 4) (Figure 2).

Medication prescribed for hypertension, diabetes, atherosclerosis and asthma was classified according to the subclasses of drugs for each condition using the Anatomical Therapeutic Chemical Classification (ATC) codes.[14]

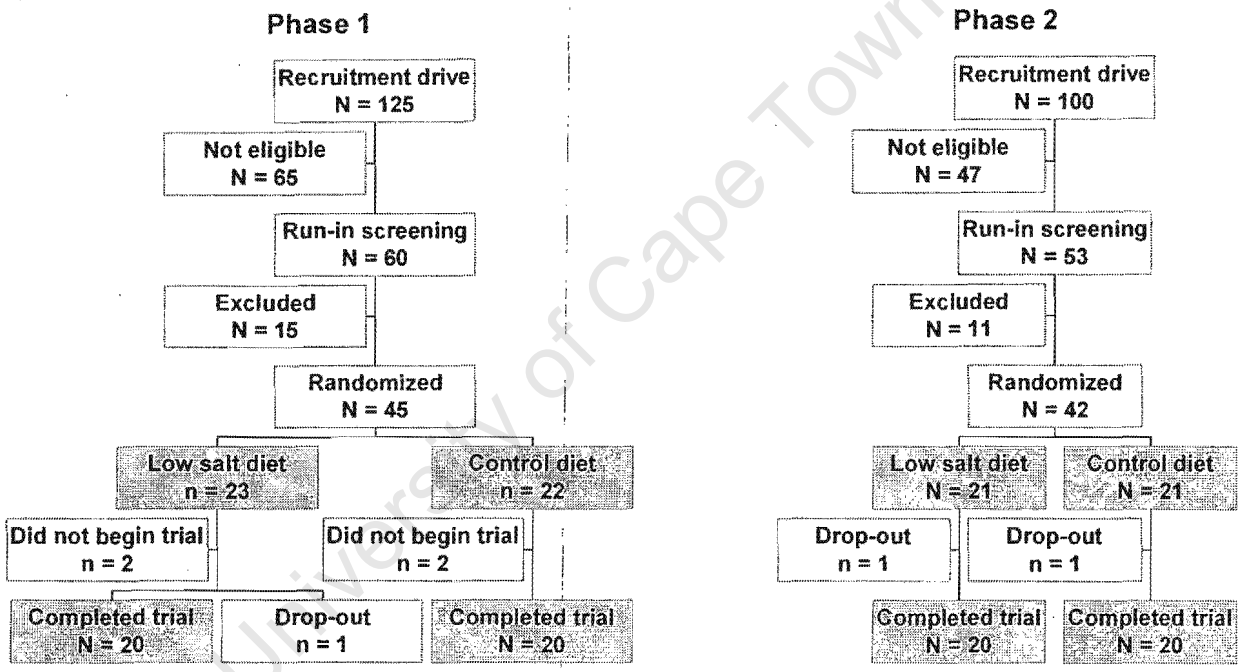


Figure 2
Recruitment and screening of subjects: Phase 1 and Phase 2

Intervention

The intervention comprised 7 commonly consumed food items, known to be important contributors to overall Na intake in the target population. The intervention diet provided a low Na salt replacement (Solo™) and reduced Na versions of bread, margarine, stock cubes, soup mixes, and Aromat, plus 500 ml/day of maas (fermented milk). The cation content of all foods except maas was modified to be reduced in Na, and increased in potassium, magnesium and calcium content. Provision of an additional 500 ml maas per

day (Paramalat) provides an additional 775 mg/day of calcium, which is almost double the current intake described in Chapter 4. A more detailed description of the intervention foods is provided in Chapter 6. The control diet provided the same quantities of these foods to subjects, but which were of standard commerical composition. Instead of maas, 500 ml artificially sweetened cooldrink was provided in the control arm, in order to ensure equivalent fluid intake between the two diets. Being a group of low socioeconomic status, it was assumed that the foods provided would be consumed by all members of the family, therefore an average household size of 5 persons was taken into account and sufficient quantities of food items were provided to ensure that the research subjects would receive their required daily allocation of each of the food products. In an attempt to prevent the purchase of any of the foods included in the intervention from shops during the trial, subjects were able to request more of the food items should the amounts provided be insufficient to meet their household's needs. Subjects were instructed to consume quantities of the test foods that they would usually use. Average habitual amounts of each of the test foods were calculated from the baseline dataset (see Chapter 4). Calculation of the cation contributions of the six intervention foods to total daily intake, according to estimated usual intakes, are shown in Table 1.

Substituting 4 grams of salt (2/3 tsp) per day (ie. habitual discretionary salt intake consumed by black subjects in baseline study) for SOLO® would reduce Na intake from 1 633 mg Na to 640 mg (27.8 mmol) (ie reduction of 61%); increase K intake by 868 mg (22.2 mmol) and increase Mg by 80 mg (3.3 mmol). Overall, compared to the control diet foods, the intervention diet foods provide a 41 % (-1611 mg/day) reduction in Na; an 826 % (+2432 mg/day) increase in K; a 388 % (+636 mg/day) increase in Ca; and a 368 % (+241mg/day) increase in Mg.

Blinding

The trial was double blinded. The corresponding food items provided in each of the intervention arms had identical packaging. Subjects were allocated a study number and randomized to a diet by a statistician. Only the statistician and the dietician responsible for packing the food items had access to the subject's randomization number. All food was provided to subjects in a large, sealed shopping bag which had a label indicating their name, study code number, address and telephone number. After being packed, the food bags were zipped and unopened until the subject received, and signed for their bag. A driver (who was also blinded to diet group allocation of subjects) was employed to deliver the food bags to subjects' homes three times a week. Neither the principal

investigator (KC) nor observers taking blood pressure and other measurements were aware of which types of foods the subjects were receiving at any given time.

Table 1
Contribution of cations from intervention and control foods, according to estimated consumption of quantities per day (independent laboratory analyses of food items)*

Food item and recommended quantity per day	Na content/ Day	K content/ Day	Ca content/ Day	Mg content/ Day
1. Solo® salt replacement (4 g/day)	640 mg	868 mg	-	80 mg
Normal table salt (4 g/day)	1 554 mg	-	-	11.6 mg
2. Reduced Na brown bread – 5 slices (5 x 30g = 150 g)	526 mg	524 mg	249 mg	131 mg
Normal Sasko brown bread (150 g/day)	735 mg	320 mg	219 mg	86 mg
3. Reduced Na margarine (Rama) 20 g/day†	109 mg	41 mg	-	8.2 mg
Normal Rama brick margarine (20g)†	161 mg	-	-	-
4. Reduced Na Aromat (3g/day)	330 mg	308 mg	0.45	33.6 mg
Normal Aromat (3g/day)	675 mg	4.2 mg	0.25	0.4 mg
5. Reduced Na soup mix (3 pkts/household/week = 180g). Average 5 persons per HH = 36g/pp/week.	180 mg	120mg	2.0 mg	18 mg
Normal Knorr soup mix (3 pkts/household/week = 180g). Average 5 persons per HH = 36g pp/week.	411 mg	9.2 mg	1.5 mg	1.4 mg
6. Reduced Na stock cubes (3 cubes/person/week = 30g)	291 mg	131 mg	0.27 mg	0.64 mg
Normal Knorrex stock cubes (3 cubes/person/week = 30g)	381 mg	1.4 mg	0.35 mg	0.26 mg
7. Maas (500 ml/day) – supplied in intervention arm only	230 mg	775 mg	605 mg	60 mg
TOTAL PER DAY Intervention diet *	**2 306 mg (100.3 mmol)**	**2 767 mg (70.9 mmol)**	**857 mg**	**331 mg (13.8 mmol)**
TOTAL PER DAY Control diet *	**3 917 mg (170.3 mmol)**	**335 mg (8.6 mmol)**	**221 mg**	**90 mg (3.7 mmol)**
% Difference between Intervention and Control diets	**- 41 %**	**+ 826 %**	**+ 388 %**	**+ 368 %**

* Amounts provided by intervention foods only
† Recipe formulation

Compliance of subjects

Subjects were advised not to change their habitual intake of foods and beverages, other than the supplied intervention foods for the duration of the trial. Compliance was monitored using three methods. Firstly, at the beginning of each week subjects returned their salt and Aromat shakers, for weighing of remaining quantities and were provided with new, full shakers of each. Secondly, subjects completed interviewer-administered 24-hour dietary recalls at weeks 4 and 8 of the intervention in order to quantify dietary

intake during the trial (average of both visits taken as intervention intake or "post"). Thirdly, corresponding 24-hour urinary electrolyte concentrations were assessed at weeks 4 and 8.

Consumer acceptability of intervention foods

Prior to commencement of the trial, the experimental reduced sodium bread was developed at the head office of Sasko Milling and Baking, Paarl. The infrastructure of Sasko's consumer testing facilities were used to test palatability of the regular brown Sasko loaf (Na content of baker's percentage of 2 % dry weight); and the reduced Na bread (Na content of baker's percentage of 1.28% dry weight) (See Chapter 6 for detailed methodology). During week 8, subjects were asked to rate the palatability of the trial foods, in comparison to the versions that they habitually consumed. This was assessed by completing a questionnaire with 5-point Likert scale ratings, according to the method described by Drewnowski and colleagues.[15]

Outcome measures

All clinical measurements and questionnaires were administered by trained fieldworkers, and interviews were conducted either in Xhosa or English, depending on subjects' preferences. All of the urinary and blood parameters were analysed by a central laboratory (National Health Laboratories Institute, based at Groote Schuur Hospital) which uses standard methods and quality control procedures.

- **Primary Outcomes**

Blood pressure

Resting blood pressure was measured according to the American Heart Association Recommendations for Human BP Determination,[16] using an automated Omron BP monitor. The Omron has been endorsed by the British Hypertension Association as being reliable for use in clinical trials. A large cuff was used for subjects with an arm circumference ≥ 33 cm. Blood pressure was measured three times on each occasion and an average of the second and third measurements was taken. Blood pressure was measured weekly during the run-in period of 3 weeks, and on the first day of intervention (prior to receiving foods). The baseline ("Pre") BP was taken as the mean of measurements taken on these 4 occasions. Thereafter, BP was measured mid-way during the intervention (i.e. at 4 weeks) and during the final, eighth week. For Phase II, BP measurements taken during the intervention period (i.e. weeks 4 and 8) were calculated as the mean of measurements obtained on two consecutive days, whereas

subjects in Phase I had BP measurements taken on single occasions only.

24-h Ambulatory Blood Pressure (ABP)

Average 24-hour ambulatory systolic and diastolic BP, awake and asleep BP, heart rate, and mean arterial pressure, was measured at baseline and during the final week of the intervention, using an Oscar 2 (SunTech Medical) ABP monitor. The monitor was programmed to take readings every 20 minutes during 'awake time' which was set between 06h00 and 22h00 (maximum of 48 readings). 'Sleep time' was between 22h00 and 06h00 and blood pressure readings were taken every 30 minutes during this interval (maximum of 16 readings). The appropriate cuff size was placed on the subject's left arm and the monitor attached to the cuff. The Oscar 2 monitor was placed in a pouch and strapped over the subject's shoulder. Detailed instructions were provided to subjects about the ABP monitoring process. Twenty-four hours later the Oscar 2 was removed and the data downloaded to a laptop computer which was pre-programmed with the monitor's software.

- **Secondary Outcomes**

Dietary Intake

Habitual dietary intake was assessed using repeated interviewer-administered, quantified 24-hour dietary recall methodology.[17] Subjects reported all food and drink items consumed during the past 24-hour period. The validated Dietary Assessment Education Kit (DAEK),[18] which includes a manual of colour photographs of typical foods consumed by the South African population was used to determine intake and to quantify food portion sizes. The recorded quantities of food consumed were converted to gram weights using the Medical Research Council (MRC) Food Quantities Manual.[19] Average daily nutrient intake was calculated using the Foodfinder III computerised dietary assessment programme, which is based on the 1991 MRC Food Composition Tables,[20] as well as later supplements to the original Tables.[21] The 24-hr dietary recalls were performed the day following each of the 24-hr urinary collections, thus corresponded to the period of urine collection. During the run-in period, habitual dietary intake was assessed from the mean of three repeated 24-hr recalls, each performed one week apart.

Urinary Analyses

Weekly 24-hr urinary samples were collected for electrolyte and creatinine analyses during the run-in period of 3 weeks, and once at week 4 and week 8. For the baseline analyses, the mean of the 3 consecutive weekly measurements were used. Completeness of 24-hr urine collection was assessed using the criterion of at least 500 ml volume **and** a urinary creatinine value ≥ 0.18 mmol/kg lean body mass/day (men) or ≥

0.12 mmol/kg lean body mass/day (women).[22] The following urinary analyses were performed using flame photometry: sodium, potassium, magnesium, calcium, and creatinine.

Biochemical analyses

Fasting blood samples were drawn once during the run-in period, and once during the last week of each of the intervention arms (i.e. at 8 weeks) for the following analyses performed on the Roche Modular auto analyser: serum creatinine (Jaffe reaction with rate-blanking and compensation); plasma glucose (enzymatic colorimetric assay with glucose oxidase (GOD-PAP method)); serum calcium (colorimetric assay with cresolphthalein complexone); and serum magnesium (colorimetric assay with xylidyl blue; Roche modular analyser. Plasma active renin (Nichols Institute IRMA kit) and plasma aldosterone (Diagnostic Products Corporation coated tube kit) were measured once in each subject, during the run-in period.

Body Composition

Anthropometric measurements were taken by trained interviewers during the run-in period, and at the end of each of the intervention arms. Standing height was recorded using a stadiometer and weight was recorded on a calibrated scale, to the nearest 100g. Body Mass Index was calculated as weight (kg)/ (height (m))^2 and classified according to World Health Organization (1997) categories.[23] Whole body bio-electrical impedance was measured at 50 kHz using a standard tetrapolar bioimpedance monitor (Bodystat™), with the subject lying supine. Fat mass was obtained by subtracting Fat Free Mass (FFM) from total body mass and the fat percentage is obtained by expressing fat mass as a percentage of total body mass.

Physical activity, alcohol intake and smoking

Current habitual physical activity levels were assessed at baseline and at the end of the intervention period, using an interviewer-administered version of the Yale Physical Activity Survey (YPAS) for older adults.[24] Individual weekly energy expenditure (kcal/week) was calculated for five activity domains: household chores, gardening, care-giving, exercise and recreation. Subjects were asked whether they were regular consumers of alcohol, and if so, mean daily alcohol intake was assessed by quantifying usual weekly intake of alcohol-containing beverages and calculating grams of alcohol divided by 7. Subjects were asked whether they were current or past smokers of tobacco.

Functional dependence and quality of life

As a measure of functional dependence, ability to perform Activities of Daily Living were assessed using both the 6-item Katz[25] ADL questionnaire and the 10-item Barthel[26] ADL questionnaire. For the Katz ADL score, the possible scoring ranges between 0 and 6,

with a score of 6 indicating full function, 4 indicating impairment, and 2 or less indicating severe impairment. For the Barthel ADL score, possible scoring ranged from 0 - 20, where a higher score indicates greater independence. The Instrumental Activities of Daily Living (IADL) assessment scale[27] was administered to determine ability to perform the more sophisticated tasks of everyday life. Scoring ranges from 0 (totally dependent) to a maximum of 16 (totally independent). Self-perceived health status was assessed using a five-scale item, as well as a visual analogue scale based on the EQ-5D instrument, on which the subject was required to rate their perceived current health status, ranging from 0 ("worst possible health state") to 100 ("best possible health state").[28]

Ethical considerations

The Research and Ethics Committee of the University of Cape Town approved the study protocol, and written informed consent was obtained from all participating subjects. Regarding potential adverse effects of Na restriction on the study subjects, patients were closely monitored for adverse effects and BP was monitored at 4 and 8 weeks in both the intervention and control arms. At all times during the study, subjects had access to both the fieldworkers and a back-up medical service. High-risk patients, such as those having previously had a cerebrovascular accident or those with impaired renal function, were excluded from the study. In addition, the BP criteria for entry to the study excluded subjects with uncontrolled hypertension at baseline (i.e. > 160/95 mm Hg). At the end of the study, all participants received a written explanatory report of their BP measurements and their relevant blood, urinary and dietary parameters that were measured. Participants with previously undiagnosed or uncontrolled diabetes were referred to their primary health-care providers during the run-in screening period.

Statistical analyses

Analyses were performed on an intention to treat basis. Comparison of the diet groups at baseline was considered unnecessary. All the subjects included in the trial met the inclusion criteria and the randomisation process was strictly followed. All statistical analyses were considered significant at the $P<0.05$ level.

For the analysis of office (Omron) BP, linear regression modelling was performed to assess the intervention effect. The model had indicators for diet group, phase and time. The model also included the appropriate interaction effects for testing the consistency of the intervention effect over the two phases and the expected differential change in BP

over time in the two diets. The analysis accounted for the repeated nature of the BP measurements within each subject by using the generalised estimation equation (GEE) approach. From the results of this analysis, the difference in Omron BP measurements between baseline (mean of 4 repeated measurements) and the intervention period (mean of 2 repeated measurements) was calculated for each subject. These pre-post changes were compared between diet groups using the 2-sample t-test as a more robust and general comparison.

For the analysis of 24-hr ABPM, a multivariate linear regression model was used to assess the intervention effect. The vector of 24-hr ABPM measurements obtained at the end of the intervention period was modelled on indicator variables for diet and phase as well as the baseline values of average 24-hr systolic and diastolic BP, 24-hr Mean Arterial Pressure (MAP). The vector of seven measurements consisted of the average systolic and diastolic BP, the wake and sleep systolic, the wake and sleep diastolic BP and the MAP. The multivariate approach utilized the strong correlation structure of this set of 24hr ABPM measurements. The set of covariates used for baseline adjustments were limited to the three variables described above to avoid the problem of co-linearity between the variables

For both the Omron BP and 24-hr ABPM the intervention effect was estimated for the pooled data over both phases as well as for each phase separately. Confidence intervals (95%) were estimated to reflect the precision of these estimates for the trial. For both Omron and 24hr BP there was no significant interaction effects between Phase and intervention. Thus, the pooled estimates were presented since this improved the precision of the estimated intervention effects.

For reported dietary intake changes and potential confounding variables (physical activity, self-rated health score, weight, % body fat), change from baseline was assessed using paired t-tests within diet groups (Pooled or Satterthwaite t-tests for parametric and non-parametric data, respectively). Between-diet group differences were investigated using independent t-tests.

Change in 24-hour urinary excretion of Na, K, Mg and Ca, from baseline values (average of collections taken at 3 visits during run-in) to week 4, week 8 and 'Post' (average of week 4 and week 8 visits), was calculated according to Phase and diet group allocation using paired t-tests. Between-diet group difference in urinary cation change was assessed using independent t-tests. Change in both systolic and diastolic blood pressure

was assessed according to change (baseline-post) in 24-hour urinary excretion of Na, K, Mg and Ca using multivariate linear regression modelling in which diet group and diet*urinary cation change interaction factors were also included.

Results

Baseline sociodemographic characteristics of the subjects and lifestyle factors associated with blood pressure control are shown in Table 2. Mean age was 61.7 (7.9) years, ranging from 50 - 76 years. When asked what they did to control their blood pressure, other than take medication, 47.5 % of subjects reported that they followed a low salt diet, 38.8 % claimed to exercise, 16.3 % took home remedies and 8.8 % controlled their weight (unprompted responses). Only one subject (low salt group, Phase 2) reported being a current consumer of alcohol, thus mean daily intake was not quantified.

Anthropometric and body composition measurements, as well as plasma renin and aldosterone concentrations are shown in Table 3. At baseline, the control group was on average 5kg heavier than the low salt group, which was reflected in their higher BMI and % fat mass. In Phase 2, control subjects had lower serum Mg concentrations than low salt subjects. Overall, 3 subjects in the control group and 2 in the low salt group had serum Mg values below the reference value of 0.65 mmol/l (reference value = 0.65 - 1.10 mmol/l).

Table 2
Baseline sociodemographic characteristics of sample, lifestyle factors associated with hypertension control, self-perceived health status

	PHASE 1		PHASE 2		TOTAL	
	Low salt N = 20	Control N = 20	Low salt N = 20	Control N = 20	Low salt N = 40	Control N = 40
Age (years) (Mean (SD))	64.0 (6.4)	59.4 (8.7)	59.7 (6.1)	61.4 (5.7)	61.8 (6.6)	60.4 (7.4)
Range	51 - 76	50 - 75	50 - 72	51 - 71	50 - 76	50 - 75
Female/male ratio	16/4	16/4	17/3	18/2	33/7	34/6
Literacy rate (ability to read) (% subjects)	100	100	95	90	97.5	95.0
Highest level of education achieved (% subjects)						
No schooling	-	-	-	5	-	2.5
Up to Grade 7 (primary)	5	35	45	35	25.0	35.0
Grade 8 - 10	50	35	25	45	37.5	40.0
Grade 11 - 12	35	10	20	10	27.5	10.0
Tertiary/diploma	10	20	10	5	10.0	12.5
Employment status (% subjects)						
Employed	10	25	15	15	12.5	20.0
Unemployed/Housewife	30	30	30	20	30.0	25.0
Social (old age)grant	55	35	50	55	52.5	45.0
Disability grant	5	10	5	10	5.0	10.0
Type of housing (% subjects)						
Formal housing (privately owned)	55	75	30	50	42.5	50.0
Council/core house	40	15	20	10	32.5	10.0
Informal shack	-	5	10	20	5.0	20.0
Hostel	5	5	35	20	20.0	20.0
No. rooms in house (mean (SD))	3.35 (1.46)	2.65 (1.56)	1.95 (1.23)	2.45 (1.60)	2.65 (1.51)	2.55 (1.57)
No. persons in HH (mean (SD))	5.40 (2.28)	4.65 (2.03)	5.0 (3.11)	5.05 (2.58)	5.05 (2.58)	4.85 (2.30)
Housing density (No. persons/rooms)	2.06 (2.03)	2.19 (1.41)	3.51 (3.11)	2.77 (1.96)	2.77 (1.96)	2.48 (1.71)
Previously diagnosed chronic conditions (% (n))						
Hypertension	100 (20)	100 (20)	100 (20)	100 (20)	100.0 (40)	100.0 (40)
Heart attack or angina	15 (3)	10 (2)	15 (3)	5 (1)	15.0 (6)	7.5 (3)
Any other heart condition	15 (3)	10 (2)	5 (1)	0	10.0 (4)	5.0 (2)
Hypercholesterolaemia	0	10 (2)	5 (1)	5 (1)	2.5 (1)	7.5 (3)
Asthma	10 (2)	15 (3)	20 (4)	20 (4)	15.0 (6)	17.5 (3)
Peripheral vascular disease	5 (1)	0	5 (1)	10 (2)	5.0 (2)	5.0 (2)
Diabetes[1]	25 (5)	0	10 (2)	35 (7)	17.5 (7)	17.5 (7)
Self-rated health status (% subjects)						
Excellent/Very good	5	5	0	10	2.5	7.5
Good	40	75	50	45	45.0	60.0
Fair	55	15	50	45	52.5	30.0
Not good	0	5	0	0	0	2.5
Self-rated health scale						
Mean score (SD)	71.8 (14.3)	60.3 (20.0)	59.8 (21.4)	70.5 (20.6)	65.7 (19.0)	65.4 (20.7)
Range	40 - 90	30 - 90	10 - 100	50 - 100	10 - 100	30 - 100
Tobacco use (% (n))						
Current tobacco use	15 (3)	5 (1)	5 (1)	10 (2)	10 (4)	7.5 (3)
Past smoker	5 (1)	0	5 (1)	10 (2)	5 (2)	0
Physical activity						
Mean (SD) (kcal/wk)	2307(1274)	2393 (1607)	1350 (957)	1676 (1450)	1828 (1214)	2034 (1554)
Range	422 - 5152	318 - 6307	110 - 3605	160 - 6377	110 - 6377	160 - 6377

[1] Data obtained from medication history during run-in period, plus one newly-diagnosed diabetic in low salt group of Phase 1.

Table 3
Weight, height, body composition, serum magnesium, plasma renin and aldosterone concentrations of subjects at baseline, by diet group and Phase

	PHASE 1		PHASE 2		TOTAL	
	Low salt N = 20	Control N = 20	Low salt N = 20	Control N = 20	Low salt N = 40	Control N = 40
Weight (kg)	84.2 (14.6)	91.7 (14.0)	82.4 (13.0)	85.8 (16.8)	83.3 (13.7)	88.8 (15.5)
Height (m)	1.60 (0.07)	1.60 (0.07)	1.59 (0.08)	1.57 (0.07)	1.60 (0.07)	1.59 (0.07)
BMI	33.0 (6.2)	36.0 (6.4)	32.8 (5.6)	34.6 (5.6)	32.9 (5.8)	35.3 (6.0)
Lean mass (kg)	46.1 (6.6)	47.5 (5.8)	44.3 (7.8)	43.3 (9.3)	45.2 (7.2)	45.3 (8.0)
Lean mass (%)	54.6 (9.0)	53.2 (11.0)	54.7 (9.4)	50.8 (6.8)	54.7 (9.1)	51.9 (9.0)
Fat mass (kg)	39.3 (12.4)	43.6 (14.8)	37.5 (11.7)	42.6 (11.0)	38.4 (12.0)	43.1 (12.8)
Fat mass (%)	45.3 (9.0)	46.8 (10.9)	45.2 (9.4)	49.2 (6.8)	45.3 (9.1)	48.1 (9.0)
Serum Mg (mmol/l)	0.81 (0.08)	0.83 (0.11)	0.84 (0.07)	0.79 (0.07)	0.83 (0.08)	0.81 (0.10)
Plasma renin (uU/ml)	35.5 (53.2)	68.1 (78.4)	47.1 (95.2)	56.4 (66.6)	41.3 (76.3)	62.3 (72.1)
Plasma aldosterone (pmol/ml)	228 (149)	202 (142)	284 (264)	216 (156)	256 (213)	209 (148)
Aldosterone:renin ratio	14.3	9.3 (9.3)	22.3 (30.2)	14.7 (19.2)	18.3 (23.8)	12.0 (15.1)

Activities of Daily Living (ADL) and Independent Activities of Daily Living (IADL)

Level of independent functioning was high, with a mean Barthel ADL Index and Katz ADL score of 19.9 (0.3) and 5.90 (0.30), respectively in the low salt group and 19.6 (2.1) and 5.87 (0.79), respectively, in the control group. All subjects scored either 19 or 20 out of a possible 20 on the Barthel Index and had a Katz ADL score ≥ 5 which indicates no impairment, with the exception of one subject who had a score of 7 on the Barthel and 1 on the Katz indices (control group, Phase 2). Out of a possible maximum score of 16 on the IADL index, mean score was 15.9 (0.6) and 15.5 (2.6) in the low salt and control groups, respectively. Regarding IADL, the same subject had a score of 0, indicating maximum impairment while most other subjects scored full points (ie.16) on the Lawton scale. All subjects reported being continent, both for bladder and bowel function. No differences in ability to perform ADL were found between diet groups in either Phase.

Baseline blood pressure

Baseline BP, measured using the Omron automated monitor and 24-hr AMBP is shown in Table 4. On average, 89 % of the number of BP readings attempted during the 24-hr period of wearing the Oscar II device were successfully recorded. AMBP measurements were repeated in 8 subjects at baseline who had less than 50 % of potential measurements.

Table 4
Baseline blood pressure, as measured by Omron and 24-hour ambulatory Oscar 2, according to Phase and diet group (mean (SD))

	PHASE 1		PHASE 2		TOTAL	
	Low salt N = 20	Control N = 20	Low salt N = 20	Control N = 20	Low salt N = 40	Control N = 40
OMRON (Office) BP						
Systolic BP	139.7 (15.7)	132.0 (19.0)	128.1 (11.1)	138.8 (13.8)	133.9 (14.6)	135.4 (16.7)
Diastolic BP	80.2 (9.6)	80.5 (7.0)	79.4 (7.6)	84.1 (7.7)	79.8 (8.6)	82.3 (7.5)
24-HOUR AMBULATORY BP						
No. of readings						
Taken	57.0 (8.0)	54.8 (8.4)	55.8 (11.2)	57.4 (7.1)	56.4 (9.6)	56.1 (7.8)
Attempted	63.1 (2.2)	63.0 (1.7)	63.3 (2.4)	63.3 (1.9)	63.2 (2.3)	63.2 (1.8)
% Obtained	90.3	87.0	88.2	90.7	89.2	88.8
Average Sys BP	135.4 (13.8)	132.6 (16.5)	134.6 (13.6)	145.3 (15.5)	135.0 (13.5)	138.9 (17.0)
Average Dias BP	78.1 (10.2)	76.6 (6.1)	80.4 (7.1)	84.2 (9.7)	79.2 (8.7)	80.4 (8.9)
Wake Sys BP	139.5 (14.0)	136.1 (18.4)	137.8 (14.4)	149.0 (15.4)	138.6 (14.0)	142.5 (18.0)
Wake Dias BP	81.0 (11.1)	79.5 (7.0)	83.1 (6.6)	87.5 (10.4)	82.0 (9.1)	83.5 (9.6)
Sleep Sys BP	123.6 (13.3)	122.9 (13.6)	124.4 (15.0)	134.0 (18.0)	124.0 (14.0)	128.5 (16.7)
Sleep Dias BP	69.0 (8.4)	68.8 (6.1)	71.7 (10.3)	74.7 (9.5)	70.4 (9.4)	71.8 (8.4)

Medication status of subjects is shown, according to diet group, in Table 5. The average number of prescribed anti-hypertensive medications was 2.1 (0.9). The most common anti-hypertensive medication was low ceiling thiazide diuretics (76.2 % subjects), followed by ACE inhibitors (55.0 % subjects). Thirty percent (n = 24) of subjects were on a monotherapy regimen (n = 15/24 were taking low ceiling diuretics,

Table 5
Number (%) of subjects taking regular prescribed medication for hypertension, diabetes and atherosclerosis

Condition and type of drug	Diet group		Total N = 80
	Low salt N = 40	Control N = 40	
Hypertension			
No. types of drugs (Mean (SD))	2.07 (0.86)	2.15 (0.92)	2.11 (0.89)
Diuretics			
Low-ceiling diuretics (thiazide, others)	29 (72.5%)	32 (77.5%)	61 (76.2 %)
Other diuretics: High-ceiling diuretics; Potassium-sparing agents; diuretics and potassium-sparing agents	4 (10 %)	4 (10 %)	8 (10 %)
Beta-blocking agents	11 (27.5 %)	13 (32.5 %)	24 (30 %)
Calcium channel blockers	11 (27.5 %)	11 (27.5 %)	22 (27.5 %)
Agents acting on renin-angiotensin system (ACE inhibitors)	23 (57.5 %)	21 (52.5 %)	44 (55.0 %)
Anti-adrenergic agents - centrally acting (Reserpine)	2 (5 %)	1 (2.5 %)	3 (3.7 %)
Agents acting on arteriolar smooth muscle	3 (7.5 %)	6 (15 %)	9 (11.2 %)
Diabetes (Biguanides/sulphonylureas)†	6 (15 %)	7 (17.5 %)	13 (16.2 %)
Atherosclerosis	8 (15 %)	6 (15 %)	14 (17.5 %)
Vasodilators; Platelet aggregation inhibitors; Aspirin; Cardiac glycosides; Antiarrhythmics			

† One subject was newly diagnosed as being diabetic in the run-in period, so was not taking hypoglycaemic agents at beginning of study.

while the most common combination of antihypertensive drugs was low ceiling diuretics and ACE inhibitors (n = 22/80; 27.5 %), followed by low ceiling diuretics, calcium channel blockers and ACE inhibitors (n = 8/80; 10 %), and then low ceiling diuretics and beta blockers (n= 7/80; 8.8 %). No subject was taking a single agent which combined diuretics with either reserpine and/or a vasodilator, an ACE inhibitor, or a beta-blocking agent, nor were peripherally acting anti-adrenergic agents or lipid-lowering drugs prescribed. Fourteen subjects (17.5 %; 7 in each diet group) had Type 2 diabetes and were treated with oral agents (17.5 %).

Blood pressure

Omron (office) blood pressure

The mean BP at baseline (Week 1 - mean of 3 weekly repeated measurements plus Day 1 of intervention), at Week 4 (after 4 weeks of consuming randomly assigned diet) and Week 8 (after 8 weeks of consuming randomly assigned diet) is shown in Table 6 and demonstrated graphically in Figures 3 and 4, and. Results are combined for Phase 1 (N = 40) and Phase 2 (N = 40).

Table 6
Mean BP measurements (Omron) at baseline, week 4 and week 8, according to diet group (Phase I and II combined)

Visit	BP	Low salt			Control		
		Valid N	Mean	SD	Valid N	Mean	SD
Baseline†	SysBP	40	133.91	14.62	40	135.43	16.71
	DiasBP	40	79.82	8.58	40	82.26	7.47
Week 4‡	SysBP	40	125.74	19.17	40	136.75	22.14
	DiasBP	40	78.12	12.39	40	81.40	8.54
Week 8‡	SysBP	40	127.50	16.73	40	132.51	17.96
	DiasBP	40	79.06	10.86	40	82.08	8.38

† Mean of run-in visits (1,2,3) and visit 4 (day 1 of intervention)
‡ Mean of 2 consecutive days' visits for Phase II subjects, 1 visit for Phase 1.

Figure 3
Mean systolic BP (Omron) at baseline (Week 0), Week 4 and Week 8 of intervention, according to diet group allocation (Confidence intervals reflect SEM)

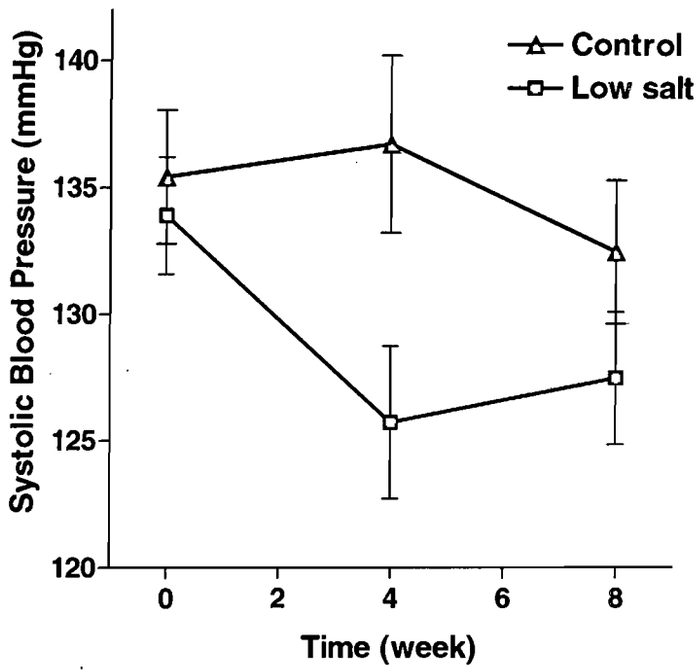
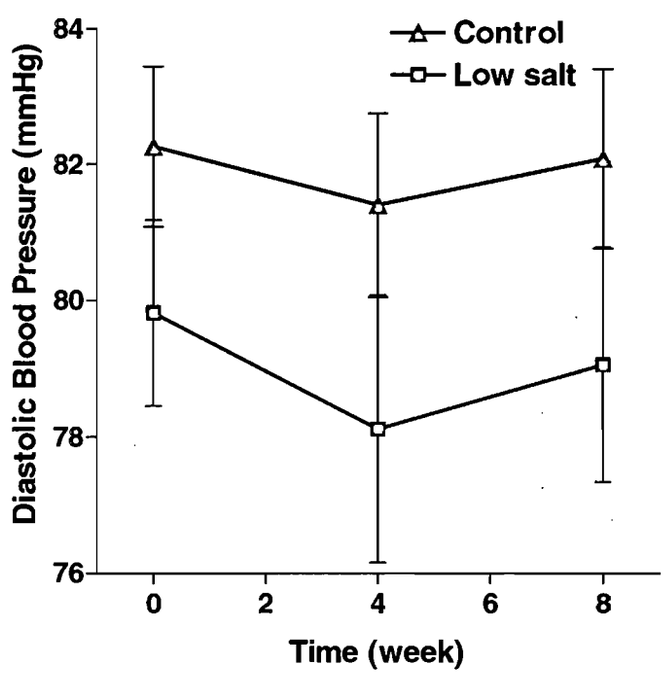


Figure 4
Mean diastolic BP (Omron) at baseline (Week 0), Week 4 and Week 8 of intervention, according to diet group allocation (Confidence intervals reflect SEM)



From Figures 3 and 4, it is evident that most of the effect of the intervention had already occurred within the first 4 weeks of the intervention. For systolic BP, the overall linear regression model showed a significant interaction (P = 0.0321) effect between diet and time which is what was expected from a successful intervention. This interaction necessitated an investigation of the BP between-diet group differences at each time point and within each diet group over time. The between-diet difference (estimated from the model) increases from 1.54 mm Hg at baseline to 10.4 mmHg at week 4 and then drops to 5.4 mmHg at week 8 (Table 7).

Table 7
Analysis of regression modelling of parameter estimates for change in Omron Systolic BP according to Diet*Time interaction effect (GEE approach)

Group	Time	Estimate	SE	P value	95 % CI estimate
Control	1 - 4	-1.368	2.554	0.5923	-6.373; 3.638
Within-group	1 - 8	1.928	2.471	0.4353	-2.916; 6.772
difference	4 - 8	3.296	2.812	0.2412	-2.215; 8.807
Low salt	1 - 4	7.521	1.977	0.0001	3.646; 11.396
Within-group	1 - 8	5.804	2.177	0.0077	1.538; 10.070
difference	4 - 8	-1.717	2.322	0.4598	-6.268; 2.835
Between-group	1	1.549	3.475	0.6557	-5.261; 8.360
difference	4	10.438	4.700	0.0264	1.226; 19.649
(Control-Low salt)	8	5.425	4.044	0.1797	-2.500; 13.351

For diastolic BP, the overall regression model did not find a significant interaction (P = 0.9163) effect between diet and time, nor were any differences in BP found for either within- or between-diet groups over time (Table 8).

Table 8
Analysis of regression modelling of parameter estimates for change in Omron Diastolic BP according to Diet*Time interaction effect (GEE approach)

Group	Time	Estimate	SE	P value	95 % CI estimate
Control	1 - 4	1.069	0.856	0.2118	-0.609; 2.748
Within-group difference	1 - 8	0.206	1.046	0.8439	-1.844; 2.256
	4 - 8	-0.863	1.220	0.4790	-3.254; 1.527
Low salt	1 - 4	1.248	1.185	0.2920	-1.073; 3.570
Within-group difference	1 - 8	0.840	1.092	0.4418	-1.300; 2.980
	4 - 8	-0.408	1.355	0.7632	-3.064; 2.247
Between-group	1	3.696	2.256	0.1015	-0.727; 8.118
difference	4	3.287	1.967	0.0947	-0.568; 7.143
(Control-Low salt)	8	2.218	2.060	0.2817	-1.821; 6.256

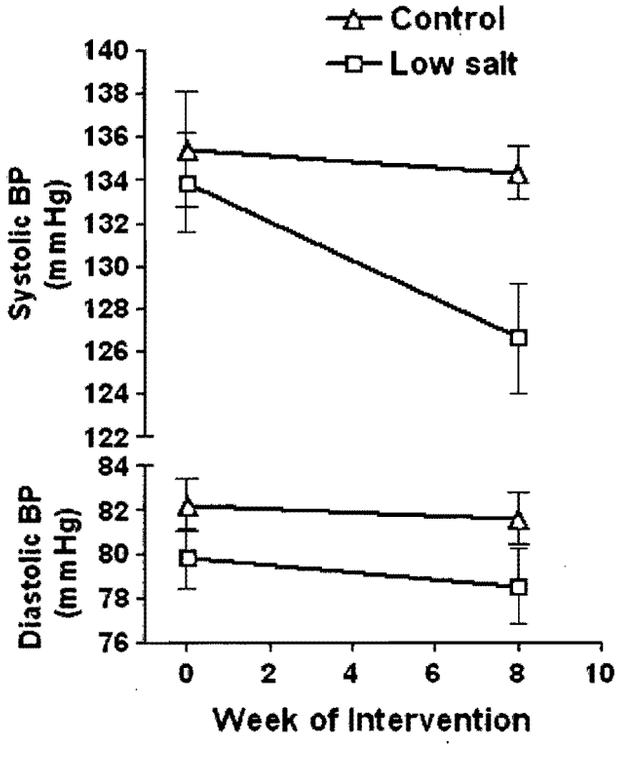
Since no difference was found in change from baseline between weeks 4 and 8 for both systolic (Table 7) and diastolic (Table 8) BP, the average BP of weeks 4 and 8 ("Post") was calculated and is shown in Table 9 and demonstrated graphically in Figure 5.

Table 9
Mean BP measurements for Pre and Post, according to diet group (Phase I and II combined)

Visit	BP	LOW SALT			CONTROL		
		Valid N	Mean	SD	Valid N	Mean	SD
Pre†	SysBP	40	133.91	14.62	40	135.43	16.71
	DiasBP	40	79.82	8.58	40	82.26	7.47
Post‡	SysBP	40	126.62	16.32	40	134.34	18.48
	DiasBP	40	78.59	10.67	40	81.63	7.62

† Baseline, mean of run-in visits (1,2,3) and visit 4 (day 1 of intervention)
‡ Mean of Week 4 and Week 8 measurements.

Figure 5
Mean systolic and diastolic BP at Pre (baseline) and Post (mean of week 4 and week 8 measurements), according to diet group allocation (Confidence Intervals reflect SEM)



The mean within-diet group difference in systolic and diastolic BP between Pre and Post intervention visits is shown in Table 10 and between-diet group differences shown in Table 11.

Table 10
Mean change from baseline in blood pressure (Omron) associated with a reduced salt, compared to control, diet in black South African hypertensives (Pre to Post)[a]

	N	Mean BP (mm Hg)	Std. Deviation	Std Error Mean
Systolic BP				
Low salt	40	-7.288	10.961	1.733
Control	40	-1.094	12.564	1.986
Diastolic BP				
Low salt	40	-1.229	6.224	0.984
Control	40	-0.634	4.514	0.714

[a] Pre = mean of run-in visits (1,2,3) and visit 4 (day 1 of intervention)
Post = mean of Week 4 and Week 8 measurements.

Table 11
Mean net difference in blood pressure (Omron) between the intervention and control diet groups (Pre to Post)[a]

	N	Mean net difference (mm Hg)	SD	SEM	95 % Confidence Interval of Difference		Independent t test statistic (tailed P-Value
					Lower	Upper	
Systolic BP	80	-6.194	23.577	2.636	-11.442	-0.945	-2.349 (0.021*
Diastolic BP†	80	-0.595	10.876	1.216	-3.019	1.829	-0.489 (0.626)

[a] Pre = mean of run-in visits (1,2,3) and visit 4 (day 1 of intervention)
Post = mean of Week 4 and Week 8 measurements.
* P<0.05; T-test performed assuming unequal variances.

24-hr Ambulatory Blood Pressure

Post-intervention, on average, 90.5 % (57.5/63.5) and 90.7 % (57.6/63.5) of the number of BP readings attempted during the 24-hr period of wearing the Oscar II device were successfully recorded for the low salt and control groups, respectively. One subject who had < 50 % potential readings recorded at first attempt repeated the AMBP measurement. Between-diet group change in 24-hr ambulatory BP from baseline is shown in Table 12. The largest diet effect was seen for wake systolic BP (mean between-group difference of -5.14 (SE = 2.40) mmHg; P<0.05), followed by average 24-hr systolic BP (-4.53 (2.27); P = 0.050).

Table 12
Between-diet group difference† in 24-hour ambulatory blood pressure (General Linear Model, adjusted for baseline average 24-hr systolic BP, diastolic BP, MAP and Phase)

	Mean net difference (mm Hg)	SEM	95 % Confidence Interval of Difference		P value†
			Lower	Upper	
Average Sys BP	-4.527	2.269	-9.047	-0.006	0.050*
Average Dias BP	-2.494	1.338	-5.160	0.173	0.066
Wake Sys BP	-5.138	2.404	-9.928	-0.348	0.036*
Wake Dias BP	-2.661	1.457	-5.565	0.242	0.072
Sleep Sys BP	-3.465	2.540	-8.527	1.596	0.177
Sleep Dias BP	-1.790	1.679	-5.134	1.555	0.290
MAP††	-3.113	1.583	-6.267	0.040	0.053

† Low salt-control
* P<0.05 for differences in BP between diet groups (baseline BP minus 8-week BP)
†† Mean arterial pressure

Compliance with dietary intervention

- **Reported dietary intake**

At baseline, there were no differences in reported energy, protein, fat, cholesterol, carbohydrate, Na, K, Ca or Mg intake between diet groups. Reported Na intake at baseline was similar for the low salt and control groups (1 694 mg/day (4.23 g NaCl) and 1 912 mg/day (4.78 g NaCl), respectively). During the intervention, reported energy and carbohydrate intake increased significantly in both the low salt and control groups (Tables 13 and 14). Reported protein, fat and cholesterol intake remained unchanged during the intervention in both diet groups.

Reported dietary Na intake increased significantly (P<0.0005) by an average of 1 252 (1403) mg (3.13 g salt) per day between baseline and intervention in the control group, but no change was seen in the low salt group. Reported K, Ca and Mg intake significantly (<0.0001) increased by 897 (781) mg, 436 (280) mg and 84 (86) mg respectively, in the low salt group, but no change was seen in the control group.

Table 13
Reported dietary intake at baseline and during week 4 and week 8 of the intervention, according to diet group (Phase 1 and 2 combined): Low salt group (N = 40)

Nutrient		Baseline[a]	Week 4	Week 8	Post[b]	Difference†	P value*
Energy (kJ)	Mean	6440.7	7537.5	7202.4	7369.9	929.3	**0.0267**
	SD	1569.4	2417.7	2405.3	2075.1	1839.7	
	Median	6437.7	7886.1	6927.4	7431.3		
Protein (g)	Mean	53.6	61.0	62.1	61.6	8.0	**0.0488**
	SD	16.1	24.7	25.2	19.6	17.9	
	Median	53.6	56.9	54.6	63.8		
Fat (g)	Mean	48.2	55.2	49.6	52.4	4.3	0.3039
	SD	18.0	25.8	23.4	18.8	18.4	
	Median	46.3	55.7	48.1	53.1		
Cholesterol (mg)	Mean	213.6	222.0	202.2	212.1	-1.5	0.9589
	SD	120.5	200.6	168.8	137.3	129.2	
	Median	205.5	162.1	150.7	170.2		
Carbohydrate (g)	Mean	200.1	251.7	241.2	246.5	46.3	**0.0051**
	SD	55.3	90.8	95.3	84.7	71.5	
	Median	197.1	242.2	234.0	236.7		
Na (mg)	Mean	1693.8	1744.7	1811.9	1778.3	84.5	0.6487
	SD	724.3	1031.4	1119.1	916.3	825.9	
	Median	1502.7	1601.2	1545.4	1656.0		
Ca (mg)	Mean	385.1	910.0	733.0	821.5	436.4	**<0.0001**
	SD	180.1	447.7	498.6	353.0	280.2	
	Median	347.8	922.9	491.2	802.4		
K (mg)	Mean	1831.8	2734.5	2723.1	2728.8	896.9	**<0.0001**
	SD	517.0	1068.6	1314.2	976.6	781.3	
	Median	1762.9	2669.3	2513.0	2729.3		
Mg (mg)	Mean	223.4	316.8	298.0	307.4	84.0	**<0.0001**
	SD	63.6	108.2	128.0	103.4	85.9	
	Median	231.6	303.2	284.0	281.8		

[a] Baseline = average of visits 1,2 and 3 during run-in; [b] Post = average of week 4 and 8.
† Difference = Post - Baseline
* Independent t-test for difference in intake from baseline; Pooled for equal variances, Satterthwaite for unequal variances. P<0.05 values highlighted in bold.

Table 14
Reported dietary intake at baseline and during week 4 and week 8 of the intervention, according to diet group (Phase 1 and 2 combined): Control group (N = 40)

Nutrient		Baseline[a]	Week 4	Week 8	Post[b]	Difference†	P value*
Energy (kJ)	Mean	6432.5	7189.7	7823.3	7506.5	1074	**0.0309**
	SD	1902.5	2569.8	2948.3	2435.9	2185.5	
	Median	6098.9	7381.9	7646.7	7329.2		
Protein (g)	Mean	57.4	56.6	61.2	58.9	1.4805	0.7933
	SD	29.6	24.3	23.2	19.7	25.172	
	Median	52.9	53.8	61.8	56.8		
Fat (g)	Mean	50.5	54.9	59.7	57.3	6.8469	0.2043
	SD	22.7	24.8	35.1	25.1	23.921	
	Median	50.6	53.1	49.0	54.8		
Cholesterol (mg)	Mean	224.2	210.0	242.1	226.0	1.8807	0.9603
	SD	185.7	195.9	195.5	148.9	168.31	

Nutrient		Baseline[a]	Week 4	Week 8	Post[b]	Difference†	P value*
	Median	155.0	137.4	170.2	216.1		
Carbohydrate (g)	Mean	194.1	236.5	255.5	246.0	51.966	**0.0049**
	SD	59.8	106.2	106.0	95.8	79.815	
	Median	190.7	227.1	232.6	215.4		
Na (mg)	Mean	1912.3	3215.2	3112.4	3163.8	1251.5	**0.0002**
	SD	922.4	1933.0	2095.1	1757.2	1403.3	
	Median	1732.4	2826.9	3096.0	2813.4		
Ca (mg)	Mean	406.9	505.0	561.3	533.1	126.26	0.1009
	SD	298.2	395.9	426.4	377.4	340.12	
	Median	326.5	352.8	424.4	426.3		
K (mg)	Mean	1825.5	1735.8	1977.9	1856.9	31.351	0.8420
	SD	587.6	879.0	936.3	798.7	701.1	
	Median	1804.0	1590.8	1742.5	1725.8		
Mg (mg)	Mean	221.5	219.4	250.7	235.0	13.561	0.4446
	SD	64.5	95.9	103.4	91.0	78.886	
	Median	211.8	218.4	232.5	223.2		

[a] Baseline = average of visits 1,2 and 3 during run-in; [b] Post = average of week 4 and 8.
† Difference = Post - Baseline
* Independent t-test for difference in intake from baseline; Pooled for equal variances, Satterthwaite for unequal variances. P<0.05 values highlighted in bold.

A significant (P<0.0005) difference between diet groups for change in intake of Na, K, Ca and Mg intake from baseline to intervention was found (Table 15). In order to account for the increase in energy intake, Na intake was expressed as mg/4 200 kJ. Mean difference in Na/4 200 kJ between intervention and baseline was a reduction of -104 (418) mg/4 200 kJ for the low salt group and an increase of 558 (1058) mg/4 200 kJ for the control group, yielding a net between-diet difference of -661 (805) mg/4 200 kJ (P=0.0006).

Table 15
Between-diet difference in reported dietary intake between baseline and during intervention (average of weeks 4 and 8) (Phase 1 and 2 combined)

Nutrient	Mean difference (Low salt group- Control group)	P value*
Energy (kJ)	-145 (1 973)	0.7437
Protein (g)	6.5 (26.3)	0.2700
Fat (g)	-2.6 (24.7)	0.6409
Cholesterol (mg)	-3.4 (183)	0.9345
Carbohydrate (g)	-5.6 (77.8)	0.7473
Na (mg)	-1 167 (1 532)	**0.0012***
Ca (mg)	310 (392)	**0.0007***
K (mg)	867 (890)	**<0.0001***
Mg (mg)	70.5 (89.0)	**0.0007***

* Independent t-test for between-diet group difference in intake from baseline; Pooled for equal variances, Satterthwaite for unequal variances. P<0.05 values highlighted in bold.

The average amount of food consumed from each food group at baseline and during the intervention is shown, according to diet group in Table 16. There were no differences

between diet groups at baseline regarding quantity of intake from any of the food groups. During the intervention, the low salt group had a significantly higher intake from the milk and dairy products group compared to the control group (P<0.0001).

Table 16
Reported amount of food (g/day) consumed from selected food groups at baseline and during the intervention, according to diet groups: mean (SD)

Food group	Low salt (N = 40)	Control (N = 40)
Grains and grain products		
Baseline[a]	394.3 (160.1)	351.2 (143.6)
Follow-up[b]	368.5 (156.2)	362.7 (175.7)
Vegetables		
Baseline	125.8 (89.7)	90.5 (71.7)
Follow-up	94.6 (106.0)*	84.2 (104.8)
Root vegetables		
Baseline	86.6 (69.5)	79.2 (65.1)
Follow-up	84.9 (64.1)	84.6 (82.8)
Fruits		
Baseline	112.8 (100.9)	130.1 (123.7)
Follow-up	145.7 (110.8)	140.8 (138.0)
Vegetable oils and spreads		
Baseline	9.7 (7.8)	12.3 (13.7)
Follow-up	16.4 (16.3)	23.5 (19.8)*
Milk and dairy products		
Baseline	128.3 (92.6)	127.0 (112.1)
Follow-up†	310.0 (174.5)**	130.0 (180.3)
Meat/poultry/offal		
Baseline	93.2 (63.8)	105.1 (76.1)
Follow-up	131.2 (103.7)	160.4 (93.5)*
Fish		
Baseline	9.7 (30.5)	11.8 (22.7)
Follow-up	13.3 (40.2)	9.9 (27.3)
Eggs		
Baseline	23.1 (27.6)	15.8 (22.9)
Follow-up	16.9 (30.5)	21.9 (30.3)
Legumes		
Baseline	10.6 (32.6)	9.0 (23.9)
Follow-up	11.4 (38.1)	8.9 (35.4)
Nuts, seeds		
Baseline	1.7 (4.8)	3.2 (6.8)
Follow-up	1.6 (5.8)	1.2 (3.4)

[a] Mean of 3 repeated 24-hr recalls; [b] Mean of single 24-hr recall at week 4 and Week 8.
* P<0.05; ** P<0.0001; Wilcoxon 2-sample t-test (two-sided) for difference between baseline and intervention for that diet group.
† P<0.0001; Wilcoxon 2-sample t-test (two-sided) for difference between diet group at intervention.

The average reported amount of individual foods that were provided as part of the intervention is shown, at baseline and during the intervention, according to diet group in Table 17. There were no differences between diet groups at baseline for quantity of intake from any of the food groups. During the intervention, the low salt group had a significantly higher intake of maas (P<0.0001) and a lower intake of margarine (P = 0.019) and cooldrink (P<0.0001) compared to the control group.

Table 17
Reported consumption of individual foods (g/day) that were provided during the study: at baseline and during the intervention, according to diet groups: mean (SD)

Food group	Low salt (N = 40)	Control (N = 40)
Bread (all types)		
Baseline[a]	85.6 (52.4)	94.7 (55.8)
Follow-up[b]	123.2 (72.2)*	129.4 (71.1)*
Margarine (brick - Rama)		
Baseline	3.0 (5.5)	3.8 (6.5)
Follow-up †	12.8 (14.6)**	21.7 (20.3)**
Maas		
Baseline	33.3 (56.7)	28.2 (70.0)
Follow-up ††	215.4 (178.4)**	59.9 (132.9)
Knorrox Stock cubes		
Baseline	0.67 (0.90)	0.93 (1.07)
Follow-up	2.38 (3.37)	3.14 (4.18)
Knorrox Soup mix		
Baseline[c]	0.4 (2.3)	5.0 (18.9)
Follow-up[d]	37.3 (89.1)*	55.4 (93.2)
Aromat		
Baseline	0.08 (1.16)	0.07 (0.24)
Follow-up	0.41 (0.50)**	0.65 (0.65)**
Table salt/Solo		
Baseline	1.12 (1.16)	1.33 (1.31)
Follow-up	0.64 (1.02)‡	1.12 (1.44)
Cooldrink		
Baseline	12.8 (35.3)	13.0 (47.8)
Follow-up ††	17.3 (59.9)	376.8 (267.2)

[a] Mean of 3 repeated 24-hr recalls; [b] Mean of single 24-hr recall at week 4 and Week 8.
[c] Dry powder [d] Reconstituted with water.
* P<0.05; ** P<0.0001; ‡P=0.054; Wilcoxon 2-sample t-test (two-sided) for difference between baseline and intervention for that diet group.
†P<0.05; †† P<0.0001; Wilcoxon 2-sample t-test (two-sided) for difference between diet group at intervention.

There was a non-significant trend for mean daily intake of maize porridge and dishes (these were not provided as part of intervention) to decrease from baseline to intervention in both the low salt (115.0 (131.6) vs 77.9 (106.5) g/day; P = 0.0593) and control groups (84.8 (117.1) vs 65.5 (116.8) g/day; P = 0.1729).

- **Average weekly usage of salt and Aromat**

In Phase 1, subjects assigned to the Control diet had a significantly higher average weighed weekly household intake of salt than subjects in the Low salt group, however this difference was not seen in Phase 2 (Table 18), and was not significant for both Phases combined (P = 0.139). No difference was found for Aromat usage between the diet groups in either Phase.

Table 18
Mean weekly intake of salt and Aromat of subjects' households, according to diet group and Phase

	Phase 1 (N = 40)	Phase 2 (N = 40)	TOTAL (N = 80)
SALT/SOLO (g/week/household)			
Low salt	65.42 (40.71)	71.79 (50.55)	68.61 (45.42)
Control	92.39 (40.71)*	74.48 (44.79)	84.43 (43.21) P=0.139
AROMAT (g/week/household)			
Low salt	42.19 (18.39)	47.25 (21.02)	44.72 (19.66)
Control	46.78 (15.51)	45.39 (14.23)	46.08 (14.71)

*P<0.05; Independent t-test for differences between low salt and control groups, for that Phase.

- **Consumer acceptability of intervention foods**

At the end of the intervention (week 8), subjects were asked to rate, on a 5 point Likert scale, each of the food items that had been provided to them, in comparison to the type of these foods that they usually ate. Most of the low salt group disliked the margarine (87.5 %) and the salt replacement (75 %), but reported preferring the other food items to the regular consumed varieties (Table 19). The control group reported that they preferred all of the food items provided, despite the fact that the foods were unaltered in cation content.

Table 19
Acceptability of foods provided in trial, compared to usual varieties eaten

Food	Like a lot better	Like a little better	Same	Dislike a little more	Dislike a lot more
Bread					
Low salt diet	47.5	30.0	5.0	10.0	7.5
Control diet	77.5	10.0	5.0	5.0	2.5
Margarine					
Low salt diet	5.0	5.0	7.5	12.5	75.0
Control diet	90.0	2.5	5.0	2.5	-
Salt					
Low salt diet	15.0	7.5	2.5	22.5	52.5
Control diet	67.5	22.5	2.5	5.0	2.5
Aromat					
Low salt diet	80.0	5.0	5.0	7.5	2.5
Control diet	82.5	12.5	-	2.5	2.5
Stock cubes					
Low salt diet	67.5	10.0	7.5	10.0	5.0
Control diet	90.0	2.5	5.0	2.5	-
Soup mix					
Low salt diet	62.5	20.0	2.5	7.5	7.5
Control diet	75.0	12.5	-	5.0	7.5

- **Urinary cation excretion**

Change in 24-hour urinary excretion of Na, K, Mg and Ca, according to Phase and diet group allocation, is shown in Table 20. At baseline, mean Na excretion was similar between diet groups in Phase1, but in Phase 2 the low salt group had lower values than the control group. During the trial, there was a trend for a reduced Na excretion in the low salt group, but the between-diet group difference was not significant. Between-diet group difference in 24-hour urinary K and urinary Mg excretion from baseline to intervention was 25 mmol/day ($P<0.0001$) and 0.68 mmol/day ($P<0.005$), respectively. No change in urinary calcium excretion was observed. Difference in excretion from baseline to visit 4 was similar to baseline-visit 5 difference for all cations investigated, except for urinary Na of control group in Phase 2 ($P = 0.045$; Wilcoxon matched pairs signed rank sum test).

The multiple regression model, in which change in BP was investigated against change in urinary cation excretion, diet group, and urinary cation*diet group interaction factors, found that for systolic BP, the R^2 value of the model indicated that 21.5 % of the variation in systolic BP change was due to the dietary components investigated (taking urinary excretion as a proxy for dietary intake). The only statistically significant item in the model was change in urinary potassium ($P = 0.002$; increase in K associated with reduction in BP), with a trend towards an interaction effect between diet and change in urinary K ($P = 0.143$). In regression models which excluded interaction terms, increase in urinary K remained the only significant predictor of both reduction in systolic BP ($P = 0.006$; model $R^2 = 0.176$) and diastolic BP ($P = 0.023$; $R^2 = 0.075$), irrespective of diet treatment arm. The association between change in urinary K excretion and change in systolic and diastolic BP is shown in Figures 6 and 7, respectively. Lines represent non-parametric smoothers to estimate the mean BP change over the urinary K change.

Table 20
Mean (SD) 24-hour urinary excretion of Na, K, Mg and Ca (mmol/day)

	Baseline[a]	Post-intervention[b]	Intervention		Mean change from baseline†	Mean difference between diet groups††
			Week 4	Week 8		
Urinary Na (mmol/24-hr)						
Phase 1						
Low salt diet	182.4 (54.4)	165.2 (70.2)	170.5 (67.2)	164.7 (81.2)	-12.4 (64.5)	-1.9 (44.5)
Control	178.6 (41.6)	168.0 (51.4)	166.3 (60.5)	171.6 (80.8)	-10.6 (43.2)	
Phase 2						
Low salt diet	161.5 (52.4)	142.8 (56.4)	139.0 (62.6)	146.8 (66.8)	-16.7 (43.8)	-15.8 (45.0)
Control	167.8 (62.0)	170.6 (65.2)	157.0 (86.5)	184.2 (69.7)	-0.9 (64.8)	
Phase 1 & 2						
Low salt diet	171.7 (53.7)	154.3 (64.0)	155.2 (66.1)	156.0 (74.1)	-14.6 (54.4)	-8.7 (46.9)
Control	173.2 (52.4)	169.3 (57.7)	161.7 (73.8)	177.9 (74.7)	-5.9 (54.3)	
Urinary K (mmol/24-hr)						
Phase 1						
Low salt diet	55.4 (11.7)	75.8 (21.8)	75.2 (18.2)	77.9 (32.0)	**+22.0 (19.9)****	**+26.8 (13.8)****
Control	52.4 (14.3)	47.6 (8.8)	45.7 (11.1)	49.7 (13.9)	- 4.8 (13.4)	
Phase 2						
Low salt diet	49.3 (9.6)	67.4 (28.8)	68.2 (28.7)	67.4 (34.3)	**+18.0 (25.7)***	**+22.4 (17.5)***
Control	53.5 (15.5)	49.8 (18.7)	48.4 (18.7)	51.2 (21.8)	- 4.3 (16.4)	
Phase 1 & 2						
Low salt diet	52.3 (11.0)	71.7 (25.5)	71.8 (23.8)	72.8 (33.1)	**+20.0 (22.7)****	**+24.6 (16.5)****
Control	52.9 (14.7)	48.6 (14.3)	47.0 (15.2)	50.4 (18.1)	-4.6 (14.8)	
Urinary Mg (mmol/24-hr)						
Phase 1						
Low salt diet	2.81 (0.85)	3.56 (1.20)	3.70 (1.28)	3.50 (1.35)	**+0.82 (0.89)****	**+0.69 (0.70)***
Control	2.79 (0.74)	2.92 (0.92)	3.17 (1.00)	2.68 (1.19)	+0.13 (0.83)	
Phase 2						
Low salt diet	2.91 (1.02)	3.83 (1.91)	3.49 (1.64)	4.25 (2.65)	**+0.94 (1.47)***	+0.68 (0.97)
Control	2.78 (1.16)	3.09 (1.23)	2.96 (1.19)	3.21 (1.42)	+0.26 (0.81)	
Phase 1 & 2						
Low salt diet	2.86 (0.93)	3.70 (1.57)	3.60 (1.45)	3.86 (2.09)	+0.88 (1.20)	**+0.68 (0.88)***
Control	2.79 (0.96)	3.00 (1.07)	3.07 (1.09)	2.95 (1.32)	+0.19 (0.81)	
Urinary Ca (mmol/24-hr)						
Phase 1						
Low salt diet	2.06 (1.52)	2.39 (1.86)	2.49 (2.02)	2.32 (1.91)	+0.36 (1.07)	0.24 (0.74)
Control	0.97 (0.66)	1.09 (0.75)	0.88 (0.66)	1.26 (1.01)	+0.12 (0.73)	
Phase 2						
Low salt diet	1.78 (1.19)	1.88 (1.28)	1.73 (1.07)	1.98 (1.81)	+0.20 (0.95)	-0.34 (0.97)
Control	1.81 (1.28)	2.38 (2.06)	2.16 (2.08)	2.61 (2.29)	+0.54 (1.40)	
Phase 1 & 2						
Low salt diet	1.92 (1.35)	2.15 (1.61)	2.03 (1.62)	2.14 (1.83)	+0.27 (1.00)	-0.05 (0.91)
Control	1.39 (1.72)	1.72 (1.65)	1.50 (1.61)	1.85 (1.86)	+0.32 (1.11)	

[a] Baseline = Average of run-in visits 1,2,3; [b] Post = Average of weeks 4 & 8.
† Baseline minus Post; †† Low salt diet minus Control.
* P <0.05; ** P < 0.001; significant differences highlighted in bold.

Figure 6
Association between change in urinary potassium and change in systolic blood pressure, according to diet group

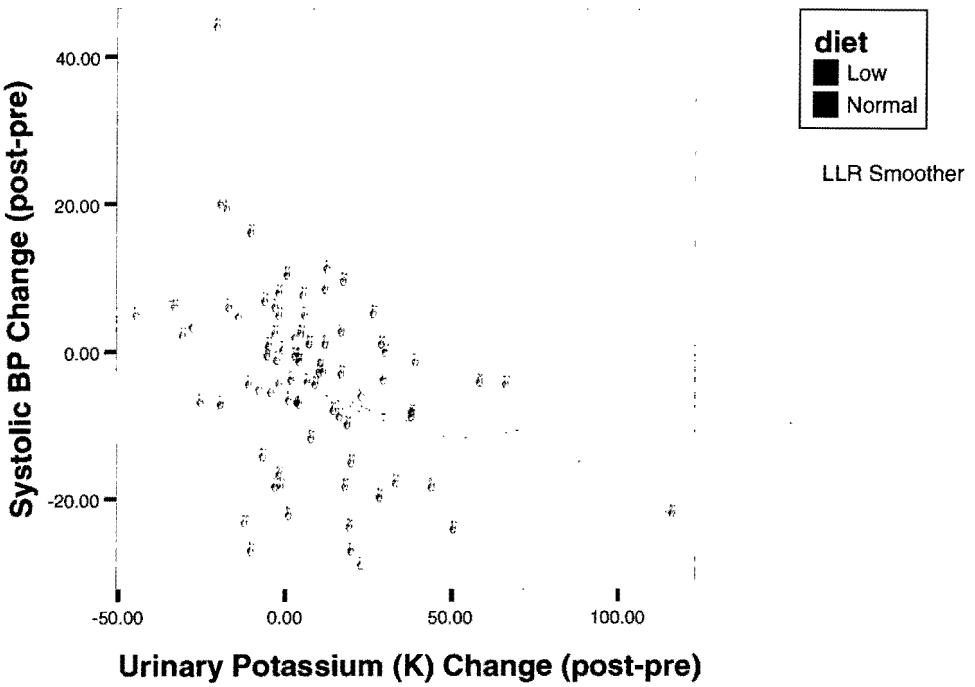
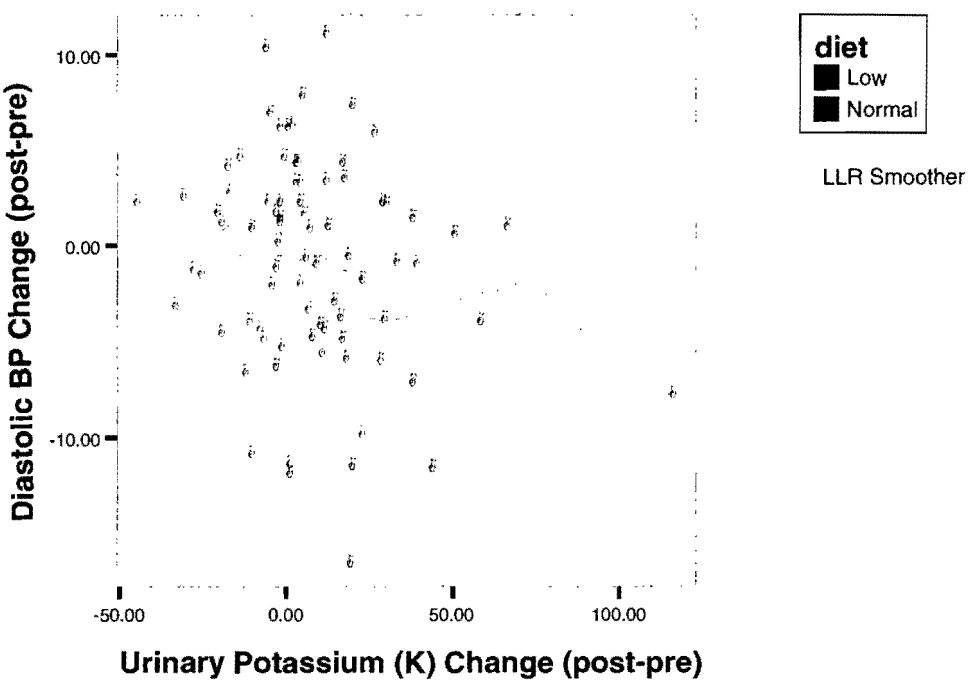


Figure 7
Association between change in urinary potassium and change in diastolic blood pressure, according to diet group



Potential confounders in the diet-BP association

A small, but non-significant weight change was seen between baseline and the end of intervention (Table 21) in both the diet and control groups (Phase 1 and 2 combined). Neither BMI nor % body fat (bioimpedance method) changed during the study, for either of the diet groups.

Table 21
Change, from baseline, in weight, % body fat, physical activity and self-perceived health status, according to Phase and diet group

	PHASE 1		PHASE 2		TOTAL	
	Low salt N = 20	**Control N = 20**	**Low salt N = 20**	**Control N = 20**	**Low salt N = 40**	**Control N = 40**
Weight (kg)						
Difference (Week 8 - Baseline)	-0.66 (1.62)	-0.45 (2.08)	0.31 (1.26)	-0.46 (1.93)	-0.18 (1.51)	-0.46 (1.98
Between group difference in weight change†	-0.21 (1.86) P = 0.7267		0.77 (1.63) P=0.1422		0.28 (1.76) P=0.4756	
BMI (kg/m^2)						
Difference (Week 8 - Baseline)	-0.24 (0.76)	-0.21 (1.09)	0.03 (0.72)	-0.22 (0.84)	-0.10 (0.74)	-0.21 (0.96
Between group difference in weight change†	-0.03 (0.94) P=0.9214		0.25 (0.78) P=0.311		0.11 (0.86) P=0.5613	
% Body fat						
Difference (Week 8 - Baseline)	-0.009 (1.80)	0.028 (0.2)	0.32 (0.98)	-0.014 (1.52)	0.11 (1.45)	0.051 (1.7
Between group difference in weight change†	-0.037 (0.190) P=0.5686		0.46 (1.29) P=0.2841		0.06 (1.61) P = 0.057	
Total physical activity (kcal/week)						
Difference (Week 8 - Baseline)	608 (1418)	-401 (2168)	**-1149 (1252)** **	-391 (1899)	-271 (1593)	-396 (2012
Between group difference in physical activity score†	1009 (1832) P = 0.0896		-758 (1608) P=0.1443		126 (1814) P=0.7578	
Health status score (self-perceived) Difference (Week 8 - Baseline)	12 (17.0)*	-2.8 (20.0)	2.3 (28.1)	6.5(30.1)	7.1 (23.4)	1.9 (25.7)
Between group difference in health score†	**14.8 (18.6)*** P = 0.0165		-4.3 (29.1) P=0.6471		5.25 (24.6) P=0.3427	

† Difference = Low salt group - Control
*P<0.05; **P<0.001 for paired t-test of differences from baseline in that group.

In all groups except for the low salt diet group in Phase 1, reported physical activity levels decreased during the intervention, but between-diet group differences were not significant. Self-perceived health status score improved significantly during the intervention in the low salt group, compared to the control group in Phase 1, but no between-diet differences were seen for the total group combined.

ADL and IADL scores remained high during the study and there were no differences in scores between baseline and intervention (data not shown). The single subject who had

a score indicative of impairment in functioning at baseline on the Barthel and Katz ADL indices, scored full points on both indices at follow-up. All subjects had a maximum Katz ADL score of 6 by the end of the intervention.

Change in anti-hypertensive medication during the trial

According to the medications that were presented to the fieldworkers at baseline and at week 8, twenty-two subjects had a change in anti-hypertensive medication during the trial (Table 22). Four categories were created to make a valid comparison between the diet groups (i.e. medications added; removed; combination of both; or no change). No significant difference in the profile of medication change was found between the two groups (Fisher's exact test P = 0.0947).

Table 22
Change in anti-hypertensive medication between baseline and week 8, according to diet group (numbers of subjects)

Type of drug	Low salt (N =40)	Control (N =40)
Agents added during trial		
One agent (n = 10)		
- ACE inhibitor	3	
- Low ceiling diuretic	2	1
- High ceiling diuretic	1	
- B-blocker	1	1
- Agent acting on arteriolar smooth muscle	1	
Two agents (n = 1)		
- ACE inhibitor + B-blocker	1	
Total - agents added	9	2
Agents removed during trial		
One Agent (n = 5)		
- ACE inhibitor		1
- Low ceiling diuretic	1	1
- Diuretic/K-sparing agent combination	1	
Anti-adrenergic, centrally acting	1	
Two agents (n = 1)		
- Diuretic/K-sparing agent combination + B-blocker		1
Total - agents removed	3	3
Agents added and removed during trial (n = 5)		
(High ceiling diuretic + B-blocker added) - (low ceiling diuretic)		1
(B-blocker added) - (low ceiling diuretic)		1
(High ceiling diuretic added) - (low ceiling diuretic)	1	
(Ca channel blocker added) - (diuretic/K sparing agent)		1
(B-blocker added) – (ACE inhibitor + low ceiling diuretic)		1
Total - agents added and removed	1	4
Anti-hypertensive medications not changed	27	31

The frequencies observed in Table 22 are thus no different from a chance event occurring in each of the medication change categories. For example, in subjects who added medication, the 95% confidence interval for having 9 subjects out of 11 (81.8 %) in one arm is 34.9 to 96.8%. This interval includes 50% - it is assumed that the outcomes that occurred across the two arms have the same underlying probability of 0.5. The 'no change' numbers are nearly identical between diet groups, which further confirms the equality between diet group allocation. The profile for the phases are the same as for the overall sample (data not shown) which again demonstrates consistency.

Discussion

This study demonstrates that in a low-income community setting in a developing African country, in this case South Africa, a food-based dietary intervention resulted in a clinically significant reduction in office systolic BP of -6.2 mmHg over 8 weeks. The decrease in diastolic BP was small and not significant (-0.6 mmHg). Twenty-four hour ambulatory blood pressure monitoring (ABPM) yielded a slightly smaller magnitude of effect for systolic BP, but demonstrated a clinically significant (and marginally statistically significant (P = 0.072)) reduction in diastolic BP (-5.14/-2.66 mmHg for waking measurements). In South Africa, the limited resources at primary health care level allocated to the prevention and management of hypertension necessitate a non-pharmacological population-based approach to curb the escalating burden of cardiovascular disease. Our study offers evidence of an approach that may be useful in a poor community setting, but that requires partnerships between public health agencies and the food industry.

The intervention was based on a limited number of affordable food items that are commonly consumed by the target population. Our baseline studies (Chapters 3 & 4) clearly demonstrated that black peri-urban dwellers in Cape Town consume excessive quantities of Na, accompanied by inadequate intakes of K, Mg and Ca. An important difference between the design of our study and that of other large food-based blood pressure trials[29,30,31,32] is that we did not attempt to alter habitual food patterns, in either quantity or quality. Instead, regular varieties of 6 foods (bread, margarine, stock cubes, soup mix, flavour enhancer, table salt) were substituted with items that had an altered content of Na, K, and Mg. In addition, provision of an additional 500 ml maas per day (a fermented milk product commonly eaten with the staple maize porridge) contributed to a higher Ca intake in the intervention group. Manipulation of the electrolyte content of these food items has provided an opportunity for co-operation between scientific researchers and the food industry to develop products with potential health benefits.

Such academia-food industry partnerships are not common in South Africa and this study is landmark in this regard. For this trial, the third largest baking and milling company in the country (Sasko Milling and Baking, Pioneer Foods) developed a reduced sodium bread which underwent extensive technological and consumer testing prior to use. Another major food company in the South African market, Unilever South Africa Foods, was responsible for developing the rest of the study food items. These two companies together contribute a large proportion of processed food in the country, and are influential trend-setters to the rest of the food industry. The scientific results of the trial can be used by the food industry partners to promote widespread consumption of the novel foods for improved blood pressure profiles, and thus provide a competitive edge in the staple food mass market, while at the same time, enabling a socially responsible approach in terms of promoting health.

The magnitude of the BP-lowering effect is similar to the findings of a 24-week randomised trial conducted in 55 – 75 year old Dutch subjects with untreated mild-to-moderate hypertension (mean reduction of -7.6/-3.3 mm Hg; N = 97).[33] In that trial, the same salt replacement used in this study (SOLO®) was provided to subjects and a number of processed foods containing the product were provided to participants, in place of the regular varieties. The DASH 8–week feeding study, demonstrated a BP reduction of -13.2/-6.1 mm Hg in the sub-group analysis for African-American hypertensives (N = 56).[34] All food and beverages were provided in the DASH study and subjects consumed one supervised meal a day at the research centre, thus increasing compliance with the allocated diet. In trials such as PREMIER,[32] where patients are required to follow the DASH diet whilst purchasing their own food, it has been demonstrated that the recommended number of daily servings of fruit and vegetables could not be met. Another important difference between the design of the DASH trial and the present study is that all hypertensives recruited to the DASH study were free of antihypertensive medication for at least 2 weeks prior to commencement of the trial, which probably magnified the BP-lowering effect of the DASH diet.

Few food-based trials have been conducted in developing countries. A randomized 8-week crossover study of low-salt and high-salt intakes conducted in Jamaica and Nigeria demonstrated a mean between-diet change in systolic BP of approximately 5 mmHg in both country sites.[35] The BP responses were consistent with studies performed in mostly white individuals in developed countries.[36] Mean change in urinary Na excretion between the low-salt and high-salt phases was 70 mmol/day, with no change in urinary K excretion. In the Nigerian site, few processed foods are commonly consumed therefore

participants were able to achieve substantial reductions in Na by simply not adding salt during meal preparation. In Jamaican participants, on the other hand, a wider variety of high sodium processed and convenience foods necessitated more extensive dietary changes to acheive a sustained 50 mmol/day reduction in Na intake. Jamaican participants substituted low-sodium alternatives for table salt, restricted their consumption of fast foods and also adopted food preparation practices aimed at reducing overall salt intake. In addition, subjects could purchase a reduced sodium lunch as the choice of low sodium foods consumed away from home are severely limited in that population.

In the present study, the reduction in systolic BP was similar or higher than blood pressure responses reported in a meta-analysis of randomised trials of either sodium reduction (N = 40 trials) or potassium supplementation (N = 27 trials),[37] despite much smaller changes in both urinary Na and K achieved in our study. The meta-analysis demonstrated that in hypertensive subjects a median Na reduction of 77 mmol/24 h is associated with a blood pressure change of -5.24/-3.69 mmHg while corresponding values for K increase (median = 44 mmol/24 h) were -3.51/-2.51 mmHg.

A food-based intervention, focussing on only a limited number of foods, has not previously been attempted in the country. It was not the purpose of the trial to determine the contribution of each dietary manipulation (ie decreased Na and increased K, Mg and Ca) to blood pressure reduction, but rather to demonstrate effectiveness of a composite dietary approach that could be of public health significance in the target population. As a measure of compliance, both 24-hr urinary excretion and reported dietary intake was measured during the intervention. It is important to note that the study sample size was not powered to detect an association between BP reduction and change in urinary excretion of cations, thereby interpretation of the analyses in this regard is limited. Despite methodological limitations, it seems that in regression models, increased urinary K excretion contributed the most to the BP-lowering impact of the intervention.

The urinary excretion data suggests that the dietary intervention did not achieve the sodium reduction that was expected from the diet formulation. The intervention diet was calculated to provide a decrease in dietary Na intake of 70 mmol/day, compared to the control diet (41 % reduction), but the urinary data demonstrates a (non-significant) mean difference of only 9 mmol/day between the groups. This suggests that subjects may not have been consuming the test foods in the anticipated quantities. Indeed, the provision of free food items may have inadvertently influenced the intake of other foods that were

not included as part of the intervention. The 24-h dietary recall data found a significant increase in energy and carbohydrate intake in both diet and control groups. Food group analyses demonstrates that bread consumption was increased in both groups during the trial. It appears that the increased bread intake displaced other staple foods, notably maize meal, to which large quantities of salt are generally added.[38]

There are discrepancies between the urinary excretion and dietary intake data which may be related to methodological difficulties associated with the collection of both these variables, despite efforts made to reduce bias and to increase validity of results. For example, to minimize error associated with undercollection of urinary samples, we excluded measurements with urinary creatinine values below sex-appropriate minimum reference ranges, expressed per kg lean body mass. Urine samples with volumes less than 500 ml per 24-hr period were also excluded from the analyses. The two interviewers who administered the 24-hr dietary recalls underwent rigorous training, including role-play to increase accuracy of dietary reporting. The dietary data found a significant between-group reported difference in Na intake of 51 mmol/day, and this difference remained even after controlling for an increased energy intake during the intervention (mean = -29 mmol per 4 200 kJ). The difference in Na intake was explained by an increase in reported Na intake in the control group, rather than a substantial reduction in the low salt group, presumably due to the increased consumption of free bread in both groups.

For potassium, the urinary excretion data is confirmed by the 24-hr dietary recall data. Mean between-group difference in 24-hr urinary K excretion was 25 mmol (575 mg), compared to an expected difference of 62 mmol/day. Similarly, the 24-hr dietary recall data found a mean between-group difference in K intake of 22 mmol/day. Urinary Mg excretion differed significantly, by on average 0.68 mmol/day, between diet groups during the intervention, whereas the dietary data found a higher between-group mean difference of 2.94 mmol/day. For calcium, no difference in urinary excretion was observed between diet groups, however dietary intake data demonstrated a significant mean increase of 310 mg in the experimental compared to control group and, mean reported intake of *maas* increased from 33 to 275 g/day in the intervention group. Caution should be exercised in the interpretation of the urinary excretion data as a proxy for intake for calcium and magnesium. Unlike sodium and potassium, where more than 98 % and 85 % of the daily ingested amount, respectively, is excreted in the urine,[39] urinary excretion of calcium and magnesium can vary widely, although it generally increases with increasing intake.[40] The biological relationships between dietary and

urinary calcium and magnesium are not quantitative and, at best, provide qualitative confirmation of compliance.[41] Urinary calcium excretion is largely influenced by sodium chloride intake,[42,43] thus in the control group who reported a substantial increase in dietary salt intake, it would be expected that calcium excretion would also be increased, regardless of dietary intake. This interaction may explain the lack of relative difference in calcium excretion between the diet groups.

Thus, considering both urinary and dietary data, the dietary intervention achieved an overall increase in K, Mg and Ca but without a substantial decrease in the Na content of the diet. In effect, our trial simulated the high sodium arm (150 mmol Na/day) of the DASH-Sodium trial.[30] Our sample had higher baseline Na excretion values (167 - 169 mmol/day) than participants in either the DASH Sodium (152 - 158 mmol/day)[30] or the original DASH diet[29] (132 - 140 mmol/day) studies. Urinary Na excretion remained around the level of 150 mmol/day for the low salt group during the intervention in the present study. Urinary K excretion of subjects in the low salt arm increased during the trial to similar levels as those described for subjects following the DASH diet in both the original DASH study (74.5 mmol/day) and the follow-up DASH-Sodium study (75.0 mmol/day). Urinary Mg levels rose to similar concentrations found in the DASH study (4.03 mmol/day), but urinary Ca was considerably lower than DASH study participants (3.64 mmol) (Neither Mg nor Ca excretion were described for the DASH-Sodium study). We achieved similar BP reductions between diet-control groups as those reported in participants in the high-Na arm of the DASH-Sodium study (-5.9/-2.9 mmHg). Thus, simply by changing the cation content of 5 commonly eaten foods, providing a salt replacement, and the daily addition of a fermented milk drink, the effects of the DASH diet can be achieved in South African black treated hypertensive subjects who consume a high salt intake.

Regarding acceptability of the novel foods with an altered cation content, with the exception of margarine and the salt replacement, most participants in the experimental group preferred the food items to the usually consumed varieties. This subjective rating of foods was supported by the weekly measurement of returned salt shakers, whereby (at least in Phase 1) the average household intake of salt was significantly lower in the low salt compared to control group. Clearly, further technological development and consumer testing of these items in the target population is required before they could be commercially launched on a large-scale level. A possible reason why the control group reported that they preferred all of the food items provided, despite the fact that the foods were unaltered in cation content, may reflect gratitude for receipt of free foods.

No change in body weight or body composition was found during the trial, despite a significantly increased reported energy intake in both diet groups. Participants were instructed to continue to consume their habitual diet, with the exception of substituting the 7 intervention foods for those provided. Average portion sizes of bread and margarine consumed by the target population were estimated using detailed dietary data obtained in the baseline survey (Chapter 4) as well as secondary analyses of other South African dietary datasets.[44] In South Africa, multi-generational households are the norm, especially in households where pensioners reside, and members of a household tend to eat from a common pot.[45,46] In order to improve compliance, sufficient quantities of food for the whole family were provided to participants. A possible explanation for a lack of weight gain is that the magnitude of reported increased energy intake (approximately 1 000 kJ/day) was probably too small to result in weight increases over an 8 week period. Most of the test foods did not contribute significantly to kilojoule intake, namely salt, Aromat (flavour enhancer) and stock cubes.

A number of methodological issues need to be considered. The most important of these is the measurement of the main outcome, blood pressure. Both office-based electronic measures and 24-hr ambulatory blood pressure monitoring (ABPM) were performed. For systolic BP, the magnitude of change associated with the dietary intervention was larger using office-based compared to ambulatory measurements. Three other studies of dietary intervention have also reported a smaller effect size measured by ABPM than that detected by standard measurements.[47,48,49] ABPM is generally considered to be a preferable alternative to traditional methods for measuring BP in clinical trials.[50,51,52,53] Its perceived benefits in this regard include enhanced precision, thus allowing for a reduced sample size and increased study power, elimination of observer bias, and identification of subjects with 'white coat' (or 'isolated office') hypertension.[54,55,56,57] Importantly, there is evidence that ABPM reduces the variability of estimates of BP change in clinical trials.[58,59,60,61] The ability of ABPM to detect BP change in comparison to multiple office based measurements has been investigated using data from the DASH feeding trial.[62] Both ambulatory and five daily pairs of random-zero (RZ) BP measurements were taken pre- and post-intervention in 321 participants. For systolic BP, the standard deviations of change in mean 24-h ambulatory BP were comparable or lower than the corresponding standard deviations of change in RZ-BP based on five daily readings. For change in mean waking ambulatory BP, the standard deviations were comparable to those obtained using three to four daily RZ readings. It was concluded that ABPM was more

efficient than three to four sets of daily RZ readings, in that a smaller sample size could detect a given BP change and less clinic visits were required.

A major limitation of the study is that office measurements (Omron) of BP were taken on only one occasion at each of the follow-up visits for Phase 1 participants, and at two occasions for Phase 2 participants. The intervention effect was calculated using the mean of three repeated measurements for the baseline measure and the mean of two repeated measurements (week 4 and week 8) as the 'post' BP measure, in an attempt to reduce variability. In contrast to other published studies,[54-57] we did not find evidence of enhanced precision using the ABPM method compared to the automated Omron office measurement in a sample of older black hypertensive South Africans from a low income community, even when only between two and four repeated office measurements were taken. A very large variability in BP change was found using either the office measurement or ABPM, and this far exceeded that reported in hypertensives in the DASH study. For example, standard deviation of change in systolic BP in the present study was 20.3 mmHg and 23.6 mmHg for ABPM and Omron measurements, respectively, compared to 8 mmHg for 24-hr ABPM and 8.7 mmHg for 4 repeated measurements of RZ-BP in DASH participants.[62] The unfamiliarity and anxiety of wearing the ABPM device may have contributed to a larger than expected variability in 24hr blood pressure measurements in our sample who were of low educational status. Indeed, in the DASH study, 50 - 55 % of participants reported that the wearing of the ABPM device interfered with their usual home and work activities, while 15 - 20 % of participants reported that the monitor interfered with their sleep 'a lot.[62] In the present study, compliance with instructions to wear the Oscar II device was good; between 88.8 % and 90.7 % of a potential 63 readings were recorded for the 24-hr periods of BP measurement. Measurements were repeated in 9 subjects who had less than 50 % of potential readings at first attempt, and in all but one of these individuals the second attempt yielded at least 80 % of valid recordings.

Vollmer *et al*[62] have demonstrated that in order to detect a between-group difference in BP of 5 mmHg (with study power of 90 %), 90 subjects per group are required if 2 sets of daily measurements are obtained. This reduces to 67 in the case of 5 daily measurements and the comparable sample size using a single 24-hr ABPM method is 54 per group. These sample size calculations, extrapolated from the DASH data, were published only after the start of our trial. Notwithstanding, our sample size (N = 40 per group) was calculated using sound statistical methods, based on variability of BP in the target group. It can be hypothesized that an even larger magnitude of BP change

associated with the dietary intervention may have been demonstrated if we had used more repeat office BP measurements.

Generalizability of the results warrants consideration, with regard to the population-wide approach being recommended for improved BP control. Participants were older, black South African hypertensive patients who were receiving drug treatment for hypertension, mostly from the public health services. Extrapolation of the findings to the normotensive population and to those with severe hypertension cannot be assumed. In order to facilitate the practical delivery of the foods, participants were recruited from only one geographical area, Langa, one of the oldest (> 60 y since settled) and more established peri-urban townships. Langa is located on the outskirts of Cape Town metropolis and has an estimated population of 46 505, mostly Xhosa-speaking Africans.[63] Like most townships, Langa is a legacy of the South African apartheid system. The reason for including older study participants (50 to 76 years) who were likely to be unemployed or retired was an attempt to improve compliance with the study protocol. Good blood pressure control is rarely achieved in this high-risk group which may be attributed to social and economic factors,[64] such as failure to return to health-care centres for follow-up visits due to transport costs and time involved. A further consideration may be a lack of knowledge of the disease and its outcomes, in particular the risk of stroke. Poor service delivery has been identified as a major factor that contributes to the inadequate treatment of hypertension in patients at primary health-care level in South Africa.[65] Further, the relatively short duration of the trial (8 weeks) raises questions regarding compliance with the dietary changes over the longer term and the impact thereof on blood pressure.

All subjects in the present study were receiving anti-hypertensive medication. It is well documented that, with the exception of calcium channel blockers,[66] there is an interaction between antihypertensive medications and dietary interventions on blood pressure outcomes. Lowering the extracellular volume by limiting salt intake can have an additional benefit, enhancing the response to most antihypertensive drugs, including the thiazide diuretics, angiotensin converting enzyme (ACE) inhibitors, and ß-adrenergic blockers. For example, the antihypertensive effect of diuretics is dependent upon initial volume depletion, an effect that is enhanced by salt restriction.[67] Salt restriction also increases renin release, making the BP more angiotensin II-dependent and therefore more responsive to therapy with an ACE inhibitor or angiotensin II receptor blocker.[68,69]

In line with the national guidelines for the management of hypertension in the primary health-care setting that recommend diuretic agents as the first-line drug for all patients with hypertension,[70,71] 86 % of subjects in the trial were being prescribed diuretics. Thiazide-type diuretics, the cheapest anti-hypertensive agent available, were being used by over three-quarters of subjects. Reserpine (centrally-acting anti-adrenergic agent) is the cheapest second-line agent suggested in the guidelines, but only 4 % subjects were being prescribed this drug. ACE-inhibitors were the second most commonly used anti-hypertensive drug in our sample. Importantly, the type of antihypertensive medication use was similar between the control and intervention groups at baseline.

Twenty-two subjects had changes to their anti-hypertensive medication during the trial (based on information provided on the packages that subjects brought to follow-up visits at the research facility). It is possible that some subjects may have forgotten to bring all of their medications to the visits, thus introducing reporter bias, but this cannot be verified. The types of medication change during the trial were similar between the intervention and control groups. The study is a pragmatic clinical trial in which analyses were conducted on an intention-to-treat basis. It would therefore have been methodologically incorrect to remove subjects from the final analyses based on medication change.

The study aimed to demonstrate effectiveness of a community-based dietary intervention, rather than its efficacy. The inclusion of treated hypertensives in the study demonstrates the ***additional*** benefits of dietary intervention in subjects receiving standard treatment. It is unethical to stop hypertensive medication for almost 6 months in study participants selected from a population known to be at high risk of cerebrovascular disease. It was also logistically not feasible to recruit a sample of untreated hypertensives in the South African context. Subjects taking more than one diuretic were excluded as this is considered to be inappropriate management, based on national and international therapeutic guidelines. Our inclusion strategy is in line with international drug trials in hypertension that currently enter patients already on multiple anti-hypertensive drugs, such as the VALUE Trial.[72]

The public health implications of the study's findings, in the context of the challenges for the various relevant roleplayers in the country such as the food industry and the Department of Health, are discussed in the following and final Chapter of the thesis.

Conclusion

This randomised controlled trial provides evidence that the manipulation of the Na, K and possibly Mg content of commonly consumed processed foods, together with the provision of a salt replacement and daily consumption of 500 ml fermented milk, lowers BP in free-living, hypertensive black South Africans. The BP reduction was of a magnitude likely to have health outcome impacts and was achieved in the setting of treated hypertensive individuals who were taking combination blood pressure-lowering drugs. This data makes a case for more active dietary intervention in people with hypertension. The food industry needs to be lobbied to lower the Na content of their products, together with a concurrent increase in K, particularly in staple foods such as bread.

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Chapter 8

Public health implications of food modification for blood pressure control in South Africa: overall conclusions and recommendations

8.1 Introduction: a need for a public health approach to prevent and manage hypertension in South Africa

In South Africa, in relation to the other competing groups of diseases, inadequate recognition is given to the magnitude of the burden of chronic diseases of lifestyle (CDL) and subsequently the prevention of unhealthy lifestyles and early diagnosis and cost-effective management of CDL risk factors are low on the list of priorities.[1] A quadruple burden of disease in this region is characterised by a combination of poverty-related diseases together with the emerging chronic diseases associated with urbanisation, industrialisation and a westernised lifestyle, accompanied by high injury rates associated with the social instability of violence and high crime rates, and by the exploding epidemic of HIV/AIDS across the African continent.[1] The impact that the HIV/AIDS epidemic might have on the anticipated increase in CDL warrants consideration in terms of future health care planning. The Actuarial Society of South Africa developed a model in 2000 (ASSA2000) to project how the AIDS epidemic could affect the patterns of mortality. Although the model projects a tremendous increase in the mortality of young adults by 2010,[22] the data also demonstrates that, irrespective of the deaths attributable to AIDS, there will continue to be a slight increase in the mortality in South Africa attributed to CDL.

The estimated age-adjusted prevalence of hypertension in the adult population is 21.1 %, which equates to an estimated 5.5 million South Africans with the condition.[2] By far, the greatest number of hypertensives are from the black African sector of the population (3.8 million) in whom BP levels are rarely well controlled below 140/90 mmHg.[6] Following stroke and ischaemic heart disease, hypertensive heart disease together with diabetes mellitus and chronic obstructive pulmonary disease were among the leading causes of non communicable diseases in 2000.[3] Hypertensive heart disease death rates were about 3 times higher in the black population compared to those for mixed ancestry and Indian South Africans, and nearly 10 times higher than the rate for the white population.[4] The same study demonstrated that the mortality rate for stroke in black South Africans was double that found among the white population.

A two-pronged approach is required to address the rising burden of chronic diseases in South Africa. The first is the promotion of healthy lifestyles for the whole population (i.e. primordial prevention), and the second, complementary approach, requires early diagnosis and cost-effective management of risk factors and disease. Both approaches need inter-sectoral collaboration, underpinned by stronger leadership from policy makers, advocates and health professionals.[5]

The work presented in this thesis has concentrated on the first approach, namely a health promotion strategy that could be implemented on a population level. The motivation for taking this approach is that in low- and middle-income countries, one of the major constraints for controlling hypertension is the limitation of resources for health care. A pragmatic CVD-Risk Management package has been developed by the World Health Organization (WHO) to facilitate cardiovascular risk assessment and management in low-resource settings.[6] However, cost-effective healthcare interventions to reduce the cardiovascular burden can only be implemented if the health services' policy, environment and financing enable implementation. The success of this approach will also depend on the capacity of primary heath care systems to deliver these interventions and serve the long-term needs of high-risk CVD patients. For many countries, including South Africa, the individual management of large numbers of patients with low CVD risk is simply not affordable. Since it is individuals at lower risk, and not those at high risk, that account for a greater share of the overall disease burden, the distribution of CVD needs to be shifted through population-wide strategies that address all major CVD risk factors, including hypertension.[7]

Although the causes of hypertension are complex and multifactorial, the adoption of an unhealthy lifestyle is recognized as being a major risk factor in its development. The WHO's global strategy on diet, physical activity and health (2004) was endorsed by resolution WHA 57.17 of the World Health Assembly.[8] This document highlights the responsibility of governments of member states, through co-operation with other stakeholders, to create an environment that empowers and encourages individuals, families and communities to make positive, life-enhancing decisions on healthy diet and physical activity. One of the four main objectives of the global strategy is *to reduce the risk factors for noncommunicable diseases that stem from unhealthy diets and physical inactivity by means of essential* ***public health action*** *and health-promoting and disease-preventive measures.* The principles for action that underpin the global strategy stipulate that priority should be given to activities that have a positive effect on the poorest population groups and communities. Again, it is emphasized that such activities *will generally require community-based action with strong government intervention and oversight.*

Regarding diet, a diversity of ethnic and cultural groups exists in South Africa, each with different eating patterns and at different stages in the nutrition transition (i.e change in diet from a traditional high carbohydrate, high fibre, low fat diet to one with a higher fat and

sugar intake and a lower carbohydrate and fibre intake[9]). Urbanisation has a profound influence on the macronutrient profile of the black population. In the early 1990s, Bourne and colleagues demonstrated that in a sample of black adults residing in Cape Town, proportion of energy from fat increased as a function of time spent in the city.[10] At present, only one nationally representative dietary study exists in South Africa, the National Food Consumption Survey, conducted in 1 - 9 year old children in 1999.[11] However, many other dietary surveys exist that are geographically and ethnically representative of the areas where they were undertaken[12] - collectively, these provide good estimates of macronutrient and micronutrient intakes, but as with all dietary surveys, fail to adequately measure salt intake. The baseline surveys in this thesis have provided valid estimates of habitual sodium intake. In South Africans from three ethnic groups, sodium intake was found to be excessive and potassium intake was inadequate. For the first time, important sources of sodium-containing foods in the local population were identified, which included food items such as bread, margarine, soup mixes, stock cubes and flavour enhancers.

Thus, given the current magnitude and the projected increase in the prevalence of hypertension in the country, the inadequacy of health care resources to effectively manage the condition, and the burden of raised blood pressure on mortality and morbidity, together with evidence of a poor quality diet that is high in sodium and low in potassium, public health measures to control blood pressure on a population level in South Africa are paramount. Even more so, perhaps, than in developed countries[13,14] it is critical that South Africa utilise its limited resources optimally and implement cost-effective health-promotion interventions timeously to prevent the predicted epidemic of CDL in the face of all the other health needs in this region.

Challenges of health promotion in South Africa - a country of disparity

At the beginning of the millennium an initiative called *Healthy People 2010* was established in the US with the objectives to reduce heart disease and stroke by 20 % and to reduce the major causal factors associated with these elevated levels, such as excess sodium intake.[15] Healthy People 2010 set two important goals: (1) increasing quality of life and years of life; and (2) eliminating health disparities. In 1993, South Africa was described by the World Bank as one of the world's most unequal economies with a Gini-coefficient for income as high as 0.58 (World Bank, cited in May, 2004).[16] Despite having been placed as one of the 50 wealthiest nations in the world in 2001, with a per capita Gross Domestic Product (corrected for purchasing power parity) of US$11 240 per year,[16] the Gini-coefficient indicator had deteriorated to 0.69 by 2000. South Africa is now listed as

the third most unequal society in the world.[17] As expected, this degree of disparity is reflected in differences in blood pressure control between various sectors of the population. Secondary analyses of the SA Demographic and Health Survey (SADHS) data demonstrated that awareness of hypertension, the use of hypertension medication and the control of hypertension (cut-off point 160/95 mmHg) among subjects with hypertension increased with increasing wealth and were highest in the richest group.[18] Antihypertensive medications can be costly, carry the potential for adverse side effects, and many patients fail to take medications as prescribed. Primary prevention of hypertension provides an attractive opportunity to prevent the continuing costly cycle of managing hypertension and its complications,[19,20] particularly in a country with huge contrasts in the socio-economic indicators of its people.

8.2 Challenge of implementing recommended dietary behaviour change in developing countries

Undoubtedly, the Dietary Attempts to Stop Hypertension (DASH) trials have demonstrated that a carbohydrate-rich diet that emphasizes fruit and vegetables, low-fat dairy products and that is reduced in saturated fat, total fat and cholesterol substantially lowers blood pressure.[21,22] This type of diet is effective, either with[20] or without sodium restriction.[21] Although not tested for blood pressure-lowering effects in African populations, the diet has been shown to be extremely efficacious in Africa American individuals.[23] Subsequently, the OmniHeart trial provided evidence that partial substitution of carbohydrate in a DASH diet with either protein or monounsaturated fat can further lower blood pressure, improve lipid levels and reduce estimated cardiovascular risk.[24]

In developing countries, dietary patterns are largely determined by cost constraints and food availability, and tend to be low in dietary diversity. A positive association between socioeconomic status and dietary diversity has been demonstrated in other developing countries, namely Botswana,[25] urban and rural Mali,[26] Nepal[27] and in a multi-country analysis of data from 10 countries.[28] Data obtained from analyses included in this thesis demonstrate that few individuals consume the recommended number of servings from the various food groups according to the DASH diet principles, and that ethnic and urban/rural differences exist in eating patterns. For example, mean frequency of intake of dairy products ranged from 3.1 - 4.3 times per day in white groups, compared to 0.9 - 1.1 times a day in black urban dwellers, and less than once a day, on average, in black rural groups. Between 15.4 and 29.5 % of urban black South Africans, compared to 0.9 - 5.8 % of their rural black counterparts consumed diary products twice or more times a day. A low

vegetable and fruit intake has been reported in many dietary surveys conducted in the adult black population. Secondary analyses of existing dietary databases in the country provide an overall picture of food group intake.[29] In the total South African adult population, only 21.4 % consumed fruit (average of 287 (SD = 187) g/day in consumers) and 44.8 % consumed vegetables (average of 166g (153) g/day in consumers) on the day of dietary reporting (24-hr recall method). The food items most commonly consumed by the adult population, in descending order, are maize porridge, white sugar, tea, brown bread, white bread, non-dairy creamer, brick margarine, chicken meat, full-cream milk, and green leaves (wild greens/spinach).[29]

Thus, promotion of the DASH eating plan in the South African context is clearly unrealistic. We propose a population-based approach to reduce sodium, while increasing potassium, magnesium and calcium through the modification of various processed foods. Data from the randomised controlled trial (RCT) included in this thesis has demonstrated that the substitution of limited number of commonly consumed food items (bread, margarine, salt, stock cubes, flavour enhancer, soup mix) in which the cation content had been modified for regular varieties, in the presence of the daily consumption of 500 ml fermented milk product (*maas*), lowered blood pressure by a clinically significant magnitude in free-living, hypertensive patients. The magnitude of the BP-lowering effect is similar to that shown by the use of diuretic therapy, however the additional benefits were observed, over and above standard pharmacotherapy use.

Our food-based study, conducted in a resource-poor community setting, showed that many of the beneficial nutrients obtained through the adoption of a DASH diet can be obtained simply by modifying existing food items. Such an approach will need to be accompanied by nutrition education activities, such as promotion of the Food Based Dietary guidelines, in order to improve the overall quality of the diet.

The generalizability of the RCT results warrants consideration, with regard to the primoridal, population-wide approach that is being recommended here for improved blood pressure control. Our study included only people with drug-treated hypertension, and a strong case has been made for more active dietary intervention in this group. However, extrapolation of the findings to the normotensive South African population may be questioned. Data from other developing countries has demonstrated that a short-term salt-restricted diet resulted in a reduction in systolic blood pressure of approximately 5 mmHg in normotensive individuals in both Jamaica and Nigeria.[30]

Meta-analyses of high quality studies of sodium restriction, in both normotensive and hypertensive samples,[31,32,33,34,35] support the blood pressure-lowering benefits of a population-wide strategy to reduce salt intake. Generally, a lower blood pressure reduction occurs in normotensive, compared to hypertensive individuals. In their meta-analysis of 32 randomized clinical trials, Cutler *et al.*[36] concluded that a daily decrease in sodium intake of 100 mmol lowers blood pressure by 5.8/2.5 mmHg in hypertensive individuals and by 2.3/1.4 mmHg among those without hypertension. This level of reduction in diastolic blood pressure could result in an overall 15 % reduction in stroke risk.[37] There is evidence that in older adults, similar falls in blood pressure can be achieved with a modest salt restriction (10 g to 5 g per day) in both normotensive and (untreated) hypertensive subjects.[38]

Original data from Chapters 3 and 4 identified that both normotensive and hypertensive South Africans from three ethnic groups had an excessive intake of sodium and an inadequate intake of potassium. A low potassium intake has been shown to amplify the blood pressure response to sodium in normotensive African-American men. A metabolically controlled study demonstrated that, when potassium intake is even marginally deficient, an elevated blood pressure response to sodium occurs in black, but not white, normotensive men.[39] The response was dose-dependently suppressed when dietary potassium was increased within its normal range. Thus, based on the findings presented in this thesis and studies elsewhere, in the South African context, a population-wide strategy to lower the sodium content of processed foods while simultaneously increasing the potassium, magnesium and calcium content thereof appears warranted to have a beneficial impact on cardiovascular risk reduction.

8.3 International trends and strategies that influence blood pressure through dietary measures

International consensus exists that a modest dietary sodium reduction for people with normal and raised blood pressure has a large enough benefit on blood pressure to justify a guideline advising restraint for the entire population.[40,41,42,43] One of the five dietary recommendations for populations and individuals outlined in the WHO's global strategy on diet, physical activity and health is *to limit salt (sodium) consumption from all sources and ensure that salt is iodized.*[8] Reduction of sodium intake to ≤ 100 mmol per day (2.4 g sodium or 6 g sodium chloride) is one of the five lifestyle-related approaches to preventing and managing hypertension outlined in the internationally accepted JNC7 guidelines.[44] Decreased sodium consumption can be accomplished by changing the population's exposure to sodium in the food supply and thereby represents a challenge most amenable

to a public health solution. However, the success of this intervention can only be achieved with close collaboration between the Department of Health, members of the food industry and food regulating bodies.

It has been recognized for some time now that policies affecting food consumption patterns are cost effective ways to change risk factors for chronic diseases. For example, the implementation of policy to reduce salt content in manufactured foods would result in a leftward shift in the population distribution of blood pressures and a surprisingly large reduction in cardiovascular disease.[45] Similarly, another population-level policy measure that would have a profound influence on the distribution of a population's BMI levels, and hence largely determine its level of type 2 diabetes, is that affecting energy intake (such as the availability and price of energy-dense foods) and/or energy expenditure (such as the level of motorization and mechanization).[46]

While global economic development may ease some of the health challenges in developing countries, chronic diseases have been exacerbated.[47] Important factors in this regard include urbanization, trade, foreign investment and promotional marketing. In South Africa, globalisation influences nutrition patterns through the establishment of multi-national corporations in the country. For example, *McDonald's* opened their first outlets in South Africa in 1995 and by the end of 2002 had about 100 branches in the country, with plans to expand to other countries in the region. The advertising and marketing budgets of these large corporations are enormous, often exceeding the budgets of some countries in sub-Saharan Africa. The annual advertising budget in 2003 in the US for Coca-Cola, Burger King and McDonald's were $473 million, $524 million and $619 million, respectively.[48] In contrast, in the same country only 2.2 % of food advertising spending is used to promote the consumption of unrefined foods, such as fruits, vegetables, wholegrains and beans.[49] The education component of the National Cancer Institute-sponsored Five-a-Day campaign to promote fruit and vegetable consumption is under $1 miilion.[50] Food marketing is particularly effective at influencing the behaviour of children and adolescents.[51]

Despite arguments that multinational food companies promote the abolishment of agricultural subsidies in developing countries, back free trade and support the importation of cheaper, frequently subsidised foods and other goods from industrialised countries,[52] globalisation may be favourable in the case of reducing the salt content of processed

foods. In this regard, international trends in the food industry may be applied to develop food products locally.

It is useful to identify strategies that have been used in other countries to improve blood pressure control through dietary measures. These will be discussed under the sub-headings of lobbying and advocacy, government support/food industry control, food labelling and health education.

- **Lobbying and advocacy**

Largely as a result of the lobbying of consumer pressure groups in other countries, particularly in the United Kingdom and other European countries, impressive strides have been made by the food industry in reducing the amount of salt in their products. In order to ensure economic viability of reduced sodium products, and to maintain market competitiveness, large food companies use consumer panels to identify the point at which the reduction in foods can be detected.

Perhaps the most successful example of food industry commitment to reduce salt content in processed foods is seen in the UK. In that country, the Food Standards Agency has worked closely with food manufacturers and their trade bodies, governmental organizations, retailers, the catering and food service sector, academia, and consumer groups to facilitate their strategy to lower salt intake on a population level. Following a public consultative process, voluntary salt reduction targets for food manufacturers and retailers were published by the Agency in March 2006 to further encourage salt reduction in a wide range of processed foods.[53] The targets apply to salt levels in the 85 food categories that contribute most to the amount of salt in the UK diet. Everyday foods such as bread, bacon, ham, breakfast cereals and cheese are included in the targets, as well as convenience foods such as pizza, ready meals, savoury snacks, cakes and pastries. The industry-related activities of the Food Standards Agency have been accompanied by an ongoing consumer salt awareness campaign (started in September 2004) and research activities to track behaviour and attitudes related to salt intake.

To date, the results of the Food Standards Agency's lobbying have been impressive. Most of the country's major supermarket chains (e.g. ASDA, Marks and Spencer, Sainsbury's, Tesco and Waitrose) offer low salt products, such as bread and ready-meals, as well as products such as canned vegetables with no added salt. The Association of Cereal Food Manufacturers has reduced the levels of salt in breakfast cereals by 33 % since 1998, the

Federation of Bakers have agreed to a further 5 % salt reduction in bread to result in an overall 30 % reduction since the late 1980s, Kraft has lowered the salt in its cheese spreads and snack products by around a third, while members of the Food and Drink Federation have sought to reduce the salt in soups and sauces by 30 %. Many other sectors of the food industry have committed to long-term salt reduction in their products. There is evidence that consumers are not only being exposed to salt reduction in processed foods, but are also adding less discretionary salt to their foods. In October 2005, an estimated 22 million people (46 % of adults) in the UK reported that they were trying to cut down on their salt intake - an increase of nearly six million (or 12 %) since the start of the Agency's salt awareness campaign in September 2004,[54] while 34 % of consumers claim to be checking food labels for salt content.[55] In addition, retail sales of salt indicate a reduction from £23m in 2003 to £20m in 2005.[56]

Unilever, the company that developed the dry goods (stock cubes, flavour enhancer and soup mix) and margarine that were modified in cation content for use in our randomized blood pressure controlled trial, has already been proactive in the salt reduction arena in other countries for some time. In the United Kingdom, in 2002, the company focused their attention on soups and sauces within their Knorr range (same range as available in South Africa) as a first step in reducing the salt content of their products. Unilever approached a number of other leading manufacturers of branded soups and sauces to develop an initiative (Project Neptune) across the industry and received widespread support and collaboration from Heinz, Baxter's, Campbells and Pataks, as well as the food industry's trade association, the Food & Drink Federation. These member companies agreed with the need to lower salt, but acknowledged that any reduction needed to be gradual in order to maintain palatability and thus prevent customers from switching to brands with higher salt contents. The group committed to a three-year programme, with the aim of reducing salt by 10% in 2003, and thereafter reductions of a similar magnitude over the following two years, subject to consumer acceptance. The project is on track to achieve its objectives. Unilever achieved a 10% reduction across soups and sauces in 2003 and a similar reduction was made in 2004. In addition, their range of Birds Eye ready-meal products now contain less than a third of the recommended daily intake (RDI) of salt for an adult and the children's range products were reduced to provide 25% of the RDI for 4-6 year olds and 15% of the RDI for 7-10 year olds.

Heinz, another food giant, recently launched a reduced sugar and salt version of its baked beans, with 50 % lower salt than its standard baked beans, in the UK and the US. The

company simultaneously introduced a "salt equivalent" labelling on each pack to increase consumer awareness and to simplify interpretation of nutritional information.

Further innovation by the food industry to develop quality food products that are in line with the WHO Global strategy on diet, physical activity and nutrition requires scientific understanding of food structure, functionality of food ingredients, composition and nutritional, physiological and psychological properties. The taste, texture and appearance of foods are central to their acceptance. Novel technologies need to be developed to deliver the appropriate sensory attributes while reducing fat, sugar and salt.

- **Examples of the role of governments in influencing the food industry**

In Finland, government intervention to regulate salt levels in some foods, together with voluntary changes by the industry, has led to an overall reduction in salt consumption. A salt substitute, Pan Salt™ (Kallo Group Ltd), which is a blend of sodium chloride, potassium chloride, magnesium sulphate and lysine hydrochloride is now being used by manufacturers in that country. Pan Salt contains 43 % less salt than common table salt. In 1996, Pan Salt™ was used in around 500 products in Finland and at the end of 1998, in at least 200 products in the UK.[57]

In the UK, over a decade ago, the 1992 Health of the Nation White Paper[58] advised a reduction in salt consumption in the context of reducing coronary heart disease and stroke, *as "excessive consumption of alcohol and sodium, together with obesity, contribute to raised blood pressure, which is the main risk factor for stroke.*" The Government's target was to reduce mean systolic blood pressure in the adult population "*by at least 5mm Hg*" by 2005. The first "Eat Well!"[59] action plan from the Nutrition Task Force (NTF) (set up to achieve the Health of the Nation targets) was published in 1994. It commented that "*the evidence for a link between salt intake and raised blood pressure was not thought strong enough to warrant a specific target at the time of the publication of the White Paper.*" But, the report noted that reaching the blood pressure target "*will require a nutritional contribution from sodium intake as well as alcohol and obesity.*" A product development project team was set up under the auspices of NTF, with a remit determined by NTF "*to support and monitor an industry led initiative for a detailed scrutiny.....which will seek to identify the scope for changes in the fat balance of products, and for reductions in salt content.*" By 1996, the Eat Well II![60] report stated that the manufacturing sector felt "*unable to accept the inclusion of salt within the 'scrutiny' at this time. The industry takes the view that the scientific evidence does not sufficiently demonstrate a benefit from reduction in*

salt consumption on a population basis. Nevertheless, a number of manufacturers and retailers are reducing the salt content of their products in response to consumer demand....." Subsequent policy (October 1997) on salt labelling on food products in the UK was in accordance with the European Economic Community (EEC) Directive 90/496 on nutrition labelling. This led the European Commission to conclude that "*the risk of the sodium component of salt in the diet is dependent on intake which, in turn, is determined by dietary habits.*" The Commission was satisfied that current regulations were adequate, but proposed to "*harmonise conditions for the use of the term "low-sodium" on the basis of relevant reports.*" In 2003, the Scientific Advisory Committee on Nutrition published a report on salt and health, under the auspices of the Food Standards Agency and the Department of Health.[61]

An excellent example of how government support can open up the food/nutrition/health debate and provide an opportunity for a partnership between Government (UK & European Union), food growers, food processors, food manufacturers, retailers, nutrition and health professionals and consumers is provided in the UK white paper '*Choosing Health: Making healthier choices easier*' (2004).[62] Accompanying the White Paper is an action plan, *Choosing a Better Diet*,[63] that summarises how the Government will deliver the commitments on nutrition presented in the public health white paper over a 3-year period. The document sets out the Government's plans to encourage and co-ordinate the action of a range of organisations to improve nutrition and health in England and includes action on advertising and promotion of foods to children, simplified food labelling, obesity education and prevention, and nutritional standards in schools, hospitals and the workplace. One of the six objectives of the action plan is to reduce average salt intake from the current 9.5 g/day to 6 g/day by 2010. The nutrition action plan is backed up by two other comprehensive action plans, "*Delivering Choosing Health: making healthier choices easier*" and "*Choosing Activity: a physical activity action plan.*"

- **Food labelling and nutrition logos**

The Codex Alimentarius is the international body established in 1962 by the Food and Agricultural Organization (FAO) and the WHO to develop international food standards. Many countries, including South Africa, base their national food regulations and food labelling laws on Codex standards. In South Africa and many other countries, food labelling is required by law only for those products that make nutritional claims such as 'low fat, "reduced sugar' or 'reduced salt.' In practice, there is a high level of voluntary

nutritional labelling among industry. The Food and Drink Federation (UK) has made more informative labelling one of the commitments in its Food and Health Manifesto.[64]

In the United Kingdom, the Department of Health is taking steps to pressure the European Union (the legislating body) to simplify and clarify food labelling, and to make it mandatory on all packaged foods.[65] In the consultative process around development of the white paper "*Choosing Health,*" there was evidence that consumers are calling for simpler and clearer labelling, that recognizes inequalities in literacy and numeracy levels, and that are more in keeping with consumers' lifestyles. The Institute of Grocery Distribution's *Consumer Watch* report of June 2003 found that 34 % of consumers identified clearer food labelling as the main way that the food industry could help them make healthier food choices.[66]

In addition to mandatory food labelling regulations that may exist, some countries have also adopted nutrition-related logos to help consumers make healthy food choices. For example, in New Zealand, the *Pick the Tick* programme of the National Heart Foundation serves as a 'nutrition signpost' for consumers and appeals to the food industry as a tool for marketing food products. In a 1-year period (1998/99), *Pick the Tick* influenced food companies to exclude about 33 tonnes of salt from the food supply in New Zealand through the reformulation or new formulation of 23 breads, breakfast cereals and margarines.[67] Similarly, in Australia in 1997, Kellogg reformulated 12 of their breakfast cereals to provide an average sodium reduction of 40 %, without compromising consumer taste appeal.[68] As a result, 235 tonnes of salt were removed annually from the food supply and five more products were able to carry the *Pick the Tick* logo (i.e. sodium content < 400mg/100g). In the United Kingdom, the House of Commons Health Committee Inquiry into obesity recommended the introduction of 'traffic lighting' or signposting.[69] Recent Food Standards Agency (FSA) research has suggested that consumers would like simple labelling signposts to help them make informed and healthier food choices.[70]

Further research is being conduced by the FSA to identify which formatting and logo choices work best, and to identify nutritional criteria for the use of the 5 A DAY logo on composite foods that contain added fat, sugar and salt in addition to fruit and vegetables.

- **Health education**

Health promotion activities can be a cost-effective way to improve lifestyles and thereby improve the health of large numbers of people.[71,72] Community interventions have used the

mass media, combined with various other methods, to reach the target population. During the 1980s in the United States, three major projects were conducted, the Stanford Five-City project,[73] the Minnesota Heart Health Programme[74] and the Pawtucket Heart Health Programme,[75] with the aims of lowering blood pressure, blood cholesterol and body weight, reducing smoking rates and encouraging greater levels of physical activity. Each programme lasted 5 - 8 years. An analysis of the combined results of the three studies found that improvements in the outcomes of interest were of very low magnitude and were not statistically significant, while the estimated risk of coronary heart disease mortality was unchanged.[76] Two other community intervention projects, the Heart to Heart project in Florence[77] and the Bootheel Heart Health project in Missouri[78] also showed little success. However, there are examples of community projects that have achieved benefit. The most well known of these is the North Karelia project in eastern Finland, in which nutrition education was an important component of the intervention.[79] Two other European studies in which positive results were demonstrated were the German Cardiovascular Prevention Study (1985 - 1992),[80] conducted in six regions of the former West Germany and Action Heart[81] in Rotherham, England.

As outlined in the UK "*Choosing Health*" white paper,[65] the problem of health education is not so much a lack of information, but that health messages can be inconsistent or uncoordinated or out of step with the way people live their lives. Surveys of consumer awareness, such as the UK Food Standards Agency (FSA) *Consumer Attitudes Survey 2003*,[82] show that most respondents know what constitutes a healthy diet, but that they often lack the ability to translate knowledge into practice. Evidence from the Unites States shows that increasing consumer awareness can influence consumption patterns. Evaluation of the National Cancer Institute's *5 A DAY for Better Health* campaign[83] found that the strongest predictors of dietary change were: knowledge of the recommendation to eat five or more servings of a variety of fruit and vegetables per day; taste preferences; and self efficacy, in particular, confidence in preparing and consuming fruit and vegetables in different situations.

Influencing people's attitudes to the choices they make is the first step in successful behaviour change associated with health education strategies.[84] In acknowledgement that food industry advertising budgets are many times that which the UK Government spends on healthy eating campaigns (£743 million was spent by the UK food industry on advertising of food, soft drinks, chain restaurants in 2003, compared to £7 million for government spending in 2004),[85] the Department of Health is exploring with the food

industry how it might contribute to promoting positive health information and education.[86] In that country, the Food Standards Agency will also continue its public health campaign to reduce high salt consumption. The first phase of the campaign, described earlier, included television and print advertising, as well as a full media relations campaign and dedicated website. The second phase was launched in late 2005. In addition, the Department of Health has also appointed the National Consumer Council,[87] an independent body, to help develop a social marketing strategy that promotes health by influencing people's attitudes to the choices they make. The social marketing strategy will build on previous marketing communications activities such as 5 A DAY, salt reduction, smoking cessation, mental wellbeing and sexual health. Another innovative way of communicating nutrition and health messages is the introduction of the Department of Health-accredited online practical health assessment, the Personal Health Guide. The guides will be tools to assist people in planning for health, and will provide opportunity for self-assessment of health, goal-setting and determining what action an individual wants to take. Further, the "*Choosing Health*" white paper outlines governmental plans to restrict advertising and promotion to children of those foods and drinks that are high in fat, sugar and salt.[88]

8.4 Food industry trends and challenges in South Africa with regard to lowering sodium content of processed foods

Salt is added to food to preserve it, and to give flavour and texture. It is widely used in dairy products, meat and fish products, canned vegetables, bakery products, confectionery, pickles and sauces, as well as savoury biscuits and crackers, crisps and snacks, and canned and packet soups. Salt's use as a preservative is related to its ability to reduce water activity thereby inhibiting or slowing the growth of food-poisoning or spoilage micro-organisms.

Bread is a staple food, together with maize meal porridge, in the black South African population. The baseline studies of this thesis indicated that bread was the single most important contributor to non-discretionary sodium intake, while also providing a large proportion of total magnesium, potassium and calcium intake. Thus, bread is an ideal food to target to replace some of the sodium with the beneficial cations. Even prior to mandatory fortification of bread flour which was introduced in October 2003, bread was already an important source of micronutrients (15.6 % of iron; 16.9 % of zinc; 19.5 % of niacin; and 15.6% of thiamin intake) in the diets of South African children aged 1 - 9 years, an age group that provides an indication of household eating patterns.[89]

In bread, salt plays an important role in flavour. Without salt, or additional sodium derived from baking soda and other leavening agents, bread and similar products are described as tasting insipid. As well as affecting the flavour profile of bread, salt also influences starch properties, dough rheology, baking properties and bread quality characteristics. It reduces crumb stickiness in dough, and bread baked without salt has a coarse, unacceptable crumb structure. However, this effect appears to be related in part to ionic strength rather than the presence of sodium ions particularly, so that up to 40 % replacement of sodium chloride with potassium, calcium or magnesium salts has been reported to have no adverse effect on baking performance, provided optimum mixing times are used, although the flavour of the test breads was poor.[90] By contrast, the need for salt is greatly reduced in rye breads and sour dough breads, and good sensory characteristics have been obtained with 0.75% NaCl.

The sodium content of bread in South Africa has remained higher than in many other countries, at the level of 2% salt, which equates to approximately 520 mg per 100g bread. From figures obtained from nutritional labeling on various types of breads, it is estimated that 3 – 4 slices of bread per day will provide between 480 – 675 mg sodium (1.2 – 1.7 g salt). The addition of 30 g margarine to this amount of bread will provide an extra 480 mg of sodium (1.2 g salt), which totals 2.9 g salt – almost half of the US dietary guideline of the maximum recommended intake of 6g salt per day.[44] In the UK, the British Retail Consortium (which represents food manufacturers) published its salt policy in February 2004, setting upper values for the salt levels in 9 key product categories for members. For bread, the upper level was 440 mg sodium (1.1 g salt) per 100g, with 50 % of plant bread containing no more than 350 mg sodium (0.9 g salt) per 100g. The UK Food Standards Agency has recommended an upper level of salt in bread as 350 mg per 100g as the target for manufacturers to aim towards. There is no equivalent body in South Africa that interfaces between government and the food industry to lobby food manufacturers to reduce salt in their products.

In collaboration with a major bread baker in South Africa, we have shown that sodium in bread can be reduced by 32 % with a simultaneous increase in potassium, magnesium and calcium of 55%, 69 % and 35 %, respectively. This modified bread demonstrated acceptable performance with regard to baking properties and physical quality of the final product, required mixing times during dough production, sensory considerations, and ease of production in baking plants. However, in order for a staple food item to have widespread uptake by the poor sector of the population, it needs to be cost competitive. The additional

cost associated with the novel bread was 8.9 cents per loaf, substantially higher than the 0.7 cents deviation that was considered to be of significant economical consequence to the bread company involved in its development. Use of a salt replacement instead of a locally formulated cation mix would have greatly increased the cost of the novel bread - SOLO™ (The Low Sodium Sea Salt Company, UK) is available at approximately 10 times the cost of normal salt to food processors (Leslie Wilson, personal communication). The cost calculation of 36 % reduced Na bread using SOLO™ as the salt replacement was an increase of 30.1 cents per loaf.

A cheaper option may be to achieve a sodium reduction of 25 % without replacement with other cations. This would incur a negligible additional cost of only R0.00169 per loaf (ingredient cost of R0.92464), assuming a salt price of R0.47/kg. The retail-selling price of this type of reduced sodium bread would be R4.6232, compared R4.6142 for the regular standard brown bread. In terms of the proposed new food labeling legislation (see later), a comparative nutritional claim could be made on the food packaging, thus providing the company with a marketing edge and enabling the consumer to make an informed food choice. However, the blood-pressure lowering effect of such a bread cannot be extrapolated from the results of our trial in which a composite food package was provided to participants for 8 weeks. Further research would be required in this regard.

It is feasible that the baking industry could produce 25 % reduced sodium bread for the mass market, while at the same time producing a more specialized product, similar to the one used in our trial, for niche markets. The findings of our randomized controlled trial suggest that, of the four cations that were modified in the food products, increased potassium intake was the greatest predictor of blood pressure reduction. It has been reported by other authors that a low potassium intake and a high urinary sodium to potassium ratio are related to rise in blood pressure in childhood and may be important in the early pathogenesis of primary hypertension.[91]

Another strategy to lower blood pressure may be through the consumption of foods that are comparatively rich in potassium yet moderate in sodium content. The use of commercial salt substitutes that contain potassium chloride, such as Solo™, in part substitution for regular salt in processed foods, is a possible way of substantially increasing the potassium content of foods, albeit at a cost premium as described above. Potassium salt substitutes supply 390 - 507 mg potassium per gram; an average teaspoon (5 g) of salt substitute contains about 2 g potassium. Increasing the ratio of potassium to

sodium in food often causes the food to have a bitter taste, although this is not detectable by all people. There is concern by some bodies such as the EU Scientific Committee on Food (SCF), that the indiscriminate use of such measures (i.e. the use of potassium salts as substitutes for sodium) could result in intakes (above 5 - 7 g/day) at which toxicity might develop in individuals with undetected renal problems and abnormal retention of potassium.[92] In our randomised controlled trial, subjects in the intervention group who received foods that had some of the sodium chloride replaced with potassium chloride, either by the use of Solo™ (soup mix, margarine, Aromat) or LoSalt™ salt replacements (stock cubes) or another cation mix (bread) reported average intakes of 2 .7 g K per day and had urinary K excretion values of 71.7 mmol/day (2.8 g) which is below the recommended daily intakes for either Europe (3.1 - 3.5 g/day)[93] or the Unites States (4.7 g/day).[94]

Public health recommendations to food manufacturers encourage a gradual reduction in the amount of added sodium across a wide range of products, in the knowledge that the preference for sodium is quickly reduced when less sodium is ingested.[95] This requires a long-term commitment by food industry members and accountability to a central regulating body that monitors salt content in various food categories.

To be an effective way to reduce blood pressure on a population-wide scale, consumers need to purchase the modified food products in place of the regular alternatives. Regarding acceptability of the novel foods with an altered cation content, no differences could be detected by a large consumer testing panel between the reduced sodium bread developed for the purpose of our study and the standard control variety. Indeed, participants in the randomised controlled trial rated most of the cation-modified food products as being preferable to the regular varieties they usually consumed, with the exception of margarine and the salt replacement.

While by no means being as progressive as many European equivalents, the South African food industry recognize the need to develop product ranges with a reduced sodium content, but the motivation to do so has been slow, probably due to the lack of pressure from government, health organizations, retailers and consumers themselves.

Unilever South Africa Foods (Pty Ltd.) have recently launched a 25% reduced salt stock cube in their Knorrox range. Under the auspices of the parent trans-national company, Unilever has in place a global commitment to reduce salt, sugar, and saturated and trans

fat in their products to bring them in line with the World Health Organization global strategy on diet, physical activity and health.[8] The company is currently reviewing their local product portfolio to see where these ingredients can be reduced without compromising on taste (Personal communication, Christine Broadhurst, Consumer Affairs Manager, Unilever South Africa Foods Pty Ltd.).

Heinz, despite being active in reducing the salt content of its products in many other countries, has yet to introduce its low salt range in South Africa. The marketing manager for Heinz Foods South Africa, Anthony West, reports that South African consumers are more concerned about issues around organics and kilojoule content than sodium, and that demand for low-salt versions of foods is presently low (cited in *Food Review*, Nov/Dec 2004).[96] This sentiment is shared by Greg Anderson, the development and research director for Tiger Brands, a company which has a major market share in grain products such as porridge oats, pasta, rice, snacks and treats, as well as some processed meats and baby foods in South Africa (cited in *Food Review*, Nov/Dec 2004).[96] He points out that South African consumers are predominantly concerned about sugar and fat levels in foodstuffs. These quotes from leaders in local food companies identify a need for public education awareness campaigns on the negative effects of salt on blood pressure.

There may be beneficial spin-offs from the development of specialist food ranges by certain retailers. For example, Woolworths, a chain which caters for the upper socioeconomic strata, began the process of measuring and reducing sodium levels of many of its products when it introduced its *Count On Us* low kilojoule range in 2002. The health range required specialist sodium measurement equipment to analyse product and now the sodium content of all products is measured and awareness has been raised regarding the need to lower salt.

8.5 Future policy needs and strategies to address dietary strategies to lower blood pressure in South Africa

Some recommendations for policies that influence the food supply which can be adopted and implemented are presented below.

- **Fiscal policies and levies**

The introduction of fiscal policies that influence the food supply to ensure that the population has access to safe and affordable foods, while discouraging the over-consumption of high fat/high sugar foods has been suggested by Swinburn *et al.* (2004).[97]

An Australian study has demonstrated that food shoppers with low levels of education, and those residing in low-income households, were least likely to purchase foods that were comparatively high in fibre and low in fat, salt and sugar.[98]

The use of taxation and subsidies, in combination with food-related legislation, is a powerful tool of the government to influence dietary behaviour change. Policy recommendations to use taxes and subsidies as a means of inducing many people to shift their diets in a healthier direction were advocated by the World Health Organization (WHO) at the Adelaide conference in 1988[99] and more recently in the 2004 WHO global strategy on diet, physical activity and health.[9] Since the primary business of the food industry is to sell food to consumers and generate profit, not to improve the health of the nation, the introduction of legislation or incentives may be necessary to encourage the food industry to be more proactive in their product development, as well as their marketing and pricing strategies.

A food-related legislative measure that has been effective in reaching the poor in South Africa is the exemption of basic foods from Value Added Tax (VAT).[100] These foods include brown bread, brown bread flour (excluding bran), eggs, dried beans, maize meal, tinned pilchards, mealie (maize meal) rice, milk, cultured milk (maas), milk powder and dairy powder blend, dried mealies, samp, fresh/frozen fruit and vegetables, lentils, rice, vegetable oil (excluding olive oil), edible legumes and pulses of leguminous plants.[101]

A lesson can be learned from the impact that effective legislation in tobacco control and pricing has had on changing smoking behaviours in the country. In 1993, South Africa passed a Tobacco Products Control Act, to which extensive amendments to strengthen the bill were made in 1998. The impact of this legislation, which among other things bans advertising and marketing of tobacco products, has led to a marked reduction in tobacco consumption shown both by a reduced prevalence of tobacco smoking and reduced amount of tobacco sold in the country.[16] There is convincing evidence that price hikes are an effective means to reduce smoking rates[102] and that this measure is a far more effective means of tobacco control than education or media campaigns.[103] It has been estimated that a 10 % increase in price reduces tobacco consumption by about 5 %, especially among the lower socioeconomic groups.[104] Similarly, alcohol intake shows a similar price elasticity to tobacco intake: a price rise of 10 % results in a decrease in consumption by 3 - 8 %.[105]

A review of tobacco control in South Africa, Brazil, Thailand, Poland, Bangladesh and Canada showed that tobacco prevalence can be reduced cost-effectively in high, middle and low income countries.[106] The high cost effectiveness of other preventive measures for chronic diseases has been demonstrated by Yach.[107]

- **Effective communication strategies to influence consumers to change dietary behaviour**

Food labelling and claims, health logos

Food labelling legislation in South Africa falls under the Foodstuffs, Cosmetics and Disinfectants Act of 1972 and is the mandate of the Directorate of Food Control of the Department of Health. The food labelling regulations are currently in the process of being revised, and a draft of the proposed new regulations was circulated for consultation in 2002.[108] In that draft, the categories that were proposed for sodium, with regard to nutrient content claims allowed on food packaging, are described in Table 1:

Table 1
Proposed nutrient content claims (draft food labelling regulations, 2002)[108] for sodium

Proposed nutrient content claim (2002)	Sodium content of food
Low sodium	Not more than 120 mg Na (300 mg salt) per 100g
Very low sodium	Not more than 40 mg Na (100 mg salt) per 100g
Virtually sodium free or sodium free	Not more than 5 mg Na (12.5 mg salt) per 100g

As well as nutrient content claims, nutrient function claims will also be allowed on food packaging provided that the nutrient of interest is present in high amounts in the food item. Nutrient content of a food is considered to be high if it meets 30% of Recommended Dietary Allowance (RDA) per 100g or per single serving. The nutrients of interest in terms of blood pressure control, and which relate to the modification of cations undertaken in the intervention foods in our randomised controlled trial are potassium, magnesium and calcium. Proposed nutrient function claims that will be allowed for these nutrients are described below in Table 2.

Foods which contain < 120mg sodium per 100g may be allowed to display the following health claim: "*Diets low in sodium may reduce the risk of high blood pressure, a disease associated with many risk factors.*" Our data suggests that foods that are high in potassium will improve blood pressure control in the management of hypertension and it is recommended that a nutrient function claim in this regard be considered.

Table 2
Proposed nutrient function claims (draft food labelling regulations, 2002)[108] for calcium, magnesium and potassium

Nutrient	Proposed nutrient function claim (2002)
Calcium	Helps maintain healthy bones and teeth, and a healthy nervous system. Important for healthy regular heartbeat. Needed for muscular growth and contraction and prevents muscle cramps. Essential in blood clotting.
Magnesium	Helps to utilise carbohydrates, proteins, fats & minerals; aids as vital catalyst in enzyme activity, especially those enzymes involved in energy production. Helps maintain a healthy muscle and nervous system. Assists in calcium and potassium uptake and plays role in formation of bone. Plays role in transmission of nerve and muscle impulses, therefore preventing irritability and nervousness. Aids in maintaining proper pH balance and normal body temperature.
Potassium	Important for healthy nervous system. Important for regular heart rhythm and maintenance of stable blood pressure. Aids in proper muscle contraction. Works with sodium to control body's water balance. Aids in transmitting electrochemical impulses.

Introduction of mandatory reporting of sodium on food labels will require a standardized approach by industry, (for example whether sodium or salt (sodium chloride) content is displayed, and whether the units are given in millimoles or milligrams), and will need to be accompanied by extensive consumer training if it is to be effective at changing dietary salt intake behaviour. The upper recommended limit for sodium intake that will appear on food labels is the internationally recognised guideline of <2 400 mg Na per day.[19-check]

Nutrition and health logos

In South Africa, non governmental organizations such as the Heart Foundation and the Cancer Association provide their logos to food products which meet certain specified health claims and nutritional standards, The purpose of these logos is to increase the awareness of consumers by acting as an easily identifiable 'nutrition signpost' and to also provide the food industry with a tool for marketing food products. In this way, the health logos may provide an incentive to some food manufacturers to improve the nutritional content of their foods.

The heartmark logo of the Heart Foundation indicates that a food is lower in cholesterol, lower in saturated fat, lower in salt and high in fibre (where applicable). The upper limit for salt content depends on the category of food. Nutritional criteria for Heart Foundation-approved products are provided in Table 3 below for three of the food item categories which were modified for the blood pressure trial that forms part of this thesis. Stock cubes

are presently not eligible for the Heartmark logo, nor are flavour enhancers such as Aromat-type products, presumably due to their very high sodium content.

Table 3
Guidelines for the nutrient content of selected food categories that would be eligible to carry the Heartmark logo of the South African Heart Foundation

Food category	Nutrient content guidelines for Heartmark approval			
	Fat	**Sodium**	**Fibre**	**Added sugar**
Bread	5 % or less	450 mg/100g or less	3g/100g or more	5 % or less
Margarine	Saturated + trans fats = maximum 28 % total fats.	400 mg/100g (salt = 1 % or less)	-	-
Soups (as reconstituted)	5 % or less	200 mg/100g or less	1.5g/100g or more	-

In addition to nutrient content, nutrient function claims, and disease-specific claims there is ongoing discussion between Medical Research Council researchers involved in the present randomized controlled blood pressure trial (Charlton, Steyn), food technologists and managers from Pioneer Foods (Sasko Milling and Baking) and the Directorate of Food Control (Antoinette Booysen) regarding approval of a composite claim on the combined effects of sodium reduction and increased potassium, magnesium and calcium on blood pressure control. Suggested wording in this regard could be *"Diets reduced in sodium and increased in potassium, magnesium and calcium can lower blood pressure."* Should such a claim be approved, this could provide an incentive to the food industry to not only lower sodium content of foods, but to replace some of the sodium with other beneficial cations.

Marketing and advertising standards

It has been demonstrated that the most frequently encountered source for information on nutrition in urban black South African women is the media, with the radio and TV being the most frequently accessed media sources.[109] Information from these sources are perceived as being highly reliable and trustworthy.[109] Consumers, particularly those with lower educational levels, may not be able to distinguish between information provided in marketing campaigns, and that of valid nutrition education activities. The high frequency of access to the media for nutrition information is to be expected, given that expenditure on electronic and print media related to food sales exponentially exceeds that available to government agencies for health education.[110,111] The impact of commercial advertising (through TV and radio media, as well as written media (magazines, newspapers, and adverts posted on billboards and public transport vehicles)) on the food choice behaviour

of South African adults and children has not received much attention and warrants investigation.

Advertising of energy-dense, high fat and high sugar foods on television to young children has been shown to be an effective way to persuade young children to make undesirable and unhealthy food choices.[97] Regulations that ban the marketing of unhealthy food items, including foods that are high in salt, may influence individuals to make better food choices. To date, no such regulations exist in South Africa. Internationally, it has been suggested that the feasibility of codes of practice in food advertising should be explored through the *Codex Alimentaris* international foods standard-setting body.[8]

Nutrition education: Directorates of Nutrition and Health Promotion

Adult black South Africans living in urban areas have been shown to consume a diet that is not in line with healthy eating guidelines,[112,113] while the National Food Consumption Survey (which was conducted in a representative sample of 3 120 children aged 1 – 9 years) found that the majority of South African children, particularly black children, have poor dietary intakes from many of the food groups.[114] It is the role of nutrition professionals to translate nutrient-based dietary goals into practical and achievable food-based dietary messages for various target audiences. The World Declaration and Plan of Action for Nutrition, adopted at the International Conference on Nutrition in Rome in 1992, includes a number of goals aimed at eliminating or substantially reducing famine and famine-related deaths, chronic malnutrition, micronutrient deficiencies and diet-related communicable and non-communicable diseases. One strategy that was suggested to achieve these goals is the promotion of appropriate diets and lifestyles. In response to this, together with recognition of a need for more effective nutrition education interventions, the World Health Organization (WHO) and Food and Agricultural Organization (FAO) of the United Nations convened an international consultation in 1995 to discuss the development of Food Based Dietary Guidelines (FBDGs) as an effective nutrition education tool.[115]

After extensive development and consumer testing across all socio-economic spectra within South Africa,[116,117,118] the first set of FBDGs for healthy South Africans aged 7 years and older was approved and adopted as national dietary guidelines by the Department of Health (Nutrition Directorate) on 9 May 2003.[119] One of the eleven FBDGs relates specifically to the reduction of salt intake ("*Use salt sparingly*")[120] while another encourages an increased intake of potassium-rich fruit and vegetables ("*Eat plenty of vegetables and fruits every day*"). [121] In line with the philosophy of food-based rather than

nutrient-based dietary guidelines, the current South African food based dietary guideline on salt intake has not adopted a quantitative approach, with regard to an upper recommended intake per day. The wording "***use*** salt ... " may impart the message to the public that it is only salt added to foods at the table and in cooking that needs to be used cautiously. However, accompanying consumer education materials produced by the Department of Health provide detailed information on food sources of salt and how to make appropriate dietary changes. Perhaps controversially, and in contrast to many other countries' FBDGs, there is no guideline that addresses calcium-containing foods.

The 2003 FBDGs are the first single set of national dietary guidelines in the country. Prior to the democratic elections of 1994, when three racially disparate Health Departments existed to address the needs of black, mixed ancestry/Indian, and white populations separately, nutrition educators throughout South Africa made use of separate nutrition education tools for various socio-economic groups. A three food-group approach was used for lower socio-economic groups, while a five food-group approach was adopted for upper socio-economic groups. The early 1990's saw the implementation of these decisions in the various publications issued at the time, such as "A Guide for Healthy Eating" (for white South Africans; Department of Health Services and Welfare, 1993), "Self-Help in Health" (for black South Africans; Department of National Health and Population Development, 1991) and "Think Healthy, Think Food, Think Three Food Groups" (for mixed ancestry South Africans; Wheat Board, 1990). The FBDGs provide an opportunity for nutrition educators to provide consistent nutrition messages in a non-segregating manner.

Most of the foods which were modified in cation content in the present body of work, apart from bread and margarine, are very high in sodium content (stock cubes, Aromat flavour enhancer, soup mix, table salt). It may be argued that consumers, especially those at high risk for hypertension and its associated health conditions, should be advised to avoid these foods altogether. In the diets of people from the lowest socioeconomic strata, there is evidence that these food items are widely used to provide taste to otherwise bland dishes, such as maize meal porridge, particularly when complementary vegetables or protein sources are unavailable.

Current confusion and poor knowledge related to diet and blood is demonstrated in a small survey conducted in 85 hypertensive adults in the Cape Metropole.[122] Despite fruit and vegetables being perceived as having a beneficial effect on hypertension, only 15 % of the group knew that the recommendation for their usage was five or more servings per day.

Knowledge regarding salt usage indicated that over a third (34.1%) of participants believed that monosodium glutamate-containing flavour enhancers, such as *Aromat* or *Fondor*, could safely be used instead of table salt.

Role of health care providers in promoting healthy eating practices

It is known that quality of health care is dependent upon the knowledge, attitudes and practices of both physicians and health-care workers. One of the critical roles of primary health care in the management of blood pressure and prevention of stroke is education of the patient. Health-care professionals at all levels need to continually reinforce the message that stroke can largely be prevented by the control of blood pressure and by maintaining a satisfactory body weight and following a healthy lifestyle.[123] In South African clinics, staff are poorly motivated and ill-equipped to adequately counsel patients on behaviour modification techniques required to bring about changes in lifestyle, including diet, required for lowering of cardiovascular risk.[128] The inclusion of community health workers (CHW) as part of the health team is being considered in many provinces. In a study which investigated the knowledge, beliefs and attitudes of community health workers about hypertension in the black townships in the Cape Peninsula, an enormous need to train CHWs in the care of patients with hypertension was identified.[124] Many of the CHWs interviewed believed in traditional medicines and home-brewed beer as the best treatment for hypertension.

Optimal control of blood pressure in patients with hypertension requires both adherence to a healthy lifestyle and the regular use of prescribed blood pressure medication. Social and economic factors contribute to the many reasons for lack of blood pressure control in South Africa.[125] Patients frequently do not return to the health-care centres for follow-up visits because of the costs incurred by transport to facilities, as well as the long waiting time involved.

In the late nineties, the Department of Health developed, in a consultative process, guidelines on the prevention management of hypertension, along with similar guidelines for diabetes, hyperlipidaemia and overweight. However, these guidelines were distributed widely in the public health sector without a systematic educational intervention, a prerequisite for change in the practice of health care providers.[126] The guidelines are largely inadequate in their content on lifestyle management related to these conditions and there is evidence that health-care workers do not access these guidelines in the daily management of patients.[123] Two qualitative studies conducted with a purposeful sample of doctors, professional nurses and

pharmacists for hypertension and diabetes management in the public health sector in Cape Town have provided insight into the determinants of care delivered to patients with these conditions.[127,128] Perceived barriers to providing adequate patient care included increasing patient numbers, dramatic budget cuts and acute staff shortages. Staff, particularly nurses, felt inadequately trained and not sufficiently empowered to be effective patient educators and counsellors. Another study in which a validated questionnaire was used to assess the knowledge and practices regarding healthy lifestyles of doctors and nurses in public sector community clinics has identified a poor practical knowledge of nutrition.[129] Added to the problem is a dearth of appropriate and culturally acceptable patient education materials.[123] It is clear that training of health care workers in the non-pharmacological management and prevention of hypertension is urgently required, accompanied by the development of educational aids in the language of the patient group being served (there are 11 official languages in South Africa).

- **Integrated approach**

Our approach (i.e. partnership with the food industry to modify the cation content of commonly consumed foods, as well as promotion of an increased consumption of fermented milk and the possible use of a salt replacement) should be used in conjunction with other policies and strategies to address the promotion of healthy diets. To date, various strategies aimed to eliminate micronutrient deficiencies in the country have been undertaken. The implementation of fortification schemes have been successfully implemented through the Department of Health. The iodation of salt became compulsory in 1995, while the fortification of maize and wheat flour to provide 33 % of the RDA per serving of 8 micronutrients (i.e. vitamin A, thiamine, riboflavin, niacin, folic acid, vitamin B6, iron and zinc) at the point of consumption[130] became compulsory in October 2003. Supplementation programmes that are currently operated through primary care health facilities include vitamin A and iron supplementation.

All health promotion activities need to embrace the principles of the Ottawa Charter for Health Promotion, within the social, economic, and political context of countries.[20] The successful implementation of health promotion materials and programmes at both the population-wide level (blanket coverage) and within the health care services (targeted coverage), requires careful consideration of the local culture and the circumstances of people living in poor settings.

The promotion of the daily consumption of 500 ml fermented milk (*maas*) is a realistic and achievable dietary message. Maas is commonly eaten with soft maize meal porridge by the black population. McCarron & Heaney (2004) generated first-year and fifth-year healthcare cost savings that would accrue if adult Americans simply increased their intake of dairy foods to the currently recommended 3 to 4 servings/day. Using conservative estimates of potential benefit and accumulated data from prospective longitudinal studies and randomized controlled trials, they projected first-year savings of approximately 26 billion dollars and 5-year cumulative savings in excess of 200 billion dollars in terms of outcomes on obesity, hypertension, type 2 diabetes, osteoporosis, kidney stones, certain outcomes of pregnancy, and some cancers.[131]

Conclusion

The group of studies presented in this thesis, which culminated in a randomised controlled trial, have demonstrated that free-living black hypertensive taking anti-hypertensive medication can lower their blood pressure further by consuming their usual foods which have been modified to contain lower levels of sodium and higher levels of potassium, magnesium and calcium. These findings have shown, for the first time in South Africa, that the improvement of the cation content of processed foods, together with the increased consumption of fermented milk, can lower blood pressure. An imperative need for South African food companies to improve staple food products in the country, particularly bread, has been identified. The recommended public health strategy approach also identifies a need for the Department of Health to ensure that the necessary regulations are in place to enforce such improvements and to develop supporting health education messages and activities to accompany the intervention.

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Appendix A: Salt intake questionnaire (N = 42 items)

NUTRITIONAL AND LIFESTYLE HABITS
The following questions are about your dietary and life-style habits. All your answers will be strictly confidential

Office use

Study number: 3

During the **PAST 7 days (1 week)** did you eat any of the following? IF YES, ASK HOW OFTEN
(*if no, circle never*) [DO NOT PROMPT THE ANSWER OPTIONS BELOW]

Food item	***NEVER***	NOT EVERY DAY		EVERY DAY			
		1-3 times per **week**	4-6 times per **week**	1 time a **day**	2 times a **day**	3+ times a **day**	
White bread/ white bread rolls	0	1	2	3	4	5	4
Brown/wholewheat bread/ Rolls	0	1	2	3	4	5	
Breakfast Cereal (processed)	0	1	2	3	4	5	
Breakfast Cereal (weetbix, muesli)	0	1	2	3	4	5	
Crackers (ProVita etc)	0	1	2	3	4	5	
Cookies, biscuits,rusks	0	1	2	3	4	5	
Cake/scone/ muffin/ puddings/pancake/fruit pie/koeksister	0	1	2	3	4	5	
Roti/ samoosa/springroll/doughnut	0	1	2	3	4	5	
Pizza	0	1	2	3	4	5	
Pasta/noodle dishes with cheese sauces (macaroni cheese, lasagne, noodle salad etc.)	0	1	2	3	4	5	
Popcorn	0	1	2	3	4	5	
Crisps (Simba and Niknaks etc.)	0	1	2	3	4	5	
Sausage (wors)	0	1	2	3	4	5	
Polony/salami/bacon/salami/pork suasages (processed meat, cooked, smoked and canned)	0	1	2	3	4	5	
Meat or chicken pies/sausage rolls	0	1	2	3	4	5	
Chicken - battered (KFC etc). and chicken burger only	0	1	2	3	4	5	
Meat and meat dishes (steaks, minced meat, cottage pie, mince, meatballs, stew, bobotie, etc.)	0	1	2	3	4	5	20
Gravy, made with stock or gravy powder	0	1	2	3	4	5	
Biltong/dry wors/bokkems	0	1	2	3	4	5	
Milk (all types, also dairy fruit juice, malted milk, milk shakes)	0	1	2	3	4	5	
Maas	0	1	2	3	4	5	
Cheese	0	1	2	3	4	5	
Yoghurt	0	1	2	3	4	5	
Eggs	0	1	2	3	4	5	
Tinned fish (pilchards/tuna, etc.)	0	1	2	3	4	5	
Other fish and seafood	0	1	2	3	4	5	
Potato chips/french fries and potato salad	0	1	2	3	4	5	
Canned vegetables, incl. Baked beans, tomato paste, sweetcorn, etc.	0	1	2	3	4	5	
Soup (all types)	0	1	2	3	4	5	
Salad dressing/mayonnaise	0	1	2	3	4	5	
Ice cream (all types)	0	1	2	3	4	5	

Margarines, all types, also butter	0	1	2	3	4	5	
Chutney / atchar/chakalaka / Worcester sauce	0	1	2	3	4	5	
Savoury sauces (mushroom, monkey gland, white,cheese)	0	1	2	3	4	5	
Tomato sauce	0	1	2	3	4	5	
Salt	0	1	2	3	4	5	
Aromat / Fondor /mustard	0	1	2	3	4	5	
Peanuts	0	1	2	3	4	5	
Peanut butter	0	1	2	3	4	5	
Marmite/Bovril	0	1	2	3	4	5	
Chocolate sweets and sauce	0	1	2	3	4	5	
Beer and cider	0	1	2	3	4	5	