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Relationship between sodium and potassium intake and blood pressure in a sample of overweight adults

Rhoda Ndanuko

University of Wollongong, rnn954@uowmail.edu.au

Linda C. Tapsell

University of Wollongong, ltapsell@uow.edu.au

Karen E. Charlton

University of Wollongong, karenc@uow.edu.au

Elizabeth Neale

University of Wollongong, elizan@uow.edu.au

Katrina M. O'Donnell

University of Wollongong, kmod202@uowmail.edu.au

See next page for additional authors

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Relationship between sodium and potassium intake and blood pressure in a sample of overweight adults

Abstract

Objective: The aim of this study was to examine the relationship between sodium and potassium intakes and blood pressure (BP) in a clinical sample.

Methods: Secondary analysis of baseline data from 328 participants (mean age: 43.6 ± 8 y, mean body mass index [BMI]: 32.4 ± 4.2 kg/m², mean systolic BP [SBP]/diastolic BP [DBP]: $124.9 \pm 14.5/73.3 \pm 9.9$ mm Hg) of the 12-mo HealthTrack randomized controlled weight loss trial was conducted. Resting BP and 24-h urine sodium and potassium were measured. Dietary intake was evaluated with 4-d food records and self-reported diet histories.

Results: Urinary sodium was positively correlated (Spearman's rho) with SBP ($r = 0.176$; $P = 0.001$) and DBP ($r = 0.150$; $P = 0.003$). The ratio of sodium to potassium was positively correlated with SBP ($r = 0.1$; $P = 0.035$). Urinary sodium ($F [4,323] = 20.381$; $P < 0.0005$; adjusted $R^2 = 0.231$) and sodium-to-potassium ratio ($F[4,323] = 25.008$; $P < 0.0005$; adjusted $R^2 = 0.227$) significantly predicted SBP after controlling for age, sex, BMI, and hypertension medication use. Dietary sodium and potassium significantly predicted urinary sodium ($B = 0.33$, $t = 4.032$, $P < 0.01$) and potassium ($B = 0.67$, $t = 8.537$, $P < 0.01$) excretion, respectively, after adjustment for energy and BMI. Median dietary sodium intake was 3197 mg/d and median dietary potassium intake was 2886 mg/d. Cereal-based products and dishes were the major contributors (22%) to total sodium intake.

Conclusions: In the present study, a high dietary sodium intake and high sodium-to-potassium ratio predicted high SBP. This suggests a need to focus dietary advice on reduction of sources of sodium and increasing sources of potassium in weight loss interventions to improve BP control.

Keywords

adults, relationship, overweight, sodium, potassium, intake, sample, between, blood, pressure

Disciplines

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Authors

Rhoda Ndanuko, Linda C. Tapsell, Karen E. Charlton, Elizabeth Neale, Katrina M. O'Donnell, and Marijka Batterham

The relationship between sodium and potassium intake and blood pressure in a sample of overweight adults

Rhoda N Ndanuko MSc ¹, Linda C Tapsell PhD ¹, Karen E Charlton PhD ¹, Elizabeth P Neale PhD ¹, Katrina M O'Donnell BSc ¹, Marijka J Batterham PhD ²

¹School of Medicine, University of Wollongong, Northfields Avenue, Wollongong NSW 2522, Australia; ²Statistical Consulting Service, School of Mathematics and Applied Statistics, University of Wollongong, Northfields Avenue, Wollongong NSW 2522, Australia

To whom correspondence should be addressed: Rhoda Ndanuko, School of Medicine, University of Wollongong, Northfields Avenue, Wollongong NSW 2522, Australia. Email: rnn954@uowmail.edu.au

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Abbreviations used: BP, blood pressure; BMI, body mass index; DBP, diastolic blood pressure; NHMRC, National Health and Medical Research Council; SBP, systolic blood pressure; WHO, World Health Organization

ABSTRACT

Objective: The aim of this study was to examine the relationship between sodium and potassium intakes and blood pressure (BP) in a clinical sample.

Methods: Secondary analysis of baseline data from 328 participants [mean age: 43.6±8.0 years, mean BMI: 32.4±4.2 kg/m², mean systolic BP (SBP)/diastolic BP (DBP): 124.9±14.5/73.3±9.9 mmHg] of the 12-month HealthTrack randomized controlled weight loss trial was conducted. Resting BP and 24-h urine sodium and potassium were measured. Dietary intake was evaluated with 4-day food records and self-reported diet histories.

Results: Urinary sodium was positively correlated (Spearman's rho) with SBP ($r=0.176$, $P=0.001$) and DBP ($r=0.150$, $P=0.003$). Sodium-to-potassium ratio was positively correlated with SBP ($r=0.1$, $P=0.035$). Urinary sodium [$F(4,323)=20.381$, $P<0.0005$; adjusted $R^2=0.231$] and sodium-to-potassium ratio [$F(4,323)=25.008$, $P<0.0005$; adjusted $R^2=0.227$] significantly predicted SBP after controlling for age, sex, BMI and hypertension medication use. Dietary sodium and potassium significantly predicted urinary sodium ($B=0.33$, $t=4.032$, $P<0.01$) and potassium ($B=0.67$, $t=8.537$, $P<0.01$) excretion respectively after adjustment for energy and BMI. Median dietary sodium intake was 3197 mg/day and median dietary potassium intake was 2886 mg/day. Cereal based products and dishes were the major contributors (22%) to total sodium intake.

Conclusions: In our sample, a high dietary sodium intake and high sodium-to-potassium ratio predicted high SBP. This suggests a need to focus dietary advice on reduction of sources of sodium and increasing sources of potassium in weight loss interventions in order to improve BP control.

Keywords: blood pressure, food sources, obese, potassium, sodium, urinary excretion

INTRODUCTION

High blood pressure (BP) or hypertension is the major single risk factor for cardiovascular disease [1]. Hypertension affects about 1 billion people globally and its prevalence is predicted to reach 29% (1.56 billion) by 2025 [2]. It is estimated that a population-level reduction of 5.0 mmHg in systolic blood pressure (SBP) and 3.0 mmHg in diastolic blood pressure (DBP) would reduce the incidence of coronary heart disease and stroke in the US by 15% and 27%, respectively [3]. The link between obesity, BP and cardiovascular disease is well established. In prospective studies, greater hospitalisations due to different types of cardiovascular disease have been observed in patients with a higher BMI [4]. The presence of various co-morbidities therefore warrants a holistic approach in their management and prevention.

From a preventive health perspective, lifestyle changes have been recommended to lower BP. These include reducing dietary sodium, following a healthy diet such as the Dietary Approaches to Stop Hypertension diet and engaging in regular physical activity [5]. Evidence supporting dietary approaches is provided in meta-analyses that have assessed the effect of sodium and potassium intake on BP. For example, reducing sodium intake to about 1720 mg/day led to a reduction in BP by 2.0 and 1.0 mmHg in SBP and DBP, respectively, in normotensive subjects. Greater reductions of 5.0 and 3.0 mmHg in SBP and DBP, respectively, were observed in hypertensive subjects [6]. On the other hand, increasing potassium intake by 780 mg/day without the use of antihypertensive medications reduced SBP and DBP by 4.9 mmHg and 2.7 mmHg, respectively [7]. The ratio of sodium to potassium may better reflect desirable food choices for lowering BP across various geographical regions and cultures [8] but food patterns that reflect an optimal ratio have not been adequately identified.

Due to the relationship between excessive sodium intake and low potassium intake with hypertension and cardiovascular disease, the WHO recommends a population intake of 2000 mg of sodium and 3510 mg of potassium per day [9]. The Australian National Health and Medical Research Council (NHMRC) further recommends a Suggested Dietary Target of 1600 mg of sodium for chronic disease prevention [10] especially since there is an increase of salt sensitivity in individuals with metabolic syndrome [11]. Despite these recommendations, excessive sodium consumption and low potassium intakes have been previously reported [12]. Food sources of sodium may vary according to the dietary habits of different cultures. In the INTERMAP study, major food sources of sodium included bread, grains and cereals in the United States and United Kingdom; added salt in China; and soy sauce, processed fish/seafood and salted soups in Japan [13].

There are few studies examining the relationship between sodium and potassium intakes and BP specifically in clinical overweight populations, whereby exposing the details of dietary-BP relationships may be informative for primary healthcare practice. The aim of this study was to examine the association between sodium and potassium intakes and BP in a sample of overweight and obese adults volunteering for a weight loss trial. To address issues of translating dietary advice to practice we also report on sodium and potassium intake as well as major food sources of these nutrients in the context of the clinical trial.

METHODS

The current study is a cross-sectional secondary analysis of baseline data from the 12-month HealthTrack randomized controlled trial, an interdisciplinary lifestyle intervention investigating whether a novel lifestyle intervention is more effective than usual care in achieving weight loss in overweight/ obese adults. The Healthtrack study was approved by the University of Wollongong/ Illawarra Shoalhaven Local Health District Human Research

Ethics Committee (HE13/189) including this analysis. The trial commenced in May 2014 and all participants provided their informed written consent to participate. The HealthTrack study was registered with the Australian and New Zealand Clinical Trial Registry (ANZCTRN 12614000581662). For this analysis, the primary outcomes were SBP and DBP and secondary outcome was BMI.

Study context: The HealthTrack Trial

Detailed study protocol is reported elsewhere [14]. In brief, the HealthTrack study enrolled adults aged between 25 and 54 years recruited through advertisements in local newspapers from the Illawarra community, a major coastal region 70 km south of Sydney, Australia. Subjects were included if they were permanent residents of the Illawarra region and had a BMI of 25-40 kg/m². Exclusion criteria included subjects that were unable to communicate in English, had severe medical conditions impairing ability to participate in the study, immunodeficiency, reported illegal drug use or regular alcohol intake (>50 g/day) associated with alcoholism, or difficulties or major impediments to participating in the components of the study.

Systolic and diastolic BP was measured in the supine position after 5 minutes of rest using an automatic BP monitor (OMRON BP-203RPE III, OMRON HEALTHCARE Co. Ltd, Kyoto, Japan) using standard techniques. All BP measurements were performed by trained health practitioners.

Participants were instructed to collect 24-h urine for estimation of sodium and potassium excretion. Detailed instructions were provided whereby participants discarded the first urine of the day and collected the rest over the 24 hours in standard plastic containers that were provided. The collected samples were delivered to Southern IML pathology and were stored upon receipt at 2-8 degrees. The total volume of urine was measured and recorded. Sodium

and potassium concentrations were determined by indirect ion-specific electrodes whilst the creatinine concentration was determined using the Jaffe reaction colorimetric method [15]. Samples that had a total volume of less than 500 mL ($n = 1$) and/or creatinine levels less than 6.0 mmol/d in volumes less than 1000 mL ($n = 1$) were excluded as they were classified as incomplete [16].

Dietary intake was assessed by using 4-day food records (including one weekend day) whereby participants recorded all foods and drinks consumed including amounts and recipes. In addition, Accredited Practising Dietitians assessed participants' dietary intake through a self-reported diet history interview. Dietary data were entered into the FoodWorks nutrient analysis software program (Xyris software, FoodWorks. 2012: Brisbane, Australia).

Anthropometric measurements were conducted by trained health practitioners. Lightly clad body weight was measured on digital scales [Tanita scales, Tanita Corporation, Tokyo, Japan, UM0703581(1)] to the closest 0.1 kg and percent body fat via bioelectrical impedance recorded to the closest 0.1 %. Height was measured using a wall-mounted stadiometer rounded to the nearest millimetre in accordance with the established anthropometric protocols [17]. Widest part of hip and narrowest waist circumference were measured in accordance with the reported acceptable protocols [17]. BMI was calculated as body weight in kilograms divided by the square of the height in metres.

Dietary data was originally analysed using AUSNUT2007 [18], which was the most recent food composition database at the time of study commencement. The recent release of AUSNUT2011-2013 [19] necessitated dietary data be categorized according to the AUSNUT2011-2013 food groups. AUSNUT2007 foods were updated to AUSNUT 2011-2013 using a systematic process described elsewhere [20].

Statistical analysis

For this secondary analysis of the Healthtrack data, statistical analysis was performed using the Statistical Package for the Social Sciences (IBM Corp., SPSS for Windows Version 21. 2012: New York, USA). Normality testing was conducted using Shapiro-Wilk test and data was log transformed where possible. For sodium and potassium intake, medians were used as they are less influenced by extreme values and skewness compared to means. Spearman's rank order correlation was performed to assess the relationship between urinary sodium and potassium excretion and sodium-to-potassium ratio with BP. Stepwise forward multiple linear regression was performed to assess prediction of BP by sodium intake, potassium intake and sodium-to-potassium ratio and prediction of urinary sodium and potassium by dietary sodium and potassium. Covariates that were controlled for include age, sex, BMI and antihypertensive medication. Initial analysis to ensure no violations of the assumptions of normality, linearity, multicollinearity and homoscedasticity were conducted. Data are expressed as mean (standard deviation) unless otherwise stated and as a percentage for the food group contribution of sodium and potassium. Data was considered statistically significant when $P < 0.05$.

RESULTS

Baseline characteristics

A total of 377 participants, predominantly Australian born (82%), were randomised to the HealthTrack study. For the present analysis we included data from 328 participants who had complete dietary data, BP data and 24-h urine collections. The main characteristics of the participants are reported in Table 1. 26% of the participants (n=85) were hypertensive and of this 46 were taking antihypertensive medication. Median urinary sodium concentrations indicated that dietary intakes were above the recommended targets set by the NHMRC [10]

and WHO [9]. Table 2 illustrates the number of participants who were compliant with the NHMRC and WHO guidelines for sodium and potassium intake.

Table 1

Characteristics of the analysis sample¹

Characteristic	Mean (SD)
Male/ female, % (n)	27/73 (89/238)
Age, years	43.6 (8.0)
Height, cm	1.7 (0.1)
Weight, kg	91.7 (15.1)
BMI, kg/m ²	32.4 (4.2)
Waist circumference, cm	103.4 (11.8)
Blood pressure	
Systolic, mmHg	124.9 (14.5)
Diastolic, mmHg	73.3 (9.9)
Hypertensives, % (n)	26 (85)
Urinary excretion	
Volume, ml/day	2029 (879)
Creatinine, mmol/day	8.2 (4.5)
Median sodium, mg/day, (IQR)	3197 (2282-4140)
Median potassium, mg/day, (IQR)	2886 (2223-3549)
Median sodium-to-potassium ratio, (IQR)	1.9 (1.5-2.4)
Median salt intake, g/day (IQR)	8.2 (5.9-10.6)
Dietary intake (4-d food records)	
Median sodium, mg/day, (IQR)	2682 (2084-3439)
Median potassium, mg/day, (IQR)	3124 (2680-3825)

¹Data are presented as mean (standard deviation), unless otherwise stated; Participants were diagnosed as hypertensive if blood pressure was $\geq 140/90$ mmHg and/or taking antihypertensives; IQR, Interquartile range.

Table 2

Number (%) meeting sodium and potassium urinary excretion targets defined in NHMRC and WHO guidelines¹

Target value	Number (%)
24-h sodium excretion	
≤6 g salt/day (2300 mg Na/day) ^a	86 (26.2)
≤5 g salt/day (2000 mg Na/day) ^b	50 (15.2)
≤4 g salt/day (1600 mg Na/day) ^c	14 (4.3)
24-h potassium excretion	
≥ 97 mmol/day, men (<i>n</i> = 89) ^e	32 (36.0)
≥ 72 mmol/day, women (<i>n</i> = 239) ^f	111 (46.4)
≥ 90 mmol/day ^b	90 (27.4)

¹Data presented for *n* = 328 unless otherwise stated; NHMRC, National Health and Medical Research Council; WHO, World Health Organization. ^aNHMRC upper level; ^bWHO population target; ^cNHMRC suggested dietary target; ^eNHMRC target for men; ^fNHMRC target for women

Food group contributions to total dietary and potassium intake

Percentage contributions of sodium and potassium by the AUSNUT2011-13 major food groups were determined (Table 3). Overall, cereal based products and dishes; cereal and cereal products; meat, poultry and game products and dishes; and milk products and dishes were the major contributors for dietary sodium. On the other hand, vegetable products and dishes; meat, poultry and game products and dishes; and milk products and dishes were the major contributors for dietary potassium intake.

1 **Table 3**

2 Percentage contribution of major food groups to sodium and potassium intake among clinically overweight adults

Food group ^a	Sodium		Potassium	
	% Sodium intake	Median (IQR) ^b mg/day	% Potassium intake	Median (IQR) mg/day
Cereal based products and dishes (biscuits, cakes, pastries)	21.61	527 (247-878)	7.37	206(100-343)
Cereal and cereal products (breads, rice, pasta, breakfast cereals)	14.53	384 (251-529)	7.78	231 (149-326)
Meat, poultry and game products and dishes	17.57	393 (203-709)	15.58	456 (307-704)
Milk products and dishes (milk, yoghurt, cheese, custard)	10.59	247 (141-418)	12.74	356 (181-575)
Savoury sauces and condiments	8.69	176 (70-333)	1.68	26 (8-76)
Vegetable products and dishes	4.72	100 (46-181)	22.02	682 (426-930)
Soup	4.27	282 (179-563)	1.93	158 (87-282)
Fish and seafood products and dishes	4.01	134 (77-262)	2.69	128 (65-217)
Non-alcoholic beverages (tea, coffee, soft drinks, juices)	3.11	66 (31-117)	9.51	254 (109-444)
Miscellaneous (yeast, herbs, spices, seasonings)	3.02	76 (23-234)	0.85	16 (3-69)
Snack foods	1.89	76 (38-155)	1.62	58 (22-155)
Legume and pulse products and dishes	1.29	124 (56-201)	0.84	81 (52-129)
Fats and oils	1.13	22 (7-51)	0.04	1 (0.3-2)
Egg products and dishes	0.86	27 (14-54)	0.67	29 (16-50)
Confectionery and cereal/nut/fruit/seed bars	0.75	22(7-41)	1.88	62 (28-123)
Alcoholic beverages	0.65	27 (13-54)	2.08	102 (43-179)
Seed and nut products and dishes	0.60	6 (1-38)	2.00	71(37-147)
Dairy & meat substitutes (soy based beverages and soups)	0.25	22 (3-69)	0.51	62 (26-238)
Special dietary foods	0.17	37 (24-59)	0.23	60(0-133)
Fruit products and dishes	0.15	3 (1-6)	7.74	231 (112-404)
Sugar products and dishes	0.14	1 (0.1-4)	0.24	4 (1-14)

3 ^aUsing AUSNUT2011-13 major food groups; ^bIQR, interquartile range.

Prediction of urinary sodium and potassium by dietary intake

A multiple linear regression model established that reported dietary sodium via the 4-day food records significantly predicted urinary sodium excretion adjusted for energy and BMI. The overall model was significant, $F(21.8, 3df) P < 0.01$ accounting for 16.0% of the explained variability in urinary sodium (Figure 1). Dietary sodium was a significant predictor, $B = 0.334$, $t = 4.032$, $P < 0.01$. Likewise, the overall model for predicting urinary potassium from dietary potassium adjusted for energy and BMI was significant, $F(29.1, 3df)$, $p < 0.01$ accounting for 20.5% of the explained variability in urinary potassium (Figure 2). Dietary potassium was a significant predictor, $B = 0.67$, $t = 8.537$, $P < 0.01$. In both sodium and potassium analysis, there was no evidence of multicollinearity and the residual diagnostics indicated the models were a good fit. Dietary intake assessed via 4-day food records showed a stronger relationship with urinary sodium and potassium compared to diet histories (data not shown).

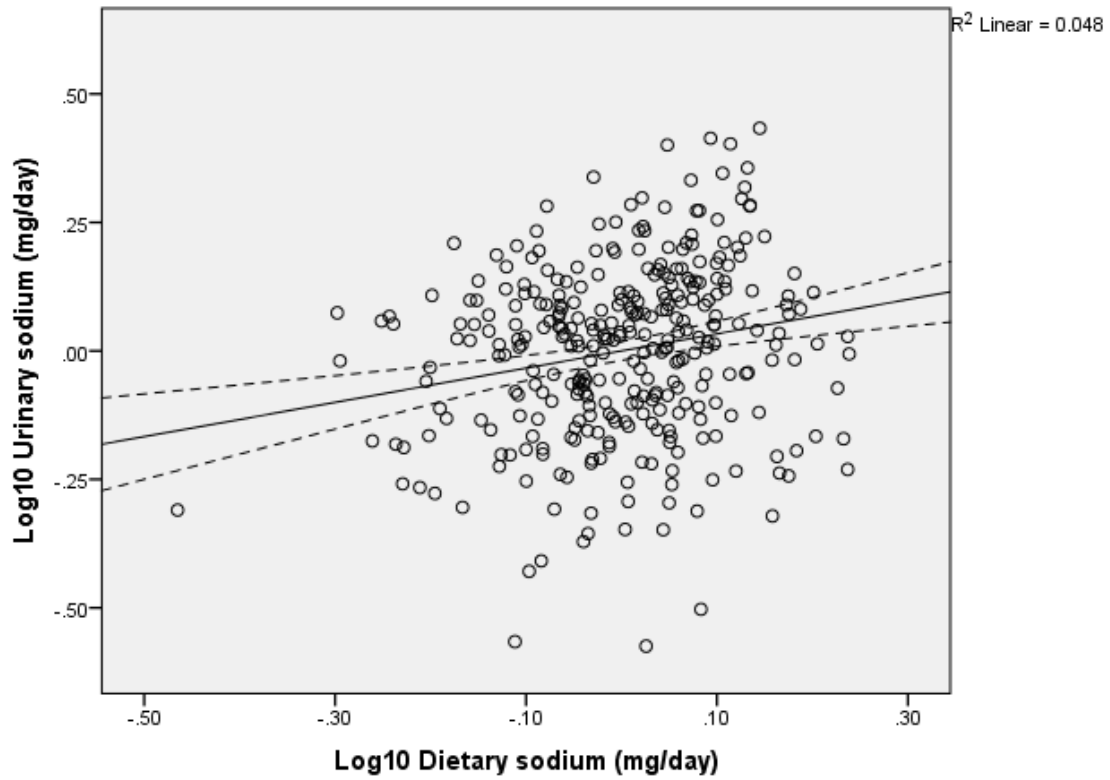


Figure 1

Relationship between dietary sodium and urinary sodium excretion. The solid line represents the regression best of line of fit while the dotted lines indicate the 95% confidence limits for mean predicted values.

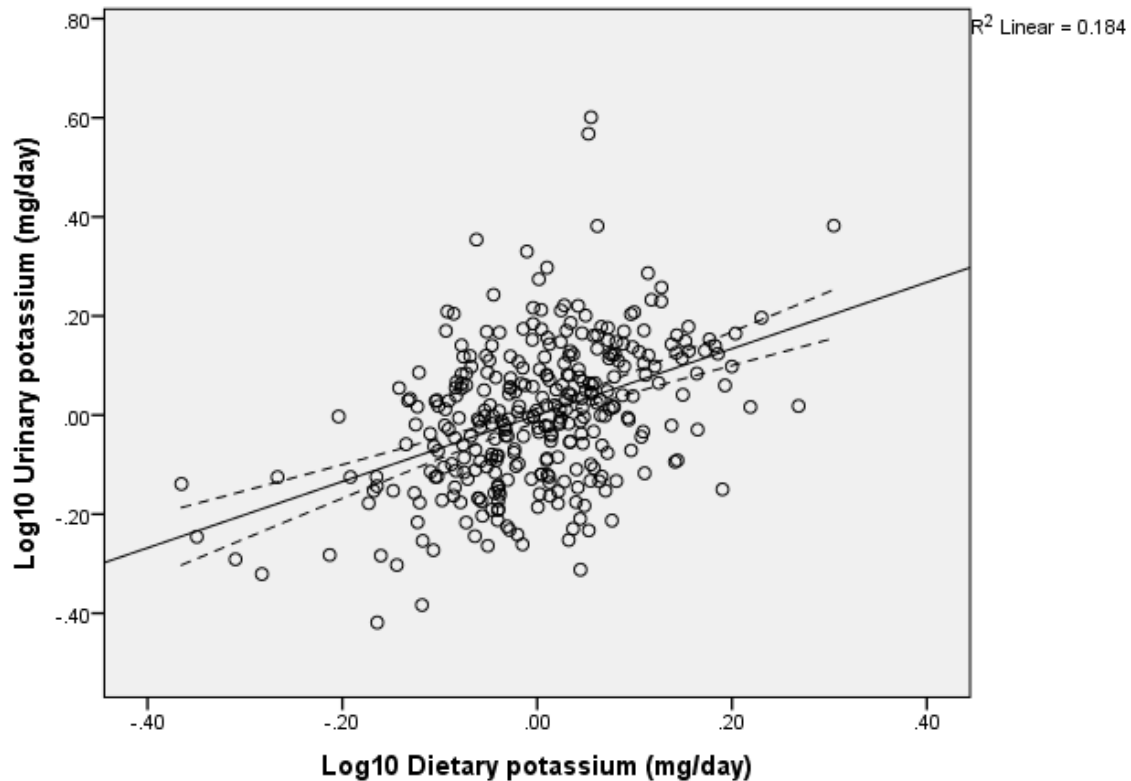


Figure 2

Relationship between dietary potassium and urinary potassium excretion. The solid line represents the regression best of line of fit while the dotted lines indicate the 95% confidence limits for mean predicted values

Relationship between sodium, potassium and sodium-potassium-ratio and blood pressure

Urinary sodium was positively correlated with SBP ($r = 0.176$, $P = 0.001$) and DBP ($r = 0.150$, $P = 0.003$). Sodium-to-potassium ratio was positively correlated with SBP ($r = 0.1$, $P = 0.035$). In multiple linear stepwise regression, urinary sodium [(F (4,323) = 20.381, $P < 0.0005$; adjusted $R^2 = 0.231$)] and sodium-to-potassium ratio [(F (4,323) = 25.008, $P < 0.0005$; adjusted $R^2 = 0.227$)] were identified as significant predictors of SBP after

controlling for age, sex, BMI and hypertension medication use. Likewise, dietary potassium was a significant predictor of DBP [(F (4,323) = 28.059, $P < 0.0005$; adjusted $R^2 = 0.249$)] (Table 4). No statistically significant associations were observed between urinary potassium and SBP, urinary sodium and sodium-to-potassium ratio and DBP, dietary sodium and dietary potassium and SBP, and dietary sodium and DBP.

Table 4

Stepwise regression analyses using SBP and DBP as the dependent variables¹

Variable	<i>B</i>	<i>SE B</i>	<i>Standardized B</i>	<i>P</i> value
SBP:				
Age	0.002	0.0003	0.367	<0.001
BMI	0.003	0.001	0.242	<0.001
Urinary sodium	0.037	0.014	0.118	0.02
Na:K ratio	0.033	0.014	0.118	0.02
DBP:				
Age	0.003	0.0004	0.358	<0.001
Sex	-0.031	0.006	-0.232	<0.001
BMI	0.003	0.001	0.230	<0.001
Dietary potassium	-0.051	0.024	-0.104	0.035

¹Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; Na:K, sodium-to-potassium ratio; SBP, systolic blood pressure; B, β standardized coefficient. Only statistically significant variables are shown; variables excluded include urinary potassium and dietary sodium.

DISCUSSION

This secondary analysis of baseline data from a weight loss clinical trial confirmed that the relationship between dietary sodium intake and sodium-to-potassium ratio as analysed using the 24h sodium excretion, and SBP can be observed within an overweight clinical sample. In addition, age and BMI significantly predicted SBP, while age, sex and BMI significantly predicted DBP. Dietary potassium as measured using food records was also significantly associated with DBP. There was a marked difference between urinary and dietary sodium and potassium intakes. It has been shown that there is a tendency for subjects with a higher BMI to under-report their dietary sodium intake and over-report dietary potassium intake [21]. Moreover, the discrepancy between estimations of reported dietary intake and urinary excretion of sodium in our sample may be an indication of discretionary salt intake.

Our study focused on a clinical obese population that was participating in a weight loss trial. About 26% of the participants were hypertensive with more than half taking anti-hypertensive medication. Lifestyle interventions such as consuming a healthy diet (such as the DASH diet and reduced sodium intake), weight loss and regular physical activity, are some of the strategies recommended for BP management [5]. In the present study, we observed a significant association between sodium intake, sodium-to-potassium ratio and SBP. This is in line with findings from a previous Australian study whereby sodium intake (3567 mg/d) and sodium-to-potassium ratio (1.99) were both positively associated with SBP [22] though the sample population was older (mean age: 64 y) and less overweight (mean BMI: 28 kg/m²) in comparison to our relatively young study population. Although dietary potassium was significantly associated with DBP in the present study, no association was found with urinary potassium. The higher dietary potassium could have been due to over-reporting of fruit and vegetable intake which has been observed in epidemiological studies [23]. In our study, the sodium-to-potassium ratio (1.9) exceeded the WHO recommendation

of a sodium-to-potassium molar ratio of 1:1 [24]. A recent review demonstrated that in various randomized controlled trials the sodium-to-potassium ratio had a stronger association with BP than sodium or potassium alone [25]. In the development of hypertension, excessive sodium and insufficient potassium intake are both shown to play a role since they result in vascular smooth muscle cell contraction which leads to increased peripheral vascular resistance thus causing high BP [26].

The major food sources of sodium that were identified in our study included cereal-based products and dishes; cereal and cereal products; meat, poultry and game products and dishes; and milk products and dishes. These four food groups provided more than 60% of the total sodium intake. This is comparable to the recent 2011-2012 population based cross-sectional Australian Health Survey (AHS) which found that cereal-based products and dishes (25%), cereal and cereal products (18%) and meat and poultry (18%) were the major sources of sodium [27]. Similar results have been found in other studies in Finland [16] and South Africa [28]. In contrast, sources of sodium may vary in other countries depending on the major foods consumed and cultural context. For instance, in Japan, most of the dietary sodium is derived from soy sauce, salted soups, processed fish/ seafood and preserved vegetables [13]. While we did not analyse meal patterns and their sodium contribution to the total daily intake, a previous Australian study found that lunch had the highest sodium density compared to dinner and breakfast [29]. Our study did not assess the use of discretionary salt, but in low and low-middle income countries, this has been found to be a significant source of sodium which may account for up to 76% of total intake in China [13].

There are a limited number of studies that have assessed food sources of potassium. We identified vegetable products and dishes; meat, poultry and game products and dishes; and milk products and dishes as major contributors, accounting for half of total potassium intake.

These results are similar to the nationally representative Australian Health Survey whereby vegetable products and dishes accounted for 17.5% of total potassium intake [27].

Nutrient intakes, including sodium and potassium, are contributed from combinations of food groups that are consumed within the context of dietary patterns and cuisines. In a recent meta-analysis, we demonstrated that consumption of diets that are rich in fruit, vegetables, wholegrains, legumes, seeds, nuts, fish and dairy, and that are low in meat, sweets and alcohol, significantly lowered SBP and DBP by 4.26 mmHg and 2.38 mmHg, respectively [30]. We have also identified specific dietary patterns that are related to BP in another of our clinical trial datasets in an entirely different population of obese adults attending for weight loss. Dietary patterns that included larger amounts of fruits & nuts and/or seafood were associated with lower BP at baseline, while patterns that were characterised by yeast extract & seasonings were associated with higher BP [31]. Hence, diets that are high in potassium and low in sodium are shown to be beneficial in lowering BP in clinical populations.

While large cohort studies have provided in principle evidence of the relationship between dietary sodium and potassium with BP [32], our analysis of data from a clinical trial provides a novel confirmation that the relationship between dietary sodium and SBP can still be observed in a relatively small sample of an at risk group. Importantly, the analysis identifies the food sources of sodium and potassium that would form the basis of dietary counselling in these settings.

The major strength of our study includes the use of 24-h urine excretion to estimate sodium and potassium intake given the recognised problems with underreporting and over-reporting of sodium and potassium respectively [21]. About 90% of the sodium or potassium consumed is excreted in the urine, thus making it a good indicator of dietary intake [33] provided that a complete 24hr sample can be collected. We ascertained completeness of collections by use of

urinary creatinine concentrations. On the other hand, the use of a single 24-h urinary sample may be considered a limitation because of large day-to-day variability [34]. Greater accuracy of sodium and potassium habitual intake would be obtained from repeated 24-h urinary collections [34]. Dietary assessment remains important as it assists in identifying food sources of sodium and potassium which may be useful in making recommendations in health education [33]. Another limitation is that the current study is a secondary analysis of a randomized controlled trial which was designed to answer a different question from the one addressed in this study and there is a possibility of the analysis being underpowered. In addition, our dataset was not large enough to group participants according to their level of sodium and potassium intake though we have reported the number of participants meeting the sodium and potassium targets. Our participants were an overweight clinical population from a regional area of New South Wales, Australia, who had volunteered to participate in a 12-month weight loss trial. The results may therefore not be generalizable to the wider adult population. In addition, the study participants were about 70% female. It has been shown that females are more likely to seek primary care services compared to males especially in the age group between 20 and 44 years [35].

Conclusions

This study has confirmed that the relationship between dietary sodium and sodium-to-potassium ratio and SBP can be observed in a clinical sample of overweight adults. In addition, dietary potassium as measured through food records was associated with DBP. While much of the evidence for this relationship is translated into population health messages, the analysis suggests there is good reason to translate this advice to clinical practice for patient groups such as overweight adults. Importantly, the identification of food sources of sodium and potassium in the usual diet enables a direct pathway to practice, with food based dietary advice specifically targeting changes for improved BP regulation.

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Author contributions

Conception and design of the study: LCT, KEC, EPN and MJB. Generation, collection, assembly, analysis and/or interpretation of data: RNN, KMO and MJB. Drafting and revision of the manuscript: RNN, LCT, KEC, EPN and MJB. All authors read and approved the final manuscript.

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