Relationship of urinary sodium and sodium-to-potassium ratio to blood pressure in older adults in Australia

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ABSTRACT

Objective: To assess the relationship between dietary sodium intake, as measured by urinary electrolyte excretion, and blood pressure within a population of older Australian adults.

Design, setting and participants: A cross-sectional study of adults enrolled in the Melbourne Collaborative Cohort Study, stratified by sex, country of birth (Italy, Greece, Australia/New Zealand) and age (50–59 and 60–75 years). Blood pressure measurements were taken in 2003–2007 and 24-hour urine collections in 2007–2008.

Main outcome measures: 24-hour urinary excretion of sodium and potassium, urinary sodium-to-potassium ratio, and clinic blood pressure measurement.

Results: The mean ±SD age of 783 participants was 64.0 ±6.3 years. Mean ± SD urinary sodium was 155.1 ±63.1 mmol/day (8.9 ±3.6 g salt/day), urinary potassium was 82.3 ±27.9 mmol/day, and urinary sodium-to-potassium ratio was 1.99 ±0.83. In the 587 participants with blood pressure measurements, urinary sodium and the sodium-to-potassium ratio were both associated with systolic blood pressure in all adjusted and unadjusted models (mmHg change per 100 mmol/day increase in sodium: regression coefficient, 2.3, 95% CI, 0.1–4.6; $P = 0.049$, adjusted for age, sex, body mass index, country of birth and antihypertensive medication use).

Conclusion: This study has demonstrated, for the first time within an Australian population sample of older adults, that sodium intake is positively associated with blood pressure. These results suggest that a population-wide reduction in sodium intake could be effective in reducing blood pressure in adults in Australia.

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Raised blood pressure (BP) is one of the most common and preventable risk factors for cardiovascular disease. It has been estimated that 33% of the Australian adult population have established hypertension,[1] and that hypertension contributes 42% of the cardiovascular disease burden in Australia.[2] There is compelling evidence across countries that total dietary sodium intake and sodium-to-potassium ratio (assessed by 24-hour urine collection, which provides an objective measure of total dietary sodium intake) are associated with an age-related rise in BP.[3] Few studies have demonstrated an effect within populations with relatively high sodium intakes.[4,5] Urinary potassium, reflecting dietary intake, has been shown to have an independent inverse association with BP.[6] A large prospective study and a randomised controlled trial have shown that high urinary sodium or a high urinary sodium-to-potassium ratio increases the risk of cardiovascular events and mortality[7-9] independently of the effects on BP.

There are limited data on sodium intake in Australia assessed using 24-hour urine collection. In 1995, a small population-based sample indicated mean urinary sodium excretion of 170 mmol/day (9.8 g salt/day) for 87 men and 118 mmol/day (6.8 g salt/day) for 107 women,[10] and small convenience samples have reported excretion of 105–210 mmol/day (6–12 g salt/day).[11-16]

We assessed 24-hour urinary sodium and potassium excretion and examined the relationship to BP in a cross-section of middle-aged and older Australian adults participating in the Melbourne Collaborative Cohort Study (MCCS), a large prospective study investigating the role of lifestyle factors in the development of common chronic diseases.

METHODS

Study population

From 1990 to 1994, the MCCS recruited 41 514 people (17 045 men) aged 27–75 years, including 5411 migrants from Italy and 4525 from Greece. Details of design, recruitment, and study procedures have been previously described.[17,18] From 2003 to 2007, 26 918 (78% of those alive and contactable) participated in a follow-up survey that included measurement of BP and a detailed medication audit.

In 2007–2008, we recruited participants from among 13 130 eligible MCCS participants residing in the Melbourne area to calibrate a new food frequency questionnaire, which included a 24-hour urine collection. Participants were randomly selected and stratified by sex, country of birth (Italy, Greece, Australia/New Zealand) and age (50–59 and 60–75 years). A total of 1771 participants were invited to participate, with equal samples selected in each stratum, except for participants born in Greece, where the eligible sample sizes were smaller.

The Cancer Council Victoria's Human Research Ethics Committee approved the study protocol (HREC 0703), and all participants gave written informed consent.

Anthropometry

At baseline (1990–1994), height was measured using a stadiometer.[18] Weight was measured during the follow-up survey, at the same time as the BP measurement, by a trained investigator using an electronic scale (HW-200GL and UC-321 Personal Precision Health Scale, A&D Mercury, Adelaide, SA). Body mass index (BMI) was calculated as weight/height^2 (kg/m^2).

Urinary sodium and potassium

In 2007–2008, participants were given written and verbal instructions for the 24-hour urine collection. The first urine of the day was discarded, and all urine over the following 24 hours, including the first urine of the following day, was collected in the bottles provided. Total volume of the collection was measured, the samples were mixed and aliquots taken and frozen at −80° C. In 2009, the samples were thawed and analysed for creatinine using the Jaffe reaction, and sodium and potassium concentrations were determined using ion-selective electrodes (Gribbles Pathology, Melbourne, Vic). Inaccurate urine collections (ie, urinary creatinine < 5.0 mmol/day for women and < 6.0 mmol/day for men, and a 24-hour urine collection of < 1 L), and extreme outliers for urinary creatinine (> 3 SD from the mean) were excluded.

To determine discretionary salt use, participants were telephoned and asked: "Do

you use salt in cooking?" and "Do you add salt at the table, ie, to food?".

BP and antihypertensive medication

BP measurements were obtained from a retrospective audit of the MCCS 2003–2007 follow-up survey. Some participants were missing BP measurements; reasons for this included follow-up interviews being conducted outside the research centre, machine failure, discomfort and medical reasons. Three consecutive BP measurements were taken using a Dinamap 1846SX automatic BP monitor (Critikon, Tampa, Fla, USA). The average of the second and third BP measurements was used in analysis. Participants had also provided information on the use of prescription medication, including antihypertensive drugs. Participants were classified as hypertensive according to the World Health Organization definition if they had a systolic BP (SBP) ⩾ 140 mmHg or a diastolic BP (DBP) ⩾ 90 mmHg or were currently taking antihypertensive medication.[19]

Statistical analysis

Statistical analyses were performed using SPSS version 17.0 for Windows (SPSS Inc, Chicago, Ill, USA). $P < 0.05$ was considered significant. Sample characteristics were compared between sexes using a Student *t* test or a χ^2 test for categorical data. Standard linear regression was used to assess associations between sodium, potassium and BP, with covariates of BMI, sex, age, country of birth and antihypertensive medication use. Adjusted means of sodium intake were analysed using a Student *t* test (sex and hypertension status), or one-way analysis of variance (country of birth). Binary logistic regression was used to estimate odds ratios (ORs).

RESULTS

Estimated sodium and potassium intakes

Of the 1771 MCCS participants invited in 2007–2008, 959 (54%) participated in this study, and 793 provided a 24-hour urine sample; 10 of these were excluded because of inaccurate urine collection or missing data. Characteristics of the 783 participants who provided complete 24-hour urine collections are shown in Box 1. The mean ± SD urinary sodium was 155.1 ± 63.1 mmol/day (8.9 ± 3.6 g salt/day), urinary potassium was 82.3 ± 27.9 mmol/day, and urinary sodium-to-potassium ratio was 1.99 ± 0.83. Compared with men, women had lower urinary sodium (mean ± standard error of the mean, 133.7 ± 2.5 v 178.4 ± 3.4 mmol/day; $P < 0.001$) and potassium (77.0 ± 1.2 v 88.1 ± 1.6 mmol/day; $P < 0.001$). Adjusting for age, BMI and hypertension did not significantly alter these results (Box 2). Only six men (1.6%) and 32 women (7.9%) (4.9% total) conformed with the Australian recommendation for chronic disease prevention of sodium intake of < 70 mmol/day (< 4 g salt/day).[20] Intake at or below the recommended upper limit of 100 mmol/day (6 g salt/day) was achieved by 41 men (10.9%) and 125 women (30.7%) (21.2% total).

Participants born in Australia/New Zealand had lower urinary sodium excretion compared with those born in Italy or Greece, after adjusting for BMI, age and sex (Box 2). Urinary potassium excretion was unfavourably lower in those born in Australian/New Zealand compared with those born in Greece, although the sodium-to-potassium ratio was also lower.

Discretionary salt use

Relative to participants born in Australia/New Zealand, those born in Italy and Greece were more likely to add salt in cooking (OR, 20.6; 95% CI, 9.7–43.5; $P < 0.001$ and OR, 36.2; 95% CI, 13.1–100.1; $P < 0.001$, respectively, after adjusting for sex and age). Use of salt in cooking was associated with a higher daily sodium excretion (157.5 ± 2.2 v 142.5 ± 5.6 mmol/day; $P = 0.016$) and a higher sodium-to-potassium ratio (2.0 ± 0.03 v 1.8 ± 0.1; $P = 0.007$), after adjusting for BMI, age, sex and country of birth. Relative to those born in Australia/New Zealand, participants born in Italy were less likely to add salt at the table (OR, 0.6; 95% CI, 0.4–0.9; $P = 0.007$).

Correlates of BP

BP measurements were available for 587 of the 783 participants with urine samples (75%) (Box 1). The BP measurements were made on average 2 years and 11 months before the urine samples were collected. Sodium excretion and sodium-to-potassium ratio were both positively associated with SBP after adjusting for age and sex (Box 3, Model A), but there was no significant association between potassium and SBP. Sodium excretion and sodium-to-potassium ratio remained significantly and positively associated with SBP in all adjusted models (Box 3, Models B–D). In the fully adjusted model, these covariates together contributed to 20% of the variance in SBP ($R^2 = 0.198$ for urinary sodium; $R^2 = 0.200$ for sodium-to-potassium ratio). There was no association with DBP and urinary excretion of sodium or potassium (Box 3).

SBP (adjusted for age and sex) was not significantly different between participants born in Australia/New Zealand, Italy and

1 Characteristics of 783 participants with complete 24-hour urine collections

Characteristic	Women (*n* = 407)	Men (*n* = 376)
Age, mean (SD)	63.7 (6.1)	64.3 (6.6)
Body mass index (kg/m^2), mean (SD)	28.0 (4.9)	28.4 (4.0)
Urine volume (L/24 h), mean (SD)	2.1 (0.8)	2.0 (0.8)
Discretionary salt intake, no. (%)		
Salt added in cooking	344 (85%)	316 (84%)
Salt added at table	153 (38%)	199 (53%)*
Country of birth, no. (%)		
Australia/New Zealand	160 (39%)	133 (35%)
Italy	117 (29%)	136 (36%)
Greece	130 (32%)	107 (29%)
Urinary excretion, range		
Sodium (mmol/day)	10.7–382.5	52.7–523.0
Potassium (mmol/day)	15.0–210.7	32.4–292.3
Sodium-to-potassium ratio	0.38–5.41	0.63–6.62
Blood pressure, mean (SD)	*n* = 298	*n* = 289
Systolic (mmHg)	129.5 (16.8)	133.0 (13.1)†
Diastolic (mmHg)	66.6 (10.4)	76.1 (9.2)†

* $P < 0.001$ (χ^2 test). † $P < 0.001$ (*t* test).

2 Adjusted means* of daily urinary sodium, potassium and sodium-to-potassium ratio, according to different strata

	No.	Sodium (mmol/day)	Potassium (mmol/day)	Sodium-to-potassium ratio
Sex†				
Female	407	133.7 ±0.9‡	77.2 ±1.4‡	1.8 ±0.04‡
Male	376	178.3 ±0.7	88.1 ±1.9	2.2 ±0.04
Country of birth				
Australia/New Zealand	293	139.3 ±3.4§	78.9 ±1.6¶	1.9 ±0.05¶
Italy	253	160.7 ±3.6	83.5 ±1.7	2.0 ±0.05
Greece	237	169.0 ±3.7	85.4 ±1.8	2.1 ±0.05
Normotensive	334	146.2 ±3.0**	81.2 ±1.5	1.9 ±0.04**
Hypertensive††	253	154.5 ±3.5	82.2 ±1.8	2.0 ±0.05

* Data are mean ±standard error of the mean, adjusted for age, sex and body mass index (BMI). † Adjusted for age and BMI only. ‡ $P < 0.05$ v male. § $P < 0.05$ v Italy and Greece. ¶ $P < 0.05$ v Greece. ** $P < 0.05$ v hypertensive. †† Defined as systolic blood pressure (BP) ⩾ 140 mmHg or diastolic BP ⩾ 90 mmHg or currently taking antihypertensive medication (88 participants who provided no information on antihypertensive medication were classified according to their actual BP measurements). ◆

Greece (mean ±SD: 131.3 ±7.0, 130.7 ±6.4 and 131.5 ±6.5 mmHg, respectively). The mean adjusted predicted values for SBP (using the fully adjusted Model D, Box 3) were significantly greater for participants born in Greece compared with those born in Australia/New Zealand (132.6 ±7.3 v 130.4 ±7.2 mmHg; $P = 0.010$).

Hypertension, urinary sodium and sodium-to-potassium ratio

Of the 587 participants with BP measurements, 253 (43%) were classified as hypertensive. Compared with normotensive participants, hypertensive participants were significantly older (mean ±SD, 63.8 ±6.45 v 58.7 ±5.3 years; $P < 0.001$), had a higher BMI (28.4 ±4.5 v 27.5 ±4.4 kg/m^2; $P < 0.001$), a higher daily sodium excretion and a higher sodium-to-potassium ratio (Box 2).

Participants with daily urinary sodium excretion in the two highest quintiles (adjusted for age and sex) were twice as likely to be hypertensive as those in the lowest quintile (Box 4). Those with a sodium-to-potassium ratio in the highest quintile were also more likely to be hypertensive than those in the lowest quintile, after adjusting for age, sex, BMI and country of birth (Box 4).

DISCUSSION

This is one of the few within-population studies[4,5] and the first study in Australian adults to demonstrate a positive association between urinary sodium or sodium-to-potassium ratio and SBP. Our findings provide supporting evidence that the current high intake of sodium in older adults in Australia is related to higher BP. This has been difficult to demonstrate within populations because the urinary sodium–BP relationship within a community is affected by regression dilution bias, as daily sodium excretion can vary considerably within individuals and BP varies throughout the day.[21] The association may be evident in this study because this population was older and ethnically diverse. Most participants were consuming excessive amounts of sodium, which appears to be making a significant contribution to elevated BP and increased rates of hypertension. In this population with a high-sodium diet that would be common in the Western world, we found that a reduction in sodium intake of 100 mmol/day (6 g salt/day) was associated with a 2.3 mmHg reduction in SBP.

This finding is consistent with the Intersalt international cross-population study,[3] which estimated that a 100 mmol/day lower sodium intake was associated with a 2.2 mmHg lower SBP. Sodium-to-potassium ratio has also been previously related to BP regulation,[8,22] and we observed that a 1 unit decrease in the sodium-to-potassium ratio was associated with a reduction in SBP of 1.8 mmHg. A population-wide fall in SBP of 2 mmHg has been predicted to lower stroke mortality by 10% and ischaemic heart disease and other vascular diseases by 7%.[23]

Epidemiological and observational studies are likely to underestimate the association between urinary sodium and BP; randomised controlled trials have demonstrated greater falls in BP for a given reduc-

3 Relationship between blood pressure (BP) and urinary sodium, potassium and sodium-to-potassium ratio

	Model A*		Model B†		Model C‡		Model D§	
	Regression coefficient (95% CI)¶	*P*	Regression coefficient (95% CI)¶	*P*	Regression coefficient (95% CI)¶	*P*	Regression coefficient (95% CI)¶	*P*
Systolic BP								
Sodium (mmol/day)	3.0 (0.8 to 5.2)	0.007	2.6 (0.3 to 4.8)	0.025	2.4 (0.1 to 4.7)	0.038	2.3 (0.1 to 4.6)	0.049
Potassium (mmol/day)	0.1 (–4.3 to 4.5)	0.7	–0.1 (–4.5 to 4.3)	0.9	–0.3 (–4.7 to 4.2)	0.9	–0.4 (–4.8 to 4.0)	0.9
Sodium-to-potassium ratio	2.2 (0.6 to 3.8)	0.006	2.0 (0.4 to 3.6)	0.016	1.9 (0.3 to 3.5)	0.021	1.8 (0.2 to 3.4)	0.025
Diastolic BP								
Sodium (mmol/day)	1.1(–0.4 to 2.5)	0.6	1.0 (–0.5 to 2.5)	0.2	1.0 (–0.6 to 2.6)	0.2	1.0 (–0.6 to 2.5)	0.2
Potassium (mmol/day)	1.1 (–1.8 to 4.1)	0.5	1.1 (1.9 to 4.0)	0.5	1.0 (–2.0 to 4.0)	0.5	1.0 (–2.0 to 4.0)	0.5
Sodium-to-potassium ratio	0.49 (–5.7 to 1.5)	0.4	0.42 (–0.66 to 1.5)	0.4	0.41 (–0.67 to 1.5)	0.4	0.40 (–0.69 to 1.48)	0.5

* Adjusted for age and sex. † Adjusted for age, sex and body mass index (BMI). ‡ Adjusted for age, sex, BMI and country of birth. § Adjusted for age, sex, BMI, country of birth and antihypertensive use. ¶ Regression coefficients show the predicted value of the change in BP with a 100 mmol/day increase in sodium or potassium or with a 1 unit increase in sodium-to-potassium ratio. ◆

4 Prevalence of hypertension* in 587 participants with blood pressure (BP) measurements, by quintiles of urinary sodium, potassium and sodium-to-potassium ratio

	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Sodium, mmol/day	< 102.08	⩾ 102.08 to < 132.60	⩾ 132.60 to < 160.00	⩾ 160.00 to < 201.85	⩾ 201.85
Salt, g/day	< 5.9	⩾ 5.9 to < 7.6	⩾ 7.6 to < 9.2	⩾ 9.2 to < 11.6	⩾ 11.6
Systolic BP, mean ± SEM	128.6 ± 1.4	130.7 ± 1.5	130.6 ± 1 .5	132.3 ± 1.5	134.2 ± 1.5
Prevalence of hypertension	36% (45/126)	46% (54/118)	36% (44/123)	48% (58/120)	52% (52/100)
Adjusted OR (95% CI) for hypertension					
Model A (age, sex)	1	1.58 (0.89–2.8)	1.13 (0.63–2.03)	2.16 (1.20–3.88)†	2.04 (1.10–3.78)†
Model B (age, sex, BMI)	1	1.44 (0.81–2.57)	1.01 (0.56–1.83)	1.86 (1.02–3.38)†	1.56 (0.82–2.98)
Model C (age, sex, BMI, country of birth)	1	1.43 (0.80–2.56)	1.00 (0.55–1.81)	1.82 (0.99–3.33)	1.56 (0.81–3.00)
Potassium, mmol/day	< 60.27	⩾ 60.27 to < 72.67	⩾ 72.67 to < 85.84	⩾ 85.84 to < 100.73	⩾ 100.73
Systolic BP, mean ± SEM	131.4 ± 1.6	129.6 ± 1.6	133.3 ± 1.5	131.3 ± 1.5	130.1 ± 1.2
Prevalence of hypertension	44% (51/117)	41% (48/118)	45% (53/117)	44% (52/118)	42% (49/117)
Adjusted OR (95% CI) for hypertension					
Model A (age, sex)	1	0.88 (0.50–1.55)	0.87 (0.49–1.54)	1.10 (0.63–1.92)	0.80 (0.45–1.43)
Model B (age, sex, BMI)	1	0.83 (0.47–1.47)	0.78 (0.43–1.39)	1.04 (0.59–1.84)	0.72 (0.40–1.30)
Model C (age, sex, BMI, country of birth)	1	0.83 (0.47–1.48)	0.78 (0.44–1.40)	1.04 (0.59–1.84)	0.73 (0.40–1.31)
Sodium-to-potassium ratio	< 1.31	⩾ 1.31 to < 1.68	⩾ 1.68 to < 2.06	⩾ 2.06 to < 2.61	⩾ 2.61
Systolic BP, mean ± SEM	129.0 ± 1.4	129.7 ± 1.3	130.1 ± 1.5	133.4 ± 1.6	133.9 ± 1.6
Prevalence of hypertension	37% (46/123)	41% (49/120)	43% (51/120)	41% (51/123)	55% (56/101)
Adjusted OR (95% CI) for hypertension					
Model A (age, sex)	1	0.97 (0.55–1.71)	1.13 (0.64–2.00)	1.20 (0.68–2.12)	2.40 (1.32–4.38)‡
Model B (age, sex, BMI)	1	0.99 (0.56–1.77)	1.07 (0.60–1.90)	1.08 (0.61–1.93)	2.11 (1.15–3.87)†
Model C (age, sex, BMI, country of birth)	1	0.99 (0.55–1.77)	1.07 (0.60–1.90)	1.06 (0.59–1.90)	2.08 (1.13–3.85)†

SEM = standard error of the mean. OR = odds ratio. BMI = body mass index. * Defined as systolic BP ⩾ 140 mmHg or diastolic BP ⩾ 90 mmHg or currently taking antihypertensive medication. † $P < 0.05$ v Quintile 1. ‡ $P < 0.01$ v Quintile 1.

tion in sodium.[24] The strength of the positive associations between sodium and the sodium-to-potassium ratio and SBP may have been underestimated in our study. BP measurements and urine collections were performed on average nearly 3 years apart. It is possible that there may have been small improvements in dietary habits, as participants were taking part in an intensive exercise to measure their food intake over this period, which would have tended to reduce any sodium–BP association. In this cohort, participants with pre-existing cardiovascular disease were more likely to have reported a healthier diet than others in the cohort.[25] However, overall, most participants were consuming excessive amounts of sodium, with only 21% of the sample at or below the upper recommended limit of 100 mmol/day. After adjusting for age and sex, the odds of hypertension in participants in the two highest quintiles of urinary sodium (⩾ 160 mmol or ⩾ 9.2 g salt/day) were twice the odds in those in the lowest quintile. This is concerning because the majority of these participants were aware of their high BP but were still consuming large amounts of salt.

The major strength of our study is that sodium excretion from 24-hour urine collection was used to estimate sodium intake in a relatively large, ethnically diverse sample of adult Australians. The fully adjusted model indicated that the covariates, including country of birth, explained 20% of the variation in SBP. Although the stratified sampling was based on age and country of birth, all participants lived in the Melbourne metropolitan area, which may limit the generalisability of these findings.

Lowering the sodium intake of populations has been predicted to shift the population distribution curve of BP towards more optimal levels. Reducing dietary salt by 3 g per day has been projected to reduce mortality by 3%–11% for adults aged 35–64 years and to have cardiovascular benefits similar to population-wide reductions in tobacco use, obesity, and cholesterol levels.[26] However, it is very difficult for individuals to effectively reduce dietary sodium intake, as over three-quarters of total intake is from sodium present in purchased foods.[27] The most effective strategy to achieve a significant reduction in population-wide salt intake would be to reduce the salt added to staple processed foods.

With health organisations around the world advocating reduction of salt intake, more targeted government and industry support is required to reduce the sodium content of processed foods, together with educational strategies to advise consumers to limit discretionary salt consumption.[28] Such public health initiatives could effectively lower sodium intake and reduce the incidence of hypertension.

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COMPETING INTERESTS

None relevant to this article declared (ICMJE disclosure forms completed).

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