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Foods contributing to sodium intake and urinary sodium excretion in a group of Australian women

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Abstract

Objectives: To identify food sources of Na in a group of community-dwelling women in Adelaide, South Australia. A secondary aim was to measure Na excretion in this group.
Design: Survey.
Setting: Community setting, Adelaide, South Australia.
Subjects: Seventy healthy women (mean age 48·6 (SD 8·1) years, mean BMI 28·6 (SD 6·3) kg/m^2) living in metropolitan Adelaide, South Australia and participating in a validation study of an FFQ. Dietary intake was derived from two 4 d weighed food records. Foods from the 4 d weighed food records were grouped according to foods or food groups to establish contributors to Na intake. Na excretion was measured in two 24 h urine samples. Completeness of urine collections was verified using creatinine excretion.
Results: Bread alone contributed 19·0 % of Na intake, with an overall contribution from the breads and cereals group of 32·5 %. Meat products contributed 14·4 % of intake, the dairy and eggs group (excluding cheese) 9·6 % and combination dishes (e.g. pizza, quiche, sandwiches and stir fry dishes) 8·4 %. Na excretion was 126 (SD 42) mmol/d, i.e. approximately 7·6 (SD 2.5) g salt/d. Seventy per cent of participants (n 48) had Na excretion ≥100 mmol/d (146 (SD 34) mmol/d).
Conclusions: Effective Na reduction could be achieved by reducing the amount in staple foods such as bread and meat products.

Keywords
Dietary sodium intake
Urinary sodium excretion
Food supply

There is considerable evidence that a high dietary salt intake is a major contributor to raised blood pressure and that reduction of salt intake at the population level has the potential to achieve meaningful reductions in cardiovascular events and medical costs[1,2]. While intervention studies with hard end points to determine the efficacy of salt reduction are difficult and expensive to conduct, there has been one long-term follow-up study 10–15 years after the end of two randomised intervention studies, the Trials of Hypertension Prevention (TOHP) Phase I and TOHP Phase II[3]. A 25 % lower risk of a cardiovascular event was observed among those in the intervention group despite relatively small reductions in Na intake and a 30 % lower risk after adjustment for baseline Na excretion and weight. The Na reductions in the intervention groups were relatively small at 44 mmol/24 h (approximately 2·6 g NaCl/d) and 33 mmol/24 h (approximately 2 g NaCl/d) in TOHP Phase I and Phase II, respectively[4,5].

There are other harmful effects of a high Na intake including impaired endothelial function, which is associated with the development of atherosclerosis, stiffening of the arteries as measured by increased pulse wave velocity and left ventricular hypertrophy[6–9]. A high Na intake has also been shown to have a negative impact on bone density[10].

In Australia, the National Health and Medical Research Council (NHMRC) states that the Upper Level (UL) of intake should be no more than 100 mmol (2300 mg) Na/d (approximately 6 g NaCl/d) for the population and 70 mmol (1600 mg) Na/d (approximately 4 g NaCl/d) for chronic disease prevention[11].

The most recent population data on Na intake as assessed by urinary Na excretion in Australia are from the 1995 Hobart Salt Study, which showed that men had an excretion of 170 mmol Na/d (approximately 10 g NaCl/d) and women 118 mmol Na/d (approximately 7 g NaCl/d)[12]. More recently, Charlton *et al.* (2010) observed that Na

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excretion was equivalent to 111 mmol Na/d (approximately 7 g NaCl/d) in a group of women living in Wollongong, New South Wales, Australia[13]. In contrast, recent data from 24 h urine collections from overweight volunteers who participated in weight-loss studies at the Commonwealth Scientific and Industrial Research Organisation (CSIRO) have shown that Na intake is relatively high in this sample of overweight people at 133 mmol Na/d (approximately 8 g NaCl/d) in women and 183 mmol Na/d (approximately 11 g NaCl/d) in men[14].

In developed countries more than 75 % of dietary Na intake comes from processed foods, making it difficult for an individual to follow a reduced or low Na diet[15,16]. Webster *et al.* (2010) collected data on Australian processed foods including 7221 products in ten food groups and found that the food groups highest in Na were sauces and spreads (1283 mg/100 g) and processed meats (846 mg/100 g)[17]. They found that cereal and cereal products (206 mg/100 g) and fruit and vegetables (211 mg/100 g) were the lowest in Na. Similar data have been reported from the New Zealand Total Diet Survey 2003–2004[18,19]. Dietary modelling conducted by Food Standards Australia New Zealand (FSANZ) found that among adults dietary Na intake is estimated to be 1900–2500 mg/d[20]. The aim of the present study was to identify the foods and food groups contributing to Na intake in a group of healthy women living in Adelaide. A secondary aim was to measure Na excretion in this group.

Experimental methods

Women living in the community in Adelaide, South Australia were recruited by advertisements in local newspapers to participate in a validation study of the CSIRO FFQ against weighed dietary intakes. That study has been described in detail elsewhere[21]. Inclusion criteria were female, age 30–60 years and healthy as determined from a self-reported questionnaire. Exclusion criteria were having undertaken food intake measurement previously, evidence of illness/disease likely to entail dietary modification (e.g. diabetes, cardiovascular, renal, liver or gastrointestinal disease), past history of gastrointestinal surgery affecting dietary intake, medications affecting gastrointestinal motility or hunger/appetite (e.g. metoclopramide, domperidone and anti-cholinergic drugs, i.e. atropine or erythromycin) and non-English speaking. The study was approved by the CSIRO Food and Nutritional Sciences Human Research Ethics Committee and participants gave informed written consent.

Dietary intake methodology

Data collection was undertaken in two phases from September to December 2007 and March to July 2008. Participants attended the CSIRO research facility. They completed two 4 d weighed food records (WFR), 4 weeks apart, which the researchers believed was enough time to forget previous responses and maximise retention of participants in the study. Participants were asked to include three weekdays and one weekend day in each food record. They were given detailed instruction by the researchers on how to weigh and record all foods and fluids contributing to their dietary intake. Participants were asked to record the use of discretionary salt (i.e. salt added in cooking and at table) on their food record (salt cellars were not provided). When they returned the food record they were asked by the researcher to estimate the amount used in comparison to a 1 g sachet of salt. The estimated amount was then included in the dietary analysis. They were provided with printed instructions and record forms. They were asked to be specific about the food eaten (e.g. note the type and brand of food, whether meat was trimmed of fat or not, whether raw or lean weight), to weigh all food or use metric cups or spoons for fluids. They were asked to record all foods eaten, to weigh leftover food and record the amount eaten. They were asked not to deliberately change their eating pattern because they were participating in the study. Electronic weighing scales (Kitchen Craft®; Thomas Plant Ltd, Birmingham, UK) were provided. Each WFR was checked by the researcher with the participant while she was at the research facility for accuracy and for clarification of specific foods or amounts of food consumed if they seemed unusually high or low. WFR data were analysed using a computerised database of Australian foods (Foodworks Professional Edition 2007, version 5) based on the Australian nutrient database NUTTAB 2006[22]. NUTTAB 2006 contains data for 2600 foods. The majority of the data are analysed values with some data from overseas food tables, supplied by the food industry or taken from food labels. The development of NUTTAB 2006 has been described by FSANZ[23]. The numbers of foods in each set are as follows: Additives and Food Ingredients, Herbs, Seasonings and Spices (25), Beverages, Alcoholic (38), Non-Alcoholic (116), Cereals and Cereal Products, Biscuits (49), Bread and Bread Products (61), Breakfast Cereals (25) Cakes, Slices and Other Battered Products (55), Flours, Grains and Starches (48), Hamburgers, Pizza and Other Takeaway Products (23), Noodles and Pasta (33), Pastries, Pies and Tarts (34), Condiments, Dressings, Pastes and Sauces (57), Spreads (5), Dairy Butter (5), Cheese (35), Cream (12), Ice Cream and Edible Ice Products (11), Milk (26), Yoghurts and Dairy Desserts (14), Dairy and Meat Alternatives (22), Edible Fats and Oils, Edible Oil Spreads (22), Fats and Oils (23), Eggs (19), Fruit (161), Indigenous Foods, Plant Foods (6), Legumes (20), Meat and Meat Products, Beef (138), Game and Other Meat (27), Lamb (121), Mutton (20), Offal (31), Pork (75), Poultry (54), Processed Meats (67), Veal (40), Nuts and Seeds (30), Restaurant Foods, Asian Foods (84), Mediterranean Foods (36), Seafood and Seafood Products, Crustacea and Molluscs (12), Fish (59), Processed Fish, Crustacea and Molluscs (55),

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Snack Foods (36), Soups (43), Sugar, Confectionery and Sweet Spreads, Chocolate Based (21), Spreads and Toppings (12), Sugar Based (22), Sugars and Sweeteners (16), Vegetables (238).

Urinary sodium excretion

Participants collected two 24 h urine samples during the course of the study. Each collection was performed on 1 d during each of the two WFR. Participants were instructed by the researchers how to carry out a 24 h urine collection and provided with written instructions when the urine bottles were given out. These instructions were reiterated before the second collection. To minimise intrusion and promote compliance with the urine collections participants decided which day suited them best to collect the 24 h urine sample. Urine collection bottles were given to the participants in a carry bag at their visits to the research unit. The bottles did not contain preservative.

Completeness of the 24 h urine sample was verified by a creatinine excretion within the normal range.

Analysis for urinary Na and creatinine was undertaken by a certified laboratory (Institute of Medical and Veterinary Science, Adelaide, Australia). Results were averaged for the two visits.

Food group analysis

Foods from the WFR were categorised into groups (Table 1) similar to those specified by FSANZ, with modifications made to suit the purpose of the study.

Statistical analysis

Average Na excretion was calculated from the two urinary Na excretions. Average Na intake from the dietary method was calculated from the 8 d of WFR. Data are presented as means and standard deviations and as percentages. Statistical analysis was performed using the statistical software package PASW Statistics 18.

Regression analysis

Prediction intervals from a linear regression of urinary Na excretion *v.* Na intake from the dietary method were used to assess the accuracy of WFR in estimating Na excretion both at the group level and for individuals.

Agreement analysis

The validity of the dietary intake in measuring Na intakes was tested by tertile classification. Individuals were ranked by tertile for both Na excretion and intake. The overall strength of agreement between tertile rankings was measured with the weighted kappa statistic.

Results

Seventy participants, with a mean age of 48·6 (SD 8·0) years, mean BMI of 28·4 (SD 6·3) kg/m^2 and mean energy intake of 8·5 (SD 1·8) MJ/d, participated in the validation study.

Foods and food groups

Na intake from the WFR for the seventy participants who completed a WFR was 2378 (SD 690) mg/d. Proportions of Na intake contributed by the food groups are shown in Table 1. Overall the breads and cereals group contributed 32·5 % of Na in the diet, with bread alone contributing 19·0 %. Meat products and combination dishes contributed 22·8 % of Na intake, while discretionary salt use contributed 1·0 % of Na intake.

Table 1 Percentage contribution of individual foods and food groups to sodium intake among a sample of healthy women (*n* 70) living in metropolitan Adelaide, South Australia, 2007–2008

Food/food group	%
Bread and cereals group: breads, focaccia, croissant, crumpets, breakfast cereals, muesli, oats, pappadams, taco shells, pasta, noodles, rice, cous cous, biscuits, buns, cakes, pastries and fruit pies, slices, muffins, doughnuts, muffins, pancakes, desserts and puddings, flour, breadcrumbs, bran, pastry, filled pastry products, cereal beverages	32·5
Meat products group: meat products, poultry, sausages, bacon, ham, salami, other deli meats, smoked meats, pate, takeaway hamburger	14·4
Dairy and eggs group: yoghurt, cream, ice cream, custard, milk, eggs	9·6
Combination dishes, pizza, quiche, sandwiches, Asian stir fry dishes	8·4
Vegetables, vegetable-containing dishes, canned vegetables, hot potato chips and wedges, baked beans and spaghetti, seeds, nuts, fruit, potato crisps, other crisps and other savoury snack foods	7·2
Sauces, dips, Asian sauces including soya sauce, pasta sauces	5·1
Soup, stock	4·8
Cheese	4·7
Canned fish, seafood, fish and fish-containing dishes, smoked fish products	3·6
Alcoholic and non-alcoholic beverages	3·5
Chutney, relish, mayonnaise and dressings, yeast spreads, spreads, jams, honey, syrups, toppings, peanut butter and chocolate spreads	3·2
Discretionary salt, pepper, herbs and spices	1·0
Butter, margarine, oil	1·2
Sugar, jelly, confectionery, snack bars and artificial sweetener	0·8

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Urinary analysis

One participant did not complete the urine collections. Urine analysis was available for sixty-nine participants. Urinary Na excretion was 126 (SD 42) mmol/d. Seventy per cent (*n* 48) had Na excretion ≥100 mmol/d (146 (SD 34) mmol/d).

Urinary creatinine excretion was 12·0 (SD 2·6) mmol/24 h. Creatinine excretion for all participants was within the reference range of 6–16 mmol/24 h[24].

Urinary sodium excretion v. *sodium intake from weighed food records*

Urinary Na excretion was measured on sixty-nine women who had also completed WFR, from which daily Na intake was calculated. Average daily dietary Na intake was 2361 (SD 680) mg/d compared with 2898 (SD 966) mg/d (126 (SD 42) mmol/d) from urinary Na excretion ($P < 0·001$ for difference). Na excretion and intake were moderately correlated ($r = 0·5$, $P < 0·001$).

Determinants of differences between the methods

Women with BMI < 25 kg/m^2 (*n* 28) had a smaller difference between Na excretion and intake than those with BMI ≥ 25 kg/m^2 (*n* 41; 215 (SD 726) *v.* 752 (SD 888) mg Na/d, $P = 0·01$). There was an inverse relationship between the difference between Na excretion and intake and energy intake, such that it was lower in those reporting a higher energy intake ($r = -0·3$, $P = 0·012$). When age, BMI and energy intake were entered in a multivariate equation, only energy intake was independently related to the difference between Na excretion and intake. As urinary Na increased the difference between the two methods increased, with a correlation of $r = 0·72$ between the two.

To assess the strength of agreement between the two methods, a linear regression was estimated of the test method (WFR) *v.* the gold standard method (urinary excretion). The linear regression was significant ($P < 0·001$, $R^2 = 0·246$). The WFR intake regression coefficient was $B = 0·705$ (SE 0·151).

Tertiles of agreement

When the data were analysed according to tertiles, approximately 42 % (*n* 24) of participants were in exactly the same tertile (weighted $\kappa = 0·4$).

Discussion

As in other studies[13,25], the main finding of the present study was that bread alone contributed 19·0 % of Na intake with the breads and cereals group overall contributing 32·5 %. These proportions are similar to the results of a previous study in Adelaide, Australia in overweight and obese men and women with diabetes, in which breads and cereals contributed 29 % of Na intake[26]. In a recent study by Charlton *et al.* (2010) the bread and cereals group contributed 27 % of Na intake but the proportion that bread alone contributed was not reported[13]. Margerison *et al.* (2004) reported that bread contributed 8 % of the Na intake of the participants in their study and meat products/dishes contributed 17 %[25]. Comparison with other studies is difficult as the methodology used to create composite food groups varies. For example, the bread and cereals group in Charlton *et al.*'s study contains bread, cereal, rice, pasta and noodles; whereas in the present study the breads and cereals group contains these foods and in addition noodles, rice, cous cous and sweet foods such as biscuits, buns, cakes, pastries and fruit pies. The sweet foods were included in a grouping of snacks/desserts/extras in the study by Charlton *et al.*, giving different percentage contributions to Na intake from the composite groups[13]. Dietary modelling from FSANZ indicates that main contributors to Na intake are bread and bread rolls (25 %), but cereal products and cereal-based dishes (e.g. biscuits and pizza) are dealt with in a different group contributing 17 % and breakfast cereals are a group on their own contributing 4 %[20].

In the present study Na excretion was on average 126 mmol/d, with the majority of participants (70 %) having Na excretion in excess of the NHMRC-recommended UL of intake of 100 mmol Na/d. In the 1995 Hobart Salt Study women had Na excretion of 118 mmol/d and 64 % had Na excretion in excess of the NHMRC-recommended UL, suggesting that Na intakes have not changed since then[12]. In contrast, Charlton *et al.* (2010) found that average Na intake was in line with the NHMRC recommendations and only 43 % of women had an intake in excess of the recommendations[13]. However, in the study by Charlton *et al.* completeness of the urine collection was not verified and it is likely that some of the participants had incomplete urine collections. The population in Charlton *et al.*'s study had an average BMI within the normal range whereas the population in the present study was on average overweight, which may also contribute to a higher proportion of women exceeding the NHMRC UL. While similar in other respects both studies were small and the discrepancies between them provide support for the need for a larger population study of dietary Na intake.

It is of interest that bread continues to be such a large contributor to Na intake given data from the Australian Longitudinal Study on Women's Health (2009) showing that on average most (75·4 %) women eat two slices or less daily, suggesting that amount of Na in bread remains high. In a recent survey of breads in Australia only four of twenty brands met the Australian Heart Foundation's benchmark of ≤400 mg/100 g, with some popular brands of bread having Na content 30 % higher than the Heart Foundation benchmark[27]. Reducing the Na content of bread to the Heart Foundation benchmark of 400 mg/100 g would decrease the average Na excretion by only 5 %. There is

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clearly an argument to be made for reducing the Heart Foundation benchmark further to achieve more substantial Na reductions. It has been shown recently that the Na content of bread can be reduced by 52 % without affecting bread consumption[28].

The most effective way to reduce Na intake at the population level is by decreasing the Na content of all processed foods[17]. Many Australian food manufacturers are already actively doing this, but 54 % of food products in the food supply have Na levels above the salt targets proposed by FSANZ in 2010[17,29].

It is of interest that those women who had high Na intakes (as assessed by urinary Na excretion) had a greater disparity between the recorded dietary intake and the urinary excretion, suggesting that they were either using more added salt or failing to report salty snacks and other salt-rich foods. This was confirmed by the association of this difference with BMI, supporting under-reporting of food as the major problem. Under-reporting of dietary intake has been reported previously and increases as BMI increases[30]. This phenomenon emphasises the need for biomarkers of dietary intake such as urinary Na to validate dietary Na intake and urinary N to validate protein intake, together with *p*-aminobenzoic acid[31] or other markers to verify completeness of 24 h urine collections[32]. Those who reported a higher Na intake (or higher energy intake) had little or no disparity between intake and output.

Robustness of dietary intake data is dependent on the food composition tables used. In the present study the most recent version of the Australian food composition tables available at the time of the study was used[33].

We conclude that bread remains a major source of dietary Na and that the majority of women in the present study had Na intake exceeding the NHMRC UL. Effective reduction of Na requires specific targeting of those foods in the food supply that contribute most to Na intake, including breads and cereals and processed meats.

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