Appetite 68 (2013) 147–151

Contents lists available at SciVerse ScienceDirect

Appetite

journal homepage: www.elsevier.com/locate/appet

Research report

Food label education does not reduce sodium intake in people with type 2 diabetes mellitus. A randomised controlled trial ☆

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ARTICLE INFO

Article history:
Received 10 February 2013
Received in revised form 29 April 2013
Accepted 30 April 2013
Available online 9 May 2013

Keywords:
Type 2 diabetes mellitus
Nutrition education
Sodium
Food labelling
Hypertension

ABSTRACT

Background: Sodium intake is high in people with type 2 diabetes (T2DM). The aim of this study was to investigate whether urinary sodium excretion can be reduced by educating people with T2DM to read food labels and choose low sodium products.
Method: In a 3 month randomised controlled trial, 78 men ($n = 49$) and women ($n = 29$) with T2DM were recruited from a Diabetes Centre at a University teaching hospital. The intervention group was educated in a single session to use the nutrition information panel on food labels to choose products which complied with the Food Standards Australia New Zealand (FSANZ) guideline of <120 mg sodium/100 g food. The control group continued on their usual diet. The primary outcome measure was 24 h urinary sodium excretion which was performed at baseline and 3 months. Data was analysed using repeated measures analysis of variance, independent samples *t*-test and Pearson's correlations.
Results: At 3 months mean urinary sodium excretion was unchanged in the intervention (174 ± 13 mmol/24 h and 175 ± 13 mmol/24 h) and control group (167 ± 15 mmol/24 h and 161 ± 13 mmol/24 h), and there was no between group difference ($p > 0.05$).
Conclusion: Sodium excretion was not reduced following the label reading education provided to this group of people with T2DM.

Introduction

Type 2 diabetes (T2DM), which is growing in prevalence worldwide, increases the risk of developing cardiovascular disease (CVD) (Booth, Kapral, Fung, & Tu, 2006; Lee, Cheung, Cape, & Zinman, 2000). Hypertension, a risk factor for CVD, affects up to 70% of individuals with T2DM (Gomes et al., 2009). Hypertension has a significant effect accounting for 35–75% of diabetic complications (Feldstein, 2002). A modest reduction in sodium intake of 74–78 mmol/24 h (approximately 1700–1800 mg) can lower blood pressure in both hypertensive (systolic blood pressure −4.97 mmHg, diastolic blood pressure −2.74 mmHg) and normotensive individuals (systolic blood pressure −2.03 mmHg, diastolic blood pressure −0.99 mmHg) (He & MacGregor, 2004). Sodium reduction can effectively reduce blood pressure in people with T2DM as shown when a reduction in dietary sodium intake of 60 mmol/day reduced systolic blood pressure by 12 mmHg (Dodson et al., 1989).

Recent Australian studies show that sodium excretion is high in people with T2DM attending a specialist diabetes clinic and in overweight study participants (Ekinci et al., 2010; Villani, Clifton, & Keogh, 2012). Thus sodium reduction presents an opportunity for people with T2DM to reduce their blood pressure using a dietary approach.

The aim of this study was to investigate whether urinary sodium excretion could be reduced by educating people with T2DM to read food labels and choose products with a low sodium content. Secondary aims were to determine whether blood pressure medications could be reduced in participants on antihypertensive medications and if blood pressure could be reduced in participants not on these medications and to understand participants' experiences of the intervention. It was hypothesised that the education provided would enable participants with T2DM to modestly reduce their sodium intake (20%) which may have a favourable effect on blood pressure.

☆ *Acknowledgements*: This study was supported by a grant from the Division of Health Sciences, University of South Australia. The authors of this study would like to acknowledge the contributions of study participants, the staff of the Royal Adelaide Hospital Diabetes Centre, the University of South Australia and Flinders University. *Conflicts of interest*: The authors declare there are no conflicts of interest.

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http://dx.doi.org/10.1016/j.appet.2013.04.028

Materials and methods

Participants and recruitment

Eligible participants were adults (>18 years) diagnosed with T2DM. There were no inclusion/exclusion criteria for blood pressure or baseline urinary sodium excretion. Those diagnosed with a medical condition affecting sodium fluid balance were excluded. Individuals prescribed diuretics were eligible for inclusion as treatment with thiazide diuretics for >4 weeks does not affect urinary sodium excretion (Ekinci et al., 2009).

Seventy-eight people, diagnosed with T2DM, were recruited from the Diabetes Centre at the Royal Adelaide Hospital, during July–December 2011. Potential participants were approached by study personnel, the response rate was not recorded. Participants were enrolled in the study by one of two study personnel and randomised to one of two parallel groups after enrolment. A random allocation sequence was generated in blocks of four, to ensure an equal allocation ratio. Study personnel were not blinded to the participant's randomisation. Written informed consent was obtained from the participants and the study was approved by Royal Adelaide Hospital Human Research Ethics Committee and the University of South Australia Human Research Ethics Committee. The trial was registered with the Australian New Zealand Clinical Trials Registry (ACTRN12611000348954).

Study design

This was a 3 month randomised controlled study. All participants completed a 24 h urine sample, for measurement of urinary sodium, potassium and creatinine, prior to attending the baseline visit and were unaware of the results until their 3 month follow-up appointment. All self-reported information and measurements were taken by one of two study personnel. Outcome measures were recorded at 0 and 3 months.

Dietary education

The dietary education which focussed on label reading was provided to participants individually in a single 10–15 min session by study personnel. Participants were educated to identify foods, using the nutrition information panel, complying with the Food Standards Australia New Zealand (FSANZ) sodium guideline of less than 120 mg per 100 g food. A booklet, developed by a qualified dietitian, based on experiences from a previous study (Ireland, Clifton, & Keogh, 2010) was used in the education session and included colour images of currently available low sodium processed food products as well as general salt reduction guidelines. As there was no low sodium bread available on the market the brand of bread with lowest sodium content was recommended (280 mg sodium/100 g). Food packages of products meeting the FSANZ guideline were used during the education session to enable participants to practice their label reading skills and demonstrate their understanding of the information provided. The control group ($n = 39$) continued on their usual diet without any extra dietary advice.

Outcome measures

Sodium, potassium and creatinine were measured in a 24 h urine sample at 0 and 3 months. All biochemical analyses were performed at the Institute of Medical and Veterinary Sciences Adelaide, South Australia, a certified commercial laboratory. The total volume of the urine sample was measured. Aliquots were taken and sodium and potassium concentrations were measured using ion-selective electrodes. Creatinine was measured using the Jaffe reaction. Completeness of the samples was assessed by urinary creatinine excretion. Samples with a urinary creatinine excretion below 6 mmol/24 h for women and 8.8 mmol/24 h for men were considered incomplete as this is the lower limit of the laboratory's reference range.

Weight was measured at 0 and 3 months using a calibrated electronic scale (Avery Berkel HL 122, Selacs Pty Ltd, Adelaide, 2002) while the participants were in light clothing. Height was measured to the nearest 0.1 cm with a stadiometer at baseline. Body mass index (BMI) was calculated as kg/m^2.

Participants self-reported all prescribed medications, including anti-hypertensive drugs at baseline. At 3 months any changes in prescribed medications or doses were reported by the participants.

Clinic blood pressure was measured at 0 and 3 months using an Ambulatory Blood Pressure Monitor (Meditech Ltd., Hungary, 2003) once the participant had been seated for 5 min. Four consecutive readings were taken, the first reading was discarded and the mean of the following three measurements was taken. Blood pressure was re-measured when readings differed by greater than 10 mmHg systolic blood pressure. Blood pressure was measured on the same arm at 0 and 3 months and in the same manner across subjects.

A 24 h recall was completed at 0 and 3 months and analysed using a computerised database of Australian foods (Foodworks Professional Edition, Version 6, Xyris Software 2009, High gate Hill, Australia). Participants in the intervention group completed a self-administered questionnaire regarding their experiences of sodium reduction, based on a previous questionnaire (Ireland et al., 2010). Participants were asked to indicate on a 5 point Likert scale the extent to which they adhered to the program (''none of the foods I ate met the requirements'' to ''all the foods I ate met the requirements'') and how they found particular aspects of the program including shopping, eating out and reading labels (''extremely hard'' to ''very easy''). Participants were also asked to rate the availability of low salt products (''very poor'' to ''very good) and the taste of low salt products (''very bad'' to ''very good''). Qualitative data was not collected from the controls.

Data analysis

Based on previous studies (Ireland et al., 2010; Villani et al., 2012) it was estimated that 60 participants (30/group) were required for 80% power to detect a 35 mmol/24 h difference in sodium excretion between groups. Independent samples t-test was used to detect between group differences at baseline. Repeated measures analysis of variance was used to determine changes in outcome measures with time as the within-subject factor and group as the between subject factor. Pearson's correlations were performed. Descriptive data about study participants is presented as the mean ± standard deviation and outcome data is presented as the mean ± standard error of the mean. Analysis was performed with SPSS for Windows (SPSS Version 19.0, SPSS Inc., Chicago, IL, 2010). Significance was set at $p < 0.05$. All tests were two tailed.

Results

Thirty-nine participants in the intervention group (mean age 62.9 ± 10.8 years; 26 men, 13 women) and 39 in the control group (mean age 61.6 ± 10.8 years; 23 men, 16 women) commenced the study. Thirty-eight participants (49%) were using oral hypoglycaemic agents and insulin concurrently to treat their T2DM and 59 (76%) were taking one or more anti-hypertensive medications. Median glycosylated haemoglobin type A1c (HbA1c) at baseline was 8.1 ± 1.7% (65 mmol/mol) and mean reported time since T2DM diagnosis was 13.5 ± 7.9 years.

At baseline sodium excretion was 188 ± 75 mmol/24 h (4255 ± 1725 mg/24 h) in men and 127 ± 62 mmol/24 h (2921 ± 1426 mg/24 h) in women. Six participants (men $n = 2$; women $n = 4$)