

randomised to the VD or HD for 14 weeks. IL-6 was measured at baseline and 14 weeks. Sodium intake was assessed using 24-hour urinary collections. Intervention data were analysed using one-way ANCOVA.

Results: Thirty-nine and thirty-four subjects completed the VD and HD respectively. Sodium intake remained unchanged in the HD (mean change -0.04 mmol/L; 95%CI: $-18.4, 18.3$; $p = 0.99$), while the VD saw a mean 35.4 mmol/L reduction (95%CI: $20.5-50.3$; $p < 0.001$) with the intervention. IL-6 decreased over time by 1.47 pg/mL (95%CI: $1.21-1.78$; $p < 0.001$) and 1.35 pg/mL (95%CI: $1.15-1.59$; $p < 0.001$) for the VD and HD respectively. However when diet groups were examined there was no association between sodium intake and IL-6 ($p = 0.52$).

Conclusions: The inflammation status of individuals at risk of heart disease, as measured using IL-6, did change over time but the effect was not driven by sodium intake in this cohort.

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EFFECTS OF INCREASED POTASSIUM AND SODIUM ON ENDOTHELIAL AND VASCULAR FUNCTION

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Background/Aims: Increased potassium intake has been related to improved endothelial function. High sodium intake is known to impair endothelial function. The effect of increasing potassium in the presence of high dietary sodium is not known. The aim was to determine the effect of increased potassium and increased sodium on postprandial endothelial function, as assessed by flow mediated dilatation (FMD).

Methods: Thirty nine healthy, normotensive volunteers (mean \pm SD age 37 ± 15 years and BMI 23.0 ± 2.8 kg/m²) received a meal with 3.1 mmol potassium and 65 mmol sodium (LKHNa), a meal with 38 mmol potassium and 65 mmol sodium (HKHNa) and a control meal (LKLNa) with 5.5 mmol sodium and 3.1 mmol potassium on three separate occasions in a randomized order. FMD, pulse wave velocity (PWV) and blood pressure (BP) were measured while participants were fasting and at 30, 60, 90 and 120 minutes after the meal. Repeated-measures ANOVA was used to assess the effects of the meal type on the dependent variables over time.

Results: The addition of potassium (HKHNa) significantly attenuated the post meal decrease in FMD when compared to the high sodium meal and control meals ($p < 0.05$). FMD was significantly lower following the LKHNa meal when compared to the HKHNa meal at 30 minutes ($p < 0.05$). There was no significant differences in PWV or BP between treatments.

Conclusions: The addition of potassium to a high sodium meal attenuates the post meal reduction in endothelial function as assessed by FMD. Increases in sodium and potassium do not affect PWV or BP in the postprandial state.

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UPDATING AN EXISTING REVIEW

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Background/Aims: A systematic review is 'time-stamped' to the date of the literature search. A quantitative result (i.e. meta-analysis) might change when relevant new studies are published and the review is updated. We describe three ways of examining whether new data alter the conclusion of He *et al* (2013) that lowering sodium intake reduces blood pressure (-4.18 mmHg, 95%CI: $-5.18, -3.18$ random effects analysis).

Methods: One new paper was found when the literature search was replicated for the period December 2012 to August 2013. Its impact on the existing result was examined using: shorthand estimation, complete re-analysis and update by combining the previous overall effect with the additional result.

Results: The new study had a mean reduction in blood pressure larger (-12 mmHg, $p < 0.01$) than the existing meta-analysis result. Therefore, this could not reduce the size of the existing result. This might be a sufficient assessment in certain situations. Complete random effects re-analysis yielded -4.32 mmHg (95%CI: $-5.33, -3.31$). However combining the new results with the previous overall result yielded -7.45 mmHg (95%CI: $-15.01, 0.11$) or -4.37 mmHg (95%CI: $-5.36, -3.39$) in a random or fixed effects analysis respectively.

Conclusions: The variation in results seen would not be observed in all datasets. Short-cut methods can be useful in certain circumstances. Care needs to be taken when interpreting the results of any analysis.

Funding source(s): N/A.

Concurrent session 8: dietary composition and metabolic outcomes

SATURATED FAT INTAKE, STATIN THERAPY AND ATHEROSCLEROTIC VASCULAR DISEASE: A PROSPECTIVE COHORT STUDY

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Background/Aims: The relationship between saturated fat (SFA), statin therapy and atherosclerotic vascular disease (ASVD) remains uncertain, particularly in the elderly population. The aim of this study was to investigate the association of SFA intake, statin therapy and risk of ASVD death in a population of elderly women.

Methods: A prospective cohort study of 1,469 women living in Perth, Western Australia, mean age 75.2 (SD 2.7) years, had SFA intake assessed using a validated food frequency questionnaire. Statin use was determined from patient report verified by medical record data. Total ASVD deaths were retrieved from the Western Australian Data Linkage System. HR was tested using Cox regression analysis.

Results: At baseline mean (SD) SFA intake was 25 (11) g/day and 268 (18%) participants were taking statin medication. Over 10 years, 134 participants died from ASVD-related causes. In the highest compared with the lowest quartile of SFA intake, the risk of ASVD death was higher in the total cohort (HR = 3.04, $p = 0.002$) and the statin non-user subset (HR = 3.55, $p = 0.001$), but not in the statin user subset (HR = 1.05, $p = 0.962$).

Conclusions: High SFA intake was associated with increased risk of 10-year ASVD death in elderly women. The risk was most evident in participants with SFA intake > 31 g/day and not taking statin medication. These data support current practice to lower SFA intake in low risk individuals and to continue statin therapy in high risk individuals.

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DIETARY INTAKE AND BEHAVIOURS IN PEOPLE WITH SEVERE MENTAL ILLNESS ACROSS FOUR COUNTRIES: COMPARISON WITH A NORMATIVE SAMPLE

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Background/Aims: Physical health is inextricably linked with mental health; people with severe mental illness (SMI) have poor physical health, high mortality and cardiovascular diseases. We investigated dietary intake and behaviours in people with SMI and compared with a normative sample.

Methods: Demographic and health behaviour data were collected from 697 people with SMI (aged 17–69 years) in Germany ($n = 387$), Middle East ($n = 200$), London ($n = 67$) and Australia ($n = 43$). Data were analysed by one-sample *t*-tests with 666 people with substance abuse disorder ($n = 224$), schizophrenia ($n = 158$), mood disorders ($n = 227$) and somatoform disorders ($n = 63$). The General Health Behaviour Questionnaire investigated dietary intake of healthy food/drinks and 'traditional' (unhealthy) food, regularity of meals, snacking, eating out, emotional eating and knowledge regarding the impact of diet on health. The normative sample was derived from a German population ($n = 495$).

Results: The whole sample had significantly lower intake of healthy food/drinks and higher intake of 'traditional' (unhealthy) food (both $p < 0.001$); these remained consistent across the four subgroups. They reported higher snacking, less regular meals, eating out, eating more when