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The relationship between the dietary inflammatory index and risk of total cardiovascular disease, ischemic heart disease and cerebrovascular disease: Findings from an Australian population-based prospective cohort study of women

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ABSTRACT

Background and aims: Recently, a pro-inflammatory diet based on a dietary inflammatory index (DII) has been related to higher CVD risk in general population, but this has not been investigated among women.
Methods: We investigated the relationship between DII and risk of total CVD and CVD subgroups (myocardial infarction, ischemic heart disease, stroke and cerebrovascular disease) in a prospective cohort of 6972 Australian women aged 50–55 years at baseline in 2001. We used clinical and procedure information from inpatient hospital separation registries, information on use of health care services, and from the causes-of-death registry to ascertain CVD outcomes during 11-year follow up. The association between baseline DII score and cardiovascular endpoints was analysed through cox-regression, with correction for demographic and cardiovascular risk factors.
Results: We identified 335 incident cases of CVD and 191 cases of ischaemic heart disease (including 69 myocardial infarctions) and 59 cases of cerebrovascular disease (including 40 cases of stroke). A statistically significant higher risk of myocardial infarction was observed in analyses using DII scores as a continuous variable with a hazard ratio of 1.46 (95% confidence interval 1.12–1.89), but this was attenuated by further adjustment for other known cardiovascular risk factors. No association was found for total CVD, ischaemic heart diseases, or cerebrovascular disease.
Conclusions: There was no statistically significant association between the dietary inflammatory index and risk of total cardiovascular disease, ischemic heart disease, myocardial infarction, cerebrovascular disease or stroke in this population of mid-aged Australian women. Associations were not different for postmenopausal women.

1. Introduction

Subclinical, chronic inflammation due to damage to the endothelial vessel wall has long been hypothesized as part of the pathophysiology of cardiovascular diseases (CVD) [1]. This could be through a relationship with cardiovascular risk factors such as hypertension [2] and metabolic syndrome [3] or through an atherothrombotic effect of inflammation itself [4]. Pro- or anti-inflammatory properties have been described for a number of foods and nutrients and an extensive literature research regarding these food items and nutrients has led to the development of a Dietary Inflammatory Index (DII) [5]. The goal of this index was to

Abbreviations: ALSWH, Australian Longitudinal Study on Women's Health; ATC code, Anatomical Therapeutical Chemical code; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; DII, dietary inflammatory index; FFQ, food frequency questionnaire; HR, hazard ratio; ICD, international classification of disease; IHD, ischemic heart disease; MBS, medicare benefits schedule; MET, metabolic equivalent of task; MI, myocardial infarction; MUFA, monounsaturated fatty acids; PBS, pharmaceutical benefits scheme; PUFA, polyunsaturated fatty acids; SD, standard deviation.

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use different food parameters, categorized into pro- or anti-inflammatory or inflammatory neutral, in order to calculate the inflammatory property of a person's total diet [5,6]. A relationship between the DII and levels of inflammatory markers has been found in multiple cohorts [5,7–10], though not all [11]. This has led to the hypothesis that a more pro-inflammatory diet according to the DII, might be associated with higher risk of CVD.

The DII score has been investigated previously with regard to cardiovascular risk factors. In a population-based cohort from Luxembourg, a relationship between DII and HDL-cholesterol was found in cross-sectional analyses [12], though no association was observed with other lipids, diastolic blood pressure or hypertension [11,12]. Studies in different cohorts have suggested a relationship between pro-inflammatory DII scores and glycemic biomarkers [8,13] and BMI and waist-hip ratio [14]– factors related to CVD. Also, a more pro-inflammatory diet has been related to total CVD in one previous study performed among a Spanish population at high risk of developing CVD, but these results might not be easily generalized to a population not at high risk of CVD and no sex-specific strata were described [15].

Based on prior research with the DII and evidence linking inflammation with CVD, this study aimed to investigate the relationship between the DII and total CVD and several subtypes, namely ischaemic heart disease (IHD), myocardial infarction (MI), stroke and cerebrovascular disease in a population-based cohort of middle-age Australian women.

2. Patients and methods

2.1. Study population

The Australian Longitudinal Study on Women's Health (ALSWH) recruited a nationally representative sample of over 40,000 women in 1996 [16]. Women were sampled from the universal health insurance scheme that includes all Australian citizens and permanent residents, Medicare Australia. Three age groups were sampled; women born in 1973–78, 1946–51, and 1921–26. Sampling of women was random, except that women from rural and remote areas were intentionally oversampled.

For this study we used the cohort of women born in 1946–51. This cohort of middle-aged women has been surveyed every 2–3 years since the start of the study in 1996. Based on the initial response of 13,715 to Survey 1 (baseline), response rates for Surveys 2, 3, 4, 5, 6 and 7 were n = 12,338 (90%), n = 11,221 (81.8%), n = 10,905 (79.5%), n = 10,638 (77.6%), n = 10,011 (73.0%) and n = 9151 (66.7%), respectively [17,18]. Dietary information was assessed at Survey 3 in 2001 and return date of this survey was therefore used as baseline for our study.

The Human Research Ethics Committee of the University of Newcastle and the University of Queensland approved the study methods. Further details on the sample and methods used by the ALSWH have been reported elsewhere [16–18] and are available at www.alswh.org.au.

The sample consisted of women living in states for which hospital information was available, namely Australian Capital Territory, New South Wales, Queensland, South Australia and Western Australia. Women with missing nutritional data (n = 593), who lived in states for which no hospital information was available (n = 3150) or who opted out of linkage with inpatient hospital separation registries (n = 473) were excluded.

We also excluded women with chronic kidney disease stage 4 or 5 during follow up (n = 13) and with prevalent CVD (n = 20), after which 6972 women remained for data analysis.

2.2. Dietary intake

At baseline in 2001, information on dietary intake over the past 12 months was obtained by use of The Dietary Questionnaire for Epidemiological Studies, version 2 (DQES v2). Based on this questionnaire, participants were asked to provide an estimate of the mean daily consumption of 101 individual food items in 18 food categories [19].

Composition tables from The Australian Food Composition Database were used to calculate energy and nutrient intakes relevant to this study [20]. The DQES has been validated in a study among 63 women of childbearing age against seven days of weighed food records, showing <10% variation in mean nutrient intakes for most nutrients [21].

The development and validation of the DII are described in detail elsewhere [6]. Developing the DII involved reviewing and scoring nearly 2000 scientific articles representing cell culture and laboratory animal experiments, and a variety of human studies on diet and six inflammatory markers (i.e., CRP, interleukin (IL)-1β, IL-4, IL-6, IL-10, tumor necrosis factor (TNF)-α). Developing the DII also entailed creation of a world standard database that involved obtaining 11 data sets from around the world to which individuals' intakes of 45 food parameters (consisting of nutrients, spices and whole foods) on which the DII is based, could then be compared.

FFQ-derived dietary data were used to calculate DII scores for all ALSWH participants. Dietary data were first linked to the previously described regionally representative world database that provided a robust estimate of a mean and standard deviation for each parameter [6]. These then became the multipliers to express an individual's exposure relative to the “standard global mean” as a z-score. This score was computed by subtracting the “standard global mean” from the amount reported and dividing this value by the “global standard deviation” of the world population as represented by the 11 data sets used for comparative purposes. To minimize the effect of “right skewing”, which commonly occurs for dietary variables, this value was then converted to a centered percentile score.

For each individual food parameter, this score was multiplied by the respective food parameter effect score, derived from the literature review, in order to obtain a food parameter-specific DII score [6]. All of the food parameter-specific DII scores were then summed to create the overall DII score for each participant in the study, DII = b1*n1+b2*n2…b29*n29, where b refers to the literature-derived inflammatory effects score for each of the evaluable food parameters and n refers to the food parameter-specific centered percentiles, which were computed from the FFQ-derived dietary data.

For the current study, data on 25 of the 45 DII food parameters could be derived from the FFQ and were thus used for calculating the DII. These include: pro-inflammatory components (energy, carbohydrate, protein, fat, saturated fat, iron, cholesterol) and anti-inflammatory components (alcohol, fibre, mono-unsaturated fat, poly-unsaturated fat, omega-3, omega-6, niacin, thiamin, riboflavin, magnesium, zinc, vitamin A, vitamin C, vitamin E, folic acid, beta-carotene, garlic and onions). Theoretically, the DII can range from the most anti-inflammatory score of −8.87 to the most pro-inflammatory score of 7.98.

2.3. Ascertainment of endpoints

We have obtained endpoints through the inpatient hospital separation registries, causes of death registry and Medicare Benefits Scheme (MBS). Mortality data were obtained through matching of participants on name, address and date of birth with the National Death Index [22]. Information on causes of death was complete