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Dietary inflammatory index or Mediterranean diet score as risk factors for total and cardiovascular mortality

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Abstract

Background and Aims—Dietary patterns are associated with risk of cardiovascular disease (CVD). We aimed to examine associations of the Dietary Inflammatory Index (DII) and the Mediterranean Diet Score (MDS) with total, cardiovascular disease (CVD) and coronary heart disease (CHD) mortality in the Melbourne Collaborative Cohort Study; and compare the strengths of the associations.

Methods and Results—In our prospective cohort study of 41,513 men and women aged 40–69 years, a food frequency questionnaire was completed at baseline and mortality data were obtained via linkage with local and national registries over an average of 19 years follow up. At baseline, questionnaires were completed and physical measures and blood samples taken. Cox proportional hazards models, adjusting for age, alcohol consumption, sex, region of origin, personal history of CVD or diabetes and family history of CVD, were used to assess associations between dietary scores and mortality

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Conflict of interest Dr. James R. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counselling and dietary intervention in clinical settings. Dr. Nitin Shivappa is an employee of CHI. The other authors declare that Cancer Council Victoria received funding from the National Health and Medical Research Council Australia which supported this work as described below.

Author contributions AMH came up with the idea for the analysis. AMH, JKB and DRE designed the analysis and JKB performed the analysis. All authors read the manuscript and commented on the analysis, and contributed to the interpretation of the results and to the discussion.

More Mediterranean or less inflammatory diets were associated with lower total, CVD and CHD mortality. The hazard ratio for total mortality comparing the highest and lowest quintiles was 1.16 (95% CI: 1.08–1.24) for DII; and 0.86 (95% CI: 0.80–0.93) comparing the highest and lowest three categories of MDS. Using the Bayesian information criterion, there was no evidence that the DII score was more strongly associated with total and CVD mortality than was the MDS.

Conclusions—The MDI and the DII show similar associations with total and cardiovascular mortality, consistent with the consensus that plant-based diets are beneficial for health.

Keywords

Dietary inflammatory index; Mediterranean diet score; mortality; cardiovascular disease; coronary heart disease; prospective; epidemiology

Introduction

Many studies have investigated associations between diet and cardiovascular disease (CVD), including several large-scale dietary interventions. A recent review concluded that an approach based on overall diet quality was preferable to focussing on a few specific nutrients, such as saturated/total fat and cholesterol [1].

The cardiovascular benefits of the MDS have been demonstrated in the PREDIMED Study, a multi-centre trial in participants at high risk of CVD randomized to Mediterranean diet plus olive oil or nuts, or a low fat control diet. Both interventions reduced the risk of major cardiovascular events by around 30% relative to the control group [2]. The Dietary Inflammatory Index (DII) is a relatively new method of assessing diet; a higher score, reflecting a more inflammatory diet, has been associated with CVD incidence and mortality [3]. Subsequently, a study of Australian women found no association between DII scores and risk of total CVD, ischaemic heart disease or cerebrovascular disease [4]. A nested case-control study found that for Swedish men, but not women, DII was positively associated with risk of myocardial infarction (MI) over 6 years [5]. In a case-control study from Italy, a DII in the top quartile was associated with a 60% greater risk of acute myocardial infarction compared with a DII in the lowest quartile [6].

We aimed to examine and compare prospective associations of the DII and the MDS, with total mortality, CVD or coronary heart disease (CHD) mortality in the Melbourne Collaborative Cohort Study.

Materials and Methods

Study population

The Melbourne Collaborative Cohort Study (MCCS) is a prospective cohort of 41,513 participants (24,469 women), of whom 99% were aged 40–69 years at baseline. Full details of the design, recruitment, and study procedures are published [7]. Recruitment occurred between 1990 and 1994 with participants approached via the electoral roll, ethnic radio, clubs, and churches. The Cancer Council Victoria Human Research Ethics Committee approved the study, and subjects gave their written informed consent to participate. At

baseline, questionnaires capturing lifestyle exposures, including dietary intake, were completed, physical measurements and blood samples were taken. Subjects missing data for dietary intake or any of the final covariates modelled were excluded from analyses. We also excluded subjects who had daily energy intakes greater than sex-specific 99th percentile or less than sex-specific 1st percentile. This left 39,532 subjects for final analysis (23,481 women, 16,051 men).

Assessment of dietary intake

Dietary information was collected using a self-administered, 121-item FFQ [8]. Nutrient intakes were calculated using food composition data from NUTTAB 95 [9], USDA [10] (carotenoids), Britain [11] (vitamin E) and RMIT [12] (fatty acids) and sex-specific standard portions. Nutrients from supplements were not included in the calculated intakes.

Baseline demographic and risk factors

A structured interview was used to obtain information on sex, country of birth, smoking, alcohol consumption, physical activity, personal history of CVD and diabetes, family history of CVD, and socioeconomic status, the latter using Socio-Economic Indexes for Areas (SEIFA) Index of Relative Socio-economic Disadvantage [13]. Height, weight and hips and waist circumferences were measured using standard protocols. Blood pressure was measured three times using Dinamap 1846SX automatic blood pressure monitors (Johnson & Johnson Medical Pty Ltd) and the mean of the second and third measurement was calculated for both systolic (SBP) and diastolic blood pressure (DBP). Mean arterial pressure (MAP) was calculated from these values [$MAP = (SBP + 2(DBP))/3$]. Total cholesterol was measured in plasma using a Kodak Ektachem DT-60 Analyser (Rochester, NY, USA).

Cohort follow-up and ascertainment of deaths

Deaths were included in the analysis if they occurred between the recruitment date and 30 November 2013. The Victorian Cancer Registry assigned codes for underlying cause of death from cancer since 2007; other causes of death were coded by the Australian Bureau of Statistics. Deaths due to CVD and CHD were defined based on underlying cause of death codes (390–459 and I00–I99 for CVD and 410–414 and I20–I25 for CHD) from the 9th and 10th revisions of the International Classification of Diseases.

The Dietary Inflammatory Index (DII)TM

The development of the DII is described in detail elsewhere [14]. Briefly, peer-reviewed primary research articles published through December 2010 on the effect of diet on inflammation were screened for possible inclusion. Qualifying articles were scored according to whether each dietary parameter increased, decreased or had no effect on six inflammatory biomarkers: IL-1 β , IL-4, IL-6, IL-10, TNF- α and C-reactive protein. The DII used here is an update of the original score first published in the SEASONS Study [15]. The current DII has been validated in three additional studies in the U.S.A. (in the Women's Health Initiative [16]), in a population of African-Americans in South Carolina [17] and in the National Health And Nutrition Examination Survey [6]. The DII was also subjected to construct validation in studies of adults in Belgium [18], and of adolescents in Europe [19].

These studies confirmed associations between the DII and inflammatory biomarkers. Of the 45 possible items in the DII, values for MCCS participants were calculated using the 29 foods and nutrients available: energy, carbohydrate, protein, fat, alcohol, fibre, cholesterol, saturated fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids omega 3 fat, omega 6 fat, trans fat, niacin, thiamine, riboflavin, vitamin B12, vitamin B6, iron, magnesium, zinc, vitamin A, vitamin C, vitamin E, folic acid, beta carotene, garlic, onion and tea.

Mediterranean Diet Score

A Mediterranean Diet Score was computed as described by Trichopoulou et al [20]. Intakes equal to or above the sex-specific medians for frequency of intake for vegetables, fruit, cereals, legumes, and fish, and below the medians for dairy and red meat (not chicken), contributed 1 point to the score; other intakes contributed zero. Daily intakes of alcohol between 10 and 50 g/d for men and 5–25 g/d for women contributed 1 point; lower or higher intakes scored zero [20]. Trichopoulou et al used the ratio of dietary monounsaturated:saturated fats as the ninth component of the Mediterranean Diet Score [20], but we have used the intake of olive oil instead because in Australia red meat is an important source of monounsaturated fatty acids [21].

Statistical analysis

Quintiles were obtained for the DII using the whole sample; the lowest quintile was used as the reference category. The MDS ranges from 0 to 9, which we categorised into 3 groups: 0–3; 4–6; and 7–9. Energy-adjusted intakes [22] of selected food groups were summarised by DII quintiles according to region of origin.

Cox regression models were fitted with age as the time axis, to estimate hazard ratios (HRs) and 95 % confidence intervals for the association between dietary scores and all-cause, CVD or CHD mortality separately. Participants were considered at risk from study entry (baseline) until the earliest of date of death, date last known to be in Australia or 30 November 2013 (date at which ascertainment of cause of death was considered complete).

Potential confounders measured at baseline were selected based on a directed acyclic graph (DAG) (Supplementary Figure X) and all models were adjusted for sex, SEIFA quintile, personal history of CVD or diabetes and family history of CVD, and alcohol consumption (lifetime abstainers; ex-drinkers; low intake (men: 1–39g and women: 1–19g/d); moderate intake (men: 40–59g and women: 20–39g/d); high intake (men: 60g/d and women: 40g/d)). Participants were divided according to region of origin (northern Europe including Australia/New Zealand/UK and southern Europe including Greece/Italy) on the assumption that the northern European group would follow similar diets, while the southern Europeans would follow variations of the Mediterranean diet.

Tests based on Schoenfeld residuals and other graphical methods (log-log plots and plots of observed and predicted survival over time) were used to identify violations of the proportional hazards assumption. As hazards were not proportional for personal and family history of CVD and sex, all models were stratified for these variables.

For CVD mortality, there was evidence that the MDS violated the proportional hazards assumption. Therefore, we included in this model a two-way interaction term between MDS and attained age (age was used as the underlying timescale) to allow the MDS to vary continuously over time, and the HRs were estimated separately for ages 72, 78 and 83 years; these ages were the 25th, 50th and 75th percentiles of the distribution of age at death from CVD. To allow comparison between models using the MDS or DII, we performed the analysis in the same way for DII.

Interactions between region of origin and the DII and the MDS were also tested. To estimate linear trends on a log-hazards scale, the DII and MDS were fitted as pseudo-continuous variables (using the median value in each category). We fitted separate models for region of origin for the CVD outcome as these models also included the two-way interaction term between DII or MDS score and attained age. We used the Bayesian information criteria (BIC) to compare models including the DII with models including the MDS.

We conducted sensitivity analyses by adjusting for cholesterol (<5.2mmol/L; 5.2 to <6.2mmol/L; 6.2mmol/L) and mean arterial pressure (MAP, <70mmHg; 70–105mmHg; 105mmHg), excluding those with a personal history of CVD, not adjusting our models for personal history of CVD or diabetes, and excluding the first 2 years of follow-up to minimise the possibility of reverse causality.

Statistical analyses were performed using Stata/SE® 14.1 (Stata Corporation, College Station, TX). All P-values were two-sided and those less than 0.05 were considered statistically significant.

Results

Table 1 shows the baseline characteristics of the study participants by DII quintiles. DII values ranged from anti-inflammatory at -5.0 to pro-inflammatory at 4.9. Compared with those in the most anti-inflammatory quintile, participants in the most pro-inflammatory DII quintile were younger, more overweight, less active, more likely to be current smokers and in the most disadvantaged SEIFA quintile. Participants of southern European origin were overrepresented in the most pro-inflammatory DII quintile as were females. Participants with the most anti-inflammatory diets were more likely to have a family history of CVD (heart attack or stroke).

The intake of energy and energy-adjusted dietary intakes of olive oil, wholemeal bread, fish, vegetables, legumes fruit, and protein tended to decrease across DII quintiles (more pro-inflammatory) (Table 2). The highest intakes of red meat, white bread and fat were in the most pro-inflammatory DII quintile. Thus a low DII anti-inflammatory diet consists mainly of plant foods. The Mediterranean Diet Score was inversely associated with the DII, being 6 for the lowest, most anti-inflammatory DII quintile and 2–3 for the most pro-inflammatory DII quintile. The frequency of cereal consumption was inversely associated with DII score in the northern European origin sub-group and positively associated in the southern Europeans. Southern Europeans tended to have much higher olive oil intakes than the northern European origin participants and less difference between DII quintiles.

Table 3 shows the HRs for death associated with DII, overall and by region of origin. Overall there was a positive association between the DII and death from all-causes ($HR_{Q5 \text{ vs } Q1} = 1.16$, 95% CI: 1.08, 1.24; P-trend <0.0001) and from CHD ($HR_{Q5 \text{ vs } Q1} = 1.24$, 95% CI: 1.02, 1.51; P-trend=0.02) (Table 3); these associations appeared largely attributable to the results in the northern European origin sub-group as there was no evidence of an association for those of southern European origin. Positive associations between the DII and CVD mortality were observed at a younger age. At 72 years the $HR_{Q5 \text{ vs } Q1}$ was 1.30 (95% CI: 1.10, 1.53; P-trend=0.001), while at age 78 it was 1.16 (95% CI: 1.01, 1.33; P-trend=0.03).

Overall, there was an inverse association between the MDS and all-cause mortality ($HR_{Q3 \text{ vs } Q1} = 0.86$, 95% CI: 0.80, 0.93; P-trend <0.0001), CVD mortality (Age 72: $HR_{Q3 \text{ vs } Q1} = 0.72$, 95% CI: 0.59, 0.86; P-trend <0.0001) and CHD mortality ($HR_{Q3 \text{ vs } Q1} = 0.81$, 95% CI: 0.65, 0.99; P-trend =0.004) (Table 4). No associations were observed for those of southern European origin except for CVD mortality in the youngest age at death sub-group. The sensitivity analyses did not materially change these findings.

There was no evidence from the BIC that the models with DII fitted better than models using the MDS. Particularly for CVD and CHD mortality, there was some evidence for the MDS models fitting better (Table 5).

Discussion

In our prospective cohort study, for each of total mortality, CVD mortality and CHD mortality, we observed a significant increase in risk for participants reporting a more pro-inflammatory diet. Consistent with these findings, consumption of a more Mediterranean diet was associated with lower risk of death. These associations were limited to participants of northern European origin and the associations with CVD mortality for both the DII and MDS were strongest for younger participants.

Strengths of our study include the large cohort with a wide range of dietary intakes, and minimal loss to follow-up. We were able to adjust for likely important confounders according to our DAG, although residual confounding cannot be ruled out. Weaknesses of this study include those inherent to self-reported dietary data, and the fact that the DII was not computed using the full range of food and nutrient intakes. Most of the items that were missing from the DII calculations were phytochemicals and herbs, which are considered anti-inflammatory. However, other studies have reported associations between DII based on fewer items and inflammatory biomarkers [18, 23], and we have previously reported DII to be weakly but significantly correlated with plasma hs-CRP in a sub-group of the MCCS [24]. A further limitation is that diet was assessed using a single FFQ administered at baseline so we were unable to account for possible changes in dietary intakes over follow-up.

Our observations that DII score was positively associated, and MDS inversely associated, with total, CVD and CHD mortality are consistent with previous studies [1, 3]. We did not find evidence that DII was more strongly associated with the outcomes than the MDS,

according to the BIC. Given that participants in our cohort consuming a diet with a low DII consumed more fruit, vegetables, legumes, fish and olive oil, and less meat, all of which are also features of a Mediterranean diet, it is possible that the DII is a marker of a more healthy diet, working through a variety of mechanisms. We have previously reported that the correlation between the DII and the MDS in our cohort is -0.45 (Spearman's rho) [24]. The PREDIMED study identified improved blood pressure, insulin sensitivity, lipid profile and inflammatory biomarker levels after 3 months on a Mediterranean diet. There was also evidence that the intervention improved the lipoprotein profile and reduced the concentration of oxidatively modified LDL particles at 3 months [25]. The DII may also work via these mechanisms, and independently of CRP as both the DII and the MDS tend to identify similar, plant-based diets as 'better'. Other possible benefits of a low-DII diet, such as improving blood lipid profile, were also noted by Boden et al. [5] as potentially contributing to the observed inverse association between DII and myocardial infarction for men.

For participants who survived to age 83, there was no association between CVD mortality and diet scores, but significant associations were seen for younger ages. Few studies have investigated whether apparent benefits of 'healthy' diets are maintained into older ages. Two cross-sectional studies examining adherence to national dietary guidelines in the US and France found associations with Metabolic Syndrome only for participants aged less than 55 years in the US [26] and less than 50 years in France [27]. In contrast, a large collaborative study of a Healthy Diet Index (HDI) based on adherence to the 2003 WHO dietary guidelines and CVD mortality using data from 10 cohorts across Europe and America found a marginally stronger association for participants over 70 years of age relative to those aged between 60 and 70 years at baseline [28].

It is of interest that neither dietary score was associated with mortality outcomes for southern Europeans. One possibility is that the dietary patterns underlying similar scores differed for the mainly Australian born and the southern European participants. Although the intakes of most foods tended to trend in the same direction across quintiles of DII for all participants in our study, for some 'healthy' items, such as olive oil, there was a tendency for intakes to be higher for Greek and Italian participants, and the intake of cereal foods trended in opposite directions depending on region of origin. We have previously noted that southern European migrants in the MCCS reported their diets with more error than participants of northern European origin [29], which may contribute to the lack of association between diet scores and mortality for these participants. Another possible explanation is that the missing items in the DII calculations, which were largely plant-based, resulted in misclassification towards higher DII for the southern Europeans relatively more than for the other participants. With regard to the MDS, it may be that southern Europeans had a smaller range of scores than participants of northern European origin. The PREDIMED Study demonstrated the benefits of Mediterranean diets in a southern European population [2] and the lack of association we report here may be due to the weaknesses in measurement and scoring of dietary data in the southern Europeans.

We have shown that stronger adherence to a Mediterranean style diet or one with low inflammatory potential is associated with reduced mortality overall and from CVD or CHD. Both diets may be associated with outcomes through inflammation as well as by other

mechanisms. Our findings are consistent with the consensus that plant-based diets are beneficial for health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Cases and their vital status were ascertained through the Victorian Cancer Registry (VCR) and the Australian Institute of Health and Welfare (AIHW), including the National Death Index and the Australian Cancer Database.

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More Mediterranean or less inflammatory diets associated with lower mortality.

DII score was not more strongly associated with mortality than was the MDS.

The results are consistent with plant-based diets being beneficial for health.

Figure 1.

Sample flowchart, The Melbourne Collaborative Cohort Study, 1990 to 2013

Table 1

Baseline characteristics by Dietary Inflammatory Index (DII) quintile, the Melbourne Collaborative Cohort Study, 1990 to 2013.

	DII quintile				
	Q1	Q2	Q3	Q4	Q5
N	7907	7906	7907	7906	7906
DII, range	-5.0, -2.4	-2.4, -1.5	-1.5, -0.5	-0.5, 0.7	0.7, 4.9
Age, mean(SD)	56.0 (8.8)	55.4 (8.7)	55.2 (8.7)	54.9 (8.6)	55.0 (8.5)
MAP mmHg, mean(SD)	96.3 (12.7)	95.9 (12.8)	96.2 (12.9)	96.7 (13.0)	97.3 (13.2)
Cholesterol mmol/L, mean(SD)	5.4 (1.0)	5.5 (1.0)	5.5 (1.0)	5.6 (1.1)	5.7 (1.1)
BMI kg/m ² , mean(SD)	26.3 (4.2)	26.5 (4.2)	26.8 (4.4)	27.1 (4.5)	27.6 (4.6)
Male, n(%)	3511 (44.4)	3264 (41.3)	3146 (39.8)	3121 (39.5)	3009 (38.1)
Country of birth, n(%)					
Australia/NZ	6319 (79.9)	5886 (74.4)	5699 (72.1)	5164 (65.3)	4246 (53.7)
UK	666 (8.4)	622 (7.9)	553 (7.0)	546 (6.9)	498 (6.3)
Italy	353 (4.5)	657 (8.3)	915 (11.6)	1275 (16.1)	1926 (24.4)
Greece	569 (7.2)	741 (9.4)	740 (9.4)	921 (11.6)	1236 (15.6)
SEIFA, n(%)					
Q1 (most disadvantaged)	1237 (15.6)	1238 (15.7)	1347 (17.0)	1541 (19.5)	1778 (22.5)
Q2	1396 (17.7)	1571 (19.9)	1625 (20.6)	1777 (22.5)	1923 (24.3)
Q3	1217 (15.4)	1224 (15.5)	1221 (15.4)	1237 (15.6)	1321 (16.7)
Q4	1591 (20.1)	1574 (19.9)	1472 (18.6)	1382 (17.5)	1315 (16.6)
Q5 (least disadvantaged)	2466 (31.2)	2299 (29.1)	2242 (28.4)	1969 (24.9)	1569 (19.8)
Education, n(%)					
Primary school	865 (10.9)	1102 (13.9)	1326 (16.8)	1738 (22.0)	2500 (31.6)
Some high/technical school	2942 (37.2)	2962 (37.5)	3123 (39.5)	3048 (38.6)	3034 (38.4)
Completed high/technical school	1768 (22.4)	1766 (22.3)	1677 (21.2)	1647 (20.8)	1341 (17.0)
Degree/diploma	2332 (29.5)	2076 (26.3)	1781 (22.5)	1473 (18.6)	1031 (13.0)
Alcohol consumption, n(%)					
Never drinker	2298 (29.1)	2185 (27.6)	2151 (27.2)	2252 (28.5)	2449 (31.0)
Former drinker	822 (10.4)	826 (10.4)	858 (10.9)	826 (10.4)	830 (10.5)

	DII quintile				
	Q1	Q2	Q3	Q4	Q5
Low intake	3794 (48.0)	3999 (50.6)	4018 (50.8)	3896 (49.3)	3759 (47.5)
Moderate intake	728 (9.2)	613 (7.8)	587 (7.4)	601 (7.6)	497 (6.3)
High intake	265 (3.4)	283 (3.6)	293 (3.7)	331 (4.2)	371 (4.7)
Smoking status, n(%)					
Never	4686 (59.3)	4759 (60.2)	4582 (57.9)	4505 (57.0)	4314 (54.6)
Former	2600 (32.9)	2503 (31.7)	2504 (31.7)	2466 (31.2)	2286 (28.9)
Current	620 (7.8)	644 (8.1)	821 (10.4)	935 (11.8)	1306 (16.5)
Physical activity, n(%)					
0 (none)	1199 (15.2)	1459 (18.5)	1661 (21.0)	1999 (25.3)	2411 (30.5)
>0 to <4 (low)	1418 (17.9)	1505 (19.0)	1629 (20.6)	1685 (21.3)	1722 (21.8)
4 to <6 (moderate)	2943 (37.2)	2940 (37.2)	2828 (35.8)	2707 (34.2)	2635 (33.3)
6 (high)	2347 (29.7)	2002 (25.3)	1789 (22.6)	1515 (19.2)	1138 (14.4)
Personal history of CVD, n(%)	551 (7.0)	463 (5.9)	453 (5.7)	402 (5.1)	459 (5.8)
Personal history of diabetes, n(%)	306 (3.9)	307 (3.9)	285 (3.6)	376 (4.8)	433 (5.5)
Family history of CVD, n(%)	4332 (54.8)	4254 (53.8)	4125 (52.2)	4053 (51.3)	3793 (48.0)

Table 2

Dietary intakes by region of origin and Dietary Inflammatory Index (DII) quintile, the Melbourne Collaborative Cohort Study, 1990 to 2013.

		DII quintile				
		Anti-inflammatory		Pro-inflammatory		
	Region of origin	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)
Olive oil ^a	N. Europe	34 (0, 61)	33 (0, 59)	32 (0, 58)	21 (0, 57)	0.0 (0.0, 55.8)
	S. Europe	104 (35, 235)	102 (35, 182)	100 (35, 178)	99 (56, 175)	97 (54, 176)
Cereal products ^b	N. Europe	33 (26, 42)	32 (25, 41)	32 (25, 41)	31 (24, 40)	30 (22, 39)
	S. Europe	24 (17, 34)	26 (19, 35)	28 (21, 36)	29 (22, 38)	30 (21, 39)
White bread ^b	N. Europe	0.6 (0.0, 4.8)	0.9 (0.0, 5.7)	1.2 (0.0, 6.6)	2.9 (0.0, 7.5)	3.9 (0.6, 8.6)
	S. Europe	4.4 (0.0, 10.5)	5.4 (0.4, 12.5)	6.3 (0.6, 14.8)	7.1 (1.1, 16.2)	8.0 (1.3, 15.8)
Wholemeal bread ^b	N. Europe	6.9 (3.1, 15.6)	6.2 (2.4, 15.0)	5.6 (0.9, 8.7)	3.7 (0.5, 8.1)	1.4 (0.0, 7.3)
	S. Europe	4.9 (0.3, 11.0)	3.7 (0.0, 7.9)	2.8 (0.0, 7.6)	1.1 (0.0, 7.7)	0.6 (0.0, 5.6)
Dairy products ^b	N. Europe	37 (25, 49)	37 (23, 49)	36 (22, 51)	36 (21, 52)	36 (18, 55)
	S. Europe	26 (15, 36)	25 (15, 36)	26 (15, 37)	25 (14, 35)	22 (12, 36)
Red meat ^b	N. Europe	6.5 (4.6, 8.8)	7.0 (5.0, 9.3)	7.3 (5.2, 9.7)	7.7 (5.6, 10.1)	8.1 (5.8, 10.7)
	S. Europe	8.0 (5.4, 11.9)	8.2 (5.7, 11.6)	8.1 (5.8, 11.2)	8.2 (5.9, 11.0)	8.4 (6.1, 11.3)
Fish ^b	N. Europe	1.6 (1.0, 2.9)	1.5 (1.0, 2.3)	1.5 (1.0, 2.1)	1.3 (0.9, 2.0)	1.2 (0.6, 1.9)
	S. Europe	2.4 (1.4, 4.2)	1.9 (1.1, 3.2)	1.7 (1.0, 2.8)	1.6 (1.1, 2.5)	1.4 (1.0, 2.2)
Vegetables ^b	N. Europe	6.4 (4.8, 8.4)	5.6 (4.2, 7.3)	4.9 (3.7, 6.5)	4.2 (3.0, 5.7)	3.2 (2.2, 4.5)
	S. Europe	8.8 (6.5, 12.0)	7.1 (5.0, 9.5)	6.0 (4.3, 7.7)	5.1 (3.7, 6.7)	3.8 (2.6, 5.3)
Legumes ^b	N. Europe	3.8 (2.6, 5.9)	3.5 (2.4, 5.4)	3.3 (1.9, 4.8)	3.1 (1.3, 4.3)	2.2 (1.1, 3.9)
	S. Europe	5.8 (2.9, 10.1)	3.9 (2.2, 6.7)	3.0 (1.9, 5.2)	2.7 (1.8, 4.2)	2.4 (1.6, 3.5)
Fruit ^b	N. Europe	4.3 (2.9, 6.2)	4.0 (2.7, 5.6)	3.5 (2.2, 5.1)	2.9 (1.9, 4.4)	2.1 (1.2, 3.3)

DII quintile						
Region of origin	Q1 Anti-inflammatory		Q2		Q3	
	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)
S. Europe	6.4 (4.5, 9.1)	5.8 (4.0, 8.4)	5.1 (3.5, 7.3)	4.4 (3.0, 6.2)	2.8 (1.6, 4.4)	
N. Europe	2.7 (0.0, 14.8)	3.1 (0.0, 15.2)	3.2 (0.0, 16.2)	3.4 (0.0, 17.2)	3.7 (0.0, 18.4)	
S. Europe	0.0 (0.0, 13.3)	0.0 (0.0, 12.4)	0.0 (0.0, 14.5)	0.0 (0.0, 16.2)	0.0 (0.0, 15.3)	
Glycaemic index						
N. Europe	48 (46, 50)	48 (46, 51)	49 (46, 52)	49 (47, 52)	50 (47, 54)	
S. Europe	47 (45, 50)	48 (46, 51)	49 (46, 52)	50 (47, 53)	50 (47, 54)	
Protein ^c						
N. Europe	99 (92, 108)	98 (90, 106)	97 (89, 105)	96 (87, 105)	94 (85, 104)	
S. Europe	111 (98, 126)	105 (95, 118)	102 (93, 113)	100 (91, 110)	98 (88, 109)	
Fat ^c						
N. Europe	79 (70, 88)	79 (71, 88)	81 (72, 89)	83 (74, 91)	87 (78, 96)	
S. Europe	81 (69, 92)	80 (70, 92)	80 (70, 90)	82 (72, 93)	88 (76, 99)	
Carbohydrate ^c						
N. Europe	256 (232, 278)	255 (231, 278)	252 (227, 276)	246 (221, 271)	234 (208, 259)	
S. Europe	241 (205, 273)	243 (213, 276)	247 (217, 276)	242 (215, 272)	231 (201, 260)	
Energy (kJ/day)						
N. Europe	11613 (9734, 13975)	9766 (8306, 11477)	8633 (7337, 10130)	7615 (6380, 9084)	6257 (5179, 7567)	
S. Europe	12832 (10225, 15514)	10581 (8740, 12422)	9055 (7720, 10709)	7894 (6679, 9277)	6079 (5033, 7430)	
Mediterranean Diet						
N. Europe	6.0 (5.0, 7.0)	5.0 (4.0, 6.0)	4.0 (3.0, 5.0)	3.0 (2.0, 4.0)	2.0 (2.0, 3.0)	
Score						
S. Europe	6.0 (5.0, 7.0)	6.0 (5.0, 7.0)	5.0 (4.0, 6.0)	4.0 (3.0, 5.0)	3.0 (2.0, 4.0)	

^a millilitres/week^b times/week^c grams/day^d N.Europe includes Australia/New Zealand/UK; S.Europe includes Greece/Italy

Table 3

All-cause, cardiovascular disease (CVD) and coronary heart disease (CHD) mortality by Dietary Inflammatory Index (DII) quintile. The Melbourne Collaborative Cohort Study, 1990 to 2013.

DII	All			N. Europe			S. Europe		
	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)
All-cause mortality									
<20pc	1633	1.00		1405	1.00		228	1.00	
20–40th	1458	0.96	(0.90, 1.03)	1156	0.96	(0.89, 1.04)	302	0.93	(0.78, 1.10)
40–60th	1471	1.00	(0.93, 1.07)	1146	1.02	(0.94, 1.10)	325	0.87	(0.73, 1.03)
60–80th	1558	1.10	(1.02, 1.18)	1093	1.13	(1.04, 1.22)	465	0.95	(0.81, 1.11)
>80th	1637	1.16	(1.08, 1.24)	986	1.24	(1.14, 1.35)	651	0.95	(0.82, 1.10)
Linear model	7757	1.04	(1.02, 1.05)	5786	1.05	(1.04, 1.07)	1971	1.00	(0.97, 1.03)
P-trend		<0.0001			<0.0001			0.96	
CVD mortality									
Age 72 years									
<20pc	438	1.00		374	1.00		64	1.00	
20–40th	423	1.12	(0.95, 1.32)	335	1.12	(0.93, 1.35)	88	1.05	(0.71, 1.55)
40–60th	370	1.03	(0.87, 1.22)	280	1.03	(0.85, 1.25)	90	0.96	(0.65, 1.40)
60–80th	416	1.19	(1.01, 1.40)	281	1.21	(1.00, 1.45)	135	1.05	(0.73, 1.51)
>80th	434	1.30	(1.10, 1.53)	264	1.40	(1.16, 1.69)	170	1.05	(0.75, 1.48)
Linear model	2081	1.05	(1.02, 1.09)	1534	1.07	(1.03, 1.11)	547	1.01	(0.95, 1.07)
P-trend		0.001			0.0005			0.70	
Age 78 years									
<20pc	438	1.00		374	1.00		64	1.00	
20–40th	423	1.07	(0.93, 1.22)	335	1.07	(0.93, 1.25)	88	1.04	(0.75, 1.44)
40–60th	370	0.96	(0.83, 1.10)	280	0.97	(0.83, 1.13)	90	0.88	(0.64, 1.22)
60–80th	416	1.13	(0.99, 1.30)	281	1.14	(0.97, 1.33)	135	1.04	(0.77, 1.41)
>80th	434	1.16	(1.01, 1.33)	264	1.29	(1.10, 1.51)	170	0.90	(0.67, 1.21)
Linear model	2081	1.03	(1.00, 1.06)	1534	1.05	(1.02, 1.09)	547	0.98	(0.93, 1.03)
P-trend		0.03			0.003			0.45	
Age 83 years									

DII	All			N. Europe			S. Europe		
	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)
<20pc	438	1.00		374	1.00		64	1.00	
20–40th	423	1.03	(0.88, 1.21)	335	1.03	(0.87, 1.23)	88	1.03	(0.69, 1.54)
40–60th	370	0.90	(0.76, 1.06)	280	0.92	(0.76, 1.11)	90	0.83	(0.55, 1.24)
60–80th	416	1.08	(0.92, 1.28)	281	1.08	(0.90, 1.31)	135	1.03	(0.72, 1.49)
>80th	434	1.05	(0.89, 1.24)	264	1.20	(0.99, 1.47)	170	0.79	(0.54, 1.14)
Linear model	2081	1.01	(0.98, 1.05)	1534	1.04	(1.00, 1.08)	547	0.95	(0.89, 1.02)
P-trend		0.45			0.08				0.17
CHD mortality									
<20pc	217	1.00		183	1.00		34	1.00	
20–40th	207	1.09	(0.90, 1.32)	158	1.07	(0.86, 1.32)	49	1.10	(0.71, 1.70)
40–60th	188	1.02	(0.84, 1.24)	140	1.03	(0.82, 1.28)	48	0.92	(0.59, 1.43)
60–80th	210	1.19	(0.98, 1.45)	144	1.24	(1.00, 1.55)	66	1.00	(0.66, 1.51)
>80th	218	1.24	(1.02, 1.51)	134	1.40	(1.12, 1.75)	84	0.94	(0.63, 1.40)
Linear model	1040	1.05	(1.01, 1.09)	759	1.08	(1.03, 1.13)	281	0.98	(0.91, 1.05)
P-trend		0.02			0.001				0.57

All models adjusted for country of birth, SEIFA quintile, alcohol intake, personal history of diabetes, stratified by sex and personal and family history of CVD; CVD model includes DII x attained age interaction. P-interaction for country of birth and DII = 0.01 (All-cause model); 0.27 (CHD model). CVD model: separate models fitted for those of northern and southern European origin

Table 4

All-cause, cardiovascular disease (CVD) and coronary heart disease (CHD) mortality by Mediterranean Diet Score (MDS) quintile. The Melbourne Collaborative Cohort Study, 1990 to 2013.

MDS	All			N. Europe			S. Europe		
	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)
All-cause mortality									
MDS 0–3	2755	1.00		2073	1.00		682	1.00	
MDS 4–6	4098	0.91	(0.87, 0.96)	3050	0.90	(0.85, 0.95)	1048	0.94	(0.86, 1.04)
MDS 7–9	904	0.86	(0.80, 0.93)	663	0.83	(0.76, 0.90)	241	0.98	(0.84, 1.13)
Linear model	7757	0.96	(0.95, 0.98)	5786	0.96	(0.94, 0.97)	1971	0.98	(0.96, 1.01)
P-trend		<0.0001			<0.0001			0.21	
CVD mortality									
Age 72 years									
MDS 0–3	775	1.00		583	1.00		192	1.00	
MDS 4–6	1062	0.81	(0.72, 0.90)	773	0.79	(0.70, 0.90)	289	0.85	(0.69, 1.05)
MDS 7–9	244	0.72	(0.59, 0.86)	178	0.68	(0.54, 0.85)	66	0.82	(0.59, 1.16)
Linear model	2081	0.93	(0.90, 0.96)	1534	0.92	(0.89, 0.95)	547	0.95	(0.90, 1.00)
P-trend		<0.0001			<0.0001			0.05	
Age 78 years									
MDS 0–3	775	1.00		583	1.00		192	1.00	
MDS 4–6	1062	0.85	(0.78, 0.94)	773	0.83	(0.74, 0.92)	289	0.96	(0.79, 1.16)
MDS 7–9	244	0.84	(0.72, 0.97)	178	0.79	(0.67, 0.94)	66	0.98	(0.73, 1.31)
Linear model	2081	0.95	(0.93, 0.98)	1534	0.95	(0.92, 0.97)	547	0.98	(0.94, 1.03)
P-trend		0.0002			0.0001			0.52	
Age 83 years									
MDS 0–3	775	1.00		583	1.00		192	1.00	
MDS 4–6	1062	0.89	(0.79, 1.00)	773	0.85	(0.75, 0.98)	289	1.06	(0.83, 1.36)
MDS 7–9	244	0.95	(0.80, 1.13)	178	0.91	(0.74, 1.11)	66	1.14	(0.79, 1.63)
Linear model	2081	0.98	(0.95, 1.01)	1534	0.97	(0.93, 1.00)	547	1.01	(0.95, 1.08)
P-trend		0.12			0.05			0.65	

MDS	All			N. Europe			S. Europe		
	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)	Deaths	HR	(95% CI)
CHD mortality									
MDS 0–3	393	1.00		295	1.00		98	1.00	
MDS 4–6	527	0.83	(0.73, 0.95)	379	0.80	(0.69, 0.94)	148	0.91	(0.70, 1.18)
MDS 7–9	120	0.81	(0.65, 0.99)	85	0.76	(0.60, 0.97)	35	0.93	(0.63, 1.38)
Linear model	1040	0.95	(0.92, 0.98)	759	0.94	(0.90, 0.98)	281	0.98	(0.92, 1.05)
P-trend		0.004			0.002			0.60	

All models adjusted for country of birth, SEIFA quintile, alcohol intake, personal history of diabetes, stratified by sex and personal and family history of CVD; CVD model includes DII x attained age interaction. P-interaction for country of birth and DII = 0.17 (All-cause model); 0.59 (CHD model). CVD model: separate models fitted for those born in northern and southern Europe

Table 5

Comparison of model fits using the Mediterranean Diet Score (MDS) or the Dietary Inflammatory Index (DII), The Melbourne Collaborative Cohort Study, 1990 to 2013.

		DII			MDS			Difference ^a		
Mortality outcome		df	AIC	BIC	df	AIC	BIC	AIC	BIC	
All-cause mortality										
All	Categorical	14	117880.7	118000.8	12	117891.1	117994.2	-10.4	6.6	
	Continuous	11	117881.8	117976.3	11	117876.2	117970.6	5.6	5.7	
Interaction with region of origin	Categorical	18	117875.6	118030.1	14	117891.5	118011.7	-15.9	18.4	
	Continuous	12	117874.1	117977.1	12	117874.6	117977.6	-0.5	-0.5	
CVD mortality ^b										
All	Categorical	18	30162.8	30317.3	14	30149.2	30269.4	13.6	47.9	
	Continuous	12	30156.6	30259.6	12	30141.1	30244.1	15.5	15.4	
N.European	Categorical	17	21378.8	21520.2	13	21367.6	21475.7	11.2	44.5	
	Continuous	11	21371.8	21463.2	11	21361.2	21452.7	10.6	10.6	
S.European	Categorical	17	6499.5	6620.9	13	6493.0	6585.8	6.6	35.1	
	Continuous	11	6490.8	6569.4	11	6488.1	6566.7	2.7	2.7	
CHD mortality										
All	Categorical	14	14741.2	14861.4	12	14735.7	14838.7	5.5	22.6	
	Continuous	11	14736.8	14831.2	11	14734.2	14828.6	2.6	2.6	
Interaction with region of origin	Categorical	18	14744.0	14898.5	14	14738.7	14858.9	5.3	39.6	
	Continuous	12	14734.1	14837.2	12	14735.0	14838.0	-0.9	-0.8	

^a A positive difference in AIC or BIC indicates that the MDS provides a better model fit than the DII

^b Separate models were fitted by region of origin as the CVD mortality models included an interaction between the dietary score and the underlying time scale to account for non-proportional hazards in the dietary score