Public Health Nutrition: 24(18), 6157–6168 doi:10.1017/S1368980021001713

Diet scores and prediction of general and abdominal obesity in the Melbourne collaborative cohort study

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Submitted 22 September 2020: Final revision received 4 March 2021: Accepted 14 April 2021: First published online 20 April 2021

Abstract

Objective: To ascertain which of the Alternative Healthy Eating Index (AHEI) 2010, Dietary Inflammatory Index (DII®) and Mediterranean Diet Score (MDS) best predicted BMI and waist-to-hip circumference ratio (WHR).

Design: Body size was measured at baseline (1990–1994) and in 2003–2007. Diet was assessed at baseline using a FFQ, along with age, sex, socio-economic status, smoking, alcohol drinking, physical activity and country of birth. Regression coefficients and 95 % CI for the association of baseline dietary scores with follow-up BMI and WHR were generated using multivariable linear regression, adjusting for baseline body size, confounders and energy intake.

Setting: Population-based cohort in Melbourne, Australia.

Participants: Included were data from 11 030 men and 16 774 women aged 40–69 years at baseline.

Results: Median (IQR) follow-up was 11·6 (10·7–12·8) years. BMI and WHR at follow-up were associated with baseline DII® (Q5 *v.* Q1 (BMI 0·41, 95 % CI 0·21, 0·61) and WHR 0·009, 95 % CI 0·006, 0·013)) and AHEI (Q5 *v.* Q1 (BMI −0·51, 95 % CI −0·68, −0·35) and WHR −0·011, 95 % CI −0·013, −0·008)). WHR, but not BMI, at follow-up was associated with baseline MDS (Group 3 most Mediterranean *v.* G1 (BMI −0·05, 95 % CI −0·23, 0·13) and WHR −0·004, 95 % CI −0·007, −0·001)). Based on Akaike's Information Criterion and Bayesian Information Criterion statistics, AHEI was a stronger predictor of body size than the other diet scores.

Conclusions: Poor quality or pro-inflammatory diets predicted overall and central obesity. The AHEI may provide the best way to assess the obesogenic potential of diet.

Keywords
Diet score
FFQ
Overall and central obesity
Prospective
Nutritional epidemiology

The prevalence of obesity is increasing at an alarming rate, across both developed and developing countries[1,2]. Obesity increases risk of many chronic diseases including type 2 diabetes, CVD, liver and kidney diseases, neurodegenerative diseases and at least thirteen types of cancer, including more common ones such as colorectal and post-menopausal breast cancer[3]. Central (abdominal) obesity is also strongly associated with morbidity and mortality[4].

In Australia in 2017–2018, 67 % of adults were overweight or obese, 10 % higher than in 1995[5]. In 2011–2012, obesity cost the Australian economy an estimated $8·6 billion, not including the cost of reduced well-being and foregone earnings[6]. Further, it has been estimated that 14 % of the 2020 burden of disease due to overweight and obesity could have been avoided if the BMI of the population at risk in 2011 was reduced by 1 kg/m^2[7].

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The global obesity epidemic is understood to be a consequence of lifestyle changes, favouring high intakes of energy-dense food and less physical activity than in the past, and often interacting with genetic susceptibility[8]. Lifestyle modification, predominantly through diet and exercise, is a frontline strategy to manage obesity and obesity-related conditions[6]. The question remains – what diet composition is best to prevent weight gain or lead to weight loss? Early research focused on macronutrient composition[9]. It is now well established that in terms of weight loss, it is energy deficit rather than diet composition that is most important[9,10]. However, energy-dense foods tend to encourage overconsumption of energy, while less energy-dense foods high in fibre and non-starch polysaccharides such as whole grains, and in water such as fruit and vegetables, may help limit energy consumption[11].

As in other areas of nutritional epidemiology, recent studies of obesity have tended to focus on measures of overall diet rather than single foods or nutrients[12]. There are many approaches to look at diet using scores. The Alternative Healthy Eating Index (AHEI)-2010 assesses adherence to the US dietary guidelines, which are based on minimising the risk of chronic disease[13]. The Dietary Inflammatory Index (DII®) is based on literature identifying foods and nutrients associated with pro-inflammatory biomarkers, which are then combined into an index of dietary inflammatory potential[14]. The Mediterranean Diet Score (MDS) was developed to assess adherence to the traditional diet of Crete which was identified as being associated with low CVD risk in the seven Countries Study[15]. Thus, these three diet scores (AHEI, MDS and DII) have very different theoretical bases, but all tend to be consistent in favouring consumption of fruit, vegetables and whole grains over meat and saturated fats.

Studies of these diet indices in relation to obesity provide some support for AHEI being inversely associated with obesity over time[16], especially in sub-groups identified as genetically susceptible to obesity[17,18]. In an 8-year follow-up of the Seguimiento University of Navarra (SUN) study cohort, a more pro-inflammatory diet, measured using the DII, was associated with greater yearly weight gain and increased risk of overweight and obesity[19]. In cross-sectional analysis of the Prevención con Dieta Mediterránea Study, a higher DII was associated with obesity[20]. A recent meta-analysis looking at DII and BMI or obesity also supported a positive association across twenty-two studies, including thirteen cohorts[21]. In the European Prospective Investigation into Cancer and Nutrition–Physical Activity, Nutrition, Alcohol Consumption, Cessation of Smoking, Eating Out of Home, and Obesity (EPIC-PANACEA) study, higher adherence to a Mediterranean diet was associated with reduced risk of weight gain and of developing overweight or obesity[22].

To our knowledge, none of the studies compared the three indices in the same cohort and looked, simultaneously, at both overall and central (abdominal) obesity. Hence, the aim of this study was to investigate which of the three diet scores – AHEI, DII and MDS – was a better predictor of obesity, assessed as BMI or waist-to-hip circumference ratio (WHR), using data from the Melbourne Collaborative Cohort Study (MCCS).

Methods

Study participants

The MCCS is a prospective cohort with 41 513 participants recruited in 1990–1994, via the electoral roll and direct approach through churches, clubs and ethnicity-specific mass media. A detailed description of the cohort, including recruitment, data collection and follow-up, has been presented elsewhere[23]. At baseline, physical measurements were taken and other information was collected using interviewer-administered questionnaires. At follow-up in 2003–2007, physical measurements (not including height) were again taken and other information was collected using self-administered questionnaires.

We excluded participants who did not attend the second follow-up, those who had other chronic diseases at baseline or developed over follow-up conditions that may affect body size, including diabetes, asthma, cancer, stroke, angina, heart attack or had cardiac surgery (Fig. 1).

Dietary assessment

Information on dietary intake was collected using a 121-item self-administered FFQ[24]. Sex-specific average portion sizes were assigned to each food item, and daily frequencies of some fruits were seasonally adjusted. Nutrient composition data were largely derived from the Australian food composition tables[25]. Because these tables do not include folate or vitamin E, we used British data for these[26]. Fatty acid data were sourced from Royal Melbourne Institute of Technology[27] and carotenoid data from the United States Department of Agriculture[28]. Mean daily nutrient intakes were obtained by multiplying the daily frequency of each food item by the nutrient composition for an average sex-specific portion size. Comparisons of antioxidant and fatty acid intakes against plasma biomarkers for this FFQ have been described previously[29,30].

Three diet scores were calculated from the estimated food and nutrient intakes. The DII is based on reviewing and scoring literature assessing the association between various dietary components including nutrients, spices and food items and six inflammatory biomarkers: IL-1β, IL-4, IL-6, IL-10, TNF-α and C-reactive protein, giving a single score assessing anti- or pro-inflammatory potential of the overall diet[14]. The DII has been validated in various populations[31–33]. DII values for MCCS participants were calculated using twenty-nine of forty-five possible foods and nutrients. The higher the score, the more pro-inflammatory the diet. The AHEI-2010 for the MCCS was

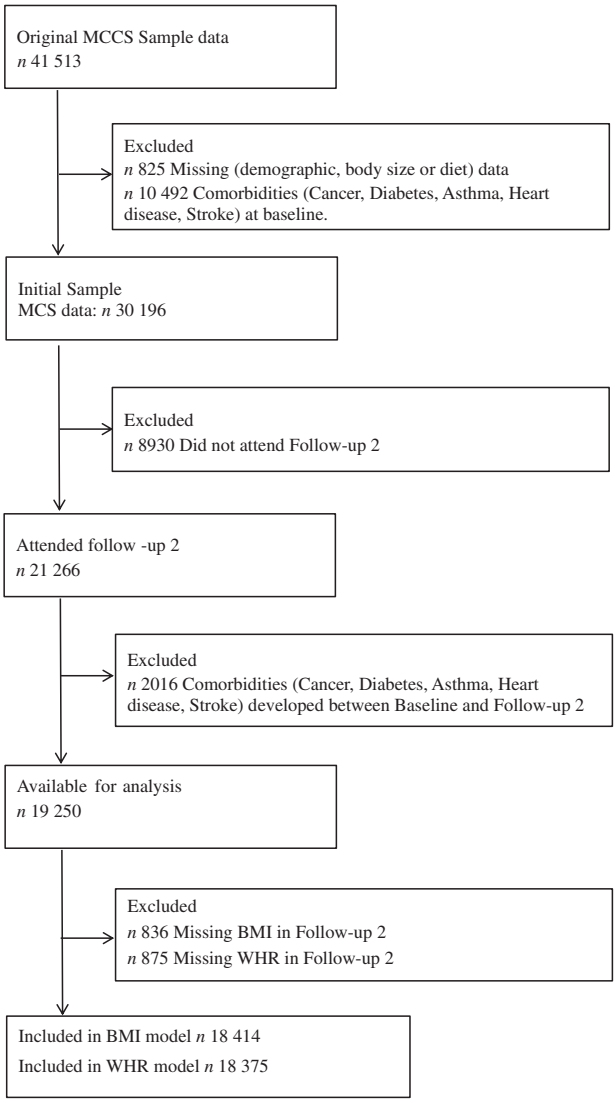

Fig. 1 Participant flow chart

calculated using the method described by Chiuve *et al.*[13], which defines eleven dietary components with scores of 0–10 for each. A higher component score represents greater consumption of vegetables, fruit, whole grains, nuts and legumes, *n*-3 and PUFA, moderate alcohol intake, and a lower consumption of sugar-sweetened beverages and fruit juice, red and processed meat, trans fat and Na. The overall AHEI-2010 score is the sum of the eleven dietary component scores, ranging from 0 (minimum score) to 110 (maximum score). The MDS assesses how closely the diet adheres to the traditional Cretan diet. Intakes of cereals, legumes, fruits, vegetables, olive oil and fish above the sex-specific medians for the cohort were scored one, intakes below or equal to the medians scored zero. Intakes of dairy and meat above the sex-specific median scored zero, and below or equal to the median scored one. Alcohol intake within the range of 10 g/d–50 g/d for men and 5 g/d–25 g/d for women also scored one. Intakes outside these ranges scored zero. Although the original MDS included dietary monounsaturated to saturated fat ratio as the ninth component, we used intake of olive oil because in Australia, red meat is an important source of MUFA[34]. The total score ranged from 0 to 9 with a higher score indicating stronger compliance with a traditional Mediterranean diet[15].

Body size assessment

Standard protocols were used to measure height, weight, waist and hip circumferences at baseline[23] from which BMI and WHR were calculated and classified as follows: BMI ≥ 25 kg/m^2 (overweight), BMI ≥ 30 kg/m^2 (obese) and WHR $\geq 0{\cdot}90$ (men); $\geq 0{\cdot}85$ (women) raised WHR. At follow-up, these measures, except height, were repeated. Baseline height was used to calculate BMI at follow-up.

We chose to use WHR rather than waist circumference as our measure of central obesity as it is less closely correlated with BMI in the MCCS (BMI and waist $r = 0{\cdot}82$, BMI and WHR $r = 0{\cdot}37$) and may represent a more distinct measure of body fat distribution.

Confounders

Information on country of birth, age, smoking, alcohol consumption and physical activity was derived from questionnaires at baseline. Participants were divided according to country of origin as Australia/New Zealand/others, UK and Southern European. Socio-economic position was represented as quintiles of Socio-Economic Indexes for Areas Index of Relative Socio-economic Disadvantage based on postcode at baseline.

Self-reported health information covering diabetes, asthma, cancer, stroke, angina, heart attack and cardiac surgery was collected at both surveys and used to exclude participants with conditions that could impact on body size. Energy intake estimated from the FFQ was also included as a confounder.

Statistical analysis

Univariate associations of baseline characteristics and the dietary indices were evaluated using one-way ANOVA for continuous variables and χ^2 test for categorical variables. Assumptions of linear relationships between dietary indices and anthropometric indices were assessed by fitting both linear and quadratic curves; the results showed linear relationship between the dietary indices with BMI and WHR. Regression coefficients and 95 % CI for the association of each dietary score at baseline with BMI and WHR at follow-up 2 were generated using linear regression models adjusted for age, energy intake, BMI or WHR at baseline as continuous measure and sex (male, female), Socio-Economic Indexes for Areas (quintiles 1–5), smoking status (never, former and current), alcohol drinking status (never, former and current) and physical activity level (0, >0 and <4, ≥4 and <6, ≥6) as categorical.

To test linear trends, we assigned the median daily equivalent frequency to each of the categories of dietary indices and used this as a continuous variable in the linear regression models. Changes in the outcome (BMI and WHR) by one category increment in the dietary score and the *P* for trend were generated. We used the Akaike information criterion and Bayesian information criteria (BIC) to compare models including each of the diet indices and the grades of evidence proposed by Raftery[35] to interpret these. Statistical analyses were performed using Stata/se release 15 (Stata Corp.).

Results

We excluded 13 679 participants with self-reported conditions at baseline, and six with missing dietary data, which left 27 834 subjects. Of those excluded, 2297 had diabetes, 7060 had asthma, 3734 had cancer, 492 had had a stroke, 845 had angina, 496 had had a heart attack and 98 had had heart bypass surgery (Fig. 1).

Median (IQR) follow-up duration of the participants included in the analysis was 11·6 (10·7–12·8) years. The characteristics of the study participants at baseline by quintiles of each dietary index are presented in Tables 1–3. In general, people with diets who were assessed as healthier were more likely to be older, female (except for DII where there was a higher proportion of women relative to men in the highest scoring (most inflammatory) group), less disadvantaged, Australian, New Zealander or of Northern European origin, non-smokers, more physically active and have WHR and BMI in the normal range.

The least inflammatory DII group (Q1) had the highest energy intake; for AHEI, the healthiest diet (Q5) had the lowest energy intake, and for MDS, the healthiest diet (high score) had the highest energy intake.

The proportion consuming alcohol increased with healthier dietary scores except for DII, where an inverted 'U' shape pattern was observed.

At baseline, 10 539 people (37·9 %) had normal weight (BMI: <25 kg/m^2), 11 991 (43·2 %) were overweight (BMI: 25–29·9 kg/m^2) and 5258 (18·9 %) were obese (BMI: ≥30 kg/m^2) (Tables 1–3). Among those who had normal weight at baseline, 27·4 % were overweight and 1·5 % were obese at follow-up. Among those who were overweight at baseline, 10·4 % were normal weight, 20·3 % were overweight at follow-up and the remainder were obese. Among those who were obese at baseline, 0·5 % were normal weight and 14·2 % were overweight at follow-up, while 85·3 % remained obese. The percentages were similar for both men and women.

At baseline, 18 520 people (66·7 %) had WHR within normal limits and 9264 people (33·3 %) were centrally obese, with high WHR (Tables 1–3). Around one-third (32·5 %) of those with normal WHR at baseline were obese at follow-up and 86·8 % of those with high WHR remained centrally obese at follow-up.

Table 4 presents the association of baseline dietary indices with follow-up BMI in multivariable adjusted models.

Baseline DII was associated with follow-up BMI in the multivariable model. Follow-up BMI increased across DII quintiles (from most least inflammatory to most inflammatory). Change in BMI for one category increment in the DII score was 0·085 (0·045, 0·125) (*P*-trend <0·001). Follow-up BMI decreased across increasing AHEI quintiles (from least healthy to most healthy). Change in BMI for one category increment in the AHEI score was −0·016 (−0·022, −0·01) (*P*-trend <0·001). No association was found between baseline MDS score and follow-up BMI (*P*-trend 0·250).

Table 5 presents the association of dietary indices with follow-up WHR in multivariable models. Follow-up WHR increased across increasing DII quintiles (from least inflammatory to most inflammatory). Change in WHR for one category increment in the DII score was 0·002 (0·001, 0·003); (*P*-trend <0·001). Follow-up WHR decreased across increasing AHEI quintiles (from least healthy to most healthy). Change in WHR for one category increment in the AHEI score was −0·00035 (−0·00044, −0·00026) (*P*-trend <0·001). Baseline MDS score also showed a significant inverse association with follow-up WHR. Change in WHR for one category increment in the MSD score was −0·0009 (−0·0014, −0·00036) (*P*-trend = 0·001).

To assess the model fit for the different dietary scores, Akaike information criterion and BIC are also presented in Tables 4 and 5. These values provide evidence that AHEI was a better predictor of both BMI and WHR at follow-up than the other diet scores, with differences for BIC AHEI relative to BIC MDS and BIC DII being around 20 and 23, respectively, for BMI and 31 and 27 for WHR.

Discussion

We describe for the first time the associations between three theoretically different dietary indices and two measures of body size in the same population to assess whether one particular diet stands out as being associated with body size, and whether overall or central obesity is differently associated with dietary patterns. After adjustment for potential confounders and baseline body size measurements, DII and AHEI were associated with BMI and WHR at follow-up, while MDS was associated with WHR but not with BMI at follow-up. The associations were positive for DII, indicating that a more inflammatory diet at baseline was associated with a higher BMI or WHR at follow-up, while the associations for AHEI and MDS were negative, indicating that diets more closely adhering to the US dietary guidelines or the traditional Cretan diet were associated with lower body size at follow-up. Poorer

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Table 1 Descriptive statistics by Dietary Inflammatory Index (DII) quintile

	DII Q1		DII Q2		DII Q3		DII Q4		DII Q5		Total		
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	Test statistics
Quintile median	−2·9		−1·9		−1·0		−0·1		1·8		−0·9		$P < 0·001$*
IQR	−3·3, −2·7		−2·2, −1·7		−1·2, −0·7		−0·2, 0·4		1·3, 2·6		−2·1, 0·5		
Age													
<50 years	1764	33·3	1951	35·2	2001	36·1	2110	37·2	2078	37·2	9904	35·6	$\chi^2 = 48·4$; df = 8, $P < 0·001$
50–59 years	1726	32·6	1872	33·8	1847	33·3	1955	34·4	1960	34·2	9360	33·7	
≥60 years	1808	34·1	1716	31·0	1701	30·7	1615	28·4	1700	26·9	8540	30·7	
Mean	55·0		54·4		54·3		53·9		54·1		54·3		$P < 0·001$*
SD	8·7		8·6		8·6		8·5		8·4		8·6		
Gender													
Male	2295	43·2	2201	39·7	2166	39·0	2238	39·4	2130	37·1	11 030	39·7	$\chi^2 = 46·2$; df = 4, $P < 0·001$
Female	3003	56·7	3338	60·3	3383	61·0	3442	60·6	3608	62·9	16 774	60·3	
SEIFA quintiles													
SEIFA Q1	813	15·5	842	15·3	897	16·2	1042	18·4	1253	21·9	4847	17·5	$\chi^2 = 372$; df = 16, $P < 0·001$
SEIFA Q2	928	17·7	1072	19·5	1120	20·3	1257	22·2	1404	24·6	5781	20·9	
SEIFA Q3	802	15·3	875	15·9	877	15·9	884	15·6	973	17·0	4411	16·0	
SEIFA Q4	1023	19·5	1097	20·0	1041	18·9	1016	18·0	924	16·2	5101	18·5	
SEIFA Q5	1686	32·1	1613	29·3	1589	28·8	1453	25·7	1158	20·3	7499	27·1	
Country of birth													
AU/NZ/Other	4100	77·4	4023	72·6	3886	70·0	3603	63·4	2910	50·7	18 522	66·6	$\chi^2 = 1400$; df = 8, $P < 0·001$
UK	410	7·7	407	7·4	352	6·3	347	6·1	317	5·5	1833	6·6	
Southern Europe	788	14·9	1109	20·0	1311	23·6	1730	30·5	2511	43·8	7449	26·8	
Smoking status													
Never	3207	60·5	3457	62·4	3289	59·3	3301	58·1	3302	57·6	16 556	59·6	$\chi^2 = 230$; df = 8, $P < 0·001$
Former	1631	30·8	1605	29·0	1657	29·9	1678	29·5	1508	26·3	8079	29·1	
Current	459	8·7	477	8·6	603	10·9	701	12·3	928	16·2	3168	11·4	
Alcohol dinking status													
Never	1521	29·0	1503	27·4	1473	26·8	1606	28·6	1761	31·2	7864	28·6	$\chi^2 = 36$; df = 8, $P < 0·001$
Former	537	10·3	558	10·2	579	10·5	557	9·9	596	10·5	2827	10·3	
Current	3180	60·7	3431	62·5	3452	62·7	3461	61·5	3295	58·3	16 819	61·1	
Physical activity score													
0	829	15·7	1031	18·6	1161	20·9	1478	26·0	1793	31·3	6292	22·6	$\chi^2 = 755$; df = 12, $P < 0·0001$
>0 and <4	981	18·5	1077	19·4	1146	20·7	1189	20·9	1235	21·5	5628	20·2	
≥4 and <6	1886	35·6	2008	36·3	1950	35·1	1871	32·9	1881	32·8	9596	34·5	
≥6	1602	30·2	1423	25·7	1292	23·3	1142	20·1	829	14·8	6288	22·6	
WHR at baseline													
Normal WHR	3664	69·2	3806	68·7	3747	67·6	3720	65·6	3583	62·5	18 520	66·7	$\chi^2 = 75$; df = 4, $P < 0·001$
Raised WHR	1633	30·8	1732	31·3	1795	32·4	1955	34·5	2149	37·5	9264	33·3	
Mean	0·83		0·83		0·83		0·84		0·84		0·84		$P < 0·001$*
SD	0·1		0·1		0·1		0·1		0·1		0·1		
BMI at baseline													
<25	2302	43·5	2323	42·0	2124	38·3	2019	35·6	1771	30·9	10 539	37·9	$\chi^2 = 331$; df = 8, $P < 0·001$
25–29·9	2205	41·6	2335	42·2	2388	43·1	2507	44·2	2556	44·6	11 991	43·2	
≥30·0	791	14·9	878	15·9	1033	18·6	1149	20·3	1407	24·5	5258	18·9	
Mean	26·1		26·3		26·6		26·9		27·5		26·7		$P < 0·001$*
SD	4·0		4·1		4·2		4·3		4·5		4·3		
Energy (kJ/d)													
Mean	12 888		10 341		9109		8031		6299		9274		$P < 0·0001$*
SD	4983		2742		2406		2129		1877		3729		

IQR, interquartile range; SEIFA, Socio-Economic Indexes for Areas; WHR, waist-to-hip circumference ratio.
*P for trend.

Table 2 Descriptive statistics by Alternative Healthy Eating Index (AHEI) quintile

	AHEI Q1		AHEI Q2		AHEI Q3		AHEI Q4		AHEI Q5		Total		
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	Test statistics
Quartile median	51		59		65		71		78		64·5		$P<0·001$*
IQR	46, 53		57, 61		64, 67		69, 72		76, 82		57, 72		
Age													
<50 years	2193	38·7	2190	36·3	1827	34·1	1798	34·1	1896	34·7	9904	35·6	$\chi^2=39$; df = 8, $P<0·001$
50–59 years	1806	31·9	1994	33·1	1855	34·7	1810	34·3	1894	34·6	9359	33·7	
≥60 years	1671	29·5	1849	30·7	1670	31·2	1669	31·6	1681	30·7	8540	30·7	
Mean	53·8		54·2		54·6		54·9		54·5		54·3		$P<0·001$*
SD	8·7		8·6		8·5		8·6		8·4		8·6		
Gender													
Male	3234	57·0	2686	44·5	1882	35·2	1692	32·1	1535	28·1	11 029	39·7	$\chi^2=1300$; df = 4, $P<0·0001$
Female	2436	43·0	3347	55·5	3470	64·8	3585	67·9	3936	71·9	16 774	60·3	
SEIFA quintiles													
SEIFA Q1	1194	21·2	1135	18·9	906	17·0	830	15·8	782	14·4	4847	17·5	$\chi^2=245$; df = 16, $P<0·001$
SEIFA Q2	1270	22·5	1267	21·1	1156	21·7	1084	20·7	1004	18·5	5781	20·9	
SEIFA Q3	904	16·0	1001	16·7	828	15·6	874	16·7	803	14·8	4410	16·0	
SEIFA Q4	1008	17·9	1075	17·9	999	18·8	957	18·3	1062	19·6	5101	18·5	
SEIFA Q5	1266	22·4	1525	25·4	1437	27·0	1498	28·6	1773	32·7	7499	27·1	
Ethnic origin													
AU/NZ/Others	3927	69·3	3913	64·9	3373	63·0	3465	65·7	3844	70·3	18 522	66·7	$\chi^2=158$; df = 8, $P<0·001$
UK	329	5·8	382	6·3	340	6·4	342	6·5	440	8·0	1833	6·6	
Southern Europe	1414	24·9	1738	28·8	1639	30·6	1470	27·9	1187	1·7	7448	26·7	
Smoking status													
Never	2934	51·8	3510	58·2	3336	62·3	3383	64·1	3393	62·0	16 556	59·6	$\chi^2=516$; df = 8, $P<0·001$
Former	1716	30·3	1723	28·6	1467	27·4	1442	27·3	1730	31·6	8078	29·1	
Current	1020	18·0	799	13·3	549	10·3	452	8·6	348	6·4	3168	11·4	
Alcohol dinking status													
Never	1641	29·6	1840	30·8	1795	33·9	1427	27·2	1161	21·3	7864	28·6	$\chi^2=259$; df = 8, $P<0·001$
Former	636	11·5	593	9·9	531	10·0	507	9·7	560	10·3	2827	10·3	
Current	3267	58·9	3544	59·3	2972	56·1	3308	63·1	3727	68·4	16 818	61·1	
Physical activity score													
0	1670	29·5	1483	24·6	1256	23·5	1038	19·7	845	15·5	6292	22·6	$\chi^2=515$; df = 12, $P<0·001$
>0 and <4	1178	20·8	1316	21·8	1054	19·7	1037	19·7	1043	19·1	5628	20·2	
≥4 and <6	1800	31·8	2035	33·7	1879	35·1	1930	36·6	1951	35·7	9595	34·5	
≥6	1022	18·0	1199	19·9	1163	21·7	1272	24·1	1632	29·8	6288	22·6	
WHR at baseline													
Normal WHR	3057	54·0	3780	62·7	3665	68·5	3798	72·0	4220	77·2	18 520	66·7	$\chi^2=801$; df = 4, $P<0·0001$
Raised WHR	2607	46·0	2248	37·3	1684	31·5	1476	28·0	1248	22·8	9263	33·3	
Mean	0·87		0·85		0·83		0·82		0·81		0·84		$P<0·001$*
SD	0·1		0·1		0·1		0·1		0·1		0·1		
BMI at baseline													
<25	1792	31·6	2114	35·1	1937	36·2	2133	40·5	2563	46·9	10 539	37·9	$\chi^2=348$; df = 8, $P<0·001$
25–29·9	2664	47·0	2671	44·3	2311	43·2	2238	42·4	2107	38·5	11 991	43·2	
≥30	1211	21·4	1245	20·7	1101	20·6	902	17·1	798	14·6	5257	18·9	
Mean	27·2		26·9		26·9		26·4		25·9		26·6		$P<0·001$*
SD	4·3		4·2		4·3		4·2		4·2		4·3		
Energy (kJ/d)													
Mean	9914		9381		9171		9064		8797		9274		$P<0·001$*
SD	3932		3779		3716		3632		3452		3728		

IQR, interquartile range; SEIFA, Socio-Economic Indexes for Areas; WHR, waist-to-hip circumference ratio.
*P for trend.

Table 3 Descriptive statistics by Mediterranean Diet Score (MDS) categories

	MDS score categories								
	Score 0–3		Score 4–6		Score 7–9		Total		
	n	%	*n*	%	*n*	%	*n*	%	Test statistics
Quartile median	2		5		7		4		
IQR	2, 3		4, 6		7, 8		3, 6		
Age									
<50 years	3491	37·1	5294	35·3	1119	33·0	9904	35·6	$\chi^2 = 21{\cdot}6$; df = 4, $P < 0{\cdot}001$
50–59 years	3068	32·6	5123	34·2	1169	34·5	9360	33·7	
≥60 years	2854	30·3	4585	30·6	1101	32·5	8540	30·7	
Mean	54·1		54·3		54·9		54·3		$P < 0{\cdot}001$*
SD	8·6		8·6		8·5		8·6		
Gender									
Male	3821	40·1	5854	39·0	1355	40·0	11 030	39·7	$\chi^2 = 6{\cdot}1$; df = 2, $P = 0{\cdot}047$
Female	5592	59·9	9148	61·0	2034	60·0	16 774	60·3	
SEIFA quintiles									
SEIFA Q1	1872	20·0	2508	16·8	467	13·9	4847	17·5	$\chi^2 = 177$; df = 8, $P < 0{\cdot}001$
SEIFA Q2	2086	22·3	3091	20·7	604	18·0	5781	20·9	
SEIFA Q3	1494	16·0	2420	16·2	497	14·8	4411	16·0	
SEIFA Q4	1667	17·8	2749	18·4	685	20·4	5101	18·5	
SEIFA Q5	2243	24·0	4146	27·8	1110	33·0	7499	27·1	
Ethnic origin									
AU/NZ/Others	6277	66·7	9995	66·6	2250	66·4	18 522	66·6	$\chi^2 = 39{\cdot}4$; df = 4, $P < 0{\cdot}001$
UK	562	6·0	970	6·5	301	8·9	1833	6·6	
Southern Europe	2574	27·4	4037	26·9	838	24·7	7449	26·8	
Smoking status									
Never	5614	59·6	8945	59·6	1997	58·9	16 556	59·6	$\chi^2 = 71{\cdot}6$; df = 8, $P < 0{\cdot}001$
Former	2574	27·4	4403	29·4	1102	32·5	8079	29·1	
Current	1225	13·0	1653	11·0	290	8·6	3168	11·4	
Alcohol dinking status									
Never	3117	33·5	4166	28·1	581	17·3	7864	28·6	$\chi^2 = 341$; df = 4, $P < 0{\cdot}001$
Former	974	10·5	1496	10·1	357	10·6	2827	10·3	
Current	5206	56·0	9184	61·9	2429	72·1	16 819	61·1	
Physical activity score 56·0									
0	2443	26·0	3286	21·9	563	16·6	6292	22·6	$\chi^2 = 202{\cdot}5$; df = 6, $P < 0{\cdot}001$
>0 and <4	1932	20·5	3023	20·2	673	19·9	5628	20·2	
≥4 and <6	3225	34·3	5171	34·5	1200	35·4	9596	34·5	
≥6	1813	19·3	3522	23·5	953	28·1	6288	22·6	
WHR at baseline									
Normal WHR	6099	64·9	10 089	67·3	2332	68·8	18 520	66·7	$\chi^2 = 23$; df = 4, $P < 0{\cdot}001$
Raised WHR	3302	35·1	4906	32·7	1056	31·2	9264	33·3	
Mean	0·84		0·83		0·83		0·84		$P < 0{\cdot}001$*
SD	0·1		0·1		0·1		0·1		
BMI at baseline									
<25	3400	36·2	5795	38·6	1344	39·7	10 539	37·9	$\chi^2 = 32$; df = 4, $P < 0{\cdot}001$
25–29·9	4115	43·8	6391	42·6	1485	43·8	11 991	43·2	
≥30	1889	20·1	2810	18·7	559	16·5	5258	18·9	
Mean	26·8		26·6		26·4		26·7		$P < 0{\cdot}001$*
SD	4·3		4·3		3·9		4·3		
Energy intake (kJ/d)									
Mean	7408		9820		12 043		9274		$P < 0{\cdot}001$*
SD	2678		3647		4072		3728		

IQR, interquartile range; SEIFA, Socio-Economic Indexes for Areas; WHR, waist-to-hip circumference ratio.
*P for trend.

quality or more inflammatory diets predicted larger increases in markers of overall and central obesity. The AHEI appeared to be a stronger predictor than the other scores for markers of both central and overall obesity.

Alternative healthy eating index

Boggs *et al.* (2013) reported that for young African American, women who had a normal BMI (18·5–24·9 kg/m^2) at baseline and a high AHEI score, reflecting a higher quality diet in both 1995 and 2001, the risk of incident obesity between 2001 and 2011 was less than for women with a lower AHEI score[16]. The association was not seen for women who were overweight at baseline. Two other studies[17,18] looked at dietary quality scores, including AHEI, in relation to obesity and interactions with genetic susceptibility to obesity. Ding *et al.*[17] analysed BMI after 2–3 years in three cohorts: Nurse's Health Study, Health Professional's Follow-up Study and the Women's Genetic Health Study, and used a genetic risk score for obesity based on ninety-seven SNP.

Table 4 Association of dietary indices with BMI adjusting for plausible confounders and baseline BMI level

	Adjusted* β	95 % CI	*P* value*	AIC	BIC
DII Quintile				97 824·6	97 996·6
DII Q1	Reference				
DII Q2	0·14	−0·02, 0·30	0·09		
DII Q3	0·20	0·03, 0·37	0·02		
DII Q4	0·34	0·16, 0·52	<0·001		
DII Q5	0·41	0·21, 0·61	<0·001		
Per unit DII	0·085	0·045, 0·125	<0·001		
AHEI Quintile				97 801·8	97 973·8
AHEI Q1	Reference				
AHEI Q2	−0·22	−0·38, −0·06	0·006		
AHEI Q3	−0·12	−0·282, 0·04	0·15		
AHEI Q4	−0·232	−0·40, −0·07	0·006		
AHEI Q5	−0·51	−0·68, −0·35	<0·001		
Per unit AHEI	−0·016	−0·022,−0·010	<0·001		
MDS categories				97 837·1	97 993·5
Score 0–3	Reference				
Score 4–6	−0·09	−0·21, 0·03	0·12		
Score 7–9	−0·05	−0·23, 0·13	0·56		
Per unit MDS	−0·019	−0·052, 0·013	0·25		

AIC, Akaike information criterion; BIC, Bayesian information criteria; DII, Dietary Inflammatory Index; AHEI, Alternative Healthy Eating Index; SEIFA, Socio-Economic Indexes for Areas; MDS, Mediterranean Diet Score.
*Adjusted for age, sex, SEIFA, smoking status, alcohol drinking status, physical activity level and BMI and average energy intake at baseline and country of birth.

Table 5 Association of dietary indices with waist-to-hip circumference ratio (WHR) adjusting for plausible confounders and baseline WHR level

	Adjusted* β	95 % CI	*P* value*	AIC	BIC
DII quintile				−53 406·8	−53 234·8
DII Q1	Reference				
DII Q2	0·002	−0·001, 0·004	0·21		
DII Q3	0·004	0·001, 0·007	0·003		
DII Q4	0·005	0·002, 0·008	0·002		
DII Q5	0·009	0·006, 0·013	<0·001		
Per unit DIIincre	0·002	0·001, 0·003	<0·001		
AHEI quintile				−53 433·6	−53 261·6
AHEI Q1	Reference				
AHEI Q2	−0·004	−0·007, −0·002	0·001		
AHEI Q3	−0·006	−0·008, −0·003	<0·001		
AHEI Q4	−0·006	−0·009, −0·003	<0·001		
AHEI Q5	−0·011	−0·013, −0·008	<0·001		
Per unit AHEI	−0·00035	−0·00044,−0·00026	<0·001		
MDS categories				−53 387·2	−53 230·9
Score 0–3	Reference				
Score 4–6	−0·003	−0·005, −0·001	0·002		
Score 7–9	−0·004	−0·007, −0·001	0·008		
Per unit MDS	−0·0009	−0·0014, −0·00036	0·001		

AIC, Akaike information criterion; BIC, Bayesian information criteria; DII, Dietary Inflammatory Index; AHEI, Alternative Healthy Eating Index; SEIFA, Socio-Economic Indexes for Areas; MDS, Mediterranean Diet Score.
*Adjusted for age, sex, SEIFA, smoking status, alcohol drinking status, physical activity level and WHR and average energy intake at baseline and country of birth.

Wang *et al.*[18] looked at BMI change over 20 years in the Nurse's Health Study and Health Professional's Follow-up Study with a seventy-seven SNP genetic risk score. In both analyses, a higher quality diet measured as AHEI tended to attenuate the effect of genetic risk on obesity and higher quality diet was most beneficial for people at higher genetic risk. These studies are consistent with the MCCS in showing benefits of a higher AHEI for minimising weight gain or obesity. The MCCS participants were predominantly non-obese at baseline, both by BMI (81·1 % with BMI < 30 kg/m^2) and WHR (66·7 % with normal WHR) which is consistent with the observations of Boggs *et al.*[16], but no information was available to assess genetic risk of obesity.

Dietary inflammatory Index

A cross-sectional analysis of Prevención con Dieta Mediterránea study data found that a higher DII was associated with a higher BMI, waist circumference and waist-to-height ratio, despite an inverse association

between DII and energy intake[20]. The association tended to be stronger for waist circumference than the other anthropometric measures. Despite the cross-sectional nature of the study, the authors concluded that the results support the hypothesis that diet has an impact on obesity via inflammatory modulatory mechanisms. Similar findings of an association of DII with WHR but not BMI were reported from cross-sectional analyses in the Polish–Norwegian Study[36]. In another cross-sectional analysis, black South African women were divided according to positive or negative DII scores and various body composition measures compared. Waist circumference, WHR and visceral adipose area were higher for those women with a positive (more inflammatory) DII, but there was no difference between groups for BMI, body weight or body fat % by DXA[37]. In one of the few longitudinal analyses of the DII and weight gain, over 7000 healthy-weight university graduates in the SUN study were followed over 8 years. A higher (more pro-inflammatory) DII score was associated with a greater risk of developing obesity or gaining weight than a diet with a lower DII[19]. This is consistent with our findings, but the SUN study did not look specifically at abdominal obesity, which appeared to be more strongly associated with DII in cross-sectional analyses[18,34,35]. While it is generally accepted that obesity drives inflammation, the SUN study authors also considered their findings to support the idea that inflammation may contribute to obesity and cited other studies where inflammatory biomarkers were associated with weight gain and the development of obesity[38]. In the MCCS, we found that a higher DII was associated with larger increases in BMI and WHR, but we did not specifically assess whether the association differed according to the outcome.

Mediterranean dietary score

In a follow-up of almost 400 000 participants across ten countries in Europe participating in the EPIC-PANACEA study, the relative MDS (which is adjusted for energy intake) was inversely associated with weight gain and the risk of overweight or obesity[22]. The studies by Ding[17] and Wang[18] both used the Alternative MDS which, unlike the AHEI-2010 and DASH diet score, was not associated with weight gain. In the EPIC-PANACEA study, the associations for three centres with relatively low adherence to a Mediterranean style diet in the UK, Netherlands and Sweden were not consistent with those for the other centres. In the American studies[17,18], variation in adherence to a Mediterranean diet may not have been sufficient to see an association. A review by Bendall *et al.*[39] of Mediterranean Diet interventions and central obesity identified eighteen studies with waist circumference, WHR or visceral fat as outcomes. Thirteen studies found some association, but seven of these included energy restriction and only three reported significant effects[39]. The studies reporting significant effects were those without a control group comparison, so overall the evidence from this review is not strong. The inclusion of southern European migrants who tend to follow a Mediterranean diet in the MCCS may have contributed to the associations we saw for MDS and WHR; it is not known why an association was not seen for BMI and MDS.

In a 3-year follow-up of 67 000 post-menopausal women from the Women's Health Initiative Observational Study, four different dietary patterns Healthy Eating Index-2010, AHEI-2010, DASH and Alternative MDS were assessed, along with weight and waist circumference. A 10 % increment in each diet score at baseline was associated with a smaller increase in waist circumference over 3 years, even accounting for weight change[40]. The authors noted that maintaining or improving diet quality may be beneficial for reducing chronic disease via central adiposity[40]. These authors did not assess whether any one diet pattern was more closely associated with waist circumference than the others, but did report that Alternative MDS was less strongly associated with change in waist circumference than were the other scores, and they noted the consistency of healthy diet components, irrespective of the score used[40].

Wirth *et al.* have previously shown that DII is inversely associated with the Healthy Eating Index, AHEI-2010 and the DASH diet score[41], confirming that a less inflammatory diet would also rate as healthier on these other scores. This is consistent with our findings that DII, AHEI and MDS were all associated with follow-up WHR, and AHEI and DII with follow-up BMI. Similar to other healthy diets, we have shown that in the MCCS, a lower DII, that is, a less inflammatory diet, contains more olive oil, more wholemeal bread, less white bread, more fruit, vegetables and legumes, and less red meat than a high DII diet[42]. We also reported a moderate inverse correlation between the DII and MDS (Spearman's rho = −0·45)[43]. These findings suggest that many versions of 'healthy' diets could be beneficial for reducing the risk of overall and abdominal obesity.

Energy intake is a major driver of weight gain, and the dietary scores we have examined had different associations with energy intake due to the way components were scored. Adjusting models for total energy intake attempts to account for this, but may be over-adjusting, as energy intake could be a mechanism through which different dietary patterns are associated with weight gain. Diet scores which adjust for energy as part of the scoring algorithm, such as the relative MDS[22] and the energy-adjusted DII[44], which are likely not associated with energy intake, may give a better understanding of how diet composition is associated with weight, but here we have chosen to assess the more commonly used unadjusted scores and include energy in our models.

Our work is the first to attempt to answer the question of whether one healthy diet is likely to be better for avoiding increases in overall and abdominal obesity, and we have

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shown that models using AHEI have better fit than those with DII or the MDS. The AHEI may also be easier to use for advising the public as it is based on existing guidelines, reflects mostly food groups rather than single items or nutrients and gives recommended quantities of items to be consumed. The MDS on the other hand, as used here, is based on intake relative to the population mean[15], and the DII is based on mostly nutrients with a few herbs and does not provide any information on how much of anything should be consumed[14], though interventions have been created around DII-based recommendations[45].

The associations we observed were seen after adjusting for energy intake, suggesting that other mechanisms were involved. However, it should be noted that in many populations, the energy-adjusted DII does a better job of predicting outcomes than does the DII[44]. Diets can modify gut microbiota, and specific phyla have been associated with obesity in animal models and humans[46]. Microbiota from obese mice can make gnotobiotic mice obese, and it is understood that healthy plant-based diets including plentiful fibre are more likely to be associated with a lean microbiome[47]. Dysbiosis is associated with low-grade inflammation which may be a mechanism relating diet with obesity and poor health outcomes beyond the effect of energy intake[47,48].

Strength and weakness

A major strength of our study is the use of a large data set and a prospective study design with adjustment for many plausible confounders. Our study used three diet scores with different theoretical bases and reached similar conclusions for each, although the MDS was not a significant predictor of BMI. Also, we had anthropometric measurements rather than relying on self-reported measures often used in similar large data sets.

One limitation of our study was that we used self-reported dietary data, which can be particularly problematic for energy calculation and may be associated with biases, particularly in energy-dense foods[49,50]. Only twenty-nine of forty-five components of DII were included for this study, which may limit comparison of the findings with other similar studies using different dietary variables. Physical activity data were self-reported, and the questions were not as detailed as in instruments commonly used today, such as IPAQ[51].

Conclusion

Poor quality, pro-inflammatory diets predicted overall and abdominal obesity, consistent with other studies that have reported increases in weight or BMI associated with low diet quality diets. However, we were the first to identify AHEI among the three diet scores we studied, as being the best predictor of both overall and abdominal obesity. Given the generally similar items that contribute to more healthy diets as assessed by the three indices, it is reasonable in practice to promote diets high in fruit, vegetables, whole grains, legumes, using unsaturated fat with minimal meat and processed meat and moderate dairy foods, as recommended by dietary guidelines. The AHEI may provide the best way to operationalise this and may best capture subtle differences between the scores.

Acknowledgements

Acknowledgements: Cases and their vital status were ascertained through the Victorian Cancer Registry and the Australian Institute of Health and Welfare, including the National Death Index and the Australian Cancer Database. *Financial support:* The Melbourne Collaborative Cohort Study (MCCS) cohort recruitment was funded by VicHealth and Cancer Council Victoria. The MCCS was further augmented by Australian National Health and Medical Research Council grants 209057, 396414 and 1074383 and by infrastructure provided by Cancer Council Victoria. B.d.C. is supported by a Royal Australasian College of Physicians Fellows Career Development Fellowship. *Conflict of interest:* A.M.H., N.K., R.L.M. and B.d.C. have no conflicts of interest to declare. Dr. J.R.H. owns controlling interest in Connecting Health Innovations LLC (CHI), a company that has licenced 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. N.S. is an employee of CHI. *Authorship:* A.M.H., N.K. and B.d.C. planned the analysis, N.K. conducted the statistical analysis, A.M.H., N.K. and B.d.C. interpreted the results, A.M.H. wrote the first draft of manuscript, A.M.H. had primary responsibility for final content. All authors read and approve the final manuscript. *Ethics of human subject participation:* This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving research participants were approved by the Cancer Council Victoria Human Research Ethics Committee. Written informed consent was obtained from all participants. The current study received approval from the Monash University Human Research Ethics Committee.

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