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Longitudinal analysis of the Alternative Healthy Eating Index-2010 and incident non-communicable diseases over 15 years in the 1973–1978 cohort of the Australian Longitudinal Study on Women's Health

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Abstract

In studies that contain repeated measures of variables, longitudinal analysis accounting for time-varying covariates is one of the options. We aimed to explore longitudinal association between diet quality (DQ) and non-communicable diseases (NCDs). Participants from the 1973–1978 cohort of the Australian Longitudinal Study on Women's Health (ALSWH) were included, if they; responded to survey 3 (S3, 2003, aged 25–30 years) and at least one survey between survey 4 (S4, 2006) and survey 8 (S8, 2018), were free of NCDs at or before S3, and provided dietary data at S3 or S5. Outcomes were coronary heart disease (CHD), hypertension (HT), asthma, cancer (except skin cancer), diabetes mellitus (DM), depression and/or anxiety, and multimorbidity (MM). Longitudinal modelling using generalised estimation equation (GEE) approach with time-invariant (S4), time-varying (S4–S8) and lagged (S3–S7) covariates were performed. The mean (± standard deviation) of Alternative Healthy Eating Index-2010 (AHEI-2010) of participants ($n = 8022$) was 51·6 ± 11·0 (range: 19–91). Compared to women with the lowest DQ (AHEI-2010 quintile 1), those in quintile 5 had reduced odds of NCDs in time-invariant model (asthma: OR (95 % CI): 0·77 (0·62–0·96), time-varying model (HT: 0·71 (0·50–0·99); asthma: 0·62 (0·51–0·76); and MM: 0·75 (0·58–0·97) and lagged model (HT: 0·67 (0·49–0·91); and asthma: 0·70 (0·57–0·85). Temporal associations between diet and some NCDs were more prominent in lagged GEE analyses. Evidence of diet as NCD prevention in women aged 25–45 years is evolving, and more studies that consider different longitudinal analyses are needed.

Key words: **Longitudinal analysis: Young women: Childbearing age: Diet quality: Non-communicable disease: Multimorbidity**

Diet is one of the most important modifiable risk factors for some non-communicable diseases (NCDs), including cardiovascular disease (CVD), some cancers and diabetes mellitus (DM)[1]. Since diet is comprised of a combination of nutrients and other bioactive compounds, where some interact with each other synergistically and/or antagonistically[2], using a single nutrient approach in epidemiological studies may underestimate the effects of nutrients on health outcomes[3]. It is necessary to take into account the inter-correlation of nutrient constituents contained in each meal[4]. A number of approaches are used to measure dietary pattern (DP)[5]. One main approach is *a posteriori* or data-driven that identifies DP from comprehensive dietary data through multivariate analysis such as principal component, exploratory factor or cluster analysis[2,6]. Another approach is *a priori* or investigator-driven in which a diet quality index (DQI) is constructed based on pre-defined scoring criteria for adherence to dietary recommendations, such as dietary guidelines[7–9] or a specific DP[2]. Based on a pre-defined 'ideal diet', a DQI can rank individuals within a population from lower to higher diet quality (DQ)[10]. It can describe the overall diet of the population of interest and can be replicated in various populations[11]. It is easy to understand, and the summation technique is more straightforward than any other statistical methods of DP analysis[11].

In studies assessing the relationship between diet and NCD, overall DQ measured by construction of DQI based on the dietary recommendations or guidelines[7–9] is widely used. The growing body of evidence suggests that DQ has a preventive role in NCD[12–18] and NCD multimorbidity (MM)[12], with studies showing that people with higher scores in DQI had reduced risk

Abbreviations: AHEI-2010, Alternative Healthy Eating Index-2010; ALSWH, Australian Longitudinal Study on Women's Health; CHD, coronary heart disease; CI, confidence interval; CVD, cardiovascular disease; DAG, Directed Acyclic Graphic; DM, diabetes mellitus; DP, dietary pattern; DQ, diet quality; DQES-v2, Dietary Questionnaire for Epidemiological Studies-version 2; DQI, diet quality index; FFQ, Food frequency questionnaire; GEE, generalised estimation equation; HT, hypertension; MM, multimorbidity; NCD, non-communicable diseases; OR, odds ratio; PUFA, polyunsaturated fatty acid; SSB, sugar-sweetened beverages.

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of disease outcomes. Of course, separating the effect of diet from other potential confounders can be difficult, and a range of study designs and statistical methods may be needed.

A number of studies have been conducted to measure the associations between DQ and incidence of NCD in a large population-based study setting, the Australian Longitudinal Study on Women's Health (ALSWH)[12,19–27]. Analyses performed were cross-sectional[12,21,22,25,27] or longitudinal[19,20,23,24,26]. Longitudinal data, which contain repeated measures for an individual participant, are correlated[28], and that correlation needs to be adjusted for in analysis[29]. There are two common approaches to longitudinal analysis: (i) subject-specific and (ii) population-averaged[28]. In the subject-specific approach, it is assumed that there is a parametric distribution in the population, and the source of the covariance among repeated observations for a subject is explained[28]. In the population-averaged approach, the response or outcome can be modelled without considering subject to subject variability, and the source of the covariance among repeated observations for a subject is only described[28].

The population-averaged or generalised estimation equation (GEE) approach provides the estimates of population average effects over follow-up periods by considering within-person correlation from repeated measurements of each individual[30]. Correlation structures considered in GEE are 'independent' structure, assuming that correlation between time points is independent, 'exchangeable' structure, assuming that there are equal correlation between all possible measurement pairs, 'autoregressive' structure, assuming the highest correlation for adjacent times and reducing correlation with increasing distance between time points, and 'unstructured', not assuming any specific correlation[31]. The appropriate correlation structure is selected according to the values of the quasi-likelihood information criterion[31]. Both time-invariant[32] and time-varying covariates[33] are used in the GEE approach. By using the GEE approach, incidence of NCD over time in study participants at population level according to proportions of exposure can be assessed[29].

Exploring the longitudinal relationship between DQ and NCD in childbearing women is of interest, because the findings from the ALSWH study suggest there is an increasing trend of NCD occurrence in women aged 25–47 years[34]. In our previous studies, we have investigated the association between baseline DQ and incident NCD of participants from the ALSWH 1946–1951 cohort[12] and 1973–1978 cohort[27], using a repeated cross-sectional design. Based on the results of our systematic review[35], we selected three dietary indices measuring various conceptual perspectives in our analysis of data from the ALSWH 1946–1951 cohort: (1) Healthy Eating Index for Australian Adults-2013 that assesses meeting Australian Dietary Guidelines-2013[36]; (2) Mediterranean Diet Score that assesses following a specific DP; and (3) Alternative Healthy Eating Index-2010 (AHEI-2010) that assesses adhering DP that highlights foods and nutrients for NCD prevention. Built on our first analysis, the AHEI-2010 was selected in the 1973–1978 cohort by considering its relevance to up-to-date dietary recommendations[37] and favourable performance because of its comprehensive measurements for dimension (adequacy, moderation and balance), both foods and nutrients components for healthy and unhealthy ingredients, metric scaling with normative cut points[35]. Longitudinal analysis testing the association between DQ and NCD is necessary to (i) improve the quality of methods used in studies of diet–disease relationships and (ii) design future effective dietary intervention for NCD prevention. The aim of the current study was therefore to examine the longitudinal relationship between DQ and NCD, measured in Australian women from the ALSWH born between 1973 and 1978 (25–30 years old at baseline) over a 15-year period. The NCD outcomes were coronary heart disease (CHD), hypertension (HT), asthma, cancer (except skin cancer), DM, depression and/or anxiety, and MM.

Materials and methods

Study sample

The current study used data from the ALSWH, an ongoing prospective cohort study that commenced in 1996[38]. Approximately 45 000 women across three cohorts were recruited: those born in 1973–1978 (aged 18–23 years), 1946–1951 (aged 45–50 years) and 1921–1926 (aged 70–75 years) were selected from the Medicare database (Australia's government-funded universal health care cover)[38]. In 2013, a new cohort of women born between 1989 and 1995 (*n* 17 012) were recruited. The Human Ethics Committees of the University of Newcastle (approval number: h–076–0795) and University of Queensland (approval number: 200400224) approved the study methods[38]. Further details about the ALSWH on recruitment details, baseline characteristics and attrition rates have been described elsewhere[38–40].

The current analysis was performed in the 1973–1978 cohort. Survey 1 (S1) was conducted in 1996 (*n* 14 247, aged 18–23 years), and these women have been followed through on a roughly 3-year rolling schedule in 2000 (survey 2, S2), 2003 (survey 3, S3), 2006 (survey 4, S4), 2009 (survey 5, S5), 2012 (survey 6, S6), 2015 (survey 7, S7) and 2018 (survey 8, S8). Dietary information was collected at S3 (aged 25–30 years) and again at S5 (aged 31–36 years), using a Food Frequency Questionnaire (FFQ) known as the Dietary Questionnaire for Epidemiological Studies-version 2 (DQES-v2).

We included women who responded at least once between S4 (2006, aged 28–33 years) and S8 (2018, aged 40–45 years). Women were excluded from the current analyses if they had missing data on diet at both S3 and S5 or had NCDs (CHD, HT, cancer and DM) recorded at S1, S2 and S3. There may be fluctuations with acute episodes in depression and/or anxiety[41], and a high prevalence of asthma in this age group[42]. Therefore, we did not exclude women who had a history of depression and/or anxiety, or asthma; however, adjustments were made for the history of these conditions and a current episode was considered an incident case. In total, 8022 women from S3 (2003) and onwards were included (Fig. 1).

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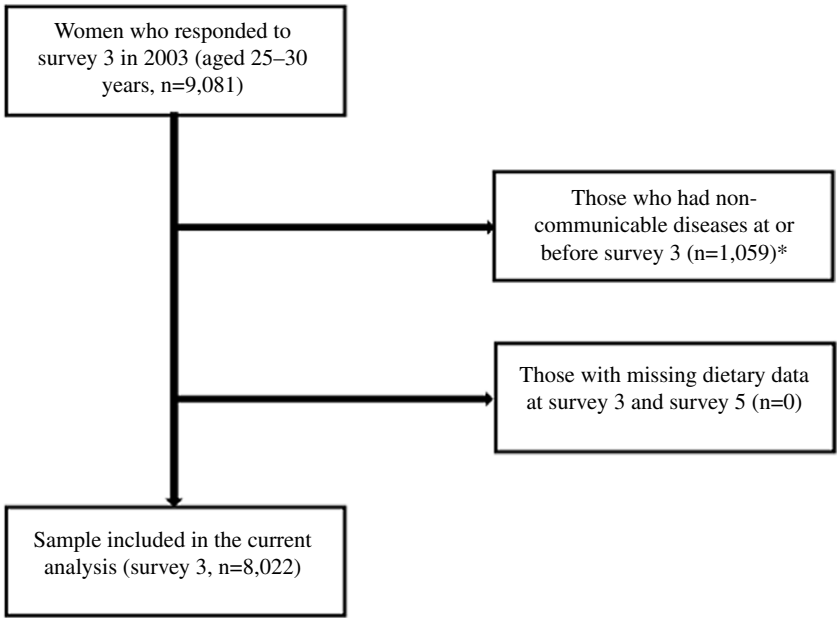

Fig. 1. Selection of participants from the Australian Longitudinal Study on Women's Health (ALSWH), born in 1973–1978. *NCDs at or before survey 3 were coronary heart disease, hypertension, cancer and diabetes mellitus.

Measurement of diet

At S3 (2003) and S5 (2009), dietary intake of the ALSWH 1973–1978 cohort was measured using the DQES-v2[43]. The participants were required to indicate their usual consumption of seventy-four food items and six alcoholic beverages in the preceding year[43]. Food items were asked on a ten-point frequency scale, 'Never', 'Less than once per month', '1 to 3 times per month', '1 time per week', '2 times per week', '3 to 4 times per week', '5 to 6 times per week', '1 time per day', '2 times per day' and '3 or more times per day'. Alcoholic beverages were asked with options 'Never', 'Less than once per month', '1 to 3 days per month', '1 day per week', '2 days per week', '3 days per week', '4 days per week', '5 days per week', '6 days per week' and 'Everyday'[44]. Additional questions were also assessed: amount of intakes for fruit, milk, bread, sugar, egg; and types of consumed food for vegetables, milk, bread, spread and cheese[44]. Portion-size photographs of vegetables, potatoes, meat and casserole dishes were provided to guide for reporting a standard serving size[44]. Nutrient intakes were computed from the Australian Food Composition Database (NUTTAB95)[45]. The validation of the FFQ was investigated in childbearing women using a 7-d weighed food record, and the results showed moderate to strong energy-adjusted correlation coefficients for nutrient intakes (ranging from 0·28 for vitamin A to 0·78 for carbohydrates)[44].

Measurement of the Alternative Healthy Eating Index-2010

In this study, DQ was measured as the AHEI-2010 at S3 and S5. Selection of the AHEI-2010 was based on its components which are linked with prevention of NCD[37], having preferable features of a DQI based on our systematic review[35], and the most sensitive index that associated with more NCD in our previous ALSWH 1946–1951 cohort analysis[12]. The scoring criteria for the AHEI-2010 is provided in online Supplementary Table 1.

The components of the AHEI-2010 are foods and nutrients being useful in prevention of CVD, some cancers and DM[46,47]. This index builds on the original Healthy Eating Index (HEI)[48] and the original AHEI[46]. It is composed of eleven components: six components for which the highest intakes are presumed to be healthy (vegetables, fruits, wholegrains, nuts and legumes, long-chain *n*-3 fats, and polyunsaturated fatty acid (PUFA)), four components for which the lowest intakes are presumed to be healthy (sugar-sweetened beverages (SSB) and fruit juice, red and processed meat, *trans*-fat, and sodium (Na))[47] and one component for which the moderate intakes are presumed to be healthy (alcohol). Each component is assigned to a minimum score of 0 and a maximum score of 10, with intermediate values scored proportionally to their intake[47].

The scoring of each component was obtained using data from the DQES-v2. Measurements of positively weighted components were performed. All vegetables intakes except potato (green vegetables, orange vegetables, cruciferous vegetables and tuber vegetables) were summed, and servings were calculated. Servings of whole fruits (citrus, melon, berry, pome, stone and tropical) and nuts and legumes of participants were calculated. Intakes of total wholegrain in grams and long-chain *n*-3 fats in milligrams were calculated. Intakes of PUFA as percentage of energy was computed. Minimum and maximum scores were assigned as: vegetables (0–5 servings per d), whole fruits (0–4 servings per d), nuts and legumes (0–1 serving per d), wholegrain (0–75 grams per d), long-chain *n*-3 fats (0–250 milligrams per d), and PUFA (2–10 % of energy).

For negatively weighted components, servings of SSB and fruit juice, and red and processed meat were computed. Intakes of *trans*-fat as percentage of energy and Na as decile

(milligrams per d) were computed. Minimum scores were assigned to higher intakes: SSB and fruit juice (≥ 1 serving/d), red and processed meat (≥ 1·5 servings/d), *trans*-fat (≥ 4 % of energy), and Na (highest decile (mg/d))[47]. Maximum scores were assigned to no SSB and fruit juice, no red and processed meat, ≤ 0·5 % of energy from *trans*-fat or lowest decile (mg/d) of Na[47]. Participants who consumed alcohol ≥ 2·5 drinks/d were assigned the minimum score and 0·5–1·5 drinks/d were assigned the maximum score[47]. All component scores are summed to obtain a total AHEI-2010, ranging from 0 (poor adherence) to 110 (excellent adherence)[47]. Detailed scoring of the AHEI-2010 has been previously reported[47].

Measurement of non-communicable diseases

In this study, incidence of NCDs (CHD, HT, asthma, cancer (except skin cancer after S4), DM, and depression and/or anxiety) and MM were the main outcome variables. An incidence case of each disease was counted by self-reported diagnosis or treatment of respective NCD in the ALSHW survey. Self-reported NCD data in the ALSWH participants have been shown to be acceptable[49,50]. At S1 in 1996, participants were asked if a doctor ever diagnosed them with heart disease, HT (high blood pressure), asthma, cancer and/or diabetes (high blood sugar). Women's depression and anxiety conditions were firstly assessed in S2 as depression (not postnatal) and anxiety disorder. Different wording was used in the assessment of NCD status such as HT other than during pregnancy for HT (from S2 to S4), cancer (specify type) (from S2 to S4), skin cancer and other cancer for cancer (from S5 to S8), and non-insulin-dependent (type 2) diabetes for DM (S2 to S8). Since self-reported data on cancer were separately asked at S5–S8, skin cancer was excluded in calculating the occurrence of cancer during these periods.

NCD were considered as enduring conditions, meaning that participants who reported NCD at any survey were regarded as having that NCD continuously in all successive surveys, except for asthma, and depression and/or anxiety. MM was considered as the presence of two or more of any combination of CHD, HT, asthma, cancer, DM, and depression and/or anxiety between S4 in 2006 and S8 in 2018.

Measurement of covariates

In epidemiological studies, potential confounders are identified and adjusted for in further statistical analyses to obtain unbiased effect sizes[51]. Various approaches such as statistical prerequisites, criteria for selecting variables and variable selection algorithms are performed in selecting covariates or confounders[52]. The Directed Acyclic Graphic (DAG), selecting variables based on background knowledge, is one of these approaches[52]. A DAG is a non-parametric diagrammatic display of the anticipated data-generating process for a group of variables in examining the causal associations between variables[53]. By viewing DAG, the planned adjustment set of variables for unbiased causal estimand (the desired causal effect of the exposure on the outcome) can be observed[54]. To specify models that are parsimonious, DAG that proposed the association between the AHEI-2010, covariates and NCDs were constructed (online Supplementary Fig. S1–S7).

In the present study, sociodemographic, lifestyle, previous disease status and childbearing variables at respective surveys were considered as covariates in model adjustments. Selection of covariates based on background knowledge and literature was reported in the previous study[27]. Details on categorisation of covariates, harmonisation of variables related to medication and childbearing have been reported[27].

Of variables measuring sociodemographic characteristics (residence status, marital status, education, occupation and ability to manage income), the correlation between the latter three variables is significant. Moreover, evidence highlights the inclusion of income as a covariate in diet–health relationship studies, rather than education[55]. Lifestyle-related variables were alcohol consumption, smoking, physical activity, prescribed medication and body mass index (BMI). Of these, alcohol consumption was measured as a component in the AHEI-2010[47], and smoking was reported to be significantly associated with physical activity[56]. Regarding prescribed medication in the diet–NCD relationship, we have suggested that its potential involvement in the pathway depends on underlying disease condition[27]. BMI was deemed as a mediator, meaning that it can be altered by DQ[57] and can impact NCD outcomes[58–64]. Previous disease status of women was considered as a potential confounder in asthma, depression and/or anxiety, and MM. Since the women were of childbearing age, we included variables measuring parity, history of breast-feeding, history of gestational diabetes mellitus for DM, and history of HT in pregnancy for CHD and HT in the adjusted models. Total energy intake has been considered in model adjustment in nutritional epidemiology[65]. However, we found that there was significant correlation between the AHEI-2010 and total energy intake of our participants ($r = -0{\cdot}21$, $P < 0{\cdot}0001$). Additionally, total energy intake of those who developed NCD were not different with those who did not.

According to above reasons, we did not include education, occupation, alcohol consumption, smoking, physical activity, prescribed medication, BMI and total energy intake for adjustments in our main analyses. The covariates included in DAG for adjusting for in the main analyses were those measuring sociodemographic characteristics such as residence, marital status, ability to manage income; previous disease status such as history of asthma for asthma, depression and/or anxiety for depression and/or anxiety, and MM; and childbearing such as parity, history of breast-feeding, history of HT in pregnancy for CHD and HT, and history of gestational diabetes mellitus for DM.

For the management of missing data (0·15–12·15 % for covariates across S3 to S8), the responses from both the preceding and subsequent surveys (for S4 to S7) and from the preceding survey (for S8) were carried forward in the first instance, or back, to fill missing items of covariates (area of residence, marital status, education, occupation, ability to manage income, physical activity and taking prescribed medicine)[66]. After filling, no variable had more than 5 % of the total data missing. Childbearing variables had missing values less than 5 %, and the carry-forward approach for missing values was not applied. Proportions of missing values, before and after

applying imputation of missing data for covariates (area of residence, marital status, education, ability to manage income, physical activity and taking prescribed medicine) across S3 to S8 were provided (online Supplementary Table 2).

Statistical analysis

Continuous data were assessed for normality and expressed as mean and standard deviation. Categorical data were described as number and percentage. Number of respondents in each survey and incidence of NCDs were reported (online Supplementary Tables 3 and 4). The sociodemographic and lifestyle variables of women at S3 according to categories of the AHEI-2010 were investigated using the ANOVA for continuous variables and χ^2 test for categorical variables.

To examine the longitudinal association between the AHEI-2010 (based on the FFQ data at S3 and S5) and incident NCDs (S4–S8), logistic regression models applying GEE approach[67] were used. A binomial distribution with logit link function using an independent correlation was applied for all analyses. To examine improved model fit, the quasi-likelihood information criterion[31] was used, with lower values indicating better fit.

Three models were constructed to analyse the longitudinal association between the AHEI-2010 and NCDs:

(i) time-invariant models,
(ii) time-varying models, and
(iii) time-lagged models.

In model (i), AHEI-2010 and covariates at S4 were used as time-invariant, such that only baseline measurements were used, across all time periods. In model (ii), time-varying covariates and outcomes were considered in analyses. Modelling was performed by using AHEI-2010 and covariates from the same survey at which NCD outcomes were reported. In our final model (iii), temporal relationships between AHEI-2010 and NCDs were investigated. Modelling was performed by assuming the effects of repeated measurements of exposure and covariates at preceding or lagged surveys (S3–S7) were related to NCDs.

The time-invariant model (i) was constructed to examine the association between AHEI-2010 at S3 and incidence of NCDs (S4–S8), adjusting for covariates at S4 (residence, marital status, ability to manage on income, parity, history of breast-feeding, history of gestational diabetes mellitus for DM and history of HT in pregnancy for CHD and HT).

The time-varying model (ii) was constructed to investigate the population-averaged effects of time-varying AHEI-2010 and covariates (S4–S8) on incidence of NCDs (S4–S8). Since diet was measured at S3 and S5, the AHEI-2010 at S3 was applied for S4, and S5 AHEI-2010 was applied for S6, S7 and S8.

The lagged model (iii) was constructed to investigate the population-averaged lagged effects of AHEI-2010 and covariates (S3–S7) on incidence of NCDs (S4–S8). Since diet was measured at S3 and S5, the AHEI-2010 at S3 was applied for S4, and S5 was applied for S6 and S7.

For asthma, two GEE analyses were performed: one model included 'history of asthma' as a covariate, and another model did not. The odds ratio (OR) and 95 % confidence interval (CI) for NCD outcomes with respect to the AHEI-2010 were calculated considering the lowest quintile as the reference category. Survey wave indicator was included in all models to investigate any secular trends in NCD risks.

To explore potential selection bias, comparison of sociodemographic and lifestyle characteristics of participants who had been excluded and included in the current study was performed (online Supplementary Table 5). Furthermore, sensitivity analyses that used education, occupation, physical activity and taking prescribed medicine as covariates in GEE analyses were performed (online Supplementary Table 6). The models with AHEI-2010 only were also performed to investigate the effects of diet (only) on NCD outcomes (online Supplementary Table 7). We tested effect modification by adding an interaction term between the survey time and AHEI-2010 in the main analysis (online Supplementary Table 8). P value $< 0{\cdot}05$ is considered significant, with consideration given to multiple comparisons, and all statistical tests are two-sided. All analyses were conducted using Stata version 15.1 (StataCorp LP).

Results

The study cohort included 8022 women who were free of NCDs at S3 in 2003, with a mean AHEI-2010 at S3 of 51·6 ± 11·0 (range: 19–91). Women with a higher AHEI-2010 score were more frequently in a married/*de facto* relationship, living in an urban area, graduated from a university, in paid employment, able to manage on income easily and did more physical activity than women with lower DQ (Table 1).

Time-invariant model (model i), time-varying model (model ii) and the lagged model (model iii) examining the association between the AHEI-2010 and risk of common NCD (including MM) are shown in Table 2.

For women with the quintile 5 (high DQ) compared with women with quintile 1 (low DQ), 29–33 % lowered odds of HT were observed in time-varying model (OR: 0·71, 95 % CI 0·50, 0·99) and in lagged model (OR: 0·67, 95 % CI 0·49, 0·91). However, no significant association between the AHEI-2010 and HT was observed in the time-invariant model.

When the prospective associations between DQ and asthma were investigated, the reduced odds of asthma were detected amongst those consuming the highest DQ quintile compared with the lowest quintile. In all models where a history of asthma was not adjusted for, lowered odds of asthma was found in time-invariant (OR: 0·77, 95 % CI 0·62, 0·96), time-varying (0·62, 95 % CI 0·51, 0·76) and lagged (0·70, 95 % CI 0·57, 0·85) models. The association was stronger in the time-varying model (model ii). In the model with history of asthma, the association was attenuated and significant in time-varying and lagged models only.

The inverse association between the AHEI-2010 and MM was found only in the time-varying model, that is, 25 % lowered odds of MM in those with the highest quintile of the AHEI-2010 when compared with those with the lowest quintile. Although lagged and time-invariant GEE analysis effects on MM were in the same direction of effect as the time-varying model, the results were not significant.

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Table 1. Sociodemographic and lifestyle variables at survey 3 (in 2003) related to the first (Q1, low diet quality) and fifth (Q5, high diet quality) quintiles of the Alternative Healthy Eating Index-2010 (AHEI-2010) of the sampled women (*n* 8022)

	AHEI-2010 quintiles										
	Q1 (*n* 1635)		Q2 (*n* 1572)		Q3 (*n* 1582)		Q4 (*n* 1636)		Q5 (*n* 1592)		
Characteristics, *n* (%) unless otherwise specified†	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*P*
Age in years											
Mean	27·50		27·55		27·53		27·63		27·64		0·02*
SD	1·5		1·5		1·5		1·5		1·4		
Marital status											< 0·001*
Never married	415	25·5	487	31·1	557	35·3	630	38·6	734	46·3	
Married/	1141	70·1	1028	65·6	975	61·7	940	57·5	811	51·1	
Separated/divorced/widowed	72	4·4	52	3·3	47	3·0	64	3·9	42	2·6	
Area of residence											< 0·001*
Urban	810	49·6	835	53·2	868	55·0	992	60·7	1002	63·2	
Inner regional	499	30·5	435	27·7	430	27·2	388	23·7	388	24·4	
Outer regional/rural	325	19·9	300	19·1	281	17·8	254	15·6	197	12·4	
Education											< 0·001*
No formal education	21	1·3	22	1·4	21	1·3	11	0·7	9	0·6	
High school certificate	582	36·1	507	32·7	452	29·0	376	23·3	289	18·4	
Apprenticeship/diploma	449	27·9	414	26·7	399	25·6	402	25·0	340	21·7	
University/higher degree	558	34·7	608	39·2	686	44·1	821	51·0	932	59·3	
Occupation											< 0·001*
No paid job	393	24·2	354	22·6	269	17·1	257	15·9	195	12·3	
Paid job	1228	75·8	1210	77·4	1304	82·9	1364	84·1	1385	87·7	
Ability to manage income											< 0·001*
Easy/not bad	885	54·2	884	56·3	905	57·4	1032	63·2	1036	65·2	
Sometimes/always difficult	747	45·8	685	43·7	672	42·6	601	36·8	552	34·8	
Physical activity											< 0·001*
Nil/sedentary	204	12·7	167	10·8	129	8·3	101	6·3	56	3·6	
Low	625	38·9	578	37·3	510	32·7	482	29·8	375	23·8	
Moderate	359	22·3	355	22·9	380	24·2	414	25·6	381	24·2	
High	420	26·1	450	29·0	543	34·8	618	38·3	761	48·4	
Taking prescribed medicine											0·22
No	1170	72·7	1107	71·5	1134	72·7	1206	74·6	1171	74·5	
Yes	440	27·3	442	28·5	425	27·3	411	25·4	401	25·5	

AHEI-2010: Alternative Healthy Eating Index-2010.
* Statistically significant ($P < 0·05$). Values for categorical variables are given as 'number (percentage): *n* (%)' and for continuous variable as 'mean (standard deviation): mean (SD).'
† Due to missing data, the sum for each characteristic may not equal *n*.

For the outcomes of CHD, cancer (excluded skin cancer), DM and depression and/or anxiety, there was no association between DQ and these NCDs in time-invariant, time-varying and lagged models (Table 2). However, there were the effects of time on these NCDs, especially in lagged models.

In performing the sensitivity analyses using physical activity, education, occupation and prescribed medicine as covariates, the odds of NCDs and MM remained consistent (online Supplementary Table 6). When GEE analyses were constructed using the AHEI-2010 only, the results were similar to the main results (online Supplementary Table 7). Compared with the primary analyses, longitudinal associations between the AHEI-2010 and NCDs were broadly coherent with the main results in models with effect modification term. However, wider CIs for ORs were observed (online Supplementary Table 8).

Discussion

Within our sample of Australian young women, longitudinal associations between the AHEI-2010 and incident NCDs such as HT, asthma and MM were observed during a 15-year follow-up. However, there was no association between the AHEI-2010 and CHD, cancer, DM, and depression and/or anxiety. Diet as a preventive factor of NCD in women aged 25–45 years was documented for some NCDs, when accounting for the analysis of time-varying covariates. The effects of time were more significant in lagged models.

In the current study, there was no association between the AHEI-2010 and incident CHD during S4–S8. Some components of the AHEI-2010 are beneficial for cardiovascular health, for example, positively weighted PUFA and negatively weighted SSB[47]. The inverse association between the AHEI-2010 and CHD incidence has been observed in previous studies: in women-only cohorts[68] and in mixed cohorts[69–71]. Compared with previous studies, women in this cohort were younger and have lower number of CHD. Another potential reason might be the limitation of the FFQ which could not be measured with high sugar content drinks such as soda drink and the effect estimates could not be accurate.

When examining the impacts of time in our adjusted models, an association between the AHEI-2010 and HT was found in time-varying and lagged models. Our findings are in line with the previous ALSWH study using different DQI[19,24]. In an ALSWH study that used time-varying GEE analysis, reduced odds of HT was found in women who had high DQ (*n* 5324,

Table 2. Longitudinal associations between the Alternative Healthy Eating Index-2010 (AHEI-2010), covariates and risk of common non-communicable diseases (including multimorbidity) for women from the 1973–1978 Australian Longitudinal Study on Women's Health cohort

	Time-invariant model (model i)		Time-varying model (model ii)		Lagged model (model iii)	
NCD	OR	95 % CI	OR	95 % CI	OR	95 % CI
CHD						
AHEI-2010+covariates†	1·26	0·51, 3·14	1·36	0·56, 3·34	1·39	0·61, 3·17
HT						
AHEI-2010+covariate†	0·77	0·55, 1·08	0·71	0·50, 0·99*	0·67	0·49, 0·91*
Asthma						
AHEI-2010+covariate‡	0·77	0·62, 0·96*	0·62	0·51, 0·76*	0·70	0·57, 0·85*
AHEI-2010+covariate§	0·82	0·65, 1·02	0·70	0·57, 0·86*	0·78	0·64, 0·95*
Cancer (excludes skin cancer)						
AHEI-2010+covariate	1·03	0·66, 1·60	1·35	0·89, 2·04	1·30	0·88, 1·94
DM						
AHEI-2010+covariate‖	0·84	0·46, 1·54	1·29	0·72, 2·31	1·21	0·70, 2·08
Depression and/or anxiety						
AHEI-2010+covariate¶	0·93	0·80, 1·08	0·92	0·80, 1·06	0·98	0·86, 1·12
Multimorbidity						
AHEI-2010+covariate¶	0·87	0·66, 1·14	0·75	0·58, 0·97*	0·81	0·63, 1·04

AHEI-2010, Alternative Healthy Eating Index-2010; CHD: coronary heart disease; DM: diabetes mellitus; NCD: non-communicable disease; HT: hypertension. OR (95 % CI) described in the table is the odds of having NCDs (each disease, multimorbidity), quintile 5 (high diet quality) compared with quintile 1 (low diet quality) of the AHEI-2010.
Model i: generalised estimation equation adjusted for time-invariant covariates at S4 only.
Model ii: generalised estimation equation adjusted for time-varying covariates at S4–S8.
Model iii: generalised estimation equation adjusted for lagged covariates at S3–S7.
Adjusted covariates were marital status, residence, ability to manage income, parity and history of breast-feeding at S4 in model (i), at S4–S8 in model (ii), and at S3–S7 in model (iii) for all NCD outcomes.
* Statistically significant ($P < 0·05$).
† History of hypertension during pregnancy at S4 in model (i), at S4–S8 in model (ii) and at S3–S7 in model (iii) were included as a covariate in each model.
‡ Model without history of asthma.
§ History of asthma at S4 in model (i), at S4–S8 in model (ii) and at S3–S7 in model (iii) were included as a covariate in each model.
‖ History of gestational diabetes mellitus at S4 in model (i), at S4–S8 in model (ii) and at S3–S7 in model (iii) were included as a covariate in each model.
¶ History of depression and/or anxiety at S4 in model (i), at S4–S8 in model (ii) and at S3–S7 in model (iii) were included as a covariate in each model.

aged 50–55 years)[24]. In another study using the lagged GEE approach conducted amongst 7169 women aged 50–55 years at baseline, those consumed pro-inflammatory diet had 24 % increased risk of HT compared with those consumed inflammatory diet[19]. Although similar statistical analyses were used in these two studies[19,24], components of DQI emphasised were different: fruits, vegetables, legumes and nuts in the Australian Recommended Food Score and Mediterranean Diet Score[24], and nutrients, spices, whole food and others in the Dietary Inflammatory Index[19]. Current evidence suggest that fruits[72], vegetables[72], legumes[73], *n*-3 fatty acids and PUFA[74] may be helpful for HT by reducing blood pressure. Despite these components in the AHEI-2010, the previous studies showed conflicting findings[12,56,75,76]. These previous studies were different in sample sizes ranging from 124[75] to 7169[19], study type such as case–control[76] and cohort[12,19,24,56,75], statistical analysis such as logistic regression[12,75,76], multinominal logistic regression[56], time-varying GEE analysis[24] and lagged GEE analysis[19]. Applying lagged GEE analysis has added to the evidence of DQ on HT or blood pressure in longitudinal analyses of cohort studies.

In our sample, women with the highest quintile of the AHEI-2010 had reduced odds of asthma by 23–38 % comparing to the lowest quintile. After accounting for history of asthma in the time-varying and lagged GEE analyses, the effect size was reduced. There is still evolving evidence on DQ and asthma in adults, showing inconclusive results[77]. The cumulative evidence from observational studies indicated potential healthful effects of fruits, vegetables, and vitamin E[78,79], as well as fibre[80], and unhealthful effects of red and processed meat[81,82] and SSB[83] on asthma. Reduced odds of asthma symptoms were documented in two French cohort studies[84,85]. In our repeated cross-sectional study, we found that women in the highest AHEI-2010 quintile had a 25 % reduction in the odds of asthma at 3 years after diet measurement[27]. By contrast, no association between the AHEI-2010 and adult-onset asthma had been reported[86,87]. These mixed findings could be partly explained by the application of different measures for asthma[77] such as continuous scoring variable[84,85] or diagnosis status[12,27,86] or present asthma condition[87], and for measures of diet such as FFQ[12,27,84,86] or 24-h recalls[85,87]. However, the evidence of DQ in adults could be expanded by longitudinal analysis using GEE approach, especially lagged model. In previous studies, various statistical analyses were performed: for example, linear and logistic regressions[87], negative binomial regression[85], mediation analysis[84], and cox proportional hazard modelling[86].

In the current study, there was no evidence of association between the AHEI-2010 and incidence of cancer. The previous analysis that examined the relationship between DQ and cancer of women from the 1946–1951 ALSWH cohort[12] and the 1973–1978 cohort[27] also did not find any association. Nevertheless, an inverse association between the AHEI-2010 and incident cancer was documented in the women-only cohort[47] and mixed cohort[88]. Given that effects of diet on

cancer may be dependent on the type of cancer[47], the impacts could not be found when overall cancer was considered as the outcome in this study. Further, self-reported data of cancer in the ALSWH 1973–1978 cohort were asked separately for skin and other cancers in later surveys (S5–S8). This might affect measurement of cancer and effect sizes in our adjusted models.

Beneficial effects of diet measured by the AHEI-2010 on DM risk have been reported across the world. The reduced risks of DM among participants with higher AHEI-2010 score compared with lower score were found in some studies: those conducted only in women[12,47,89] and in both sexes[90–92]. In contrast to these previous findings, no association was observed in this 1973–1978 ALSWH cohort[27], and the recent analysis performed in the Atherosclerosis Risk in Communities (ARIC) study participants[69]. Collective evidences showed the increased DM risk was related with high intakes of SSB[93], red and processed meat[94], *trans*-fat[95], yet no strong evidence of the protective consequence of PUFA and long-chain *n*-3 fatty acids intakes on DM[96]. A possible explanation of the null finding in this cohort could partly be the low intakes of Australians' *trans*-fat intake which is made up of only 0·6 % of total daily energy[97]. This low intake might not be captured in the scoring of the AHEI-2010, thereby affecting the discriminating power to detect different levels of *trans*-fat in our sample.

DP loaded with vegetables, fruits and wholegrains have been recognised as preventive factors to depression[98]. Vitamins and other micronutrients present in vegetables and fruits have supportive effects on the nervous system[99]. Additionally, diet high in *n*-3 PUFA can reduce pro-inflammatory cytokines[100]. Regarding red and processed meat and SSB, their possible harmful impacts on depression have been suggested in previous studies[101,102]. The scoring of the AHEI-2010 reflects high intakes of favourable diet and low intakes of unfavourable diet[47]. However, inconsistent findings with regard to the association between the AHEI-2010 and depressive symptoms and/or anxiety were reported: no association[12,27,103] and inverse association[104,105]. In an ALSWH study that used GEE lagged analysis for exploring the effects of diet on depression, women who consumed anti-inflammatory diet had lower odds of depression[20]. Comparisons between studies were not directly made because of differences in measurements of diet and statistical analyses. However, longitudinal analysis with time-lagged GEE approach in a cohort study may be useful in nutritional epidemiology.

To date, evidence concerning dietary factors and NCD MM is still evolving[12,106–113]. Previously, a few cross-sectional[107,108] and prospective[109–111] studies demonstrated the reduced odds of NCD MM among those consumed high vegetable and fruits compared with those consumed low intakes. In addition, an analysis of data obtained from 36 663 Australians aged ≥ 16 years indicated that soft drink consumption increased the risk of MM[106]. There were a few studies that investigated the link between DQ and MM[12,112,113]. Preventive potential of diet and MM were demonstrated amongst European adults with high in the modified Mediterranean Diet Score[112,113] and Australian women high in the Healthy Eating Index for Australian Adults-2013 and AHEI-2010[12]. In the present study, longitudinal association between the AHEI-2010 and NCD MM was documented only in the time-varying GEE approach. It may be the effects of time-varying covariates on the occurrence of NCD MM. In finding the effects of diet on NCD MM, longitudinal analysis with time-varying covariates may be useful.

It has been acknowledged that evidence from other research design with appropriate statistical analysis can be used for measuring causal effects in instances where randomised controlled trials are unfeasible[114]. Nutritional epidemiological studies assessing diet–health outcome relationships are no exception, and well-conducted prospective cohort studies with long follow-up periods could be alternatives[115,116]. Although it is challenging to obtain an unbiased estimate from longitudinal studies, appropriate adjustment of time-varying covariates in statistical analysis is an option[117]. In exploring the longitudinal relationship between exposure and outcome, GEE analysis with time-invariant and lagged covariates were used in other epidemiological settings such as in sport medicine[118], occupational health[119] and elderly health[120]. These previous studies observed that the estimates obtained from lagged GEE analysis that accounted for effects of exposure, covariates and outcome over time were more precise than time-invariant models[119,120]. Although we did not find longitudinal association between DQ and some NCDs such as CHD, cancer, DM, and depression and/or anxiety, the findings from longitudinal time-lagged modelling showed that there were effects of time on these outcomes.

A major strength in the application of the GEE estimate is that it has intuitive population-averaged interpretation compared with those of linear regression and allows for the adjustment of time-invariant, time-varying and lagged models in predicting NCD outcomes. In previous ALSWH studies assessing diet–NCD relationships, modellings with GEE approach were used[19,20,24]. One study used GEE time-varying analysis for CVD and HT[24], and two studies used GEE lagged analyses for HT and depression[19,20]. To date, there was no study using different GEE analyses in investigating the relationship between diet and NCD extensively. The findings of the longitudinal relationship between the AHEI-2010 and some NCDs in the 1973–1978 cohort aged 25–45 years, using different GEE analysis, add to the body of evidence related with DQ. Further, it was beneficial that selecting a sample from the data obtained through a nationally representative population of childbearing age women[121], who were free of NCDs before the start of the study. Longitudinal analysis allowing for adjustment of the minimal set of time-varying covariates provides another strength for the current study and the recommended use of GEE for analysing this complex survey data.

There are some study limitations worth noting. Compared with the original sampled population at S1 (*n* 14 247), those excluded from the study due to their NCD status and missing FFQ data (*n* 1059) had low socio-economic and health profile. For example, excluded women had no formal education, no paid job, financial hardship and less physically active (online Supplementary Table 5). Therefore, there may be skewed results towards more healthy participants. Additionally, they were less likely to be represented in higher DQ quintiles such as

quintiles 4 and 5 (data not shown). This distribution could affect the investigation of DQ variations between different groups, making it challenging to identify slight variations which might be biased towards the null. Like other measures of dietary assessment, the self-reported FFQ are subject to measurement error[122]. Since the DQES-v2 included food available in the late 1980s[123], it is likely to lack representative food consumption in the 2000s, particularly in terms of foods rich in *trans*-fat, packaged food, SSB, etc. Hence, there is the possibility of under-reporting in this study. However, Blumfield et al. (2011) have previously shown very similar daily mean energy intakes from the DQES-v2 compared with intakes reported by women aged 25–44 years in the Australian 1995 National Nutrition Survey[124]. Regarding data presentation and analysis in our study, we classified the AHEI-2010 into quintiles which could lead to loss of information and inaccurate effect size[125]. Nevertheless, most nutritional epidemiological studies have been applied categorical data[65]. Diet data were not available for every survey; therefore, measurement of DQ at S3 was used for S4, and measurement of DQ at S5 was used for S6, S7 and S8. However, variables adjusted for in the multivariable analyses were time-varying in model (ii) and model (iii). The outcome assessments were completely relied on self-reported diagnosis of NCD; however, it was shown that self-reported NCD data in the ALSWH participants were acceptable to use[49,50]. The modest number of cases in CHD and DM might lead to wide CIs of our effect sizes. Number of participants with incident cancer might not be accurate, since there was no available separate self-reported data for skin cancer and other cancers in S1–S4. However, when percentages of skin cancer cases in other cancer cases during later surveys were examined, there were not more than 5 % (data not shown). Even though a minimal set of covariates were used in adjustments, possibility of residual confounding cannot be ruled out.

Conclusion

In the longitudinal analysis of diet as a preventive factor of NCD, high DQ measured by the AHEI-2010 was associated with incidence of HT, asthma and MM. A temporal association between diet and some NCDs was evident in lagged GEE analyses. However, the longitudinal analysis of diet and NCDs using GEE analyses is limited. Additionally, direct evidence of the relationship between diet and NCD in the age group of 25 to 45 years is evolving. Further epidemiological analyses performed in young cohorts with updated dietary data focusing on longitudinal analysis with time-varying and lagged covariates are needed to better understand diet–NCD relationships.

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Supplementary material

For supplementary material/s referred to in this article, please visit https://doi.org/10.1017/S0007114523001605

References

1. World Cancer Research Fund/American Institute for Cancer Research (2018) *Continuous Update Project Expert Report 2018. Diet, Nutrition, Physical Activity and Colorectal Cancer*. London: World Cancer Research Fund International.
2. Ocké MC (2013) Evaluation of methodologies for assessing the overall diet: dietary quality scores and dietary pattern analysis. *Proc Nutr Soc* **72**, 191–199.
3. Jacobs DR & Tapsell LC (2007) Food not nutrients, is the fundamental unit in nutrition. *Nutr Rev* **65**, 439–450.
4. Jannasch F, Kroger J & Schulze MB (2017) Dietary patterns and type 2 diabetes: a systematic literature review and meta-analysis of prospective studies. *J Nutr* **147**, 1174–1182.
5. Schulze MB, Martínez-González MA, Fung TT, *et al.* (2018) Food based dietary patterns and chronic disease prevention. *BMJ* **361**, j2396.
6. Tapsell LC, Neale EP & Probst Y (2019) Dietary patterns and cardiovascular disease: insights and challenges for considering food groups and nutrient sources. Curr Atheroscler Rep 21, 9.
7. Newby PK & Tucker KL (2004) Empirically derived eating patterns using factor or cluster analysis: a review. *Nutr Rev* **62**, 177–203.
8. Kourlaba G & Panagiotakos DB (2009) Dietary quality indices and human health: a review. *Maturitas* **62**, 1–8.
9. Kant AK (2004) Dietary patterns and health outcomes. *J Am Diet Assoc* **104**, 615–635.
10. Hodge A & Bassett J (2016) What can we learn from dietary pattern analysis? *Public Health Nutr* **19**, 191–194.
11. Moeller SM, Reedy J, Millen AE, *et al.* (2007) Dietary patterns: challenges and opportunities in dietary patterns research. *J Am Diet Assoc* **107**, 1233–1239.
12. Hlaing-Hlaing H, Dolja-Gore X, Tavener M, *et al.* (2021) Diet quality and incident non-communicable disease in the 1946–1951 cohort of the Australian Longitudinal Study on Women's Health. *Int J Environ Res Public Health* **18**, 11375.

13. Altun A, Brown H, Szoeke C, *et al.* (2019) The Mediterranean dietary pattern and depression risk: a systematic review. *Neurol Psychiatr Brain Res* **33**, 1–10.
14. Cowell OR, Mistry N, Deighton K, *et al.* (2021) Effects of a Mediterranean diet on blood pressure: a systematic review and meta-analysis of randomized controlled trials and observational studies. *J Hypertens* **39**, 729–739.
15. Martínez-González MA, Gea A & Ruiz-Canela M (2019) The Mediterranean diet and cardiovascular health: a critical review. *Circ Res* **124**, 779–798.
16. Martín-Peláez S, Fito M & Castaner O (2020) Mediterranean diet effects on type 2 diabetes prevention, disease progression, and related mechanisms. A review. *Nutrients* **12**, 2236.
17. Morze J, Danielewicz A, Przybyłowicz K, *et al.* (2021) An updated systematic review and meta-analysis on adherence to Mediterranean diet and risk of cancer. *Eur J Nutr* **60**, 1561–1586.
18. Shafiei F, Salari-Moghaddam A, Larijani B, *et al.* (2019) Adherence to the Mediterranean diet and risk of depression: a systematic review and updated meta-analysis of observational studies. *Nutr Rev* **77**, 230–239.
19. Vissers LET, Waller M, van der Schouw YT, *et al.* (2017) A pro-inflammatory diet is associated with increased risk of developing hypertension among middle-aged women. *Nutr Metab Cardiovasc Dis* **27**, 564–570.
20. Shivappa N, Schoenaker DAJM, Hebert JR, *et al.* (2016) Association between inflammatory potential of diet and risk of depression in middle-aged women: the Australian Longitudinal Study on Women's Health. *Br J Nutr* **116**, 1077–1086.
21. Vissers LET, Waller MA, van der Schouw YT, *et al.* (2016) 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. *Atherosclerosis* **253**, 164–170.
22. Lai JS, Hure AJ, Oldmeadow C, *et al.* (2017) Prospective study on the association between diet quality and depression in mid-aged women over 9 years. *Eur J Nutr* **56**, 273–281.
23. Lai JS, Oldmeadow C, Hure AJ, *et al.* (2016) Longitudinal diet quality is not associated with depressive symptoms in a cohort of middle-aged Australian women. *Br J Nutr* **115**, 842–850.
24. Jackson JK, MacDonald-Wicks LK, McEvoy MA, *et al.* (2020) Better diet quality scores are associated with a lower risk of hypertension and non-fatal CVD in middle-aged Australian women over 15 years of follow-up. *Public Health Nutr* **23**, 882–893.
25. Alhazmi A, Stojanovski E, McEvoy M, *et al.* (2014) Diet quality score is a predictor of type 2 diabetes risk in women: the Australian Longitudinal Study on Women's Health. *Br J Nutr* **112**, 945–951.
26. Lee M, Bradbury J, Yoxall J, *et al.* (2022) Is dietary quality associated with depression? An analysis of the Australian Longitudinal Study on Women's Health data. *Br J Nutr* **129**, 1380–1387.
27. Hlaing-Hlaing H, Dolja-Gore X, Tavener M, *et al.* (2022) Alternative Healthy Eating Index-2010 and incident non-communicable diseases: findings from a 15-year follow up of women from the 1973–1978 cohort of the Australian Longitudinal Study on Women's Health. *Nutrients* **14**, 4403.
28. Zeger SL, Liang K-Y & Albert PS (1988) Models for longitudinal data: a generalized estimating equation approach. *Biometrics* **44**, 1049–1060.
29. Carrière I & Bouyer J (2002) Choosing marginal or random-effects models for longitudinal binary responses: application to self-reported disability among older persons. *BMC Med Res Methodol* **2**, 1–10.
30. Hanley JA, Negassa A, Edwardes MD, *et al.* (2003) Statistical analysis of correlated data using generalized estimating equations: an orientation. *Am J Epidemiol* **157**, 364–375.
31. Hin LY & Wang YG (2009) Working-correlation-structure identification in generalized estimating equations. *Stat Med* **28**, 642–658.
32. Zeger SL, Liang K-Y & Self SG (1985) The analysis of binary longitudinal data with time independent covariates. *Biometrika* **72**, 31–38.
33. Stiratelli R, Laird N & Ware JH (1984) Random-effects models for serial observations with binary response. *Biometrics* **40**, 961–971.
34. Harris ML, Egan N, Forder PM, *et al.* (2021) Increased chronic disease prevalence among the younger generation: findings from a population-based data linkage study to inform chronic disease ascertainment among reproductive-aged Australian women. *PLoS One* **16**, e0254668.
35. Hlaing-Hlaing H, Pezdirc K, Tavener M, *et al.* (2020) Diet quality indices used in Australian and New Zealand adults: a systematic review and critical appraisal. *Nutrients* **12**, 3777.
36. National Health and Medical Research Council (Australia) (2013) *Australian Dietary Guidelines*. Canberra, Australia: National Health and Medical Research Council.
37. Shivappa N, Hebert JR, Kivimaki M, *et al.* (2017) Alternative Healthy Eating Index 2010, Dietary Inflammatory Index and risk of mortality: results from the Whitehall II cohort study and meta-analysis of previous Dietary Inflammatory Index and mortality studies. *Br J Nutr* **118**, 210–221.
38. Dobson AJ, Hockey R, Brown WJ, *et al.* (2015) Cohort profile update: Australian Longitudinal Study on Women's Health. *Int J Epidemiol* **44**, 1547.
39. Lee C, Dobson AJ, Brown WJ, *et al.* (2005) Cohort profile: the Australian longitudinal study on women's health. *Int J Epidemiol* **34**, 987–991.
40. Loxton D, Tooth L, Harris ML, *et al.* (2018) Cohort profile: the Australian longitudinal study on Women's health (ALSWH) 1989–1995 cohort. *Int J Epidemiol* **47**, 391–392e.
41. Ayuso-Mateos JL, Nuevo R, Verdes E, *et al.* (2010) From depressive symptoms to depressive disorders: the relevance of thresholds. *Br J Psychiatry* **196**, 365–371.
42. Dobson A, Forder P, Hockey R, *et al.* (2020) The Impact of Multiple Chronic Conditions: Findings from the Australian Longitudinal Study on Women's Health. https://alswh.org.au/wp-content/uploads/2020/09/ALSWH-Major-Report-Multimorbidity-2020.pdf (accessed 15 March 2022).
43. Ireland P, Jolley D, Giles G, *et al.* (1994) Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving an ethnically diverse cohort. *Asia Pac J Clin Nutr* **3**, 19–31.
44. Hodge A, Patterson AJ, Brown WJ, *et al.* (2000) The Anti Cancer Council of Victoria FFQ: relative validity of nutrient intakes compared with weighed food records in young to middle-aged women in a study of iron supplementation. *Aust N Z J Public Health* **24**, 576–583.
45. Lewis J, Milligan GC & Hunt A (1995) *NUTTAB95: Nutrient Data Table for Use in Australia*. Barton, Australia: Food Standards Australia New Zealand.
46. McCullough ML, Feskanich D, *et al.* (2002) Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. *Am J Clin Nutr* **76**, 1261–1271.

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47. Chiuve SE, Fung TT, Rimm EB, *et al.* (2012) Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr* **142**, 1009–1018.
48. Kennedy TE, Ohls J, Carlson S, *et al.* (1995) The Healthy Eating Index: design and applications. *J Am Diet Assoc* **95**, 1103–1108.
49. Cristina TJN, Williams JAS, Parkinson L, *et al.* (2016) Identification of diabetes, heart disease, hypertension and stroke in mid- and older-aged women: comparing self-report and administrative hospital data records. *Geriatr Gerontol Int* **16**, 95–102.
50. Stavrou E, Vajdic CM, Loxton D, *et al.* (2011) The validity of self-reported cancer diagnoses and factors associated with accurate reporting in a cohort of older Australian women. *Cancer Epidemiol* **35**, e75–80.
51. Hébert JR, Frongillo EA, Adams SA, *et al.* (2016) Perspective: randomized controlled trials are not a panacea for diet-related research. *Adv Nutr* **7**, 423–432.
52. Heinze G, Wallisch C & Dunkler D (2018) Variable selection–a review and recommendations for the practicing statistician. *Biom J* **60**, 431–449.
53. Tennant PW, Murray EJ, Arnold KF, *et al.* (2021) Use of directed acyclic graphs (DAGs) to identify confounders in applied health research: review and recommendations. *Int J Epidemiol* **50**, 620–632.
54. Textor J, Hardt J & Knüppel S (2011) DAGitty: a graphical tool for analyzing causal diagrams. *Epidemiology* **22**, 745.
55. Temple N (2015) The possible importance of income and education as covariates in cohort studies that investigate the relationship between diet and disease. *F1000Research* **4**, 690.
56. Al-Ibrahim AA & Jackson RT (2019) Healthy Eating Index *v.* Alternate Healthy Index in relation to diabetes status and health markers in US adults: NHANES 2007–2010. *Nutr J* **18**, 26.
57. Asghari G, Mirmiran P, Yuzbashian E, *et al.* (2017) A systematic review of diet quality indices in relation to obesity. *Br J Nutr* **117**, 1055–1065.
58. Peacock AS, Bogossian F, McIntyre HD, *et al.* (2014) A review of interventions to prevent type 2 diabetes after gestational diabetes. *Women Birth* **27**, e7–e15.
59. Mongraw-Chaffin ML, Peters SA, Huxley RR, *et al.* (2015) The sex-specific association between BMI and coronary heart disease: a systematic review and meta-analysis of 95 cohorts with 1·2 million participants. *Lancet Diabetes Endocrinol* **3**, 437–449.
60. Jackson C, Herber-Gast G-C & Brown W (2014) Joint effects of physical activity and BMI on risk of hypertension in women: a longitudinal study. *J Obes* **2014**, 271532.
61. Peters U, Dixon AE & Forno E (2018) Obesity and asthma. *J Allergy Clin Immunol* **141**, 1169–1179.
62. Tyrrell J, Mulugeta A, Wood AR, *et al.* (2019) Using genetics to understand the causal influence of higher BMI on depression. *Int J Epidemiol* **48**, 834–848.
63. Bhaskaran K, Douglas I, Forbes H, *et al.* (2014) Body-mass index and risk of 22 specific cancers: a population-based cohort study of 5.24 million UK adults. *Lancet* **384**, 755–765.
64. Gondek D, Bann D, Brown M, *et al.* (2021) Prevalence and early-life determinants of mid-life multimorbidity: evidence from the 1970 British birth cohort. *BMC Public Health* **21**, 1319.
65. Willett W (2013). *Nutritional Epidemiology*. 3rd ed. New York, United States: Oxford University Press Inc.
66. Engels JM & Diehr P (2003) Imputation of missing longitudinal data: a comparison of methods. *J Clin Epidemiol* **56**, 968–976.
67. Liang K-Y & Zeger SL (1986) Longitudinal data analysis using generalized linear models. *Biometrika* **73**, 13–22.
68. Shan Z, Li Y, Baden MY, *et al.* (2020) Association between healthy eating patterns and risk of cardiovascular disease. *JAMA Intern Med* **180**, 1090–1100.
69. Xu Z, Steffen LM, Selvin E, *et al.* (2020) Diet quality, change in diet quality and risk of incident CVD and diabetes. *Public Health Nutr* **23**, 329–338.
70. Trebuchet A, Julia C, Fezeu L, *et al.* (2019) Prospective association between several dietary scores and risk of cardiovascular diseases: is the Mediterranean diet equally associated to cardiovascular diseases compared to National Nutritional Scores? *Am Heart J* **217**, 1–12.
71. Hu EA, Steffen LM, Coresh J, *et al.* (2020) Adherence to the healthy eating index–2015 and other dietary patterns may reduce risk of cardiovascular disease, cardiovascular mortality, and all-cause mortality. *J Nutr* **150**, 312–321.
72. Gibbs J, Gaskin E, Ji C, *et al.* (2021) The effect of plant-based dietary patterns on blood pressure: a systematic review and meta-analysis of controlled intervention trials. *J Hypertens* **39**, 23–37.
73. Martini D, Godos J, Marventano S, *et al.* (2021) Nut and legume consumption and human health: an umbrella review of observational studies. *Int J Food Sci Nutr* **72**, 871–878.
74. Colussi G, Catena C, Novello M, *et al.* (2017) Impact of *n*-3 polyunsaturated fatty acids on vascular function and blood pressure: relevance for cardiovascular outcomes. *Nutr Metab Cardiovasc Dis* **27**, 191–200.
75. Wu PY, Huang CL, Lei WS, *et al.* (2016) Alternative health eating index and the Dietary Guidelines from American Diabetes Association both may reduce the risk of cardiovascular disease in type 2 diabetes patients. *J Hum Nutr Diet* **29**, 363–373.
76. Neelakantan N, Naidoo N, Koh W-P, *et al.* (2016) The Alternative Healthy Eating Index is associated with a lower risk of fatal and nonfatal acute myocardial infarction in a Chinese adult population. *J Nutr* **146**, 1379–1386.
77. Bédard A, Li Z, Ait-Hadad W, Camargo Jr CA, *et al.* (2021) The role of nutritional factors in asthma: challenges and opportunities for epidemiological research. *Int J Environ Res Public Health* **18**, 3013.
78. Guilleminault L, Williams EJ, Scott HA, *et al.* (2017) Diet and asthma: is it time to adapt our message? *Nutrients* **9**, 1227.
79. Leynaert B, Le Moual N, Neukirch C, *et al.* (2019) Environmental risk factors for asthma developement. *Presse Medicale* (Paris, France: 1983) **48**, 262–273.
80. Andrianasolo RM, Hercberg S, Kesse-Guyot E, *et al.* (2019) Association between dietary fibre intake and asthma (symptoms and control): results from the French national e-cohort NutriNet-Santé. *Br J Nutr* **122**, 1040–1051.
81. Andrianasolo RM, Hercberg S, Touvier M, *et al.* (2020) Association between processed meat intake and asthma symptoms in the French NutriNet-Santé cohort. *Eur J Nutr* **59**, 1553–1562.
82. Li Z, Rava M, Bédard A, Dumas O, *et al.* (2017) Cured meat intake is associated with worsening asthma symptoms. *Thorax* **72**, 206–212.
83. DeChristopher LR & Tucker KL (2018) Excess free fructose, high-fructose corn syrup and adult asthma: the Framingham Offspring Cohort. *Br J Nutr* **119**, 1157–1167.
84. Li Z, Kesse-Guyot E, Dumas O, *et al.* (2017) Longitudinal study of diet quality and change in asthma symptoms in adults, according to smoking status. *Br J Nutr* **117**, 562–571.
85. Andrianasolo RM, Kesse-Guyot E, Adjibade M, *et al.* (2018) Associations between dietary scores with asthma

symptoms and asthma control in adults. *Eur Respir J* **52**, 1702572.
86. Varraso R, Chiuve SE, Fung TT, *et al.* (2015) Alternate Healthy Eating Index 2010, risk of chronic obstructive pulmonary disease among US women, men: prospective study. *BMJ* **350**, h286.
87. Han Y-Y, Jerschow E, Forno E, *et al.* (2020) Dietary patterns, asthma, and lung function in the Hispanic Community Health Study/Study of Latinos. *Ann Am Thorac Soc* **17**, 293–301.
88. Lavalette C, Adjibade M, Srour B, *et al.* (2018) Cancer-specific and general nutritional scores and cancer risk: results from the prospective NutriNet-Sante cohort. *Cancer Res* **78**, 4427–4435.
89. Cespedes EM, Hu FB, Tinker L, *et al.* (2016) Multiple healthful dietary patterns and type 2 diabetes in the Women's Health Initiative. *Am J Epidemiol* **183**, 622–633.
90. Jacobs S, Harmon BE, Boushey CJ, *et al.* (2015) A priori-defined diet quality indexes and risk of type 2 diabetes: the Multiethnic Cohort. *Diabetologia* **58**, 98–112.
91. Hodge AM, Karim MN, *et al.* (2021) Association between diet quality indices and incidence of type 2 diabetes in the Melbourne Collaborative Cohort Study. *Nutrients* **13**, 4162.
92. Chen G-C, Koh W-P, Neelakantan N, *et al.* (2018) Diet quality indices and risk of type 2 diabetes mellitus: the Singapore Chinese Health Study. *Am J Epidemiol* **187**, 2651–2661.
93. Malik VS & Hu FB (2019) Sugar-sweetened beverages and cardiometabolic health: an update of the evidence. *Nutrients* **11**, 1840.
94. Papier K, Fensom GK, Knuppel A, *et al.* (2021) Meat consumption and risk of 25 common conditions: outcome-wide analyses in 475 000 men and women in the UK Biobank study. *BMC Med* **19**, 53.
95. Wang Q, Imamura F, Ma W, *et al.* (2015) Circulating and dietary trans fatty acids and incident type 2 diabetes in older adults: the Cardiovascular Health Study. *Diabetes Care* **38**, 1099–1107.
96. Brown TJ, Brainard J, Song F, *et al.* (2019) *n*-3, *n*-6, and total dietary polyunsaturated fat for prevention and treatment of type 2 diabetes mellitus: systematic review and meta-analysis of randomised controlled trials. *BMJ* **366**, l4697.
97. Wu J, Downs S, Catterall E, *et al.* (2017) *Levels of Trans Fats in the Food Supply and Population Consumption in Australia: an Expert Commentary Rapid Review Brokered by the Sax Institute The National Heart Foundation of Australia*. Sydney: Sax Institute.
98. Rahe C, Unrath M & Berger K (2014) Dietary patterns and the risk of depression in adults: a systematic review of observational studies. *Eur J Nutr* **53**, 997–1013.
99. Li Y, Lv M-R, Wei Y-J, *et al.* (2017) Dietary patterns and depression risk: a meta-analysis. *Psychiatry Res* **253**, 373–382.
100. Kiecolt-Glaser JK (2010) Stress, food, and inflammation: psychoneuroimmunology and nutrition at the cutting edge. *Psychosom Med* **72**, 365.
101. Hu D, Cheng L & Jiang W (2019) Sugar-sweetened beverages consumption and the risk of depression: a meta-analysis of observational studies. *J Affect Disord* **245**, 348–355.
102. Nucci D, Fatigoni C, Amerio A, *et al.* (2020) Red and processed meat consumption and risk of depression: a systematic review and meta-analysis. *Int J Environ Res Public Health* **17**, 6686.
103. Adjibade M, Lemogne C, Julia C, *et al.* (2018) Prospective association between adherence to dietary recommendations and incident depressive symptoms in the French NutriNet-Santé cohort. *Br J Nutr* **120**, 290–300.
104. Sánchez-Villegas A, Henríquez-Sánchez P, Ruiz-Canela M, *et al.* (2015) A longitudinal analysis of diet quality scores and the risk of incident depression in the SUN Project. *BMC Med* **13**, 197.
105. Saneei P, Hajishafiee M, Keshteli AH, *et al.* (2016) Adherence to Alternative Healthy Eating Index in relation to depression and anxiety in Iranian adults. *Br J Nutr* **116**, 335–342.
106. Shi Z, Ruel G, Dal Grande E, *et al.* (2015) Soft drink consumption and multimorbidity among adults. *Clin Nutr ESPEN* **10**, e71–e76.
107. Jeong D, Kim J, Lee H, *et al.* (2020) Association of cardiometabolic multimorbidity pattern with dietary factors among adults in South Korea. *Nutrients* **12**, 2730.
108. Pereira BP, Bortolotto CC, Tomasi E, *et al.* (2020) Food consumption and multimorbidity among non-institutionalized elderly people in Pelotas, 2014: a cross-sectional study. *Epidemiol Serv Saude* **29**, e2019050.
109. Ruel G, Shi Z, Zhen S, *et al.* (2014) Association between nutrition and the evolution of multimorbidity: the importance of fruits and vegetables and whole grain products. *Clin Nutr* **33**, 513–520.
110. Wikström K, Lindström J, Harald K, *et al.* (2015) Clinical and lifestyle-related risk factors for incident multimorbidity: 10-year follow-up of Finnish population-based cohorts 1982–2012. *Eur J Intern Med* **26**, 211–216.
111. Dekker LH, de Borst MH, Meems LM, *et al.* (2019) The association of multimorbidity within cardio-metabolic disease domains with dietary patterns: a cross-sectional study in 129 369 men and women from the Lifelines cohort. *PLoS One* **14**, e0220368.
112. Freisling H, Viallon V, Lennon H, *et al.* (2020) Lifestyle factors and risk of multimorbidity of cancer and cardiometabolic diseases: a multinational cohort study. *BMC Med* **18**, 1–11.
113. Kyprianidou M, Panagiotakos D, Faka A, *et al.* (2021) Adherence to the Mediterranean diet in Cyprus and its relationship to multi-morbidity: an epidemiological study. *Public Health Nutr* **24**, 4546–4555.
114. West SG, Duan N, Pequegnat W, *et al.* (2008) Alternatives to the randomized controlled trial. *Am J Public Health* **98**, 1359–1366.
115. Satija A, Yu E, Willett WC, *et al.* (2015) Understanding nutritional epidemiology and its role in policy. *Adv Nutr* **6**, 5–18.
116. Allman-Farinelli M, Byron A, Collins C, *et al.* (2014) Challenges and lessons from systematic literature reviews for the Australian dietary guidelines. *Aust J Prim Health* **20**, 236–240.
117. Clare PJ, Dobbins TA & Mattick RP (2019) Causal models adjusting for time-varying confounding—a systematic review of the literature. *Int J Epidemiol* **48**, 254–265.
118. Twisk J (1997) Different statistical models to analyze epidemiological observational longitudinal data: an example from the Amsterdam Growth and Health Study. *Int J Sports Med* **18**, S216–S224.
119. Hoogendoorn W, Bongers P, De Vet H, *et al.* (2002) Comparison of two different approaches for the analysis of data from a prospective cohort study: an application to work related risk factors for low back pain. *Occup Environ Med* **59**, 459–465.
120. Lin K-C, Chen P-C, JWR T, *et al.* (2010) Time-varying nature of risk factors for the longitudinal development of disability in older adults with arthritis. *J Epidemiol* **20**, 460–467.
121. Powers J & Loxton D (2010) The impact of attrition in an 11-year prospective longitudinal study of younger women. *Ann Epidemiol* **20**, 318–321.

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122. Dietary Assessment Primer. Food Frequency Questionnaire at a Glance. National Institutes of Health, National Cancer Institute. https://dietassessmentprimer.cancer.gov/profiles/questionnaire/ (accessed 23 May 2023).
123. Cancer Council Victoria (2019) *Dietary Questionnaire for Epidemiological Studies Version 2 (DQES v2): User Guide*. Victoria, Australia: Cancer Epidemiology Division-Cancer Council Victoria.
124. Blumfield ML, Hure AJ, MacDonald-Wicks LK, *et al.* (2011) Disparities exist between National food group recommendations and the dietary intakes of women. *BMC Womens Health* **11**, 1–9.
125. Bennette C & Vickers A (2012) Against quantiles: categorization of continuous variables in epidemiologic research, and its discontents. *BMC Med Res Methodol* **12**, 1–5.