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Diet quality and progression from health to chronic disease, multimorbidity and mortality in the UK Biobank

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Abstract

Background: Diet quality may influence the progression from health to chronic disease, multimorbidity, and death, but current evidence is limited.

Methods: We aimed to assess the association between diet quality and the risk of transitioning among four health states using multi-state models: from a healthy (disease-free) state to developing one predefined chronic disease, to multimorbidity and lastly, to death. A total of 84,293 healthy individuals (40-70 years) from the UK Biobank were included and diet quality was assessed through four separate exposures using well-established scores: the Alternate Mediterranean Diet Index (aMED), the Dietary Approaches to Stop Hypertension (DASH), the Alternative Healthy Eating Index 2010 (AHEI-2010), and the healthful Plant-based Diet Index (hPDI).

Results: Over 11.2 years follow-up, 22,723 participants developed one predefined chronic disease and 4368 progressed to multimorbidity. A total of 2886 deaths occurred: 770 after multimorbidity, 1512 after one chronic condition, and 604 among participants who did not develop any of the diseases under study. Higher adherence to aMED, DASH, and AHEI-2010 was associated with a reduced risk of developing one chronic disease [HR (95% CI) for highest vs. lowest tertile: 0.92 (0.89, 0.95), 0.94 (0.91, 0.98), and 0.92 (0.89, 0.95)]. aMED was also associated with lower risk of death without any disease [HR: 0.72 (0.56, 0.92)]. aMED and DASH were associated with lower risk of progression to multimorbidity [HRs: 0.92 (0.86, 0.98) and 0.90 (0.83, 0.98)]. aMED and hPDI were associated with lower risk of death after a first disease [HRs: 0.89 (0.79, 1.00) and 0.87

(0.77, 0.98)]. All scores except hPDI were associated with a reduced risk of death after multimorbidity [0.76 (0.61, 0.95) for aMED, 0.71 (0.59, 0.86) for DASH, and 0.80 (0.65, 0.97) for AHEI-2010].

Conclusions: Our findings indicate that greater adherence to healthy dietary patterns was associated with a reduced risk of progression towards one predefined chronic condition, multimorbidity and death, highlighting their potential protective role in long-term health.

Keywords: multimorbidity; chronic disease; mortality; dietary patterns; transitions; multi-state models

Background

The increase in life expectancy has made the occurrence of chronic diseases, frequently several at once, a common situation in older adults (1). Multimorbidity, defined as the coexistence of two or more chronic conditions in an individual, is associated with reduced quality of life, disability and increased risk of mortality (2,3). A growing range of lifestyle factors, including smoking status, alcohol intake, and decreased physical activity, have all been associated with the development of multimorbidity (4-6); however, more information is needed to explain multimorbidity clusters and their trajectories over time and how lifestyles modify such trajectories (7).

Higher diet quality, as assessed by different dietary patterns, has consistently been associated with a lower risk of developing individual chronic diseases (8-10), whereas unhealthy dietary patterns have shown the

opposite direction (8). More recently, higher diet quality has been associated with a lower risk of multimorbidity (11-13).

Understanding whether diet quality is equally important throughout all stages on the progression towards multimorbidity and death can provide a more comprehensive view of how diet influences health. Although diet quality is well known to influence major chronic diseases, previous studies have examined this relationship with incident multimorbidity considered as a single outcome or in terms of specific multimorbidity clusters (14-17). Much less is known about how diet quality relates to the progression of health over time, including the progression from being disease-free to developing a first chronic condition and subsequently multimorbidity and mortality.

Several dietary scores have been developed to assess adherence to evidence-based dietary recommendations. We selected four well-established indices: the Alternate Mediterranean Diet score (aMED), the Dietary Approaches to Stop Hypertension (DASH) score, the Alternative Healthy Eating Index-2010 (AHEI-2010), and the healthful Plant-Based Diet Index (hPDI), because they capture complementary dimensions of diet quality that have been consistently linked to chronic disease prevention. The aMED score reflects adherence to the Mediterranean dietary pattern (18); the DASH score focuses on dietary components relevant to cardiometabolic health (19); the AHEI-2010 score incorporates food- and nutrient-based

recommendations associated with lower chronic disease risk (20); and the hPDI characterises the quality of plant-based dietary patterns (21).

Therefore, the aim of this study was to assess the associations between diet quality, as measured with four dietary indexes, and the risk of transitioning between four health states using a multi-state modelling approach: from a healthy state (disease-free) to developing one chronic disease, multimorbidity and lastly, death.

Methods

Study design and participants

The UK Biobank Study is a large prospective population-based cohort study in the United Kingdom, with more than 500,000 participants aged 40-70 years, where baseline information on demographics, health status and lifestyle was obtained between 2006 and 2010 (22,23). Participants also provided biological samples and underwent a physical examination. Subsequent follow-ups were conducted in 2012-2013, 2014-2019, and 2019-2022. This study was conducted under the ethical approval obtained by the UK Biobank from the NHS National Research Ethics Service (reference 11/NW/0382, June 17, 2011). Written and signed informed consent was provided by all study participants.

Dietary assessment

Food consumption data was collected using the Oxford Web-Q questionnaire (24,25). This computerized questionnaire was administered at five distinct

time points between 2009 and 2012: at study baseline in 2009-2010 to 210,842 participants, and up to four additional rounds in 2011-2012. Only participants with a known email address were invited by the UK Biobank to complete the questionnaires, so food consumption data was not available for the whole cohort. The Web-Q includes questions about the intake of 200 commonly consumed foods and beverages and participants are asked if they have consumed any of them during the previous day; positive responses trigger follow-up questions in which participants select the type and amount of food consumed based on standard serving categories or portions. The data collection approach used in this tool could be defined as a hybrid between a 24-hour dietary recall and a food frequency questionnaire (25,26). The Web-Q automatically calculates nutrient intakes from UK-specific food composition tables (27,28). Correlations between macronutrients assessed with an interviewer-administered 24-hour dietary recall and the Oxford Web-Q range between 0.59 and 0.66 (25).

Only participants who completed at least two diet questionnaires were included in our study, and average food consumption and nutrient intake from all available Web-Qs were calculated for each participant to reflect their habitual diet (29). Four diet quality indexes based on the information obtained were calculated: the Alternate Mediterranean Diet score (aMED) (18), the Dietary Approaches to Stop Hypertension (DASH) (19), the Alternative Healthy Eating Index (AHEI-2010) (20) and the healthful Plant-Based Diet Index (hPDI) (21). These scores were selected because they have

been widely validated as reliable measures of overall diet quality that reflect well-established dietary guidelines. These indexes positively score the consumption of food groups or nutrients known to provide health benefits and negatively score the components associated with a higher risk of chronic disease. For the aMED, the median consumption of each item by the study participants was used to define adherence. For the DASH and hPDI, scores were assigned according to quintiles of participants' consumption. The AHEI-2010 uses predefined cut-off points for each item in the index. For all the diet quality scores, a higher overall score indicates greater adherence to the dietary pattern.

Some modifications were made to calculate the dietary patterns according to the dietary information available in the UK Biobank. When it was not possible to categorize participants into quintiles of individual items consumption, tertiles or above/below the median were calculated. For the AHEI-2010, fish intake was considered as a proxy for eicosapentaenoic and docosahexaenoic acids (EPA + DHA) intake, whereas polyunsaturated fatty acids (PUFAs) were calculated as the sum of n-3 and n-6 fatty acids (as reported by the UK Biobank). For the hPDI, the vegetable oil item was not calculated because the UK Biobank does not provide information on the quantity of oil intake. Lastly, the unhealthful Plant-Based Diet Index (uPDI) was also calculated as a secondary exposure to compare the results of the healthy dietary pattern considered with this unhealthy pattern, as it captures a diet characterised by higher intake of less-healthful plant foods

(**Additional file 1: Table S1 and Table S2**). The correlations between the dietary scores were moderate (aMED with DASH: 0.67; aMED with AHEI-2010: 0.65; aMED with hPDI: 0.40; DASH with AHEI-2010: 0.71; DASH with hPDI: 0.61; and AHEI-2010 with hPDI: 0.57).

Assessment of chronic diseases and multimorbidity occurrence

Participants were followed up to assess the occurrence of nine chronic diseases previously used in the Whitehall II study (30) and the UK Biobank (31), to define chronic disease incidence and multimorbidity: cancer, chronic obstructive pulmonary disease (COPD), dementia, Parkinson's disease, stroke, depression, osteoarthritis, diabetes, and coronary heart disease (CHD).

Cancer diagnoses were obtained through linkage with national cancer registries (International Classification of Diseases (ICD)-10 codes C00-C97, or ICD-9 codes 140.1-208.9). COPD, dementia, Parkinson's disease and stroke cases were identified using the UK Biobank algorithmically defined outcomes (32), which are a combination of medical conditions reported at baseline and hospital admissions records (diagnoses and procedures). COPD was defined through hospital diagnoses ICD-10 codes J43-J44, or ICD-9 codes 492.0, 492.8, 492.9, 496.9) or self-report. Dementia was defined through hospital diagnoses (ICD-10 codes A81.0, F00-F03, F05.1, F10.6, G30-G30.9, G31.1, G31.2, G31.8, I67.3, or ICD-9 codes 290.2-290.4, 291.2, 294.1, 331.0-331.2, 331.5). Parkinson's disease was defined through hospital diagnoses (ICD-10 codes G20- G23, G25.9, G26, G90.3, or ICD-9

codes 332.0, 332.1, 333.0). Stroke was defined through hospital diagnoses (ICD-10 codes I60-I61, I63-I64, or ICD-9 codes 430.9, 431.9, 434.9, 436.9).

Depression, osteoarthritis, diabetes and coronary heart disease were defined by a combination of ICD-10 and ICD-9 codes, self-reported information and medications used (except for CHD). Depression was defined as hospital diagnoses (ICD-10 codes F32-33), self-report or use of antidepressants (fluoxetine, citalopram, escitalopram, sertraline, lustral, paroxetine, or bupropion). Osteoarthritis was defined by hospital diagnoses (ICD-10 codes M15-19 or ICD-9 codes 715) and self-report. Diabetes was defined as hospital diagnoses (ICD-10 codes E10-14), or HbA1c ≥ 48 mmol/mol, self-report, or use of diabetes medication (insulin, nateglinide, repaglinide, glipizide, glimepiride, metformin, rosiglitazone, pioglitazone, or acarbose). CHD was defined through hospital diagnoses (ICD-10 codes I20-25, or ICD-9 codes 413.9, 414.0, 429.2, 412.9, 414.1, 414.8, 414.9, 415.1, 416.0, 416.1, 417.8, 417.9), operative procedures according to the Office of Population Censuses and Surveys Classification of Interventions and Procedures version 4 (K40-49, K50, K75, U19) and self-report. As no medication start date was available, we used the date of self-reported illness; otherwise, we used the date of the interview when participants first reported taking the medication.

We included only participants without any prevalent condition at baseline, that is, those who were free of the nine chronic diseases considered. Information on chronic disease incidence was considered from the date of

the first available 24-hour food recall until May 2022. Multimorbidity was defined as the development of two or more of these chronic diseases in the same individual (33), and the date of multimorbidity onset was the date of diagnosis of the second incident disease. Since there is not a consensus on how to define multimorbidity (34), we used a definition including leading causes of death in high-income countries that seemed adequate for our data (we lacked information of diagnosis from Primary Care for all the participants), which had previously been used in other studies in the UK (30,31).

Mortality

All-cause mortality data was obtained from death certificates held by the National Health Service Information Centre (England and Wales) and the National Health Service Central Register Scotland (Scotland) up to May 2022 (35).

Other variables

Baseline information included age, sex, educational level (primary education or less, secondary education, or university level), the Townsend deprivation index, and smoking status (never, former or current smoker). Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared, with weight and height measured under standardized conditions. Physical activity was assessed using the Short International Physical Activity Questionnaire in metabolic equivalent tasks-hours/week (METs-h/wk) (36).

Sedentary time was considered as the sum of television hours, computer hours, and driving time per day. Sleep time was obtained from the following question: “About how many hours sleep do you get in every 24 hours? (please include naps)”. Energy intake (kcal/d), macronutrient intake (% of total energy intake), and alcohol consumption (g/d) were obtained from the 24-hour food recalls.

Statistical Analysis

In the sample of 210,842 participants with dietary information, we excluded 84,100 participants with less than two 24-hour food recalls and 107 with implausible (<800 or >5000 kcal/d for men, and <500 or >4000 kcal/d for women) (30) or missing energy intake. Of the 126,635 eligible individuals, we also excluded 39,621 participants with any prevalent chronic disease at baseline. Finally, 1396 individuals were excluded if the date of the second dietary recall was after the chronic disease diagnosis, 347 with missing data on covariates, and 979 participants because their first and second chronic disease diagnoses had the same date. The analytical sample comprised 84,293 participants at baseline (**Additional file 1: Fig. S1**).

Participants were categorized into tertiles of the dietary pattern scores. Person-years of follow-up were calculated from the first available 24-hour food recall until the occurrence of the first chronic condition, multimorbidity, death, or the end of the study follow-up (May 2022). Analyses were performed with multi-state models (four possible states starting from a healthy state) using the Weibull distribution to estimate

hazard ratios (HRs) and their 95% confidence intervals (CIs) for the association of each tertile of the four calculated dietary scores and transitions from: (1) a healthy state at baseline to developing one predefined chronic disease, (2) a healthy state at baseline to death, (3) having one predefined chronic disease to multimorbidity, (4) having one predefined chronic disease to death, and (5) multimorbidity to death (**Figure 1**). Survival time for each transition was defined as the time from baseline (first dietary assessment between 2009-2012) to the occurrence of the next transition. Participants were censored at the earliest of: end of follow-up, lost to follow-up, or death (if death was not the outcome of the transition being modelled). Participants who did not progress to a particular state contributed with risk time until their last known status. Death was the absorbing state. This modelling approach allows us to explicitly model disease progression though intermediate states, while keeping a unified time scale and risk structure across transitions (37-39). A parametric Weibull distribution was chosen because it allows for monotonic hazards, provides stable estimates across multiple transitions, and showed adequate fit to these data. We implemented a unidirectional multi-state model with four possible states (healthy, one chronic disease, multimorbidity, death). Transitions were restricted to forward movement only. The model was specified under a Markov proportional hazards framework. The time scale was time since the first dietary assessment. Individuals entered the model

in the healthy state and were followed until a transition occurred or they were censored.

Two models were sequentially fitted. The first model was adjusted for age and sex. A second model was subsequently adjusted for educational level (primary education or less, secondary education, and university degree), Townsend deprivation index, tobacco use (current, former, or never smoker), BMI (<25, 25-29.9, ≥30 kg/m^2), physical activity (quintiles of METs h/wk), sedentary time (quintiles of h/d), sleep time (<7, 7-8, >8 h/d), total energy intake (quintiles of kcal/d), and alcohol consumption (quintiles of g/d). Covariates with known non-linear associations with the outcomes, including BMI, physical activity, sedentary time, sleep duration, total energy intake and alcohol intake, were modelled using quintiles rather than continuous terms. This strategy avoids imposing linearity assumptions and allows for flexible adjustment, and is an approach commonly used in nutritional epidemiology (29).

Stratified analyses by age (<60 years and ≥ 60 years) and sex were also performed to examine the association of 1-SD increase in dietary scores and transitions between each possible state according to these subgroups. Interaction was assessed with likelihood ratio tests comparing models with and without an interaction term, defined as the cross-product of the dietary quality score and the stratification variable. Further analyses to examine the association of each tertile of the uPDI with transitions between states were also performed to compare the results with those obtained from the

healthy dietary quality scores. In sensitivity analysis, diagnoses within the first 2 years of follow-up were excluded to account for reverse causality (N = 81,760). We fitted fully adjusted models for a 1-SD increase and for quintiles of each dietary score to better understand the dose-response relationship. The main fully adjusted models were re-estimated including continuous covariates as continuous terms rather than categorical ones. We also adjusted the models for medication use and ethnicity at baseline, and we fitted a model where we excluded adjustment for alcohol intake. We further stratified the analyses by BMI and physical activity categories.

To explore potential disease-specific pathways, we conducted additional exploratory analyses. The nine chronic conditions were grouped into five disease categories: cardiometabolic (coronary heart disease, stroke, diabetes), respiratory (chronic obstructive pulmonary disease), neuropsychiatric (depression, dementia, Parkinson's disease), cancer, and musculoskeletal (osteoarthritis). We identified the first diagnosed chronic condition within each disease category. We then examined associations between diet quality (modelled per 1-SD increase in each dietary score) and transitions from a disease-free state to a first chronic condition within each category, as well as transitions from there to multimorbidity.

All tests were two sided, with $p < 0.05$ considered statistically significant. Analyses were performed with Stata (version 17.0; Stata Corp., College Station). This manuscript follows the recommendations of the

Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) (40).

Results

Mean age of the study participants was 54.9 years (SD 7.85) and 56.0% of them were women. Baseline characteristics of the participants across tertiles of the diet scores are shown in **Table 1**. Compared to participants with the lowest adherence, those with the highest adherence to the dietary patterns (highest scores) were older and women. They were also more educated and less likely to be current smokers, with a lower BMI and reporting greater levels of physical activity and less sedentary time. The highest tertiles of adherence to the diet quality scores were associated with lower energy intake, except for the aMED score; higher carbohydrate and protein intakes, and lower total fat intakes, except for the AHEI-2010 score, but higher intakes of polyunsaturated fat, n-3 and n-6 fatty acids. Regarding monounsaturated fat, individuals in the highest tertiles for the DASH and hPDI scores had a lower intake, whereas for the aMED and AHEI-2010 scores, they had a higher intake. Mean alcohol intake was lower among those with higher adherence to the diet quality scores.

Over a median follow-up of 11.2 years (range 10.4 - 13.1), 22,723 participants developed one chronic disease, and 4368 subsequently progressed to multimorbidity. In total, 2886 deaths occurred: 770 after multimorbidity, 1512 after one chronic disease without prior

multimorbidity, and 604 among participants who did not develop any of the chronic diseases under study (**Figure 1**).

As shown in **Table 2**, higher scores on all the diet indexes except the hPDI were associated with lower risk of developing one predefined chronic disease (Transition 1). The adjusted hazard ratios (HRs) with 95% confidence intervals (CIs) for tertile 3 (T3) vs. tertile 1 (T1) were 0.92 (0.89, 0.96) for the aMED score, 0.94 (0.91, 0.98) for the DASH score, and 0.92 (0.89, 0.95) for the AHEI-2010. For the transition from a healthy state to mortality (Transition 2), only the aMED score showed an association in the fully adjusted models [0.72 (0.56, 0.92)].

Individuals who had already developed one chronic condition could develop another chronic disease during follow-up (Transition 3). For this transition, individuals with higher adherence to the DASH score showed an inverse association with progression to multimorbidity [HR 95% T3 vs. T1: 0.90 (0.83, 0.98)]. Among these individuals, moderate adherence to the dietary scores was associated with a reduced risk of death, but no association was found for the highest level of adherence (Transition 4). Finally, among individuals who developed multimorbidity, those in the highest tertiles of the aMED, DASH, and AHEI-2010 scores had lower risk of mortality (Transition 5) [0.76 (0.61, 0.95), 0.71 (0.59, 0.86), and 0.80 (0.65, 0.97), respectively].

In subgroup analyses by age, the inverse association seen for the DASH score with the risk of developing a chronic disease was only observed for participants ≥60 years (Transition 1), as well as in the transition from a healthy state to mortality (Transition 2). In this transition, higher adherence to the hPDI was associated with higher mortality among those <60 years. For the progression from multimorbidity to mortality (Transition 5), the inverse association was observed for the AHEI-2010 and hPDI among younger participants, but not for the older participants. In subgroup analyses by sex, no significant interactions were found in any of the transitions **(Table 3)**.

When we examined adherence to the uPDI score in relation to the adverse health events considered, individuals in the highest tertile of adherence showed an increased probability of progressing from a healthy state to having one chronic disease [HR 95% T3 vs. T1: 1.08 (1.05, 1.12)], from a healthy state to mortality [1.22 (1.00, 1.50)] and from having one chronic disease to developing multimorbidity [1.09 (1.01, 1.18)] (**Additional file 1: Table S3**). In sensitivity analysis without the first 2 years of follow-up the results remained the same (**Additional file 1: Table S4**). Models further adjusted for medication use at baseline and ethnicity showed similar results to the main analysis **(Additional file 1: Table S5)**. When we excluded alcohol intake from the adjustment set of covariates results remained similar to the main analyses **(Additional file 1: Table S5)**. The models where dietary scores were categorised in quintiles **(Additional file 1:**

Table S6) and as continuous variables per 1-SD increase **(Additional file: Table S7)** were directionally consistent with the primary analyses. However, some differences in statistical significance were observed depending on the dietary score. For the hPDI in Transition 1, associations were weaker and non-significant when modelled in tertiles, but modest inverse associations were observed when modelling hPDI in quintiles (driven by the highest quintile) or per SD. Similar differences in statistical significance were observed between the analyses in tertiles and quintiles/per SD for aMED in Transition 5 and for DASH in Transition 2. Results including covariates as continuous variables instead of categorised into quintiles were largely consistent with the main analyses (**Additional file 1: Table S8**). In stratified analyses by BMI and physical activity, the results remained in the same direction (**Additional file 1: Table S9**). Results from the exploratory analyses by the type of first chronic condition experienced are shown in **Additional file 1: Fig. S2 and S3**. Overall, the patterns observed were consistent with the main analyses examining transitions to any first chronic disease and subsequently to multimorbidity.

Discussion

In this prospective study in the UK Biobank, we examined the association between four diet quality scores and transitions from a healthy state (disease-free from the disease at study) to several adverse health outcomes. We found that, among healthy individuals, a higher adherence to the aMED,

DASH and AHEI-2010 scores was associated with a decreased risk of developing one chronic disease, and that a higher adherence to the aMED score was associated with a reduced risk of mortality. For individuals with a chronic disease, higher adherence to the DASH score was associated with lower risk of developing multimorbidity. For individuals with multimorbidity, a higher adherence to the aMED, DASH and AHEI-2010 scores was associated with reduced risk of mortality. These results suggest that diet quality, as measured by different scores that capture complementary dimensions of diet quality, is associated with transitions at multiple stages of health and disease.

Diet quality has been extensively studied in relation to the development of chronic diseases, the onset of multimorbidity, and mortality. However, its role in shaping the progression between different health and disease outcomes remains crucial to understand. Multimorbidity is a particularly complex outcome in which multiple diseases interact, and the underlying mechanisms are not yet fully understood. In the transition from a healthy state to the development of one chronic disease, we found that all scores except hPDI showed an inverse association, which is consistent with the current evidence that diet plays an important role in reducing the risk of developing some chronic conditions (8-10).

In the progression from having one chronic disease to multimorbidity, only the aMED and DASH scores showed an inverse association. Another study

in the EPIC cohort, found that a higher adherence to the Mediterranean diet, measured by a pyramid-based scale (per 1-SD increase), was inversely associated with the risk of developing cardiometabolic multimorbidity transitioning from a first cardiometabolic disease [HR 0.67 (0.53, 0.84), and 0.80 (0.70, 0.92)] in follow-ups between 10-15 years, respectively (17). In addition, other findings from the UK Biobank showed that the quality of dietary macronutrients influences the risk of developing multimorbidity beyond their quantity. Unhealthy versions of low-carbohydrate and low-fat diets, including high amounts of animal protein, unhealthy fats, and low-quality carbohydrates, were associated with an increased rate of multimorbidity (41). Moreover, findings from a study using the EPIC and UK Biobank cohorts, found that a ten-point increment in the hPDI score was associated with a lower risk of multimorbidity in both cohorts [HR 0.89 (0.83; 0.96), and HR 0.81 (0.76; 0.86)] in the EPIC and UK Biobank, respectively, which contrasts to our findings were we only found significant associations for the DASH and aMED, but not for hPDI and progression to multimorbidity (16).

We also found that all scores except the hPDI showed an inverse association in the transition from multimorbidity to mortality, highlighting the importance of maintaining a high-quality diet in individuals with multiple chronic conditions. A previous study in the UK Biobank found that a healthy diet was not associated with a lower risk of mortality nor with an increase in life years in individuals with multimorbidity. However, this study defined a

healthy diet as consuming at least 5 servings of fruit and vegetables per day (42), which could undermine the effects of other healthy dietary components. Another study in the Sidney Memory and Ageing Study also found no protective associations between a healthy diet, define as rich in protein, fibre, iron, zinc, magnesium, potassium, and folate and reduced mortality risk in individuals with multimorbidity; nonetheless, when this diet was combined with other healthy lifestyles including some physical activities, sleep quality, or social engagement, a protective association was found (43). These studies did not measure diet quality using widely validated scores and used simpler definitions of diet quality; the advantage of the use of well-defined dietary patterns is that they can reflect interactions among food components and nutrients.

Across the five transitions, all four dietary scores generally pointed towards a lower risk. Associations were consistently stronger for early transitions, particularly the transition from healthy state to one chronic disease, where the aMED, DASH, and AHEI-2010 showed similar inverse associations, while the hPDI showed weaker effects. For mortality from a healthy state, inverse associations were observed for the aMED, but estimates for other scores were non-significant. In contrast, later transitions like the one from one chronic disease to multimorbidity showed smaller, and in most cases non-significant associations, with more variation across scores. The aMED, DASH and AHEI-2010 were generally associated with a lower risk of mortality after multimorbidity. These differences suggest that dietary scores

may capture different aspects of diet quality and that they may not influence all stages of disease progression to the same extent.

The hPDI is a plant-based score that favours healthier plant-based foods but penalizes all animal-based food groups. However, not all animal-based foods are associated with poorer health outcomes. For example, fish consumption, especially oily fish, has been associated with some protective effects including a decrease in all-cause and cause specific mortality (44,45) and multimorbidity risk (12), whereas dairy products (46,47) and poultry (44,47) have yielded mixed results. In addition, animal protein is thought to be of higher quality and more bioavailable than plant protein, which may also influence the overall effect of the diet (48,49). Furthermore, the uPDI showed that for Transitions 1, 2 and 3, greater adherence to this dietary pattern was associated with a higher rate for these transitions, supporting the idea that a plant-based pattern in which the foods included are of low quality will also pose health risks, just as a plant-based pattern that equally penalizes all animal-based foods may have a somewhat weaker protective effect. In the EPIC and UK Biobank cohorts (16), a higher adherence to an unhealthy plant-based diet, measured with the uPDI, was also found to be positively associated with multimorbidity risk in UK Biobank. However, this association was not replicated in the EPIC, so this relationship should be further explored.

In some transitions, the second tertile showed a significant association that was lost in the highest tertile, which is consistent with non-linear relationships or threshold effects, suggesting that a moderate diet quality could be more beneficial compared to a lower diet quality, but adherence to higher diet quality is not associated with stronger benefits. Other possible explanations are that this is due to fewer events in tertile 3, residual confounding, or measurement error in dietary exposures that could attenuate the observed associations. The per 1-SD and quintile analyses pointed in the same direction, showing these non-linear trends. The differences in statistical significance found for some dietary scores when modelling them in quintiles/per SD versus tertiles could be due to broader categories, such as tertiles, including a wider range of diet quality scores reducing statistical power more than modelling in quintiles or per SD, which could dilute the contrast with the lowest category.

Potential underlying mechanisms of the effects of these dietary patterns on the health-disease process are related to inflammation, oxidative stress, and metabolic effects (50,51). Lower quality diets characterised by higher intakes of refined and added sugars, saturated and trans fats, sodium, or ultra-processed foods may promote insulin resistance, dyslipidaemia, and other altered physiological processes that cause tissue damage and increase the risk of developing chronic diseases. One of the most important mechanisms is chronic low-grade inflammation, which can promote the release of proinflammatory cytokines such as TNF-α, IL-6 or IL-1β, which

may contribute to these altered processes. In addition, excess nutrients such as glucose or free fatty acids can induce mitochondrial oxidative stress, which promotes the accumulation of reactive oxygen species (ROS) and damage to cellular tissues. Moreover, a diet low in antioxidant nutrients and bioactive compounds, which are mainly found in high quality diets, reduces the organism's ability to neutralize these ROS.

Regarding age groups, lower risk associations were found for the aMED, DASH, and AHEI-2010 in Transition 1 for those aged ≥60 years, highlighting the potential beneficial impact of a healthy diet in older adults, who are at higher risk of developing chronic diseases. This association was also found between these dietary scores in Transition 2, showing that in older individuals, a higher diet quality has an inverse association with all-cause mortality even after adjustment for other relevant confounders; and only for the DASH score in Transition 5, supporting our findings for the whole cohort, where DASH shows some of the stronger lower risk associations in most of the transitions. On the other hand, DASH, AHEI-2010 and hPDI showed an inverse association in Transition 5 in individuals < 60 years, which could be explained by the fact that among younger adults, diet quality could have a greater relevance when individuals have already developed several diseases. Current evidence suggests that multimorbidity accumulates most rapidly in midlife (around 55–65 years) (52), and at this stage diet could have a greater impact on disease trajectories. Other studies have reported that the association between diet

quality and disease accumulation vary by age group, indicating heterogeneity in the relationship between age and diet (53).

The strengths of this study are the large sample size and the length of follow-up. Only participants with at least two completed 24-hour food recalls were included in the study to better reflect their habitual dietary composition. In addition, we used four well-established and widely used dietary quality scores to assess diet quality. We obtained information on chronic disease diagnoses from multiple sources (hospital records, self-report, medication). The use of multi-state models allowed us to identify transitions between different health-disease states considering multiple pathways. Lastly, the analyses were adjusted for relevant confounders. This study also has several limitations. Diet information was self-reported, so there might be a certain level of misclassification in food consumption. Also, dietary intake was measured before the onset of any of the conditions included in our study thus, diet was not updated after each transition, so potential changes in the exposure were not considered in the analysis. However, we excluded the first two years of follow-up to reduce the influence of early diagnoses on dietary behaviour. Moreover, although some covariates have repeated measurements in UK Biobank, these updates were not consistently available nor time-aligned with the dietary assessments period. As a result, changes in lifestyle factors or health status during follow-up may not be fully captured which could potentially bias the results. Other definitions of multimorbidity are possible, including those based on

wider disease lists, different thresholds, or weighted approaches. Nonetheless, the cut-off of two or more conditions is widely used, especially considering that we included nine diseases in total. We opted to include nine high-burden, well-ascertained chronic diseases, consistent with those used in previous UK-based studies such as the Whitehall II (30), which was available at the time the study was conceived. However, this definition excludes other important chronic conditions, such as chronic kidney and liver disease, that substantially contribute to the overall disease burden and have been included in alternative definitions. Narrower lists may underestimate multimorbidity and may not fully capture its heterogeneity. Moreover, diseases commonly diagnosed in primary care (e.g., depression) may be under-ascertained as this information was not available for the whole cohort. This limitation should be considered when interpreting our results. The role of medication on the transitions from having chronic conditions to subsequent outcomes may strongly be significant. Unfortunately, data in the cohort only included self-reported information on medication obtained from specific questionnaire updates, without the possibility to use up-to-date medication when the transition happened. Finally, like in any observational study, some degree of residual confounding may remain despite adjustment for potential confounders. Menopausal status and hormone therapy use, which are factors commonly considered in studies of diet and chronic disease among women were not available and could not be incorporated. Furthermore, it is challenging to

ascertain the role of socioeconomic factors; previous studies have shown inequalities in the risk of developing multimorbidity (30). Although UK Biobank was designed to investigate exposure-disease relationships rather than to be representative of the general population, the selective recruitment of participants may nevertheless influence effect estimates. UK Biobank participants tend to be healthier and of a higher socioeconomic status than the general UK population (54). If diet-disease associations differ by socioeconomic status, as suggested by previous research, the observed associations may disproportionately reflect those present in higher socioeconomic groups, meaning they may not be applicable to more socioeconomically deprived populations. This potential selection bias should therefore be considered when interpreting our findings. Furthermore, we did not consider the impact of genetic factors on multimorbidity.

Conclusions

In conclusion, our findings indicate that higher diet quality assessed by multiple dietary scores is inversely associated with progression towards one chronic condition, multimorbidity and death. Across all transitions, the aMED and DASH showed stronger and more consistent inverse associations, while the AHEI-2010 and particularly the hPDI showed weaker or non-significant patterns. This suggests that these scores may capture distinct dimensions of diet quality with varying relevance for the transitions examined. In older adults, diet quality appeared to have a greater impact among healthy individuals, whereas in younger adults, diet quality seemed

to have a stronger inverse association in individuals with established multimorbidity.

List of abbreviations

AMED: Alternate Mediterranean Diet Index

AHEI-2010: Alternative healthy eating index

BMI: Body mass index

CHD: Coronary heart disease

COPD: Chronic obstructive pulmonary disease

DASH: Dietary Approaches to Stop Hypertension

DHA: Docosahexaenoic acid

EPA: Eicosapentaenoic acid

HPDI: Healthful Plant-based Diet Index

ICD-10: International Classification of Diseases 10

ICD-9: International Classification of Diseases 9

MEDAS: Mediterranean Diet Adherence Screener

METs: Metabolic equivalent tasks

NHS: National Health Service

PUFAS: Polyunsaturated fatty acids

UPDI: Unhealthful Plant-Based Diet Index

Additional file description

Additional file 1. Figure S1. Flow-chart. Figure S2. Exploratory associations between diet quality and transitions to specific first chronic conditions. Figure S3. Exploratory associations between diet quality and transitions from specific first chronic conditions to multimorbidity. Table S1. Components and scoring of the aMED, DASH, aHEI-2010, hPDI and uPDI diet quality scores Table S2. Dietary descriptives of the aMED, DASH, aHEI-2010, hPDI and uPDI diet quality scores. Table S3. Hazard ratios (95% confidence interval) of the transitions from a healthy state to adverse health events across tertiles of the unhealthful Plant-based diet score. Table S4. Hazard ratios (95% confidence interval) for the transitions from a healthy

state to adverse health events across tertiles of the diet quality scores excluding cases within the first two years of follow-up. Table S5. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events across tertiles of the diet quality scores including i) adjustments for ethnicity and number of medications taken, and ii) without adjustment for alcohol intake. Table S6. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events across quintiles of the diet quality scores. Table S7. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events per 1-SD increment. Table S8. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events across tertiles of the diet quality scores with adjustment for confounders as continuous variables. Table S9. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events per 1-SD increment by BMI and physical activity.

Declarations

Ethics approval and consent to participate

This study was conducted under the ethical approval obtained by the UK Biobank from the NHS National Research Ethics Service (reference 11/NW/0382, June 17, 2011). Written informed consent was signed by all study participants.

Consent for publication

Not applicable.

Data Availability

The datasets and analytic code described in the manuscript are not publicly available because the UK Biobank has proprietary rights of the data. External researchers can apply to use the UK Biobank data set by

registering and applying at the website: http://www.ukbiobank.ac.uk/register-apply.

Competing Interests

The authors have no conflicts.

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Authors’ contributions

AV-F, FFC and EL-G designed the research. AV-F performed the statistical analyses. AV-F and EL-G drafted the manuscript. All authors reviewed the

manuscript for substantial intellectual content. All authors read and approved the final manuscript. EL-G supervised the conduct of research and had primary responsibility for final content.

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Table 1. Baseline characteristics across tertiles of diet quality scores in the UK Bioba

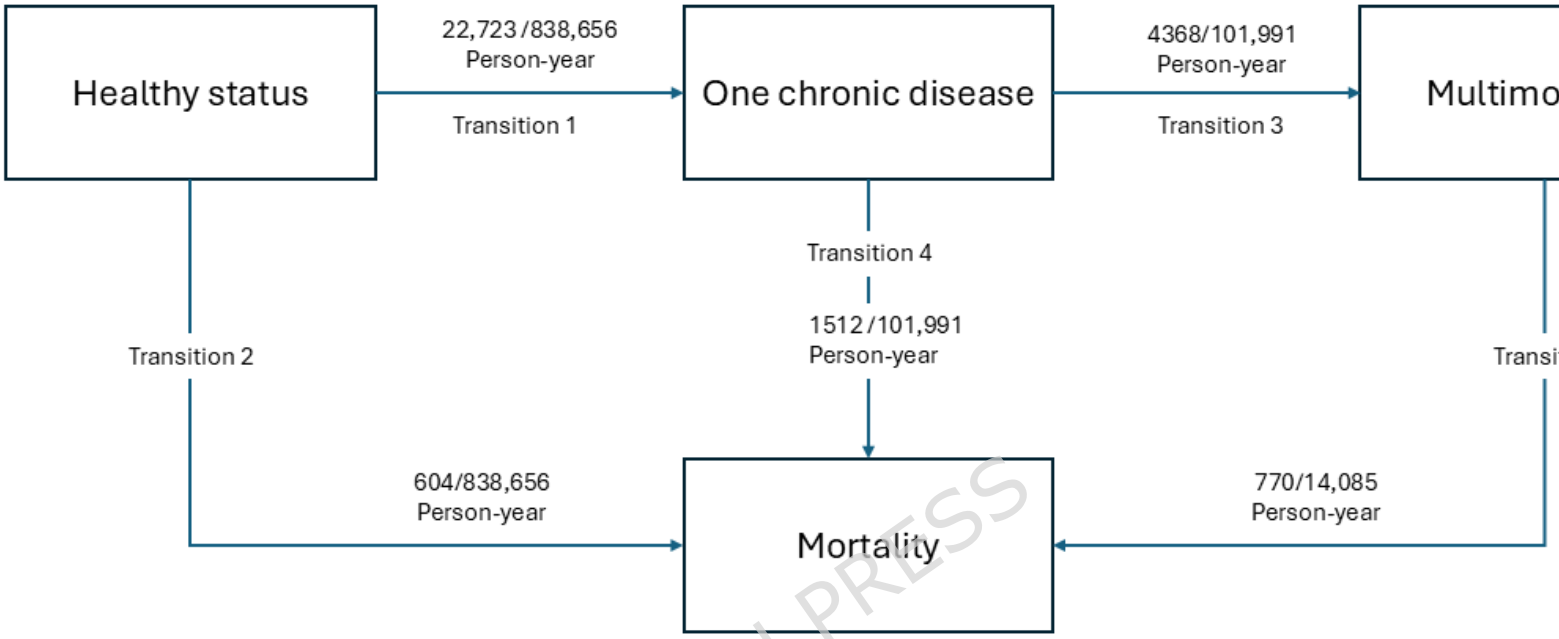
		aMED		DASH		AHEI-20	
	Overall population	Lowest adherence	Highest adherence	Lowest adherence	Highest adherence	Lowest adherence	H ad
Score range		0-3	6-9	8-21	27-40	10-38	4
Participants, n	84,293	36,480	15,374	29,877	23,764	28,098	28
Age, y	54.9 (7.85)	54.3 (7.94)	55.8 (7.65)	53.5 (8.02)	56.2 (7.48)	54.1 (7.98)	55
Sex, female, %	56.0	50.7	64.2	44.4	68.6	43.7	6
Educational level, primary or less, %	5.41	6.00	4.19	5.80	4.78	5.33	5.
Townsend deprivation index, points	-1.66 (2.82)	-1.69 (2.82)	-1.57 (2.86)	-1.62 (2.86)	-1.63 (2.83)	-1.67 (2.82)	-1
Current smoker, %	6.66	8.39	4.36	9.30	4.13	9.16	4.
Body mass index, kg/m^2	26.2 (4.21)	26.7 (4.29)	25.4 (3.97)	26.9 (4.33)	25.4 (4.01)	26.9 (4.25)	25
Physical activity, METs-h/wk*	14.1 (6.02-27.9)	12.5 (4.97-25.8)	16.9 (8.20-30.9)	12.4 (4.92-25.9)	16.2 (7.59-30.6)	13.1 (5.43-26.7)	15 29
Sedentary time, h/d	4.53 (2.25)	4.75 (2.33)	4.17 (2.13)	4.88 (2.38)	4.16 (2.09)	4.81 (2.34)	4.
Sleep duration, h/d	7.16 (0.92)	7.15 (0.93)	7.17 (0.90)	7.14 (0.94)	7.17 (0.91)	7.15 (0.93)	7.
Dietary intake							
Total energy intake, kcal/d	2066 (506)	2041 (516)	2119 (498)	2162 (529)	1981 (463)	2234 (513)	19
Carbohydrate intake, % energy	49.2 (7.35)	48.5 (7.52)	50.5 (6.71)	47.8 (7.39)	51.0 (7.05)	47.7 (7.36)	50
Fat intake, % energy	31.8 (5.77)	32.0 (5.77)	31.7 (5.65)	32.8 (5.60)	30.7 (5.79)	31.8 (5.55)	31
Monounsaturated fat	11.5 (2.44)	11.3 (3.34)	11.8 (2.48)	11.9 (2.31)	11.2 (2.53)	11.4 (2.22)	11
Polyunsaturated fat	5.66 (1.56)	5.16 (1.36)	6.50 (1.62)	5.42 (1.41)	6.00 (1.70)	5.05 (1.22)	6.
n-3 fatty acids	0.88 (0.32)	0.78 (0.27)	1.03 (0.35)	0.83 (0.29)	0.94 (0.35)	0.77 (0.24)	1.
n-6 fatty acids	4.78 (1.40)	4.38 (1.22)	5.47 (1.49)	4.59 (1.25)	5.06 (1.54)	4.28 (1.09)	5.
Protein intake, % energy	15.8 (3.02)	15.8 (3.09)	15.9 (2.84)	15.6 (2.99)	16.0 (3.01)	15.5 (2.79)	16
Alcohol intake, g/d*	11.9 (0.04-26.6)	14.5 (0.02-29.6)	9.53 (3.07-18.7)	13.6 (0.06-30.6)	9.06 (0.02-21.7)	22.3 (0.05-38.1)	9. 16

Values are means (SD) unless otherwise indicated.
* Value is the median (IQR, interquartile range)
aMED: Alternate Mediterranean Diet Index; DASH: Dietary Approaches to Stop Hypertension; AHEI-2010: Alternative Healthy Ea
Diet Index; MET: metabolic equivalent.

Figure 1. Incidence per person-years of the transitions from a healthy state to adverse health conditions (one predefined chronic disease, multimorbidity, and mortality) (N = 84,293). Figures are cases/person-years.

tios (95% confidence interval) for the transitions from a healthy state to adverse health events across tertiles of the diet
,293)

	aMED			DASH			AHEI-2010				
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T



p											
151	698	542	276	567	524	421	555	470	487	563	5
	1.0	0.86 (0.77, 0.97)	0.93 (0,81, 1.07)	1.0	0.87 (0.77, 0.98)	0.88 (0.77, 1.00)	1.0	0.87 (0.77, 0.98)	0.93 (0.82, 1.06)	1.0	0.85 0.9
	1.0	0.89 (0.79, 1.00)	0.98 (0.85, 1.13)	1.0	0.91 (0.80, 1.02)	0.94 (0.82, 1.07)	1.0	0.90 (0.79, 1.02)	0.98 (0.86, 1.13)	1.0	0.87 0.9
70	373	289	108	335	261	174	289	270	211	295	2
	1.0	0.96 (0.83, 1.13)	0.75 (0.60, 0.93)	1.0	0.81 (0.69, 0.96)	0.69 (0.57, 0.84)	1.0	0.97 (0.82, 1.15)	0.87 (0.73, 1.04)	1.0	0.88 1.0
	1.0	0.97 (0.83, 1.13)	0.76 (0.61, 0.95)	1.0	0.83 (0.70, 0.98)	0.71 (0.59, 0.86)	1.0	0.91 (0.76, 1.09)	0.80 (0.65, 0.97)	1.0	0.86 1.0

terranean Diet Index; DASH: Dietary Approaches to Stop Hypertension; AHEI-2010: Alternative Healthy Eating Index 2010; hPDI: h

multi-state (Weibull distribution) model: adjusted for age (years) and sex.
additionally adjusted for educational level (primary or less, secondary and university), Townsend Deprivation Index (points), smoking
y mass index (<25; 25-29.9; ≥ 30 kg/m^2), physical activity (quintiles of METs h/wk), sedentary time (quintiles of h/d), sleeping time (<
al/d) and alcohol intake (quintiles of g/d).

Table 3. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events per 1-SD increment in adherence to the diet quality scores, by age groups and sex, in the UK Biobank (N = 84,293)

	Transition 1 Healthy to 1 chronic disease	**Transition 2** Healthy to mortality	**Transition 3** One chronic disease to multimorbidity	**Transition 4** One chronic disease to mortality	**Transition 5** Multimorbidity to mortality
	1-SD increment	1-SD increment	1-SD increment	1-SD increment	1-SD increment
Age group					
Age < 60 years (n = 55,470)					
N / n cases	55,470/11,444	55,470/259	11,444/1684	11,444/589	1684/227
aMED	0.99 (0.97, 1.01)	1.06 (0.93, 1.20)	0.99 (0.94, 1.04)	1.02 (0.94, 1.11)	0.89 (0.78, 1.03)
DASH	1.02 (1.00, 1.04)†	1.09 (0.96, 1.24)†	0.98 (0.93, 1.03)	1.04 (0.95, 1.13)	0.82 (0.71, 0.94)
AHEI-2010	0.99 (0.97, 1.01)	1.09 (0.95, 1.25)	0.99 (0.94, 1.05)	1.03 (0.94, 1.12)	0.77 (0.66, 0.90)†
hPDI	1.00 (0.98, 1.02)	1.14 (1.00, 1.30)†	0.97 (0.92, 1.02)†	1.05 (0.97, 1.15)	0.76 (0.66, 0.98)†
Age ≥ 60 years (n = 28,823)					
N / n cases	28,823/11,279	28,823/345	11,279/2684	11,279/923	2684/543
aMED	0.97 (0.95, 0.99)	0.85 (0.76, 0.95)	0.97 (0.93, 1.01)	0.98 (0.91, 1.05)	0.95 (0.87, 1.04)
DASH	0.98 (0.96, 0.99)†	0.84 (0.75, 0.94)†	1.01 (0.96, 1.05)	0.96 (0.90, 1.03)	0.91 (0.83, 1.00)
AHEI-2010	0.96 (0.94, 0.98)	0.85 (0.75, 0.97)	1.00 (0.96, 1.05)	0.95 (0.88, 1.03)	0.97 (0.87, 1.07)†
hPDI	0.99 (0.97, 1.01)	0.90 (0.81, 1.01)†	1.02 (0.98, 1.06)†	0.98 (0.91, 1.05)	0.99 (0.90, 1.09)†
Sex					
Men (n = 33,071)					
N / n cases	37,071/11,028	37,071/392	11,028/2360	11,028/827	2360/450
aMED	0.97 (0.95, 0.98)	0.86 (0.78, 0.96)	0.98 (0.94, 1.02)	0.99 (0.92, 1.06)	0.95 (0.86, 1.05)
DASH	0.96 (0.94, 0.98)	0.86 (0.77, 0.96)	0.99 (0.94, 1.03)	0.97 (0.90, 1.05)	0.86 (0.78, 0.95)
AHEI-2010	0.95 (0.93, 0.97)	0.91 (0.81, 1.03)	0.99 (0.94, 1.04)	1.01 (0.93, 1.10)	0.89 (0.79, 1.00)
hPDI	0.97 (0.95, 0.99)	1.01 (0.91, 1.13)	1.01 (0.97, 1.06)	1.00 (0.93, 1.08)	0.90 (0.81, 0.99)
Women (n = 47,222)					
N / n	47,222/11,695	47,222/212	11,695/2008	11,695/685	2008/320

cases					
aMED	0.97 (0.95, 0.99)	1.03 (0.90, 1.19)	0.96 (0.92, 1.01)	0.98 (0.91, 1.06)	0.90 (0.80, 1.01)
DASH	0.98 (0.96, 0.99)	1.01 (0.97, 1.17)	0.96 (0.92, 1.01)	0.97 (0.90, 1.06)	0.90 (0.80, 1.02)
AHEI-2010	0.97 (0.95, 0.99)	0.96 (0.82, 1.12)	0.98 (0.93, 1.03)	0.91 (0.84, 1.00)	0.90 (0.79, 1.02)
hPDI	0.99 (0.97, 1.01)	0.94 (0.81, 1.08)	0.97 (0.92, 1.02)	0.99 (0.91, 1.07)	0.95 (0.84, 1.07)

aMED: Alternate Mediterranean Diet Index; DASH: Dietary Approaches to Stop Hypertension; AHEI-2010: Alternative Healthy Eating Index 2010; hPDI: healthful Plant-based Diet Index; MET: metabolic equivalent.

Score means (SD): aMED 3.86 (1.74); DASH: 23.5 (5.15); AHEI-2010: 42.9 (10.3); hPDI: 53.4 (7.10).

Fully adjusted multi-state (Weibull distribution) model: adjusted for age, sex, educational level (primary or less, secondary and university), Townsend Deprivation Index, smoking status (current, former, or never), body mass index (<25; 25-29.9; ≥ 30 kg/m^2), physical activity (quintiles of METs h/wk), sedentary time (quintiles of h/d), sleeping time (<7 h/d, 7-8 h/d, >8 h/d, energy (quintiles of kcal/d) and alcohol intake (quintiles of g/d), except for the stratification variable.

† indicates that the interaction between diet quality score and age or sex (respectively) was statistically significant.

Figure 1. Incidence per person-years of the transitions from a healthy state to adverse health conditions (one predefined chronic disease, multimorbidity, and mortality) (N = 84,293). Figures are cases/person-years.

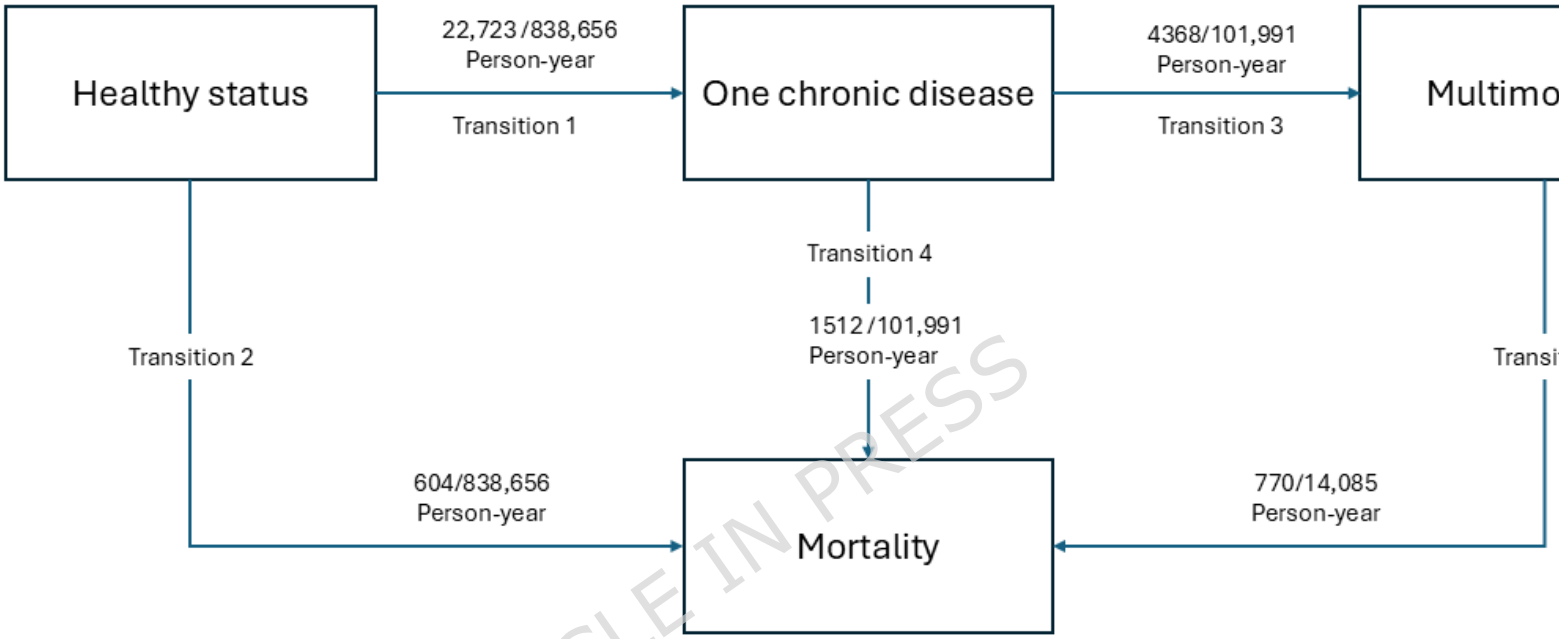


Table 1. Baseline characteristics across tertiles of diet quality scores in the UK Biobank (N = 84,293).

		aMED		DASH		AHEI-2010		
	Overall population	Lowest adherence	Highest adherence	Lowest adherence	Highest adherence	Lowest adherence	Highest adherence	Lowest adherenc
		0-3	6-9	8-21	27-40	10-38	47-93	25-50
	84,293	36,480	15,374	29,877	23,764	28,098	28,097	28,543
	54.9 (7.85)	54.3 (7.94)	55.8 (7.65)	53.5 (8.02)	56.2 (7.48)	54.1 (7.98)	55.5 (7.67)	54.4 (8.06
	56.0	50.7	64.2	44.4	68.6	43.7	66.4	45.8
	5.41	6.00	4.19	5.80	4.78	5.33	5.21	5.76
	-1.66 (2.82)	-1.69 (2.82)	-1.57 (2.86)	-1.62 (2.86)	-1.63 (2.83)	-1.67 (2.82)	-1.57 (2.87)	-1.69 (2.8
	6.66	8.39	4.36	9.30	4.13	9.16	4.52	7.59
	26.2 (4.21)	26.7 (4.29)	25.4 (3.97)	26.9 (4.33)	25.4 (4.01)	26.9 (4.25)	25.4 (4.03)	26.7 (4.35
	14.1 (6.02-27.9)	12.5 (4.97-25.8)	16.9 (8.20-30.9)	12.4 (4.92-25.9)	16.2 (7.59-30.6)	13.1 (5.43-26.7)	15.4 (6.78-29.6)	12.9 (5.31 26.3)
	4.53 (2.25)	4.75 (2.33)	4.17 (2.13)	4.88 (2.38)	4.16 (2.09)	4.81 (2.34)	4.24 (2.16)	4.80 (2.33
	7.16 (0.92)	7.15 (0.93)	7.17 (0.90)	7.14 (0.94)	7.17 (0.91)	7.15 (0.93)	7.16 (0.92)	7.15 (0.93
	2066 (506)	2041 (516)	2119 (498)	2162 (529)	1981 (463)	2234 (513)	1938 (478)	2236 (513
	49.2 (7.35)	48.5 (7.52)	50.5 (6.71)	47.8 (7.39)	51.0 (7.05)	47.7 (7.36)	50.6 (7.20)	48.6 (6.73
	31.8 (5.77)	32.0 (5.77)	31.7 (5.65)	32.8 (5.60)	30.7 (5.79)	31.8 (5.55)	31.9 (6.06)	32.6 (5.24
at	11.5 (2.44)	11.3 (3.34)	11.8 (2.48)	11.9 (2.31)	11.2 (2.53)	11.4 (2.22)	11.6 (2.68)	11.7 (2.13
ed	5.66 (1.56)	5.16 (1.36)	6.50 (1.62)	5.42 (1.41)	6.00 (1.70)	5.05 (1.22)	6.36 (1.74)	5.45 (1.31
	0.88 (0.32)	0.78 (0.27)	1.03 (0.35)	0.83 (0.29)	0.94 (0.35)	0.77 (0.24)	1.00 (0.37)	0.86 (0.28
	4.78 (1.40)	4.38 (1.22)	5.47 (1.49)	4.59 (1.25)	5.06 (1.54)	4.28 (1.09)	5.36 (1.59)	4.59 (1.17
	15.8 (3.02)	15.8 (3.09)	15.9 (2.84)	15.6 (2.99)	16.0 (3.01)	15.5 (2.79)	16.0 (3.19)	15.6 (2.70
d*	11.9 (0.04-26.6)	14.5 (0.02-29.6)	9.53 (3.07-18.7)	13.6 (0.06-30.6)	9.06 (0.02-21.7)	22.3 (0.05-38.1)	9.04 (0.04-16.9)	12.5 (1.28 28.1)

) unless otherwise indicated.
(IQR, interquartile range)
iterranean Diet Index; DASH: Dietary Approaches to Stop Hypertension; AHEI-2010: Alternative Healthy Eating Index 2010; hPDI: h
abolic equivalent.

tios (95% confidence interval) for the transitions from a healthy state to adverse health events across tertiles of the diet c
,293)

	aMED			DASH			AHEI-2010				
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T
c											
22,	9959	8763	4001	8058	8253	6412	7819	7635	7269	7820	8
	1.0	0.94 (0.91, 0.97)	0.87 (0.84, 0.91)	1.0	0.91 (0.88, 0.94)	0.88 (0.85, 0.91)	1.0	0.93 (0.90, 0.96)	0.87 (0.84, 0.90)	1.0	0.98 (1.0
	1.0	0.96 (0.93, 0.99)	0.92 (0.89, 0.96)	1.0	0.95 (0.92, 0.98)	0.94 (0.91, 0.98)	1.0	0.96 (0.93, 0.99)	0.92 (0.89, 0.95)	1.0	1.01 (1.0
604	290	228	86	240	213	151	227	205	172	233	2
	1.0	0.86 (0.72, 1.02)	0.68 (0.53, 0.86)	1.0	0.82 (0.68, 0.99)	0.77 (0.62, 0.95)	1.0	0.92 (0.76, 1.11)	0.79 (0.65, 0.97)	1.0	0.85 (1.0
	1.0	0.89 (0.74, 1.06)	0.72 (0.56, 0.92)	1.0	0.87 (0.72, 1.05)	0.84 (0.68, 1.04)	1.0	0.95 (0.78, 1.16)	0.85 (0.68, 1.05)	1.0	0.87 (1.0
o											
436	2013	1609	746	1652	1539	1177	1570	1468	1330	1572	1
	1.0	0.88 (0.83, 0.94)	0.88 (0.80, 0.95)	1.0	0.87 (0.81, 0.93)	0.83 (0.77, 0.90)	1.0	0.95 (0.88, 1.02)	0.89 (0.83, 0.96)	1.0	0.94 (1.0
	1.0	0.92 (0.86, 0.98)	0.95 (0.87, 1.04)	1.0	0.91 (0.85, 0.97)	0.90 (0.83, 0.98)	1.0	0.99 (0.91, 1.06)	0.95 (0.88, 1.03)	1.0	0.97 (1.0
o											
151	698	542	276	567	524	421	555	470	487	563	5
	1.0	0.86 (0.77, 0.97)	0.93 (0,81, 1.07)	1.0	0.87 (0.77, 0.98)	0.88 (0.77, 1.00)	1.0	0.87 (0.77, 0.98)	0.93 (0.82, 1.06)	1.0	0.85 (0.9
	1.0	0.89 (0.79, 1.00)	0.98 (0.85, 1.13)	1.0	0.91 (0.80, 1.02)	0.94 (0.82, 1.07)	1.0	0.90 (0.79, 1.02)	0.98 (0.86, 1.13)	1.0	0.87 (0.9
70	373	289	108	335	261	174	289	270	211	295	2
	1.0	0.96 (0.83, 1.13)	0.75 (0.60, 0.93)	1.0	0.81 (0.69, 0.96)	0.69 (0.57, 0.84)	1.0	0.97 (0.82, 1.15)	0.87 (0.73, 1.04)	1.0	0.88 (1.0
	1.0	0.97 (0.83, 1.13)	0.76 (0.61, 0.95)	1.0	0.83 (0.70, 0.98)	0.71 (0.59, 0.86)	1.0	0.91 (0.76, 1.09)	0.80 (0.65, 0.97)	1.0	0.86 (1.0

terranean Diet Index; DASH: Dietary Approaches to Stop Hypertension; AHEI-2010: Alternative Healthy Eating Index 2010; hPDI: h

multi-state (Weibull distribution) model: adjusted for age (years) and sex.
additionally adjusted for educational level (primary or less, secondary and university), Townsend Deprivation Index (points), smoking
y mass index (<25; 25-29.9; ≥ 30 kg/m^2), physical activity (quintiles of METs h/wk), sedentary time (quintiles of h/d), sleeping time (<
al/d) and alcohol intake (quintiles of g/d).

Table 3. Hazard ratios (95% confidence interval) for the transitions from a healthy state to adverse health events per 1-SD increment in adherence to the diet quality scores, by age groups and sex, in the UK Biobank (N = 84,293)

	Transition 1 Healthy to 1 chronic disease	**Transition 2** Healthy to mortality	**Transition 3** One chronic disease to multimorbidity	**Transition 4** One chronic disease to mortality	**Transition 5** Multimorbidity to mortality
	1-SD increment	1-SD increment	1-SD increment	1-SD increment	1-SD increment
Age group					
Age < 60 years (n = 55,470)					
N / n cases	55,470/11,444	55,470/259	11,444/1684	11,444/589	1684/227
aMED	0.99 (0.97, 1.01)	1.06 (0.93, 1.20)	0.99 (0.94, 1.04)	1.02 (0.94, 1.11)	0.89 (0.78, 1.03)
DASH	1.02 (1.00, 1.04)†	1.09 (0.96, 1.24)†	0.98 (0.93, 1.03)	1.04 (0.95, 1.13)	0.82 (0.71, 0.94)
AHEI-2010	0.99 (0.97, 1.01)	1.09 (0.95, 1.25)	0.99 (0.94, 1.05)	1.03 (0.94, 1.12)	0.77 (0.66, 0.90)†
hPDI	1.00 (0.98, 1.02)	1.14 (1.00, 1.30)†	0.97 (0.92, 1.02)†	1.05 (0.97, 1.15)	0.76 (0.66, 0.98)†
Age ≥ 60 years (n = 28,823)					
N / n cases	28,823/11,279	28,823/345	11,279/2684	11,279/923	2684/543
aMED	0.97 (0.95, 0.99)	0.85 (0.76, 0.95)	0.97 (0.93, 1.01)	0.98 (0.91, 1.05)	0.95 (0.87, 1.04)
DASH	0.98 (0.96, 0.99)†	0.84 (0.75, 0.94)†	1.01 (0.96, 1.05)	0.96 (0.90, 1.03)	0.91 (0.83, 1.00)
AHEI-2010	0.96 (0.94, 0.98)	0.85 (0.75, 0.97)	1.00 (0.96, 1.05)	0.95 (0.88, 1.03)	0.97 (0.87, 1.07)†
hPDI	0.99 (0.97, 1.01)	0.90 (0.81, 1.01)†	1.02 (0.98, 1.06)†	0.98 (0.91, 1.05)	0.99 (0.90, 1.09)†
Sex					
Men (n = 33,071)					
N / n cases	37,071/11,028	37,071/392	11,028/2360	11,028/827	2360/450
aMED	0.97 (0.95, 0.98)	0.86 (0.78, 0.96)	0.98 (0.94, 1.02)	0.99 (0.92, 1.06)	0.95 (0.86, 1.05)
DASH	0.96 (0.94, 0.98)	0.86 (0.77, 0.96)	0.99 (0.94, 1.03)	0.97 (0.90, 1.05)	0.86 (0.78, 0.95)
AHEI-2010	0.95 (0.93, 0.97)	0.91 (0.81, 1.03)	0.99 (0.94, 1.04)	1.01 (0.93, 1.10)	0.89 (0.79, 1.00)
hPDI	0.97 (0.95, 0.99)	1.01 (0.91, 1.13)	1.01 (0.97, 1.06)	1.00 (0.93, 1.08)	0.90 (0.81, 0.99)
Women (n = 47,222)					

N / n cases	47,222/11,695	47,222/212	11,695/2008	11,695/685	2008/320
aMED	0.97 (0.95, 0.99)	1.03 (0.90, 1.19)	0.96 (0.92, 1.01)	0.98 (0.91, 1.06)	0.90 (0.80, 1.01)
DASH	0.98 (0.96, 0.99)	1.01 (0.97, 1.17)	0.96 (0.92, 1.01)	0.97 (0.90, 1.06)	0.90 (0.80, 1.02)
AHEI-2010	0.97 (0.95, 0.99)	0.96 (0.82, 1.12)	0.98 (0.93, 1.03)	0.91 (0.84, 1.00)	0.90 (0.79, 1.02)
hPDI	0.99 (0.97, 1.01)	0.94 (0.81, 1.08)	0.97 (0.92, 1.02)	0.99 (0.91, 1.07)	0.95 (0.84, 1.07)

aMED: Alternate Mediterranean Diet Index; DASH: Dietary Approaches to Stop Hypertension; AHEI-2010: Alternative Healthy Eating Index 2010; hPDI: healthful Plant-based Diet Index; MET: metabolic equivalent.

Score means (SD): aMED 3.86 (1.74); DASH: 23.5 (5.15); AHEI-2010: 42.9 (10.3); hPDI: 53.4 (7.10).

Fully adjusted multi-state (Weibull distribution) model: adjusted for age, sex, educational level (primary or less, secondary and university), Townsend Deprivation Index, smoking status (current, former, or never), body mass index (<25; 25-29.9; ≥ 30 kg/m^2), physical activity (quintiles of METs h/wk), sedentary time (quintiles of h/d), sleeping time (<7 h/d, 7-8 h/d, >8 h/d, energy (quintiles of kcal/d) and alcohol intake (quintiles of g/d), except for the stratification variable.

† indicates that the interaction between diet quality score and age or sex (respectively) was statistically significant.