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Life expectancy can increase by up to 10 years following sustained shifts towards healthier diets in the United Kingdom

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Adherence to healthy dietary patterns can prevent the development of non-communicable diseases and affect life expectancy. Here, using a prospective population-based cohort data from the UK Biobank, we show that sustained dietary change from unhealthy dietary patterns to the Eatwell Guide dietary recommendations is associated with 8.9 and 8.6 years gain in life expectancy for 40-year-old males and females, respectively. In the same population, sustained dietary change from unhealthy to longevity-associated dietary patterns is associated with 10.8 and 10.4 years gain in life expectancy in males and females, respectively. The largest gains are obtained from consuming more whole grains, nuts and fruits and less sugar-sweetened beverages and processed meats. Understanding the contribution of sustained dietary changes to life expectancy can provide guidance for the development of health policies.

In the United Kingdom, unhealthy diets are estimated to cause more than 75,000 premature deaths each year, including almost 17,000 deaths in the age group 15–70 years[1]. Evidence on the mortality benefits of food choices is essential for the United Kingdom to achieve Sustainable Development Goal target 3.4, which is to reduce premature mortality from non-communicable diseases by one-third by 2030 (ref. 2) Internationally, the Global Burden of Diseases and Injuries consortium and the EAT–*Lancet* commission encourage healthy eating and quantify the population health that is associated with unhealthy eating[3–5]. Furthermore, Public Health England and the UK Government encourage the population to follow the diet pattern recommended in the Eatwell Guide to achieve a healthy and balanced diet[6].

Life expectancy is a measure of expected years an individual has left to live and is a commonly used metric for population health. Higher adherence to the recommendations of the Eatwell Guide is associated with reduced mortality in the United Kingdom[7], but it is not known how a sustained improvement in dietary patterns translates into gains in life expectancy at different stages of life. Estimating such gains in life expectancy would provide policymakers with a measure of the health gains that are possible in a population and provide guidance on which policies would be the most effective. Furthermore, health personnel would also benefit in identifying key risks related to unhealthy dietary patterns with the highest potential for gain when guiding people to prioritize relevant behaviour changes. In addition, most people do not

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	Q1 (lowest)	Q2	Q3 (typical)	Q4	Q5 (highest)
Whole grains	1	0.78 (0.74–0.81)	0.77 (0.73–0.8)	0.82 (0.78–0.86)	0.82 (0.79–0.86)
Vegetables	1	0.95 (0.9–0.99)	0.94 (0.9–0.98)	0.92 (0.87–0.96)	0.93 (0.89–0.97)
Fruit	1	0.88 (0.84–0.91)	0.84 (0.79–0.89)	0.85 (0.81–0.89)	0.86 (0.82–0.9)
Nuts	1	0.82 (0.76–0.89)	0.91 (0.8–1.03)	0.89 (0.73–1.08)	0.81 (0.2–3.24)
Legumes	1	0.91 (0.83–0.98)	1.02 (0.87–1.21)	1.02 (0.6–1.72)	0.72 (0.1–5.11)
Fish	1	0.97 (0.92–1.02)	0.96 (0.92–1.00)	1.03 (0.98–1.09)	0.99 (0.94–1.03)
Egg	1	0.82 (0.73–0.93)	0.85 (0.78–0.93)	0.90 (0.83–0.96)	1.08 (0.95–1.23)
Milk	1	0.99 (0.85–1.16)	0.98 (0.85–1.13)	0.95 (0.82–1.1)	0.93 (0.8–1.08)
Refined grains	1	1.20 (1.12–1.28)	1.17 (1.11–1.23)	1.23 (1.18–1.28)	1.16 (1.11–1.21)
Meat, red	1	1.02 (0.95–1.09)	1.05 (0.99–1.13)	1.18 (1.07–1.29)	1.21 (1.08–1.37)
Meat, processed	1	1.02 (0.96–1.08)	1.13 (1.06–1.2)	1.25 (1.14–1.37)	1.47 (1.27–1.69)
Meat, white	1	0.97 (0.90–1.04)	0.91 (0.85–0.98)	1.00 (0.88–1.15)	0.97 (0.71–1.33)
Sugar-sweetened beverages	1	0.91 (0.83–1)	1.02 (0.9–1.16)	1.22 (0.98–1.52)	1.59 (1.1–2.31)

Fig. 1 | Hazard ratio for all-cause mortality per food group for each quintile (Q1–Q5) among UK Biobank participants. Data are presented as hazard ratios and their 95% confidence intervals. The reference groups were the lowest quintile of intake for each food group. The analyses were adjusted for age, sex, area-based socio-demographic deprivation, smoking, alcohol consumption and physical activity level. The unhealthy categories are shown in red, the longevity associated are shown in green and dark green, and the Eatwell recommendations are shown in blue. The dark green category had large uncertainties; thus, the robust version of the healthiest dietary patterns is in green (not dark green).

adhere to healthy eating guidelines[8], with research showing that less than 0.1% of the UK population adheres to all recommendations of the Eatwell Guide[7]. Therefore, it is important to estimate the gains in life expectancy that are expected from different types of dietary change and various degrees of adherence to the recommendations.

Recently, we developed a model for estimating age- and sex-specific gains or losses in life expectancy following sustained change (that is, dietary changes for remaining lifespan) in the consumption of major food groups with measured intakes[9]. In this paper, we use this model to estimate life expectancy gains from a sustained change from median or unhealthy dietary patterns in the United Kingdom to the longevity-associated dietary pattern, or to the recommendations of the Eatwell Guide.

The median dietary patterns in the United Kingdom were categorized as the intake level with the mid-quintile intake of each of the food groups from the UK Biobank data, from 467,354 participants. The longevity-associated dietary patterns were the quintiles for each food group with the lowest mortality risk estimates or second lowest if the confidence intervals were very wide (Fig. 1). For the unhealthy eating pattern, we used the quintiles for each food group with the highest mortality association, while for the Eatwell Guide, we chose the quintiles of daily intakes fitting best the Eatwell Guide. A detailed description of the methodology is described in Supplementary Information.

Results

Our results showed that the longevity-associated dietary pattern had moderate intakes of whole grains, fruit, fish and white meat; a high intake of milk and dairy, vegetables, nuts and legumes; a relatively low intake of eggs, red meat and sugar-sweetened beverages; and a low intake of refined grains and processed meat (Fig. 1). Analyses adjusting also for body mass index and energy (Supplementary Information) showed slight reductions in inverse associations with mortality for whole grains, vegetables and fruits, reductions in positive associations with mortality for red meat, and stronger inverse associations for both nuts and white meat. For several of the food groups associated with reduced mortality, the lowest intake quintiles were substantially different from the other quintiles. The unhealthy dietary pattern (that is, the quintile with the highest mortality associations) contained no or limited amounts of whole grains, vegetables, fruits, nuts, legumes, fish, milk and dairy, and white meat and substantial intakes of processed meat, eggs, refined grains and sugar-sweetened beverages. The strongest positive associations with mortality were for sugar-sweetened beverages and processed meat, while the strongest inverse associations with mortality were for whole grains and nuts.

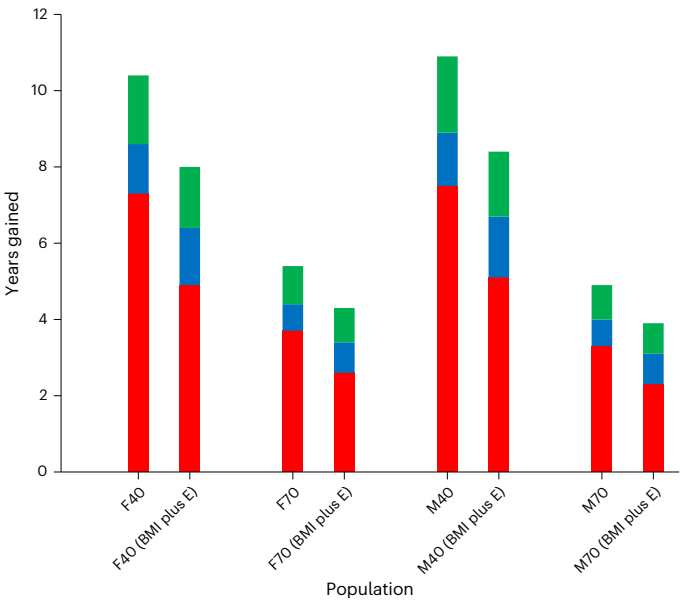

Fig. 2 | Expected life years gained from dietary changes. Expected life years gained after changing from unhealthy median dietary patterns (red), changing from median dietary patterns to the Eatwell Guide (blue) and changing from the Eatwell Guide to longevity-associated dietary patterns (green) for 40-year-old female and male adults (F40 and M40, respectively) and 70-year-old female and male adults (F70 and M70, respectively) from the United Kingdom. Both core-adjusted models (adjusted for age, sex, socio-demographic area, smoking, alcohol consumption and activity level) and mediation models (adjusted for energy and body mass index (BMI plus E)) are presented.

We present life expectancy estimates with uncertainty intervals (UI, indicating the lowest and highest population means that are likely) associated with various dietary patterns in Fig. 2, Table 1 and Supplementary Information. The life expectancy (that is, the estimated remaining years to live) of a 40-year-old with a median dietary pattern was 44.7 years for females and 41.5 years for males. Similarly, the life expectancy of a 70-year-old with a median dietary pattern was 17.6 years for females and 15.5 years for males. Estimated gains from simulated sustained dietary change from a median UK diet pattern to the longevity-associated diet pattern were 3.1 years (UI 1.3–4.9 years) for 40-year-old females and 3.4 years (UI 1.4–5.3 years) for 40-year-old males. Correspondingly, for sustained change to the Eatwell dietary

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Table 1 | Life expectancy associated with various dietary patterns among UK females and males aged 40 and 70 years, and life expectancy gains with uncertainty intervals (UI) changes in dietary patterns

Group	MUK (years)	UUK (years)	EUK (years)	LUK (years)	MUK ➔ LUK (UI years)	UUK ➔ LUK (UI years)	MUK ➔ EUK (UI years)	UUK ➔ EUK (UI years)
UK females, 40 years old	44.7	37.4	46	47.8	3.1 (1.3, 4.9)	10.4 (8.2, 11.3)	1.3 (0.1, 2.4)	8.6 (6.8, 10.2)
UK females, 70 years old	17.6	13.9	18.4	19.3	1.7 (0.7, 2.6)	5.4 (4.4, 6.0)	0.7 (0.0, 1.3)	4.4 (3.6, 5.4)
UK males, 40 years old	41.5	34	42.9	44.8	3.4 (1.4, 5.3)	10.8 (8.8, 12.0)	1.4 (0.1, 2.6)	8.9 (7.2, 10.8)
UK males, 70 years old	15.5	12.2	16.2	17.1	1.6 (0.7, 2.5)	5.0 (4.2, 5.6)	0.7 (0.0, 1.2)	4.0 (3.4, 5.1)
UK females, 40 years old[a]	44.7	39.7	46.1	47.7	3.1 (1.3, 5.1)	8.0 (5.3, 9.7)	1.5 (−0.2, 2.9)	6.4 (4.1, 8.8)
UK females, 70 years old[a]	17.6	15.1	18.4	19.3	1.7 (0.7, 2.7)	4.3 (2.8, 5.2)	0.8 (−0.1, 1.5)	3.4 (2.2, 4.7)
UK males, 40 years old[a]	41.5	36.3	43	44.8	3.3 (1.4, 5.4)	8.4 (5.7, 10.4)	1.6 (−0.2, 3.1)	6.7 (4.3, 9.3)
UK males, 70 years old[a]	15.4	13.1	16.2	17.1	1.6 (0.7, 2.6)	3.9 (2.7, 4.9)	0.8 (−0.1, 1.5)	3.1 (2.1, 4.3)

[a]Extensively adjusted models also adjusted for energy and body mass index. Dietary changes presented: MUK ➔ LUK: from median UK diet (MUK) to longevity-associated diet patterns (LUK); UUK ➔ LUK: from unhealthy UK diet patterns (UUK) to LUK; MUK ➔ EUK: from MUK to the Eatwell Guide dietary pattern (EUK); UUK ➔ EUK: from UUK to EUK.

pattern, estimated gains were 1.3 years (UI 0.1–2.4 years) for females and 1.4 years (UI 0.1–2.6 years) for males. Estimated gains from sustained dietary changes from an unhealthy UK diet pattern to the longevity-associated diet pattern were 10.4 years (UI 8.2–11.3 years) for 40-year-old females and 10.8 years (UI 8.8–12.0 years) for 40-year-old males. Correspondingly, estimated gains from sustained dietary changes from an unhealthy UK diet pattern to the longevity-associated diet pattern were 5.4 years (UI 4.4–6.0 years) for 70-year-old females and 5.0 years (UI 4.2–5.6 years) for 70-year-old males.

Estimated gains from simulated sustained dietary changes from an unhealthy UK diet pattern to full adherence to the Eatwell Guide were 8.6 years (UI 6.8–10.2 years) for 40-year-old females and 8.9 years (UI 7.2–10.8 years) for 40-year-old males. Corresponding gains for 70-year-old females and males were 4.4 years (UI 3.6–5.4 years) and 4.0 years (UI 3.4–5.1 years), respectively.

In sensitivity analyses, sex-stratified analyses of food groups and associations with mortality generally showed similar associations across the sexes except for white meat, which seemed to be more beneficial among females. A range of other sensitivity analyses are presented in Supplementary Information. To reduce potential reverse causation, we performed a landmark analysis that excluded events that occurred within the previous 2 years.

Discussion

In this paper, we present a method for estimating changes in life expectancy following changes in food choices, considering correlation between mortality and food group intakes, and effect delay. Such estimates may be useful particularly for policy purposes and for underpinning both guidance and interventions for improving public health. Our results indicate that UK adults aged 40 years with median dietary patterns can expect to gain approximately 3 years in life expectancy from sustained changes to the longevity-associated dietary patterns. Importantly, the estimated gain in life expectancy is approximately a decade for those shifting from the unhealthiest to the longevity-associated dietary patterns. Overall, the bigger the changes made towards healthier dietary patterns, the larger the expected gains in life expectancy are.

Consuming less sugar-sweetened beverages and processed meats and eating more whole grains and nuts were estimated to result in the biggest improvements in life expectancy. Sensitivity analysis also adjusting for body mass index and energy consumption indicated that body mass index and energy consumption might partially mediate and/or confound a possible beneficial effect between life expectancy and whole grains, vegetables and fruits, and inversely for red meat and eggs. For white meat, associations were stronger when adjusting for energy intake and body mass index, while the situation was mixed for legumes. These estimates correspond well with meta-analyses on associations between intakes of food groups and mortality[10–15]. Our estimates from the UK Biobank are also strengthened by meta-analyses of randomized trials on the consumption of various food groups and scoring of biomarkers for disease[16], mirroring our estimates with nuts, legumes and whole grains performing most beneficially and sugar-sweetened beverages and red meat performing worst. We have also presented estimated health gains associated with adhering to the Eatwell Guide recommendations, showing that the Eatwell Guide does well on the longevity perspective. The life expectancy gains from changes from unhealthy eating to the Eatwell Guide achieve 82–83% of the potential compared with those from sustained change to longevity-associated patterns, which strengthens the evidence base for the promotion of the dietary targets in the Eatwell Guide in public health guidance and interventions. The Eatwell Guide might also be a more realistic target for dietary changes than the statistically set longevity-associated dietary patterns.

Unsurprisingly, predicted gains in life expectancy are lower when the dietary change is initiated at older ages, but these remain substantial. For example, we estimated that people at the age of 70 years could expect to benefit from about half of the life expectancy gain predicted for adults at the age of 40 years, equivalent to a gain in 1.5 years when optimizing median dietary patterns and 4–5 years for those shifting from the unhealthiest dietary patterns. The UK population currently has a life expectancy at birth of 83.6 years for females and 79.9 years for males, and a 3 year gain in life expectancy associated with changes from median to longevity-optimized dietary patterns from the age of 40 years (ref. 17). Life expectancies have steadily increased over time[18], and the observed increase is parallel to the changes in life expectancy observed in the United Kingdom over the past 15 years[17]. A large shift towards healthy dietary patterns could contribute substantially to meeting Sustainable Development Goal target 3.4 that aims to cut premature mortality by one-third.

The governmental food strategy in the United Kingdom to address chronic diseases emphasizes a shared responsibility, with the industry having a responsibility to promote and supply healthier foods, the government having a role in making targeted regulatory interventions to support change, and individual consumers being empowered with better information about healthier choices and thus demanding and seeking healthier foods[19]. With respect to potential policy actions, a recent paper presented approaches to address the substantial inequalities in health in the United Kingdom[20]. The paper argues for five principles, including healthy-by-default and easy-to-use initiatives, long-term and multisector action, locally designed focus, targeting disadvantaged communities and matching of resources to need. The paper further identifies various actions that could contribute to improvements,

such as health-oriented food taxes and subsidies aiming to reduce the cost of healthy foods but not that of unhealthy foods. Other actions were related to improving food environments in school, public and working places by removing vending machines and banning the sale of sugar-sweetened beverages and snacks high in fat, sugar or salt. Such policy measures, informed by the up-to-date estimates on potential gains in life expectancy that we provide in this paper, could guide the deployment of resources to improve healthy eating patterns across the population.

Limitations of our study include correlation between the associations between food groups and mortality. We addressed this through model adjustments presenting a conservative model as the main analyses. As for most cohort studies, confounders can have an impact. However, we have adjusted our core models for several potential confounders such as age, sex, area-based socio-demographic deprivation, smoking, alcohol consumption and physical activity level. We have also added sensitivity analyses investigating for mediation and possible confounding from body mass index and energy intake. Our models are based on population data with the uncertainty measure being on the population-level mean. Nevertheless, even though the individual uncertainty is likely to be larger than uncertainty at the group level, one could expect the mean life expectancy differences of groups of individuals with similar characteristics but variations in group size to be comparable (with uncertainty being higher in smaller groups). Thus, our estimates could be useful also in clinical settings. We model sustained and prolonged dietary changes. However, maintaining lifestyle changes over time with dietary improvements can be challenging, and for many, dietary patterns fluctuate over time. We have not modelled potential changes in life expectancy of fluctuating changes. Furthermore, unhealthy and longevity-associated dietary patterns could be seen as dietary 'constructs' (as associations between dietary patterns and mortality are used to set these). This is not necessarily overlapping with typically presented dietary patterns such as the Mediterranean or the Nordic diets. Furthermore, the UK Biobank does not measure consumption of rice, which is particularly important for many migrant groups. Overall, the UK Biobank data under-represent non-white populations compared to the UK population. Even though the UK Biobank data contain about nearly half a million participants in our analyses, the size of the data is not sufficient to achieve precise estimates across all quintiles and there seems to be some random fluctuations between some of the quintiles. Thus, the exact quintile threshold should be interpreted with caution, and more emphasis should be placed on the general trends. There were limited and selective data in the dietary recall that could result in biases.

In conclusion, for middle-aged adults in the United Kingdom, sustained dietary improvement is predicted to increase life expectancy by about 3 years for both females and males. Importantly, for those with the least healthy dietary patterns, change to the longevity-associated dietary pattern is predicted to translate into approximately 10 years gain in life expectancy. Changing from an unhealthy dietary pattern to eating in line with the Eatwell Guide was associated with an 8 year life expectancy gain. Gains in life expectancy are lower the longer the delay in the initiation of dietary improvements, but even for those initiating dietary change at age 70 years, the gain in life expectancy is about half of that achieved by 40-year-old adults. The biggest gains in life expectancy are associated with increased intake of whole grains and nuts, and with reduced intake of sugar-sweetened beverages and processed meats. Our findings suggest that these food groups should be specific targets for clinicians in the guidance of patients and policymakers in developing public health policy.

Methods

The study complies with all relevant ethical regulations (with approval obtained by UK Biobank from the National Health Service National Research Ethics Service (reference 11/NW/0382). All data included are from participants who provided written informed consent for use of their data. Detailed methods beyond the shortened description in this Brief Communication are provided in Supplementary Information.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

Requests for the dataset can be sent through UK Biobank.

Code availability

The R code is available in Supplementary Information.

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Author contributions

L.T.F., C.C.-M., F.K.H., J.P.P., K.A.J. and J.C.M. designed the study. L.T.F., C.C.-M., J.-M.Ø., S.P.-S. and Ø.A.H. conducted the statistical analysis. R.B., E.J.A. and Ø.A.H. contributed to the modelling. L.T.F. wrote the first draft. C.C.-M., J.-M.Ø., S.P.-S., K.M.L., F.K.H., J.P.P., R.B., E.J.A., Ø.A.H., K.A.J. and J.-M.Ø critically revised the paper. L.T.F., C.C.-M. and J.-M.Ø are the guarantors of the paper and accept full responsibility for the work and/or the conduct of the study, had access to the data and controlled the decision to publish. The corresponding author attests that all listed authors meet authorship criteria and that no authors meeting the criteria have been omitted.

Competing interests

The authors declare no competing interests.

Additional information

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Reporting on sex and gender	*Sex-specific estimates are provided. UKBiobank included 56% females among over 500,000 participants.*
Population characteristics	*Age, sex and population indicators are included in the model. UKBiobank participants were aged 37-73 years at baseline*
Recruitment	*UK Biobank is a prospective cohort study. A total of over 500,000 participants aged 37-73 years at baseline were enrolled, from the general population. In brief, between 2006 and 2010, participants attended one of 22 assessment centres across Scotland, England, and Wales. All participants completed a touch-screen questionnaire, had physical measurements taken, and provided blood, urine, and saliva samples at baseline. A subset of 467,354 participants in UK Biobank for whom diet was assessed at least once using the web-based 24-hour dietary questionnaire Oxford WebQ were included in this study (Text S1, Table S1).*
Ethics oversight	*This study was performed under generic ethical approval obtained by UK Biobank from the National Health Service National Research Ethics Service (approval letter ref 11/NW/0382, 17 June 2011).*

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

☐ Life sciences ☒ Behavioural & social sciences ☐ Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/documents/nr-reporting-summary-flat.pdf

Life sciences study design

All studies must disclose on these points even when the disclosure is negative.

Sample size	*See below*
Data exclusions	*See below*
Replication	*N/A*
Randomization	*N/A*
Blinding	*N/A*

Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	*UK Biobank is a prospective cohort study (quantitative). Our study used modeling with these background data.*
Research sample	*A subset of 467,354 participants in UK Biobank for whom diet was assessed at least once using the web-based 24-hour dietary questionnaire Oxford WebQ were included in this study (56% females).*
Sampling strategy	*A population-based random sample from >500,000 participants aged 37-73 years at baseline were enrolled, from the general population. In brief, between 2006 and 2010, participants attended one of 22 assessment centres across Scotland, England, and Wales. All participants completed a touch-screen questionnaire, had physical measurements taken, and provided blood, urine, and saliva samples at baseline.*
Data collection	*The baseline touchscreen questionnaire that was completed by participants at the assessment centre, was based on the food frequency questionnaire (FFQ), which included 29 items about diet and 18 questions about alcohol. The questionnaire included data on the frequency of consumption of the main food groups over the previous year, including fruits and vegetables, fish, meat, and cheese (details in Table S4). A 24-hour recall-based method, based on the Oxford WebQ, which captured information on up to 206 food and 32 drink items, was introduced towards the end of the recruitment period. Thus, participants recruited between April 2009 and September 2010 completed this at their assessment centre baseline visit. In addition, between February 2011 and June 2012, there were 4 cycles, separated by 3-4 months, where participants were invited via email to complete the 24-hour dietary recall at home. Over 200,000 participants completed at least one 24-hour recall. Further details about the dietary assessments, including*

	reproducibility and agreement between the two methods have been published. In the present study, we used data from the 24-hour recall (Oxford WebQ) to assess adherence to the recommendations on the intakes of nuts, eggs, and sugar-sweetened drinks. We used FFQ data for fruits and vegetables, whole and refined grain, fish, diary, proceed and unprocessed meat and vegetables oils recommendations where the cut-offs are described as intake 'per week' so as not to over or underestimate consumption of these foods. The participants were blinded to the hypotheses of this study.
Timing	*Participants were recruited between April 2009 and September 2010 completed this at their assessment centre baseline visit. In addition, between February 2011 and June 2012, there were 4 cycles, separated by 3-4 months*
Data exclusions	*Persons without dietary assessments or heart disease diagnoses were excluded (approximately 81,645).*
Non-participation	*Of >500,000 participants enrolled in UK Biobank, 467,354 participants had dietary assessments and were included in this study (while the remainder where excluded)*
Randomization	*No randomization was conducted for the study.*

Ecological, evolutionary & environmental sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	*Briefly describe the study. For quantitative data include treatment factors and interactions, design structure (e.g. factorial, nested, hierarchical), nature and number of experimental units and replicates.*
Research sample	*Describe the research sample (e.g. a group of tagged Passer domesticus, all Stenocereus thurberi within Organ Pipe Cactus National Monument), and provide a rationale for the sample choice. When relevant, describe the organism taxa, source, sex, age range and any manipulations. State what population the sample is meant to represent when applicable. For studies involving existing datasets, describe the data and its source.*
Sampling strategy	*Note the sampling procedure. Describe the statistical methods that were used to predetermine sample size OR if no sample-size calculation was performed, describe how sample sizes were chosen and provide a rationale for why these sample sizes are sufficient.*
Data collection	*Describe the data collection procedure, including who recorded the data and how.*
Timing and spatial scale	*Indicate the start and stop dates of data collection, noting the frequency and periodicity of sampling and providing a rationale for these choices. If there is a gap between collection periods, state the dates for each sample cohort. Specify the spatial scale from which the data are taken*
Data exclusions	*If no data were excluded from the analyses, state so OR if data were excluded, describe the exclusions and the rationale behind them, indicating whether exclusion criteria were pre-established.*
Reproducibility	*Describe the measures taken to verify the reproducibility of experimental findings. For each experiment, note whether any attempts to repeat the experiment failed OR state that all attempts to repeat the experiment were successful.*
Randomization	*Describe how samples/organisms/participants were allocated into groups. If allocation was not random, describe how covariates were controlled. If this is not relevant to your study, explain why.*
Blinding	*Describe the extent of blinding used during data acquisition and analysis. If blinding was not possible, describe why OR explain why blinding was not relevant to your study.*

Did the study involve field work? ☐ Yes ☐ No

Field work, collection and transport

Field conditions	*N/A*
Location	*N/A*
Access & import/export	*N/A*
Disturbance	*N/A*

Reporting for specific materials, systems and methods

We require information from authors about some types of materials, experimental systems and methods used in many studies. Here, indicate whether each material, system or method listed is relevant to your study. If you are not sure if a list item applies to your research, read the appropriate section before selecting a response.

Materials & experimental systems

n/a	Involved in the study
☒	☐ Antibodies
☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology and archaeology
☒	☐ Animals and other organisms
☒	☐ Clinical data
☒	☐ Dual use research of concern

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging

Antibodies

Antibodies used: N/A

Validation: N/A

Eukaryotic cell lines

Policy information about cell lines and Sex and Gender in Research

Cell line source(s): N/A

Authentication: N/A

Mycoplasma contamination: N/A

Commonly misidentified lines (See ICLAC register): N/A

Palaeontology and Archaeology

Specimen provenance: N/A

Specimen deposition: N/A

Dating methods: N/A

☐ Tick this box to confirm that the raw and calibrated dates are available in the paper or in Supplementary Information.

Ethics oversight: *Identify the organization(s) that approved or provided guidance on the study protocol, OR state that no ethical approval or guidance was required and explain why not.*

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Animals and other research organisms

Policy information about studies involving animals; ARRIVE guidelines recommended for reporting animal research, and Sex and Gender in Research

Laboratory animals: *For laboratory animals, report species, strain and age OR state that the study did not involve laboratory animals.*

Wild animals: *Provide details on animals observed in or captured in the field; report species and age where possible. Describe how animals were caught and transported and what happened to captive animals after the study (if killed, explain why and describe method; if released, say where and when) OR state that the study did not involve wild animals.*

Reporting on sex: *Indicate if findings apply to only one sex; describe whether sex was considered in study design, methods used for assigning sex. Provide data disaggregated for sex where this information has been collected in the source data as appropriate; provide overall numbers in this Reporting Summary. Please state if this information has not been collected. Report sex-based analyses where performed, justify reasons for lack of sex-based analysis.*

Field-collected samples: *For laboratory work with field-collected samples, describe all relevant parameters such as housing, maintenance, temperature, photoperiod and end-of-experiment protocol OR state that the study did not involve samples collected from the field.*

Ethics oversight	*Identify the organization(s) that approved or provided guidance on the study protocol, OR state that no ethical approval or guidance was required and explain why not.*

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Clinical data

Policy information about clinical studies

All manuscripts should comply with the ICMJE guidelines for publication of clinical research and a completed CONSORT checklist must be included with all submissions.

Clinical trial registration	*Provide the trial registration number from ClinicalTrials.gov or an equivalent agency.*
Study protocol	*Note where the full trial protocol can be accessed OR if not available, explain why.*
Data collection	*Describe the settings and locales of data collection, noting the time periods of recruitment and data collection.*
Outcomes	*Describe how you pre-defined primary and secondary outcome measures and how you assessed these measures.*

Dual use research of concern

Policy information about dual use research of concern

Hazards

Could the accidental, deliberate or reckless misuse of agents or technologies generated in the work, or the application of information presented in the manuscript, pose a threat to:

No	Yes	
☒	☐	Public health
☒	☐	National security
☒	☐	Crops and/or livestock
☒	☐	Ecosystems
☒	☐	Any other significant area

Experiments of concern

Does the work involve any of these experiments of concern:

No	Yes	
☒	☐	Demonstrate how to render a vaccine ineffective
☒	☐	Confer resistance to therapeutically useful antibiotics or antiviral agents
☒	☐	Enhance the virulence of a pathogen or render a nonpathogen virulent
☒	☐	Increase transmissibility of a pathogen
☒	☐	Alter the host range of a pathogen
☒	☐	Enable evasion of diagnostic/detection modalities
☒	☐	Enable the weaponization of a biological agent or toxin
☒	☐	Any other potentially harmful combination of experiments and agents

ChIP-seq

Data deposition

☐ Confirm that both raw and final processed data have been deposited in a public database such as GEO.

☐ Confirm that you have deposited or provided access to graph files (e.g. BED files) for the called peaks.

Data access links *May remain private before publication.*	*For "Initial submission" or "Revised version" documents, provide reviewer access links. For your "Final submission" document, provide a link to the deposited data.*
Files in database submission	*Provide a list of all files available in the database submission.*
Genome browser session (e.g. UCSC)	*Provide a link to an anonymized genome browser session for "Initial submission" and "Revised version" documents only, to enable peer review. Write "no longer applicable" for "Final submission" documents.*

Methodology

Replicates	*Describe the experimental replicates, specifying number, type and replicate agreement.*
Sequencing depth	*Describe the sequencing depth for each experiment, providing the total number of reads, uniquely mapped reads, length of reads and whether they were paired- or single-end.*
Antibodies	*Describe the antibodies used for the ChIP-seq experiments; as applicable, provide supplier name, catalog number, clone name, and lot number.*
Peak calling parameters	*Specify the command line program and parameters used for read mapping and peak calling, including the ChIP, control and index files used.*
Data quality	*Describe the methods used to ensure data quality in full detail, including how many peaks are at FDR 5% and above 5-fold enrichment.*
Software	*Describe the software used to collect and analyze the ChIP-seq data. For custom code that has been deposited into a community repository, provide accession details.*

Flow Cytometry

Plots

Confirm that:

☐ The axis labels state the marker and fluorochrome used (e.g. CD4-FITC).

☐ The axis scales are clearly visible. Include numbers along axes only for bottom left plot of group (a 'group' is an analysis of identical markers).

☐ All plots are contour plots with outliers or pseudocolor plots.

☐ A numerical value for number of cells or percentage (with statistics) is provided.

Methodology

Sample preparation	*Describe the sample preparation, detailing the biological source of the cells and any tissue processing steps used.*
Instrument	*Identify the instrument used for data collection, specifying make and model number.*
Software	*Describe the software used to collect and analyze the flow cytometry data. For custom code that has been deposited into a community repository, provide accession details.*
Cell population abundance	*Describe the abundance of the relevant cell populations within post-sort fractions, providing details on the purity of the samples and how it was determined.*
Gating strategy	*Describe the gating strategy used for all relevant experiments, specifying the preliminary FSC/SSC gates of the starting cell population, indicating where boundaries between "positive" and "negative" staining cell populations are defined.*

☐ Tick this box to confirm that a figure exemplifying the gating strategy is provided in the Supplementary Information.

Magnetic resonance imaging

Experimental design

Design type	*Indicate task or resting state; event-related or block design.*
Design specifications	*Specify the number of blocks, trials or experimental units per session and/or subject, and specify the length of each trial or block (if trials are blocked) and interval between trials.*
Behavioral performance measures	*State number and/or type of variables recorded (e.g. correct button press, response time) and what statistics were used to establish that the subjects were performing the task as expected (e.g. mean, range, and/or standard deviation across subjects).*

Acquisition

Imaging type(s) *Specify: functional, structural, diffusion, perfusion.*

Field strength *Specify in Tesla*

Sequence & imaging parameters *Specify the pulse sequence type (gradient echo, spin echo, etc.), imaging type (EPI, spiral, etc.), field of view, matrix size, slice thickness, orientation and TE/TR/flip angle.*

Area of acquisition *State whether a whole brain scan was used OR define the area of acquisition, describing how the region was determined.*

Diffusion MRI ☐ Used ☐ Not used

Preprocessing

Preprocessing software *Provide detail on software version and revision number and on specific parameters (model/functions, brain extraction, segmentation, smoothing kernel size, etc.).*

Normalization *If data were normalized/standardized, describe the approach(es): specify linear or non-linear and define image types used for transformation OR indicate that data were not normalized and explain rationale for lack of normalization.*

Normalization template *Describe the template used for normalization/transformation, specifying subject space or group standardized space (e.g. original Talairach, MNI305, ICBM152) OR indicate that the data were not normalized.*

Noise and artifact removal *Describe your procedure(s) for artifact and structured noise removal, specifying motion parameters, tissue signals and physiological signals (heart rate, respiration).*

Volume censoring *Define your software and/or method and criteria for volume censoring, and state the extent of such censoring.*

Statistical modeling & inference

Model type and settings *A detailed description is provided in the supplementary files*

Effect(s) tested *A detailed description is provided in the supplementary files*

Specify type of analysis: ☐ Whole brain ☐ ROI-based ☐ Both

Statistic type for inference (See Eklund et al. 2016) *Specify voxel-wise or cluster-wise and report all relevant parameters for cluster-wise methods.*

Correction *Describe the type of correction and how it is obtained for multiple comparisons (e.g. FWE, FDR, permutation or Monte Carlo).*

Models & analysis

n/a	Involved in the study
☐	☐ Functional and/or effective connectivity
☐	☐ Graph analysis
☐	☒ Multivariate modeling or predictive analysis

Functional and/or effective connectivity *Report the measures of dependence used and the model details (e.g. Pearson correlation, partial correlation, mutual information).*

Graph analysis *Report the dependent variable and connectivity measure, specifying weighted graph or binarized graph, subject- or group-level, and the global and/or node summaries used (e.g. clustering coefficient, efficiency, etc.).*

Multivariate modeling and predictive analysis *A detailed description is provided in the supplementary files*