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Dietary pattern trajectories from youth to adulthood and adult risk of impaired fasting glucose: a 31-year cohort study

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Abstract

Context The influence of dietary pattern trajectories from youth to adulthood on adult glucose metabolism is unknown.

Objectives To identify dietary pattern trajectories from youth to adulthood and examine their associations with adult impaired fasting glucose (IFG).

Design, Setting, and Participants 31-year population-based cohort study among 1,007 youths aged 3-18 years at baseline in Finland.

Exposures Diet intake was assessed in 1980, 1986, 2001, 2007 and 2011. Group-based trajectory modelling was used to identify dietary pattern (identified by factor analysis) trajectories.

Main outcome measures Adult IFG was measured by the latest available data from 2001, 2007 and 2011.

Results Among 1,007 participants, 202 (20.1%) developed IFG and 27 (2.7%) developed T2D in adulthood (mean follow-up of 30.7 years; mean (SD) age=40.5 (5.0) years). Three dietary patterns were identified at baseline and were retained in 1986 and 2001: 'Traditional Finnish', 'High-carbohydrate' and 'Vegetables and dairy products'. Three different patterns were identified in 2007, which remained similar in 2011: 'Traditional Finnish and high-carbohydrate', 'Red meat', and 'Healthy'. Trajectories of increased or stably medium 'red meat' pattern scores from youth to adulthood were detrimentally associated with IFG (relative risk=1.46, 95% confidence interval: 1.12-1.90 for M-stable/M-large increase vs. Low-stable trajectory) after adjusting for confounders. This association was slightly reduced after further adjusting for long-term dietary fibre intake.

Conclusions Trajectories of an increased or stably moderate adherence to a 'red meat' dietary pattern from youth to adulthood are associated with higher risk of adult IFG. This association is partly explained by low dietary fibre intake.

Introduction

Type 2 diabetes (T2D) is a major health issue with a global prevalence of diabetes (T2D accounting for >85%) estimated to be 9.3% (463 million) in 2019 and projected to increase to 10.9% (700 million) by 2045(1). Although the global prevalence of T2D in young adults remains low (<5% among those aged <40 years)(1), the number of individuals with prediabetes as assessed by impaired glucose tolerance is alarmingly high in this age group (106 million under age 40 years)(1), leading to significantly increased risk of developing T2D later in life(2). Thus, early prevention is critically important(3-5) with growing evidence showing that early life factors may have profound and lasting impacts on adult glucose metabolism(6-8).

Diet plays a paramount role in the prevention of T2D(9) and dietary pattern analysis are important for assessing the effect of overall diet on health outcomes(10). However, previous studies have been limited to dietary patterns from a single time point during adulthood in relation to glucose metabolism and T2D(11). Only one study using data from the Nurses' Health Study II examined the association between dietary patterns during high school and adult risk of T2D but the diet was retrospectively measured at least 16-35 years later, introducing significant recall bias(12). Nonetheless, the developmental trajectories of dietary patterns from youth to adulthood and their influences on adult glucose metabolism have not been assessed. Addressing this evidence gap could improve current strategies for preventing T2D at an early stage of life through a life-course approach. Therefore, we aimed to identify dietary patterns and their trajectories from youth to adulthood and examine whether these trajectories are associated with adult impaired fasting glucose (IFG).

Subjects and Methods

Study design and participants

The prospective Cardiovascular Risk in Young Finns Study (YFS) began in 1980 (baseline) and was followed up in 1986, 2001, 2007 and 2011. At baseline, 3596 participants (83.2% of those invited) aged 3-18 years were randomly selected from the national register of the study areas. In 1980, a 50% random sample of the participants was selected to participate in the 48-hour dietary recall interview

(N=1798). Of whom, 1767 participated and were reinterviewed in 1986 (n=1212) and 2001 (n=1037). All participants were invited to complete the food frequency questionnaire assessment in 2007 (n=1996) and 2011 (n=1736). Participants with at least three observations of diet data were included to identify dietary pattern trajectories (see methods), (n=1256). To assess the association between dietary pattern trajectories and IFG/T2D, participants were included if they had dietary and complete risk factor data from baseline (1980) and adult fasting glucose or T2D available from either the 2001, 2007 or 2011 surveys but did not have type 1 diabetes and were not pregnant at either the 2001, 2007 or 2011 surveys (n=1007). A flowchart of participation is given in Figure 1. All participants gave written informed consent, and local ethics committees approved the study.

Impaired fasting glucose (IFG) and type 2 diabetes (T2D)

Fasting serum glucose concentration was determined by the enzymatic hexokinase method (Glucose Olympus System Reagent, Olympus, Dublin, Ireland). IFG was defined as having a fasting serum glucose \geq 5.6 but \leq 6.9 mmol/L using the latest available measurement without meeting the criteria for T2D(13). Participants were classified as having T2D if they met one of the following: fasting plasma glucose \geq 7 mmol/L (126 mg/dl); T2D diagnosed by a physician(14); HbA1c \geq 6.5% (48 mmol/mol) at the 2011 follow-up; use of glucose-lowering medication at 2007 or 2011 follow-ups; or being confirmed by National Social Insurance Institution Drug Reimbursement Registry.

Diet intake

Details of diet assessment in 1980, 1986 and 2001 have been described in detail elsewhere (15). Briefly, diet was assessed by trained dietitians using a 48-hour dietary recall method by recording the type and amount of food eaten by the participant during the two days prior to the interview. In 2007 and 2011, diet was assessed by a validated 128-item food frequency questionnaire as described in detail elsewhere(16,17). Participants were asked to fill in the form about their usual eating habits during the past 12 months, which were presented under 12 subgroups (e.g., dairy products, vegetables, and fruits and berries). Food consumption was calculated in grams per day by the National Food Composition Database(18).

Other factors

All measures were from baseline unless otherwise stated. Height and weight were measured in 1980, 2001, 2007 and 2011 and body mass index (BMI) calculated as weight/(height²) (kg/m²). The latest available measures from 2001, 2007 and 2011 were used as adulthood BMI. Serum 25hydroxyvitamin D (250HD) concentrations were analysed by radioimmunoassay (DiaSorin, Stillwater, Minnesota)(19). Long-term dietary fibre intake was calculated as the mean of fibre intake in 1986, 2001, 2007 and 2011 measured using the National Food Composition Database in Finland (18). Smoking habits were asked during a health examination in a solitary room. Participants aged 12 years at baseline were considered non-smokers. For those aged 12-18 years at baseline, youth smoking was defined as regular cigarette smoking on a weekly basis (or more often). Additionally, youth smoking was defined as regular cigarette smoking on a daily basis based on follow-up data (follow-ups after 3, 6, 9 and 12 years) when they were aged 12-18 years. Questionnaires were used to collect physical activity information and an age-standardised physical activity index was calculated(20), which has been shown to be reliable and valid(21). We used a parent-completed questionnaire for participants aged 3 and 6 years and self-reported questionnaire for those aged 9 to 18 years. Questionnaires were also used to obtain information on parental history of T2D and years of education (as a measure of socio-economic status).

Statistical analysis

Dietary pattern analysis

All food items were classified into 23 food groups based on similarity and habitual culinary use as done previously(22). Exploratory factor analysis was used to identify dietary patterns for each survey years based on eigenvalues (>1.25) and interpretability of the factors. Three dietary patterns were identified in 1980 with the largest eigenvalues (>1.4)(23). For consistency, three dietary patterns with the largest eigenvalues were retained for all follow-up surveys. Dietary patterns were similar from 1980 to 2001 but had changed by 2007 and then remained similar in 2011. Thus, six different dietary patterns were retained (three each in 1980/1986/2001 and 2007/2011) to broadly represent the

different patterns identified across the five survey years. Dietary pattern scores for the six dietary patterns were calculated for all five survey years by summing the intake of food groups with absolute factor loadings \geq 0.2, weighted by the factor loadings of the food groups(12). To avoid the influence of using different factor loadings on dietary pattern scores for the same dietary pattern, the factor loadings for the three dietary patterns observed in 1980 and the three dietary patterns observed in 2011 were consistently applied to all survey years. At each time point, dietary pattern scores were adjusted for total energy intake using the residual method to account for under- or over-reporting and eliminate the confounding effect of the amount of food consumed(22). Moreover, energy adjusted dietary pattern scores were further standardised by survey years to improve comparability across study years.

Group-based trajectory modelling was used to examine trajectories of dietary pattern scores across surveys(24), using the 'traj' plug-in in Stata version 15.1. This approach assesses the variation of the developmental courses of the variable of interest between groups of individuals in the population. Details for model selection of trajectory analysis are given in the supplemental documents (page 7)(25).

Descriptive and main analyses

Mean (standard deviation) and number (%) were used, as appropriate, to describe variables. Radar charts were used to plot the evolution of dietary patterns from 1980 to 2011 ('Traditional Finnish' to 'Traditional Finnish and high-carbohydrate'; 'High-carbohydrate' to 'Red meat'; 'Vegetables and dairy products' to 'Healthy'). Univariable and multivariable modified Poisson regression models were used to assess the association between trajectories of dietary patterns from youth to adulthood and adult IFG. Given the small number of T2D cases (n=27), we performed sensitivity analyses by repeating these analyses using the combination of IFG and T2D as the outcome. We selected potential confounders based on the biological plausibility of an association of a factor with both the outcome and the exposure of interest. Model 1 was unadjusted and model 2 adjusted for age, sex, BMI, serum 250HD levels, total energy intake, parental history of diabetes physical activity, smoking, socio-economic status at baseline and adult BMI. Due to nonsignificant interactions between sex and the

dietary pattern trajectories, analyses were not stratified by sex. Trajectory categories with a relatively small number of participants were combined with the adjacent category for analyses (e.g., M-large increase for the 'Healthy' pattern). To explore the potential mechanism for the association of the 'red meat' pattern trajectory with IFG, a post-hoc analysis was conducted to further adjust for long-term dietary fibre intake in above-mentioned Model 2.

Missing data for adult BMI (n=3) was imputed using sex, baseline age and BMI as predictors (multiple imputation using chained equations; 20 datasets imputed). Inverse probability of weighting was used to account for missing data(26). Briefly, BMI and energy intake were used to predict the participants' probabilities of being a complete case using logistic regression. We assumed all values were missing at random.

All analyses were performed in Stata version 15.1 (Stata Corporation, Texas, USA). A two-tailed p value <0.05 was considered statistically significant.

Results

Identification of dietary patterns and their trajectories

Three dietary patterns were identified at baseline and named, for descriptive purpose, as 'Traditional Finnish' (characterised by high consumption of rye, potatoes, butter, milk, coffee and sausages but low consumption of fruit and berries), 'High-carbohydrate' (by high consumption of wheat, margarine and oils, sugar, milk, beef and eggs) and 'Vegetables and dairy products' (by high consumption of vegetables, fruits, cheese, other dairy products, tea, beef and alcoholic beverages but low consumption of milk) (Supplemental Table 1(25)). These patterns were similar between 1980 and 2001 (Figure 2, Supplemental Figure 1 and 2, and Supplemental Tables 1-3(25)). By 2007 the patterns had significantly changed and these patterns remained stable in 2011 (age in 1980 and 2011 (years): mean (SD)= 10.5 (5.0) and 41.5 (5.0), respectively; range 3-18 and 34-49, respectively): 'Traditional Finnish and high-carbohydrate' (characterised by high wheat, other grain products, rye, potatoes, butter, sausages and sugar; 'Red meat' (characterised by high consumption of pork, other meats, sausages, eggs, fish, potatoes and alcoholic beverages but low consumption of tea); 'Healthy' (by high

consumption of vegetables, legumes and nuts, fruits, fish, cheese, other dairy products, tea, other meats, eggs) (Figure 2, Supplemental Figure 1 and 2, and Supplemental Tables 4 and 5(25)).

Details for model selection of trajectories are given in the supplemental documents (page 7-8)(25). Three trajectories were identified for each dietary pattern and named based on the starting level and change over time (Figure 3). For example, the H-stable trajectory of 'Traditional Finnish' was named as it started at a high level and remained stable over time (Figure 3, A).

Participants' characteristics and associations of dietary pattern trajectories with IFG

Of the 1,007 participants (53% female), 202 (20.1%) developed IFG and 27 (2.7%) developed T2D in adulthood (mean follow-up=30.7 years). **Table 1** shows participants' characteristics in youth and adulthood by trajectory groups of 'red meat' pattern. After adjusting for confounders, trajectories of increased or stably medium adherence to the 'red meat' pattern from youth to adulthood were associated with increased risk of IFG (**Table 2** Model 2; relative risk=1.46, 95% confidence interval: 1.12-1.90 for M-stable/M-large increase vs. L-stable). Post-hoc analysis further adjusting for long-term dietary fibre intake showed a slightly reduced association (relative risk=1.38, 95% confidence interval: 1.06-1.79). There were no significant associations between the other dietary pattern trajectories and IFG (**Table 2**). Sensitivity analyses combining IFG with T2D and analyses that considered inverse probability weighting showed similar results (data not shown).

Discussion

Our study for the first time examined the long-term evolution of dietary patterns from youth to middle adulthood, identified trajectories of these patterns from youth to middle adulthood and examined the association of these trajectories with adult risk of IFG. Dietary patterns remained stable from youth to early adulthood but significantly changed during the transition to middle adulthood. The trajectories of an increased or stably medium adherence to a 'red meat' dietary pattern from youth to middle adulthood (among more than one third of the participants) were associated with higher risk of adult IFG compared to those who had a stably low trajectory. This finding suggests that interventions to

prevent the increased or high adherence to a dietary pattern characterised by high intake of red meat from youth to middle adulthood may reduce the risk of IFG in adulthood and T2D later in life.

Previous data from our group showed that dietary patterns remained consistent from youth to early adulthood(22) but the current study suggested significant changes in major components of these patterns during the transition from early to middle adulthood. For example, the traditional Finnish pattern appeared to evolve to integrate with a high-carbohydrate pattern. Interestingly, a dietary pattern characterised by high consumption of red meat was seen in adulthood, which does not seem to stem from any patterns observed in youth. These findings confirm that a healthy diet habit should be established as early as in childhood but further suggest that the transition from early to middle adulthood may provide another opportunity for interventions to improve diet behaviours.

Although cohort studies have investigated the association of dietary patterns with glucose metabolism and T2D in adults(11,27), none have assessed dietary pattern trajectories from early life to adulthood or their associations with glucose metabolism in adulthood. The finding from the current study that trajectories of increased or stably medium 'red meat' pattern scores from youth to adulthood were associated with higher risk adult IFG is novel and biologically plausible. Previous systematic review and meta-analyses of observational and intervention studies have consistently shown a detrimental association of red meat intake and risk of T2D in adults(28,29). A recent study in adults showed that the association between the intake of red meat and the risk of T2D was partially explained by the increased content of dietary heme-iron(30). Moreover, a higher red meat intake means a potentially lower intake of fibre, which has been consistently associated with increased risk of T2D(31). Consistent with this finding, when we adjusted for dietary fibre intake we observed a reduction in the association of red meat pattern trajectory with IFG. Our finding has also major public health implication as a large proportion of participants were classified into these 'unfavourable' trajectories (34.9%). Of note, these differences between trajectories were apparent in youth or became distinct in early adulthood. This suggests that interventions to prevent the higher adherence to a dietary pattern characterised by high intake of red meat should start as early as possible, which might most effectively reduce the risk of IFG in adulthood and T2D later in life.

10

Although not statistically significant, an increased adherence to traditional Finnish and highcarbohydrate pattern may have a clinically important association with IFG (29% risk reduction for slight increase vs. stable/large decrease). This may be partly explained by the high intake of whole grains, which have been consistently associated with reduced risk of T2D in recent systematic reviews and meta-analyses of observational and intervention studies(31-33). Not surprisingly, whole grains are rich in dietary fibre, antioxidants, and other important micronutrients and vitamins, which can all play a role in glucose metabolism(32). For example, a recent systematic review and meta-analysis of observational and intervention studies showed that there was moderate evidence for a beneficial association of dietary fibre and T2D in adults(31). Altogether, our finding suggests that improving high quality carbohydrate diet from childhood to adulthood may be important for the prevention of IFG later in life.

Only one cohort study examined the association of early life dietary patterns with adult risk of T2D(12), showing that a Western dietary pattern during adolescence (characterized by a high consumption of desserts, processed meats, and refined grains) was associated with an increased risk of T2D in young to middle age but a prudent pattern (characterised by high consumption of vegetables, fruit, legumes, fish, and better-quality grains and low consumption of snacks and soda) was not. However, a major limitation of that study was the potential recall bias of the adolescence dietary data, which was retrospectively assessed 16-35 years later. Moreover, it is unclear if the adolescent patterns changed during adulthood and whether any changes impacted the risk of T2D. Another study assessed the association of dietary pattern trajectories with glucose metabolism(34), but it was limited to adulthood and assessed only one 'healthy' dietary pattern (characterised by a high consumption of wheat products and soy milk and low consumption of rice, legumes, poultry, eggs and fish) that might be only seen in specific populations. They found that dietary pattern trajectories with higher scores (vs. lower) were beneficially associated with HbA1c but not insulin resistance or diabetes. However, a direct comparison with our findings might not be feasible due to differences in the study design and population characteristics.

11

Few randomised controlled trials (RCT) have determined the effect of dietary pattern interventions for preventing T2D and they have generally focused on improving the intake of fruits and vegetables and maintaining a low carbohydrate diet (35). A large US RCT showed no benefit of an intervention promoting a low-fat (20% total energy) dietary pattern with increased vegetables, fruits and grains for preventing diabetes in 48,835 generally healthy postmenopausal women(35). However, there was a trend toward reduced risk with greater decreases in total fat intake and weight loss. These findings are generally consistent with ours for the 'healthy' pattern. In contrast, another large US RCT among adults with elevated fasting and post-load plasma glucose (n=3,234) showed that those who received a lifestyle intervention of a healthy low-calorie, low-fat diet, and improved physical activity of moderate intensity, reduced the incidence of T2D by 58% compared with the placebo group(36). This suggests that an intensive lifestyle intervention, including diet, may be optimal in the prevention of T2D, particularly in those at high risk.

The strength of this study is the use of data from a long-term cohort, consisting of five measurements of diet intake, in a population-based sample that allowed trajectories of dietary patterns with adult health outcomes to be examined. The advantage of the trajectory analysis is that multiple observations of an exposure could be used to model the long-term development. This provides detailed information about the change of the exposure during the study period and its influence on health outcomes that could not be achieved by the traditional method of using change in the exposure between two time points. Our study has limitations. Causality could not be inferred given the observational design. However, we adjusted for many important confounders or mediators, including smoking, physical activity and BMI in both youth and adulthood. Method for diet measurement was changed from 48-hour dietary recall to FFQ in 2007 and 2011, which may have partly affected the change dietary patterns and trajectories. However, the food items included in the two different methods were largely comparable and were all grouped in the same way(22). Moreover, dietary pattern scores were standardised by survey years to make the values comparable between different methods and survey years. We were not able to examine the association of dietary pattern trajectories with T2D alone because of a small number of T2D patients (n=27). Therefore, studies with longer follow-up or larger

12

sample size are needed to examine this topic. Participants were lost to follow-up, but we have previously shown that these samples are representative of the original cohort(37,38). Moreover, inverse probability weighting was used to account for missingness and results remained largely similar, suggesting minimum impact of loss-to-follow-up.

In conclusion, trajectories of an increased or stably medium adherence to a 'red meat' dietary pattern from youth to adulthood were associated with higher risk of adult IFG, suggesting that interventions to prevent these unfavourable dietary pattern trajectories may substantially reduce the risk of IFG in adulthood and T2D later in life. As differences between these trajectories already existed in youth or started to become distinct in early adulthood, interventions should be implemented before this period to maximise the benefits. Acknowledgements: We thank all the volunteers and participants involved in the present study.

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A.J., T.L., K.P, S.P.R., T.R., N.H., M.K., T.L., J.S.A.V., and O.T. R. were responsible for data

collection and management. F.W. performed data analysis and drafted the manuscript, in consultation

with C.G.M., K.J.S., S.P.R., T.R. and M.J.B.. All authors revised manuscript content and approved

the final manuscript and had access to the data. J.S.A.V. contributed to the initial design of Young

Finns. O.T.R. leads Young Finns and contributed to obtaining funding and to the study design.

C.G.M. and O.T.R. are the guarantors of the study and accept full responsibility for the finished

article, had access to any data, and controlled the decision to publish.

Data Availability: All datasets generated during and/or analyzed during the current study are not

publicly available but are available from the corresponding author on reasonable request.

References

- 1. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, Colagiuri S, Guariguata L, Motala AA, Ogurtsova K, Shaw JE, Bright D, Williams R, Committee IDFDA. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9(th) edition. *Diabetes Res Clin Pract*. 2019;157:107843.
- 2. Tabak AG, Herder C, Rathmann W, Brunner EJ, Kivimaki M. Prediabetes: a high-risk state for diabetes development. *Lancet*. 2012;379(9833):2279-2290.
- Diabetes Prevention Program Research G, Knowler WC, Fowler SE, Hamman RF, Christophi CA, Hoffman HJ, Brenneman AT, Brown-Friday JO, Goldberg R, Venditti E, Nathan DM. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet*. 2009;374(9702):1677-1686.
- 4. Lindstrom J, Peltonen M, Eriksson JG, Ilanne-Parikka P, Aunola S, Keinanen-Kiukaanniemi S, Uusitupa M, Tuomilehto J, Finnish Diabetes Prevention S. Improved lifestyle and decreased diabetes risk over 13 years: long-term follow-up of the randomised Finnish Diabetes Prevention Study (DPS). *Diabetologia*. 2013;56(2):284-293.
- 5. Li G, Zhang P, Wang J, Gregg EW, Yang W, Gong Q, Li H, Li H, Jiang Y, An Y, Shuai Y, Zhang B, Zhang J, Thompson TJ, Gerzoff RB, Roglic G, Hu Y, Bennett PH. The long-term effect of lifestyle interventions to prevent diabetes in the China Da Qing Diabetes Prevention Study: a 20-year follow-up study. *Lancet*. 2008;371(9626):1783-1789.
- 6. Wu FT, Juonala M, Pitkanen N, Jula A, Lehtimaki T, Sabin MA, Pahkala K, Hutri-Kahonen N, Kahonen M, Laitinen T, Viikari JSA, Magnussen CG, Raitakari OT. Both youth and long-term vitamin D status is associated with risk of type 2 diabetes mellitus in adulthood: a cohort study. *Ann Med.* 2018;50(1):74-82.
- Jaaskelainen P, Magnussen CG, Pahkala K, Mikkila V, Kahonen M, Sabin MA, Fogelholm M, Hutri-Kahonen N, Taittonen L, Telama R, Laitinen T, Jokinen E, Lehtimaki T, Viikari JSA, Raitakari OT, Juonala M. Childhood Nutrition in Predicting Metabolic Syndrome in Adults. *Diabetes Care*. 2012;35(9):1937-1943.

- 8. Pitkanen N, Juonala M, Ronnemaa T, Sabin MA, Hutri-Kahonen N, Kahonen M, Lehtimaki T, Viikari JSA, Raitakari OT. Role of Conventional Childhood Risk Factors Versus Genetic Risk in the Development of Type 2 Diabetes and Impaired Fasting Glucose in Adulthood: The Cardiovascular Risk in Young Finns Study. *Diabetes Care*. 2016;39(8):1393-1399.
- 9. American Diabetes A. 3. Prevention or Delay of Type 2 Diabetes: Standards of Medical Care in Diabetes-2020. *Diabetes Care*. 2020;43(Suppl 1):S32-S36.
- 10. Newby P, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. *Nutr Rev.* 2004;62(5):177-203.
- 11. Jannasch F, Kroger J, Schulze MB. Dietary Patterns and Type 2 Diabetes: A Systematic Literature Review and Meta-Analysis of Prospective Studies. *J Nutr.* 2017;147(6):1174-1182.
- 12. Malik VS, Fung TT, van Dam RM, Rimm EB, Rosner B, Hu FB. Dietary patterns during adolescence and risk of type 2 diabetes in middle-aged women. *Diabetes Care*. 2012;35(1):12-18.
- Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Kahn R, Kitzmiller J, Knowler WC, Lebovitz H, Lernmark A, Nathan D, Palmer J, Rizza R, Saudek C, Shaw J, Steffes M, Stern M, Tuomilehto J, Zimmet P, Expert Committee on the D, Classification of Diabetes M. Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care*. 2003;26(11):3160-3167.
- 14. Pitkänen N, Juonala M, Rönnemaa T, Sabin MA, Hutri-Kahonen N, Kahonen M, Lehtimaki T, Viikari JS, Raitakari OT. Role of Conventional Childhood Risk Factors Versus Genetic Risk in the Development of Type 2 Diabetes and Impaired Fasting Glucose in Adulthood: The Cardiovascular Risk in Young Finns Study. *Diabetes Care*. 2016;39(8):1393-1399.
- 15. Rasanen L, Laitinen S, Stirkkinen R, Kimppa S, Viikari J, Uhari M, Pesonen E, Salo M, Akerblom HK. Composition of the diet of young Finns in 1986. *Ann Med.* 1991;23(1):73-80.
- 16. Paalanen L, Mannisto S, Virtanen MJ, Knekt P, Rasanen L, Montonen J, Pietinen P. Validity of a food frequency questionnaire varied by age and body mass index. *J Clin Epidemiol*. 2006;59(9):994-1001.
- 17. Mannisto S, Virtanen M, Mikkonen T, Pietinen P. Reproducibility and validity of a food frequency questionnaire in a case-control study on breast cancer. *J Clin Epidemiol*. 1996;49(4):401-409.
- 18. Reinivuo H, Hirvonen T, Ovaskainen ML, Korhonen T, Valsta LM. Dietary survey methodology of FINDIET 2007 with a risk assessment perspective. *Public Health Nutr*. 2010;13(6A):915-919.
- 19. Juonala M, Voipio A, Pahkala K, Viikari JS, Mikkila V, Kahonen M, Hutri-Kahonen N, Jula A, Burgner D, Sabin MA, Marniemi J, Loo BM, Laitinen T, Jokinen E, Taittonen L, Magnussen CG, Raitakari OT. Childhood 25-OH vitamin D levels and carotid intima-media thickness in adulthood: the cardiovascular risk in young Finns study. *J Clin Endocrinol Metab*. 2015;100(4):1469-1476.
- 20. Telama R, Viikari J, Välimäki I, Siren-Tiusanen H, Akerblom HK, Uhari M, Dahl M, Pesonen E, Lahde PL, Pietikainen M, et al. Atherosclerosis precursors in Finnish children and adolescents. X. Leisure-time physical activity. *Acta Paediatr Scand Suppl.* 1985;318:169-180.
- 21. Telama R, Yang X, Leskinen E, Kankaanpaa A, Hirvensalo M, Tammelin T, Viikari JS, Raitakari OT. Tracking of physical activity from early childhood through youth into adulthood. *Med Sci Sports Exerc*. 2014;46(5):955-962.
- 22. Mikkila V, Rasanen L, Raitakari OT, Pietinen P, Viikari J. Consistent dietary patterns identified from childhood to adulthood: the cardiovascular risk in Young Finns Study. *Br J Nutr*. 2005;93(6):923-931.
- 23. Wu F, Wills K, Laslett LL, Oldenburg B, Jones G, Winzenberg T. Associations of dietary patterns with bone mass, muscle strength and balance in a cohort of Australian middle-aged women. *Br J Nutr*. 2017;118(8):598-606.
- 24. Nagin DS, Odgers CL. Group-based trajectory modeling in clinical research. *Annu Rev Clin Psychol.* 2010;6:109-138.
- 25. Wu F, Pahkala K, Juonala M, Rovio S, Sabin M, Rönnemaa T, Buscot M, Smith K, Männistö S, Jula A, Lehtimäki T, Hutri-Kähönen N, Kähönen M, Laitinen T, Viikari J, Raitakari OT, Magnussen CG. Data from: Dietary pattern trajectories from youth to adulthood and adult risk

of impaired fasting glucose: a 31-year cohort study. Figshare 2020. Deposited 13 June 2020. https://doi.org/10.6084/m9.figshare.12476483.v2.

- 26. Wu F, Callisaya M, Wills K, Laslett LL, Jones G, Winzenberg T. Both Baseline and Change in Lower Limb Muscle Strength in Younger Women Are Independent Predictors of Balance in Middle Age: A 12-Year Population-Based Prospective Study. J Bone Miner Res. 2017;32(6):1201-1208.
- 27. McEvoy CT, Cardwell CR, Woodside JV, Young IS, Hunter SJ, McKinley MC. A posteriori dietary patterns are related to risk of type 2 diabetes: findings from a systematic review and meta-analysis. *J Acad Nutr Diet*. 2014;114(11):1759-1775 e1754.
- 28. Yang X, Li YQ, Wang CJ, Mao ZX, Zhou W, Zhang LL, Fan MY, Cui SY, Li LL. Meat and fish intake and type 2 diabetes: Dose-response meta-analysis of prospective cohort studies. *Diabetes & Metabolism.* 2020;46(5):345-352.
- 29. Neuenschwander M, Ballon A, Weber KS, Norat T, Aune D, Schwingshackl L, Schlesinger S. Role of diet in type 2 diabetes incidence: umbrella review of meta-analyses of prospective observational studies. *BMJ*. 2019;366:12368.
- Talaei M, Wang YL, Yuan JM, Pan A, Koh WP. Meat, Dietary Heme Iron, and Risk of Type
 2 Diabetes Mellitus: The Singapore Chinese Health Study. *Am J Epidemiol*. 2017;186(7):824-833.
- 31. Reynolds A, Mann J, Cummings J, Winter N, Mete E, Te Morenga L. Carbohydrate quality and human health: a series of systematic reviews and meta-analyses. *Lancet*. 2019;393(10170):434-445.
- 32. Della Pepa G, Vetrani C, Vitale M, Riccardi G. Wholegrain Intake and Risk of Type 2 Diabetes: Evidence from Epidemiological and Intervention Studies. *Nutrients*. 2018;10(9).
- 33. Hu Y, Ding M, Sampson L, Willett WC, Manson JE, Wang M, Rosner B, Hu FB, Sun Q. Intake of whole grain foods and risk of type 2 diabetes: results from three prospective cohort studies. *BMJ*. 2020;370:m2206.
- 34. Batis C, Mendez MA, Sotres-Alvarez D, Gordon-Larsen P, Popkin B. Dietary pattern trajectories during 15 years of follow-up and HbA1c, insulin resistance and diabetes prevalence among Chinese adults. *J Epidemiol Community Health*. 2014;68(8):773-779.
- 35. Tinker LF, Bonds DE, Margolis KL, Manson JE, Howard BV, Larson J, Perri MG, Beresford SA, Robinson JG, Rodriguez B, Safford MM, Wenger NK, Stevens VJ, Parker LM, Women's Health I. Low-fat dietary pattern and risk of treated diabetes mellitus in postmenopausal women: the Women's Health Initiative randomized controlled dietary modification trial. *Arch Intern Med.* 2008;168(14):1500-1511.
- 36. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM, Diabetes Prevention Program Research G. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.* 2002;346(6):393-403.
- 37. Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, Srinivasan SR, Daniels SR, Davis PH, Chen W, Sun C, Cheung M, Viikari JS, Dwyer T, Raitakari OT. Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N Engl J Med.* 2011;365(20):1876-1885.
- 38. Mikkila V, Rasanen L, Laaksonen MM, Juonala M, Viikari J, Pietinen P, Raitakari OT. Longterm dietary patterns and carotid artery intima media thickness: the Cardiovascular Risk in Young Finns Study. *Br J Nutr*. 2009;102(10):1507-1512.

Figure legend

Figure 1. Flowchart of study participants. IFG, impaired fasting glucose; T2D, type 2 diabetes. # from a random sample of 50% of the participants in 1980. * all participants from the orginal cohort were invited. [§] participants who had less than three diet assessments available from baseline, 1986, 2001, 2007 and 2011 follow-ups.

Figure 2. The radar chart for the evoluation of the 'Traditional Finnish' dietary pattern (1980, 1986 and 2001) to the 'Traditional Finnish and high-carbohydrate' dietary pattern (2007 and 2011) from youth (aged 3 to 18 years) to adulthood (aged 34 to 49 years). The factor loading ranges from -1.0 (centre) to 1.0 (the largest circle), with a difference of 0.2 between the two adjacent circles.

Figure 3. The trajectories from youth to adulthood of six dietary patterns derived by diet in 1980 (A-C) and 2011 (D-F). A: Traditional Finnish; B: High-carbohydrate; C: Vegetables and dairy products; D: Traditional Finnish and high-carbohydrate; E: Red meat; F: Healthy. Values of the y-axis are survey year-standardised z-scores of each pattern. % in brackets indicate the percentage of participants allocated to each trajectory group. L=low, M=medium, H=high, indicating starting level of each trajectory.

| | Trajectories of Red meat pattern | | |
|---|------------------------------------|-------------|--|
| | L-stable M-stable/M-large increase | | |
| | (n=649) | (n=358) | |
| Youth | | | |
| Age (year) | 9.9 (4.9) | 11.4 (5.1) | |
| Females, (%) | 65 | 32 | |
| BMI (kg/m ²) | 17.3 (2.9) | 18.3 (3.1) | |
| 25OHD (nmol/L) | 53 (15) | 51 (16) | |
| Physical activity index (z score) | -0.05 (0.94) | 0.14 (1.02) | |
| Parental history of diabetes, n (%) | 17 (2.6) | 5 (1.4) | |
| Smokers, n (%) | 108 (16.6) | 85 (23.7) | |
| Parental years of education | 10.5 (3.4) | 9.1 (2.7) | |
| Adulthood ^a | | | |
| Age (year) | 40.9 (4.9) | 42.4 (5.1) | |
| BMI (kg/m^2) | 25.6 (4.9) | 26.9 (4.6) | |
| Smokers, n (%) | 94 (15) | 100 (28) | |
| Education status, n (%) | | | |
| Grammar school | 82 (13) | 68 (20) | |
| College or vocational school | 274 (45) | 176 (52) | |
| University degree | 258 (42) | 95 (28) | |
| Long-term dietary fibre intake ^b | 20.08 (7.0) | 21.2 (6.6) | |
| Fasting glucose (mmol/L) | 5.26 (0.88) | 5.47 (0.69) | |
| Glucose metabolism categories, n (%) | | | |
| NFG | 538 (82.9) | 240 (67.0) | |
| IFG | 98 (15.1) | 104 (29.1) | |
| T2D | 13 (2.0) | 14 (3.9) | |

Table 1 Participants' characteristics in youth (1980) and adulthood in the YFS (n=1,007)

L=low, M=medium, indicating starting point of each trajectory.

Data are mean (standard deviation) unless otherwise stated.

Abbreviations: NFG, normal fasting glucose; IFG, impaired fasting glucose (cut-off 5.6 mmol/L); T2D, type 2 diabetes; BMI, body mass index; 25OHD, 25-hydroxyvitamin D.

^a all variables used data from the latest available values in adulthood (from 2001, 2007 or 2011). There were statistical differences between groups for all variables except for parental history of diabetes, p<0.05.

^b calculated as the mean of dietary fibre intake in 1986, 2001, 2007 and 2011.

| | | | IFG (vs | s NFG) |
|--|---|---------------------------------|--|--|
| | | | Model 1 ^c | Model 2 ^d |
| Dietary patterns | Trajectory categories | n/N (%) ^e | RR (95% CI) | RR (95% CI) |
| Traditional Finnish ^a | L-slight decrease | 79/429 (18.4) | Reference | Reference |
| | M-slight increase | 100/477 (20.1) | 1.14 (0.87 to 1.48) | 1.00 (0.76 to 1.31) |
| | H-stable | 23/74 (31.1) | 1.69 (1.14 to 2.50) | 1.03 (0.67 to 1.58) |
| High-carbohydrate ^a | M-slight decrease M-stable/M-large increase | 139/633 (22.0) 63/347 (18.2) | Reference 0.83 (0.63 to 1.08) | Reference 0.80 (0.62 to 1.04) |
| Vegetables and dairy | L-moderate decrease | 44/199 (22.1) | Reference | Reference |
| products " | M-stable M-moderate increase | 146/726 (20.1) 12/55 (21.8) | 0.82 (0.61 to 1.11) 0.96 (0.51 to 1.81) | 1.01 (0.57 to 1.36) 1.00 (0.57 to 1.74) |
| Traditional Finnish and high carbohydrate ^b | M-stable/M-large decrease | 175/807 (21.7) | Reference | Reference |
| lingii-carbonydrate | M-slight increase | 27/173 (15.6) | 0.72 (0.50 to 1.04) | 0.71 (0.50 to 1.01) |
| Red meat ^b | L-stable M-stable/M-large increase | 98/636 (15.4) 104/344 (30.2) | Reference 1.96 (1.54 to 2.50) | Reference 1.46 (1.12 to 1.90) |
| Healthy ^b | L-stable M-stable/M-large increase | 151/677 (22.3) 51/303 (16.8) | Reference 0.75 (0.57 to 1.01) | Reference 0.91 (0.69 to 1.21) |

Table 2 Associations of dietary pattern trajectories from youth to adulthood with adult risk of IFG (n=980)

L=low, M=medium, H=high, indicating starting level of each trajectory.

Abbreviations: RR, relative risk; CI, confidence interval; NFG, normal fasting glucose; IFG, impaired fasting glucose (cut-off 5.6 mmol/L).

Bold denotes statistical significance, p<0.05.

^a dietary pattern scores used for trajectory analysis were calculated using factor loadings from dietary patterns in 1980.

^b dietary patterns scores used for trajectory analysis were calculated using factor loadings from dietary patterns in 2011.

^c unadjusted;

^d adjusted for age, sex, body mass index and total energy intake, serum 25OHD levels, parental history of diabetes, physical activity, smoking, and socioeconomic status (parental education years) at baseline and body mass index in adulthood.

^e cases of IFG/total number of participants in that category.





Figure 3



L=low, M=medium, H=high, indicating starting level of each trajectory.

Page 2- 6, Supplemental tables for factor loadings of dietary patterns identified from each survey years.

Page 7-8, Model selection and Supplemental tables for group-based trajectory modelling (GBTM).

Page 10, Figure legend for Supplemental Figures 1-2.

| Food groups | Foods included in the group | Traditional | High- | Vegetables |
|--------------------|--------------------------------------|-------------|--------------|------------|
| | | FIIIIISII | carbonydrate | products |
| Rve | Rye bread, rye porridge | 0.68 | -0.01 | 0.14 |
| Wheat | Wheat bread, pasta | 0.14 | 0.62 | -0.02 |
| Other grain | Cereals other than rye and wheat, | | | |
| products | breakfast cereals, biscuits, starch, | | | |
| • | rice | -0.21 | 0.28 | -0.06 |
| Legumes and nuts | Peas, beans, other legumes, nuts, | | | |
| | seeds, soya products | -0.03 | 0.10 | 0.04 |
| Potatoes | Potatoes, potato products | 0.54 | 0.27 | 0.01 |
| Root vegetables | Root vegetables | 0.12 | -0.13 | 0.33 |
| Other vegetables | Leaf vegetables, onions, | | | |
| | cabbages, tomatoes, cucumbers, | | | |
| | canned vegetables, mushrooms | -0.01 | 0.18 | 0.54 |
| Fruit and berries | Fresh fruits, canned fruits, | | | |
| | berries, fruit and berry juices | -0.32 | 0.21 | 0.20 |
| Margarine and oils | Soft margarine, low-fat spreads, | | | |
| | oil | -0.02 | 0.68 | 0.15 |
| Butter | Butter, butter-oil spreads, lard | 0.71 | -0.02 | -0.09 |
| Milk | Milk | 0.36 | 0.40 | -0.40 |
| Cheese | Cheese | 0.19 | 0.17 | 0.53 |
| Other dairy | Cream, sour milk products, | | | |
| products | yoghurt ice cream | -0.13 | -0.10 | 0.53 |
| Pork | Pork | 0.05 | 0.11 | 0.06 |
| Other meat | Beef, lamb, game, poultry, meat | | | |
| | products | 0.14 | 0.36 | 0.23 |
| Sausages | Sausages, frankfurters | 0.35 | 0.16 | -0.03 |
| Offal | Liver, kidney, other offal | 0.03 | 0.09 | -0.01 |
| Fish | Fish, shellfish, fish products | 0.02 | 0.13 | 0.03 |
| Eggs | Eggs | -0.01 | 0.45 | 0.08 |
| Coffee | Coffee | 0.46 | 0.09 | 0.02 |
| Tea | Tea | 0.10 | 0.12 | 0.26 |
| Alcoholic | Alcoholic beverages | | | |
| beverages | | 0.16 | 0.01 | 0.33 |
| Sugar | Sugar, syrup, sweets, chocolate | -0.02 | 0.51 | -0.16 |
| Variance (%) | | 8.7 | 8.7 | 6.5 |

Supplemental Table 1 Rotated factor loadings for the three dietary patterns identified from exploratory factor analysis based on diet intake in 1980.

| Food groups | Traditional | High-carbohydrate | Vegetables and red |
|----------------------|-------------|-------------------|--------------------|
| | Finnish | | meat |
| Rye | 0.49 | 0.05 | 0.16 |
| Wheat | 0.14 | 0.66 | -0.04 |
| Other grain products | -0.03 | -0.04 | -0.04 |
| Legumes and nuts | -0.12 | 0.16 | -0.06 |
| Potatoes | 0.30 | -0.08 | 0.69 |
| Root vegetables | -0.15 | -0.09 | 0.63 |
| Other vegetables | -0.16 | 0.27 | 0.37 |
| Fruit and berries | -0.25 | 0.18 | 0.08 |
| Margarine and oils | -0.29 | 0.47 | 0.20 |
| Butter | 0.77 | 0.15 | 0.02 |
| Milk | 0.60 | -0.19 | 0.08 |
| Cheese | 0.04 | 0.44 | 0.14 |
| Other dairy products | 0.00 | -0.08 | 0.00 |
| Pork | -0.02 | 0.22 | 0.60 |
| Other meat | 0.10 | -0.13 | 0.26 |
| Sausages | 0.26 | 0.24 | -0.03 |
| Offal | 0.07 | 0.20 | -0.01 |
| Fish | -0.01 | -0.02 | 0.03 |
| Eggs | 0.19 | 0.47 | -0.18 |
| Coffee | 0.34 | 0.10 | 0.03 |
| Tea | -0.10 | 0.36 | 0.04 |
| Alcoholic beverages | -0.07 | 0.01 | 0.10 |
| Sugar | 0.13 | 0.19 | 0.00 |
| Variance (%) | 7.8 | 7.1 | 6.9 |

Supplemental Table 2 Rotated factor loadings for the three dietary patterns identified from exploratory factor analysis based on diet intake in 1986.

| Food groups | Traditional | High- | Vegetables and |
|----------------------|-------------|--------------|----------------|
| | Finnish | carbohydrate | dairy products |
| Rye | 0.53 | 0.29 | 0.25 |
| Wheat | -0.17 | 0.68 | -0.13 |
| Other grain products | 0.23 | 0.78 | 0.12 |
| Legumes and nuts | -0.41 | 0.18 | 0.02 |
| Potatoes | 0.30 | 0.16 | -0.24 |
| Root vegetables | 0.13 | 0.03 | 0.47 |
| Other vegetables | -0.46 | 0.32 | 0.17 |
| Fruit and berries | -0.10 | 0.13 | 0.32 |
| Margarine and oils | 0.33 | 0.35 | -0.04 |
| Butter | -0.05 | 0.51 | -0.11 |
| Milk | 0.48 | 0.25 | -0.22 |
| Cheese | -0.21 | 0.33 | 0.07 |
| Other dairy products | -0.07 | 0.04 | 0.31 |
| Pork | 0.20 | 0.27 | -0.11 |
| Other meat | -0.09 | 0.16 | -0.18 |
| Sausages | 0.32 | 0.27 | -0.31 |
| Offal | -0.07 | 0.03 | -0.06 |
| Fish | -0.22 | 0.12 | 0.05 |
| Eggs | -0.10 | 0.34 | -0.08 |
| Coffee | 0.16 | 0.09 | -0.56 |
| Теа | 0.00 | 0.07 | 0.66 |
| Alcoholic beverages | -0.33 | 0.02 | -0.36 |
| Sugar | -0.08 | 0.32 | 0.16 |
| Variance (%) | 6.9 | 10.1 | 7.5 |

Supplemental Table 3 Rotated factor loadings for the three dietary patterns identified from exploratory factor analysis based on diet intake in 2001.

| Food groups | Traditional Finnish + | Red | Healthy |
|----------------------|-----------------------|-------|---------|
| | High-carbohydrate | meat | |
| Rye | 0.37 | -0.16 | 0.13 |
| Wheat | 0.84 | 0.04 | -0.02 |
| Other grain products | 0.84 | 0.06 | 0.22 |
| Legumes and nuts | 0.00 | 0.09 | 0.63 |
| Potatoes | 0.54 | 0.34 | 0.09 |
| Root vegetables | 0.11 | -0.01 | 0.74 |
| Other vegetables | 0.10 | 0.02 | 0.75 |
| Fruit and berries | 0.29 | -0.20 | 0.50 |
| Margarine and oils | 0.52 | 0.17 | 0.13 |
| Butter | 0.68 | 0.11 | 0.08 |
| Milk | 0.34 | 0.14 | -0.17 |
| Cheese | 0.28 | -0.03 | 0.15 |
| Other dairy products | 0.29 | -0.21 | 0.22 |
| Pork | 0.46 | 0.57 | 0.22 |
| Other meat | 0.39 | 0.39 | 0.26 |
| Sausages | 0.41 | 0.47 | -0.14 |
| Offal | 0.13 | 0.23 | 0.13 |
| Fish | 0.17 | 0.33 | 0.46 |
| Eggs | 0.37 | 0.37 | 0.21 |
| Coffee | 0.09 | 0.40 | -0.21 |
| Tea | 0.09 | -0.44 | 0.25 |
| Alcoholic beverages | -0.07 | 0.56 | -0.03 |
| Sugar | 0.56 | -0.11 | -0.03 |
| Variance (%) | 17.4 | 8.6 | 10.7 |

Supplemental Table 4 Rotated factor loadings for the three dietary patterns identified from exploratory factor analysis based on diet intake in 2007.

| Food groups | Traditional Finnish + | Red meat | Healthy |
|----------------------|-----------------------|----------|---------|
| | High-carbohydrate | | |
| Rye | 0.51 | -0.13 | 0.07 |
| Wheat | 0.78 | 0.10 | -0.06 |
| Other grain products | 0.86 | 0.09 | 0.13 |
| Legumes and nuts | -0.01 | 0.09 | 0.72 |
| Potatoes | 0.54 | 0.38 | 0.00 |
| Root vegetables | 0.11 | 0.03 | 0.73 |
| Other vegetables | -0.00 | 0.04 | 0.75 |
| Fruit and berries | 0.33 | -0.13 | 0.39 |
| Margarine and oils | 0.42 | 0.15 | 0.18 |
| Butter | 0.53 | 0.19 | 0.04 |
| Milk | 0.35 | 0.21 | -0.12 |
| Cheese | 0.18 | 0.03 | 0.35 |
| Other dairy products | 0.28 | -0.19 | 0.26 |
| Pork | 0.31 | 0.66 | 0.09 |
| Other meat | 0.25 | 0.52 | 0.26 |
| Sausages | 0.41 | 0.49 | -0.19 |
| Offal | 0.07 | 0.46 | 0.08 |
| Fish | 0.03 | 0.31 | 0.42 |
| Eggs | -0.05 | 0.54 | 0.22 |
| Coffee | 0.18 | 0.29 | -0.19 |
| Tea | 0.02 | -0.28 | 0.32 |
| Alcoholic beverages | -0.08 | 0.46 | -0.12 |
| Sugar | 0.42 | -0.14 | 0.01 |
| Variance (%) | 14.1 | 9.9 | 10.9 |

Supplemental Table 5 Rotated factor loadings for the three dietary patterns identified from exploratory factor analysis based on diet intake in 2011.

Model selection for GBTM.

Dietary pattern scores for each participant from all surveys were used as dependent variables and the survey years as independent variables. To be included in the trajectories, individuals were required to have dietary scores from at least three survey years (99% of participants had dietary scores at both baseline and at least one of the adulthood follow-ups). For trajectory modelling, dietary pattern scores were modelled with the normal distribution. The model selection was based on the Bayesian information criterion (a higher value indicating better fit), but was complemented by the following diagnostic criteria: a) an average posterior probability (AvePP) value >0.7 for each group; (b) the odds of correct classification >5 for all groups; (c) reasonably close correspondence between estimated group probabilities and the proportion of sample assigned to the group; (d) reasonably narrow confidence intervals; and (e) adequate sample numbers in each group (n>10)(1). Models with different number of groups (2-4) and trajectory shapes (zero-order, linear and quadratic) were tested until the best fitting model was established. No extra groups were tested as one or more of the groups had a very small proportion of observations or no meaningful category was additionally identified in the 4-group model.

An example of model selection for 'Red meat' pattern is shown in Supplemental Table 6 (A-C). Among the models with the largest BIC, those with better OCC were selected for the 'Traditional Finnish' (2 1 0), 'High-carbohydrate' (1 2 1), and 'Red meat' (1 1 2) patterns. The models with the biggest BIC were selected for the 'vegetables and dairy products' (1 1 1), 'Traditional Finnish and high-carbohydrate' (1 1 1), and 'Healthy' pattern (0 2 2) patterns. All trajectory groups had an AvePP value above 0.7 and reasonably narrow confidence intervals, with moderate to very close correspondence between each group's estimated probability and the proportion of study participants assigned to it (Figure 3). The OCCs were above 5 for all trajectories, except for the group with the largest proportion of participants for each pattern (2.2 to 4.0); however, their confidence intervals are the narrowest without overlapping with other trajectories, suggesting an overall good classification.

| Number of groups | Trajectory shapes ¹ | BIC^2 (N = 1256) | BIC^{3} (N = 5014) |
|------------------|--------------------------------|--------------------|----------------------|
| 2 | 0.0 | -6996.82 | -6999.59 |
| 2 | 01 | -6974.32 | -6977.78 |
| 2 | 02 | -6976.53 | -6980.68 |
| 2 | 11 | -6977.83 | -6981.98 |
| 2 | 12 | -6979.95 | -6984.80 |
| 2 | 22 | -6981.99 | -6987.53 |
| 3 | 000 | -6945.74 | -6949.89 |
| 3 | 011 | -6895.30 | -6900.84 |
| 3 | 012 | -6895.97 | -6902.20 |
| 3 | 022 | -6898.05 | -6904.98 |
| 3 | 111 | -6907.87 | -6914.10 |
| 3 | 112 | -6896.69 | -6903.61 |
| 3 | 121 | -6898.51 | -6905.43 |
| 3 | 122 | -6898.00 | -6905.62 |
| 3 | 210 | -6929.41 | -6935.64 |
| 3 | 211 | -6909.33 | -6916.25 |
| 3 | 212 | -6898.00 | -6905.62 |
| 3 | 221 | -6899.09 | -6906.70 |
| 3 | 222 | -6899.55 | -6907.85 |
| 4 | 0 0 0 0* | -6932.28 | -6937.82 |

Supplemental Table 6A. BIC for GBTM of the **'Red meat'** pattern according to number of groups and trajectory shapes (based on factor loadings in Supplemental Table 5).

1Trajectory shapes; 0 = zero-order; 1 = linear; 2 = quadratic.

2BIC = Bayesian information criterion (for the total number of participants)

3BIC = Bayesian information criterion (for the total number of observations)

*One or more groups had a very small proportion of participants or no meaningful category was additionally identified compared with the 3-group model.

Supplemental Table 6B. Average posterior probability (AvePP) value and odds of correct classification for **'Red meat'** pattern GBTM groups

| Trajectory groups of the 'Red meat' pattern | L-stable | M-stable | M-large increase |
|---|----------|----------|------------------|
| Average posterior probability value | 0.84 | 0.78 | 0.89 |
| Odds of correct classification | 3.3 | 6.3 | 576.4 |

L=low, M=medium, indicating starting point of the trajectory.

Supplemental Table 6C. **'Red meat'** pattern trajectory groups' estimated probability and the proportion of Study members classified to each group according to the maximum posterior probability assignment rule

| Group | Estimated group probability | Proportion assigned to group according to the maximum posterior probability assignment rule |
|------------------|-----------------------------|---|
| L-stable | 61.9 | 65.0 |
| M-stable | 36.8 | 33.5 |
| M-large increase | 1.4 | 1.4 |

L=low, M=medium, indicating starting point of the trajectory.

Reference 1. Nagin DS, Odgers CL: Group-based trajectory modeling in clinical research. Annu Rev Clin Psychol 2010;6:109-138

Supplemental Figure 1. The radar chart for the evolution of the 'High-carbohydrate' dietary pattern (1980, 1986 and 2001) to the 'Red meat' dietary pattern (2007 and 2011) from youth (aged 3 to 18 years) to adulthood (aged 34 to 49 years). The jagged lines (distance from the centre) indicate factor loadings for each food groups of each dietary patterns in 1980, 1986, 2001, 2007 and 2011. The factor loading ranges from -1.0 (centre) to 1.0 (the largest circle), with a difference of 0.2 between the two adjacent circles.

Supplemental Figure 2. The radar chart for the evolution of the 'Vegetables and dairy products' dietary pattern (1980, 1986 and 2001) to the 'Healthy' dietary pattern (2007 and 2011) from youth (aged 3 to 18 years) to adulthood (aged 34 to 49 years). The jagged lines (distance from the centre) indicate factor loadings for each food groups of each dietary patterns in 1980, 1986, 2001, 2007 and 2011. The factor loading ranges from -1.0 (centre) to 1.0 (the largest circle), with a difference of 0.2 between the two adjacent circles.

Supplemental Figure 1



Supplemental Figure 2

