Ultra-processed food consumption and adiposity trajectories from childhood: a prospective analysis of the ALSPAC birth cohort

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KEY POINTS

Question: Is consumption of ultra-processed foods in childhood associated with worse adiposity trajectories tracing into early adulthood?

Findings: In this prospective cohort study of 9025 British children, growth trajectories of body mass index, fat mass index, weight and waist circumference from ages 7 to 24 years were significantly greater among children with the highest (vs lowest) quintile of ultra-processed food consumption.

Meaning: Radical and effective public health actions that reduce children's exposure to and consumption of ultra-processed foods, and remove barriers to accessing minimally processed foods are urgently needed to counteract the growing burden of obesity in England and globally.

ABSTRACT

Importance

Growing evidence have reported associations between higher ultra-processed food consumption and elevated risks of obesity, non-communicable diseases, and mortality in adults. However, its associations with long-term adiposity trajectories have never been investigated in children.

Objective

To assess longitudinal associations between ultra-processed food consumption and adiposity trajectories from childhood to early adulthood.

Design

Prospective birth cohort study of children participated in the Avon Longitudinal Study of Parents and Children (ALSPAC). Children were followed up from ages 7 to 24 years (1998-2017). Data analysis was conducted between March 2020 and January 2021.

Setting

Population-based in Avon County, south-west England.

Participants

Children with baseline dietary intakes collected using 3-day food diaries and repeated measures of adiposity during the study period.

Exposure

Consumption of ultra-processed foods (applying the NOVA food classification system) was computed as a percentage of its weight contribution (gram per day) in the total diet for each participant and categorized into quintiles.

Main outcomes and measures

Repeated recordings of objectively assessed anthropometrics (body mass index, weight, waist circumference) and dual-energy X-ray absorptiometry measurements (fat and lean mass index,

body fat percentage). Associations were evaluated using linear growth curve models and adjusted for study covariates.

Results

A total of 9025 children (4,481 [49.6%] female) were followed up over a median (IQR) of 10.2 (5.2-16.4) years. Mean (SD) ultra-processed food consumption at baseline from the lowest to highest consumption quintiles was 23.2% (5.0%), 34.7% (2.5%), 43.4% (2.5%), 52.7% (2.8%) and 67.8% (8.1%). Trajectories of body mass index, fat mass index, weight and waist circumference increased significantly by an additional 0.06 (95% CI, 0.04-0.08) kg/m^2, 0.03 (95% CI, 0.01-0.05) kg/m^2, 0.20 (95% CI, 0.11-0.28) kg and 0.17 (95% CI, 0.11-0.22) cm per year among those in the highest quintile of ultra-processed food consumption compared with their lowest quintile counterpart.

Conclusions and relevance

These findings provide important and novel evidence that higher ultra-processed food consumption is associated with greater increases in adiposity from childhood to early adulthood. Robust public health measures that promote minimally processed foods and discourage ultra-processed food consumption among children are urgently needed to reduce obesity in England and globally.

INTRODUCTION

Growing evidence on the potential harmful effects of ultra-processed food (UPF) consumption on health has directed attention towards the public health significance of industrial food processing.[1-8] UPFs, as defined by the NOVA food classification system, are industrial formulations of ingredients that undergo a series of physical, chemical and biological processes.[9] They typically lack intact healthy food components and include various additives.[9] UPFs tend to be more energy-dense and nutritionally poorer (high in free sugar, salt and saturated fats but low in protein, dietary fiber and micronutrients) compared with less processed alternatives, and are designed to be cheap, palatable, durable, convenient and appealing.[9] These products are aggressively marketed by the food industry to promote purchasing and shape dietary preferences, and children are a key target market.[9,10]

The rapid expansion of global and industrialized food systems has gradually displaced traditional dietary patterns based on fresh and minimally-processed foods, in favor of ready-to-consume UPFs.[9,10] Currently, UPFs represent 65.4% and 66.2% of daily calorie intake among UK and US school-aged children, respectively.[11,12] The growing consumption worldwide, including in low- and middle-income countries, has mirrored a parallel rise in the prevalence of childhood and adult obesity globally,[9,10,13] suggesting that UPF consumption may be a key underlying driver of the obesity epidemic and diet-related non-communicable diseases.[9,10,14,15]

A recent clinical trial has shown that UPF consumption leads to excess calorie intake and weight gain in adults,[1] and cohort studies have reported associations between higher consumption and elevated risks of obesity,[2,3] type 2 diabetes,[4,5] cardiovascular disease,[6] cancer,[7] and mortality in adults.[8] Evidence for its associations with adiposity in children and adolescents remains scarce, with only few previous small-scale studies available.[16-20] This study investigates prospective associations between UPF consumption and objectively assessed adiposity measurements from childhood to early adulthood in a large cohort of British children.

METHODS

Data source

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a prospective birth cohort study that initially enrolled 14541 pregnant women residents in Avon, England with an expected date of delivery between April 1991 and December 1992.[21,22] Further enrolments post-1998 resulted in a sample of 14888 children from singleton/twin pregnancies.[23] ALSPAC participants provided written informed consent, and ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. ALSPAC's study website contains details of all data that is available through a fully searchable data dictionary and variable search tool: http://www.bristol.ac.uk/alspac/researchers/our-data/. Since 2014, study data were collected and managed using REDCap electronic data capture tools hosted at the University of Bristol.[24,25]

Outcome measures

Children were invited to a total of 10 clinic assessments, almost annually between ages 7-17 years and then at 24 years (eTable 1 in the Supplement). Adiposity outcomes were measured following standardized procedures.[26] Primary outcomes include body mass index (BMI, kg/m^2), fat mass index (FMI, kg/m^2), lean mass index (LMI, kg/m^2), and total body fat (%). Secondary outcomes are BMI z-score, weight (kg), waist circumference (cm), fat mass (FM, kg), and lean mass (LM, kg). Height was measured using the Harpenden Stadiometer; weight using the Tanita Body Fat Analyzer; and waist circumference using a tape at the minimum circumference of the abdomen between iliac crests and lowest ribs.[26] Total body FM and LM were assessed using a Lunar Prodigy Dual-energy X-ray Absorptiometry (DXA) scanner.[26] BMI was computed as weight divided by height in meters squared. FMI and LMI were calculated using DXA-measured FM/LM and divided by height in meters squared, respectively. Total body fat was computed as the percentage of FM over body mass. Age-sex standardized BMI z-score was calculated for ages 7-17 years as the British 1990 Growth Reference is only available up to 23 years of age.[27]

Dietary exposure & degree of industrial food processing

Three-day food diary was sent to parents prior to child's clinic assessment for parent completion at 7 years and child completion at 10 and 13 years.[26] Respondents were instructed to record all food and beverage items the child consumed over two weekdays and one weekend day (not necessarily consecutive).[26] Dietary data were reviewed by a nutritionist and intakes were coded using the DIDO (Diet In, Data Out) computer program and were linked to the fifth edition of McCance and Widdowson's British food composition tables.[26,28]

We applied the NOVA food classification and categorized each food/beverage item into one of the four food groups based on their extent and purpose of industrial food processing[9]: (1) *unprocessed/minimally processed foods* are fresh, frozen, ground, pasteurized or (non-alcoholic) fermented foods after separation from nature, e.g. fruit, vegetable, milk, meat, legumes; (2) *processed culinary ingredients* are substances extracted from foods and used in common culinary preparation, cooking and seasoning of group 1 foods, e.g. table salt, sugar, vegetable oils and butter; (3) *processed foods* are made by adding salt, sugar or other group 2 ingredients to group 1 foods, e.g. canned vegetables in brine, canned fish, freshly made breads and cheeses; and (4) *UPFs* are food/drink formulations of multiple substances, mostly of exclusive industrial use (e.g. high-fructose corn syrup), and are manufactured through a series of complex industrial processes (e.g. hydrogenation) and often contain cosmetic additives (e.g. colors) that disguise any undesirable sensorial properties of the final product.[9] Some examples are carbonated or dairy-based drinks, industrial-processed packaged breads with added preservatives or emulsifiers, and pre-prepared frozen/shelf-stable meals made with modified starches, stabilizers or flavor enhancers (full list of UPFs are presented in eFigure 1 in the Supplement).

Study covariates

Covariates include children's age at clinic assessment, sex (male/female), ethnicity (white/non-white), birth weight (<2500g/2500-3999g/≥4000g), baseline physical activity (moderate-to-vigorous physical activity [MVPA] per day≥60 minutes/otherwise) and mean total energy intake (continuous, kcal/day), and quintiles of the Index of Multiple Deprivation (IMD) 2004. IMD is the most common measure of deprivation for each small area of England based on seven domains.[29] Physical activity was based on the earliest recording of accelerometry data (collected at ages 11, 13 and 15 years) where children were instructed to wear a uniaxial ActiGraph 7164 accelerometer for seven days. We categorized accelerometry data into two groups according to the UK government's recommendation for children to accumulate ≥60 minutes MVPA per day.[26,30,31]

Mother's self-reported data at baseline include pre-pregnancy BMI (<18.5/18.5-24.9/25-29.9/≥30kg/m^2), marital status (single/married or living with partner), highest educational attainment (CSE or none/vocational/O level/A level/Degree or above) and socio-economic position based on the UK National Statistics Socioeconomic Classification (higher managerial, administrative and professional/intermediate/routine and manual occupation).[32]

Statistical Analysis

A total of 9025 children were included in the study after excluding 4581 children who did not participate in any clinic assessment; 1271 children with no dietary data; and 11 children with no outcome measurement at or before their dietary data collection (eFigure 2 in the Supplement). Those included were more likely to be female, white (vs non-white) and from higher socio-economic background (eTable 2 in the Supplement). Individual's age at completion of their first food dietary was considered as the baseline, thus 80.4%, 16.8% and 2.6% of the cohort were followed up from 7, 10 and 13 years old, respectively. For each child, we calculated the proportion of UPFs consumed in the total diet (g/day) and expressed as a percentage. This was considered as the primary exposure as it better captures UPFs with zero calorie content such as artificially sweetened beverages (ASBs). However, we also derived for sensitivity analysis a secondary exposure defined as the percentage of calorie contribution from UPFs relative to the total energy intake (kcal/day). We categorized individuals' baseline UPF consumption into quintiles, based on the cut-off points derived from age 7 dietary data since most children were followed up from 7 years old. We further compared this with quintiles derived from age 10, and age 13 dietary data. The quintiles were found largely similar and no gender-specific differences were identified.

Differences in baseline characteristics by UPF quintiles were compared using χ^2 tests and the analysis of variance where appropriate. Linear growth curve models were used to investigate the longitudinal associations between baseline UPF quintile and trajectories of adiposity outcomes. These two-level linear regression models allow for individual-specific random intercept and random slope modelled with age as the underlying timescale. The models included three key variables – age, UPF quintile, and an interaction term between age and UPF quintile that examines the difference in mean growth trajectories of those in higher UPF quintile compared with the lowest quintile reference group. We assessed non-linearity by fitting a quadratic age term in both the fixed and random parts of the growth models. These terms were retained if there was evidence of improved model fit.

We used multiple imputation by chained equation to impute missing covariates data (ranging 1.8%-27.7%) under the assumption of missing at random. Five imputed datasets were generated where the analytical models were performed on each and results combined using Rubin's rule. Analyses based on complete data were conducted for comparison. Study covariates were included in a stepwise manner: Model 1 was not adjusted for any covariates; Model 2 was adjusted for individual's sex, ethnicity, birth weight, level of physical activity and IMD quintile; Model 3 was additionally adjusted for mother's pre-pregnancy BMI, marital status, highest educational attainment and socio-economic position; and Model 4 was additionally adjusted for baseline daily energy intake.

Sensitivity Analyses

We performed a series of sensitivity analyses including further adjustment for baseline fruit and vegetable intake (g/day); intakes of saturated fat (g/day), sugar (g/day), fiber (g/day) and sodium (mg/day); restricting analyses to individuals with follow-up data; excluding twin children from the study cohort; stratifying by boys and girls; and re-categorizing baseline UPF consumption into five groups per 20% absolute increment in their percentage of weight contribution towards daily food intake.

All statistical analyses were performed using Stata SE version 12.1. All statistical tests were two-sided, and a $P<.05$ was considered significant.

RESULTS

A total of 9025 children (4481 [49.6%] female) were followed up over a median (IQR) of 10.2 (5.2-16.4) years. The mean (SD) UPF consumption at baseline from the lowest (Q1) to highest (Q5) quintile was 23.2% (5.0%) of the total daily food intake (g/day) in Q1, 34.7% (2.5%) in Q2, 43.4% (2.5%) in Q3, 52.7% (2.8%) in Q4, and 67.8% (8.1%) in Q5 (eFigure 3 in the Supplement). Children assigned to differing UPF quintiles were not significantly different by sex, ethnicity or birth weight (Table 1). However, children with higher UPF consumption were more likely to have lower maternal socio-economic profile compared with those in lower UPF quintiles. Major sources of UPFs among children in Q5 included fruit-based beverages (22.2%), carbonated beverages (11.5%), ready-to eat/heat foods (8.6%) and industrial-processed breads and buns (5.9%) (eFigure 1 in the Supplement). By contrast, diets among children in Q1 were largely based on minimally-processed foods including water and tea (22.2%), milk and plain yoghurt (20.2%), and fruit (6.0%).

Findings from the growth models remained consistent while adjusting for covariates in multiple steps (eTable 3-4 in the Supplement). Fully-adjusted results for the longitudinal associations between baseline UPF quintile and adiposity outcomes are presented in Table 2, and the fitted trajectories of primary adiposity outcomes are shown in Figure 1. Mean BMI at baseline (age 7 years) did not significantly differ across baseline UPF quintiles (e.g. β, 0.08 kg/m^2 for Q5 vs Q1; 95% CI, -0.09-0.24 kg/m^2). Mean BMI among children in Q1 increased by 0.55 (95% CI, 0.53-0.56) kg/m^2 per year. However, increases in BMI were significantly greater among the three highest UPF quintiles with evidence of a dose-response relationship, e.g. BMI increased by an additional 0.06 (95% CI, 0.04-0.08) kg/m^2 in Q5 compared with Q1.

Mean FMI at baseline (age 9 years) was significantly higher in Q5 by 0.27 (95% CI, 0.09-0.45) kg/m^2 compared with Q1. Mean FMI increased by 0.22 (95% CI, 0.20-0.23) kg/m^2 per year in Q1, and this growth trajectory was found significantly greater in Q5 than Q1 by an additional 0.03 (95% CI, 0.01-0.05) kg/m^2 per year. At baseline (age 9 years), mean body fat percentage was significantly higher among children of the three highest UPF quintiles. However, the growing trajectories of body fat percentage were not significantly different across UPF quintiles. Mean LMI was estimated to grow at an annual rate of 0.55 – 2*0.02*year (kg/m^2) from 9 years old, but neither the LMI at 9 years old nor its growth trajectory was found significantly different among children of varying UPF quintiles.

Mean levels of BMI z-score, weight and waist circumference were not significantly different at baseline (age 7 years) across UPF quintiles except for weight among Q2 (Table 2, Figure 2). However, when compared with children in Q1, increases in weight and waist circumference trajectories were significantly greater in the two and three highest UPF quintiles respectively, with evidence of a dose-response relationship. Trajectories of BMI z-score were only significantly greater in Q5. Results for FM and LM were found similar to FMI and LMI findings, respectively.

Results of sensitivity analyses were largely consistent with the main findings (eTable 5-6 and eFigure 4-6 in the Supplement). Girls were observed with a steeper trajectory of body fat measures than boys although their BMI trajectories were similar. Analyses using the secondary exposure showed that the mean UPF consumption in the study cohort was 61.4% out of the daily energy intake, and major contributors of energy intake were ultra-processed ready-to-eat/heat foods and industrial-processed breads and buns.

DISCUSSION

In this large prospective study following up British children from age 7 to 24 years, growth trajectories of BMI, FMI, weight and waist circumference increased by an additional 0.06 (95% CI, 0.04-0.08) kg/m^2, 0.03 (95% CI, 0.01-0.05) kg/m^2, 0.20 (95% CI, 0.11-0.28) kg and 0.17 (95% CI, 0.11-0.22) cm each year among children with the highest (vs lowest) UPF consumption. Evidence of dose-response relationships were consistently observed for BMI, weight and waist circumference trajectories among those in the two highest UPF quintiles. By the age of 24 years, a clinically important difference was observed in e.g. BMI by 1.18 (95% CI, 0.78-1.57) kg/m^2, FMI by 0.78 (95% CI, 0.46-1.08) kg/m^2 and body fat percentage by 1.53% (0.81%-2.25%) greater among those with the highest (vs lowest) UPF consumption.

Previous cohort studies of children/adolescents (sample size, 307-3454) had shorter follow-up and provide inconsistent findings.[16-20] Two studies found no significant associations between UPF consumption at 4 years old and BMI measures 3-4 years later whereas one study reported no differences in BMI growth between 16-18 years of age.[16,17,20] However, a Portuguese study reported a 0.028 increase in BMI z-score at 10 years of age per 100 kcal/day higher UPF consumption at 4 years old,[19] and a Brazilian study reported a 0.20 kg/m^2 and 0.14 kg/m^2 increase in BMI and FMI, respectively, between ages 6-11 years per 100 g/day increase in UPF consumption.[18] Our findings were based on multiple adiposity measurements between ages 7-24 years and detailed 3-day food diaries whereas previous studies were largely based on food frequency questionnaires that may have limited ability to accurately capture UPFs. Notably, British children have a prominently high UPF consumption than previous studies based in Brazil, Portugal or Spain (ranging 27.3%-42.0% of daily calorie intake).[16,18,19] The positive longitudinal association between childhood consumption of sugar-sweetened beverages (SSBs) and adiposity has been widely documented,[33] and our results are reflective of this as SSBs and ASBs constituted a great proportion of UPF consumption especially in those with the highest quintile (33.7%).

The increasing availability and variety of UPFs have reshaped global food systems displacing dietary patterns previously based on fresh and minimally-processed foods.[9,10] Of particular concern is the growing consumption among children and adolescents who are leading consumers including in middle-income countries.[11,12,34,35] These have major public health implications with higher UPF consumption associated with excess calorie intake[1] and elevated risk of obesity,[2,3] type 2 diabetes,[4,5] hypertension,[36] cardiovascular disease,[6] cancer,[7] and mortality.[8] Our findings add novel evidence showing positive associations between UPF consumption and adiposity outcomes throughout childhood, which is crucially important as lifelong dietary patterns develop from childhood and may lead to widespread consequences on health and well-being throughout the life course.[37]

The UPF industry is highly profitable through the use of low-cost supply chains and aggressive marking strategies to promote excess consumption.[14,15] Global economic policies and trade agreements which favor the interests of transnational food corporations have further enhanced their central role in the global transformation of food systems and undermined implementation of effective policies to curb UPF consumption.[10,15] Nevertheless, policies are emerging that explicitly target UPFs.[10] Public health authorities of Brazil, Uruguay, Ecuador, Peru, France, Canada and Israel have amended their national dietary guidelines with recommendations to limit UPF consumption.[10,38,39] France has set an ambitious target to reduce UPF consumption by 20% by 2022. Action on UPFs in the UK and elsewhere remains limited, instead emphasizing on reducing certain nutrient.[14,40] Voluntary product reformulations have been shown ineffective,[10,40] and even bolder regulatory action will not address their health harms as they may miss out several UPFs (e.g. ASBs) that contain industrial *trans* fatty acids,[41] food additives or toxic contaminants,[42,43] even when their calorie, salt and sugar are reduced to limit. Only mandatory policies that target UPFs holistically, with cooperative actions globally to strengthen regulations and trade agreements aim at reducing the supply and consumption of UPFs will counteract the substantial burden of UPF consumption on the environment and health care systems worldwide.[14,40,44]

Limitations

Our study has several limitations. First, some individuals had fewer adiposity measurements collected and no data collection was conducted between ages 17 and 24 years. However, completeness of outcome data was high in the study cohort (89.5%-99.9%), and a mean of 3.9-6.5 repeated measurements across study outcomes were available. Second, there may be potential misclassification of food/beverage items by the NOVA classification, but this is likely minimal given the detailed food diaries used. Third, major changes in UPF consumption may contribute to a shift in adiposity trajectories but we did not use a time-varying exposure because of the modest changes in UPF consumption between ages 7-13 years. A total of 7072 children (78.4%) provided follow-up dietary data but only 1,288 children (14.2%) were observed with an absolute change in UPF consumption of greater than ±20% between ages 7 and 10 years; and 1831 children (20.2%) between ages 10 and 13 years. Fourth, availability of multiple food diaries lowers measurement bias, and only 8% of the cohort completed on a single occasion while most participants completed two or more days. Fifth, we examined potential dietary mis-reporting based on the ratio of energy intake over estimated energy expenditure.[45] The results remained closely consistent after the exclusion of 1314 (14.6%) under-reporters and 715 (7.9%) over-reporters. Sixth, missing data may introduce bias, but we used multiple imputation while auxiliary variables were included as appropriate. Comparison of main findings to complete case analysis showed largely similar results. Finally, although a wide range of factors have been accounted for, the observational nature of the study means that residual confounding may have affected our results.

Conclusion

Our findings provide novel and important evidence that higher consumption of UPFs in childhood is associated with more rapid progression of BMI, FMI, weight and waist circumference into adolescence and early adulthood. More radical and effective public health actions that reduce children’s exposure and consumption of UPFs are urgently needed to address childhood obesity in England and internationally.

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Author Contributions

Dr Chang had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: Khanpur, Millett, Chang, Vamos.

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Conflict of Interest Disclosures

All authors report no conflict of interest.

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REFERENCES

1. Hall KD, Ayuketah A, Brychta R, et al. Ultra-Processed Diets Cause Excess Calorie Intake and Weight Gain: An Inpatient Randomized Controlled Trial of Ad Libitum Food Intake. *Cell Metabolism* 2019.
2. Rauber F, Chang K, Vamos EP, et al. Ultra-processed food consumption and risk of obesity: a prospective cohort study of UK Biobank. *European Journal of Nutrition* 2020: 1-12.
3. Beslay M, Srour B, Méjean C, et al. Ultra-processed food intake in association with BMI change and risk of overweight and obesity: A prospective analysis of the French NutriNet-Santé cohort. *PLoS medicine* 2020; **17**(8): e1003256.
4. Srour B, Fezeu LK, Kesse-Guyot E, et al. Ultraprocessed Food Consumption and Risk of Type 2 Diabetes Among Participants of the NutriNet-Santé Prospective Cohort. *Jama Internal Medicine* 2020; **180**(2): 283-91.
5. Levy RB, Rauber F, Chang K, et al. Ultra-processed food consumption and type 2 diabetes incidence: a prospective cohort study. *Clinical Nutrition* 2020.
6. Srour B, Fezeu LK, Kesse-Guyot E, et al. Ultra-processed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). *BMJ* 2019; **365**.
7. Fiolet T, Srour B, Sellem L, et al. Consumption of ultra-processed foods and cancer risk: results from NutriNet-Santé prospective cohort. *BMJ* 2018; **360**.
8. Schnabel L, Kesse-Guyot E, Allès B, et al. Association between ultraprocessed food consumption and risk of mortality among middle-aged adults in France. *JAMA internal medicine* 2019; **179**(4): 490-8.
9. Monteiro CA, Cannon G, Levy RB, et al. Ultra-processed foods: what they are and how to identify them. *Public health nutrition* 2019; **22**(5): 936-41.
10. Baker P, Machado P, Santos T, et al. Ultra-processed foods and the nutrition transition: Global, regional and national trends, food systems transformations and political economy drivers. *Obesity Reviews* 2020.

11. Onita BM, Azeredo CM, Jaime PC, Levy RB, Rauber F. Eating context and its association with ultra-processed food consumption by British children. *Appetite* 2020; **157**: 105007.
12. Neri D, Martinez-Steele E, Monteiro CA, Levy RB. Consumption of ultra-processed foods and its association with added sugar content in the diets of US children, NHANES 2009-2014. *Pediatric obesity* 2019; **14**(12): e12563.
13. Abarca-Gómez L, Abdeen ZA, Hamid ZA, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128· 9 million children, adolescents, and adults. *The Lancet* 2017; **390**(10113): 2627-42.
14. Adams J, Hofman K, Moubarac J-C, Thow AM. Public health response to ultra-processed food and drinks. *BMJ* 2020; **369**.
15. White M, Aguirre E, Finegood DT, Holmes C, Sacks G, Smith R. What role should the commercial food system play in promoting health through better diet? *BMJ* 2020; **368**.
16. Costa C, Rauber F, Leffa P, Sangalli C, Campagnolo P, Vitolo M. Ultra-processed food consumption and its effects on anthropometric and glucose profile: A longitudinal study during childhood. *Nutrition, Metabolism and Cardiovascular Diseases* 2019; **29**(2): 177-84.
17. Bawaked RA, Fernández-Barrés S, Navarrete-Muñoz EM, et al. Impact of lifestyle behaviors in early childhood on obesity and cardiometabolic risk in children: Results from the Spanish INMA birth cohort study. *Pediatric Obesity* 2020; **15**(3): e12590.
18. Costa CdS, Assunção MCF, Loret de Mola C, et al. Role of ultra-processed food in fat mass index between 6 and 11 years of age: a cohort study. *International journal of epidemiology* 2020.
19. Vedovato GM, Vilela S, Severo M, Rodrigues S, Lopes C, Oliveira A. Ultra-processed food consumption, appetitive traits and BMI in children: a prospective study. *British Journal of Nutrition* 2020: 1-25.
20. Cunha DB, da Costa THM, da Veiga GV, Pereira RA, Sichieri R. Ultra-processed food consumption and adiposity trajectories in a Brazilian cohort of adolescents: ELANA study. *Nutrition & diabetes* 2018; **8**(1): 1-9.

21. Boyd A, Golding J, Macleod J, et al. Cohort profile: the ‘children of the 90s’—the index offspring of the Avon Longitudinal Study of Parents and Children. *International journal of epidemiology* 2013; **42**(1): 111-27.
22. Fraser A, Macdonald-Wallis C, Tilling K, et al. Cohort profile: the Avon Longitudinal Study of Parents and Children: ALSPAC mothers cohort. *International journal of epidemiology* 2013; **42**(1): 97-110.
23. Northstone K, Lewcock M, Groom A, et al. The Avon Longitudinal Study of Parents and Children (ALSPAC): an update on the enrolled sample of index children in 2019. *Wellcome open research* 2019; **4**.
24. Harris PA, Taylor R, Thielke R, Payne J, Gonzalez N, Conde JG. Research electronic data capture (REDCap)—a metadata-driven methodology and workflow process for providing translational research informatics support. *Journal of biomedical informatics* 2009; **42**(2): 377-81.
25. Harris PA, Taylor R, Minor BL, et al. The REDCap consortium: Building an international community of software platform partners. *Journal of biomedical informatics* 2019; **95**: 103208.
26. ALSPAC data dictionary. http://www.bristol.ac.uk/alspac/researchers/our-data/ (Last accessed: 01/Dec/2020).
27. Cole TJ, Freeman JV, Preece MA. British 1990 growth reference centiles for weight, height, body mass index and head circumference fitted by maximum penalized likelihood. *Statistics in medicine* 1998; **17**(4): 407-29.
28. Holland B, Welch A, Unwin I, Buss D, Paul A, Southgate D. McCance & Widdowson's The Composition of Foods. 5th ed. Cambridge: Royal Society of Chemistry; 1991.
29. Noble M, Wright G, Dibben C, et al. The English Indices of Deprivation 2004. April 2004. In: The Office of the Deputy Prime Minister. London: Neighbourhood Renewal Unit, editor.
30. Evenson KR, Catellier DJ, Gill K, Ondrak KS, McMurray RG. Calibration of two objective measures of physical activity for children. *Journal of sports sciences* 2008; **26**(14): 1557-65.
31. Department of Health and Social Care. UK Chief Medical Officers' Physical Activity Guidelines. September 2019.

32. Office for National Statistics. The National Statistics Socio-economic classification (NS-SEC). https://www.ons.gov.uk/methodology/classificationsandstandards/otherclassifications/thenationalstatisticssocioeconomicclassificationnssecrebasedonsoc2010 (Last accessed: 16/Dec/2020).
33. Marshall TA, Curtis AM, Cavanaugh JE, Warren JJ, Levy SM. Child and adolescent sugar-sweetened beverage intakes are longitudinally associated with higher body mass index z scores in a birth cohort followed 17 years. *Journal of the Academy of Nutrition and Dietetics* 2019; **119**(3): 425-34.
34. Marrón-Ponce JA, Sánchez-Pimienta TG, da Costa Louzada ML, Batis C. Energy contribution of NOVA food groups and sociodemographic determinants of ultra-processed food consumption in the Mexican population. *Public health nutrition* 2018; **21**(1): 87-93.
35. Cediel G, Reyes M, da Costa Louzada ML, et al. Ultra-processed foods and added sugars in the Chilean diet (2010). *Public health nutrition* 2018; **21**(1): 125-33.
36. Mendonça RdD, Lopes ACS, Pimenta AM, Gea A, Martinez-Gonzalez MA, Bes-Rastrollo M. Ultra-processed food consumption and the incidence of hypertension in a Mediterranean cohort: the Seguimiento Universidad de Navarra Project. *American Journal of Hypertension* 2017; **30**(4): 358-66.
37. Mikkilä V, Räsänen L, Raitakari O, Pietinen P, Viikari J. Consistent dietary patterns identified from childhood to adulthood: the cardiovascular risk in Young Finns Study. *British Journal of Nutrition* 2005; **93**(6): 923-31.
38. Haut Conseil de la santé publique. Avis Relatif à La Révision Des Repères Alimentaires Pour Les Adultes Du Futur Programme National Nutrition Santé 2017-2021. https://www.hcsp.fr/Explore.cgi/Telecharger?NomFichier=hcspa20170216_reperesalimentairesactua2017.pdf (Last accessed: 01/Dec/2020). 2017.
39. The Israeli Ministry of Health. Dietary Guidelines. https://www.health.gov.il/PublicationsFiles/dietary%20guidelines%20EN.pdf (Last accessed: 14/Jan/2021). 2019.
40. Seferidi P, Millett C, Laverty AA. Industry self-regulation fails to deliver healthier diets, again. *BMJ* 2021; **372**: m4762.

41. World Health Organization. Countdown to 2023: WHO report on global trans-fat elimination 2020. https://apps.who.int/iris/handle/10665/334170 (Last accessed: 01/Dec/2020). 2020.
42. ECHA. Agreement of the Member State Committee on the identification of 4, 4′-isopropylidenediphenol (bisphenol A) as a substance of very high concern. https://echa.europa.eu/documents/10162/1c8a321c-7b59-7fe8-69f4-bfcce6de5cfe (Last accessed: 01/Dec/2020). 2017.
43. Golestanzadeh M, Riahi R, Kelishadi R. Association of exposure to phthalates with cardiometabolic risk factors in children and adolescents: a systematic review and meta-analysis. *Environmental Science and Pollution Research* 2019: 1-17.
44. Seferidi P, Scrinis G, Huybrechts I, Woods J, Vineis P, Millett C. The neglected environmental impacts of ultra-processed foods. *The Lancet Planetary Health* 2020; **4**(10): e437-e8.
45. Gomes D, Luque V, Xhonneux A, et al. A simple method for identification of misreporting of energy intake from infancy to school age: Results from a longitudinal study. *Clinical Nutrition* 2018; **37**(3): 1053-60.

LIST OF FIGURES AND LEGENDS

Figure 1. Trajectories of primary outcomes by baseline quintile of ultra-processed food consumption among 9025 ALSPAC children

Abbreviation: UPF%, percentage of daily food intake (g/day) contributed by ultra-processed foods (UPFs) at baseline and was further categorized into quintiles (Q1-Q5 represents lowest to highest quintile of UPF consumption). Trajectories were plotted for the predicted values estimated from the growth curve models at each age (wave) of clinic assessment. All linear growth models were fitted with individual-specific random intercept and random slope using age (and quadratic age for lean mass index outcome) as the underlying timescale, and included baseline UPF quintile, an interaction term between age and baseline UPF quintile, and were further adjusted by child's sex (male/female), ethnicity (white/non-white), birth weight (<2500g/2500-3999g/≥4000g), physical activity (moderate-to-vigorous physical activity per day≥60 minutes/otherwise), quintiles of Index of Multiple Deprivation, mother's pre-pregnancy BMI (<18.5/18.5-24.9/25-29.9/≥30kg/m^2), marital status (single/married or living with partner), highest educational attainment (CSE or none/vocational/O level/A level/Degree or above), socio-economic status based on UK National Statistics Socioeconomic Classification (higher managerial, administrative and professional/intermediate/routine and manual occupation), and child's total energy intake (continuous, kcal/day) at baseline. Baseline refers to 7 years old for body mass index (BMI), and 9 years old for fat/lean mass index and body fat percentage outcomes.

Figure 2. Trajectories of secondary outcomes by baseline quintile of ultra-processed food consumption among 9025 ALSPAC children

Abbreviation: UPF%, percentage of daily food intake (g/day) contributed by ultra-processed foods (UPFs) at baseline and was further categorized into quintiles (Q1-Q5 represents lowest to highest quintile of UPF consumption). Trajectories were plotted for the predicted values estimated from the growth curve models at each age (wave) of clinic assessment. All linear growth models were fitted with individual-specific random intercept and random slope using age (and quadratic age for weight, waist circumference and lean mass outcomes) as the underlying timescale, and included baseline UPF quintile, an interaction term between age and baseline UPF quintile, and were further adjusted by child's sex (male/female), ethnicity (white/non-white), birth weight (<2500g/2500-3999g/≥4000g), physical activity (moderate-to-vigorous physical activity per day≥60 minutes/otherwise), quintiles of Index of Multiple Deprivation, mother's pre-pregnancy BMI (<18.5/18.5-24.9/25-29.9/≥30kg/m^2), marital status (single/married or living with partner), highest educational attainment (CSE or none/vocational/O level/A level/Degree or above), socio-economic status based on UK National Statistics Socioeconomic Classification (higher managerial, administrative and professional/intermediate/routine and manual occupation), and child's total energy intake (continuous, kcal/day) at baseline. Baseline refers to 7 years old for BMI z-score, weight and waist circumference outcomes; and 9 years old for fat/lean mass and body fat percentage outcomes.

Table 1. Socio-demographic characteristics by baseline quintile of ultra-processed food consumption among 9025 ALSPAC children (1998-2017), England

		Quintile[a] of baseline ultra-processed food consumption					
	Overall	**1 (lowest)**	**2**	**3**	**4**	**5 (highest)**	***P* value[c]**
N	9025	1708	1759	1923	1777	1858	
Range of UPF%	0-100	0-29.9	30.0-38.9	39.0-47.9	48.0-57.9	58.0-100	
UPF% (mean, SD)	44.7 (15.9)	23.2 (5.0)	34.7 (2.5)	43.4 (2.5)	52.7 (2.8)	67.8 (8.1)	
Total energy intake at baseline (mean, SD)							
kcal/day	1729 (347)	1698 (342)	1753 (345)	1737 (332)	1731 (335)	1726 (376)	<.001
Age at baseline[b], **No. (%)**							
7 years	7264 (80.4)	1327 (77.6)	1435 (81.4)	1584 (82.3)	1460 (82.0)	1458 (78.4)	<.001
10 years	1519 (16.8)	292 (17.0)	270 (15.3)	296 (15.3)	297 (16.6)	364 (19.5)	
13 years	242 (2.6)	89 (5.2)	54 (3.0)	43 (2.2)	20 (1.1)	36 (1.9)	
Sex, **No. (%)**							
Male	4544 (50.2)	821 (48.0)	884 (50.1)	966 (50.2)	927 (52.0)	946 (50.8)	.18
Female	4481 (49.6)	887 (51.8)	875 (49.6)	957 (49.7)	850 (47.7)	912 (49.0)	
Ethnicity, **No. (%)**							
Non-white	780 (8.6)	152 (8.8)	165 (9.3)	170 (8.8)	157 (8.8)	136 (7.3)	.47
White	8029 (88.8)	1512 (88.4)	1553 (88.1)	1704 (88.5)	1585 (89.0)	1675 (90.1)	
Missing	216 (2.3)	44 (2.5)	41 (2.3)	49 (2.5)	35 (1.9)	47 (2.5)	
Birth weight, **No. (%)**							
<2500g	409 (4.5)	67 (3.9)	86 (4.8)	88 (4.5)	84 (4.7)	84 (4.5)	.22
2500 - 3999g	6905 (76.4)	1339 (78.3)	1337 (75.9)	1450 (75.3)	1385 (77.8)	1394 (74.9)	
≥4000g	1112 (12.3)	201 (11.7)	219 (12.4)	235 (12.2)	207 (11.6)	250 (13.4)	
Missing	599 (6.6)	101 (5.9)	117 (6.6)	150 (7.7)	101 (5.6)	130 (6.9)	
Physical activity, **No. (%)**							
MVPA<60 minutes	4076 (45.1)	821 (48.0)	812 (46.1)	840 (43.6)	784 (44.0)	819 (44.0)	.02
MVPA≥60 minutes	2453 (27.1)	468 (27.3)	476 (27.0)	542 (28.1)	481 (27.0)	486 (26.1)	
Missing	2496 (27.6)	419 (24.5)	471 (26.7)	541 (28.1)	512 (28.7)	553 (29.7)	
Index of Multiple Deprivation 2004, **No. (%)**							
Quintile 1 (Least derived)	2855 (31.6)	537 (31.4)	585 (33.2)	629 (32.6)	552 (31.0)	552 (29.6)	<.001
Quintile 2	2113 (23.3)	460 (26.9)	413 (23.4)	454 (23.5)	404 (22.6)	382 (20.5)	
Quintile 3	1795 (19.8)	339 (19.8)	352 (19.9)	401 (20.8)	348 (19.5)	355 (19.0)	
Quintile 4	1198 (13.2)	192 (11.2)	217 (12.3)	222 (11.5)	267 (15.0)	300 (16.1)	
Quintile 5 (Most deprived)	899 (9.9)	142 (8.3)	161 (9.1)	180 (9.3)	177 (9.9)	239 (12.8)	
Missing	165 (1.8)	38 (2.2)	31 (1.7)	37 (1.9)	29 (1.6)	30 (1.6)	

Table 1. Socio-demographic characteristics by baseline quintile of ultra-processed food consumption among 9025 ALSPAC children (1998-2017), England (continued)

		Quintile[a] of baseline ultra-processed food consumption					
	Overall	**1 (lowest)**	**2**	**3**	**4**	**5 (highest)**	***P* value[c]**
N	9025	1708	1759	1923	1777	1858	
Range of UPF%	0-100	0-29.9	30.0-38.9	39.0-47.9	48.0-57.9	58.0-100	
UPF% (mean, SD)	44.7 (15.9)	23.2 (5.0)	34.7 (2.5)	43.4 (2.5)	52.7 (2.8)	67.8 (8.1)	
Mother's self-reported pre-pregnancy BMI, No. (%)							
Underweight (<18.5kg/m^2)	334 (3.6)	74 (4.3)	65 (3.6)	68 (3.5)	54 (3.0)	73 (3.9)	<.001
Normal (18.5-24.9kg/m^2)	5752 (63.6)	1153 (67.4)	1171 (66.4)	1203 (62.5)	1159 (65.1)	1066 (57.3)	
Overweight (25-29.9kg/m^2)	1150 (12.7)	177 (10.3)	200 (11.3)	255 (13.2)	223 (12.5)	295 (15.8)	
Obese (≥30kg/m^2)	393 (4.3)	48 (2.8)	63 (3.5)	88 (4.5)	88 (4.9)	106 (5.7)	
Missing	1396 (15.4)	256 (14.9)	260 (14.7)	309 (16.0)	253 (14.2)	318 (17.1)	
Mother's marital status, No. (%)							
Single	1625 (17.9)	298 (17.4)	298 (16.9)	313 (16.2)	353 (19.8)	363 (19.5)	.05
Married/living with partner	7203 (79.7)	1374 (80.3)	1423 (80.8)	1561 (81.1)	1393 (78.2)	1452 (78.1)	
Missing	197 (2.1)	36 (2.1)	38 (2.1)	49 (2.5)	31 (1.7)	43 (2.3)	
Mother's highest educational attainment, No. (%)							
CSE/none	738 (8.1)	99 (5.7)	110 (6.2)	167 (8.6)	148 (8.3)	214 (11.5)	<.001
Vocational	662 (7.3)	92 (5.3)	123 (6.9)	119 (6.1)	144 (8.0)	184 (9.8)	
O level	3189 (35.2)	468 (27.3)	560 (31.8)	700 (36.3)	696 (39.1)	765 (41.1)	
A level	2421 (26.7)	497 (29.0)	529 (30.0)	497 (25.8)	470 (26.4)	428 (23.0)	
Degree	1569 (17.3)	462 (27.0)	362 (20.5)	340 (17.6)	236 (13.2)	169 (9.0)	
Missing	446 (4.9)	90 (5.2)	75 (4.2)	100 (5.1)	83 (4.6)	98 (5.2)	
Mother's NSSEC, No. (%)							
1.Higher managerial, administrative and professional	2822 (31.2)	667 (39.0)	624 (35.4)	607 (31.5)	487 (27.3)	437 (23.5)	<.001
2.Intermediate occupations	2716 (30.0)	446 (26.0)	503 (28.5)	564 (29.3)	580 (32.5)	623 (33.5)	
3.Routine and manual occupations	2598 (28.7)	418 (24.4)	479 (27.2)	557 (28.9)	544 (30.5)	600 (32.2)	
Missing	889 (9.8)	177 (10.3)	153 (8.6)	195 (10.1)	166 (9.3)	198 (10.6)	

Abbreviations: UPF%, percentage of daily food intake (g/day) contributed by ultra-processed foods (UPFs) at baseline; SD, standard deviation; MVPA, moderate-to-vigorous physical activity; BMI, body mass index; NSSEC, (UK) National Statistics Socioeconomic Classification.

[a] Quintile of UPF consumption was first computed for age 7, 10 and 13 dietary data separately and were found largely similar across waves, thus a set of cut-off points for the baseline quintiles of UPF consumption was derived based on age 7 data and defined at 30%, 39%, 48% and 58% of daily food intake (g/day).

[b] Age when baseline UPF consumption was collected, this indicates that >80% of children were followed up from 7 years old.

[c] Chi-square tests were used to compare socio-demographic characteristics and ANOVA were used to compare mean total energy intake between children grouped by UPF quintiles.

Table 2. Longitudinal associations[a] between baseline ultra-processed food consumption and adiposity among 9025 ALSPAC children (1998-2017), England

		Body Mass Index (kg/m^2) *n=9020*	BMI z-score *n=9018*	Fat Mass Index (kg/m^2) *n=8078*	Fat Mass (kg) *n=8085*	Total fat percentage (%) *n=8085*
		Coeff (95% CI)	**Coeff (95% CI)**	**Coeff (95% CI)**	**Coeff (95% CI)**	**Coeff (95% CI)**
Baseline UPF Quintile[b]	Q1	0 [reference]	0 [reference]	0 [reference]	0 [reference]	0 [reference]
	Q2	0.06 (-0.10 to 0.23)	0.06 (-0.01 to 0.13)	0.08 (-0.09 to 0.26)	0.11 (-0.31 to 0.52)	0.65 (-0.01 to 1.30)
	Q3	0.006 (-0.16 to 0.17)	0.03 (-0.04 to 0.10)	0.11 (-0.06 to 0.28)	0.10 (-0.32 to 0.51)	0.67 (0.02 to 1.32)[e]
	Q4	0.02 (-0.15 to 0.19)	0.05 (-0.02 to 0.12)	0.17 (-0.01 to 0.34)	0.20 (-0.22 to 0.62)	1.02 (0.35 to 1.67)[f]
	Q5	0.08 (-0.09 to 0.24)	0.05 (-0.02 to 0.12)	0.27 (0.09 to 0.45)[f]	0.51 (0.08 to 0.93)[e]	1.47 (0.81 to 2.13)[f]
Age[c]	per year	0.55 (0.53 to 0.56)[f]	0.02 (0.01 to 0.02)[f]	0.22 (0.20 to 0.23)[f]	0.96 (0.92 to 1.00)[f]	0.39 (0.35 to 0.43)[f]
Interaction[d]	Q1*Age	0 [reference]	0 [reference]	0 [reference]	0 [reference]	0 [reference]
	Q2*Age	0.02 (-0.001 to 0.04)	0.0003 (-0.006 to 0.007)	0.005 (-0.01 to 0.02)	0.03 (-0.02 to 0.09)	-0.03 (-0.08 to 0.02)
	Q3*Age	0.03 (0.005 to 0.04)[e]	0.002 (-0.005 to 0.009)	0.01 (-0.01 to 0.02)	0.06 (-0.003 to 0.11)	-0.02 (-0.07 to 0.03)
	Q4*Age	0.04 (0.01 to 0.06)[f]	0.003 (-0.004 to 0.009)	0.01 (-0.01 to 0.03)	0.07 (0.01 to 0.13)[e]	-0.04 (-0.10 to 0.01)
	Q5*Age	0.06 (0.04 to 0.08)[f]	0.01 (0.003 to 0.01)[f]	0.03 (0.01 to 0.05)[f]	0.15 (0.08 to 0.21)[f]	0.004 (-0.05 to 0.06)

		Weight (kg) *n=9021*	Waist Circumference (cm) *n=9021*	Lean Mass Index (kg/m^2) *n=8078*	Lean Mass (kg) *n=8085*
Baseline UPF Quintile[b]	Q1	0 [reference]	0 [reference]	0 [reference]	0 [reference]
	Q2	0.35 (0.007 to 0.69)[e]	0.26 (-0.14 to 0.66)	0.005 (-0.06 to 0.07)	0.13 (-0.16 to 0.42)
	Q3	0.30 (-0.03 to 0.63)	0.03 (-0.36 to 0.42)	0.009 (-0.06 to 0.07)	-0.01 (-0.30 to 0.28)
	Q4	0.34 (-0.007 to 0.68)	0.22 (-0.18 to 0.62)	-0.01 (-0.08 to 0.05)	-0.07 (-0.36 to 0.23)
	Q5	0.30 (-0.04 to 0.65)	0.16 (-0.25 to 0.56)	-0.01 (-0.08 to 0.05)	0.07 (-0.23 to 0.37)
Age[c]	per year	5.46 (5.38 to 5.53)[f]	3.36 (3.30 to 3.41)[f]	0.55 (0.53 to 0.55)[f]	4.44 (4.38 to 4.49)[f]
Age^2[c]	per year	-0.12 (-0.12 to -0.11)[f]	-0.11 (-0.11 to -0.10)[f]	-0.02 (-0.02 to -0.01)[f]	-0.17 (-0.17 to -0.16)[f]
Interaction[d]	Q1*Age	0 [reference]	0 [reference]	0 [reference]	0 [reference]
	Q2*Age	0.06 (-0.02 to 0.14)	0.05 (-0.008 to 0.10)	0.008 (-0.003 to 0.01)	0.02 (-0.04 to 0.08)
	Q3*Age	0.04 (-0.03 to 0.12)	0.06 (0.006 to 0.11)[e]	-0.003 (-0.01 to 0.008)	-0.007 (-0.07 to 0.05)
	Q4*Age	0.10 (0.01 to 0.18)[e]	0.08 (0.02 to 0.14)[f]	0.009 (-0.002 to 0.02)	0.03 (-0.03 to 0.10)
	Q5*Age	0.20 (0.11 to 0.28)[f]	0.17 (0.11 to 0.22)[f]	0.004 (-0.007 to 0.01)	-0.04 (-0.11 to 0.02)

Abbreviations: Coeff, coefficient; CI, confidence interval; UPF, ultra-processed food consumption was defined as the proportion of its weight contribution relative to daily food intake measured in g/day and was categorized into quintiles (Q1-Q5 represents lowest to highest quintile of UPF consumption).

[a] Linear growth curve models were employed with individual-specific random intercept and random slope using age (and quadratic age where appropriate) as the underlying timescale, and included baseline UPF quintile, an interaction term between age and baseline UPF quintile, and were further adjusted by child's sex (male/female), ethnicity (white/non-white), birth weight (<2500g/2500-3999g/≥4000g), physical activity (moderate-to-vigorous physical activity per day≥60 minutes/otherwise), quintiles of Index of Multiple Deprivation, mother's pre-pregnancy BMI (<18.5/18.5-24.9/25-29.9/≥30kg/m^2), marital status (single/married or living with partner), highest educational attainment (CSE or none/vocational/O level/A level/Degree

or above), socio-economic status based on UK National Statistics Socioeconomic Classification (higher managerial, administrative and professional/intermediate/routine and manual occupation), and child's total energy intake (continuous, kcal/day) at baseline**.** Baseline refers to 7 years old for body mass index (BMI), BMI z-score, weight and waist circumference outcomes; and 9 years old for fat/lean mass index, fat/lean mass and body fat percentage outcomes.

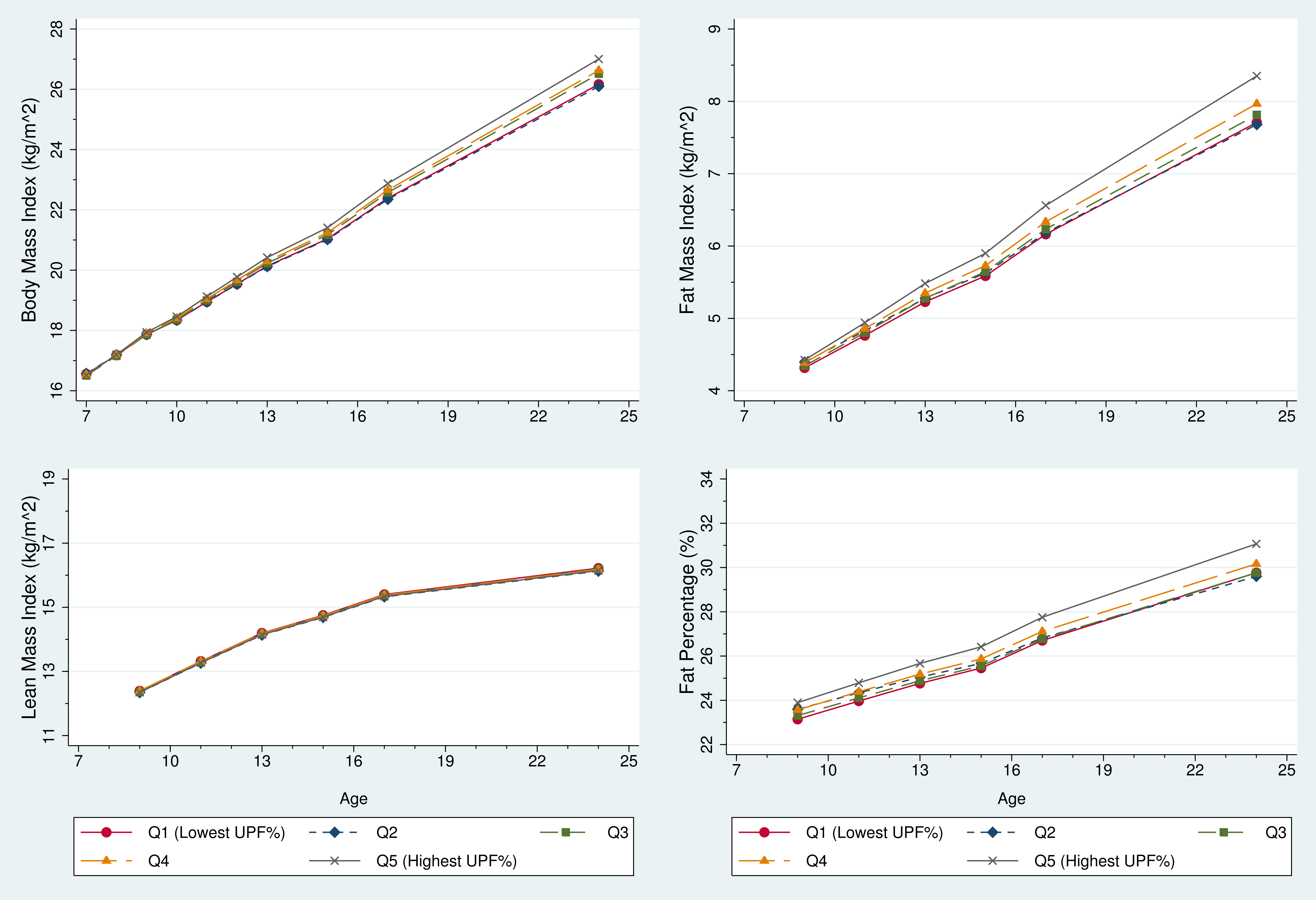
[b] Coefficient of Baseline UPF quintile: assesses the difference in mean adiposity outcomes at baseline among those of higher UPF consumption quintile compared with the lowest UPF quintile reference group.

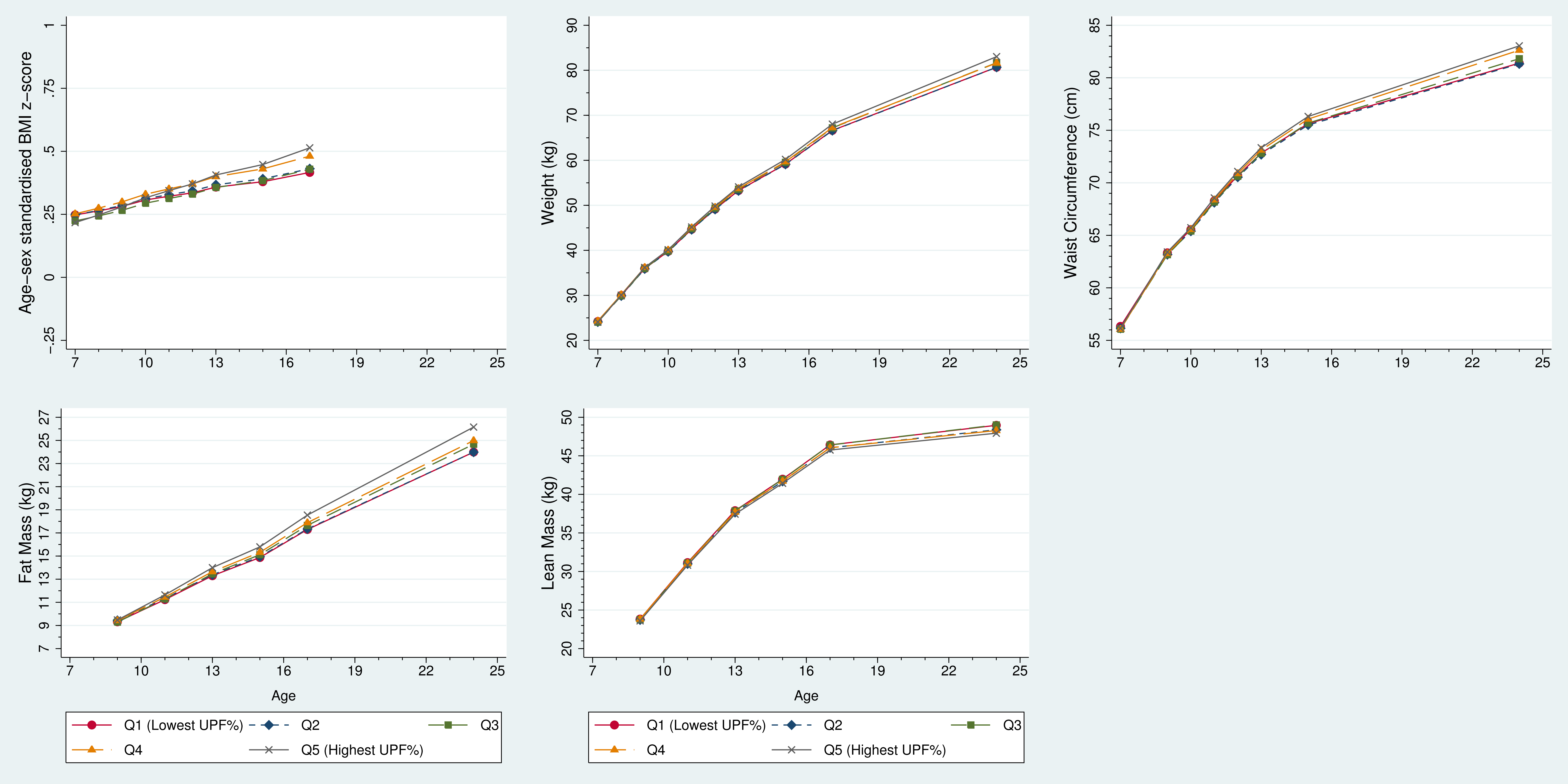
[c] Coefficient of Age and Age^2: captures the average yearly growth in adiposity outcomes for the reference group and were centered at baseline age of each outcome (described above).

[d] Coefficient of Interaction term: examines the difference in average growth trajectories of higher UPF consumption quintile compared with the lowest UPF quintile reference group.

[e] $P < .05$

[f] $P < .01$


Body Mass Index (kg/m^2)
Fat Mass Index (kg/m^2)
Lean Mass Index (kg/m^2)
Fat Percentage (%)
Age
Age
Q1 (Lowest UPF%)
Q2
Q3
Q4
Q5 (Highest UPF%)
Q1 (Lowest UPF%)
Q2
Q3
Q4
Q5 (Highest UPF%)


Age−sex standardised BMI z−score
Weight (kg)
Waist Circumference (cm)
Fat Mass (kg)
Lean Mass (kg)
Age
Age
Q1 (Lowest UPF%)
Q2
Q3
Q4
Q5 (Highest UPF%)
Q1 (Lowest UPF%)
Q2
Q3
Q4
Q5 (Highest UPF%)