# **Childhood dietary patterns and body composition at age 6 years: the Children of SCOPE study**

Angela C Flynn1, John M DThompson2,3, Kathryn V Dalrymple1,Clare Wall4,Shahina Begum1, Jaijus Pallippadan Johny3, Wayne S Cutfield5, Robyn North5, Lesley M E McCowan3, Keith M Godfrey6, Edwin AMitchell2,Lucilla Poston1, on behalf of the SCOPE Consortium.

1Department of Women and Children’s Health,School of Life Course Sciences, King’s College London, UK. 2Department of Paediatrics, Child & Youth Health, Faculty of Medical and health Sciences, University of Auckland, New Zealand. 3Department of Obstetrics and Gynaecology, Faculty of Medical and Health Science, University of Auckland, New Zealand. 4Department of Nutrition, School of Medical Sciences, University of Auckland, New Zealand. 5Liggins Institute, University of Auckland. 5Department of General Medicine, Auckland City Hospital, Auckland New Zealand. 6MRC Lifecourse Epidemiology Unit and NIHR Southampton Biomedical Research Centre, University of Southampton and University Hospital Southampton NHS Foundation Trust, UK.

**Address for correspondence:** Dr Angela C Flynn: angela.flynn@kcl.ac.uk; Department of Women and Children’s Health, King’s College London, 10th floor North Wing St Thomas’ Hospital, London SE1 7EH.

**Short title:** Dietary patterns in Children of Scope

**Keywords:** dietary patterns, body composition, pregnancy, childhood obesity

# **Abstract**

Dietary patterns describe the quantity, variety, or combination of different foods and beverages in a diet and the frequency of habitual consumption. Better understanding of childhood dietary patterns and antenatal influences could inform intervention strategies to prevent childhood obesity. We derived empirical dietary patterns in 1142 children (average age 6.0 (0.2) years) in Auckland, New Zealand whose mothers had participated in the Screening for Pregnancy Endpoints (SCOPE) cohort study and explored associations with measures of body composition. Participants (Children of SCOPE) had their diet assessed by food frequency questionnaire (FFQ) and empirical dietary patterns were extracted using factor analysis. Three distinct dietary patterns were identified; ‘Healthy’, ‘Traditional’ and ‘Junk’. Associations between dietary patterns and measures of childhood body composition (waist, hip, arm circumferences, body mass index (BMI), bioelectrical impedance analysis (BIA) derived body fat percentage, and sum of skinfold thicknesses (SST)) were assessed by linear regression, with adjustment for maternal influences. Children who had higher ‘Junk’ dietary pattern scores had 0.24cm greater arm (0.08 SD (95%CI 0.04, 0.13)) and 0.44cm hip (0.05 SD (95% CI 0.01, 0.10)) circumferences, 1.13cm greater SST (0.07 SD (95%CI 0.03, 0.12)) and were more likely to be obese (OR=1.74 (95%CI 1.07, 2.82)); those with higher ‘Healthy’ pattern scores were less likely to be obese (OR=0.62 (95%CI 0.39, 1.00)). In a large mother-child cohort, a dietary pattern characterised by high sugar and fat foods was associated with greater adiposity and obesity risk in children aged 6 years, while a ‘Healthy’ dietary pattern offered some protection against obesity. Targeting unhealthy dietary patterns could inform public health strategies to reduce the prevalence of childhood obesity.

# **Introduction**

Childhood obesity impacts on both health in childhood, and adult life (1). Global estimates from the World Health Organization indicate that 18% of children and adolescents aged 5-19 years were overweight or obesein 2016 (2). Diet is a key determinant of childhood obesity (3); as dietary habits established in early childhood may track into adulthood (4,5), interventions which change dietary intake early in life have the potential to improve lifelong health. Effective intervention strategies will depend on better understanding of the specific dietary elements which contribute to obesity risk.

The assessment of dietary patterns provides a practical alternative to the more conventional measurement of isolated nutrients and dietary components*,* the limitations of which are appreciated (6). Dietary patterns can be derived by one of two approaches; theoretically, in which dietary variables are grouped according to *a priori* criteria of nutritional health, e.g. a dietary index, or empirically, where dietary variables are condensed into clusters of variables using statistical modelling and examined *a posteriori* (7). This study used the empirical method as an assessment of total diet as it is more likely to be informative when addressing dietary risk factors for childhood obesity than diet indexes which focus on selected foods or nutrients.

Despite being widely explored in adult cohorts (8), relationships between empirical dietary patterns and childhood obesity risk have seldom been studied (9,10), especially in the context of contemporary mother-child cohorts. Ideally, associations between childhood dietary patterns and obesity are best examined in studies with information on the potentially confounding effects of maternal obesity, gestational diabetes and the mother’s own dietary patterns (11,12), and have potential to inform the development of complex interventions for mother and child.

Here we describe a study in which we have determined empirical dietary patterns of 6-year-old children from the Children of SCOPE (CoS) study. Their mothers were participants in the Auckland New Zealand centre of the International Screening for Pregnancy Endpoints (SCOPE) study (13). The overall objective of CoS was to identify the early life modifiable determinants of obesity and insulin resistance in children at 5 to 6 years of age. We have examined relationships between dietary patterns and measures of childhood obesity as assessed by childhood body composition (waist, hip, and arm circumferences, body mass index (BMI), bioelectrical impedance analysis (BIA) derived body fat percentage, and sum of skinfold thicknesses (SST)) with the aim of informing new targets for intervention. Associations of maternal influences on childhood dietary patterns were also examined and taken into account as potential confounders for associations between childhood diet and adiposity.

# **Methods**

## *Children of SCOPE*

Children of SCOPE is a follow-up study of the offspring of women who participated in the Auckland arm of the Screening for Pregnancy Endpoints cohort study. SCOPE is a prospective international cohort study which aimed to identify factors in early pregnancy that predicted women at high risk of developing pre-eclampsia, spontaneous preterm birth and fetal growth restriction (13). Nulliparous women with a singleton pregnancy were recruited at 15 weeks’ gestation and a second research visit carried out at 20 weeks’ gestation; detailed demographic and clinical data were recorded at each visit and at delivery, and for any pregnancy complications. Details of the SCOPE study have been previously described (13).For a child to be eligible to participate in CoS, the mother agreed to be contacted about the follow-up study, and the child was aged between 5 and 6.25 years. Children with major health problems that might affect growth were excluded.

From the 2,032 mothers included in the Auckland SCOPE cohort, 1,208 (59%) mothers and their children were recruited. We previously reported no difference in maternal BMI between participants in CoS and the original complete Auckland cohort, however, the mothers in CoS were more likely to be older, White, have a higher educational attainment and higher socio-economic status, were less likely to smoke in their first trimester and were less likely to have delivered before 37 weeks (14). Of the 1,208 mother-child dyads, 35 were excluded due to missing or implausible percentage body fat. The final study population included 1,173 mother-child dyads and a total of 1142 children had information on dietary intake collected to extract dietary patterns. All women provided consent for participation of their children. Ethical approval was granted by the Auckland Ethics Committee (AKX/02/00/364).

## *Dietary assessment and dietary patterns*

At the follow-up visit, a research nurse asked the child’s main caregiver to complete a Food Frequency Questionnaire (FFQ) about the child’s diet. This was based on an FFQ developed for the New Zealand Children's National Nutrition Survey (15) and requests information on the consumption of 118 food and beverage items. Accompanying this list was a multiple response grid which estimated frequency of consumption of the food and beverages in the preceding month, ranging from ‘never or less than once per month to ‘2 or more times per day’. Each food and beverage item had a standard serving size (e.g., slice of bread, apple). Where individual food items were unanswered, we imputed data using the MICE methodology. We used 10 burn-ins, followed by the construction of 10 imputed datasets. Data was imputed if less than 20% of items were missing.

Factor analysis with varimax rotation was performed to derive dietary patterns in each of the imputed datasets. The number of factors was chosen using the scree plot of eigenvalues and the interpretability of the factor loadings. Foods with a factor loading of ≥ ± 0.30 were considered to have a strong association with that factor. We then averaged factor loading for the dietary patterns across the imputed dataset. These factor loadings were then applied across an averaged imputed dataset constructed by averaging the responses across the imputed datasets. Sensitivity analyses were carried out to assess the MICE methodology to simple imputation of making an assumption that missed items were not answered because they were not consumed.

## *Childhood body composition*

At the follow up visit, the child’s body composition was assessed using anthropometric and BIA measurements. Arm circumference (cm) was measured at the mid-point between the edge of the shoulder bone and bony tip of the right elbow. Waist circumference (cm) was assessed at the mid-point between the lowest rib and top of the hip. Hip circumference (cm) was taken as the distance around the maximal diameter of buttocks*.* For estimation of BMI (kg/m2), weight (kg) was measured in light clothing without shoes to the nearest 0.1kg and height (cm) assessed in a standing position with a stadiometer. Childhood obesity was defined as BMI z-score>2SD according to WHO’s child growth standards (16). Body fat percentage was determined by BIA (ImpediMed SFB7 tetra polar bioimpedance spectroscopy). Fat free mass (FFM) was calculated using: FFM = 0.65 (height2/impedance) + 0.686 \* age + 0.15 (17). Body fat percentage was estimated using: (weight-FFM)/weight\*100. Subscapular, triceps, biceps, and suprailiac skinfold thicknesses (mm) were measured using Holtain skinfold callipers and summed to give sum of skinfold thicknesses. All measurements were taken in triplicate and mean values calculated.

## *Covariates*

In the SCOPE study, maternal demographic, clinical and lifestyle data were recorded. This included maternal age, BMI (kg/m2) at 15 weeks’ gestation, ethnicity, socioeconomic status [New Zealand Socio-economic Index 1996, education (years)], alcohol use pre-pregnancy, smoking during the 1st trimester, multivitamin use pre-pregnancy, maternal mood (Edinburgh Postnatal Depression Scale at 15 week visit), television (number of hours watched per day in the last month, evaluated at the 15 week visit), gestational hypertension (defined as systolic BP≥140 mmHg or diastolic BP≥90 mmHg on at least 2 occasions after the 20 week visit and before onset of labour), gestational diabetes mellitus diagnosis (any of :1) 75g OGTT fasting glucose ≥5.5 mmol/L and/or 2 hour ≥9.0 mmol/L, or 2) Polycose 1h glucose >11.0 mmol/L and no GTT available, or 3) Fasting Glucose ≥5.5 mmol/L and no polycose or GTT available, or 4) Random Glucose >11 mmol/L and no polycose, GTT or fasting glucose), biomarkers measured from maternal blood samples including placental growth factor (PlGF). At the time of birth, data were collected on premature birth, birthweight (grams)*,* gestational age (weeks), child's age and sex and breastfeeding (exclusive breastfeeding at discharge form obstetric unit).

## *Statistical analysis*

Descriptive data are presented as mean (standard deviation), frequency (percentage) or median (interquartile range) as appropriate. Multiple regression analysis was carried out to examine the associations between maternal influences (including maternal BMI, ethnicity, cigarette smoking in the first trimester, multivitamin use pre-pregnancy, breastfeeding, socio-economic status, education and age (12,18) and children’s dietary patterns. To investigate the relationship between childhood dietary patterns and body composition, linear regression was used. Two models were generated; model 1 where simple linear regression with no adjustment for confounding variables was performed and model 2, multiple linear regression including adjustment for confounders. Confounders were chosen *a priori* based on known associations with obesity (14,19,28,29,20–27). The maternal and early post-natal exposures used in the adjusted model were maternal BMI, ethnicity, socioeconomic status, multivitamin use pre-pregnancy, alcohol intake pre-pregnancy, cigarette smoking in first trimester, time spent watching television in first trimester, depression in first trimester, gestational hypertension, GDM diagnosis, maternal plasma PlGF at 15 weeks’ gestation, breastfeeding, and infant sex. Standardised β-coefficients are presented which represent the SD in body composition measure per SD change in dietary pattern score, enabling comparison across outcomes. Data were analysed using Stata software, version 15.0 (StataCorp, College Station, Texas).

# **Results**

## *Characteristics of the study population*

## The characteristics of the women and children are presented in **Table 1**. At 15 weeks’ gestation, the average age of the mothers was 31.0 (SD 4.4) years and the median BMI was 23.9 (21.8-26.6) kg/m2. The majority of women were of White ethnicity (87.6%). The average age of the children at the follow up visit was 6.0 (0.2) years. A total of 176 (15.1%) children were overweight or obese according to WHO standards.

## *Dietary patterns*

1142 children had complete data in the imputed datasets. Factor analysis identified three distinct dietary patterns at age 6 years; for ease of description we have termed these as healthy, traditional and junk in line with other previously published studies (30). The factor loadings for the dietary patterns are shown in **Table S1.** The ‘Healthy’ diet was characterised by high loadings including a number of fruits (berries, stone fruits, feijoas/kiwifruit, pineapple) and vegetables (avocado, tomato, green leafy vegetables, celery, cucumber, green beans, pepper). The ‘Traditional’ diet was based on a number of meat (meat and poultry in casseroles, roasted as part of a dish) and carbohydrate (potato, taro, kumara, and pumpkin either steamed, boiled or roasted). The ‘Junk’ diet was characterised by high loadings on candy bars, potato crisps, lollies or sweets, sausage rolls, and bought cake or muffin **(Table 2)**. A number of other items in the Junk dietary pattern had loadings between 0.25 and 0.30 but were not consistently above 0.3 across factor analyses of the imputed datasets.

In sensitivity analyses, a factor analysis using data imputed by replacing missing data with never, resulted in very similar dietary patterns, and loadings. Examination of the imputed dataset showed that in general a large proportion of missing values on individual items were imputed as never.

## *Associations between maternal influences and dietary patterns*

The dietary patterns had differing associations with maternal influences, which were robust to adjustment **(Table 3)**. Children who had higher ‘Healthy’ dietary pattern scores were more likely to have mothers who had a lower early pregnancy BMI, of Indian ethnicity (and less likely to be Maori), who had a higher level of educational attainment and who took multivitamins pre-pregnancy (all *P*<0.05). Children who had higher ‘Traditional’ dietary pattern scores were more likely to have mothers of Pacific Islander ethnicity, who had a lower socioeconomic status were younger (all *P*<0.05). Children who had higher ‘Junk’ dietary pattern scores were more likely to have mothers of Indian or Pacific Islander ethnicity, that smoked in the first trimester and had a lower educational attainment (all *P*<0.05).

## *Associations between children’s dietary patterns and body composition*

The association between children’s dietary patterns and body composition at aged 6 years is shown in **Table 4.** Children who had higher ‘Healthy’ pattern scores were less likely to be obese according to WHO child growth standards in Models 1 and 2 (adjusted OR=0.62; 95% CI 0.39, 1.00).

Children who had higher ‘Junk’ pattern scores had greater arm (adjusted (a)β=0.08; 95%CI 0.04, 0.13) and hip (aβ=0.05; 95%CI 0.01, 0.10) circumferences, and SST (aβ=0.07; 95%CI 0.03, 0.12).Furthermore, children who had higher ‘Junk’ pattern scores were more likely to be obese (OR=1.74; 95%CI 1.07, 2.82). These associations were robust to adjustments for confounders.

Children who had higher ‘Traditional’ dietary pattern scores had greater arm, waist and hip circumferences, body fat percentage and BMI in the univariable analysis. These associations were not robust to adjustments for confounders.

**Discussion**

In this large contemporary mother and child cohort, we found one distinct empirical childhood dietary pattern to be associated with greater adiposity and obesity risk in children aged 6 years. This pattern was characterised by high intakes of foods and beverages high in sugar and/or fat including saturated fat. A healthy dietary pattern characterised by a number of fruits and vegetables was associated with a reduced risk of obesity in a small number of children. Furthermore, we were able to characterise influences of maternal antenatal exposures on childhood dietary patterns which included maternal ethnicity, age, BMI, education, socioeconomic status and smoking in pregnancy.

The majority of reports of childhood dietary patterns have addressed the hypothesis that dietary behaviours in early childhood can influence the risk of obesity in later childhood. Several studies have shown a strong relationship between unhealthy dietary patterns characterised by energy dense, low fibre and high fat foods and their later risk of obesity (31,32). Few studies have examined the relationship between empirical dietary patterns and childhood body composition measured simultaneously. In one of the few to have specifically addressed this association, the Lifeways Cross- Generational Cohort study in Irish children aged 5 years showed that theupper tertiles of a "cereal and juice" dietary pattern and the middle tertile of a ‘pasta and veg’ dietary pattern were negatively associated with overweight and obesity (33). Our findings, together with observations that diet in early childhood is a risk for later obesity may reflect unchanging dietary patterns throughout early and mid-childhood (4), suggesting that interventions which specifically target foods and snacks high in sugar and fat in early childhood could play a role in reducing later risk of obesity. The observation that the relationship between the Junk and Healthy patterns and BMI observed in this study was only evident in children classified as obese is particularly relevant to public health measures for the prevention of childhood obesity.

The three dietary patterns identified in this study (‘Traditional’, ‘Junk’ and ‘Healthy’) are consistent with those described in the New Zealand birth cohort study which assessed dietary patterns in Auckland children aged 3.5 and 7 years (30). Similarities were observed for high factor loadings for the ‘traditional’ diet of meat, vegetables and potatoes, the ‘healthy’ diet of fruit, vegetables and nuts, and the junk diet of confectionary.

We identified important associations between maternal influences and childhood dietary patterns. Better quality diets were more common in children who had mothers with higher educational attainment, and who had a lower BMI, similar to observations in the Southampton Women’s Survey (12,34). Consistent with ALSPAC data, poorer diets were more common in children who were born to mothers who had lower educational attainment (18). Furthermore, the mothers of children with poorer diets were more likely to smoke in pregnancy, which is similar to the New Zealand birth cohort study (30). Recently, we reported on the relationship between maternal BMI and childhood obesity risk in the Children of SCOPE cohort. Maternal BMI was independently associated with childhood BMI z-score, sum of skinfolds and percentage body fat (all *P*<0.001) (14). The global increase in obesity, which is reflected in women who present for antenatal care has led to the concern that obesity in pregnancy may contribute to childhood obesity risk, with evidence from observational studies to support this link (35). Exposure *in utero* to the metabolic consequences of a high maternal BMI including dyslipidaemia, inflammatory markers, hyperinsulinaemia and hyperleptinaemia appears to influence offspring obesity risk (36,37). This highlights the need for public health interventions to promote a healthy BMI and better quality diet in reproductive aged women.

Strengths of the current study include this being an addition to a very sparse literature which has defined the dietary patterns associated with childhood obesity. Whilst the assumption is often made that foods and snacks high in sugar and fat are major contributors, there is limited evidence to support this widely held assumption in paediatric cohorts. Furthermore, our observations have been made in a large cohort, compared to previous studies of childhood obesity in mother child cohorts. By focusing on obesity in the children, the Children of SCOPE protocol prospectively included detailed measurements of childhood adiposity and a comprehensive dataset to examine maternal influences on childhood diet. By characterising dietary patterns, we have been able to capture the complexity of the diet while overcoming the limitations of single nutrient assessment (6).

Whilst this study provides evidence that specific dietary patterns in children are linked to childhood obesity, there are some limitations. At follow-up, data from 1,208 (59%) of the 2,032 mothers and their children from the original Auckland arm of the SCOPE cohort were available, which could lead to erroneous conclusions if the relationships between dietary patterns and childhood body composition differ between those included in follow up and those not. Being observational in design, we are only able to examine associations and not infer causation between dietary patterns and childhood obesity development. We assessed dietary intake using an FFQ, which may be prone to bias (38). Factor analysis was used to derive empirical dietary patterns which involves several subjective decisions including the number of factors to extract, rotation method and naming of the factors (39).We adjusted for several potentially confounding variables in the assessment of the relationship between maternal influences and childhood dietary patterns and between childhood dietary patterns and obesity. However, the possibility of residual confounding due to unmeasured lifestyle factors cannot be excluded. The relationship between physical activity and inactivity was not assessed in the present study. WHO guidelines on childhood obesity prevention include promoting physical activity among children in the first few years of life in addition to focusing on dietary intake (40). Evidence from cross-sectional studies examining the association between physical activity in children and adiposity suggest that higher levels of physical activity are related to reduced adiposity (41). Associations with sedentary time are less consistent (42). Future studies should include physical activity in assessing the relationship between childhood health behaviours and obesity risk. Finally, the majority of women in this study were of European descent and were well educated and were not a representative cohort of New Zealand women and children.

# **Conclusions**

This mother-child cohort study with multiple measures of childhood body composition provides support for a relationship between empirical dietary patterns representing overall diet and childhood adiposity. A dietary pattern characterised by consumption of high sugar and fat including saturated fat foods was associated with greater body circumferences, sum of skinfolds, and obesity risk in children aged 6 years, while a “Healthy” dietary pattern offered some apparent protection against obesity. We also identified a number of maternal exposures that were associated with eating patterns in early childhood providing insights into important maternal influences on children’s dietary intake. Our data adds to the growing evidence that targeting maternal obesity and unhealthy dietary patterns associated with early childhood adiposity could inform public health strategies to reduce the prevalence of childhood obesity.

## **Acknowledgements and financial support**

SCOPE funding: University of Auckland, the New Enterprise Research Fund, Foundation for Research Science and Technology, Health Research Council of New Zealand, Evelyn Bond Fund, Auckland District Health Board Charitable Trust.

Children of SCOPE funding: Health Research Council of New Zealand and Cure Kids. EAM and JMDT were supported by Cure Kids.

We are grateful to the women and children who participated in the Children of SCOPE study. We thank the SCOPE Project Manager Rennae Taylor and research assistants (Noleen Van Zyl, Elin Granrud, and Desley Minahan) for their contributions throughout the study. KMG is supported by the UK Medical Research Council (MC\_UU\_12011/4), the National Institute for Health Research (NIHR Senior Investigator (NF-SI-0515-10042), NIHR Southampton 1000DaysPlus Global Nutrition Research Group) and NIHR Southampton Biomedical Research Centre), the European Union (Erasmus+ Programme Early Nutrition eAcademy Southeast Asia-573651-EPP-1-2016-1-DE-EPPKA2-CBHE-JP), the US National Institute On Aging of the National Institutes of Health (Award No. U24AG047867) and the UK ESRC and BBSRC (Award No. ES/M00919X/1). LP is supported by the Biomedical Research Centre at Guy’s and St. Thomas’ NHS Foundation Trust and King’s College London and Tommy’s Charity. The funders had no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

## **Conflict of Interest**

LP is part of an academic consortium that has received research funding from Abbott Nutrition and Danone. KMG reports reimbursement from Nestle Nutrition Institute, has a patent Phenotype prediction issued, a patent Predictive use of CpG methylation issued, a patent Maternal Nutrition Composition pending, a patent Vitamin B6 in maternal administration for the prevention of overweight or obesity in the offspring issued, and is part of an academic consortium that with WSC has received research funding from Abbott Nutrition, Nestec and Danone. CW has received funding for an investigator initiated trial from Danone Nutricia. The other authors declare no conflict of interest.

## **Authorship**

AF, SB, JMDT, JPJ analysed and interpreted the data. EAM directed the Children of Scope Study. CW, RN were involved in data collection, investigation and analysis. AF, KVD completed additional analyses for the data. AF, KVD, LP had overall responsibility for the manuscript. All authors have read and approved the ﬁnal manuscript.

# **References**

1. Reilly JJ, Kelly J (2011) Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: Systematic review. *Int J Obes* **35**, 891–8.

2. World Health Organization. Report of the Commission on Ending Childhood Obesity. Geneva 2016. https://www.who.int/gho/ncd/risk\_factors/overweight\_obesity/overweight\_adolescents/en/. Accessed March 2019.

3. Lobstein T, Baur L, Uauy R (2004) Obesity in children and young people: a crisis in public health. *Obes Rev* **5**, 4–85.

4. Northstone K, Emmett PM (2008) Are dietary patterns stable throughout early and mid-childhood? A birth cohort study. *Br J Nutr* **100**, 1069–76.

5. Mikkilä V, Räsänen L, Raitakari OT *et al.* (2005) Consistent dietary patterns identified from childhood to adulthood: the cardiovascular risk in Young Finns Study. *Br J Nutr* **93**, 923–31.

6. Hu FB (2002) Dietary pattern analysis: A new direction in nutritional epidemiology. Curr *Opin Lipidol* **13**, 3–9.

7. Newby PK, Tucker KL (2004) Empirically derived eating patterns using factor or cluster analysis: a review. *Nutr Rev* **62**, 177–203.

8. Rezagholizadeh F, Djafarian K, Khosravi S *et al.* (2017) A posteriori healthy dietary patterns may decrease the risk of central obesity: findings from a systematic review and meta-analysis. *Nutr Res* **41**, 1–13.

9. Ambrosini GL (2014) Childhood dietary patterns and later obesity: A review of the evidence. *Proc Nutr Soc* **73**, 137–46.

10. Rocha NP, Milagres LC, Longo GZ *et al.* (2017) Association between dietary pattern and cardiometabolic risk in children and adolescents: a systematic review. *J Pediatr* **93**, 214–22.

11. Logan KM, Gale C, Hyde MJ *et al.* (2017) Diabetes in pregnancy and infant adiposity: systematic review and meta-analysis. *Arch Dis Child Fetal Neonatal Ed* **102**, F65–72.

12. Fisk CM, Crozier SR, Inskip HM *et al.* (2011) Influences on the quality of young children’s diets: the importance of maternal food choices. *Br J Nutr* **105**, 287–96.

13. Kenny LC, Black MA, Poston L *et al.* (2014) Early pregnancy prediction of preeclampsia in nulliparous women, combining clinical risk and biomarkers: The Screening for Pregnancy Endpoints (SCOPE) international cohort study. *Hypertension* **64**, 644–52.

14. Dalrymple KV, Begum S, Thompson JMD *et al.* (2019) Relationships of Maternal Body Mass Index and Plasma Biomarkers with Childhood Body Mass Index and Adiposity at 6 years; the Children of SCOPE study. *Paediatr Obes* e12537*.*

15. Metcalf PA, Scragg RKR, Sharpe S *et al.* (2003) Short-term repeatability of a food frequency questionnaire in New Zealand children aged 1-14 y. *Eur J Clin Nutr* **57**, 1498–503.

16. World Health Organization. Training Course on Child Growth Assessment: WHO Child Growth Standards. Geneva 2008. http://www.who.int/childgrowth. Accessed February 2019.

17. Schaefer F, Georgi M, Zieger A *et al.* (1994) Usefulness of bioelectric impedance and skinfold measurements in predicting fat-free mass derived from total body potassium in children. *Pediatr Res* **35**, 617–24.

18. Northstone K, Emmett P (2005) Multivariate analysis of diet in children at four and seven years of age and associations with socio-demographic characteristics. *Eur J Clin Nutr* **59**, 751–60.

19. Power ML, Schulkin J (2008) Sex differences in fat storage, fat metabolism, and the health risks from obesity: possible evolutionary origins. *Br J Nutr* **99**, 931–40.

20. Taveras EM, Gillman MW, Kleinman K et al. (2010) Racial/ethnic differences in early-life risk factors for childhood obesity. *Pediatrics* **125**, 686–95.

21. Falconer CL, Park MH, Croker H *et al.* (2014) Can the relationship between ethnicity and obesity-related behaviours among school-aged children be explained by deprivation? A cross-sectional study. *BMJ Open* **4**, e003949.

22. Szeto IMY, Das PJ, Aziz A *et al.* (2009) Multivitamin supplementation of Wistar rats during pregnancy accelerates the development of obesity in offspring fed an obesogenic diet. *Int J Obes* **33**, 364–72.

23. Patel N, Dalrymple KV, Briley AL *et al.* (2018) Mode of infant feeding, eating behaviour and anthropometry in infants at 6-months of age born to obese women - a secondary analysis of the UPBEAT trial. *BMC Pregnancy Childbirth* **18**, 355.

24. Lampard AM, Franckle RL, Davison KK (2014) Maternal depression and childhood obesity: a systematic review. *Prev Med* **59**, 60–7.

25. Mizutani T, Suzuki K, Kondo N *et al.* (2007) Association of maternal lifestyles including smoking during pregnancy with childhood obesity. *Obesity* **15**, 3133–9.

26. Leng J, Li W, Zhang S *et al.* (2015) GDM Women’s Pre-Pregnancy Overweight/Obesity and Gestational Weight Gain on Offspring Overweight Status. *PLoS One* **10**, e0129536.

27. Zheng JS, Liu H, Ong KK *et al.* (2017) Maternal Blood Pressure Rise During Pregnancy and Offspring Obesity Risk at 4 to 7 Years Old: The Jiaxing Birth Cohort. *J Clin Endocrinol Metab* **102**, 4315–22.

28. Strauss RS (1997) Effects of the intrauterine environment on childhood growth. *Br Med Bull* **53**, 81–95.

29. Pervanidou P, Chouliaras G, Akalestos A *et al.* (2014) Increased placental growth factor (PlGF) concentrations in children and adolescents with obesity and the metabolic syndrome. *Hormones* **13**, 369–74.

30. Wall CR, Thompson JMD, Robinson E *et al.* (2013) Dietary patterns of children at 3.5 and 7 years of age: a New Zealand birth cohort study. *Acta Paediatr* **102**, 137–42.

31. Johnson L, Mander AP, Jones LR *et al.* (2008) Energy-dense, low-fiber, high-fat dietary pattern is associated with increased fatness in childhood. *Am J Clin Nutr* **87**, 846–54.

32. Ambrosini GL, Emmett PM, Northstone K *et al.* (2012) Identification of a dietary pattern prospectively associated with increased adiposity during childhood and adolescence. *Int J Obes* **36**, 1299–305.

33. Murrin CM, Heinen MM, Kelleher CC (2015) Are Dietary Patterns of Mothers during Pregnancy Related to Children’s Weight Status? Evidence from the Lifeways Cross- Generational Cohort Study. *AIMS public Heal* **2**, 274–96.

34. Okubo H, Crozier SR, Harvey NC *et al.* (2015) Diet quality across early childhood and adiposity at 6 years: the Southampton Women’s Survey. *Int J Obes* **39**, 1456–62.

35. Heslehurst N, Vieira R, Akhter Z *et al.* (2019) The association between maternal body mass index and child obesity: A systematic review and meta-analysis. *PLoS Med* **16**, 1–20.

36. Catalano PM, Farrell K, Thomas A *et al.* (2009) Perinatal risk factors for childhood obesity and metabolic dysregulation. *Am J Clin Nutr* **90***,* 1303–13.

37. Nelson SM, Matthews P, Poston L (2009) Maternal metabolism and obesity: Modifiable determinants of pregnancy outcome. *Hum Reprod Update* **16,** 255–75.

38. Serdula MK, Alexander MP, Scanlon KS *et al.* (2002) What Are Preschool Children Eating? A Review of Dietary Assessment. *Annu Rev Nutr* **21**, 475–98.

39. Martínez ME, Marshall JR, Sechrest L (1998) Invited commentary: Factor analysis and the search for objectivity. *Am J Epidemiol* **148**, 17–9.

40. World Health Organization. Interim report of the commission on ending childhood obesity. Geneva 2015. https://www.who.int/end-childhood-obesity/publications/echo-report/en/. Accessed December 2019.

41. Jimenez-Pavon D, Kelly JRJ (2010) Associations between objectively measured habitual physical activity and adiposity in children and adolescents: Systematic review. I*nt J Pediatr Obes* **5**, 3–18.

42. Ekelund U, Hildebrand M, Collings PJ (2014) Physical activity, sedentary time and adiposity during the first two decades of life. *Proc Nutr Soc* **73**, 319–29.

**Table 1 Maternal and child characteristics in SCOPE and the Children of SCOPE studies (n=1142)**

|  |  |  |
| --- | --- | --- |
| **Maternal characteristics (15 weeks' gestation)** |  | **Mean (SD), median (IQR) or number (%)** |
| **Age (years)** |  | 31.0 (4.4) |
| **BMI (kg/m2)** |  | 23.9 (21.8-26.6) |
| **BMI (categorical)** |  |  |
| < 18.5 |  | 8 (0.7%) |
| 18.5 to 24.9 |  | 698 (61.2%) |
| 25.0 to 29.9 |  | 321 (28.1%) |
| ≥30 |  | 113 (9.9%) |
| **Ethnicity** |  |  |
| White |  | 1001 (87.6%) |
| Asian |  | 44 (3.9%) |
| Indian |  | 36 (3.2%) |
| Māori |  | 34 (2.9%) |
| Other |  | 16 (1.4%) |
| Pacific |  | 11 (0.9%) |
| **Socio-economic status (NZSEI)** |  |  |
| High (56-90) |  | 315 (27.6%) |
| Medium (32-55) |  | 677 (59.3%) |
| Low (10-31) |  | 150 (13.1%) |
| **Education (years)** |  |  |
| <12 |  | 65 (5.7%) |
| 12-13 |  | 1058 (92.6%) |
| >13 |  | 19 (1.6%) |
| **Smoking in first trimester** |  | 107 (9.4%) |
| **Alcohol intake pre-pregnancy** |  | 631 (54.5%) |
| **Multivitamin use pre-pregnancy** |  | 377 (33.3%) |
| **Gestational diabetes** |  | 18 (1.7%) |
| **Gestational hypertension** |  | 61 (5.3%) |
| **TV (hours watched per day)** |  |  |
| <2 |  | 483 (42.4%) |
| 2 - 4 |  | 586 (51.5%) |
| >4 |  | 70 (6.2%) |
| **Characteristics at follow up** |  |  |
| **Maternal BMI (categorical)** |  |  |
| < 18.5 |  | 23 (2.0%) |
| 18.5 to 24.9 |  | 622 (54.5%) |
| 25.0 to 29.9 |  | 327 (28.7%) |
| ≥30 |  | 169 (14.8%) |
| **Child's age** |  | 6.0 (0.2) |
| **Child's sex (female)** |  | 578 (50.6%) |
| **Child’s height (cm)** |  | 117.9 (4.9) |
| **Child’s weight (kg)** |  | 21.7 (20.0-23.7) |
| **Child's BMI (z-score)** |  | 0.28 (0.91) |
| **Child overweight or obese (BMI z-score ≥1SD)\*** |  | 176 (15.4) |
| **Child overweight (BMI z-score ≥1SD, <2SD)** |  | 145 (12.7) |
| **Child obese (BMI z-score ≥2SD)** |  | 31 (2.71) |
| **Child’s percentage body fat** |  | 22.8 (6.3) |
| **Child’s arm circumference (cm)** |  | 18.6 (1.7) |
| **Child’s waist circumference (cm)** |  | 55.4 (4.0) |
| **Child’s hip circumference (cm)** |  | 61.6 (4.9) |
| **Child’s sum of skinfolds (mm)** |  | 30.5 (9.3) |

Abbreviations: SES - socio-economic status, NZSEI - New Zealand Socio-Economic Index, BMI - body mass index, \*World Health Organisation (2007)

**Table 2 Dietary patterns identified using factor analysis**

|  |  |  |  |
| --- | --- | --- | --- |
| **Variable** | **Healthy** | **Traditional** | **Junk** |
| **Avocado** | **0.45** | 0.03 | -0.09 |
| **Green beans** | **0.34** | 0.15 | -0.07 |
| **Berries** | **0.49** | -0.13 | -0.02 |
| **Cake/muffin/bun** | 0.15 | 0.05 | **0.32** |
| **Brown rice** | **0.36** | 0.03 | -0.09 |
| **Candy bars** | 0.05 | 0.10 | **0.46** |
| **Celery** | **0.38** | 0.11 | 0.01 |
| **Cucumber** | **0.34** | -0.06 | -0.03 |
| **Dried fruit** | **0.32** | 0.06 | -0.02 |
| **Feijoas/kiwifruit** | **0.38** | 0.12 | 0.02 |
| **Fish fillets not in breadcrumb/batter** | **0.31** | 0.11 | -0.03 |
| **Green leafy vegetables** | **0.41** | 0.25 | -0.08 |
| **Kumera/pumpkin roasted** | 0.18 | **0.42** | 0.01 |
| **Kumera/pumpkin steamed or boiled** | 0.19 | **0.37** | -0.03 |
| **Lentils/chickpeas/other beans** | **0.37** | 0.09 | -0.12 |
| **Lollies/sweets** | -0.03 | -0.09 | **0.31** |
| **Meat as part of dish (beef/pork/lamb)** | 0.05 | **0.36** | 0.02 |
| **Meat casseroles (beef,pork,lamb)** | 0.08 | **0.45** | 0.04 |
| **Meat roasted (beef/pork/lamb)** | 0.02 | **0.42** | 0.12 |
| **Mixed vegetables** | 0.18 | **0.40** | 0.09 |
| **Nuts** | **0.33** | -0.01 | -0.02 |
| **Other fruit** | **0.33** | 0.11 | -0.02 |
| **Pepper** | **0.34** | 0.05 | -0.05 |
| **Pineapple** | **0.37** | 0.09 | 0.08 |
| **Potato/other crisps** | -0.17 | 0.06 | **0.34** |
| **Potato/taro roasted** | 0.09 | **0.34** | 0.08 |
| **Potato/taro steamed or boiled** | 0.03 | **0.36** | 0.01 |
| **Poultry as part of dish** | 0.11 | **0.38** | 0.02 |
| **Poultry casseroles** | 0.15 | **0.44** | 0.07 |
| **Poultry roasted** | 0.01 | **0.32** | 0.11 |
| **Sausage rolls** | 0.07 | 0.19 | **0.30** |
| **Stone fruit** | **0.45** | -0.06 | 0.01 |
| **Tomato** | **0.43** | 0.00 | -0.03 |
| **Yoghurt** | **0.31** | 0.02 | -0.01 |

Factor loadings ≥± 0.3 for each identified dietary pattern

**Table 3 Multiple regression analysis of the maternal influences associated with child’s dietary patterns at 6 years (n=1142).**

|  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Characteristics | Adjusted coefficient (β) (95% CI) | *P* | Adjusted coefficient (β) (95% CI) | *P* | |  | Adjusted coefficient (β) (95% CI) | | *P* |  |
|  | **Healthy** |  | **Traditional** |  | |  | **Junk** | |  |  |
| Early-pregnancy BMI (kg/m2) | **-0.03 (-0.04 to -0.02)** | **<0.001** | 0.01 (-0.001 to 0.02) | | 0.071 |  | 0.01 (-0.003 to 0.02) | 0.140 | |  |
| Ethnicity |  |  |  |  | |  |  | |  |  |
| White | Reference | **0.002** | Reference | **<0.001** | |  | Reference | | **0.002** |  |
| Asian | -0.10 (-0.36 to 0.15) |  | 0.14 (-0.10 to 0.37) |  | |  | 0.002 (-0.23 to 0.23) | |  |  |
| Indian | **0.32 (0.04 to 0.61)** |  | -0.05 (-0.31 to 0.21) |  | |  | **0.28 (0.03 to 0.54)** | |  |  |
| Maori | -**0.32 (-0.61 to -0.02)** |  | 0.22 (-0.05 to 0.48) |  | |  | 0.12 (-0.14 to 0.38) | |  |  |
| Other | 0.64 (0.21 to 1.07) |  | 0.60 (0.20 to 0.99) |  | |  | 0.47 (0.08 to 0.85) | |  |  |
| Pacific Islander | -0.21 (-0.73 to 0.30) |  | **0.79 (0.32 to 1.26)** |  | |  | **0.70 (0.24 to 1.16)** | |  |  |
| Cigarette smoking in first trimester | 0.10 (-0.08 to 0.27) | 0.274 | 0.01 (-0.15 to 0.17) | 0.942 | |  | **0.25 (0.09 to 0.41)** | | **0.002** |  |
| Multivitamin use pre-pregnancy | **0.13 (0.02 to 0.24)** | **0.016** | -0.09 (-0.19 to 0.01) | 0.064 | |  | -0.01 (-0.10 to 0.08) | | 0.833 |  |
| Breastfeeding | 0.10 (-0.02 to 0.22) | 0.098 | -0.02 (-0.13 to 0.08) | 0.688 | |  | 0.01 (-0.10 to 0.11) | | 0.920 |  |
| Socio-economic status |  |  |  |  | |  |  | |  |  |
| High | -0.02 (-0.13 to 0.10) | 0.311 | **-0.21 (-0.31 to -0.10)** | **<0.001** | |  | 0.02 (-0.08 to 0.12) | | 0.290 |  |
| Medium | Reference |  | Reference |  | |  | Reference | |  |  |
| Low | -0.12 (-0.28 to 0.04) |  | 0.09 (-0.05 to 0.24) |  | |  | 0.11 (-0.03 to 0.26) | |  |  |
| Education (years) | **0.12 (0.04 to 0.20)** | **0.002** | -0.04 (-0.11 to 0.03) | 0.225 | |  | **-0.09 (-0.16 to -0.02)** | | **0.010** |  |
| Age (years) | 0.01 (-0.01 to 0.02) | 0.363 | **-0.02 (-0.03 to -0.01)** | **<0.001** | |  | 0.004 (-0.01 to 0.01) | | 0.496 |  |

All of the maternal characteristics have been adjusted for each other in the multivariate analysis.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Table 4 Multiple regression analysis of the dietary patterns associated with body composition at age 6. | | | | | | |
|  | **Model 1 (unadjusted)** | | | **Model 2 (adjusted)** | | |
| Body composition measure | **n** | **Standardised coefficient (β)/odds ratioc (95% CI)** | ***P*** | **n** | **Standardised coefficient (β)/ odds**  **ratioc (95% CI)** | ***P*** |
| Healthy pattern |  |  |  |  |  |  |
| Percentage body fat | 1142 | -0.04 (-0.09 to 0.01) | 0.101 | 1025 | -0.02 (-0.07 to 0.04) | 0.482 |
| BMI z-scorea | 1142 | -0.05 (-0.11 to 0.002) | 0.060 | 1025 | -0.01 (-0.07 to 0.05) | 0.677 |
| Arm (cm) | 1142 | -0.02 (-0.07 to 0.03) | 0.417 | 1025 | 0.01 (-0.04 to 0.06) | 0.728 |
| Waist (cm) | 1142 | 0.001 (-0.05 to 0.05) | 0.966 | 1025 | 0.02 (-0.03 to 0.08) | 0.362 |
| Hip (cm) | 1142 | -0.02 (-0.07 to 0.03) | 0.370 | 1025 | 0.01 (-0.04 to 0.06) | 0.711 |
| Sum of skinfolds (mm) b | 1133 | -0.04 (-0.09 to 0.01) | 0.120 | 1019 | -0.03 (-0.08 to 0.03) | 0.327 |
| Overweight or obese (BMI z-score >1SD) c | 1142 | **0.76 (0.63 to 0.92)** | **0.005** | 1025 | 0.85 (0.69 to 1.05) | 0.125 |
| Obese (BMI z-score >2SD) c | 1142 | **0.52c (0.33 to 0.80)** | **0.003** | 971 | **0.62c (0.39 to 1.00)** | **0.050** |
|  |  |  |  |  |  |  |
| Traditional pattern |  |  |  |  |  |  |
| Percentage body fat | 1142 | **0.06 (0.02 to 0.11)** | **0.006** | 1025 | 0.04 (-0.01 to 0.09) | 0.097 |
| BMI z-scorea | 1142 | **0.08 (0.03 to 0.13)** | **0.003** | 1025 | 0.04 (-0.02 to 0.09) | 0.176 |
| Arm (cm) | 1142 | **0.05 (0.002 to 0.09)** | **0.042** | 1025 | 0.02 (-0.03 to 0.07) | 0.479 |
| Waist (cm) | 1142 | **0.07 (0.02 to 0.12)** | **0.003** | 1025 | 0.05 (-0.002 to 0.09) | 0.060 |
| Hip (cm) | 1142 | **0.06 (0.01 to 0.11)** | **0.011** | 1025 | 0.02 (-0.03 to 0.07) | 0.396 |
| Sum of skinfolds (mm) b | 1133 | 0.03 (-0.02 to 0.08) | 0.220 | 1019 | 0.01 (-0.04 to 0.06) | 0.679 |
| Overweight or obese (BMI z-score >1SD) c | 1142 | **1.34 (1.09 to 1.64)** | **0.006** | 1025 | 1.19 (0.95 to 1.50) | 0.135 |
| Obese (BMI z-score >2SD) c | 1142 | 0.93**c** (0.59 to 1.45) | 0.740 | 971 | 0.89**c** (0.53 t0 1.49) | 0.660 |
|  |  |  |  |  |  |  |
| Junk pattern |  |  |  |  |  |  |
| Percentage body fat | 1142 | **0.04 (0.0002 to 0.09)** | **0.049** | 1025 | 0.02 (-0.03 to 0.07) | 0.486 |
| BMI z-scorea | 1142 | **0.05 (0.004 to 0.10)** | **0.034** | 1025 | 0.02 (-0.03 to 0.08) | 0.372 |
| Arm (cm) | 1142 | **0.10 (0.06 to 0.15)** | **<0.001** | 1025 | **0.08 (0.04 to 0.13)** | **<0.001** |
| Waist (cm) | 1142 | **0.05 (0.01 to 0.10)** | **0.016** | 1025 | 0.03 (-0.01 to 0.08) | 0.158 |
| Hip (cm) | 1142 | **0.07 (0.03 to 0.11)** | **0.002** | 1025 | **0.05 (0.01 to 0.10)** | **0.028** |
| Sum of skinfolds (mm) b | 1133 | **0.09 (0.05 to 0.13)** | **<0.001** | 1019 | **0.07 (0.03 to 0.12)** | **0.002** |
| Overweight or obese (BMI z-score >1SD) c | 1142 | **1.36 (1.11 to 1.67)** | **0.003** | 1025 | 1.24 (0.99 to 1.57) | 0.066 |
| Obese (BMI z-score >2SD) c | 1142 | **1.82c (1.19 to 2.80)** | **0.006** | 971 | **1.74c (1.07 to 2.82)** | **0.025** |
| Standardised coefficient (β), representing the standard deviation (SD) change and 95% confidence interval in body composition measure per SD change in dietary pattern score enabling comparison across outcomes; Model 1 (simple regression) - association between dietary patterns and childhood body composition at age 6 years, Model 2 (multiple regression) – association between dietary patterns and childhood body composition, adjusting for infant mode of feeding, infant sex, and maternal ethnicity, BMI, socioeconomic status, multivitamin use pre-pregnancy, alcohol intake pre-pregnancy, cigarette smoking in first trimester, time spent watching television in first trimester, depression in first trimester, gestational hypertension, GDM diagnosis, and plasma placental growth factor. a World Health Organisation (2007) BMI for age z-score; bsum of triceps, biceps, subscapular and suprailiac skinfold thicknesses; c odds ratio | | | | | | |