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## Working Paper The Formation and Malleability of Dietary Habits: A Field Experiment with Low Income Families

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## The Formation and Malleability of Dietary Habits: A Field Experiment with Low Income Families

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FEBRUARY 2018

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## ABSTRACT

# The Formation and Malleability of Dietary Habits: A Field Experiment with Low Income Families<sup>\*</sup>

We conduct a field experiment to evaluate the extent to which dietary habits are malleable early on in childhood and later in life. We implement two treatments one that targets what people eat, the other that targets the timing and frequency of food intake. 285 low income families with young children were recruited and assigned either to a control group or one of the two treatments, each of them lasting for 12 consecutive weeks. In one treatment, families received food groceries at home for free for 12 weeks and were asked to prepare five specific healthy meals per week. In the other treatment, families were simply asked to reduce snacking and eat at regular times. We collected a range of measures of food preferences, dietary intake, as well as BMI and biomarkers based on blood samples. We find evidence that children's BMI distribution shifted significantly relative to the control group, i.e. they became relatively "thinner". We also find some evidence that their preferences have been affected by both treatments. On the other hand, we find little evidence of effects on parents. We conclude that exposure to a healthy diet and regularity of food intake possibly play a role in shaping dietary habits, but influencing dietary choices later on in life remains a major challenge.

JEL Classification:	112, 114, 118
Keywords:	diet, field experiments, habit formation, biomarkers

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### I. Introduction

Poor diet is a major issue in most developed and developing countries. It is estimated that 11.3 million deaths per year can be attributed to a poor diet (Global Disease Risk 2013 Collaborators, (2013)). While there are many policies targeting diet<sup>1</sup>, such as information campaigns and more recently, a series of interventions based on insights from behavioural economics, most studies show that long term changes are difficult to achieve. This is one reason why many interventions target children, presumably at a stage where dietary habits are still forming. This paper evaluates two types of interventions targeted at young children and their families. The two interventions are linked to two possible factors for poor dietary choices and the current obesity crisis: i) what people eat and ii) how people eat – particularly erratic eating habits. The first intervention consists of a strong and invasive intervention, where families receive food and recipes at home to cook 5 meals a week over a period of twelve weeks. The second is a much simpler intervention where families are instructed to avoid (adults) or engage in regular and healthy (children) snacking between meals and adhere to a pattern of food intake during the day.

We evaluate these interventions with a randomized controlled field experiment with 285 families, conducted in two different areas of the UK – Colchester (England) and Edinburgh (Scotland). These treatments should not be seen as policy proposals, but rather a way of gauging the extent to which dietary preferences are malleable particularly early on in life. The motivation for the first treatment comes from a number of studies claiming that dietary preferences are formed early on in childhood and that repeated exposure to certain foods can increase liking (see Birch (1999) for a review). This claim inspired a number of recent experimental studies targeting children (Just and Price 2013, Loewenstein et al. 2016, Belot et al. 2016, List and Samek 2015). However, to evaluate the effect of early exposure, one needs an exogenous source of variation in diet early on in life and longer term measures of dietary choices. To our knowledge, there is, in fact, little evidence of such a causal relationship. We propose a protocol that generates an exogenous source of variation in exposure. Indeed, randomization at the household level allows us to test a "treatment dose" that would be of much greater intensity than a realistic policy intervention that would be much more expensive to carry out (Ludwig, Kling and Mullainathan 2011).

<sup>&</sup>lt;sup>1</sup>See Lang et al. (2009), Capacci et al. (2012) for reviews, French et al. (2003) for a discussion or pricing policies in nutrition, Ciliska et al. (2000), Harnack et al. (2009), Drichoutis et al. (2009), Downs et al. (2009), Capacci and Mazzochi (2011), Robertson (2008), Verplanken and Wood (2006), Croker et al. (2012) for recent studies on the effects of public information campaigns (such as the five-a-day campaign or the provision of calorie labelling information).

This first intervention has a number of key elements that were chosen to maximize the chances that children do get exposed to new foods for these three months. First, the protocol ensures convenience and limits non-financial costs that could be important obstacles in adopting a healthy diet. Families do not have to plan for these meals, i.e. they do not have to search for suitable recipes, organize the shopping, etc. The food is delivered at their home and they receive a weekly booklet of recipes using the ingredients delivered. Also, the recipes have been chosen for their simplicity of execution and the protocol has been deliberately chosen over stricter protocols that would impose constraints on families on all meals and food consumed to ensure that the protocol was feasible. All families did however receive a "healthy eating booklet" (see Section II and Appendix A.2 for more details) and were recommended to follow the UK dietary guidelines as much as possible. The convenience of the protocol echoes behavioural interventions that aim at making healthy choices easier. Second, the food is provided free of charge and the costs of the meals have been calibrated to the average weekly budget of low SES families in the UK, so they should help families learn how to adopt a healthier diet within their budget.

The second intervention aims at changing the frequency and regularity of food intake during the day. This draws on evidence of how snack foods are often calorie rich and nutrient poor, and irregular and unstructured eating patterns are associated with poorer diets overall. Children and adults often consume a large proportion of calories in the form of snacks, which are usually calorie dense and nutrient poor (Piemas and Popkin 2010; Bellisle 2014). Snacks are less likely to be planned and the object of conscious decisions, which mean that snacking may make us more vulnerable to biases highlighted by behavioural economists, i.e. we may be more likely to engage in 'mindless eating' (Wansink 2006; Wansink et al. 2009). Snacking is often referred to as a possible culprit for rising obesity rates (Cutler et al. 2003; St-Onge et al. 2003) there is however mixed evidence on the effects of snacking on BMI (Field et al. 2004; Larson and Story 2013). A number of studies have also shown an association between meal irregularity and poor dietary outcomes more generally (Laska et al. 2014; Leech et al. 2015; Hume et al. 2016), and there may be metabolic advantages to eating at more regular and structured intervals (Alhussain et al. 2016; Murakami and Livingstone 2015). Key behavioural hypotheses are that people appear more likely to choose healthier foods when they select them in advance than when they select them at the moment when they will be consumed (Read and van Leeuwen 1998; Naughton et al. 2015). In light of this evidence, a protocol encouraging reduction of snacking and more regular food consumption was expected to lead to positive dietary outcomes.

Generating an exogenous variation in snacking patterns and frequency and regularity of meals is challenging. It is difficult to monitor overall food intake. We implemented this second protocol by requesting parents to follow specific instructions. Parents were instructed to provide food to their children at regular times and avoid giving additional snacks in between. Parents were themselves asked to adhere to regular times as well and to avoid snacking between these times. Families were allowed "one day off the protocol" to increase the chances of compliance for the rest of the time. This "one day off" is also inspired by a common practice in some countries such as Sweden, where children are allowed to eat sweets one day per week ("Saturday sweets"). The idea here is again to generate a source of exogenous variation in the timing of food intake patterns, which should occur even if participants have not fully complied. We will come back to the issue of compliance later in the analysis.

Our main objective is to evaluate how both treatments affected dietary choices of children and their main carer (most often their mother). Diet is however a complex object to measure and most studies rely on partial measures of dietary choices (such as isolated one- shot choices or consumption of specific items). It is in fact very difficult to obtain a complete picture of dietary choices, which then also makes it difficult to evaluate what is driving them and to identify successful policy interventions. Downs and Loewenstein (2012) identify this as a key shortcoming of existing studies, writing that "the true success of such measures will remain unclear until researchers are able to measure an individuals total food intakenot only calories at a single meal or in a single episode of snacking." To address this issue, we collected a range of measures, some are based on self-reports, others are objective (Body Mass Index and blood biomarkers the latter only for adults), and an incentivized measure of food choice for adults. We invited the participating families to the facilities at the Universities of Edinburgh and Essex several times to collect information before during and after the treatments. This set of measures collectively should in principle provide us with a more reliable picture of dietary choices than each of them would individually.<sup>2</sup>

We focus on low income families because there is well documented evidence of a strong socio-economic gradient in chronic diseases and in obesity. Low SES individuals appear to be up to twice as likely to be affected by some chronic diseases relative to high SES individuals (Dalstra et al. 2005). Socioeconomic status has also been shown to be correlated with nutritional deficiencies. For example, the 2012 UK Low Income Diet and Nutrition Survey (LIDS) shows that low-income households have diets that are deficient

 $<sup>^{2}</sup>$ We will come back in Section 4.7 on the issue of multiple measures and hypothesis testing.

in fresh fruit and vegetables, deficient in iron folate and vitamin D and high in sugar and saturated fats.

In this paper we present the evaluation of the immediate effects of the treatments, as well as the impact observed one year after the start of the experiment. Overall and perhaps notably, we do not find large differences across treatments. Also, we do not find significant and robust effects for parents their dietary habits seem unaffected by either treatment. Children, on the other hand, respond more. We find that childrens self-reported preferences for certain food groups changed in response to the treatment in the short run, but only for certain food groups and not always in the direction one would expect. Those exposed to the "meal" treatment report liking less processed foods, bread and cheese, but report liking sweets more. Those exposed to the "regular food intake" treatment, also report liking sweets more immediately after the intervention, while no other changes are observed for other food groups. Reported preferences for fruit and vegetables in particular remain very similar before and after the intervention. We find no significant differences in their overall dietary intake (as reported by the main caring parent) except for the added sugars intake in the longer run. In contrast, we find evidence for significant changes in their body mass index (measured as the percentile in the distribution of their age and gender cohort) for both treatments. Children in both treatment groups appear to have moved down in the distribution, that is, they have a relatively lower body mass index than the children in the control group.

These findings show in fact little evidence for malleability of dietary choices among adults. The first (meal) treatment is a very invasive intervention which is certainly far above the upper bound of policy instruments that could be considered. Still, we find little evidence of any change. The second treatment is very cheap, but appears harder to follow, and again, does not lead to changes in diet choices or preferences of adults. Children appear to be more responsive, and the changes we observe in BMI are notable. However, we cannot conclude that our treatment led to substantial changes in dietary preferences – that is, we do not have strong evidence that dietary choices can be altered by repeated exposure, even early on in life.

This paper relates to the recent body of experimental work on health-related behaviours and dietary choices in particular. Cawley et al. (2016) and List et al. (2015) conduct field experiments in collaboration with a supermarket and test different types of interventions targeting the prices of nutritious vs. less nutritious foods (subsidy, taxes, information) in order to increase the consumption of the former. These experiments show that framing matters (low income families purchased more of both nutritious and lessnutritious food under the subsidy framing (Cawley et al. 2016)) and that incentives can lead to sustained changes in the purchase of fruit and vegetables even when the incentives are removed, suggesting habit formation had taken place (List et al. (2015)). However, overall spending in the store was low, suggesting that most other food purchases were taking place elsewhere. It is therefore not clear what the overall effect was on diet.

The subsequent sections in the paper are structured as follows: in Section II we present the experimental design. Section III describes the different measures collected. We present the empirical analysis in Section IV. Finally, we conclude in Section V.

### II. Experimental Design

Sample and Recruitment — We recruited families with young children living on low incomes from the areas around Edinburgh (Scotland) and Colchester (England).<sup>3</sup> Based on our eligibility criteria, families would need to: have a household income below the median income £26,426 for Scotland, £26,600 for England; have a child between 2 and 6 years old at the start of the study; own a fridge and a hob; live in Edinburgh or Colchester.<sup>4</sup> Recruitment began 4 weeks prior to the start of the experiment.<sup>5</sup>

Participants received only general information about the study – such as the study being related to health and dietary choices and the study duration of 3 years. Families were not yet informed about the details of the two treatments. We excluded families for whom we considered the study to not be suitable, for example, families with individuals with pre-existing medical conditions, such as Diabetes Type I and II, or those with severe food allergies (see Appendix A Table A.1 for the full list of exclusion criteria). We collected data on at least two people per household: The youngest child in the family who was between 2 and 6 and his/her main carer (most often female). Whenever possible, we collected data on both parents. Regardless of the number of children in the household, the "study child" for which we collected measures was the youngest child of the household being aged between 2 and 6. Consent forms were obtained for each participant and from the main carer for the child.

Randomisation — Families were randomly allocated to the treatments and control groups

<sup>&</sup>lt;sup>3</sup>The experiment was conducted with ethical approval from the University of Edinburgh.

<sup>&</sup>lt;sup>4</sup>Exclusion criteria are available in Appendix A, Table A.1.

<sup>&</sup>lt;sup>5</sup>We used a range of different recruitment strategies which consisted of adverts, posters and stalls in community centers, nurseries; and shopping malls; letters sent to school principals; advertisements in buses and on radio. Samples of our recruitment materials (leaflet and poster) can be found in Appendix A. Recruitment took place in January and February 2015 for Edinburgh, and in July and August 2015 for Colchester, the interventions were conducted in March-June 2015 in Edinburgh and September-December 2015 in Colchester.

prior to treatment. When registering to take part, participants were asked to indicate several dates where they would be available to come to our facilities for the first session of measurements. All initial sessions had been randomly pre-assigned to a specific treatment (control or one of the two treatments), and participants were randomly assigned to one of their selected dates, without knowing these corresponded to the two different treatments and/or the control group (nor did families know that there were different treatments or what these were).

Timing — Overall, 91 families in Edinburgh, and 194 in Colchester took part in the study. Table 1 provides further details on attendance and attrition. Each treatment lasted for twelve weeks, and the baseline and post experiment measurements were collected during 2-week time windows before and after this twelve week treatment period, for each of the three groups (treatments and control). The 12-week treatment started in March 2015 (Edinburgh), and September 2015 (Colchester), and an additional one-year followup session has taken place in February (Edinburgh) and August (Colchester) 2016. The families were not asked to follow specific guidelines beyond the twelve weeks of treatment for the two treatment groups.

The attrition rate has been extremely low (3.85%). A year after (1-year follow-up), the attrition rate (compared to the before session) has reached only 13

Treatments — The first treatment, hereafter the "Meal" treatment, consists of providing ingredients and recipe booklets every week, for twelve weeks, directly at participants homes for five main meals for the whole family. The main objective was to maximize the chances that families, and more importantly children, are exposed to a range of healthy meals for a period of twelve weeks. We are then interested in evaluating whether these changes are sustained in the longer run. The protocol has been designed around multiple dimensions that have been highlighted as potential determinants of unhealthy dietary choices. First, there is a related literature in nutrition on the formation of food preferences, which suggests that repeated exposure to certain foods can increase liking (see Birch, 1999 for a review), particularly in childhood. While this mechanism is often mentioned in related studies, there are in fact few studies that provide causal evidence of exposure to foods and dietary patterns later in life.

Second, the protocol ensures convenience and limits non-financial costs that could be important obstacles in adopting a healthy diet. Families do not have to plan for these meals, i.e. they do not have to search for suitable recipes, organize the shopping, etc. The food is delivered at home and families receive a weekly booklet of recipes for the ingredients delivered. The recipes have been chosen by a nutritionist for their simplicity of execution, which ensured that the food families were exposed to would be part of the usual British cuisine, diminishing the likelihood for them of not knowing the food they were asked to cook. An isocaloric comparison (fixed at 365 calories, the average calories of the meals) between the recommended nutritional guidelines and our recipes shows that our recipes are overall consistent with the recommendations, and are lower than the maximum thresholds on sugar and fat (and saturated fat), compensating for these calories via higher carbohydrate and protein contents. This can be seen in the Table B.2, in Appendix B, by comparing the second and the third columns. A similar analysis on the participants diet will be discussed in section 4.4.

Convenience and ease of implementation may be particularly relevant for families on a low income, who may have other priorities to focus on other than food. For example, Mullainathan and Shafir (2013) argue that poorer individuals are likely to be confronted with a range of competing problems to resolve and may prioritize problems that require immediate attention over issues that have consequences in the more distant future (such as health or saving). The protocol has been deliberately chosen over stricter protocols that would impose constraints on families on all meals and food consumed, to ensure that the protocol was feasible. These design choices aimed at maximizing the chances of implementation in the short run and of sustainability in the longer run.

Third, the food is provided free of charge, which addresses the potential obstacle of perceived unaffordability of "healthy foods" (e.g. Dibsdall et al. (2003)). Recent survey data from the UK suggest that 36% of low income households indicated they could not afford balanced meals. In addition, low income parents may be somewhat risk averse and less willing to try to cook new meals for their children for fear of the children not liking the food (Dowler et. al. 2001). By providing the food for free, we alleviate the potential costs of wasting food that may discourage parents from buying and trying new foods. Furthermore, the costs of the meals have been calibrated to the average weekly budget of low SES families in the UK so it should in principle be possible for families to continue buying the ingredients and recipes once the treatment is over. According to the ONS statistical bulletins on Family Spending in the UK from 2015, a household composed of one adult and one child spend on average  $\pounds 42.5$  per week on food and non-alcoholic drinks. When not distinguishing by the size of households, the average spending on food only, in the UK, in 2015 is  $\pounds 39.2$  for the households below the median income (this number is not available according to the household size). Those figures exclude spending in eating out or take away. By assuming that they consume this food for about 9 meals per week (4 during week-ends and 5 evening meals during the week), we can estimate that British households spend about £21.7 for 5 meals. Note that the last two dimensions are most relevant for the adults, and mainly the mothers, who are usually in charge of food provision for the family. Tackling these obstacles should in principle maximize the chances that both children and adults get exposed to the healthy meals.

Families could select between regular or vegetarian food baskets. To maximize compliance families were asked to take photos of their meals (we provided cameras and SD cards) and to fill in a feedback sheet reporting on how easy it was to cook the meals (on a 4-point likert scale) and whether families liked them (again on a 4-point likert scale). An example of the first page of this feedback sheet can be found in Appendix A, Table A.4. Food baskets rotated on a four-weekly basis, so families received the same food baskets and recipes three times in the twelve week treatment in order to allow for possible habituation and changes in food preference. With this group, we also talked through, and provided a handout about, general advice on healthy eating which also included advice about alcohol consumption (Appendix A, document A.2.).

The second treatment, hereafter the "Snack" treatment, consists of regulating the timing of food intake, again for twelve consecutive weeks. Adults in the family were asked to eat three meals per day, at regular times (selected by participants) and consume no food or calorific drinks between meals. For the children, the treatment involved consuming three meals (not provided by us) and two snacks (provided by us) at regular times, without any further snacking in the day. The snacks we delivered were approved by a nutritionist. The list of those snacks can be found in Appendix A, Table A.3. Snacks are arguably less likely to be results of conscious decisions, and snacking may make us more likely to engage in 'mindless eating' (Wansink 2006, Wansink et al. 2009). Piemas and Popkin (2010) find that children in a US sample get 27% of their daily calorie intake through snacks, which are often nutrient poor, and high in sugar and saturated fats. A review paper by Bellisle (2014) suggests that snacking often seems to contribute calories but little nutrition, especially among obese children and adults. Factors which determine nutritionally poor snacking include choosing energy-dense foods, eating when not hungry or in an irregular fashion, and eating in contexts which promote 'mindless eating, such as watching TV (Bellisle 2014). A review on changes in childhood food consumption patterns by St-Onge et al. (2003) suggests that the rising proportion of calories coming from snack foods, which are in turn associated to higher sugar and fat consumption, may be a contributor to rising overweight and obesity in children. Although snacking is often held responsible for rising obesity rates (Cutler et al. 2003) research on the effects of snacking on BMI is not unanimous (Field et al. 2004; Larson and Story 2013).

Our protocol aims to address the detrimental effects of snacking within the context of imposing a more structured meal pattern, with meals eaten at regular intervals. There appears to be an association between meal irregularity and poor dietary outcomes. For example, a study of US college students found that meal routines most strongly associated with healthy diets included meal regularity (i.e. routine consumption of evening meals and breakfast), while eating on the run was associated with poorer dietary quality (Laska et al 2014). Yet, a review of how meal patterns are associated to diet found that only skipping breakfast was consistently associated with poorer diets across studies (Leech et al 2015). A randomised controlled trial on healthy participants found that compared to an irregular meal treatment, those on a regular meal protocol experienced metabolic responses which may favour weight management and metabolic health (Alhussain et al. 2016). With respect to children, a recent study on UK survey data focusing on metabolic markers rather than food consumption found that larger variability in eating frequency was associated with higher total and LDL cholesterol concentrations in children aged 410 years, but there was no association with BMI, waist-to-height ratio, and commonly tested biomarkers (Murakami and Livingstone 2015). A related body of literature in biology hypothesizes that irregularity of food intake could have a significant impact on diet and total calories, although this hypothesis is not supported in animal experiments (Hume et al. 2016). People choose healthier foods when selecting foods in advance compared to spur of the moment decisions (Read and van Leeuwen (1998)) and that dietary planning and self-regulation are argued to be good strategies to deal with habit driven impulsive consumption of unhealthy food (Naughton et al. 2015). In light of the evidence, a protocol encouraging reduction of snacking and more regular food consumption was expected to lead to positive dietary outcomes.

Other than being given recommendations about timing of food consumption, families were not given any additional instructions or recommendations as to what they should eat. This protocol is of course difficult to enforce, and so the main goal of this intervention is to create a source of exogenous variation in frequency and regularity of food intake across groups, and study how that has an impact on dietary composition and total calorie intake. To increase compliance, families were to follow this protocol for 6 days each week, and were allowed one day off to eat as desired. Adults were asked to fill in a diary we provided listing the times when they and their children had their meals and snacks, and if they had deviated from the treatment (see Appendix A, Table A.5.).

In addition to treatment specific compliance measures, participants from both treat-

ments were asked questions about the protocol they were involved in when coming back to our facilities after the 12 weeks of treatment. Specifically, they were asked whether they experienced any difficulties in implementing the protocol, and if they liked and ate the food delivered. Finally, our control group consists of participants recruited in the same way as those for the treatments, but were instructed to just carry on as usual with their daily routines.

Monetary Compensation— Families are receiving £350 in Edinburgh, and £400 in Colchester for completing the entire study. The total amount was altered for the Colchester arm of the study to increase sample size, in light of recruitment difficulties encountered in Edinburgh. The total monetary compensation was subdivided into smaller amounts so families are given an incentive for every measurement session they attend.<sup>6</sup>

### III. Data

We collected a range of measures to provide a complete picture of the diet and health of participants. Those measures were collected at baseline, after completion of the twelve week treatments and one year after the interventions had taken place. Note that these should not be seen as multiple outcomes we wish to study independently, but rather as a range of measures that aim at capturing diet and health in different ways. The goal of the empirical analysis will be to identify a consistent and robust pattern across these different measures. The first set of measures we collected was intended to provide a picture of childrens dietary preferences and intake. The tools we have used are suited to the age of the children population in our sample. Two of these measures are based on self-reports. The third is an objective measure of body mass index, which could reflect changes in dietary intake in a more objective manner. To facilitate the comparison with parents, we collected the exact same measures for them. Furthermore, we inlcuded two additional measures for adults that are not self-reported. One is a set of biomarkers based on blood samples, the other is an incentivized measure of food choice.

#### III.A. Base measures (children and adults)

<sup>&</sup>lt;sup>6</sup>Families received £50 for attending the first session (before intervention) in Edinburgh, £100 in Colchester. They then received £20 for a follow-up session that took place during the intervention in both locations and finally £130 for attending the session just after the intervention in Edinburgh, £100 in Colchester. Participants received £50 for attending each follow-up, once a year until 2018, in both locations.

Food Preferences and Diet — The first measure is a direct measure of dietary preferences. Due to the young age of children, we opted for a simple non-incentivised measure of preferences. We conducted a simple survey asking children and adults independently to rate their liking of a set list of foods. The questionnaire included 20 food items aimed at capturing a range of different food groups and 5 recipes that featured in the Meal treatment. The chosen items cover different food groups (see Appendix B, Table B.3. for the full list of items). For each item, participants had to answer on a 4-point scale how much they liked the item (really dislike to really like), with the additional possibility of an 'allergic' or 'never tried' option. Items were then grouped into food categories following the 'eatwell plate'<sup>7</sup> food categories (fruit, vegetables, meat/fish/eggs, cheese, bread, unhealthy processed food and sweets). The second measure is a measure of dietary intake based on a well-known method in the nutrition literature, called the "24 hour diet recall". Participants are asked to recall in detail what they have eaten in the last 24 hours. They are helped and guided by a professional nutritionist, trained to collect data using this method. For children, we collect information from the childs parent, primarily the mother. The data was first recorded face-to-face with nutritionists, and then entered into a nutritional analysis software (NetWISP 4), which computed measures of dietary intake based on a large database of food items available in the UK. This provided us with caloric intake estimates, as well as diet composition in terms of macro-nutrient breakdown.

Studies validating the 24-hour diet recall as a method for measuring dietary intake compare it to energy expenditure measured by doubly labelled water. These studies show that the 24-hour recall underreports from 1% to 17% depending on a number of factors including the number of consecutive recalls obtained (each additional consecutive recall gives more accuracy), and whether these have been done in person or over the phone (Hill and Davies, 2001, Livingstone et al. 2003; Ma et al. 2009). While three consecutive recalls are recommended to assess individual intake, one recall does capture the average intake of a group fairly well (Biro et al., 2002).

For the one-year follow-up we used Intake24 - a computer-based recall method designed for the British population (https://intake24.co.uk/). Unlike the nutritionist led face-toface 24-hour dietary recall described above, with Intake24, the participants recall their own intake using the software. Outcomes are similar and can thus be compared to the face-to-face recall.

Body Mass Index — Adults and children were also weighed and measured by a member of

 $<sup>^{7}</sup>$ The eatwell plate is a policy tool used to define the British government recommendations on eating healthily and achieving a balanced diet.

our team. Height and weight data were used to calculate BMI, and age-adjusted BMI for children using BMI cut-offs (based on the percentiles) recommended by the Childhood Obesity Working Group of the International Obesity Taskforce (Vidmar et al. 2004). Each of these measures was taken up to three times for better accuracy. The average of these measures is used in the analysis.

#### III.B. Additional measures (adults only)

We collected two additional measures before and after the treatment in adults that are not based on self-reports. We used an incentivized measure of food choices in Colchester and we collected data on a range of blood biomarkers for adults in Edinburgh.<sup>8</sup>

Incentivized measure of food choices— In Colchester only, every adult participant was asked to pick two combinations of a snack and a drink, one of low calorie (less than 100 Kcal) and one of high calorie (more than 200 Kcal). They were endowed with £4 and were asked to spend part of this money in buying the pair of snacks. They had 7 choices to make in which they had to decide whether they wanted to buy the low-calorie pair or the high-calorie pair of snacks. The price of the low calorie pair of snacks was set to £2 for all 7 choices. The high calorie pair of snacks price ranged between £1.40 and £2.60, with an increment of 40 pence for each choice. The task is shown in Figure 1.

Choices made in this task tell how much the participant is willing to pay to receive the high-calorie option compared to the low-calorie one. From choices 1 to 3 the high calorie option is more expensive than the low-calorie option, choice 4 displays the same price for both, choices from 5 to 7 displays a lower price for the high calorie option.

Blood biomarkers and blood pressure — In Edinburgh only, study participants (excluding children) provided fasted blood samples prior to and after the twelve week treatment. The full list of biomarkers screened and their short description is presented in the Appendix in Table B.3.

## **IV** Empirical Analysis

#### IV.A. Empirical Strategy

In order to evaluate the impact of the two treatments on the range of outcomes we have, we estimate the following intent to treat (ITT) effects:

 $<sup>^8 {\</sup>rm For}$  logistical and time constraints reasons, it was not possible to collect both measures in both samples.

$$\begin{aligned} \text{Outcome}_{it} &= \alpha_i + \beta_1 A fter_{it} + \beta_2 \ \text{1-year}_{it} + \beta_3 M_i \times A fter_{it} + \beta_4 S_i \times A fter_{it} \\ &+ \beta_5 M_i \times \text{1-year}_{it} + \beta_6 S_i \times \text{1-year}_{it} + \epsilon_{it}, \end{aligned}$$

Where  $\alpha_i$  is an individual fixed effect,  $After_{it}$  indicates the period is immediately after the 12 week treatment, with 1 - year indicating being 1 year after the treatment.  $M_i$  and  $S_i$  are the two treatment assignments, meal and snack respectively. The estimation of the ITT effects are  $\beta_3$  through to  $\beta_6$ .

#### **IV.B.** Descriptive Statistics

Table 2 presents the descriptive statistics of our sample for the different groups. Overall, there are no statistically significant differences between the control and the treatment groups at baseline. As defined by the recruitment criteria, the average age of the children is about 4 years old, and the average income is below the English and Scottish median income. Every household receives at least one type of benefit. Our sample contains more women than men; most of the time they were single mothers or the father was not available to attend the session.

#### **IV.C.** Compliance

The experiment is an intention-to-treat. For the Meal treatment, families had to prepare five meals per week; while for the Snack treatment, families were requested to stick to regular eating times. Neither protocols were directly incentivised and we do not have a direct measure of compliance. Nevertheless, we used several strategies to encourage compliance. For the first treatment, we asked families to take pictures of the meals they prepared and fill in a feedback leaflet on the recipes (asking which meal they prepared on each day, how easy it was to prepare and to rank how it tasted, see Appendix A Table A.4.). The main reason for providing this leaflet was to encourage compliance, as they were asked to bring back the leaflets at the end of the study. For the second treatment, families were also asked to fill in a leaflet indicating the precise times the main carer and the child ate on each day of the week, which day was chosen as the "day off", and whether they deviated from the protocol (see Appendix A Table A.5.). We told all families in both treatment groups that we were interested in learning how easy the protocols were to follow and would value feedback on the difficulties they have encountered. To make sure that families understood well what was expected from them, we met with each of them one-toone and provided face-to-face instructions about the intervention. We explained in detail what was expected from them, and handed out the leaflets and cameras (for the meal

treatment). We also organized an additional short session in the middle of the twelve weeks (both for control and treatment groups), with the main purpose of maintaining compliance and preventing attrition. All families were asked to fill in a short survey, families in the Meal treatment were asked to bring back an SD card as well as the first part of feedback leaflet, families in the Snack treatment were asked to bring back the feedback leaflet.

We propose three alternative ways of gauging the degree to which families complied. First, participants from both treatments were asked questions about the protocol they were assigned to when coming back to our facilities after the twelve week treatment. In particular, they were asked whether they experienced any difficulties in implementing the protocol, and if, in general, adults and children liked and ate the food they were delivered. Hence, in addition to the feedback leaflets, the cameras and the photos, these self-reported answers inform on the motivation to follow and opinions about the treatments that have been implemented.

Table 3 shows differences in self-reported measures regarding the ease of implementation of the protocols. We find interesting differences between the two treatment groups. For instance, 42.6% of the Snack sample found difficult or very difficult not to snack between the meals. In contrast, 83.7% of those in the Meal treatment say they found it easy or very easy to cook the recipes. Complying with the Snack protocol has not been straightforward and probably meant a substantial change in routine for some participants.

Table 4 presents several variables capturing how participants felt they were affected by the protocols. We find that 46.2% of the people assigned to the Snack treatment felt they were eating less food during the day. In the Meal treatment, 64.3% of the adults self-report and 79.5% of the children (reported by the main carer) report having tried new food they had never tried before. This table shows that participants seem to perceive an effect of the protocol on their food habits. They also admit (58.2% of the Meal sample) that they had to adjust the recipes to their taste.

After the treatment, we also asked Meal participants how many recipes they intended to continue cooking and how many they actually did continue to cook. Just after the treatment, 125 individuals answered this question. On average, they planned to continue cooking 9.4 out of 19<sup>9</sup> recipes. One year later, this average dropped to 6.8 recipes for 114 participants responses.

The second measure of compliance we propose is based on the number of photographs provided by participants in the Meal treatment. Since they were supposed to cook 5

 $<sup>^915</sup>$  recipes in total for vegetarian families who represent 8% of the sample.

meals per week during 12 weeks and to photograph each of them, a complete set of pictures would include 60 photographs. On average, we received 38 unique pictures back (hence 53%). This could of course be an underestimate of the meals that were cooked and eaten, it could well be the case that a meal was cooked but participants forgot to take the picture. Conversely, it could be the case that a family cooks the meal, takes the picture, and does not eat it, implying that compliance is lower that the rate of picture returns suggest. While it is a possibility, the family is much less likely to cook and the meal and forget to take the picture than cook and not eat the meal. Hence, the compliance measure from the return of the unique photographs is likely to be an underestimate. Furthermore, only 11% of the meal households gave us no pictures back at all. These figures suggest that compliance was relatively high.

Finally, the last measure of compliance we have is based on the information provided in the leaflets. Regarding the Meal treatment, 80.6% of the households who came back after the intervention brought their leaflet back to us, which is a high rate and those leaflets were completed with care. They report an mean liking of the meals of 2.9 (0.38 s.d.) (on a 4 point likert scale). When taste has been different for the children they also reported it yielding an average liking by the children of 2.7 (0.67 s.d.). Children report liking the recipes significantly less overall than the adults (a Wilcoxon signed-rank test yields a p-value=0.000). Turning to the difficulty, from a 5-point likert scale (from very easy to very difficult) adults report an average of 1.7 (s.d=0.5). Those results corroborate the self-reports displayed in Table 3 showing that this treatment has been perceived as relatively easy to follow.

In the Snack treatment, among the families that came back after the intervention, 69.0% brought the leaflet back, which is a lower rate than in the Meal treatment. Additionally, it was possible to evaluate the extent to which the forms were filled in in a "robotic" fashion. We use two main criteria to characterise the households as filling the leaflet out in an automatic manner or not: first if they were writing out the same times of the meal over the 12 weeks, with the same pen and without any noticeable differences on each of the pages. Second, because the families could deviate from the regular food intake one day of their choice every week, another "robotic" attitude with this aspect would be to tick every week the same day, with the same pen, and without ever deviating to choose another day from the protocol. We find that 37.5% of the families tend to fill the leaflet out with the same times of the meals in an extremely regular way. As far as the second measure is concerned, 20.8% seem to always report the same day off, with no variation in the writing style.

Returning to the "day-off" allowed within the Snack treatment, if every family was taking this option, this would mean that out of the 84 days of the treatment duration, 12 (14.3%) should be marked as a day-off. We find that 14.5% of the days of the leaflets have been reported as the day off. Families also had the opportunity to inform about additional deviations of the protocol. We find that 19.9% of the days were reported as additional days where families did not follow the treatments requirements. The leaflet also allows us to check the regularity in the meal times as participants were reporting the time of the three (five for the children) meals they had during the 12 weeks. For each week, we set the time mode as the regular time and we look at the frequency of a 30 minute deviation from this mode. 18.7% of the adults breakfast deviated from their mode, 16.53% for the children. This proportion becomes 19.7% for the adults lunches, 18.2% for the childrens lunches. Finally, dinner seems to be the most consistent as 13.9% of the meals deviated from the adults time mode, 11.8% for the children. This shows a degree of irregularities of the Snack treatment which corroborate results from Table 3 and Table 4.

The three compliance measures show that the Meal treatment tended to be easier to follow for the families compared to the Snack treatment. Families in the Meal treatment were then more compliant and conscientious with filling out the leaflet.

### IV.D. Analysis of the effects on dietary preferences and intakes

We now turn to the main analysis and start with the evaluation of the effects of both treatments on dietary preferences and intakes. For each variable of interest, we first present summary statistics at baseline and across groups, and then present the Intent to Treat (ITT) results.

Table 5 presents the self-reported food preferences of both the children and the adults at baseline (before the twelve week treatment). We report the mean of each category of item. These items are ranked based on the control groups answers. There are overall no significant differences in liking at baseline between the treatment groups. It is worth pointing out that the ranking follows an expected pattern for children with sweets, bread and processed food at the top, while the meals are situated at the bottom of the ranking. For parents, in contrast, the ranking is perhaps more surprising, with processed foods and sweets appearing relatively low in the ranking. One might worry that adults are less likely to report their true preferences with such questionnaire, and are perhaps tempted to report desired preferences instead. This is speculative, of course, but worth keeping in mind when we turn to the results. We present the ITT estimates of food preferences in Table 6 for two points in time: in the short run, right after the treatment ("After") and in the long run, one year after the treatment ("1 year"). For space reasons, we only report the results for the different food groups. The results for individual food items and meals are reported in the Appendix B, Tables B.6., B.7. and B.8.

We find that self-reported preferences remained quite stable and that the treatments have a limited effect on those self-reported measures both in the short and the longer run. The estimates are quite precisely estimated and close to zero. There are a few significant differences, for example, children in the Meal treatment report a lower level of liking of processed food, as well as for cheese, which are two categories for which consumption are advised to be reduced because of their salty and fatty composition. One year after, the differences found in the short-run disappear. Adults in this group report liking more processed food on average than the control group right after they have been treated although this is only statistically significant at the 10% level. Finally, we found no significant changes were found in meal preferences for children and adults who were exposed to those meals, compared to the control group (see Table B.8. in Appendix B).

We now turn to the analysis of dietary intakes. Table 7 presents the baseline statistics (before the treatment) for different categories of food intakes and average quantities: total calories intake, number of fruits and vegetables, quantities (in grams) of fruits and vegetables, total fat, carbohydrate, protein, saturated fat (typically referred to as unhealthy fats), sugar, Non-Milk Extrinsic Sugar (NMES, also called free sugars, which are generally considered to be added sugar), fibre, sodium and alcohol. The first column of Table 7 shows the daily recommendation given by the National Obesity Observatory Document Standard evaluation framework for dietary treatments<sup>10</sup> and the Manual of Dietetic Practice (Thomas et. al. 2007). We distinguish between total fat and saturated fat as well as total sugar and NMES. On average, the self-reported intakes imply that a male adult participant consumes 2216 calories over 24 hours, whereas a female adult consumes 1907 calories. The average calorie intake in children is 1434 calories. These numbers are below the recommended total daily calorie intake in the UK. However, it is likely that participants under-report their food intake (Poslusna et al. 2009).

Diets low in saturated fats and sugars and high in fruit and vegetables are typically recommended for preventing diet related causes of morbidity and mortality. Nevertheless, at baseline, we find no significant differences in calorie intakes or other macro-nutrient

<sup>&</sup>lt;sup>10</sup>British Nutrition Foundation (BNF), 2015. Nutrition Requirements. Available at: https://tinyurl.com/nutrition-requirements

intakes between our groups.<sup>11</sup> By comparing the different intakes with the daily recommendations, we see that a relatively large proportion of food intake for our sample comes from carbohydrates. The intake of protein is above the minimum requirement, the intake of saturated fats and sugars exceeds the recommended amounts. The participants also fail to meet the recommended intake of fruit, vegetables and fibre.<sup>12</sup>

We also look at the baseline diet of the participants compared to the recipes participants have been provided in the Meal treatment. This allow us to check for a possibility of improvement in the diet of the Meal participants. In an isocaloric comparison, held at 365calories, of our participants consumption and our recipe profiles, we note that our participants ate twice the amount of recommended fat (15g versus 8g) and twice the amount of recommended sugars (20g versus 10g) (see Appendix B, Table B.2.). Participants diets at baseline were lower in carbohydrate and protein than our recipes. Our recipes were thus appropriate in aiming to modify participant diets by targeting sugar and fat consumption.

We now turn to the analysis of the two treatments on diet intake. Table 8 reports the estimates for calorie and macronutrient intakes allowing us to test for any treatment effect on those variables in both the short and the long run. To facilitate interpretation, the first row in the table indicates the sign of the difference between the UK recommendation and the average calorie/macronutrient at baseline. If it is positive (negative), participants consumptions were below (above) the recommendation and a positive (negative) treatment effect would indicate that they come closer to the recommendation. The data collection session for the post-treatment period was conducted at least one week after the 12-week treatments were finished.

Overall, we do not find many significant changes. We find that childrens fruit consumption becomes significantly lower when exposed to the Snack treatment compared to the control in the long run. We also see decrease in NMES intakes, compared to the control group, in both treatment groups in the long run. The signs of coefficients are mostly going in the expected direction except for the fruit and vegetable intakes, i.e. the coefficients are negative for calorie intakes, fat, sugars, proteins and sodium, but also negative for fruit and vegetable intake.

<sup>&</sup>lt;sup>11</sup>The report from the National Diet and Nutrition survey that is designed to assess the diet, nutrient intake and nutritional status of the general population aged 1.5 years and over living in private households in the UK reports an average calorie intake of 2107kcal for men, 1595kcal for women, and between 1108 and 1400kcal for children aged from 1.5 to 10 year old. https://www.gov.uk/government/uploads/system/uploads/attachment\_data/file/551352/NDNS\_Y5\_6\_UK\_Main\_Text.pdf

<sup>&</sup>lt;sup>12</sup>Another way of presenting the diet composition of an individual is to look at the average contribution of each macro-nutrient to the total calories. We report this Table in the Appendix B, Table B.9. The outcomes are obviously similar.

As for the adult intakes, no consistent nor significant patterns are found in contrast to the children. These results suggest that dietary intake may have changed for children, but we fail to find significant effects for most variables of interest, while for adults, we find no convincing evidence that their dietary intake has changed in the direction we would expect.

#### IV.E. Analysis of the effects on Body Mass Index

We now turn to the analysis of BMI, which is the only objective health measure we have for children. Table 9 shows the mean BMI and proportion of each weight category of our sample at baseline. Around 64% of our adult sample is overweight or obese (32%). These figures are in line with the national rates reported in the National Diet and Nutrition Survey.<sup>13</sup> Regarding the children, the obesity rate of our sample is also in line with national statistics and represents 5.3% of the children in our sample. We do not find significant differences in the distribution of weight categories between the three groups at baseline. However, women in the control group have significant higher BMI than women in the Snack treatment (a Wilcoxon signed rank test yields a p-value of 0.04).

Table 10 presents the results of the impact of the experiment on BMI. A lower BMI after the treatment in adults would indicate weight loss. Note that both treatments were not weight-loss programmes and so we would not necessarily expect large changes in BMI, at least in the short run. For children, we find a precisely estimated and negative treatment effect on the BMI percentile, seen in the first column, in the short run but also one year after. There appears to, therefore, have been a sustained impact. The size of the effects (between 4 and 6 percentage points) is similar across both treatments. Children in both treatment groups appear to have moved down in the distribution, that is, they are relatively thinner than the children in the control group. We do not find that they are more or less likely to be overweight or obese (Column 2), however the percentage of children in this category was low to start with. For adults on the other hand, we find no evidence of significant change in BMI, whether we look at BMI directly (Column 3) or the probability of being overweight or obese (Columns 4 and 5).

### IV.F. Additional measures

Incentivised measure of food choices - In the Colchester (England) sample, we included an incentivised measure of food choices, before and after the treatment. The measure is

<sup>&</sup>lt;sup>13</sup>https://www.gov.uk/government/uploads/system/uploads/attachment\_data/file/310995/ NDNS\_Y1\_to\_4\_UK\_report.pdf

described in section III.C. Table 11 indicates the changes in the number of times participants chose the low-calorie option over the high-calorie option. We find that participants are significantly less likely to pick the low-calorie option after the treatment, compared to the control group. This means that compared to before the treatment, they are willing to pay a higher price for the high-calorie snack and drink after the treatment. A possible interpretation of this could be that participants experience a rebound effect after having implemented a healthy meal or snack plan for 12 weeks and allow themselves to buy an unhealthy snack at a more expensive price to reward themselves, or it could be a manifestation of cravings after the twelve-week programme for this high-calorie snack (Fishbach and Dhar, 2005).

*Health biomarkers* — In Edinburgh, adults were asked to provide a fasted blood sample before and after the treatment (although not at the 1 year follow-up). Table 12 reports the levels of the different blood biomarkers levels at baseline, compared to the normal ranges as advised in the UK. Overall, our participants have normal levels for all biomarkers. This is not surprising as they are non-elderly adults (aged 35 on average) with no serious health conditions (one of the recruitment inclusion criteria). However, the mean Low-density lipoprotein (LDL) reaches the upper limit of the normal range in the control group and is significantly higher than in the Meal and Snack treatment. C-reactive protein (CRP) is produced by the liver, and rises when there is inflammation throughout the body. A CRP level higher than 3.0 mg/L is considered a marker of increased risk of cardiovascular disease, and studies show that CRP is lower when fibre intake is higher (Ajani et al. 2004, Johansson-Persson et al. 2014).

ITT estimates are reported in Table 13 showing two main treatment effects. First, the estimate of the level of LDL (sometimes considered as the bad cholesterol) is positive and statistically significant for the Meal participants compared to the Control group. Second, the estimate of the glucose level is positive it is not particularly precisely estimated (being only significant at the 10% level) for the Snack participants, compared to the control group.

In Table 8 we noted that no significant differences emerged in adults for the Meal treatment post treatment in terms of calorie and macronutrient intake. The changes in coefficients point to a slight increase in calories (88.4 calories), and a small increase in carbohydrates (11.8 grams) though other macronutrient changes remain in the single figures. Based on the above changes in diet, it is unclear why the Meal group experienced a small statistically significant rise in LDL values after the study. LDL has been shown to be elevated in diets higher in saturated fats (Mensink et al. 2003), yet post treatment

there was no significant change in the amount of saturated fats the Meal treatment were eating. Table 8 showed no significant differences in calorie and macronutrient intakes for the Snack treatment post treatment. The direction change of the coefficients point to a slight drop in calories (178 calories), a drop in total carbohydrates (24.5 grams) mainly caused by a drop in sugars (23.2 grams), and a fall in sodium (370 mg, approximately equivalent to 0.9 grams of salt). The above dietary changes appear to be somewhat consistent with changes in fasting blood glucose, which for the Snack group increased slightly but significantly post treatment. Fasting glucose levels tend to be higher on lowglycemic index diets than on high-glycemic index diets (Sacks et al. 2014), so a drop in blood glucose would be consistent with a post-treatment diet lower in sugars, which we indeed observe for this group albeit these are not precisely estimated.

Among other studies which have sought to quantify blood biomarkers, a study by Purkins et al. (2004) reported that after 8 days where healthy participants ate a high carbohydrate high calorie diet or a high fat high calorie diet equal to approximately twice the calories needed for subsistence, cholesterol rose by 15% and 7% respectively, but all mean results remained within recommended normal ranges. Triglyceride levels on the other hand were far more sensitive to dietary change, and were 99% higher among the high carbohydrate high calorie diet than the high fat - high calorie diet, with values for most subjects exceeding the upper limit of the reference range. In our study, it is unclear what level of change we may expect from our treatments which have not explicitly been designed to alter cholesterol or calorie intake. As for triglyceride levels, while they appear to be very sensitive particularly to sharp changes in carbohydrate intake, they also adjust very quickly to diet change (Purkins et al. (2004) reported change after 1 day). This means that if participants reverted to their usual dietary habits post study treatment, treatment driven changes in triglycerides may not have been picked up in our blood samples collected within a 2-week window post treatment.

#### IV.G. Correlation between parents and children

As the experiment is focused on the family, we are also interested in behaviour within the family unit, and also whether the changes in behaviour move in the same or different directions for different members of the family. In particular, in this section we examine the correlation of body size, food preferences and food intake and investigate to what extent to the latter two become closer or further apart as a result of the experiment. We may expect that with the meal treatment that preferences and food intake converges between the parent and child. Body measurements — Panel A of Figure B.1 shows the scatter plot of the child and main adults BMI. We find a positive correlation between the BMI of the child and main adult which is statistically significant. In panel B and C we examine the components of BMI: height and weight. We find that the positive correlation of BMI is driven by a positive correlation of weight between the parent and child and not height. We do not find a statistically significant correlation between height whereas we do for weight.

Food Preference Questionnaire — We begin by calculating the correlation of food preferences for each of the 25 items in our food preference questionnaire between the main adult and child, these are shown in Table B.9. We find a positive correlation in preferences with one exception, that of carrots which is negatively correlated but this is statistically insignificant. The correlations range from -0.043 (carrots) to 0.244 (melon), these estimates appear to be in line with earlier evidence on the resemblance of food preferences between parents and children, Pliner (1983). Just over a third of the items are positively correlated and statistically significant, with a mix of items not limited to just one food category including chips, broccoli, strawberries and peas. To examine whether the experiment led to parents and childrens preferences becoming more similar we re-estimate equation 1 where the dependent variable takes a 1 if the preferences of the parents and children are the same and zero otherwise. We present the results of exercise in Figure B.2. In summary, these figures show that the preferences of most foods have not become more alike because of the experiment, either immediately after the intervention or one-year later.

24 hour diet recall — Examining the baseline period we see a positive correlation between the food intake of the main parent and the child. Figure B.3 shows scatter plots of food intake with the childs intake on the y-axis and the main adult on x-axis, with a linear fit through those points. We find that this correlation is statistically significant for energy intake, for fruits and vegetables and almost all the macronutrients. The only exception is for protein intake, which could be due to young child not eating as much meat as their parents due to the texture. However, the correlations were the main weak with most estimates being around 0.2, the exception being vegetables which was slightly higher at 0.49. These results are of a similar magnitude to evidence from the US (Beydoun and Wang 2009, Wang et al. 2011). To examine whether this correlation changes we calculate the absolute difference in intake, be that overall energy or a specific macronutrient, between the adult and child. In particular, we again estimate equation 1 with the absolute difference as the dependent variable to examine the impact the treatments have had on this gap. Table B.10 presents the results of this analysis where we find that there is a statistically significant increase in absolute gap with respect to overall energy consumption after the intervention. From panel B, which examines the actual difference (adults intake – childs intake), we find the absolute gap is driven by an increase in the main parents calories, although this difference is not statistically significant. Panel A also shows there was a significant increase the distance between adults and children with respect to carbo-hydrates. Overall, there is a positive correlation between parents and child in the intake prior to the experiment and we find that the gap in overall energy intake between the parent and child increases also there is not a statistically significant difference for almost all the food types or macronutrients.

#### IV.H. Overall picture from multiple outcomes and hypothesis

We have collected a wide range of different measures to get the most accurate picture possible of dietary changes that may have taken place as a result of the two treatments we consider. Of course, with such a large number of variables considered, there is a danger of identifying individual coefficients that are statistically significant, purely by chance. However, these variables are not independent from each other and we can therefore exploit these multiple measures to identify consistent patterns across these variables. The question we ask here is: Do the estimated coefficients provide a consistent picture of dietary change?

Let us start with children. For the meal treatment, we observe changes in self-reported food preferences for processed foods, bread and cheese (all decreasing) and for sweets (increased preference). For dietary intakes, the point estimates for calorie intake are negative and relatively large (-37 calories immediately after and -128 one year later), and certainly well in line with the changes in numbers we observe in weight and BMI. We observe a 5 and 6 percentile drop in weight after treatment in the Meal and Snack groups respectively, which is sustained at the 1-year follow-up. To put this decrease into context, a 5-year old girl on the 50th centile for height and weight would be 108cm tall and weigh 18.10kg. To be on the 45th centile, this same girl would need to weigh 17.88kg, i.e. 220grams less, keeping height constant (NHS Healthy Weight Calculator). Based on calculations developed for adults (Hall et al. 2011), a weight loss of approximately 220 grams, would require a 770 calorie deficit over 12 weeks (the treatment period), equating to a mere 9 calorie deficit per day. This is generally in line, albeit lower, with the observed post treatment calorie deficits of -37 and -53 for the Meal and Snack groups respectively.

We find significant decreases in the intake of "added sugar" (NMES) which appears to be a key reason behind calorie reduction. At the one-year follow-up, the reduction in NMES for the Meal and Snack groups at -22g and -19g respectively accounts for 66% and 50% of the reported drop in calories (at -128 and -147 respectively). We find no effect on fats and no increase intake of fruit or vegetables. Altogether, a consistent story could be that children consumed fewer foods high in sugar (and perhaps therefore value them more) and this translated into lower BMIs. The story is somewhat similar for children assigned to the second treatment, although we also see here a significant decrease in the number of fruit consumed, and for self-reported preferences, we only find an increase in the preference for sweets. Thus, there is less of a consistent story for the Snack treatment than for the Meal treatment.

Turning to adults, it is much harder to find a consistent picture here. We find no change in self-reported preferences (all are close to zero and quite precisely estimated). The changes in calorie and macronutrient intakes are going in different directions: we find a significant increase in calorie intake for the Meal treatment one year after the treatment, as well as for the Snack treatment, although the effects are not statistically significant. There is no clear picture emerging from the point estimates of the coefficients on macronutrients, and there is no effect on BMI (coefficient is zero and quite precisely estimated). We find that adults in both treatments are more likely to choose a high calorie snack after the intervention, and their blood biomarkers do not give a clear picture either of changes in dietary choices. Thus, there is no consistent picture for adults and we find no indication that the treatments have had an effect on dietary intake and choices.

Regarding compliance, we have presented a set of different way to assess compliance of families: self-report feedback after the treatment had taken place, taking pictures of the meals, filling out feedback leaflets during the treatment phase. We have shown that compliance outcomes are going in the same direction within treatments but that they differ between the treatments. For instance, participants in the Meal treatment found easier to follow the protocol than participants in the Snack. There was also a difference in the rates of bringing back the feedback leaflet which was higher in the Meal than in the Snack treatment.

### V Conclusion

In this paper, we evaluate two treatments in a randomized controlled trial that aim at influencing what and how people eat: the Meal treatment through repeated food exposure and the Snack treatment through the regularity of food intakes. We gathered a large set of measures allowing us to have a multi-dimensional picture of dietary intakes, food preferences both incentivized and not incentivized, anthropometric measures and blood biomarkers. Families were asked to come to our facilities before, right after and one year after the treatments had taken place which enables us to estimate average treatment effect in both the short and long run.

We consider our treatments to be quite invasive and demanding, influencing food habits both through what participants eat (recipes, new ingredients, introducing more fruits and vegetables in the diet) and through the way they eat (cooking from scratch or regulating the timing of food intake). Our rich data was collected in a lab setting so we could use methods to limit as much as possible self-reported biases: height and weight were measured by a professional instead of being reported by the participants, dietary recall was assessed with the 24h dietary recall method, face-to-face with nutritionist the first year, limiting underreporting. Surveys were conducted in a computer lab so participants could ask questions if something was misunderstood.

We show that prior to the treatments both adults and children had diets that would be considered out of the national recommendations that contained too much saturated fat and sugar, and not enough fruits and vegetables. This unbalanced diet is corroborated by a high proportion of overweight and obese individuals in our sample. We do not find any consistent patterns in adults eating habits, and even perhaps a rebounding effect — the incentivised food choice task shows that participants in the treatment are willing to pay a higher price for a high-calorie snack, compared to the control group, after the 12-week treatment. No changes in weight, food preferences nor intakes were found as a result of the treatments.

Having a treatment on the family level, rather than only on the adults, was primarily to expose children to either a healthier diet or a new routine, within the family. Even though results are not so strong there is a significant pattern found here. In the short run, food preferences of children are decreasing for high-calorie food items (processed foods, bread and cheese). NMES intakes decrease significantly in the long run, in both groups, compared to the control group. And finally, children in both groups are moving down the distribution in terms of weight and BMI meaning that overall they become relatively thinner than the children in control group. A result that is found for both the short and long run. However, our treatment did not alter consumption or preferences of low-calorie recommended foods such as fruits and vegetables.

This paper raises different questions that would need to be addressed. On one hand, a heavy and intrusive treatment on diet does not seem to induce significant dietary changes in adults. On the other hand, an experimental measure such as the incentivised snack choice shows an effect of the treatments which mean that the treatments might trigger different underlying mechanisms that are more likely to be revealed with some behavioural and objective measures. The fact that results are not completely similar between children and adults shows that treatment on children, hence early on in life, might be a better way to modify dietary habits in order to prevent obesity.

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### Tables and Figures

	Control	Meal	Snack	Total
Essex baseline $(t=0)$	76	66	52	194
Essex after $(t=1)$	74	64	47	185
Essex 1 year follow-up $(t=2)$	67	55	39	161
Edinburgh baseline (t=0)	35	37	19	91
Edinburgh after $(t=1)$	35	37	17	89
Edinburgh 1 year follow-up $(t=2)$	33	37	17	87
Total baseline	111	103	71	285
Total after	109	101	64	274
Total 1 year follow-up	100	92	56	248

Table 1: Sample size. Number of participating households.

*Note*: "Baseline" refers to before the treatments, and after to just after the treatments.

	Control	Meal	Snack	P-value	P-value
	Mean (std)			(1) = (2)	(1) = (3)
Sample size (families)	111	103	71	-	-
(Present in before)					
% Female adults	72.2	79.6	75.3	0.15	0.59
% Female pregnant	8.1	6	1.9	0.62	0.13
# Adults in household	1.7	1.61	1.7	0.43	0.85
	(0.85)	(0.6)	(0.7)		
# Children in household	1.9	1.8	1.9	0.47	0.9
	(0.9)	(1.0)	(1.0)		
Age (adults)	35.1	34.7	34	0.67	0.23
	(7.5)	(6.5)	(6.9)		
Age (study child)	3.9	4	3.9	0.99	0.75
	(1.7)	(1.7)	(1.4)		
Mean annual household income	20,855	21167	23,928	0.87	0.15
(GBP)	(10,056)	(19, 227)	(21, 844)		
% Receiving child benefit	86.5	86.4	85.9	0.98	0.91
% Receiving tax credit	76.6	70.9	77.5	0.34	0.89
% Receiving job allowance	3.6	3.9	2.8	0.91	0.77
% Receiving housing benefits	37.8	41.7	38.0	0.56	0.98
% Receiving income support	22.5	17.5	22.5	0.36	0.99
% Receiving other benefits	8.1	7.8	5.6	0.92	0.53
% higher degree	21.0	19.2	15.0	0.72	0.25
% No qualifications	2.7	3.1	3.2	0.85	0.81

Table 2: Demographic characteristics at baseline and across groups

*Note*: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a t-test of equality of estimated parameters in Col. (1) and (2) and in Col. (1) and (3) respectively. Higher Degree includes higher grade, andvanced higher, CSYS, A level, GNVQ/GSVQ advanced, SVQ level 3First Degree, Higher degree, SVQ Level. No Qualifications corresponds to respondents who ticked the No Qualifications option. A descriptive statistics table for panel A Edinburgh and panel B Colchester can be found in Table B.4. in Appendix B. Pregnant women at baseline: 6 in the control group, 4 in the Meal treatment, 1 in the Snack treatment.

	Very easy/ easy	Neutral	Difficult/ Very difficult
A. Snack (N=80)			
To stick to meal times	41.2	30	28.8
To stick to meal and snack times (child)	57.5	25	17.5
Not to snack	33.7	23.8	42.5
Not to snack (child)	27.4	41.3	31.3
B. Meal (N=123)			
To cook meals	83.7	13	3.3
To stick to the recipe	61	25.2	13.8

Table 3: Self-reported feedback on the ease of implementation of the protocols

*Note*: Information collected after the 12-week treatment. All numbers are in percentages.

	Strongly disagree /Disagree	Neither agree nor disagree	Agree/ Strongly agree
A. Snack (N=80)			
I found myself eating more at meal times	21.3	30	48.7
I was surprised at how much I used	13.7	21.3	65
to snack before starting the study			
I felt less hungry between meals	26.2	31.3	42.5
I generally felt I ate less food overall during the day	22.5	31.3	46.2
I have tried new foods that I had never tried before	27.8	7.4	64.8
Cooking the recipes was time consuming	44.3	30.3	25.4
B. Meal (N=123)			
My child has tried new foods he/she	10.6	9.8	79.6
had never tried before			
I have liked an ingredient that I thought	18.9	13.9	67.2
I did not like before			

Table 4: Self-reported feedback on effects of the protocols

*Note*: Information collected after the 12-week treatment. All numbers in percentages.

	Control	Meal	Snack	$\begin{array}{c} \text{P-value} \\ (1) = (2) \end{array}$	$\begin{array}{c} \text{P-value} \\ (1) = (3) \end{array}$
A. Children					
I. Item categories					
Sweets	3.6(0.7)	3.6(0.8)	3.6(0.6)	0.25	0.99
Bread	3.4(0.9)	3.6(0.6)	3.3(0.9)	0.03	0.65
Processed food	3.3(0.6)	3.5(0.5)	3.3(0.7)	0.03	0.67
Fruit	3.2(0.8)	3.3(0.7)	3.1(0.8)	0.28	0.47
Cheese	3.1(1.1)	3.4(1.0)	3.4(0.9)	0.02	0.04
Meat/Fish/Eggs	2.6(0.9)	2.7(0.9)	2.8(0.9)	0.77	0.32
Vegetables	2.6(0.8)	2.6(0.8)	2.5(0.8)	0.88	0.35
II. Meals					
Tuna pasta	2.5(1.3)	2.7(1.3)	2.7(1.2)	0.33	0.36
Omelette	2.3(1.3) 2.4(1.3)	2.7(1.3) 2.3(1.3)	2.7(1.2) 2.2(1.2)	0.55	$0.30 \\ 0.42$
Baked potato	2.4(1.3) 2.4(1.2)	2.3(1.3) 2.2(1.2)	2.2(1.2) 2.3(1.2)	0.37	0.63
Turkey stir fried	2.1(1.2) 2.2(1.3)	2.1 (1.2) 2.1 (1.2)	1.9(1.1)	0.65	0.26
Salmon with onions	2.1 (1.2)	2.3(1.2)	2.2 (0.2)	0.27	0.20 0.67
	~ /	~ /	~ /		
B. Adults					
I. Item categories					
Fruit	3.4(0.5)	3.5(0.5)	3.3(0.5)	0.11	0.21
Meat/Fish/Eggs	3.3(0.6)	3.3(0.6)	3.3(0.6)	0.62	0.87
Cheese	3.3(0.7)	3.5(0.7)	3.5(0.7)	0.11	0.06
Vegetables	3.2(0.6)	3.2(0.6)	3.2(0.5)	0.96	0.81
Bread	3.2(0.7)	3.2(0.7)	3.3(0.7)	0.93	0.29
Processed food	3.1(0.5)	3.0(0.5)	3.1(0.5)	0.71	0.35
Sweets	2.8(0.7)	2.8(0.6)	2.9(0.6)	0.68	0.59
II. Meals					
Turkey stir fried	3.2(0.9)	3.2(1.0)	3.3(0.9)	0.62	0.38
Salmon with onions	3.2(0.3) 3.1(1.1)	3.2(1.0) 3.0(1.1)	3.1(1.1)	$0.02 \\ 0.52$	$0.38 \\ 0.83$
Omelette	3.1(1.1) 3.1(1.0)	3.0(1.1) 3.2(0.9)	3.0(1.0)	0.52 0.77	$0.85 \\ 0.54$
Tuna pasta	3.1(1.0) 3.1(1.0)	3.0(1.0)	2.9(1.0)	0.45	$0.34 \\ 0.21$
Baked potato	3.0(0.8)	3.2(0.8)	3.1 (0.9)	0.03	0.16

Table 5: Baseline food preferences

Note: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a t-test of equality of estimated parameters in Col. (2), against those in columns (3) and (4) respectively. An item that has never been tried or for which the participants declares to be allergic to is considered missing. 1 corresponds to not liking at all, 4 to liking very much.

			Meat Fish	Processed			
	Fruits	Vegetables	Eggs	Food	Sweets	Bread	Cheese
Panel A: Children							
After	$0.1^{*}$	-0.1	0.0	0.1	-0.1**	0.1	$0.2^{**}$
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
1-year	0.0	0.0	0.2	0.1	-0.1	0.0	0.0
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Meal x After	-0.1	0.0	-0.1	-0.2***	$0.2^{**}$	-0.2*	-0.3***
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Meal x 1-year	0.0	0.0	-0.1	-0.1	0.1	-0.1	-0.1
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.2)
Snack x After	0.0	0.1	-0.1	0.0	$0.2^{**}$	0.2	-0.2
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.2)
Snack x 1-year	0.2	0.1	-0.3	0.0	0.0	0.2	-0.1
	-0.1	-0.1	-0.2	-0.1	-0.1	-0.1	-0.2
Constant	$3.2^{***}$	$2.6^{***}$	$2.7^{***}$	$3.4^{***}$	$3.5^{***}$	$3.5^{***}$	3.3***
	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)
# Obs	700	700	699	700	695	697	692
R-squared	0.02	0	0.01	0.02	0.01	0.02	0.02
# individuals	286	286	286	286	285	286	284
Panel B: Adults							
After	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	(0.0)	(0.0)	(0.0)	(0.0)	(0.1)	(0.1)	(0.0)
1-year follow up	0.0	0.0	0.0	0.0	0.0	-0.1*	-0.1*
	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.1)	(0.1)
Meal*After	0.1	0.0	0.0	$0.1^{*}$	0.0	-0.1	0.00
	(0.0)	(0.0)	-0.1	(0.0)	(0.1)	(0.1)	(0.1)
Meal*1-year	0.0	0.0	-0.1	0.0	0.0	0.1	0.1
	(0.0)	(0.0)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Snack*After	0.1	0.1	-0.1	0.0	0.0	-0.1	-0.1
	(0.1)	(0.0)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Snack*1-year	0.0	0.0	-0.1	0.0	0.1	0.0	0.0
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Constant	3.4***	3.2***	3.3***	3.1***	$2.8^{***}$	3.2***	3.4***
	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)
# Obs	1,029	1,029	1,026	1,029	1,024	1,006	1,010
R-squared	0.01	0	0	0.01	0	0.02	0.01
# individuals	379	379	379	379	378	377	376

Table 6: The impact of meal and snack treatment on food preferences

 $\overline{Note}$ : Each column is from a separate regression. All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\* p<0.01, \*\* p<0.05, \* p<0.1.

	UK daily Recommendation	Control	Meal	Snack	$\begin{array}{l} \text{P-value} \\ (1) = (2) \end{array}$	$\begin{array}{c} P-value\\ (1)=(3 \end{array}$
A: Children						
Total calorie intake (Kcal)	1800	1438.9	1463.8	1383.2	034	0.93
rotal calorie intalle (rotal)	1000	(538.6)	(475.4)	(378.0)	0.101	0.00
# fruit	5 portions fruits and	0.9	1.1	1.1	0.42	0.45
	-	(1.26)	-1.4	-1.42		
# vegetables	Veg.	0.3	0.4	0.4	0.26	0.23
		(0.58)	(0.85)	(0.78)		
Fruit and veg (g)	Min 400	101.5	122.4	123.5	0.23	0.27
		(124.7)	(126.8)	(141.1)		
Total Fat (g)	Max 70	56.5	59.5	55.1	0.4	0.9
~		(24.0)	(25.8)	(20.7)		
Carbohydrate (g)	Max 220	194.7	190.2	182.2	0.77	0.91
		(86.7)	(65.3)	(50.5)	0.00	0 <b>-</b>
Protein (g)	Min 24	47.8	52.5	48.9	0.08	0.5
Saturatos (g)	Mar 20	(18.8)	(20.2)	(16.7)	0.99	0.04
Saturates (g)	Max 20	23.9	25.9	23.8	0.23	0.94
Suman (m)	Mor. 95	(11.9)	(12.9)	(11.6)	0.9	0.92
Sugar (g)	Max 85	94.2	97.0	87.3 34.3	0.2	0.83
NMES (g)	Max 23	(58.6) 18	$(47.1) \\ 25.9$	-34.3 18.1	0.21	0.41
NMES (g)	Max 25	(22.6)	(34.8)	(20.1)	0.21	0.41
Fibre AOAC (g)	Min 15	(22.0) 11.0	(34.8) 10.5	(20.1) 12.0	0.77	0.24
Pible AOAO (g)	Will 19	(5.1)	(5.2)	(5.7)	0.11	0.24
Sodium (mg)	2000-3000	(5.1) 1575.9	(5.2) 1621.7	1625	0.93	0.71
Sourium (mg)	2000-0000	(699.9)	(899.8)	(692.8)	0.55	0.11
# Obs		112	104	73		
			-			
B: Adults (main & second)		00000 1	1048.0	2026 5	0.07	0.01
Total calories intake (Kcal)	2000-2500	2036.1	1843.9	2036.5	0.07	0.91
	F C 1	(798.1)	(685.2)	(809.2)	0 59	0 70
Portions of fruit	5 portions fruits and	0.94	0.81	1.03	0.53	0.78
Doutions of remotables	Veg.	(1.86)	(1.48)	(2.67)	0.49	0.69
Portions of vegetables		0.77	0.88	0.87	0.48	0.62
Fruit and veg (g)	Min 400	(1.15)	$(1.35) \\ 135.6$	(1.03) 151.9	0.93	0.66
Fruit and veg (g)	MIII 400	137.5			0.95	0.00
Total Fat (g)	Max 70	(184.8) 84	$(160.9) \\ 74.7$	$(295.6) \\ 83.4$	0.13	0.9
10000 100 (8)	IVIGA TU	(42.7)	(35.0)	(42.3)	0.10	0.9
Carbohydrate (g)	Max 260	(42.1) 241	(35.0) 223.9	(42.3) 248.9	0.45	0.68
Carsonjarate (8)	1110A 200	(118.6)	(90.0)	(122.1)	0.10	0.00
Protein (g)	Min 45	79.2	70.1	69.1	0.05	0.21
(0)		(55.1)	(32.6)	(27.2)	0.00	J. <b>_</b> 1
Saturates (g)	Max 20	30.2	28.8	30.2	0.63	0.69
(6)		(17.5)	(16.1)	(16.0)		
Sugars (g)	Max 90	107.1	97.9	116.1	0.76	0.65
0 (0)		(88.2)	(56.7)	(99.1)		
NMES (g)	Max 30	33.2	31.5	41.3	0.08	0.06
		(61.7)	(41.0)	(63.0)		
Fibre AOAC (g)	24	13.8	13.5	14.6	0.95	0.72
		(6.9)	(6.2)	(8.2)		
Sodium (mg)	2400	2329.4	2139.1	2440.4	0.17	0.88
		(1246.3)	(1244.6)	-1817		
Alcohol (g)	0	7.1	6.0	9.4	0.71	0.09
		(22.5)	(16.6)	(21.1)		
# Obs		134	124	79		

Table 7:	Baseline	measures	of	dietarv	intake

Note: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a Wilcoxon test of equality of means. 1 portions of fruit or veg  $\approx 80g$ .

	Energy (cal)	Fruits (g)	Veg (g)	Total fat (g)	Carbs (g)	Protein (g)	Sat. fat (g)	Total Sugar (g)	$\frac{\rm NMES}{\rm (g)}$	Fibre (g)	Sodium (mg)	Alcohol (g)
A: Children Sign of the UK recommendation - baseline consumption	+	+	+	+	+	-	-	-	+	+	+	
After	-67.6 (51.3)	3.6 (5.6)	10 (12.6)	-2.6 (2.6)	-13.7 (8.4)	1.6 (2.2)	-1.6 (1.2)	-8.6(5.5)	-3.5 (3.0)	0.1 (0.7)	-79.8 (84.3)	
1 year follow-up	(31.3) $128.8^{*}$ (74.3)	(3.0) $49.6^{***}$ (13.9)	(12.0) $105.5^{***}$ (20.8)	(2.0) 0.3 (3.2)	(3.4) $32.6^{***}$ (11.8)	(2.2) 2.7 (3.1)	(1.2) -1.6 (1.4)	(3.3) $23.2^{***}$ (7.3)	(5.0) 57.7*** (6.2)	(0.7)	(34.3) 158.9 (112)	
Meal*After	-37.2 (70.2)	(13.3) -9.9 (9.4)	-4.6 (18.7)	(3.2) -4.1 (3.7)	(11.0) 4.3 (10.8)	(3.1) -3.6 (3.4)	(1.4) -2.4 (1.8)	(7.3) -0.4 (7.8)	(0.2) -4.6 (5.2)	0.7 (0.9)	-61.1 (114.)	
Meal*1 year	(10.2) -127.8 (106.4)	(5.4) 17.7 (25.3)	(10.7) 0.4 (42.3)	(5.7) -7.1 (5.3)	(10.8) -12.2 (15.6)	(3.4) -4.8 (4.4)	(1.8) -3.5 (2.5)	(7.3) $-17.1^{*}$ (10.1)	(3.2) $-21.7^{***}$ (8.0)	(0.3)	(114.) -104 (159.4)	
Snack*After	(100.4) -53.1 (75.1)	(25.5) -6.5 (11.1)	(42.3) -15.5 (20.8)	(3.5) -3.5 (4.1)	(15.0) 0.3 (11.6)	(4.4) -4.7 (3.4)	(2.3) -1.3 (2.1)	(10.1) -2.1 (7.3)	(0.0) -0.7 (4.7)	-0.6 $(1.1)$	(103.4) -120.6 (137.6)	
Snack*1 year	(13.1) -147.4 (99.4)	(11.1) -40.2** (17.4)	(20.8) -27.9 (34.9)	(4.1) -3.4 (4.8)	(11.0) -23.9 (14.9)	(3.4) -6.7 (4.2)	(2.1) -1.5 (2.4)	-14.8 (10)	(4.7) -19.2** (8.7)	(1.1)	(137.0) -159.3 (173.9)	
Constant	(33.4) 1,417.2*** (18.8)	(11.4) $31.7^{***}$ (2.7)	(54.5) 87.1*** (5.6)	(4.0) 56.7*** (1)	(14.5) $187.4^{***}$ (2.8)	(4.2) $49.4^{***}$ (0.8)	(2.4) 24.5*** (0.5)	$ \begin{array}{c} (10) \\ 92.3^{***} \\ (2) \end{array} $	(0.1) 20.8*** (1.5)	$11.0^{***}$ (0.2)	$\begin{array}{c}(110.5)\\1,565.4^{***}\\(31.0)\end{array}$	
# Obs.	804	675	738	804	804	804	804	804	802	560	804	
R-squared # of ind.	$0.03 \\ 292$	$0.13 \\ 292$	$0.12 \\ 290$	$0.02 \\ 292$	$0.07 \\ 292$	$\begin{array}{c} 0\\ 292 \end{array}$	$0.03 \\ 292$	$0.07 \\ 292$	$0.41 \\ 291$	$\begin{array}{c} 0\\ 289 \end{array}$	$0.02 \\ 292$	
<b>B: Adults</b> Sign of the UK recommendation-baseline consumption	+	+	+	-	+	-	-	-	-	+	+	-
After	$-250.9^{***}$ (86.6)	-15.1 (9.4)	$-28.8^{*}$ (15.5)	-9.5* (5.0)	$-32.3^{***}$ (10.4)	-6.9 (6.1)	-2.5 (2.1)	$-20.6^{***}$ (7.6)	-10.7* (5.6)	$-2.0^{**}$ (0.8)	$-271.3^{*}$ (153.5)	-2.2 (1.6)
1 year follow-up	$-422.9^{***}$ (109.6)	(9.4) $68.5^{***}$ (21.5)	(15.5) $126.5^{***}$ (24.4)	(5.0) -26.6*** (5.4)	(10.4) -20.2 (14.3)	(0.1) -22.6*** (6.5)	(2.1) -8.0*** (2.6)	-0.3 (10.2)	(3.0) $39.2^{***}$ (8.4)	(0.8)	(133.3) -449.1** (181.2)	(1.0) $31.3^{**}$ (12.4)
Meal*After	(105.0) 88.4 (117.1)	(21.5) -2.5 (15.5)	(24.4) -4.4 (19.7)	(5.4) 3 (6.4)	(14.5) 11.6 (15)	(0.5) 2 (7.4)	(2.0) -1.2 (2.8)	(10.2) 3.4 (9.6)	(0.4) -0.7 (7.2)	1.3 (1.2)	(101.2) -11 (216.2)	(12.4) 1 (2.3)
Meal*1 year	(117.1) $314.3^{*}$ (186.3)	(10.0) -32.3 (30)	(13.7) 10.8 (36)	(0.4) 13.7 (8.4)	(13) 29.3 (23.9)	(1.4) $15.8^{*}$ (9)	(2.8) 1.6 (3.8)	5.6 (12.7)	(1.2) -4.1 (9.9)	(1.2)	(210.2) 235.7 (302.8)	(2.5) -7.5 (14.7)
Snack*After	(130.3) -177.5 (133.2)	-20.4 (18.6)	(30) -12.4 (43)	(7.2)	(23.5) -24.5 (19.1)	(3) -3.7 (7.3)	(3.8) -3.8 (3.1)	(12.7) -23.2 (14.2)	(9.5) -10.5 (9.6)	-0.8 (1.3)	-367.9 (274.7)	(14.7) -0.5 (3.7)
Snack*1 year	(135.2) 78.8 (165.8)	(13.0) -17 (37.5)	(43) 49 (65.7)	(7.2) 3.4 (8.1)	(19.1) -4 (24.2)	(7.3) 21.1** (8.4)	(3.1) 1.5 (3.6)	(14.2) -18.1 (16.8)	(5.0) -15.8 (12.9)	(1.5)	(274.7) -177.7 (325.0)	(3.7) -16.1 (15.6)
Constant	(105.3) 1,963.1*** (35.7)	(37.3) $70.8^{***}$ (4.3)	(05.7) $80.1^{***}$ (6.8)	(0.1) $80.2^{***}$ (1.7)	$\begin{array}{c} (24.2) \\ 236.6^{***} \\ (4.8) \end{array}$	(3.4) $73.9^{***}$ (1.9)	(3.0) 29.5*** (0.7)	(10.8) $105.8^{***}$ (3)	$(12.9) 34.2^{***} (2.2)$	$14.2^{***}$ (0.2)	(323.0) 2,304.4*** (64)	(15.0) $7.5^{***}$ (0.7)
# Obs.	926	781	783	925	926	926	925	926	921	628	926	677
R-squared # of ind.	$0.06 \\ 359$	$\begin{array}{c} 0.09\\ 347 \end{array}$	$0.23 \\ 347$	$\begin{array}{c} 0.08\\ 358 \end{array}$	$\begin{array}{c} 0.04 \\ 359 \end{array}$	$0.05 \\ 359$	$\begin{array}{c} 0.05 \\ 358 \end{array}$	$\begin{array}{c} 0.05\\ 359 \end{array}$	$0.22 \\ 359$	$\begin{array}{c} 0.05 \\ 338 \end{array}$	$\begin{array}{c} 0.04\\ 359 \end{array}$	$0.18 \\ 339$

Table 8: The impact of meal and snack treatments on total calorie intake and intake of macronutrients

*Note*: All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\* p<0.01, \*\* p<0.05, \* p<0.1. Intake of fibre is not available one year follow up as they are not calculated by the diet recall software (Intake24) used in the 1 year follow up surveys

	Control	Meal	Snack	$\begin{array}{l} \text{P-value} \\ (1) = (2) \end{array}$	$\begin{array}{c} \text{P-value} \\ (1) = (3) \end{array}$
A. Children					
% Underweight	3.9	3.1	4.6	0.99	0.98
% Normal weight	71.8	78.6	78.5		
% Overweight	18.5	12.2	13.8		
% Obese	5.8	6.1	3.1		
#  Obs	103	98	65		
B. Adults (main & second)	27.0	07.0	20	0 51	0 70
Mean BMI Men	27.9	27.6	28	0.71	0.76
	(4.8)	(5.2)	(4.5)		
Mean BMI Women	29.5	27.8	27	0.14	0.04
	(7.5)		· · ·		
% Underweight	0.7	1.5	2.2	0.65	0.65
(BMI < 18)					
% Normal weight	29.3	38.6	38		
(BMI 18-25)					
% Overweight	32.7	28.1	32.6		
(BMI > 25)					
% Obese	37.3	31.8	27.2		
(BMI > 30)					
# Obs	150	132	92		

Table 9: Descriptive statistics of body measurements

Note: To calculate BMI categories we categorize children from 2 to 18 years as normal weight, overweight or obese, using BMI cut-offs recommended by the Childhood Obesity Working Group of the International Obesity Taskforce. BMI is in  $kg/m^2$ . The categories are based on cut-offs from British 1990 growth reference see page 5 Underweight: 2nd centile for population monitoring and clinical assessment, Overweight: 85th centile for population monitoring, 91st centile for clinical assessment, Obese: 95th centile for population monitoring, 98th centile for clinical assessment. 11 women in our sample are pregnant and are thus removed from this analysis (6 in the control group, 4 in the meal, 1 in the snack treatments). P-values from Kolmogorv-Smirnov test of distribution are reported to compare the BMI categories distribution between groups, signed rank tests were performed to compare BMI levels.

		Children		Adults	
	Perc. BMI	Overweight or Obese	BMI	Overweight or Obese	Obese
After	0.02	0.01	0.09	0.00	0.00
	(0.01)	(0.03)	(0.15)	(0.02)	(0.02)
1 year follow up	0.00	-0.05	$0.28^{*}$	0.00	0.00
	(0.01)	(0.03)	(0.16)	(0.02)	(0.02)
Meal*After	-0.05**	-0.05	-0.01	-0.01	-0.01
	(0.02)	(0.05)	(0.22)	(0.03)	(0.03)
Meal*1 year	-0.06***	0.02	0.04	-0.01	-0.01
-	(0.02)	(0.05)	(0.23)	(0.03)	(0.03)
Snack*After	-0.06***	-0.07	-0.15	0.02	0.02
	(0.02)	(0.05)	(0.25)	(0.04)	(0.04)
Snack*1 year	-0.04*	0.08	-0.05	0.04	0.04
	(0.02)	(0.06)	(0.27)	(0.04)	(0.04)
Constant	0.63***	0.20***	28.21***	0.63***	0.63***
	(0.01)	(0.01)	(0.07)	(0.01)	(0.01)
# Obs	785	762	1,020	1,026	1,026
R-squared	0.05	0.02	0.01	0	0
# individuals	288	283	380	380	380

Table 10: The impact of the meal and snack treatment on BMI, overweight and obesity

*Note*: All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\* p<0.01, \*\* p<0.05, \* p<0.1. Column (1) is a continuous variable of the BMI percentile in children. In column (2) the outcome variable is equal to 1 for overweight and obese adults, 0 otherwise and is performed. The independent variable in columns (3) is a continuous variable corresponding to the BM. We use the same dummy variable as in column (2) but for adults in column (4). In column (5) the Obese variable takes value of 1 is participants are obese, 0 otherwise. Linear probability models (LPM) models are performed for dummy variables.

	Number of low
	calorie choices
After	0.1
	-0.2
Meal*After	-0.9***
	-0.3
Snack*After	-0.7**
	-0.3
Constant	$4.4^{***}$
	-0.1
#  Obs	503
# ind.	268
R-squared	0.07

Table 11: Number of low-calories choices, incentivized

Note: All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\* p<0.01, \*\* p<0.05, \* p<0.1. Sample includes only adults from Colchester

	Normal ranges ranges	Control	Meal	Snack	$\begin{array}{c} \text{P-value} \\ (1)=(2) \end{array}$	$\begin{array}{l} \text{P-value} \\ (1)=(3) \end{array}$
Nefa (nmol/L)	0.00-0.72	0.4(0.2)	0.4(0.2)	0.4(0.2)	0.87	0.94
Insulin $(mIU/L)$	< 25	13.2(1.1)	11.4(5.4)	11.5(9.1)	0.40	0.58
Triglyceride (nmol/l)	< 2	1.1(0.9)	1.2(0.9)	0.9(0.4)	0.70	0.31
HDL cholesterol (nmol/L)	> 1	1.5(0.4)	1.4(0.4)	1.4(0.4)	0.53	0.71
Glucose (nmol/L)	< 6.1	4.6(0.7)	4.5(0.5)	4.4(0.6)	0.88	0.28
LDL chol $(nmol/L)1$	< 3	3.0(0.7)	2.5(0.6)	2.6(2.3)	0.00	0.04
CRP (mg/L)	< 3	4.5 (9.8)	3(4.5)	4.8(7.1)	0.37	0.91
Total Antioxidant Status	1.3 - 1.77	1.5(0.2)	1.5(0.2)	1.6(0.09)	0.62	0.07
#  Obs		34	40	23		

Table 12: Baseline health biomarkers (based on fasted blood samples) Levels

*Note*: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a t-test of equality of estimated parameters in Col. (1) and (2) and in Col. (1) and (3) respectively. Sample is for adults only in Edinburgh. LDL calculated by: Total cholesterol-HDL-(Triglyceride/2.2)

	Nefa	Triglycerides	HDL	LDL	Glucose	Insulin	CRP	TAS
After	0.0	-0.1	0.0	$-0.2^{*}$	-0.2**	0.8	-2.1	0.1
	(0.0)	(0.1)	0.0	(0.1)	(0.1)	(1.3)	(1.7)	(0.0)
Meal * After	0.0	0.0	0.0	$0.3^{**}$	0.1	-1.7	1.2	0.0
	(0.1)	(0.1)	(0.0)	(0.1)	(0.1)	(1.8)	(1.9)	(0.1)
Snack * After	0.0	0.1	0.0	0.2	$0.3^{*}$	4.8	-0.9	-0.1
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(5.7)	(2.6)	(0.1)
Constant	0.4***	1.1***	1.5***	2.7***	4.5***	11.7***	4.0***	1.5***
	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.6)	(0.4)	(0.0)
# Obs.	195	195	195	195	195	195	195	195
# ind.	106	106	106	106	106	106	106	106
R-squared	0.04	0.04	0	0.06	0.08	0.04	0.06	0.04

Table 13: The impact of the meal and snack treatment on blood biomarkers

 $\overline{Note}$ : All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\* p<0.01, \*\* p<0.05, \* p<0.1. Sample includes adults only from Edinburgh.

# **ONLINE APPENDIX**

## **Supplementary Material**

# The Formation and Malleability of Dietary Habits: A Field Experiment with Low Income Families

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## Appendix A: Recruitment and experimental materials given to the participants

Figure A.1A: Leaftlet and poster for recruitment in Edinburgh



#### Families sought for University of Edinburgh Health Study

Do you have one child or more between the age of 2 and 6? Do you want to earn some money while participating in a study?

We are running a study looking at **nutrition, lifestyle and health** among families on low incomes in Edinburgh. A compensation of  $\pounds_{350}$  in total will be offered to each family.

What does it involve? You will have to visit our study premises for six 3-hour sessions over the course of 3 years. The study includes questionnaires; weight and height measurement, blood sample collection for adults (twice only). Some people (selected at random) will be asked to follow specific nutritional advice for 12 weeks (in March-May 2015). Costs associated with being in the study are covered by us.

**When?** You would visit us 6 times, (1) Feb 2015, (2) Mar-Apr 2015, (3) Jun 2015, and (4) Feb 2016, (5) Feb 2017, (6) Feb 2018.

Where? Sessions take place at the University of Edinburgh campus (address overleaf). Interested? Register on http://bluehealthstudy.ed.ac.uk

If you require more info email <u>healthstudy@ed.ac.uk</u> If you have no internet access text us on 07981112493



#### To take part you must:

- have lived in Edinburgh for at least 2 years
- $\checkmark$  Have a child who is between 2 and 6
- ✓ be fluent in English
- ✓ Having an annual income below £26,426 per household
- ✓ own a fridge and a hob
- ✓ not be pregnant

#### Facebook: www.facebook.com/HealthstudyEdinburghUni

Twitter: @bluehealthstudy

Address: 31 Buccleuch Place, EH8 9JT Edinburgh (close to Clerk Street and The Meadows)

Bus routes: 2, 3, 5, 7, 8, 29, 30, 31, 33, 37, 41, 42, 47, 49, 67

#### NO WHEELCHAIR ACCESS





#### Figure A.1B: Leaftlet and poster for recruitment in Colchester

#### An exciting new Colchester-based research study

If you have a child between the age of 2 and 6 or are expecting a baby, you could be eligible to take part in a new research study involving leading universities across Europe.

The study aims at understanding the drivers of food preferences among children and how they change over time. As a participant, you'll visit our study centre at the University of Essex where we'll ask you and your child questions about your food habits and take body measurements.

You will receive  $\pounds 100$  for the first visit. You will be invited to come six times over three years (for a total compensation of  $\pounds 400$ )

#### Taking part

In order to take part you must:

- Have a child between 2 and 6 or be pregnant (due in 2016)
- Have lived in Colchester area for at least 2 years and have a postcode starting with CO
- Be fluent in English
- · Have a gross annual household income below £26,600
- Own a fridge and a hob

Find out more and register at www.bluehealthstudy.ed.ac.uk of call 07981 112 493.

The University of Edinburgh is a charitable body, registered in Scotland, with registration number SC005336.



Table A.1. Exclusion criteria

Adults: pregnancy, existing diagnosis of serious illness (e.g. Diabetes Type I or Type II, Cancer, Stroke, epilepsy, heart attack or angina), having received medical advice to change diet in the previous 12 months. Also excluded, adults with self-reported prior or existing severe food allergies where they are required to carry an epipen and self-reported eating disorders within the last year. Parents/students younger than 16 years old will also be excluded from the study.

Children: existing diagnosis of serious illness (e.g. Diabetes Type I or Type II, Cancer), having received medical advice to change diet in the previous 12 months. Also excluded, children with prior or existing severe food allergies where they are required to carry an epipen.

Participant families are excluded where different family members follow very different diets form each other due to health or choice reasons, which make it logistically very difficult to cater for (e.g. 4 family members where each member has different stringent dietary requirements).

Families who do not own a hob and a fridge for cooking, due to the cooking element involved in the protocol.



# Healthy Eating Information Leaflet

This leaflet summarises general recommendations for a healthy diet. These recommendations are based on research in nutrition and are in accordance with the current UK guidelines. As our study aims at understanding what drives dietary choices and how diet affects health for people of different age groups we provide you with this leaflet just for your own information. You do not have to follow these guidelines if you do not feel like it. You probably know these recommendations already, but we summarise them here again for information.

#### **The Eatwell Plate**

On the next page is a picture of what is called the "Eatwell Plate", which highlights the different types of food that make up our diet, and shows the right proportions to have a well-balanced and healthy diet. The balance doesn't need to be right at every meal but overtime such as a whole day or week.

A simple rule of thumb for a healthy diet is to **go for options that are lower in fat, salt and sugar and to** <u>avoid processed foods</u> as much as possible. Processed foods are foods that are not in their natural state (example: ready meals, chocolate bars, candies, crisps, sodas), which often contain high amounts of salt, sugar and fat; examples of non-processed foods are vegetables, fruit, nuts, seeds, legumes, uncooked and unprepared meat or fish, eggs. Non-processed foods are generally healthier. Shifting the diet towards unprocessed foods is one of the easiest ways of making sure we know what you are actually eating. Also, pasta with brown rice, brown bread and wholegrain pasta are healthier options than white rice, white bread and white pasta.

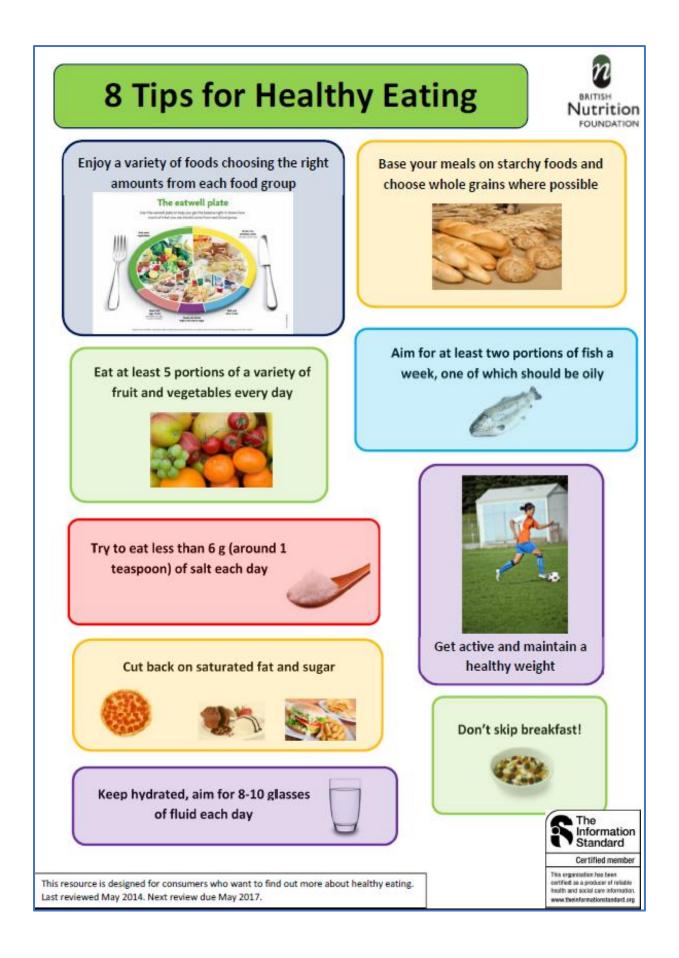
Note that the eatwell plate doesn't apply to children under the age of two because they have different nutritional needs. Between the ages of two and five, children should gradually move to eating the same foods as the rest of the family, in the proportions shown on the eatwell plate.

# The eatwell plate



Use the eatwell plate to help you get the balance right. It shows how much of what you eat should come from each food group.





#### Food labels can help

Food labels can help to understand the fat, salt and sugar content of a specific food item.

The table below provides a helpful guide to what is considered 'low' and 'high' for sugar, fat, saturates and salt or sodium.

For example, to cut down on saturated fat, it is important to decrease the consumption of foods that have more than 5g of saturated fat per 100g, as these are considered high in saturated fat.

#### High and low

Nutrient	Low (per 100g)	High (per 100g)
Sugar	5g or less	22.5 g or more
Fat	3g or less	17.5 g or more
Saturates (saturated fat)	1.5g or less	5g or more
Salt (sodium)	0.3g salt (0.1g sodium) or less	1.5g salt (0.4g sodium) or more

# The following simple swaps are an example on how to cut down the consumption of fat and sugar:

- ✓ Sugary cereals to plain cereals
- Whole milk to semi-skimmed milk (REMEMBER CHILDREN UNDER THE AGE OF 2 MUST DRINK FULL FAT MILK)
- ✓ Semi-skimmed milk to 1% fat or skimmed milk
- Butter to lower fat or spread
- Cheese to reduce fat cheese (REMEMBER CHILDREN UNDER THE AGE OF 2 MUST EAT FULL FAT CHEESE)

#### **Useful websites:**

http://www.nutrition.org.uk/

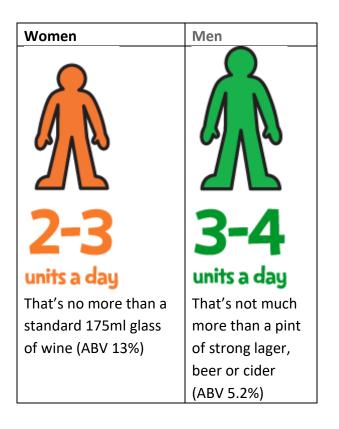
http://www.nhs.uk/Livewell/Goodfood/Pages/eatwell-plate.aspx

http://tna.europarchive.org/20100929190231/http://www.eatwell.gov.uk/healthydiet/eatwellplate

#### Alcohol units and guidelines

Alcohol should also be taken into account in evaluating a diet – alcoholic drinks also contain calories and nutrients. It can be a bit tricky to understand and remember how much alcohol is in drinks, and how this can affect our health. The lower risk guidelines can help with this. There's one for women and one for men.

No one can say that drinking alcohol is absolutely safe, but by keeping within these guidelines, there's only a low risk of causing harm in most circumstances.

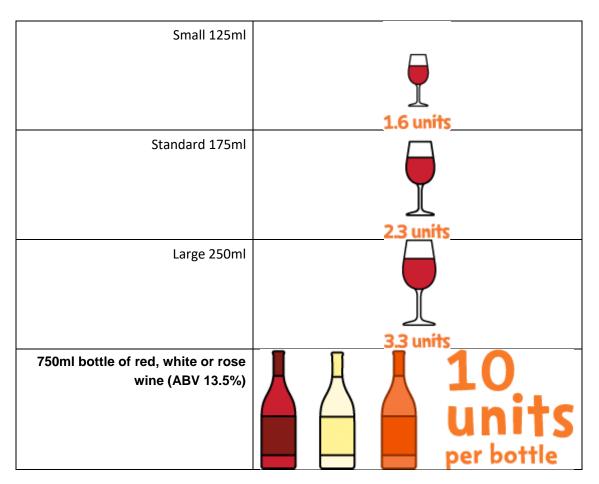


\* "Regularly" means drinking this amount most days or every day.

#### Counting the units

The amount of alcohol in drinks can vary quite widely. To calculate how many units are in the usual tipple we must refer to the "ABV" (available on the side of the bottle or can )which indicates the percentage of alcohol in the drink.

#### Glass of red, white or rose wine (ABV 13%)



#### Beer, lager and cider

Regular (ABV 4%)	lager	1.8 units	2.3 units
Strong (ABV 5.2%)	lager	2.2 units	3 units
Extra strong (ABV 8%)	lager	3.5 units	4.5 units

### Other drinks (ABV varies)

25ml single spirit and mixer (ABV 40%) 1 units 275ml bottle of alcopop (ABV 5.5%) 1.5 units

http://www.nhs.uk/change4life/Pages/alcohol-lower-risk-guidelines-units.aspx

	List of snacks per week	Quantities for one snack
	6 pieces of fruit (e.g. bananas, apple, pear, orange)	1 piece of fruit
Dogular baskat	1 low fat soft cheese (200g) with 1 packet of oat cakes (should be enough for 2 snacks)	soft cheese with oat cakes according to your child appetite and needs
Regular basket	4 yogurts of 125g each	1 snack. Please do not add any sugar or honey.
	1 low fat hummus (200g) with 1 packet batons carrots (should be enough for two snacks)	hummus with carrots according to your child appetite
Dairy from	7 pieces of fruit (e.g. bananas, apple, pear, orange)	1 piece of fruit
Dairy free	1 low fat hummus (300g) with 1 packet batons carrots	hummus with carrots according to your child appetite and needs
	1 avocado with 1 packet of oat cakes	Half of an avocado with oat cakes

### A.3. Snacks and recommendations for children (Snack treatment)

### A reminder of the main recommendations for children:

• Stick to regular meal times (same as for the parents)



- In addition to the main meals, children can also get a snack in the morning and in the afternoon. Toddlers (2-4 year old) can consume a third additional snack during the day
- You will try to provide your children with snacks <u>at very</u> <u>regular times</u> during the day (for example 10 am, 3 pm).

## A.4. Feedback leaflet to be filled in by the participants in the Meal treatment

### Example for week 1

WEEK 1 - from September 14th Recipe description	Preparation Difficulty 1 very easy, 2 easy, 3		<b>Did it taste good?</b> (Circle as appropriate)		
	moderate, 4 difficult, 5 very difficult				
Date:		XP	••	•••	:;
Meal:		YUCK!	MEH	GOOD	AWESOME!
Date:					
Meal:		× P	•••	•••	<u>ت</u>
		YUCK!	MEH	GOOD	AWESOME!
Date:					
Meal:		× P		•••	9
		YUCK!	MEH	GOOD	AWESOME!
Date:		>	••	•••	
Meal:					<u> </u>
		YUCK!	MEH	GOOD	AWESOME!
Date:		<b>*</b>	••	•••	::
Meal:		YUCK!	MEH	GOOD	AWESOME!

## A.5. Feedback leaflet to be filled in by the participants in the Snack treatment

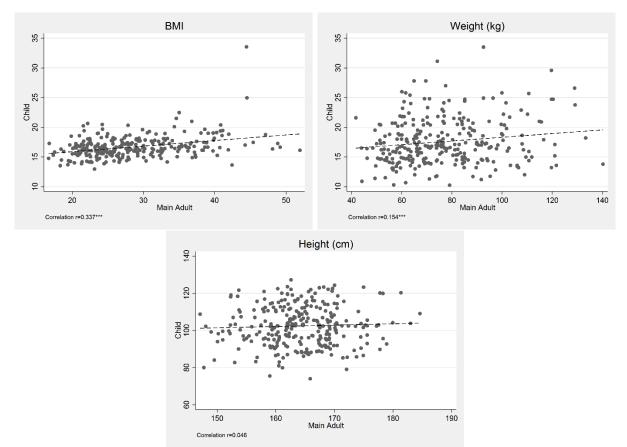
<u>MAIN STUDY</u> ADULT	Day off Protocol (Tick appropriate day)	Breakfast time	Lunch time	Dinner time	Deviations from protocol (snacks between meals) Yes / No
Monday		8:00	13:00	17:30	No
Tuesday		8:00	13:00	17:30	No
Wednesday		8:00	13:00	17:30	No
Thursday	Х	8:30	12:00	17:30	Day off
Friday		8:00	13:00	17:30	Yes
Saturday		13:00	13:00	17:30	No
Sunday		13:00	13:00	17:30	No

MAIN STUDY CHILD	Day off Protocol	Breakfast time	Morning snack time	Lunch time	Afternoon snack time	Dinner time	Deviations from protocol (Additional snacks)
Monday		8:00	10:30	13:00	15:00	17:30	No
Tuesday		8:00	10:30	13:00	15:00	17:30	No
Wednesday		8:00	10:30	13:00	15:00	17:30	No
Thursday	Х	8:30	10:30	12:00	15:00	17:30	Day off
Friday		8:00	10:30	13:00	15:00	17:30	Yes
Saturday		8:00	10:30	13:00	15:00	17:30	No
Sunday		8:00	10:30	13:00	15:00	17:30	No

Example for one week

#### Appendix B Additional Analysis

Figure B.1: Scatter plot of BMI, height and weight between child and main adult.



*Note:* The dotted line is a plot of the prediction from a linear regression of the child measurement on the main adults measurement. Pearson correlation coefficient is shown in the bottom left of the figure; \*\*\*, \*\*,\* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.

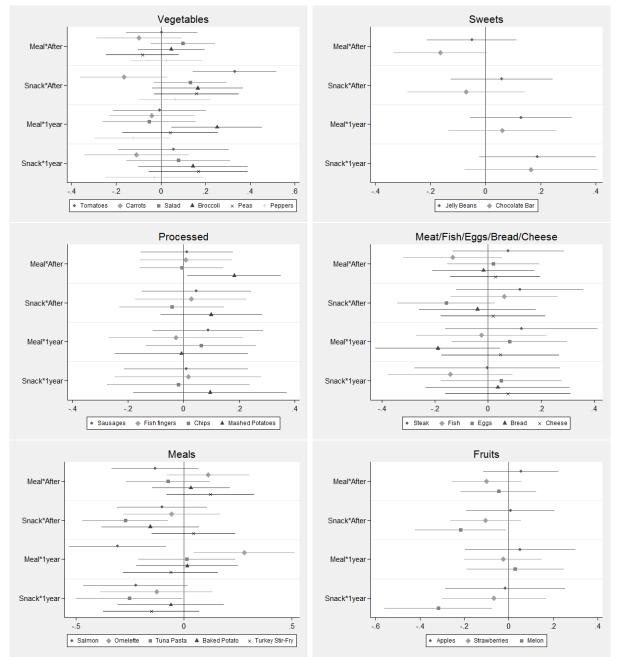


Figure B.2: The impact of the experiment interventions on the probability of parents and children reporting the same preference

Note: In each panel, each dot shape comes from a separate regression. The lines represent the 95% confidence interval.

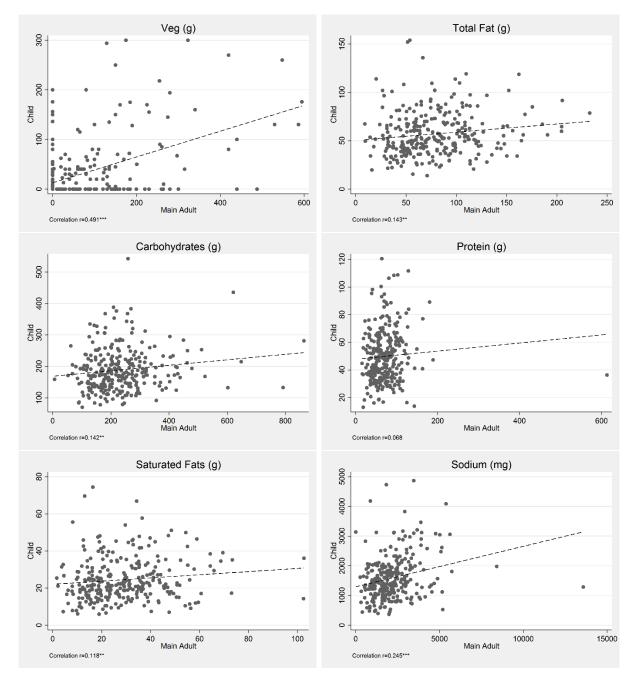


Figure B.3: Scatter plot of food intakes of child and main adult by energy, food types and macronutrients

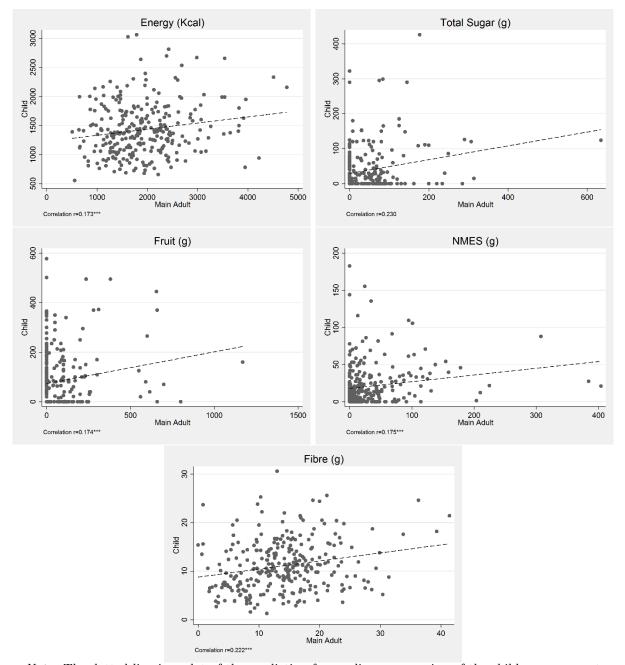


Figure B.3: Scatter plot of food intakes of child and main adult by energy, food types and macronutrients (cont.)

*Note:* The dotted line is a plot of the prediction from a linear regression of the child measurement on the main adults measurement. Pearson correlation coefficient is shown in the bottom left of the figure; \*\*\*, \*\*,\* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.

	Daily recommendations	Macro-nutrient content in the recipes (average)	Recommendations based on 364.87kcal	Self-report At baseline (all groups)	Self-report intakes based on 364.87kcal
Average kcal	2000	364.87	364.87	1970.15	364.87
Fat (g)	70	7.94	12.77	80.58	14.92
Saturates (g)	20	2.23	3.65	29.68	5.5
Carbohydrate (g)	260	51.97	47.43	237.21	43.93
Sugars (g)	90	9.53	16.42	106.01	19.63
Protein (g)	50	23.44	9.12	73.73	13.65
Sodium (mg)	2466	491.29	449.88	2303.85	426.67

 Table B2: Recipes Calibration

*Note*: Daily recommendations have been taken from https://www.nutrition.org.uk/attachments/article/907/Nutrition% 20Requirements\_Revised%20June%202016.pdf. The average calorie intake of the recipes is 364.87Kcal. An isocaloric comparison is then performed on the daily recommendations and on the self-reported macronutrient intakes

Category	Items from the questionnaire
Fruits	Apples, strawberries, melon
Vegetables	Tomato, carrot, salad, broccoli, peas, pep-
-	pers
Cheese	Cheddar
Bread	Sliced white bread
Meat/fish/eggs	Eggs, white fish, steack
Unhealthy processed food	Fishfingers, mashed potatoes, sausages, chips
Sweets	Jellybeans, chocolate
Recipes	Salmon with spring onions, omelette with vegetables, tuna sweet corn pasta, baked potato with mince, turkey pepper stir fry.

Table B.3. List of items by category in the food preference questionnaire

Blood biomarker	Description
NEFA	(Non-esterified Free Fatty Acid): The plasma concentration increases in fasting as fatty acids are released from adipose tissue as a metabolic fuel. Elevated NEFA concentration is a risk factor for cardiovascular disease and could be pathogenically involved in the atherosclerotic process (Carlsson et al. 2000)
Fasting insulin & glucose	Insulin is an anabolic hormone that promotes glucose uptake, glycogenesis, lipogenesis, and protein synthesis of skeletal muscle and fat tis- sue. If insulin is raised and glucose is normal and/or moderately raised, then there may be some insulin resistance. If the insulin is low and the glucose is high, then most likely there is in- sufficient insulin being produced by the body. If insulin levels are normal or raised and glucose levels are low, then the participant is hypogly- caemic due to excess insulin. (Wilcox 2005)
Triglyceride	Triglycerides are another type of fat, and they're used to store excess energy from your diet. High levels of triglycerides in the blood are associated with atherosclerosis. Elevated triglycerides can be caused by overweight and obesity, physical inactivity, cigarette smoking, excess alcohol consumption and a diet very high in carbohydrates (more than 60 percent of total calories) (American Heart Association 2017)
HDL	HDL cholesterol is considered "good" choles- terol because it helps remove LDL cholesterol from the arteries. Experts believe HDL acts as a scavenger, carrying LDL cholesterol away from the arteries and back to the liver, where it is broken down and passed from the body. One-fourth to one-third of blood cholesterol is carried by HDL. A healthy level of HDL choles- terol may also protect against heart attack and stroke, while low levels of HDL cholesterol have been shown to increase the risk of heart disease (American Heart Association 2017)

Table B.4. Blood biomarkers meaning and interpretation

Blood biomarker	Description
LDL	LDL cholesterol is considered the "bad" choles- terol because it contributes to plaque, a thick, hard deposit that can clog arteries and make them less flexible. This condition is known as atherosclerosis. If a clot forms and blocks a nar- rowed artery, heart attack or stroke can result. Another condition called peripheral artery dis- ease can develop when plaque build-up narrows an artery supplying blood to the legs. (Ameri- ann Heart Association 2017)
CRP	can Heart Association 2017) C-reactive protein (CRP) is produced by the liver. The level of CRP rises when there is inflammation throughout the body. You are at high risk for cardiovascular disease if your hs-CRP level is higher than 3.0 mg/L (https://medlineplus.gov/ency/article/003356.htm
TAS	Oxidative stress is an imbalance between the production of reactive oxygen radicals and the ability of the organism's natural protective mechanisms to cope with these radicals and to prevent adverse effects. The oxidation of lipids, nucleic acids, or protein is thought to be asso- ciated with the etiology of several age-related chronic diseases, including cancer, cardiovascu- lar disease, cataract, and age-related macular degeneration. (Talegawkar et al. 2009)

Table B.4. Blood biomarkers meaning and interpretation (cont.)

	England	Colchester	p-value of test of the difference	Scotland	Edinburgh	p-value of test of the difference
	(Survey)	(Experiment)		(Survey)	(Experiment)	
% Female adults	70.8	72.7	0.5	66.4	82.6	0.03
Age (adults)	33.5	35.3	0.04	33.2	34.4	0
	(7.2)	(6.7)		(8.42)	(7.19)	
Age (study child)	4	3.7	0.79	3.71	4.4	0.86
	(1.41)	(1.61)		(1.23)	(1.5)	
Number of adults/hh	1.7	1.7	0.62	1.5	1.6	0.42
	(0.56)	(0.6)		(0.61)	(0.9)	
Number of children/hh	1.9	1.9	0.86	1.8	1.8	0.86
	(0.91)	(0.97)		(0.9)	(0.97)	
Annual household Income						
(mean, GBP)	$15,\!857$	20,498	0	$16,\!884$	$20,\!692$	0.02
	(6,221)	(15, 342)		(6, 447)	(14, 259)	
% Receiving child benefit	95.9	89.7	0	94	79.1	0
% Receiving tax credit	79.6	77.3	0.43	68.7	69.2	0.91
% Receiving job seekers allowance	5.3	3.1	0.17	3.7	4.4	0.73
% Receiving housing benefits	46.4	38.1	0.02	36.6	41.8	0.31
% Receiving income support	17.9	18	0.96	15.7	26.4	0.01
% Receiving other benefits	7.5	7.7	0.9	18.7	6.6	0
% degree	16.6	21.1	0.92	26.9	27.4	0.05
% No qualifications	22.6	9.43	0	10.4	8.5	0.52
Observation (adults)	319	267		134	109	
Observation (children)	265	205		185	91	

Table B.5: Demographic characteristics at baseline Edinburgh and Colchester compared to the English and Scottish Health Surveys

 $\overline{Note:}$  English data is from the Health Survey for England (HSE) from 2014 and for Scotland from the Scottish Health Survey (SHS). The sample is restricted to those households with a child aged between 2 and 6, and those with a household income below 26000. p-value of test of the difference tests the hypothesis that Ho: Experiment=Survey.

	(1) Fish	(2) Tomato	(3) Apple	(4) Carrot	(5) Salad	(6) Broccoli	(7)Peas	(8) Strawberries	(9) Melon	(10) Peppers	(11) Eggs
A : Children											
After	0.065	-0.206*	0.106	-0.262**	-0.057	0.123	0.080	0.040	0.127	-0.080	0.046
	(0.141)	(0.122)	(0.084)	(0.120)	(0.138)	(0.114)	(0.111)	(0.091)	(0.128)	(0.122)	(0.119)
1-year follow up	$0.283^{*}$	-0.216*	-0.001	-0.052	0.147	0.134	-0.148	0.026	0.011	0.028	0.188
• -	(0.145)	(0.127)	(0.086)	(0.125)	(0.141)	(0.118)	(0.114)	(0.093)	(0.131)	(0.126)	(0.125)
Meal x After	-0.338*	0.130	-0.063	0.094	0.205	-0.156	-0.179	-0.023	-0.260	0.125	-0.062
	(0.202)	(0.177)	(0.121)	(0.177)	(0.200)	(0.167)	(0.160)	(0.132)	(0.185)	(0.177)	(0.173)
Meal x 1year	-0.447**	$0.325^{*}$	-0.095	-0.039	0.034	-0.079	0.086	0.016	-0.003	-0.035	-0.107
v	(0.208)	(0.184)	(0.125)	(0.183)	(0.204)	(0.173)	(0.165)	(0.136)	(0.191)	(0.184)	(0.181)
Snack x After	0.033	0.234	-0.089	0.271	0.206	0.080	0.150	0.226	-0.104	-0.071	-0.160
	(0.229)	(0.202)	(0.138)	(0.200)	(0.225)	(0.190)	(0.182)	(0.151)	(0.215)	(0.204)	(0.199)
Snack x 1year	-0.161	0.109	-0.037	0.278	0.057	0.134	0.399**	0.174	0.328	-0.238	-0.260
J	(0.237)	(0.209)	(0.143)	(0.208)	(0.232)	(0.196)	(0.188)	(0.156)	(0.221)	(0.212)	(0.206)
Constant	2.662***	2.463***	3.485***	3.136***	2.050***	2.675***	2.942***	3.419***	2.795***	2.267***	2.896**
	(0.062)	(0.054)	(0.037)	(0.054)	(0.061)	(0.051)	(0.049)	(0.041)	(0.057)	(0.055)	(0.053)
Observations	750	760	780	775	758	776	771	768	730	744	762
R-squared	0.018	0.014	0.010	0.019	0.011	0.011	0.018	0.012	0.016	0.006	0.008
N (ind)	288	289	289	289	288	289	288	288	285	284	287
( )	-00	200	200	200	-00	200	-00	200	-00	-01	201
B : Adults											
After	-0.071	-0.046	0.007	0.018	-0.007	0.084	-0.007	0.004	-0.041	-0.031	-0.014
	(0.058)	(0.058)	(0.054)	(0.049)	(0.053)	(0.052)	(0.054)	(0.043)	(0.058)	(0.055)	(0.054)
1-year follow up	-0.077	-0.017	0.044	-0.014	0.005	0.054	0.009	-0.000	-0.041	-0.071	0.084
	(0.062)	(0.062)	(0.057)	(0.052)	(0.057)	(0.055)	(0.058)	(0.045)	(0.061)	(0.059)	(0.057)
Meal x After	0.071	0.095	$0.152^{*}$	-0.042	-0.079	-0.023	-0.001	-0.036	0.046	0.056	-0.079
	(0.087)	(0.086)	(0.079)	(0.073)	(0.079)	(0.077)	(0.080)	(0.063)	(0.085)	(0.082)	(0.079)
Meal x 1year	0.115	0.059	0.018	0.019	-0.018	0.010	-0.066	-0.052	-0.005	0.118	$-0.192^{*}$
	(0.091)	(0.089)	(0.083)	(0.076)	(0.082)	(0.080)	(0.084)	(0.066)	(0.089)	(0.085)	(0.083)
Snack x After	-0.034	0.034	0.046	0.026	0.020	0.058	0.134	-0.003	$0.242^{**}$	0.096	0.014
	(0.098)	(0.098)	(0.091)	(0.082)	(0.090)	(0.088)	(0.092)	(0.072)	(0.097)	(0.094)	(0.091)
Snack x 1year	0.017	-0.037	-0.089	0.070	-0.112	0.032	-0.075	-0.069	0.096	0.024	-0.101
	(0.106)	(0.105)	(0.097)	(0.088)	(0.096)	(0.094)	(0.098)	(0.077)	(0.104)	(0.100)	(0.098)
Constant	3.243***	$3.094^{***}$	3.374***	3.311***	$3.274^{***}$	$3.211^{***}$	3.143***	3.714***	3.181***	$3.134^{***}$	3.341**
	(0.026)	(0.026)	(0.024)	(0.022)	(0.024)	(0.023)	(0.024)	(0.019)	(0.026)	(0.025)	(0.024)
Observations	987	1,009	1,017	1,025	1,026	1,016	1,029	1,015	1,022	1,019	1,007
R-squared	0.007	0.003	0.015	0.002	0.008	0.013	0.011	0.004	0.013	0.006	0.012
N (ind)	368	374	376	379	379	376	379	375	379	378	375

Table B.6: Changes in food preferences for low calorie items

*Note*: Each column is from a separate regression. All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\*, \*\*,\* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.

	(1) Bread	(2) Cheese	(3) Fish- fingers	(4) Mashed potatoes	(5) Sausages	(6)Steak	(7) Jelly- beans	(8) Chocolate bars	(9) Chips
A : Children									
After	0.061	$0.205^{**}$	$0.305^{***}$	0.009	0.010	-0.003	-0.120	-0.136*	0.038
	(0.090)	(0.097)	(0.101)	(0.121)	(0.093)	(0.171)	(0.115)	(0.080)	(0.082)
1-year follow up	-0.027	0.006	$0.202^{*}$	0.146	-0.000	0.170	-0.121	-0.133	-0.001
	(0.093)	(0.100)	(0.105)	(0.124)	(0.096)	(0.174)	(0.119)	(0.083)	(0.085)
Meal x After	-0.230*	-0.348**	-0.601***	-0.004	-0.203	-0.130	0.173	0.121	-0.195
	(0.132)	(0.142)	(0.148)	(0.175)	(0.137)	(0.249)	(0.171)	(0.117)	(0.121)
Meal x 1year	-0.067	-0.067	-0.368**	-0.122	0.024	-0.175	0.126	0.171	-0.030
•	(0.136)	(0.146)	(0.153)	(0.180)	(0.141)	(0.253)	(0.175)	(0.122)	(0.124)
Snack x After	0.227	-0.179	-0.119	0.240	-0.146	-0.322	$0.373^{*}$	0.024	0.143
	(0.150)	(0.161)	(0.166)	(0.199)	(0.154)	(0.279)	(0.193)	(0.133)	(0.138)
Snack x 1year	0.162	-0.109	0.127	0.087	-0.020	-0.320	0.160	-0.107	0.002
0	(0.155)	(0.167)	(0.173)	(0.206)	(0.159)	(0.286)	(0.203)	(0.138)	(0.143)
Constant	3.469***	3.286***	3.310***	2.979***	3.488***	2.379***	3.235***	3.788***	3.599**
	(0.040)	(0.044)	(0.045)	(0.054)	(0.042)	(0.076)	(0.053)	(0.036)	(0.037)
Observations	781	777	767	765	763	614	710	773	778
R-squared	0.020	0.018	0.046	0.010	0.014	0.012	0.011	0.018	0.015
# ind.	289	287	289	289	288	260	280	289	289
B : Adults									
After	0.011	0.019	-0.025	0.004	-0.104*	0.063	-0.036	-0.021	0.004
	(0.059)	(0.053)	(0.061)	(0.056)	(0.059)	(0.061)	(0.064)	(0.056)	(0.054)
1-year follow up	-0.133**	-0.095*	-0.019	-0.065	-0.082	0.077	-0.026	-0.079	-0.040
	(0.063)	(0.057)	(0.065)	(0.059)	(0.063)	(0.064)	(0.068)	(0.059)	(0.057)
Meal x After	-0.137	0.023	0.202**	-0.016	0.104	-0.033	0.140	-0.029	0.078
	(0.088)	(0.078)	(0.090)	(0.082)	(0.087)	(0.091)	(0.096)	(0.082)	(0.080)
Meal x 1year	0.084	0.071	0.104	0.008	0.029	-0.180*	0.033	0.018	0.058
	(0.092)	(0.082)	(0.094)	(0.086)	(0.092)	(0.095)	(0.099)	(0.086)	(0.083)
Snack x After	-0.117	-0.089	0.039	0.079	-0.071	-0.270***	-0.023	0.001	-0.080
	(0.101)	(0.089)	(0.103)	(0.094)	(0.098)	(0.102)	(0.108)	(0.095)	(0.091)
Snack x 1year	0.024	0.025	0.009	0.011	-0.021	-0.222**	0.071	0.081	0.018
·	(0.108)	(0.096)	(0.110)	(0.100)	(0.106)	(0.110)	(0.115)	(0.101)	(0.097)
Constant	3.216***	3.409***	2.782***	3.259***	3.148***	3.321***	2.326***	3.340***	3.092***
	(0.027)	(0.024)	(0.027)	(0.025)	(0.027)	(0.027)	(0.029)	(0.025)	(0.024)
Observations	1,006	1,010	980	1,020	960	947	1,002	1,014	1,027
R-squared	0.019	0.011	0.012	0.009	0.015	0.020	0.007	0.005	0.006
# ind.	377	376	367	377	364	359	376	377	379

Table B.7: Changes in food preferences for high calorie items

*Note*: Each column is from a separate regression. All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\*, \*\*,\* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.

	(1)	(2)	(3)	(4)	(5)
	Salmon	Omelette	Tuna Pasta	Baked potato	Turkey
A: Children					
After	0.041	-0.145	0.102	-0.327**	-0.100
111001	(0.146)	(0.154)	(0.147)	(0.146)	(0.144)
1-year follow up	0.043	0.014	-0.015	-0.148	-0.060
i joar ronon ap	(0.151)	(0.163)	(0.154)	(0.150)	(0.153)
Meal x After	0.040	0.036	0.024	0.301	0.237
	(0.209)	(0.228)	(0.215)	(0.212)	(0.213)
Meal x 1year	-0.145	-0.080	-0.045	-0.171	0.035
J	(0.216)	(0.237)	(0.227)	(0.221)	(0.225)
Snack x After	0.076	0.153	-0.253	0.085	0.060
	(0.237)	(0.254)	(0.242)	(0.232)	(0.238)
Snack x 1year	-0.219	-0.135	0.000	0.077	-0.195
U	(0.250)	(0.269)	(0.251)	(0.241)	(0.259)
Constant	2.162***	2.286***	2.659***	2.343***	2.102***
	(0.065)	(0.071)	(0.067)	(0.065)	(0.067)
	· · · ·	· · · ·	× ,	· · · ·	· · · ·
Observations	654	664	713	646	643
R-squared	0.011	0.006	0.007	0.031	0.010
# ind.	270	272	284	277	272
B: Adults					
After	-0.011	0.030	0.018	0.083	-0.027
	(0.068)	(0.068)	(0.072)	(0.066)	(0.068)
1-year follow up	-0.062	-0.029	-0.081	-0.068	-0.007
J	(0.071)	(0.072)	(0.076)	(0.070)	(0.073)
Meal x After	0.090	-0.231**	-0.156	-0.020	0.103
	(0.099)	(0.100)	(0.105)	(0.100)	(0.102)
Meal x 1year	$0.172^{*}$	-0.167	-0.059	0.065	0.086
v	(0.104)	(0.104)	(0.110)	(0.104)	(0.106)
Snack x After	-0.024	0.111	0.087	-0.049	0.041
	(0.113)	(0.114)	(0.121)	(0.111)	(0.115)
Snack x 1year	0.073	0.087	0.122	0.106	-0.020
v	(0.121)	(0.123)	(0.131)	(0.119)	(0.122)
Constant	3.065***	3.129***	2.987***	3.075***	3.228***
	(0.030)	(0.030)	(0.032)	(0.030)	(0.031)
Observations	955	987	962	934	938
R-squared	0.006	0.020	0.012	0.010	0.003
# ind.	367	375	364	362	360

Table B.8: Changes in meals preferences

*Note*: Each column is from a separate regression. All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\*, \*\*,\* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.

	Daily	Male	Female	Child
	recommendation			
Sat. fat	11	13.3	13.1	15
Non-sat. fat	24	22.2	22.5	19.8
Protein	15	14.3	15.3	13.6
Carbs. (w/o nmes)	50	40.2	41	46.1
Nmes	5	6.7	6.1	5.5
Alcohol	0	3.3	2	0

Table B.9: Average contribution (in %) total calories of macro nutrients at baseline

	(1)	(2)	(3)	(4)	(5)
	Carbo-	Proteins	Sat.	Carbs	NMES
	hydrates		Fat	without sugars	
A: Children					
After	-0.4	1.4***	-0.6	0.3	-1.0
	(1.0)	(0.5)	(0.5)	(1.1)	(0.7)
1-year follow up	4.7***	-0.1	-2.4***	-9.9***	13.8***
v i	(1.5)	(0.6)	(0.6)	(1.2)	(1.3)
Meal x After	1.2	-1.0	-0.7	2.2	-0.6
	(1.5)	(0.7)	(0.7)	(1.7)	(1.2)
Meal x 1year	0.0	-0.3	-0.7	4.8***	-3.8**
	(1.8)	(0.8)	(0.8)	(1.7)	(1.6)
Snack x After	0.5	-0.9	0.3	1.0	-0.2
	(1.7)	(0.8)	(0.9)	(1.8)	(1.3)
Snack x 1year	-2.2	-0.8	0.5	2.3	-3.6*
	(2.0)	(0.7)	(1.1)	(1.8)	(1.9)
Constant	$51.6^{***}$	$13.6^{***}$	15.1***	$46.1^{***}$	5.5***
	(0.4)	(0.2)	(0.2)	(0.4)	(0.3)
Observations	804	804	804	802	802
R-squared	0.1	0.0	0.1	0.3	0.5
# ind.	292	292	292	291	291
B: Adults					
After	1.1	0.7	0.2	1.2	-1.1
	(1.6)	(0.7)	(0.6)	(1.4)	(0.9)
1-year follow up	10.2***	-0.6	-2.0***	-4.5***	12.3***
· -	(2.6)	(1.0)	(0.7)	(1.6)	(1.4)
Meal x After	-1.8	0.0	-0.8	0.5	-1.2
	(2.0)	(1.0)	(0.8)	(1.8)	(1.2)
Meal x 1year	-2.8	0.3	-0.1	0.5	-3.0*
	(3.7)	(1.4)	(1.0)	(2.1)	(1.8)
Snack x After	-0.9	0.1	-0.3	2.0	-1.9
	(2.2)	(0.9)	(1.0)	(2.2)	(1.5)
Snack x 1year	-5.8*	$3.4^{*}$	1.4	1.7	-5.8***
	(3.3)	(1.8)	(1.1)	(2.4)	(2.0)
Constant	47.1***	$15.2^{***}$	13.0***	41.1***	6.2***
	(0.6)	(0.3)	(0.2)	(0.5)	(0.3)
Observations	926	926	925	921	921
R-squared	0.1	0.0	0.0	0.1	0.4
# ind.	359	359	358	359	359

Table B.10: The impact of meal and snack treatments on contributiuon (%) of macronutirent in total calorie intake.

Note: Each column is from a separate regression. All regressions include individual fixed effects. Standard errors in parentheses are clustered at the household level, \*\*\*, \*\*, \* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.

	Correlation	p-value
Bread	0.12	-0.055
Eggs	0.052	-0.409
Cheese	0.08	-0.197
Fish Fingers	0.008	-0.903
Fish	0.122	-0.056
Tomatoes	0.116	-0.067
Apples	0.17	-0.006
Carrots	-0.043	-0.488
Salad	0.135	-0.031
Broccoli	0.218	0
Peas	0.201	-0.001
Mashed Potatoes	0.189	-0.002
Strawberries	0.151	-0.016
Melon	0.244	0
Sausage	0.013	-0.837
Peppers	0.146	-0.022
Steak	0.048	-0.516
Jelly Beans	0.119	-0.073
Chocolate	0.024	-0.707
Chips	0.232	0
Meal: Salmon	0.144	-0.048
Meal: Omellette	0.057	-0.42
Meal: Tuna pasta	0.14	-0.039
Meal: Jacket Potato	0.201	-0.006
Meal: Turkey	0.107	-0.151

Table B.11: Correlation of food preferences between child and main adult

Note: Correlation value from the Spearman rank correlation coefficient with the p-value is from the test of the null hypothesis that the childs and parents preferences are independent.

	(1) Energy (kcal)	(2) Fruit (g)	(3) Veg (g)	(4) Total Fat (g)	(5) Carbo- hydrates (g)	(6) Protein (g)	(7) Saturates (g)	(8) NMES (g)	(9) Total Sugar (g)	(10) Fibre (g)	(11) Sodium (mg)
A. Absolute I	Difference										
Meal x After	$225.9^{**}$	-1.346	6.289	7.026	$36.10^{***}$	8.756	-1.763	4.656	-14.29	0.426	-40.71
	(96.47)	(22.13)	(13.00)	(5.420)	(12.30)	(7.166)	(2.316)	(6.670)	(11.48)	(0.968)	(168.6)
Snack x After	52.52	-18.88	7.292	-0.562	5.838	9.115	-3.452	3.489	-2.141	1.184	-255.6
	(114.9)	(30.62)	(16.91)	(6.049)	(16.89)	(7.016)	(2.679)	(7.977)	(12.65)	(1.149)	(247.4)
Meal x 1-year	$214.3^{*}$	50.68	-46.66	2.429	$29.93^{*}$	10.59	$-5.249^{**}$	-10.77			-201.5
	(119.9)	(48.77)	(32.99)	(5.764)	(17.47)	(7.539)	(2.587)	(9.126)			(210.8)
Snack x 1-year	19.23	-14.74	-30.38	-4.886	-0.102	14.62**	-4.907	-8.684			-445.8*
	(136.0)	(42.37)	(39.68)	(6.919)	(20.89)	(7.413)	(3.134)	(11.73)			(241.4)
1-year	-126.4	29.77	41.91*	-4.398	-6.916	-12.65**	2.601	23.92***			126.5
•	(90.77)	(23.73)	(22.43)	(4.460)	(14.21)	(6.308)	(2.059)	(7.711)			(162.1)
After	-300.3***	-2.854	-12.53	-9.918**	-38.93***	-12.92**	-0.977	-11.57***	-4.192	-1.041	-64.74
	(69.25)	(15.31)	(8.041)	(4.165)	(8.642)	(6.255)	(1.747)	(4.345)	(6.999)	(0.742)	(108.8)
Observations	787	660	632	787	787	787	787	782	550	550	787
R-squared	0.052	0.035	0.031	0.025	0.055	0.026	0.022	0.113	0.021	0.012	0.013
# of children	286	286	286	286	286	286	286	286	286	286	286
B. Difference	(Adult-Ch	ild)									
Meal x After	163.9	0.198	6.811	9.825	8.137	8.627	2.110	4.011	-28.02**	0.544	-12.75
	(125.1)	(26.61)	(15.55)	(6.811)	(17.76)	(7.955)	(3.028)	(8.265)	(14.14)	(1.453)	(213.0)
Snack x After	-77.65	29.20	-19.43	-0.477	-29.85	7.519	-2.136	-6.173	-9.796	0.00285	-185.7
	(144.5)	(34.60)	(22.68)	(7.673)	(21.89)	(7.950)	(3.429)	(9.084)	(14.58)	(1.617)	(285.2)
Meal x 1-year	356.0**	4.189	-66.89	19.95**	31.65	17.66*	4.852	14.70	,		133.3
	(162.8)	(58.50)	(46.40)	(7.918)	(23.11)	(9.469)	(3.945)	(10.92)			(271.8)
Snack x 1-year	278.3	116.0**	-7.495	11.18	22.42	28.86***	4.007	11.75			-2.361
v	(190.5)	(50.24)	(42.76)	(9.156)	(27.70)	(9.428)	(4.297)	(13.70)			(363.7)
1-year	-568.8***	3.722	30.35	-28.12***	-57.00***	-26.69***	-6.404**	-21.91**			-497.1**
	(122.9)	(31.91)	(26.64)	(5.770)	(18.06)	(7.775)	(2.905)	(8.457)			(201.0)
After	-219.6**	-45.09**	-19.65**	-8.948*	-19.77	-13.02**	-1.311	-6.847	5.558	-2.066*	-186.2
	(89.61)	(17.47)	(9.763)	(4.985)	(13.00)	(6.595)	(2.130)	(4.856)	(8.270)	(1.064)	(139.5)
Observations	787	660	632	787	787	787	787	782	550	550	787
R-squared	0.079	0.054	0.041	0.074	0.051	0.052	0.019	0.027	0.021	0.033	0.031
# of children	286	286	286	286	286	286	286	286	286	286	286

Table B.12: The impact of meal and snack protocol on the gap in intake between child and main adult

*Note*: All regressions include family fixed effects. Standard errors, clustered at the household level in parenthesis. \*\*\*, \*\*,\* denote statistical significance at the 1 percent, 5 percent, and 10 percent confidence levels, respectively.