Forming Dietary Habits in Childhood:

A Field Experiment With Low Income Families[1]

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Abstract

This paper evaluates with a field experiment the extent to which dietary habits are malleable early on in childhood and later in life. We evaluate two treatments – one that targets *what* people eat, the other that targets *how* people eat. 285 low income families with young children were recruited and assigned either to a control group or one of two treatments, each of them lasting for 12 consecutive weeks. In the first 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 second 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 some evidence that children's preferences have been affected by both treatments, and we find that their BMI distribution shifted significantly relative to the control group, i.e. they became relatively "thinner". On the other hand, we find little evidence of any effects on adults and these effects are not consistent with adopting a healthier diet. 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 in the short and long run remains a major challenge.

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1. 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[6], 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 very hard 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 targeting young children and their families. The two interventions are linked to two possible factors for poor dietary choices and the current obesity crisis: One is linked to *what* people eat, the other is linked to *how* people eat, and in particular erratic eating habits. The first intervention consists of a strong and relatively invasive intervention, where families receive food and recipes at home to cook 5 meals a week over a period of three months. The second is a much simpler intervention where families are instructed to avoid (adults) or regularise (children) snacking between meals and adhere to a pattern of food intake during the day. We evaluate these interventions with a randomized controlled experiment, conducted in two different areas of the UK – Edinburgh and Colchester (Essex). 285 families participated in the study. 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 is what 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). But 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. The protocol 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 "Experimental Design" and Appendix) 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

[6]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).

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 and 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 & 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 the evidence, a protocol encouraging reduction of snacking and regularised 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 however, as it is difficult to monitor overall food intake. We implemented this 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 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.

We recruited 285 families in the UK and assigned them to one of the two treatments or a control group. We conducted our experiment in two different areas of the UK – Edinburgh and Colchester (Essex). 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 individual's total food intake—not 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 University facilities 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. We will come back in Section 4.7 on the issue of multiple measures and hypothesis testing. 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 in fresh fruit and vegetables, deficient in iron folate and vitamin D and high in sugar and saturated fats.

We evaluate the immediate effects of the treatments, as well as the effects observed one year after the start of the experiment. Overall and perhaps notably, we do not find large differences in effects *across* treatments. Also, we fail to 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 children's 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) but for the added sugars intake in the long 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 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 hard to follow, and again, fails to deliver any effects. Children appear to be more responsive, and the changes we observe in BMI are notable. However, we cannot conclude that our treatment lead to significant 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 less-nutritious 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 2 we present the experimental design. Section 3 describes the different measures collected. We present the empirical analysis in Section 4. Finally, we conclude in Section 6.

2. Experimental Design

Sample and Recruitment - The experiment was conducted with ethical approval from the University of Edinburgh. We recruited families with young children living on low incomes from the areas around Edinburgh (Scotland) and Colchester (England). Based on our eligibility criteria, families would need to: have a household income inferior to 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. Exclusions criteria are available in Appendix B, Table B.1. Recruitment started 4 weeks prior to the start of the experiment and 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.[7]

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 B for a 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 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" on which we collected measures was the youngest child of the household aged being 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 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,

[7] 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, and the interventions were conducted in March-June 2015 in Edinburgh and September-December 2015 in Colchester.

without knowing these corresponded to the two different treatments and/or the control group (nor did families know that there were different treatments and what these were).

Timing - Overall, 91 families in Edinburgh, and 194 in Colchester took part in our study. Table 1 provides further details on attendance and attrition. Each treatment lasted for 12 weeks, and the baseline and post experiment measurements were collected during 2-week time windows before and after this 12-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 follow-up session has taken place in February or August 2016 for each sample. 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% (248 returning families).

Treatments - The first treatment, hereafter the "*Meal*" treatment, consists in 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 three months. 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 365cal, 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 in fact lower than the maximum thresholds on sugar and fat (and sat fat), compensating for these calories via higher carbohydrate and protein contents. This can be seen in the Appendix B, Table B.2.**,** 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). Note that 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 maximize 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 said 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.

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 £42.5 per week on food and non-alcoholic drinks. When not distinguishing by the size of households, the average spending in food only in the UK in 2015 is £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 (4-point likert scale) and whether families liked them (4-point likert scale). An example of the first page of this feedback sheet can be found in the Appendix A, Table A.4. Food baskets rotated on a 4-weekly basis, so families received the same food baskets and recipes 3 times in the 12-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 in 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 and 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' (while 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 control 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 4–10 years, but there was no association with BMI, waist-to-height ratio, and commonly tested biomarkers (Murakami & 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 regularised 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 protocol is to create a source of exogenous variation in frequency and regularity of food intake across groups, and study how that impacts 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 treatments 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 in 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.[8]

3. 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 12-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 children's 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.

We added two additional measures for adults that are not based on self-reports. One is a set of biomarkers based on blood samples, the other is an incentivized measure of food choice.

3.1 Base measures (children and adults)

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 recipes booklets. The chosen items cover different food groups (see in Appendix B, Table B.3.). 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"[9] food categories (fruit, vegetables, meat/fish/eggs, cheese, bread, unhealthy processed food and sweets).

[8] 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.

[9] The eatwell plate is a policy tool used to define the British government recommendations on eating healthily and achieving a balanced diet.

The second measure is a measure of dietary intake based on a well-known method in Nutrition, called the "24hr 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 child's 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 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 1-year follow-up we used Intake24 - a computer-based recall method fitted for the British population (https://intake24.co.uk/). Unlike the nutritionist led face-to-face 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 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.

3.2 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 Essex and we collected data on a range of biomarkers for adults in Edinburgh.[10]

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 (<100 Kcal) and one of high calorie (>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 0.40p for each choice. The task is shown in Figure 1.

[10] For logistic and time constraints reasons, it was not possible to collect both measures in both samples.

Choices made in this task tell how much the participant is ready to pay to get 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 12-week treatment. The full list of biomarkers screened and their short description is presented in the Appendix in Table B.3.

4. Empirical Analysis

4.1 Empirical Strategy

We evaluate the effects of the two treatments on diet and health using a difference-in-differences strategy, with individual fixed effects. More precisely, let $\boldsymbol{Y_{it}}$ be the outcome of interest for individual *i* at time *t* where $\boldsymbol{t = 0}$ before the treatment, *t* = 1 immediately after the treatment, *t* = 2 one year after the treatment. $\boldsymbol{M}$ is a dummy variable equal to 1 for the individuals in the *Meal* treatment, 0 for the individuals in the control group, $\boldsymbol{S}$ is a dummy variable =1 for participants who belong to the *Snack* treatment, 0 for the individuals in the control group. We estimate the following equation: We have a set of indicators for our two treatments. M is a dummy variable that signifies being in the meal treatment, and S represents being in the snack treatment.

$$Y_{it} = \beta_0 + \beta_1 M_{it} + \beta_2 S_{it} + \beta_3 . After_{it} + \beta_4 1year_{it} + \beta_5 M_{it} . After_{it} + \beta_6 S_{it} . After_{it} + \beta_7 M_{it} . 1year_{it} + \beta_8 S_{it} . 1year_{it} + \gamma_i + \varepsilon_{it}$$

Where $\boldsymbol{\beta_5}$through to $\boldsymbol{\beta_8}$ are our coefficients of interest and represent the treatment effect of our treatment once the treatment has finished.

4.2 Descriptive Statistics

Table 2 presents the descriptive statistics of our sample for the different groups. Overall, there are no 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, 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.

4.3 Compliance

The experiment is an intention-to-treat. For the first treatment, (*Meal*) families had to prepare five meals per week; while for the second (*Snack*), 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-to-one and provided face-to-face instructions about the protocol. We explained in detail what was expected from them, and handed out the leaflets and cameras (for the first treatment). We also organized an additional short session in the middle of the 12 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 12 weeks of 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 back, 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-reports 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 to 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[11] recipes. One year later, this average dropped to 6.8 recipes for 114 participants' responses.

[11]15 recipes in total for vegetarian families who represent 8% of the sample

The second measure of compliance we propose is based on the number of pictures provided by participants in the *Meal* treatment. Since they were supposed to cook 5 meals per week during 12 weeks and to photograph each of them, a complete set of pictures would include 60 pictures. On average, we received 38 unique pictures back (hence 53%). Only 11% gave us 0 pictures back. 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 relatively high rate and those leaflets were completed with care. They report an average liking of the meals of 2.9 (4 point likert scale, s.d=0.38), based on 4516 observations. When taste has been different for the children they also reported it yielding an average liking in children of 2.7 (s.d=0.67, N=834). Children report liking significantly less the recipes overall that the adults (Wilcoxon signed-rank test yields a p-value=0.000). Turning to the difficulty, from a 5-point likert scale (very easy so 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 and yielding a moderate liking of the recipes.

In the *Snack* treatment, among the families that came back after the intervention, 69.0% brought the leaflet back, which is already a much lower rate than in the *Meal* treatment. Additionally, it was possible to evaluate the "robotic" attitude in filling out the leaflet. We use two main different 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 pages after 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 every week tick the same day, with the same pen and without never deviating 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 extreme 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 manner whatsoever.

Coming back to the day off allowed within the treatment, if every family was taking this option, this means that out of the 84 days of the treatment duration, 12 (14.3%) should be marked as a day off. From our record 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 treatment's 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 min 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 children's 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 substantial amount of irregularities during the *Snack* treatment which also corroborate results from Table 3 and Table 4.

Those three compliance measures show that the *Meal* treatment tended to be easier

to follow by the families compared to the *Snack* treatment. Families in the Meal treatment were then more compliant and conscientious with filling out the leaflet.

4.4 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 results of the difference-in-differences (DID) analysis.

Table 5 presents the effects on self-reported food preferences, more precisely, on the ranked mean liking levels of each category of item based on the control group's answers, for the children and adults, before the 12-week treatment. 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 could 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 difference-in-difference estimates of those 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"), both compared to before the treatment. 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 treatment seemed to have a limited effect on those self-reported measures both in the short and the longer run. The estimates are quite precise 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 (1% level significant). One year after, the differences found in the short-run disappear. Adults in this same group report liking more processed food on average than the control group right after they have been treated (10% level significant). 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 the Appendix)

We now turn to the analysis of dietary intakes. Table 7 presents the baseline statistics for different categories of food intakes and average quantities, before the treatment: total calories intake, number of fruits and vegetables, quantities (in grams) of fruits and vegetables, total fats, carbohydrates, proteins, saturated fats (typically referred to as unhealthy fats), sugars, Non-Milk Extrinsic Sugars (NMES), fibres, 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[12] and the Manual of Dietetic Practice

[12] British Nutrition Foundation (BNF), 2015. *Nutrition Requirements.* Available at: https://www.nutrition.org.uk/attachments/article/907/Nutrition%20Requirements_Revised%20June%202016.pdf [30/06/2017]

(Thomas et. al., 2007). We distinguish total fat to saturated fat and total sugar to NMES (non-milk extrinsic sugar, also called free sugars, which are generally considered to be added sugar in different foods). On average, the self-reported intakes imply that a male adult participant consumes 2216.4kcal over 24 hours, whereas a female adult consumes 1906.6kcal. The average calorie intake in children is 1433.8kcal. 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 intakes between our groups[13]. By comparing the different intakes to 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 (in grams). 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 results are similar.

We also look at the baseline diet of the participants compared to the recipes participants have been given 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 (at 365kcal) 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 difference-in-differences analysis. 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 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. We should point out that 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 coefficients. We find that children's fruit consumption becomes significantly lower when exposed to the *Snack* treatment compared to the control in the long run. We also see 5% significant decrease in NMES intakes, compared to the control group, in both treatment groups in the long run. Other significant changes fail to

[13] 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

reach significance in both the short and long run although coefficient signs are mostly going in the right direction except for the fruit and vegetable intakes, i.e. negative for calorie intakes, fat, sugars, proteins and sodium, but also negative for fruit and vegetable intake.

As for the adult intakes, no consistent nor significant patterns are found in contrast to the children, there is also no consistency found in the coefficients' signs. 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.

4.5 Analysis of the effects on Body Mass Index

We now turn to the analysis of BMI, which is the only objective measure we have for children. Table 9 shows the mean BMI and proportion of each weight category of our sample at baseline.

About 64% of our adult sample is overweight or obese, including 32% of obese. These figures are in line with the national rates reported in the National Diet and Nutrition Survey report[14]. 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 (Wilcoxon signed rank test yields p=0.04).

Table 10 presents the results of the DID analysis of BMI. A lower BMI after the treatment in adults would mean 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 first, we find a significant and negative treatment effect (5% to 1% significant) on BMI percentile (Column 1), in the short run but also one year after. Interestingly the size of the effects (between 4 and 6 percentage points) is similar across both treatments. This means that compared to the children in the control group, the treated children's percentile BMI decrease, shifting the distribution. 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), but of course 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).

4.6 Additional measures

Incentivised measure of food choices - In the Colchester sample, we added an incentivised measure of food choices, before and after the treatment.

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

[14] https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/310995/NDNS_Y1_to_4_UK_report.pdf

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 pair of snacks after the treatment. A possible interpretation could be that participants experience a sort of 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. This might be a reward behaviour for being in the study for 12 weeks, or a manifestation of cravings 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 (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 young adults (aged 35 on average) with no serious health conditions (one of the recruitment inclusion criteria). However, the level of LDL reaches the upper limit of the normal range in the control group and is significantly higher (at a 5% level) than in the *Meal* and *Snack*. 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).

DID estimates are reported in Table 13 showing two main treatment effects. First, the diff-in-diff estimate of the level of LDL (sometimes considered as the "bad cholesterol") is positive and significant for the *Meal* participants, compared to the Control group. Second, the diff-in-diff estimate of the glucose level is positive for the Snack participants, compared to the control group albeit imprecisely estimated.

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 non-significant changes in coefficients point to a slight increase in calories (88.4kcal), and a small increase in carbohydrates (11.8 grams) though other macronutrient changes remain in the single digit 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 where 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 (177.5kcal), a drop in total carbohydrates (24.5grams) mainly caused by a drop in sugars (23.2 grams), and a drop in sodium (370mg, 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 low-glycemic 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 at a non-statistically significant level.

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.

4.7 Correlation between parents and children

As the experiment is focused on the family, we are interested in behaviour within the 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 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 children's preferences becoming more similar we 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 child's 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 estimate equation 1 (from section 4) 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 – child's intake), we find the absolute gap is driven by an increase in the main parent's 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 carbohydrates. 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.

4.8 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 drop 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 1-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. 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 strong difference in the rates of bringing back the feedback leaflet which was higher in the *Meal* than in the *Snack* treatment. Even though it is a difficult task to follow what people do during the king of experiment we propose here, we have developed a set of compliance tools that together provide a good sense of how the treatments were taken into consideration by the participants.

5. 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 on both the short and long

terms.

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 done 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: 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 people in our sample.

We do not find any consistent patterns in adults eating habits, and even perhaps a sort of rebound effect. No changes in weight, nor food preferences or intakes were found according to the treatments. A sort of rebound effect can even be considered: the incentivised food choice task shows that participant 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, compared to before its implementation.

Having a treatment on the family level, rather than only on the adults, was mainly 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, children food preferences 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 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-calories 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

Table 1 Sample size. Number of participating households.

	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

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

Table 2 Demographic characteristics at baseline and across groups

	(1) Control	(2) T1 (Meal)	(3) T2 (Snack)	(4) P-value (1)=(2)	(5) P-value (1)=(3)
	Mean (std)	Mean (std)	Mean (std)		
Sample size (families) (Present in before)	111	103	71	-	-
% 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	0.90
	(0.9)	(1.0)	(1.0)		
Age (adults)	35.1	34.7	34.0	0.67	0.23
	(7.5)	(6.5)	(6.9)		
Age (study child)	3.9	4.0	3.9	0.99	0.75
	(1.7)	(1.7)	(1.4)		
Average annual household Income (GBP)	20,855.2 (10,056.1)	21167.4 (19,227.2)	23,928.44 (21,844.3)	0.87	0.15
% 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	21.00%	19.23%	15.00%	0.72	0.25
% No qualifications	2.7%	3.08%	3.23%	0.85	0.81

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. in Appendix B. Pregnant women at baseline: 6 in the control group, 4 in the *Meal* treatment, 1 in the *Snack* treatment.

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

		Very easy and Easy	Neutral	Difficult/Very difficult	Total	# Obs
Snack	To stick to meal times	41.2	30	28.8	100	80
	To stick to meal and snack times (child)	57.5	25	17.5	100	80
	Not to snack	33.7	23.8	42.5	100	80
	Not to snack (child)	27.4	41.3	31.3	100	80
Meal	To cook meals	83.7	13	3.3	100	123
	To stick to the recipe	61	25.2	13.8	100	123

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

Table 4 Self-reported feedback on effects of the protocols

		Strongly disagree/Disagree	Neither agree nor disagree	Agree/Strongly agree	Total
Snack	I found myself eating more at meal times	21.3	30	48.7	100
	I was surprised at how much I used to snack before starting the study	13.7	21.3	65	100
	I felt less hungry between meals	26.2	31.3	42.5	100
	I generally felt I ate less food overall during the day	22.5	31.3	46.2	100
	I have tried new foods that I had never tried before	27.8	7.4	64.8	100
	Cooking the recipes was time consuming	44.3	30.3	25.4	100
Meal	My child has tried new foods he/she had never tried before	10.6	9.8	79.6	100
	I have liked an ingredient that I thought I did not like before	18.9	13.9	67.2	100

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

Table 5 Baseline average preferences (scale 1 to 4)

		(2) Control	(3) Meal	(4) Snack	(5) P-value (1)=(2)	(6) P-value (1)=(3)
Children	**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
	Meals					
	Tuna pasta	2.5 (1.3)	2.7 (1.3)	2.7 (1.2)	0.33	0.36
	Omelette	2.4 (1.3)	2.3 (1.3)	2.2 (1.2)	0.80	0.42
	Baked potato	2.4 (1.2)	2.2 (1.2)	2.3 (1.2)	0.37	0.63
	Turkey stir fried	2.2 (1.3)	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	0.67
Adults	**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
	Meals					
	Turkey stir fried	3.2 (0.9)	3.2 (1.0)	3.3 (0.9)	0.62	0.38
	Salmon with onions	3.1 (1.1)	3.0 (1.1)	3.1 (1.1)	0.52	0.83
	Omelette	3.1 (1.0)	3.2 (0.9)	3.0 (1.0)	0.77	0.54
	Tuna pasta	3.1 (1.0)	3.0 (1.0)	2.9 (1.1)	0.45	0.21
	Baked potato	3.0 (0.8)	3.2 (0.8)	3.1 (0.9)	0.03	0.16

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 very much liking.

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

	(1) Fruits	(2) Vegetables	(3) Meat/Fish/Eggs	(4) Processed	(5) Sweets	(6) Bread	(7) 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 follow up	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 1year	-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 1year	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	0.00	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	0.0
	(0.0)	(0.0)	(0.1)	(0.0)	(0.1)	(0.1)	(0.1)
Meal*1year	-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*1year	-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	0.00	0.00	0.01	0.00	0.02	0.01
# individuals	379	379	379	379	378	377	376

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.

Table 7 Baseline measures of dietary intake

	UK daily Recommendation	(1) Control	(2) Meal	(3) Snack	(4) P-value (1)=(2)	(5) P-value (1)=(3)
Panel A: Children						
Total calorie intake (Kcal)	1800	1438.9 (538.6)	1463.8 (475.4)	1383.2 (378)	0..34	0.93
# fruit	5 portions fruits and	0.9 (1.26)	1.1 (1.4)	1.1 (1.42)	0.42	0.45
# vegetables	Veg.	0.3 (0.58)	0.4 (0.85)	0.4 (0.78)	0.26	0.23
Fruit and veg (g)	Min 400	101.5 (124.7)	122.4 (126.8)	123.5 (141.1)	0.23	0.27
Total Fat (g)	Max 70	56.5 (24)	59.5 (25.8)	55.1 (20.7)	0.40	0.90
Carbohydrate (g)	Max 220	194.7 (86.7)	190.2 (65.3)	182.2 (50.5)	0.77	0.91
Protein (g)	Min 24	47.8 (18.8)	52.5 (20.2)	48.9 (16.7)	0.08	0.50
Saturates (g)	Max 20	23.9 (11.9)	25.9 (12.9)	23.8 (11.6)	0.23	0.94
Sugar (g)	Max 85	94.2 (58.6)	97 (47.1)	87.3 (34.3)	0.20	0.83
NMES (g)	Max 23	18.0 (22.6)	25.9 (34.8)	18.1 (20.1)	0.21	0.41
Fibre AOAC (g)	Min 15	11 (5.1)	10.5 (5.2)	12 (5.7)	0.77	0.24
Sodium (mg)	2000-3000	1575.9 (699.9)	1621.7 (899.8)	1625 (692.8)	0.93	0.71
# Obs		112	104	73		
Panel B: Adults (main & second)						
Total calories intake (Kcal)	~2000-2500	2036.1 (798.1)	1843.9 (685.2)	2036.5 (809.2)	0.07	0.91
Portions of fruit	5 portions fruits and	0.94 (1.86)	0.81 (1.48)	1.03 (2.67)	0.53	0.78
Portions of vegetables	Veg.	0.77 (1.15)	0.88 (1.35)	0.87 (1.03)	0.48	0.62
Fruit and veg (g)	Min 400	137.5 (184.8)	135.6 (160.9)	151.9 (295.6)	0.93	0.66
Total Fat (g)	Max 70	84 (42.7)	74.7 (35)	83.4 (42.3	0.13	0.90
Carbohydrate (g)	Max 260	241 (118.6)	223.9 (90)	248.9 (122.1)	0.45	0.68
Protein (g)	Min 45	79.2 (55.1)	70.1 (32.6)	69.1 (27.2)	0.05	0.21
Saturates (g)	Max 20	30.2 (17.5)	28.8 (16.1)	30.2 (16)	0.63	0.69
Sugars (g)	Max 90	107.1 (88.2)	97.9 (56.7)	116.1 (99.1)	0.76	0.65
NMES (g)	Max 30	33.2 (61.7)	31.5 (41.0)	41.3 (63.0)	0.08	0.06
Fibre AOAC (g)	24	13.8 (6.9)	13.5 (6.2)	14.6 (8.2)	0.95	0.72
Sodium (mg)	2400	2329.4 (1246.3)	2139.1 (1244.6)	2440.4 (1816.7)	0.17	0.88
Alcohol (g)	0	7.1 (22.5)	6 (16.6)	9.4 (21.1)	0.71	0.09
# Obs		134	124	79		

Note: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a Wilcoxon test of equality of means. Source: 24h dietary recall data, WISP output. 1 portions of fruit or veg~80g.

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

	(1) Energy kcal (kcal)	(2) Fruits (g)	(3) Veg (g)	(3) Total fat (g)	(4) Carbs (g)	(5) Protein (g)	(6) Sat. fat (g)	(7) Total Sugar (g)	(8) NMES (g)	(9) Fibre (g)	(10) Sodium (mg)	(11) Alcohol (g)
Panel A: Children												
Sign of the UK recommendation-baseline consumption	+	+	+	+	+	-	-	-	+	+	+	
After	-67.6	3.6	10.0	-2.6	-13.7	1.6	-1.6	-8.6	-3.5	0.1	-79.8	
	(51.3)	(5.6)	(12.6)	(2.6)	(8.4)	(2.2)	(1.2)	(5.5)	(3.0)	(0.7)	(84.3)	
1 year follow-up	128.8*	49.6***	105.5***	0.3	32.6***	2.7	-1.6	23.2***	57.7***		158.9	
	(74.3)	(13.9)	(20.8)	(3.2)	(11.8)	(3.1)	(1.4)	(7.3)	(6.2)		(112.0)	
Meal*After	-37.2	-9.9	-4.6	-4.1	4.3	-3.6	-2.4	-0.4	-4.6	0.7	-61.1	
	(70.2)	(9.4)	(18.7)	(3.7)	(10.8)	(3.4)	(1.8)	(7.8)	(5.2)	(0.9)	(114.3)	
Meal*1 year	-127.8	17.7	0.4	-7.1	-12.2	-4.8	-3.5	-17.1*	¥-21.7***		-104.0	
	(106.4)	(25.3)	(42.3)	(5.3)	(15.6)	(4.4)	(2.5)	(10.1)	(8.0)		(159.4)	
Snack*After	-53.1	-6.5	-15.5	-3.5	0.3	-4.7	-1.3	-2.1	-0.7	-0.6	-120.6	
	(75.1)	(11.1)	(20.8)	(4.1)	(11.6)	(3.4)	(2.1)	(7.3)	(4.7)	(1.1)	(137.6)	
Snack*1 year	-147.4	-40.2**	-27.9	-3.4	-23.9	-6.7	-1.5	-14.8	¥-19.2**		-159.3	
	(99.4)	(17.4)	(34.9)	(4.8)	(14.9)	(4.2)	(2.4)	(10.0)	(8.7)		(173.9)	
Constant	1,417.2***	31.7***	87.1***	56.7***	187.4***	49.4***	24.5***	92.3***	20.8***	11.0***	1,565.4***	
	(18.8)	(2.7)	(5.6)	(1.0)	(2.8)	(0.8)	(0.5)	(2.0)	(1.5)	(0.2)	(31.0)	
# Obs.	804	675	738	804	804	804	804	804	802	560	804	
R-squared	0.03	0.13	0.12	0.02	0.07	0.00	0.03	0.07	0.41	0.00	0.02	
# of ind.	292	292	290	292	292	292	292	292	291	289	292	

Panel B: Adults												
Sign of the UK recommendation-baseline consumption	+	+	+	-	+	-	-	-	-	+	+	-
After	-250.9***	-15.1	-28.8*	-9.5*	-32.3***	-6.9	-2.5	-20.6***	-10.7*	-2.0**	-271.3*	-2.2
	(86.6)	(9.4)	(15.5)	(5.0)	(10.4)	(6.1)	(2.1)	(7.6)	(5.6)	(0.8)	(153.5)	(1.6)
1 year follow-up	-422.9***	68.5***	126.5***	-26.6***	-20.2	-22.6***	-8.0***	-0.3	39.2***		-449.1**	31.3**
	(109.6)	(21.5)	(24.4)	(5.4)	(14.3)	(6.5)	(2.6)	(10.2)	(8.4)		(181.2)	(12.4)
Meal*After	88.4	-2.5	-4.4	3.0	11.6	2.0	-1.2	3.4	-0.7	1.3	-11.0	1.0
	(117.1)	(15.5)	(19.7)	(6.4)	(15.0)	(7.4)	(2.8)	(9.6)	(7.2)	(1.2)	(216.2)	(2.3)
Meal*1 year	314.3*	-32.3	10.8	13.7	29.3	15.8*	1.6	5.6	-4.1		235.7	-7.5
	(186.3)	(30.0)	(36.0)	(8.4)	(23.9)	(9.0)	(3.8)	(12.7)	(9.9)		(302.8)	(14.7)
Snack*After	-177.5	-20.4	-12.4	-7.5	-24.5	-3.7	-3.8	-23.2	-10.5	-0.8	-367.9	-0.5
	(133.2)	(18.6)	(43.0)	(7.2)	(19.1)	(7.3)	(3.1)	(14.2)	(9.6)	(1.3)	(274.7)	(3.7)
Snack*1 year	78.8	-17.0	49.0	3.4	-4.0	¥21.1**	1.5	-18.1	-15.8		-177.7	-16.1
	(165.8)	(37.5)	(65.7)	(8.1)	(24.2)	(8.4)	(3.6)	(16.8)	(12.9)		(325.0)	(15.6)
Constant	1,963.1***	70.8***	80.1***	80.2***	236.6***	73.9***	29.5***	105.8***	34.2***	14.2***	2,304.4***	7.5***
	(35.7)	(4.3)	(6.8)	(1.7)	(4.8)	(1.9)	(0.7)	(3.0)	(2.2)	(0.2)	(64.0)	(0.7)
# Obs.	926	781	783	925	926	926	925	926	921	628	926	677
R-squared	0.06	0.09	0.23	0.08	0.04	0.05	0.05	0.05	0.22	0.05	0.04	0.18
# of ind.	359	347	347	358	359	359	358	359	359	338	359	339

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 (Intke24) used in the 1 year follow up surveys. ¥ indicates that the sign and significance of the coefficient is the same in the DID analysis on macronutrient percentage changes reported in Appendix B, table B.10.

Table 9: Descriptive statistics of body measurements

	(1) Control	(2) Meal	(3) Snack	P-value (1)=(2)	P-value (1)=(3)
Children					
% Underweight	3.9	3.1	4.6		
% Normal weight	71.8	78.6	78.5		
% Overweight	18.5	12.2	13.8	0.99	0.98
% Obese	5.8	6.1	3.1		
# Obs	103	98	65		
Adults (main & second)					
Mean BMI Men	27.9	27.6	28.0	0.71	0.76
	(4.8)	(5.2)	(4.5)		
Mean BMI Women	29.5	27.8	27.0	0.14	0.04
	(7.5)	(6.6)	(6.3)		
% Underweight (BMI < 18)	0.7	1.5	2.2		
% Normal weight (BMI 18-25)	29.3	38.6	38.0		
% Overweight (BMI > 25)	32.7	28.1	32.6	0.65	0.65
% Obese (BMI > 30)	37.3	31.8	27.2		
# Obs	150	132	92		

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. The categories are based on cut-offs from British 1990 growth reference see page 5 http://www.noo.org.uk/uploads/doc/vid_11601_A_simple_guide_to_classifying_BMI_in_children.pdf
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.

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

	(1) Perc. BMI (children)	(2) Overweight or Obese (children)	(3) BMI (adults)	(4) Overweight or Obese (adults)	(5) Obese (adults)
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	0.00	0.00
# individuals	288	283	380	380	380

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.

Table 11 Number of low-calories choices, incentivized

	(1) 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

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

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

	(1) Normal ranges	(1) Control	(2) Meal	(3) Snack	P-value (1)=(2)	P-value (1)=(3)
Adults (main & second)						
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)	0.4	0.58
Triglyceride (nmol/l)	< 2	1.1(0.9)	1.2(0.9)	0.9 (0.4)	0.7	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		

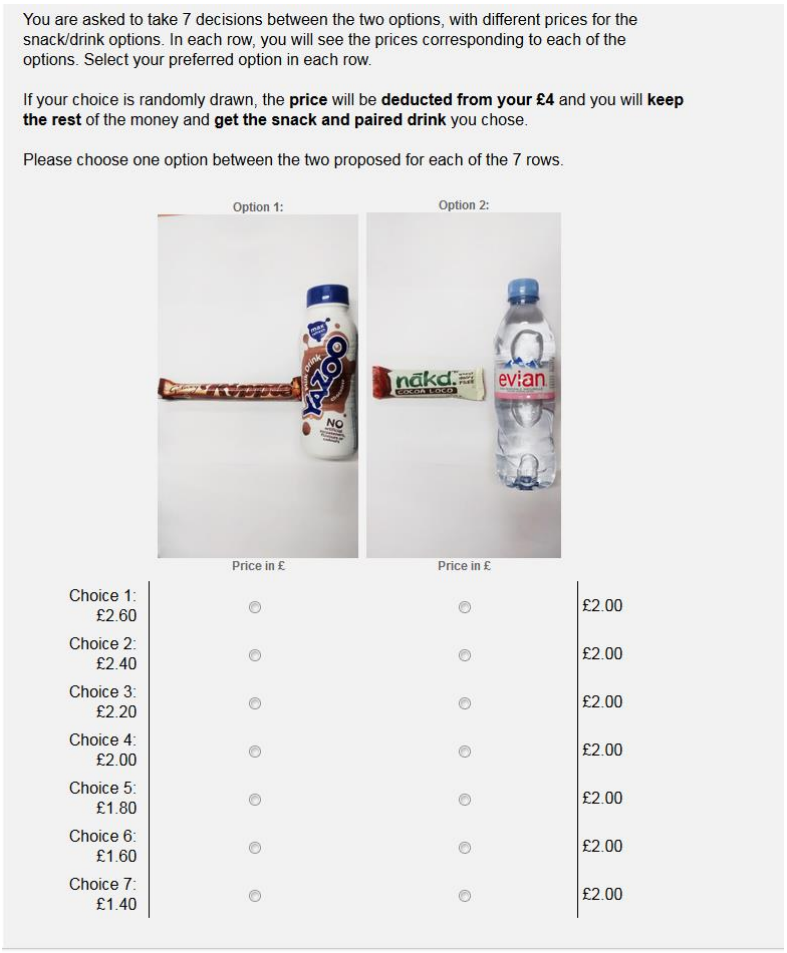
Note: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a t-test of equality of estimated parameters in Col. (1) and (2) and in Col. (1) and (3) respectively. Sample is for adults only in Edinburgh.
[1] Calculated values: Total cholesterol-HDL-(Triglyceride/2.2)

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

	(1) Nefa	(2) Triglyceride	(3) HDL	(4) LDL	(5) Glucose	(6) Insulin	(7) CRP	(8) 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	0.00	0.06	0.08	0.04	0.06	0.04

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.

Figure 1. **Incentivized measures of food choices. In the lab (Colchester sample)**



Note: Source: Computer screenshot. Option 1 is a sample of a high-calorie pair of snack (>200kcal), Option 2 is a sample of a low-calorie pair of snack (<100kcal).